

SUPPLEMENT TO

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Alcohol-Nutrient Interactions in Cancer Etiology

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The cancers for which there is the most compelling epidemiologic evidence of associations with both diet and alcohol are oral, pharyngeal, laryngeal, esophageal, and liver cancer. For lung, breast, stomach, and colorectal cancer, there is reasonably strong epidemiologic evidence of associations with diet, but only moderate or equivocal evidence of associations with alcohol. For pancreatic cancer there is suggestive evidence of associations with both exposures. It is probable that the quantitative relationship and the underlying biological mechanisms of the diet-alcohol interaction will not be the same for all cancers. Heavy alcohol consumption and generally poor nutrition, possibly a deficiency of several micronutrients and food groups, were the major risk factors for esophageal cancer in a case-control study of the unusually high rates for this cancer among Washington, DC black men. It is proposed that alcohol might increase risk of esophageal cancer, in part, by reducing nutrient intake. Two descriptive studies are presented that suggest that as the percent of caloric intake from alcohol increases, the daily consumption of protein, carbohydrate, fiber, and many micronutrients steadily and significantly decreases. Alcohol consumption and low fruit and vegetable intake, as well as snuff dipping and cigarette smoking, were shown to be risk factors for oral-pharyngeal cancer in a case-control study of the high rates of these cancers among North Carolina women. Since these women were not heavy drinkers, it is not likely that alcohol functioned by altering nutritional status. A number of mechanisms for the action of alcohol in cancer etiology are presented.

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FOR A NUMBER OF CANCERS, both diet and alcohol are postulated to be risk factors (Table 1); and the question of interaction between the two exposures can be addressed. The cancers for which there is the most compelling evidence of associations with both diet and alcohol are oral, pharyngeal, laryngeal, esophageal, and liver cancer. For all these cancers there is strong epidemiologic evidence for associations with alcohol¹ and except perhaps for laryngeal cancer, strong epidemiologic evidence for associations with diet although the dietary patterns associated with increased risk of these cancers are not the same. For lung, breast, stomach, and colorectal cancer, there is reasonably strong epidemiologic evidence of associations with diet, but only moderate or equivocal evidence of associations with alcohol.¹ Recently several studies have highlighted apparent associations between

lung cancer^{2,3} and breast cancer⁴⁻⁶ and alcohol intake. However, it is quite possible that these associations with alcohol are not causal and may have been generated by underlying associations with diet or, in the case of lung cancer, smoking, that were not adequately controlled in the analysis. Finally, for pancreatic cancer there is suggestive epidemiologic evidence of associations with both diet and alcohol.¹

The dietary risk factors for these cancers are not the same. The risks of oral,⁷⁻⁹ pharyngeal,⁷⁻⁹ and laryngeal¹⁰ cancer are increased by low vegetable and fruit intake, possibly because of the carotenoids and vitamin C concentrated in these foods. But risk of esophageal cancer seems related to generally poor nutrition,^{11,12} and the dietary risk factor for liver cancer seems to be aflatoxin-contaminated grains and legumes.¹³ Like oral, pharyngeal, and laryngeal cancer, lung cancer risk increases with low vegetable and fruit intake.¹⁴⁻¹⁶ But breast and colorectal cancer seem related to "affluent" dietary patterns: breast cancer to a high-fat diet^{16,17} and colorectal cancer to a high-fat, low-fiber diet.^{16,18} Stomach cancer is associated with high intake of smoked, pickled, or salted foods and low intake of vegetables and fruits.¹⁹ The dietary risk factors for pancreatic cancer have not been identified.²⁰ With this diversity in etiology, it is probable that the nature of the diet-alcohol interaction will not be the same for all these cancers, in terms of both the quantitative relationships and the underlying biological mechanisms.

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TABLE 1. Cancers Associated With Diet and Alcohol

Site	Diet	Alcohol
Oral cavity	Reduced risk with high vegetable and fruit intake	Strong evidence for association
Pharynx	Reduced risk with high vegetable and fruit intake	Strong evidence for association
Larynx	Possibly reduced risk with high vegetable and fruit intake	Strong evidence for association
Esophagus	Increased risk with generally poor nutrition	Strong evidence for association, especially with spirits
Liver	Increased risk with aflatoxin exposure	Strong evidence for association
Lung	Reduced risk with high vegetable and fruit intake	Equivocal evidence for association, possibly not causal
Breast	Probably reduced risk with low-fat, less "affluent" diet	Moderate evidence for association, possibly not causal
Colon-rectum	Probably reduced risk with high-fiber, low-fat diet	Moderate evidence for association, especially between beer and rectal cancer
Stomach	Possibly increased risk with intake of smoked, pickled salted foods; possibly decreased risk with high vegetable and fruit intake	Equivocal evidence for association
Pancreas	Association with diet presumed but not adequately investigated	Equivocal evidence for association

