Editorials

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High Fiber Intake

Indicator of a Healthy Lifestyle

In this issue of *JAMA*, Rimm and coworkers[1] at the Harvard School of Public Health report that "fiber, independent of fat intake, is an important dietary component for the prevention of coronary disease." Their conclusion is based on observations within a cohort of more than 40 000 male health professionals followed up for 6 years to an end point of either myocardial infarction (MI) or coronary death. If correct, the finding is important because it could provide the basis for nutritional advice that will affect many groups in our society, including average Americans, practicing physicians, and the food industry, among others. Some might even interpret it as justification for advising Americans to eat more high-fiber food without necessarily making other dietary modifications.

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Their conclusion all but implies a causal relationship and invites a low-fiber diet to take its place alongside cigarette smoking, high serum cholesterol, and high blood pressure as an independent risk factor for coronary disease. Therefore, while the health benefits of fiber are indisputable, a few words of caution may be in order regarding the interpretation of these results.

First, the investigation, while meticulously conducted, is an observational study and not a clinical trial. Judgment of causality rests not only on the difficult questions of precision and accuracy that affect questionnaire-based dietary assessment, but also on possible problems of population selection, statistical methodology, and biological plausibility. Furthermore, the accuracy of nutritional histories has been much debated for both cohort and retrospective studies. The fact that in this study the data were obtained from health professionals also raises the possibility that generally acknowledged healthy choices like fiber may have been systematically overreported while socially undesirable choices like fat may have been underreported. Even if such biases affected all quintiles similarly, they would tend to undermine the reliability of quantitative dose responses such as the conclusion that an increment in total dietary fiber of 10 g/d is associated with a specific risk decrement.

Second, the statistically significant association between fiber intake and coronary risk was confined to men in the highest quintile. These men were characterized by extreme levels of a constellation of correlated behaviors that affected the entire cohort and might be collectively termed a "healthy lifestyle effect." For instance, only 3.8% of men in the fifth quintile were cigarette smokers, compared with 19.7% in the first quintile and with 29.2% of men aged 45 to 64 years in a nationally representative sample.[2] In addition, median fiber intake by men in the fifth quintile was 28.9 g/d, compared with 12.4 in the first quintile and with a national mean of about 13.0 g/d for white men aged 55 to 64 years.[3] Even within this cohort of health professionals, the high-fiber quintile exhibited extreme levels of a variety of low-risk behavior gradients, as the Table shows.

The Association Between Fiber Intake and Coronary Risk Factors

Risk Factor	Dietary Fiber, Quintile 1†	2	3	4	5
Odds Ratio for Association of Fiber Intake With Risk Factor					
Use of vitamin E supplements	1.00	1.14	1.30	1.69	2.26
Current smokers	1.00	0.55	0.36	0.26	0.16
Value of Risk Factor in Highest Quintile Divided by Value in Lowest Quintile					
Physical activity, METs*/wk	1.00	1.03	1.11	1.21	1.42
Total fat intake, g/d	1.00	1.02	0.99	0.93	0.79
Saturated fat intake, g/d	1.00	0.98	0.93	0.84	0.69
Dietary cholesterol intake, mg/d	1.00	0.99	0.93	0.87	0.74

*METs indicates metabolic equivalents.
†Reference group.

These observations, along with a statistically significant reduction in risk of coronary death or nonfatal MI confined to the fifth quintile and absence of a trend in decreasing risk with increasing fiber, argue that the highest fiber quintile contains a high proportion of men with a group of unusually healthy behaviors that account for a low coronary risk quite apart from fiber intake. Although the authors do not report how long their subjects consumed diets with the indicated fiber levels, we would not be surprised to learn that many of these men have previously undergone major lifestyle changes in an effort to control a previously diagnosed coronary risk factor such as high cholesterol, which affected a higher proportion of men in the fifth quintile. The extraordinarily low prevalence of current smoking might also signal a pattern of behavioral changes, especially since smoking cessation is strongly protective against early fatal MI, which might have contributed to their significantly lower fatal coronary rate.

Third, subjects were ranked by dietary fiber intake values that had been individually adjusted for total energy intake. This adjustment tends to confound the effect of fiber with fat intake, inasmuch as fat is usually positively correlated with total energy and both total energy and fat are inversely associated with fiber. This might explain why only energy-adjusted measures of fiber intake showed a protective effect, and it further weakens the conclusion that risk reduction was independent of dietary fat intake.

