of General Internal Medicine), Deborah S. Main, PhD (Department of Family Medicine), Frederick A. Masoudi, MD, MSPH (Division of Cardiology, Denver Health Medical Center), and John S. Rumsfeld, MD, PhD (Section of Cardiology, Denver VA Medical Center).

REFERENCES

1. Elwyn GJ. So many precious stories: a reflective narrative of patient based medicine in a general practice, Christmas 1996. *BMJ*. 1997;315:1659-1663.
2. Mitchell WJT, ed. *On Narrative*. Chicago, Ill: University of Chicago Press; 1981.
3. Bruner J. *Making Stories*. Cambridge, Mass: Harvard University Press; 2002.
4. Kleinman A. *Writing at the Margin: Discourse Between Anthropology and Medicine*. Berkeley: University of California Press; 1995.
5. Kleinman A. *The Illness Narratives*. New York, NY: Basic Books; 1988.
6. Charon R. Narrative and medicine. *N Engl J Med*. 2004;350:862-864.
7. Smith BH. Narrative versions, narrative theories. In: Mitchell WJT, ed. *On Narrative*. Chicago, Ill: University of Chicago Press; 1981:209-232.
8. Scholes R. Language, narrative and anti-narrative. In: Mitchell WJT, ed. *On Narrative*. Chicago, Ill: University of Chicago Press; 1981:200-208.
9. Hamm RM, Hicks RJ, Bemben DA. Antibiotics and respiratory infections: are patients more satisfied when expectations are met? *J Fam Pract*. 1996;43:56-62.
10. Greenhalgh T. Narrative based medicine in an evidence based world. *BMJ*. 1999;318:323-325.
11. Sackett DL. Bias in analytic research. *J Chronic Dis*. 1979;32:51-63.
12. Vandenbroucke JP. In defense of case reports and case series. *Ann Intern Med*. 2001;134:330-334.
13. Woo EJ, Ball R, Bostrom A, et al. Vaccine risk perception among reporters of autism after vaccination: Vaccine Adverse Event Reporting System, 1990-2001. *Am J Public Health*. 2004;94:990-995.
14. Spertus JA, Jones P, McDonell M, Fan V, Fihn SD. Health status predicts long-term outcome in outpatients with coronary disease. *Circulation*. 2002;106:43-49.
15. DeSalvo KB, Fan VS, McDonell MB, Fihn SD. Predicting mortality and healthcare utilization with a single question. *Health Serv Res*. 2005;40:1234-1246.
16. Giacomini MK, Cook DJ. Users' guides to the medical literature, XXIII: qualitative research in health care, A: are the results of the study valid? *JAMA*. 2000;284:357-362.
17. Inui TS, Frankel RM. Evaluating the quality of qualitative research. *J Gen Intern Med*. 1991;6:485-486.
18. Mays N, Pope C. Rigour and qualitative research. *BMJ*. 1995;311:109-112.
19. Mullan F. Me and the system: the personal essay and health policy. *Health Aff (Millwood)*. 1999;18:118-124.
20. McDonough JE. Using and misusing anecdote in policy making. *Health Aff (Millwood)*. 2001;20:207-212.
21. Bordiga E, Nisbett RE. The differential impact of abstract vs. concrete information on decisions. *J Appl Soc Psychol*. 1977;7:258-271.
22. Tversky A, Kahneman D. Judgement under uncertainty: heuristics and biases. *Science*. 1974;185:1124-1131.
23. Newman TB. The power of stories over statistics. *BMJ*. 2003;327:1424-1427.
24. Madsen KM, Hvid A, Vestergaard M, et al. A population-based study of measles, mumps, and rubella vaccination and autism. *N Engl J Med*. 2002;347:1477-1482.
25. Sharf BF. Out of the closet and into the legislature: breast cancer stories. *Health Aff (Millwood)*. 2001;20:213-218.

EDITORIALS

Editorials represent the opinions of the authors and *JAMA* and not those of the American Medical Association.

