
A Call for Higher Standards of Evidence for Dietary Guidelines

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Abstract: Dietary guidelines, especially those designed to prevent the diseases of dietary excess, are a relatively new phenomenon in the United States. National dietary guidelines have been promulgated based on scientific reasoning and indirect evidence. In general, weak evidentiary support has been accepted as adequate justification for these guidelines. This low standard of evidence is based on several misconceptions, most importantly the belief that such guidelines could not cause harm. Using guidelines against dietary fat as a case in point, an analysis is provided that suggests that harm indeed may have been caused by the widespread dissemination of and adherence to these guidelines, through their contribution to the current epidemic of obesity and overweight in the U.S. An explanation is provided of what may have gone wrong in the development of dietary guidelines, and an alternative and more rigorous standard is proposed for evidentiary support, including the recommendation that when adequate evidence is not available, the best option may be to issue no guideline.

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Many public health recommendations are not truly evidence-based.¹ While some public health decisions can and should be made in the face of inconclusive data, many should not. The need for restraint may be especially salient when considering dietary guidelines. Dietary fat recommendations are a case in point, as they may have led to significant and harmful unintended consequences.

In this article, the history of and evidence for dietary recommendations is reviewed, the potential is demonstrated for unanticipated negative impacts on health outcomes, an explanatory model is provided, and a mechanism is suggested to limit the potential for harm through dietary guidelines.

A Brief History of Dietary Guidelines for Fat Intake

National dietary guidelines were introduced in 1894. Initially designed to prevent dietary deficiency, the goals were modified in the late 1970s to include recommendations designed to reduce the burden of cardiovascular disease (CVD).² This made sense because, first, heart attacks, strokes, and renal disease had become the leading causes of morbidity and mortality in the United States, and second, the importance of CVD risk factors, including hyperlipidemia, had been firmly es-

tablished. Since the relationship of lipids to disease was continuous, it made sense to attempt a population-wide downward shift in adverse lipid profiles despite the lack of direct evidence regarding either the ability to accomplish this shift or its cardiovascular and overall effects. This population-based approach was especially attractive since it was presumed to carry little risk. Indeed, enthusiasm was increased by the idea that a low-fat diet also could reduce obesity, as evidenced by statements like: “Populations like ours with diets high in fat have more obesity.”³ These statements were supported by ecologic data that showed a correlation between percentage of energy from fat and obesity across different countries.⁴

By Congressional mandate, the U.S. Department of Agriculture (USDA) and U.S. Department of Health and Human Services (USDHHS) issue revised guidelines every 5 years. The 1980 and 1985 dietary guidelines recommended reduced intake of all fats. In 1990, a recommendation was added that <30% of total caloric intake should come from fat. Each of these reports, however, included important qualifying statements reflecting substantial scientific uncertainty, such as: “There is controversy about what recommendations are appropriate for healthy Americans.”^{5,6}

The 1995 edition, however, abandoned this cautious approach. Noting that these dietary guidelines were intended for “readers with different levels of scientific sophistication,” the Dietary Guidelines Advisory Committee decided in 1995 to remove qualifying words, such as “the probably’s, whereas’s, could’s and might’s,” [sic] to make the messages “clear and unambiguous.” Thus, the

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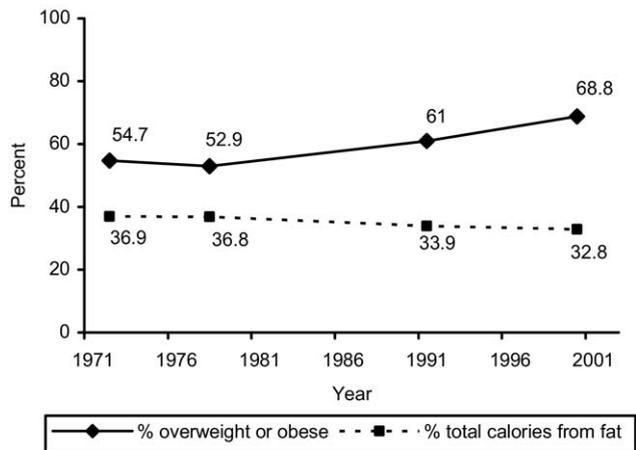


Figure 1. Trends over time for fat consumption and overweight in the U.S. population: men.¹¹