Finally, the authors used a multivariate model containing nine discrete and two continuous covariates to adjust for possible confounding by other risk factors. Because the entire number of nonfatal MIs observed in the high-fiber quintile

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was only 83, it is possible that the main finding may be driven by a small number of highly unusual individuals. In any event, statistical adjustment cannot eliminate problems of selection bias, particularly since lifestyle factors are likely to have a synergistic effect on health.

These limitations underscore the need for further studies before dietary fiber as a protective factor against coronary disease independent of fat can be said to have been established. While Rimm et al argue that their data do not support the hypothesis suggested by others[4] that fiber may lower the risk of coronary disease simply by displacing dietary fat, we believe this point is far from settled and would support a coronary intervention trial, especially if it included a short-term end point of lowered serum cholesterol. In fact, a recent clinical trial showed that the effect of fiber on chloesterol level was probably attributable to the concurrent reduction in fat intake.[4]

Studies of mechanisms must also be included in any research program. Cold cereal, which constituted a major source of dietary fiber for the cohort, contains largely insoluble fiber, whereas it is largely soluble fiber that has been shown to have a cholesterol-lowering effect.[5] The way in which insoluble fiber might lower cardiovascular risk, other than simply by replacing fat in the diet, is still unknown. Since adults who regularly consume ready-to-eat cereal at breakfast have on average lower daily intakes of fat and cholesterol,[6] it is conceivable that in this study the behavior of simply eating breakfast cereals may be a hallmark of a healthy lifestyle. Eating breakfast can be beneficial because it may set the nutritional "thermostat" for the day[7] and is likely to represent the first of at least three daily meals. Resnicow[7] reported that skipping breakfast was associated with high cholesterol in children, partly because they made up during the day with more high-fat meals, and Jenkins[8] has shown that "nibbling" or "grazing" rather than "gorging" can itself reduce cholesterol levels and, therefore, lower the risk of MI.

While we do not agree that fiber alone protects against heart disease, we join Rimm and his associates in recommending fiber as a "component for the prevention of coronary disease," provided that dietary fat is also considered. We have called our basic recommendation "25/25" (ie, 25 g of fiber per day in a diet that derives no more than 25% of its energy from fat).[9] We strongly support a multifaceted approach to achieving such a diet. We further advocate that this be a lifelong behavior, beginning in early childhood, as proposed by Williams[10] using the formula "age plus 5," meaning that children, beginning about age 2 years, should consume an amount of dietary fiber equal to their age (in years) plus 5 (g/d), which will result in a daily fiber consumption of 25 g/d by age 20 years.

We urge that fiber in the form of breakfast cereal, fruits, and vegetables be routinely recommended by physicians for their patients, by teachers for their students, and by parents for themselves and their children. With a proper strategy by public health leaders and the medical profession and with the cooperation of the food industry, this can be one component in a program to reduce nutrition-related morbidity and mortality.

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Preventing Multidrug-Resistant Tuberculosis

In the 5 years since the first major outbreaks of multidrug-resistant tuberculosis (MDR TB) in the United States were reported, the epidemiology of MDR TB has become clearer.[1,2] Clinically, the course of illness can be devastating for both human immunodeficiency virus (HIV)–infected and noninfected persons, especially when diagnosis and adequate therapy are delayed.[1,3] However, prompt diagnosis and appropriate therapy can improve the likelihood of a successful outcome.[4-6] Directly observed therapy (DOT) has increasingly become the standard of care[7] and has been accompanied by dramatic reductions in morbidity and drug resistance, including MDR TB.[8,9] Evidence supports DOT as the adherence-promoting strategy of choice par excellence.

Reflecting an improvement in control efforts is the increase in the proportion of patients completing therapy. In 1993, 82% of patients in the United States completed therapy within 12 months, compared with 77% in 1992 (Centers for Disease Control and Prevention [CDC], unpublished data, 1993). This is the first significant increase in completion rates since short-course chemotherapy was recommended in the mid 1980s.

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After years of neglect, TB research has finally entered the molecular age with a growing arsenal of diagnostic techniques. Several DNA fingerprinting methods serve as powerful tools for epidemiologic investigation and have been used to suggest that recent transmission of *Mycobacterium tuberculosis* gives rise to a larger proportion of total morbidity than previously estimated.[10,11] It is now possible to identify clinically significant mutations in the tubercle bacillus responsible for drug resistance down to the level of a single nucleotide. The study by Mr Bifani and colleagues[12] in this issue of THE JOURNAL demonstrates how identifying mutations of the tubercle bacillus facilitates the study of *M tuberculosis* transmission and spread. It

This article is one of a series addressing emerging and reemerging global microbial threats.

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