Dietary Fiber and Colorectal Cancer
An Ongoing Saga

John A. Baron, MD

DIETARY FIBER HAS HAD A LONG AND COMPLICATED relationship with colorectal cancer. The idea that intake of fiber might protect against this malignancy dates back at least to the late 1960s, when Burkitt[1] proposed that the low rates of colorectal cancer he observed in southern Africa (as well as the low rates of appendicitis, diverticular disease, and colorectal adenomas) were related to high-fiber intake. Subsequently, hemorrhoids, constipation, hypertension, hyperlipidemia, cardiovascular disease, and diabetes have been added to the list of problems that may be prevented by dietary fiber intake.

Of these disorders and diseases, colorectal cancer arguably has the most confusing association with fiber. Animal studies have variably suggested that fiber has reduced risks, increased risks, or had no effect on experimental bowel cancer.[2] Epidemiological studies have found intake of dietary fiber to be either protective, to have no effect,[3-6] or even rarely to confer an increase in risk.[7] Intervention trials (with adenoma end points) have found either no effect or increased risks.[8-12] It is certainly reasonable to ask the question: why has this been so difficult?

Part of the problem is dietary fiber itself. The term came into use in the 1970s, but the precise definition continues to evolve.[13-15] Although the term fiber suggests a single entity, fiber actually represents a group of plant products that may have very different properties.[2,13-15] Measuring dietary fiber intake is as uninformative as knowing that a patient with pneumonia took "some antibiotic or other." Clearly dietary "fibers" would be a better way to refer to these substances, and it would be best to consider fibers in biologi-

See also p 2849.

Author Affiliation: Dartmouth Medical School, Lenanon, NH.
Corresponding Author: John A. Baron, MD, Department of Medicine, Dartmouth-Hitchcock Medical Center, Evergreen #300, 46 Centerra Pkwy, Lebanon, NH 03766F (john.a.baron@dartmouth.edu).

cally meaningful groups, even if it is not practical to think of them individually. To add to the confusion, the biological effects of specific fibers may vary depending on whether a fiber is studied in isolation or in combination with other cell wall constituents.[15] Furthermore, there is a need for improvements in the measurement of the various fibers.[14]

In epidemiological studies, fibers are often categorized by the foods that contain them (eg, cereal fiber, fruit fiber, vegetable fiber). This is a convenient method of categorization, but these groupings retain substantial heterogeneity. A more physiologically helpful breakdown would be grouping fibers into categories of soluble and insoluble.[3] Insoluble fibers have the stool-bulking effect that is often associated in the public mind with fiber, but soluble fibers typically do not have this effect[2] because they are readily broken down in the large bowel. Fruit and vegetable fiber is largely soluble, but cereal fiber may be either soluble (eg, oat bran) or insoluble (wheat bran).

Dietary fibers often do not conform to their popular image. Despite the name, most are not fibrous, are certainly not just roughage. Most are polysaccharides. Although one feature dietary fibers share is a resistance to breakdown by alimentary enzymes,[13] much dietary fiber, in fact, *is* digested by bacteria in the colon.

The heterogeneity of dietary fibers has been a major contributor to the confusion regarding associations with colorectal cancer. In animal models, insoluble fibers such as wheat fiber have typically resulted in inhibition of bowel carcinogenesis, but soluble fibers such as pectins have tended to increase the numbers of tumors.[2] In human epidemiological studies that have found an inverse association with risk, the opposite pattern has been observed—fruit and vegetable (largely soluble) fibers more often have been found to be protective, while cereal fibers have not.[3] On the other hand, ecological studies (that use data on groups rather than individuals) have tended to find cereal fibers protective.[3] In general, prospective studies have shown no association. Clinical trials of wheat bran (with an adenoma end point) have been uniformly negative[8,9,11,12]; one trial that used a soluble fiber (psyllium) reported increased adenoma risks.[10] Thus, the relationship between intake of dietary fibers and colorectal cancer risk has depended on the type of fiber under discussion and the research design used. These discrepant findings do not permit a clear picture of how dietary fiber, or even some fiber groups, affect cancer of the large bowel.

In addition to the physiological complexity of the different fibers, epidemiological studies confront several challenges. One problem is that whatever dietary fiber is, it seems to be a highly confounded lifestyle factor. That is, fiber intake is related to consumption of other nutrients and to personal characteristics and traits that are associated with risk of colorectal cancer. This mixing of exposures can lead to distortion of the observed fiber-cancer association. Of course statistical adjustments can be made if these other factors are measured, but sometimes they can not be assessed or (worse) have not been identified. As a result, in epidemiological studies the relative risks for fiber intake seem to vary in important ways, depending on what variables are controlled.[5,6,16] Epidemiological studies of associations with intake of dietary fibers are also hampered by the measurement error that is inherent in dietary assessment.[17] These errors form a statistical uncertainty that generally tends to obscure associations. This problem can be quite severe, hiding even moderately strong associations.

