

from the unexpected results of a single trial that are contrary to the totality of the evidence.

CHARLES H. HENNEKENS, M.D.
JULIE E. BURING, Sc.D.
Boston, MA 02115
Brigham and Women's Hospital

RICHARD PETO, F.R.S.
Oxford University
Oxford OX2 6HE,
United Kingdom

LUNG CANCER AND VITAMIN SUPPLEMENTATION

To the Editor: A randomized nutritional-intervention trial similar in scope and size to the Finnish alpha-tocopherol and beta carotene trial¹ has been completed in Linxian, China, an area with one of the world's highest rates of esophageal and stomach cancer and habitually low intake of several nutrients.² We have previously reported³ that among the nearly 30,000 male and female participants, rates of mortality from cancer were significantly lower (relative risk, 0.87) among those who received daily supplementation from 1986 to 1991 with a combination of beta carotene (15 mg), alpha-tocopherol (30 mg), and selenium (50 µg). In this population, over 85 percent of the cancers arose in the esophagus or stomach, but 31 deaths were attributed to lung cancer. As shown in Table 1, the risk of death from lung cancer was reduced by 45 percent ($P = 0.11$) among those receiving the beta carotene, alpha-tocopherol, and selenium supplements. Three nonfatal lung cancers were diagnosed, all among those in the group given supplements. Although the small

Table 1. Relative Risk of Death from Lung Cancer among Subjects Taking Beta Carotene, Alpha-Tocopherol, and Selenium Supplements, 1986 to 1991.

BETA CAROTENE, ALPHA-TOCOPHEROL, AND SELENIUM SUPPLEMENTS	PERSON-YEARS OF OBSERVATION	NO. OF LUNG- CANCER DEATHS	RELATIVE RISK	95% CI*
No	74,749	20	1.0	—
Yes	75,024	11	0.55	0.26–1.14

*CI denotes confidence interval.

numbers of events preclude detailed evaluation according to subgroup, the reduction in risk associated with supplementation was greater among the 30 percent (almost all men) of the participants who smoked cigarettes than among the 70 percent who were nonsmokers. Although clearly not definitive, these findings provide additional relevant data for the evaluation of the potential effect of vitamin supplementation on the risk of lung cancer.

Bethesda, MD 20892

WILLIAM J. BLOT, Ph.D.
National Cancer Institute

JUN-YAO LI, M.D.
Chinese Academy
of Medical Sciences
Beijing 100021, China

Bethesda, MD 20892

PHILIP R. TAYLOR, M.D., Sc.D.
National Cancer Institute

BING LI, M.D.
Chinese Academy
of Medical Sciences
Beijing 100021, China

1. The Alpha-Tocopherol, Beta Carotene Cancer Prevention Study Group. The effect of vitamin E and beta carotene on the incidence of lung cancer and other cancers in male smokers. *N Engl J Med* 1994;330:1029-35.
2. Li B, Taylor PR, Li JY, et al. Linxian nutrition intervention trials: design, methods, participant characteristics, and compliance. *Ann Epidemiol* 1993;3:577-85.
3. Blot WJ, Li J-Y, Taylor PR, et al. Nutrition intervention trials in Linxian, China: supplementation with specific vitamin/mineral combinations, cancer incidence and disease-specific mortality in the general population. *J Natl Cancer Inst* 1993;85:1483-92.

MORE ON CORONARY HEART DISEASE: THE DIETARY SENSE AND NONSENSE

To the Editor: In his review of *Coronary Heart Disease: The Dietary Sense and Nonsense* (March 31 issue),¹ Dr. Stone writes about the report of one very large review² that it is "truly critical and comprehensive." However, that report, in dealing with migration studies, omits those that do not fit the diet-heart hypothesis — for example, the studies by Day et al.³ and Stanhope et al.⁴ This omission is deliberate and explicit ("Only representative publications have been cited here" [page 178]²) and not due to limitations in the number of references, since some 1000 references are cited in this section. It is doubtful whether such selectivity can be labeled "critical and comprehensive."

Dr. Stone writes that the Oslo Study Diet and AntiSmoking Trial "showed the importance of a cholesterol-lowering diet low in saturated fat in reducing both morbidity and total mortality from coronary heart disease." The study did not show that.⁵ In addition to a drop in saturated-fat intake, cigarette consumption dropped by half and weight went down by an average of 5 kg, without any concomitant reduction in energy intake; thus, the participants must have started exercising. It is not obvious that of these several interventions, a reduction in saturated fat played any part. The conclusion of the investigators differs from that of Dr. Stone: "If this had been a diet trial only, the difference in MI incidence between the two groups would probably not have reached statistical significance."

S-244 24 Kävlinge,
Sweden

OLOV H. HOLMQUIST, Ph.D.
Swedish Meat Research Institute

1. Stone NJ. Review of: *Coronary heart disease: the dietary sense and nonsense — an evaluation by scientists*. *N Engl J Med* 1994;330:943-4.
2. Committee on Diet and Health, Food and Nutrition Board, Commission on Life Sciences, National Research Council. *Diet and health: implications for reducing chronic disease risk*. Washington, D.C.: National Academy Press, 1989:159-258.
3. Day J, Carruthers M, Bailey A, Robinson D. Anthropometric, physiological and biochemical differences between urban and rural Maasai. *Atherosclerosis* 1976;23:357-61.
4. Stanhope JM, Sampson VM, Prior IA. The Tokelau Island Migrant Study: serum lipid concentration in two environments. *J Chronic Dis* 1981;34:45-55.
5. Hjermann I, Velve Byre K, Holme I, Leren P. Effect of diet and smoking intervention on the incidence of coronary heart disease: report from the Oslo Study Group of a randomised trial in healthy men. *Lancet* 1981;2:1303-10.

To the Editor: In the guise of a review of George Mann's book, Stone pitches the diet-heart hypothesis. Since Muldoon et al. have shown that all properly controlled trials of cholesterol lowering have only raised the death rate,¹ Stone resorts to three ploys to muddy the waters.

First, he cites the Oslo trial, in which subjects had 45 percent less tobacco use, and terms it "one of the best clinical