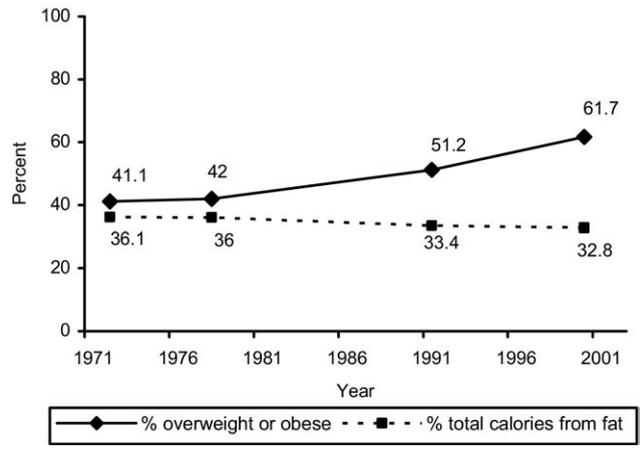


Figure 2. Trends over time for fat consumption and overweight in the U.S. population: women.¹¹

Committee wrote more confidently that “the Guidelines accurately reflect current knowledge” and that “the advice is scientifically sound as well as prudent and practical.”⁷ No new scientific evidence linking fat intake and obesity was cited to justify these changes.

The 1995 edition did begin to recognize the disconnect between population-based trends in heart disease versus obesity and their relationship to fat intake: “More Americans are now eating less fat, saturated fat, and cholesterol-rich foods than in the recent past, and fewer people are dying from the most common form of heart disease. Still, many people continue to eat high-fat diets, and the number of overweight people has increased.”⁷

Then, in 2000, the Dietary Guideline Advisory Committee reversed itself and suggested that the recommendation to lower fat intake had perhaps been ill-advised and might actually have some potential for harm: “The committee further held the concern that the previous priority given to a “low-fat intake” may lead people to believe that, as long as fat intake is low, the diet will be entirely healthful. This belief could engender an overconsumption of total calories in the form of carbohydrates, resulting in the adverse metabolic consequences of high-carbohydrate diets. Further, the possibility that overconsumption of carbohydrates may contribute to obesity cannot be ignored. The committee noted reports that an increasing prevalence of obesity in the United States has corresponded roughly with an absolute increase in carbohydrate consumption.”⁸

Health Trends

As these dietary fat guidelines were promulgated from 1980 to 2000, cardiovascular mortality rates continued their established decline in the U.S.⁹ A Cochrane Collaboration meta-analysis, published in

2001, showed that low-fat diets had a marginally (and nonsignificantly) favorable effect on CVD mortality in randomized trials,¹⁰ consistent with the notion that if a population-wide reduction in dietary fat had occurred, it might have contributed to this decline in mortality. Of course, this decline had begun in the 1960s, prior to the dietary guidelines,⁹ and other clinical interventions (e.g., statins for hypercholesterolemia, bypass surgery, and angioplasty) also had contributed. Moreover, this favorable trend in coronary heart disease (CHD) was counterbalanced by an alarming increase in obesity¹¹ and attendant diabetes¹² that coincided with the promulgation of the 1980 dietary guidelines (Figures 1 and 2).

How might this disconnect—increasing rates of obesity as relative fat consumption declined—have occurred? As Figures 3 through 6 demonstrate, the recommendation to reduce relative fat intake produced mixed results. The major contributor to reductions in the percent of calories from fat was not a reduction in the numerator (absolute fat intake), but an increase in the denominator (total caloric intake). For instance, absolute fat intake decreased by only 5% in men, while relative fat intake declined by 11%. In women, the difference was even starker: absolute fat intake actually increased by 11% while relative fat intake declined by 9%. The increase in total calories reflected increased carbohydrate consumption despite relatively stable or declining absolute fat consumption.¹¹ This may have been influenced by the effective marketing of low-fat foods, as well as the food pyramid, which suggested that low-fat foods could be eaten without any concern.¹³ Of course, the trends in marketing low-fat foods were not driven exclusively by the changes to the U.S. Dietary Guidelines; our concern is that the Guidelines may have provided an apparent governmental “seal of approval” for products