One major advantage of a combined analysis such as that reported by Park and colleagues[16] in this issue of *JAMA* is that the large number of participants included in the analysis will enable investigators to detect relatively modest associations and to assess effects in subgroups—for example, individuals with very high or very low fiber intake. However, the large sample size will not help with confounding, nor will it overcome measurement error.

In their combined analysis of 13 prospective studies, Park et al[16] found evidence of an increased risk of colorectal cancer among individuals with very low intake of total dietary fiber (about the lowest 11%). After adjustment for measurement error, the relative risk for intakes of less than 10 g per day vs 10 or more g per day increased from 1.22 to 2.16. These findings suggest that colorectal cancer might be a sort of "fiber deficiency disease," such that a relatively modest minimum intake prevents an increased risk. For colorectal cancer overall, the authors did not detect any further reduction in risk at higher intakes. Nonetheless, for rectal cancer there were suggestions that fiber intake at the high end of the scale conferred a modest decrease in risk (about 15%) compared with moderate intake. The contrast between the highest and lowest quintiles of fiber intake (a more commonly used comparison) was even greater, although not detailed explicitly. It would be particularly interesting to see this estimate corrected for measurement error, as the authors did for the association between overall colorectal cancer risk and low fiber intake. If the rectal cancer analysis was as strongly affected by measurement error as that one, the result could be a substantial gradient from lowest to highest intake. If so, exploration of the types of fiber involved would be important.

The combined analysis by Park et al[16] yields an "average" effect size across the studies that contributed data. But of course it cannot reflect studies that are not included, such as the recently published EPIC cohort analysis.[4] This study is a combined analysis of separate cohorts which together had about 50% more cases than the largest of the studies in the analysis by Park et al.[16] In an error-corrected analysis, the EPIC investigators found a more than 40% reduction in risk of colorectal cancer for individuals in the highest quintile of dietary fiber intake vs the lowest, a much greater difference than the (uncorrected) results reported by Park et al.[16]

The findings by Park et al[16] and the results of the EPIC[4] analysis provide at least some indications that dietary fiber of some sort is related in some way to colon or rectal can-

cer risk. However, the 2 studies have not addressed exactly the same issues and, therefore, the extent of agreement or disagreement is not well-defined. Over the short term, wheat fiber or psyllium interventions do not seem to affect colorectal carcinogenesis, but understanding longer-term relationships with any type of fiber will require more work. Studies like that of Park et al[16] provide valuable help, but unfortunately there is more to do.

