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Diet and Lung Cancer

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VITAMIN A AND β-CAROTENE

Interest in diet and lung cancer began with a cohort study in Norway which showed that an index of vitamin A intake was negatively associated with lung cancer incidence.3,4 Others showed similar associations.3,4

With a finding of a negative association of β-carotene but not preformed vitamin A with lung cancer incidence,3 and some understanding on how β-carotene might be protective,4 attention shifted to β-carotene. Several authors have found evidence of protective effects of β-carotene.5-11 Further, some of the strongest associations in earlier work were with "vitamin A" indices derived from plant foods. Others had also found evidence of a protective effect of vegetables on lung cancer.10,11,13 However, it cannot be assumed that all, if any, of the protective effect comes from β-carotene, as many inhibitors of carcinogenesis are to be found in plants, and thus vegetables.12 Whether or not β-carotene consumption is protective rather than some other factor associated with vegetable consumption will be clarified when results from several ongoing chemoprevention trials are available.

All investigators have been careful to adjust their analyses for smoking. Several have also attempted to evaluate interaction with smoking. In one study the reduced relative risk of lung cancer associated with vitamin A was most evident among men who smoked heavily.13 Hiraizumi14 suggested that green-yellow vegetable consumption might hasten the effect of cessation of cigarette smoking and might also affect the risk due to passive smoking. Samet et al6 found effects only in Anglos rather than Hispanics and only in former rather than current cigarette smokers. Ziegler et al10 found the protective effect of vegetables was limited to current and recent cigarette smokers. Pisani et al16 found an increased risk of lung cancer for smokers who did not consume carrots, but no corresponding effect for exsmokers or nonsmokers. Byers et al11 found the risk reduction associated with vitamin A from fruits and vegetables was most evident for light or exsmokers. Bond et al10 found the strongest inverse association of a vitamin A index with lung cancer risk among cigarette smokers.

Some investigators have also attempted to evaluate different risks according to histologic type. Several found associations that were strongest for squamous cell cancer.4,10,11,12 Byers et al11 also found associations with small cell cancer and Ziegler et al10 with adenocarcinoma for current and recent smokers.

Not all studies, however, have found a protective effect of β-carotene. In a cohort study of 10,473 residents of Los Angeles followed up for 5 years, 56 lung cancers occurred. Little evidence was found that increased intake of vitamin A or β-carotene protected against the development of lung cancer.15 It is possible that the follow-up period was too short to detect an effect. However, a recent case-control study in Toronto was also negative in respect to these micronutrients (see below).

OTHER DIETARY FACTORS

In spite of the strong and generally consistent finding of a protective effect of β-carotene, other dietary factors have been implicated in lung cancer. Hinds et al16 found evidence of increasing risk with increasing consumption of dietary cholesterol. There was some indication of a protective effect of vitamin C in this study in males but not in females.7 Byers et al11 had inconsistent findings for dietary cholesterol, though there was some indication of increasing risk with increasing consumption of fat, especially in males. Wynder et al10 performed an ecologic study using food and tobacco disappearance data from 43 countries and correlated this with male and female lung cancer mortality rates. There was a highly significant association between fat calories and lung cancer rates (r for males = 0.81). In a multivariate analysis only fat calories and tobacco disappearance remained highly statistically significantly associated with lung cancer.

In a case-control study recently completed in Toronto, sufficient dietary data were collected to enable quantitative estimates to be made of consumption of vitamin A, retinol, β-carotene, other carotenes, vitamin C, vitamin E, α-tocopherol, animal fat, saturated fatty acids, cholesterol, and nitrate. The study was designed primarily to study lung cancer in women, though we selected at random a male case to match each female, and population controls for each. The total number of female and male pairs analyzed was 469 and 472, respectively. There was no evidence of a protective effect from any of these dietary factors except nitrate. Indeed, in males retinol consumption appeared to increase risk. Nitrate, being largely derived from vegetables, may be acting as a marker for another factor in vegetables (other than β-carotene, for example) that is protective. However animal fat, saturated fatty acids, and dietary cholesterol all increased risk in males, with cholesterol also increasing risk in females. The odds ratio for each 100-mg consumption of dietary cholesterol in both sexes was 1.12.

There is increasing evidence that some dietary factors may be protective for lung cancer, and some may increase risk. Although the most consistent evidence relates to a protective effect of β-carotene, some more recent work suggests that other protective factors may be found in vegetables, while dietary cholesterol and possibly animal fat may increase risk. Fortunately, these findings are consistent with guidelines for dietary modification in Western societies to reduce cancer and cardiovascular disease risk. Neverth-
less, in all studies, the effect of dietary factors is far less than that of cigarette smoking, and it is clear that the major preventive for lung cancer remains prevention of initiation of smoking, and smoking cessation.

REFERENCES


The National Cancer Institute’s Smoking, Tobacco, and Cancer Program*

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In the past 20 years, a solid scientific base of information has been codified showing that the majority of cancers are closely linked to lifestyle and environmental factors. To act on this knowledge, however, and modify the conditions for the public to profit from this knowledge is a major challenge. In the mid-1980s the National Cancer Institute (NCI) began a new initiative to reduce cancer mortality 50% by the end of this century. This effort is aimed at saving hundreds of thousands of lives partly through primary prevention measures, and particularly by reducing the prevalence of tobacco use.

PROBLEM

The first conclusive evidence linking smoking to cancer is nearly 4 decades old. In 1950, Wynder and Graham published sufficient evidence that smoking causes lung cancer. A decade and a half later, the Surgeon General of the United States published his first Report on Smoking and Health. This monograph was a landmark document in the US. It became the base from which a series of additional reports on varying aspects on smoking and disease evolved.

Although encouraging changes have taken place in smoking behavior in the US—as many as 37 million Americans have stopped smoking in the past several decades—approximately 54 million Americans continue to smoke, and the prevalence of smoking behavior among females remains at levels that foreshadow continued increases in lung cancer mortality in women for at least another decade.

The need for new knowledge about innovative, effective, and long lasting ways to control tobacco use is paramount. Efforts to generate this knowledge must be given a high priority from now and the end of this century.

NCI’s RESPONSE—AN INTENSIVE INTERVENTION RESEARCH PROGRAM

In 1982 the NCI launched its new Smoking, Tobacco, and Cancer Program (STCP). A commitment was made to develop and test intervention strategies to reduce tobacco use and then to apply the best of these strategies in large target populations. The STCP now includes 55 prevention and cessation trials, affecting over 10 million people. It is being carried out in 26 states and in over 200 North American cities. Each of these trials lasts about 5 years and most are characterized as phases 3 and 4 of the cancer control phases (Fig 1). The cost for these trials is about $155 million.

These trials encompass a broad program testing 4 intervention channels, schools, physicians and dentists, self-help, and the media in 6 target populations, blacks, Hispanics, women, youth, smokeless tobacco users, and heavy smokers (Fig 2).

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CHEST/96/1/JULY, 1989/Supplement 95