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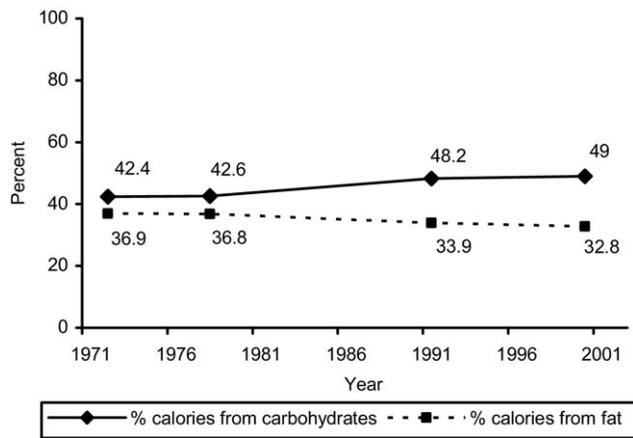


Figure 3. Dietary trends over time in U.S. men: percent.¹¹

such as fat-free cookies, which may have enhanced the appeal of these products. People also may eat larger amounts of low-fat foods; some suggest that fat intake is related to satiety, providing a biologically plausible rationale that the recommendation to choose lower-fat foods could have led to increased energy consumption.¹⁴

Whatever the mechanism, it is clear that as dietary fat-reduction recommendations were promulgated, relative intake of fat was reduced, consistent with the guidelines; however, total calorie (and carbohydrate) consumption increased, absolute fat intake did not decrease substantially, and an epidemic of obesity began in the U.S. This temporal association does not prove causation, but raises the possibility of a net harmful effect of seemingly innocuous dietary advice.

What Went Wrong?

The dietary fat story underscores several weaknesses in public health decision making. The following attempts to explain where current thinking might go wrong.

Prevention Through the “Population Strategy” Is a Two-Edged Sword

The late British epidemiologist Sir Geoffrey Rose argued that the clinical approach to prevention—the high-risk strategy—is effective, but costly and inefficient.¹⁵ Most heart attacks occur in that majority of individuals who are at average or low risk based on any given risk factor; indeed, about half of those suffering a coronary event have no established risk factors.¹⁶ Thus, Rose argued for a population strategy where even a small positive shift in a risk factor, when equally distributed across the entire population, could have an extremely large total effect.¹⁷ An example of this strategy is universal restriction of sodium intake, justified inferentially: (1) the higher the blood pressure, the greater the risk for CVD; (2) higher sodium intake is associated with higher blood pressure; (3) reduced sodium intake

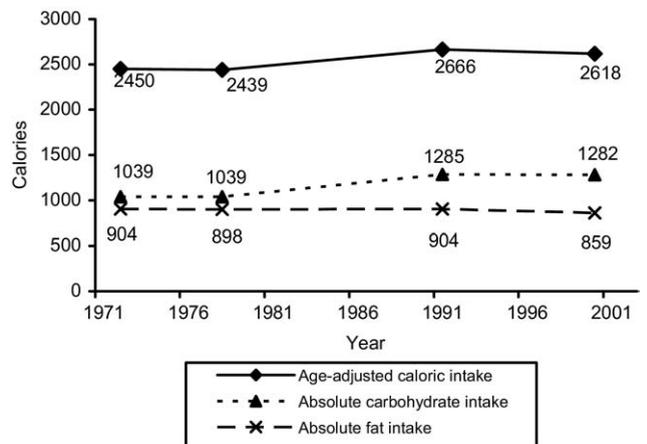


Figure 4. Dietary trends over time in U.S. men: absolute intake.¹¹

can lower blood pressure; (4) lowering blood pressure with medications reduces CVD risk; therefore (5) reducing sodium intake will reduce CVD risk.

The argument is logical, notwithstanding the absence of direct evidence supporting the conclusion. In fact, there is evidence that sodium restriction may not lead to improved health outcomes.¹⁸ Nevertheless, most dietary guidelines recommend salt restriction, presumably because their authors have discounted the possibility of harm. Quoting again from Professor Rose:

The level of evidence appropriate to a particular decision depends on the consequences of making the wrong decision. For example, there is substantial evidence, but still well short of proof, that a reduction in national salt consumption leads to a somewhat lower mean blood pressure, with important expected health benefits. The change is safe and its cost minimal (except to a small but noisy section of the business community). The evidence for this policy is imperfect, but one may judge it to be sufficient.¹⁹