Diet, Alcohol, and Esophageal Cancer

The interaction of diet and alcohol in cancer etiology has been investigated more thoroughly for esophageal cancer than for most other cancers. However, before discussing the nature of the interaction, it is first necessary to clarify the nature of the relationship with diet. Generally poor nutrition has been suspected to be a cause of esophageal cancer for several reasons. (1) In Iran,²¹ the Soviet Union,²² South Africa,²³ China,²⁴ and the Carribean,^{25,26} esophageal cancer is endemic in regions with limited diets and impoverished agriculture. (2) Case-control studies in the United States^{27,28} and Iran²⁹ and a prospective cohort study in Japan³⁰ have demonstrated an association between reduced consumption of certain basic food groups and esophageal cancer. (3) These studies, as well as case-control studies in Puerto Rico²⁵ and Singapore,³¹ also have shown an association between low socioeconomic status and esophageal cancer. Within the United States mortality rates for esophageal cancer at the county level are inversely related to indices of socioeconomic status and are higher among blacks than whites.³² (4) Until recently esophageal cancer was unusually common in women from the rural, northern areas of Europe, many of whom also had the Plummer-Vinson syndrome, which is associated with iron and other micronutrient deficiencies.^{11,33} (5) Esophageal cancer has been reported as a sequel of celiac disease, a malabsorption disorder of the small intestine.^{34,35}

Several years ago Dr. William Blot, Linda Pottern, Linda Brown, Dr. Robert Hoover, and I, all at the National Cancer Institute, conducted a case-control study of esophageal cancer among black men living in Washington, DC,^{12,36} which at the time was the US metropolitan area with the highest esophageal cancer mortality rate for nonwhite men. The next of kin of 120 esophageal cancer cases who died during 1975 through 1977 and of 250 of Washington DC black men who died of other causes were interviewed. They were asked about their usual adult frequency of consumption, excluding the last few years, of

31 food items and of beer, wine, and hard liquor.¹² The food frequencies were converted into the food groups and nutrient indices shown in Table 2. Three major food groups—dairy products and eggs, fruit and vegetables, and fresh and frozen meat and fish—were each significantly and inversely correlated with the relative risk of esophageal cancer. Associations with other food groups, such as carbohydrates and precooked or processed meat and fish,

TABLE 2. Ethanol-Adjusted Relative Risk of Esophageal Cancer by Consumption of Food Groups and Micronutrients

Nutrition index	Level of consumption		
	High	Moderate	Low
Food groups			
Meat, fish, eggs, and cheese	1.0	1.7	1.3
Meat and fish	1.0	1.3	1.2
Dairy products and eggs	1.0	1.7	1.9†
Fruits and vegetables	1.0	1.7	2.0†
Vegetables	1.0	1.5	1.6*
Green vegetables	1.0	1.0	1.3
Yellow vegetables	1.0	1.0	1.7
Fruits	1.0	2.4	2.0†
Carbohydrates	1.0	1.1	1.2
Bread	1.0	1.1	1.1
Fresh or frozen meat and fish	1.0	1.6	2.2†
Precooked or processed meat and fish	1.0	0.9	0.9
Nitrite-containing foods	1.0	1.1	1.0
Micronutrients			
Vitamin A	1.0	1.5	1.5
Carotene	1.0	1.3	1.3
Vitamin C	1.0	1.2	1.8†
Thiamin	1.0	1.2	1.2
Riboflavin	1.0	1.0	1.7†

* *P* for trend <0.10.

† *P* for trend <0.05.

TABLE 3. Ethanol-Adjusted Relative Risk of Esophageal Cancer by an Overall Measure of Food Consumption Patterns

Food consumption pattern*	No. of cases	No. of controls	RR
HHH	2	20	1.0
HHM HMM	24	65	3.8
HHL MMM HML HLL	32	68	4.5
MML MLL	36	46	6.7
LLL	11	8	15.0

* Concurrent level of consumption of fresh or frozen meat and fish, fruits and vegetables, and dairy products and eggs, each rated as high (H), moderate (M), or low (L). For example, HML indicates high consumption of one of the three food groups, moderate consumption of a second, and low consumption of a third.