REFERENCES

1. Burkitt DP. Related disease–related cause? *Lancet*. 1969;2:1229-1231.
2. Harris PJ, Ferguson LR. Dietary fibres may protect or enhance carcinogenesis. *Mutat Res*. 1999;443:95-110.
3. Kaaks R, Riboli E. Colorectal cancer and intake of dietary fibre: a summary of the epidemiological evidence. *Eur J Clin Nutr*. 1995;49(suppl 3):S10-S17.
4. Bingham SA, Norat T, Moskal A, et al. Is the association with fiber from foods in colorectal cancer confounded by folate intake? *Cancer Epidemiol Biomarkers Prev*. 2005;14:1552-1556.
5. Michels KB, Fuchs CS, Giovannucci E, et al. Fiber intake and incidence of colorectal cancer among 76,947 women and 47,279 men. *Cancer Epidemiol Biomarkers Prev*. 2005;14:842-849.
6. Terry P, Giovannucci E, Michels KB, et al. Fruit, vegetables, dietary fiber, and risk of colorectal cancer. *J Natl Cancer Inst*. 2001;93:525-533.
7. Potter JD, McMichael AJ. Diet and cancer of the colon and rectum: a case-control study. *J Natl Cancer Inst*. 1986;76:557-569.
8. Alberts DS, Martinez ME, Roe DJ, et al. Lack of effect of a high-fiber cereal supplement on the recurrence of colorectal adenomas: Phoenix Colon Cancer Prevention Physicians' Network. *N Engl J Med*. 2000;342:1156-1162.
9. DeCosse JJ, Miller HH, Lesser ML. Effect of wheat fiber and vitamins C and E on rectal polyps in patients with familial adenomatous polyposis. *J Natl Cancer Inst*. 1989;81:1290-1297.
10. Faivre J, Bonithon-Kopp C, Kronborg O, Rath U, Giacosa A. A randomized trial of calcium and fiber supplementation in the prevention of recurrence of colorectal adenomas: a European intervention study. *Gastroenterology*. 1999;116:A56.
11. MacLennan R, Macrae F, Bain C, et al. Randomized trial of intake of fat, fiber, and beta carotene to prevent colorectal adenomas: the Australian Polyp Prevention Project. *J Natl Cancer Inst*. 1995;87:1760-1766.
12. McKeown-Eyssen GE, Bright-See E, Bruce WR, et al. A randomized trial of a low fat high fibre diet in the recurrence of colorectal polyps: Toronto Polyp Prevention Group. *J Clin Epidemiol*. 1994;47:525-536.
13. DeVries JW. On defining dietary fibre. *Proc Nutr Soc*. 2003;62:37-43.
14. McCleary BV. Dietary fibre analysis. *Proc Nutr Soc*. 2003;62:3-9.
15. Ferguson LR, Chavan RR, Harris PJ. Changing concepts of dietary fiber: implications for carcinogenesis. *Nutr Cancer*. 2001;39:155-169.
16. Park Y, Hunter DJ, Spiegelman D, et al. Dietary fiber intake and risk of colorectal cancer: a pooled analysis of prospective cohort studies. *JAMA*. 2005;294:2849-2857.
17. Kipnis V, Freedman LS, Brown CC, Hartman AM, Schatzkin A, Wacholder S. Effect of measurement error on energy-adjustment models in nutritional epidemiology. *Am J Epidemiol*. 1997;146:842-855.

Creating a Safer Health Care System
Finding the Constraint

Stephen G. Pauker, MD
Ellen M. Zane, BA, MA
Deeb N. Salem, MD

PRIMUM NON NOCERE. IN *EPIDEMICS*,[1] HIPPOCRATES urged all physicians to provide safe and effective care. Of course, that is not always possible because adverse events or bad outcomes sometimes occur. But physicians are driven to minimize the likelihood of harm, especially adverse events that more careful reasoning, better practices, and better systems might prevent. The Institute of Medicine called attention to the ubiquitous nature of errors in medicine and issued the challenge to develop safer systems of care.[2-4] Safety, the flip side of doing the right thing at the right time, means not doing the wrong thing at any time, especially if the wrong action occurs inadvertently—by mistake—because the clinician or the system did not prevent an error before its effect reached a patient and caused harm.

Now, 5 years after the Institute of Medicine report,[2] several authors have questioned the system's progress toward patient safety.[5-8] In this issue of *JAMA*, Longo and colleagues[9] provide data about the pace of implementing clinical safety systems. When a system had a relatively high rate of implementation initially, subsequent improvement was difficult[10]; when a safety system did not have high penetrance initially, more rapid change might be expected subsequently. There has been steady progress in patient safety, but it has been perhaps unexpectedly slow.

Why, then, are these important improvements taking so long? Is the problem inertia, inattention, a lack of resources, or conflicting demands on time or energy? Medicine is no longer an island; physicians need to learn from other domains of practice, other industries. Of course, no industry has a uniform record of success; they all must learn from each other.

Over the past 2 decades, a set of tools and concepts loosely called the *theory of constraints*[11-15] evolved from an initial focus in manufacturing. The theory of constraints requires explicitly framing the desired goal, the necessary conditions for achieving that goal, and the measurements that will monitor progress toward that goal, and then establishing a clear plan for making change happen. For medicine, the goal is

See also p 2858.

Author Affiliations: Department of Medicine (Drs Pauker and Salem) and Office of the President (Ms Zane), Tufts–New England Medical Center, Boston, Mass. **Corresponding Author:** Stephen G. Pauker, MD, Box 302, Tufts–New England Medical Center, 750 Washington St, Boston, MA 02111 (spauker@tufts-nemc.org).