Despite all the caveats about the limitations of the evidence, dietary recommendations apparently fall below the bar at which concern for potential harm emerges. However, as has been shown, there is potential for harm in seemingly innocuous “lifestyle recommendations.” Dietary fat recommendations may have generated harm in the form of obesity that outweighs any benefit in serum cholesterol, and there is a plausible mechanism (fat intake is related to satiety). Potential harm from sodium restriction also has been demonstrated,²⁰ perhaps because a blood pressure benefit may be trumped by harmful effects on plasma renin, insulin resistance, sympathetic nerve activity, and aldosterone levels.²¹ A recent follow-up subgroup analysis of an earlier randomized clinical trial detected an apparent benefit among those randomized to a low-sodium diet; however, more compelling was the finding of no

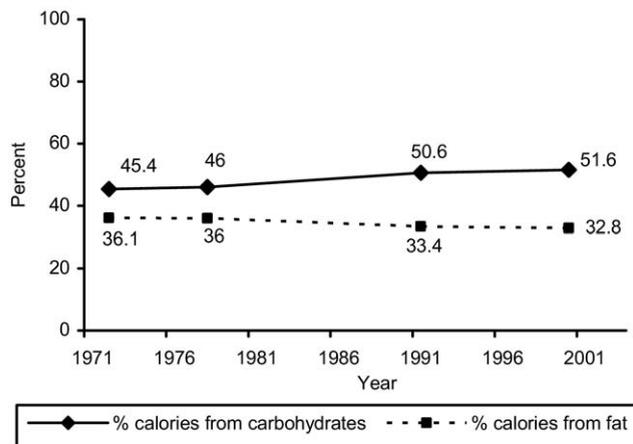


Figure 5. Dietary trends over time in U.S. women: percent.¹¹

difference in total mortality between the total randomized groups.²² Thus, the value of sodium reduction remains an open question.

Rose did caution that: “In mass prevention each individual has usually only a small expectation of benefit, and this small benefit can easily be outweighed by a small risk.”¹⁷ Potential hazards are the inverse of Rose’s population strategy of prevention; while a tiny, immeasurable improvement in individual risk can have a dramatic population benefit, substantial harm can be observed at a population level as the sum of tiny, immeasurable harms to individuals.

Although there is no proof that recommendations to decrease dietary fat directly led to obesity, the data supporting this inference are similar to those used for 20 years to justify a low-fat diet.⁴ These dietary recommendations did not necessarily cause harm; but rather, there is a realistic possibility that they may have. The hypothesis that dietary fat admonitions actually caused the current U.S. obesity epidemic is consistent with the data, logically sound, and plausible on the basis of both behavioral (e.g., food marketing and net dietary change) and biological (e.g., fat intake and satiety) mechanisms.

Distinguishing “The Public” from “Patients”

The first edition of *Nutrition and Your Health: Dietary Guidelines for Americans* claimed that such the pronouncements were created “in response to the public’s desire for authoritative, consistent guidelines on diet and health.”²³ It is not clear how this desire was assessed. When a patient seeks advice from a physician, it is the patient who asks; when the government sets standards, no one is asking. Such standard setting is often defended by the assertion that “people want to know,” but the public health community is believed to have created this market by its propensity for issuing guidelines. Indeed, the notion that the government should tell people what and how much to eat is inherently paternalistic, and is a relatively recent phenomenon.

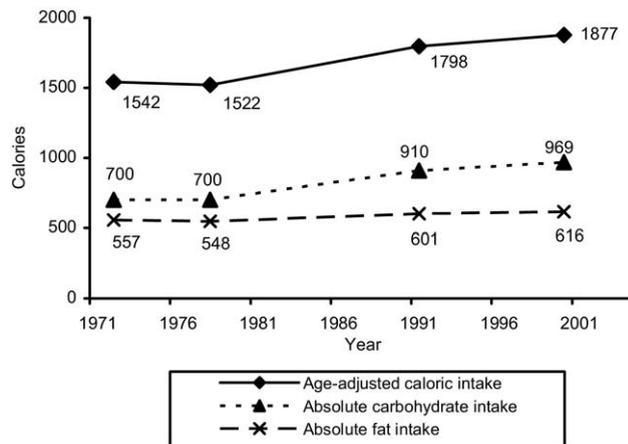


Figure 6. Dietary trends over time in U.S. women: absolute intake.¹¹

Public health guidelines are one-size-fits-all pronouncements that fail to account for variations in genetics, behavior, and environment. A physician can weigh such factors and make individual adjustments; dietary guidelines cannot.