RR: relative risk.

which includes lunch meat, frankfurters, canned fish, *etc.*, were not apparent. Thus only those food groups that are traditionally associated with a sensible, nutritious diet were related to reduced risk. The least nourished third of the study population, defined by any of these three food groups, was at twice the risk of the most nourished third. Individuals who consumed low levels of any two of these three food groups had about four times the risk of those who consumed high levels of the same two food groups. When the three food group consumption measures were combined into a single comprehensive nutrition index, as shown in Table 3, the relative risk between extremes, those low in consumption of all three food groups relative to those high in consumption of all three, reached 15. The risk of esophageal cancer decreased steadily with improving patterns of food consumption. The association of esophageal cancer with these measures of poor nutrition appeared to be independent of any associations with alcohol, smoking, or socioeconomic status, as measured by education. Because the association with poor nutrition was independent of socioeconomic status, it is unlikely

that unidentified aspects of lifestyle correlated with diet were primarily responsible.

As shown in Table 2, estimates of the intake of vitamin A, carotenoids, vitamin C, thiamin, and riboflavin were inversely associated with relative risk; but each micronutrient was less strongly associated with risk than were the broad food groups that provide most of the micronutrient. For example, risk was elevated among individuals with a low intake of vitamin C or carotenoids but the risk was somewhat less than that for low consumption of fruits and vegetables, which are the major source of vitamin C and carotenoids. Similarly, risk was elevated among those with a low intake of riboflavin, but not as markedly as among those with low consumption of dairy products and eggs, the major sources of riboflavin. Thus no specific micronutrient deficiency was identified. Instead, generally poor nutrition, possibly a deficiency of several micronutrients or food groups, seemed to be the major dietary predictor of risk of esophageal cancer among these urban black men.

Alcohol consumption was the other major risk factor in our study of Washington, DC black men.³⁶ Drinkers of alcoholic beverages had 6.4 times the risk of nondrinkers. The relative risk increased steadily with the amount of ethanol consumed and was highest among drinkers of hard liquor, although risk also was elevated among consumers of wine and beer. The mechanism by which alcohol increases the risk of esophageal cancer or the risk of any other cancer, for that matter, is not known.³⁷ We proposed that since poor nutrition clearly appeared to be a risk factor for esophageal cancer, alcohol might increase risk, at least in part, by reducing nutrient intake. Beer, wine, and hard liquor provide a share of the daily caloric needs and consequently reduce appetite but provide almost none of the daily requirements for micronutrients and protein. In Table 4 is shown the relative risk of esophageal cancer by kilocalories of alcoholic beverages consumed per week. A nutritionist might refer to these alcoholic calories as empty calories. The relative risk of esophageal cancer rose steadily from 1.0 to 4.1 to 6.4 as the percent of the estimated caloric need of the average man, 51 to 75 years of age, that was being supplied by

TABLE 4. Relative Risk of Esophageal Cancer by Consumption of the Empty Calories in Alcoholic Beverages

Kcal of beer, wine, and hard liquor consumed weekly	Percent of caloric needs filled by alcoholic beverages*	No. of cases	No. of controls	RR by empty calories	RR by empty calories/weight†
<500	<0.03	5	55	1.0	1.0
500-3360	0.03-20	16	43	4.1	4.5
3361-6720	21-40	18	31	6.4	5.6
6721-13440	41-80	28	49	6.3	6.4
>13440	>80	23	35	7.2	7.1

* Daily caloric need of each individual was assumed to be 2400 kcal, on the basis of the National Academy of Sciences' recommendation for US men 51-75 yr of age.³⁸

† Empty calories/weight was cut into strata that were almost identical in size to those chosen for empty calories.
RR: relative risk.

alcoholic beverages rose from less than 0.03% to 20%, and then to 40% and 80%. It is likely that some of these men who were consuming more than 20% of their daily caloric needs in the form of alcoholic beverages continued to eat other foods, exceeded their caloric needs, and put on weight. According to our hypothesis, only those heavy drinkers who exceeded their caloric needs would be able to approach reasonable intakes of a variety of nutritious food groups. Indeed, this proved to be true. Those men in our study population who were distinctly heavier than the average US black man had about 50% the risk of esophageal cancer of the men of average weight.