A Risk Factor Is Not Necessarily a Cause

The guideline to reduce fat intake began with a focus on the CVD risk associated with hypercholesterolemia. Risk factors are not direct causes of disease; not everyone with hypercholesterolemia has a heart attack, and not every heart attack occurs in a patient with hypercholesterolemia. Some drugs that reduce serum cholesterol, like statins, reduce mortality²⁴; some, like clofibrate, increase mortality.²⁵ Thus, appreciation of an association between diet and cholesterol does not indicate that dietary modification will invariably improve health. Hypercholesterolemia is not a disease; it neither causes symptoms nor affects quality of life. It is only important because it is known, on average, to portend a greater risk of disease and death from cardiovascular (and perhaps other) diseases. So the important question is not whether dietary changes can improve risk factors; it is: Will these dietary changes reduce disease outcomes? In the case of governmental guidelines against fat, such a direct link between diet and disease outcome was (and is) lacking.

A Change in a Specific Risk Factor, and a Beneficial Effect on a Single Outcome, Does Not Establish the Net Health Effect of an Intervention

The dietary fat observations above underscore the limitations of focusing on any single intermediate or proxy measure of an intervention. A diet changed to reduce cholesterol may have other effects. Even if a dietary intervention could reduce serum cholesterol, hypercholesterolemia is merely one among many pos-

Table 1. Proposed “strength of recommendation” grid for dietary guidelines (after U.S. Preventive Services Task Force²⁸)

Quality of evidence	Net benefit			
	Substantial	Moderate	Small	Zero/negative
Good	A ^a	B	C	D
Fair	B	B	C	D
Poor	I	I	I	I

^aSuggested language for dietary guidelines:

A=The ___ [organization promulgating the guideline] strongly recommends that all Americans carefully follow these guidelines. (The ___ found good evidence that following these guidelines will improve important health outcomes and concludes that benefits substantially outweigh harms.)

B=The ___ recommends that Americans follow these guidelines. (The ___ found at least fair evidence that these guidelines will improve important health outcomes and concludes that benefits outweigh harms.)

C=There is substantial debate regarding these guidelines. (The ___ found at least fair evidence that these guidelines may improve health outcomes but concludes that the balance of the benefits and harms is too close to justify a general recommendation.)

D or I=No recommendation.

sible intermediate endpoints. The sum of multiple intermediate effects determines the health outcome of an intervention. In the case of an effort to eat less fat, there is the possibility that something else may be lost, and the substitute may be even worse. A changed diet may reduce an individual’s serum cholesterol (or the average serum cholesterol in the population) at the expense of increasing his weight (or the average weight in the population). It is the sum of all these changes that produces the intervention’s net health effects. Logic and good intentions are not a substitute for solid evidence of the totality of the effects of any public health intervention.

Inaction Is Sometimes the Best Option

It is certainly reasonable to use a combination of public health and individually-based (prevention and treatment) efforts to reduce CVD morbidity and mortality. Happily, both morbidity and mortality from CVD have been declining steadily over the last 40 years,⁹ and Americans are living longer.²⁶ Declining (less than “expected”) rates undermine any notion of a public health emergency, where action can be justified in the face of inconclusive evidence. In the case of CVD, any wholesale recommendation for a change so central to our lifestyle should be made only on solid evidence of efficacy and safety. *Primum non nocere* applies to public health, too. Given the potential impact of even minimal harm, the lack of any specific request for information, and the unpredictable effects on multiple intermediate outcomes, the alternative is simple: Do not issue a dietary guideline without sufficient evidence.

What Can Be Done?

Dietary guidelines need explicit standards of evidence. Rating scales, such as those used by the U.S. Preventive Services Task Force (USPSTF) and other bodies, have been reviewed recently.²⁷ Rather than bury the complexity and uncertainty that may underlie dietary recommendations in order to make the message clear and

unambiguous,⁷ guidelines should always include a clear and thoughtful assessment and disclosure of the evidence.

For dietary guidelines, the strengths and limitations of evidence should be provided in easy-to-understand language. This may lead to guidelines that are laden with caveats and disclaimers, but this is preferable to resolute guidelines supported by equivocal evidence. When the evidence is murky, public health officials may be served best by exercising restraint, reflected by making no recommendation at all.

To achieve parity with the thoughtful guidelines of the USPSTF,²⁸ an adaptation is suggested of their evidence ratings (Table 1), which consider both the quality of the evidence and the net benefit of an intervention (net benefit recognizes the potential for harm). Their proposed “standard recommendation language” is adapted further to dietary guidelines (Table 1), including a broader category for which no recommendation should be made. This revised language, and the proposed higher standard of evidence, is intended to provide the appropriate level of caution and humility in the process of issuing guidelines.

The Current War on Trans Fat

Unfortunately, neither humility nor caution characterizes much of the current public health approach to diet. The campaign against trans fat is a case in point. Although there is good evidence linking dietary trans fat and CVD,²⁹ the situation is analogous to that for dietary fat guidelines: (1) trans fat intake has been associated with CVD (which is already in decline), but **not** with obesity (which is an epidemic); (2) trans fats will be replaced by something (unclear what), and the net effect on health is not known; and (3) marketing of trans fat-free foods will affect dietary behavior in unpredictable ways (e.g., it has been reported that Katz’s Delicatessen in New York City has been “trans fat free” for years³⁰; Doritos, Kentucky Fried Chicken, and

other high-calorie foods are marketed as “0 grams trans fat”).

As the vilification of dietary trans fat continues, it should not be forgotten that its consumption increased dramatically in the U.S. as margarine was promoted as the “healthy” alternative to butter, since butter was “known” to be a dangerous source of saturated fats. Indeed, margarine has been independently associated with CHD risk^{31,32} and remains the single largest source of dietary trans fat.³³ In the case of trans fat, public health officials are not exercising caution; in fact, legal restrictions have been established in Denmark and in New York City.

The public health issue is not whether there are adverse health effects of dietary trans fats (for which the evidence is good), but rather what would be the health result of eliminating these agents (for which evidence is lacking)? There is no direct evidence that regulatory limits on trans fat in restaurants will improve health. Even accepting that replacing dietary trans fats with alternative fats likely will reduce coronary risk, the net effect of legal restrictions is unknown. Since the current public health crisis is overweight and obesity, it is particularly important to note that no one suggests that replacing trans fat will lead to weight reduction. Indeed, a recent review suggested that “aggressive marketing of products that contain ‘0 grams trans fat’ leads consumers to think that they can eat them with impunity.”³⁴ Thus, this well-meaning effort may lead to improvement in CHD risk (or not) or increase in obesity rates (or not). Certainly, the net effect of this intervention cannot be known a priori.

Conclusion

Ironically, it now seems that the U.S. dietary guidelines recommending fat restriction might have worsened rather than helped the obesity epidemic and, by so doing, possibly laid the groundwork for a future increase in CVD. Unfortunately, when public health officials recognized this possibility, they did not cease issuing guidelines; they issued new ones. The new recommendation was: “choose a diet low in saturated fat and cholesterol, and a diet moderate in fat.”³⁵ In addition, the food pyramid, which had strongly suggested that dietary fat was the culprit and that carbohydrates were healthful, was changed (see www.mypyramid.gov/). The new food pyramid has at least one major advantage over the old: the old pyramid, with its horizontal bars, with carbohydrates at the base, gave a clear and unambiguous message that carbohydrates should make up the bulk of the diet; the new pyramid, with its vertical bars and colors instead of images, is ambiguous in the weighting of “acceptability” of various dietary components. In this case, where high-quality evidence is missing, a lack of clarity is the preferable alternative.

The data provided and arguments made throughout this paper are not intended to suggest that fat is “good for you.” Indeed, it is accepted that extensive evidence suggests such truths as: increased dietary fat, especially saturated fat, leads to elevation of serum cholesterol, which is in turn a risk factor for CHD³⁶; lowering dietary fat can reduce serum cholesterol³⁷; dietary trans fats have a particularly deleterious effect on serum lipids.²⁹ What is argued, however, is that knowledge of the untoward effects of particular dietary components, and the potential benefit of individual-level dietary modification, does not yield direct knowledge of the net population effect of a dietary guideline. Guidelines may have unpredicted effects, through the complexities of public perception, marketing, and behavioral changes. Since some changes may be beneficial and some may be detrimental, the net effect of these changes may be either harmful or helpful; the outcome can not be predicted in advance.

In sum, dietary guidelines aimed at the U.S. population traditionally been issued without pre-specified and transparent evidentiary support for disease outcomes across all population groups. Such guidelines could have unanticipated and adverse effects. To avoid this possibility in the future, specific and transparent classification of the quality of the evidence should attend guideline development and promulgation. Public health, just like personal-encounter medicine, should be guided by the dictum “first, do no harm.” Sometimes, in the absence of clear and convincing evidence of net benefit, that will mean: do not issue dietary guidelines at all.

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