Table 5 shows the interaction between nutritional status and ethanol consumption in determining the risk of esophageal cancer. Nutritional status is measured by concurrent consumption of fresh or frozen meat and fish, fruit and vegetables, and dairy products and eggs. The risks for poor nutrition and ethanol intake remained distinct. The elevated risk associated with heavy ethanol consumption could be seen at all three levels of nutritional status, and the elevated risk associated with poor nutrition could be detected at both levels of ethanol consumption. From a public health perspective, what this implies is that although a wholesome diet could reduce the risk associated with alcohol consumption in both light and heavy drinkers, it could not totally eliminate it. Alcohol remained a potent risk factor, with risk proportional to its intake. Similarly, decreasing alcohol consumption could reduce the risk associated with poor nutrition, but not totally eliminate it. It was not possible to determine whether poor nutrition was a risk factor among nondrinkers since so few of the men in the study population, only five of the cases, did not drink.

Technically, the interaction shown in the table is multiplicative since the relative risk in the poorly nourished, heavy drinkers is about equal to the product of multiplying the risk in well-nourished, heavy drinkers by the risk in poorly nourished, light drinkers. However, with different divisions of the nutrition and ethanol variables or different nutrition indices, combined effects were often less than multiplicative. Nonetheless, the poorly nourished, heavy drinkers remained the group at highest risk. The partial independence of poor nutrition and ethanol intake demonstrated in the table is not inconsistent with the proposal that alcohol consumption increases the risk of esophageal cancer by reducing nutrient intake. Our measure of nutritional status was a partial measure of nutrient intake; alcohol consumption was also a partial measure of nutrient intake; and heavy drinkers with a low nutrition index had the lowest nutrient intake of all.

Before leaving the subject of esophageal cancer, it must be emphasized that although poor nutrition, heavy drinking, and possibly smoking appear to be the major risk factors for this cancer in the United States, in other coun-

TABLE 5. Relative Risk of Esophageal Cancer by Nutritional Status and Ethanol Consumption

Ethanol consumption in hard liquor equivalents	Nutritional status*		
	High	Moderate	Low
0-5.9 fl oz/day	1.0 (6, 43)†	1.7 (6, 25)	3.0 (8, 19)
6.0-80.0 fl oz/day	2.7 (13, 34)	4.1 (21, 37)	8.0 (29, 26)

* Concurrent level of consumption of fresh or frozen meat and fish, fruits and vegetables, and dairy products and eggs. High, moderate, and low nutritional status were defined as food consumption patterns HHH, HHM, and HMM; patterns HHL, MMM, HML, and HLL; and patterns MML, MLL, and LLL, respectively.

† Nos. in parentheses are nos. of cases and controls.
fl oz: fluid ounces.

tries alcohol and tobacco may not play as important a role. For example, China's extremely high rates of esophageal cancer have been attributed to very poor nutrition and ingestion of toxins in moldy foods²⁴; and Iran's extremely high rates of esophageal cancer have been attributed to very poor nutrition and opium use.¹¹

Mechanisms of Action for Alcohol in Cancer Etiology

Unfortunately, epidemiologists can usually only propose, but not prove, the mechanism of action of a risk factor for a disease. However, several descriptive studies have recently examined the dietary patterns of moderate and heavy drinkers. A group from Washington State University has analyzed 24-hour dietary recalls and alcohol histories from 179 men, ranging from randomly selected undergraduates and nonfaculty university employees to men convicted of driving while intoxicated or receiving inpatient treatment for alcoholism.³⁹ In Table 6 are shown total energy intake and energy sources for this population, divided into thirds by the level of alcohol consumption. The light drinkers reported consuming less than the equivalent of 0.7 fluid ounces (fl oz) of hard liquor per day, the moderate drinkers reported the equivalent of 0.7 to 3.1 fl oz of hard liquor per day, and the heavy drinkers

TABLE 6. Total Energy Intake and Energy Sources by Ethanol Consumption

Energy intake	Ethanol consumption in hard liquor equivalents/d		
	<0.7 fl oz	0.7-3.1 fl oz	>3.1 fl oz
Total energy in kcal/d	2667 ± 1025*	2609 ± 1446	2710 ± 1506
% Alcohol	0.5 ± 1.8	6.1 ± 13.5	23.8 ± 28.1
% Protein	15.0 ± 4.0	14.1 ± 5.6	11.6 ± 6.7
% Carbohydrate	43.1 ± 10.4	43.8 ± 12.4	34.8 ± 14.2
% Fat	41.4 ± 9.7	36.0 ± 13.7	29.8 ± 18.7

* Mean ± SD.
fl oz: fluid ounces.

TABLE 7. Nutrient Intake by Ethanol Consumption

Nutrient	Ethanol consumption in hard liquor equivalents/d		
	<0.7 fl oz	0.7-3.1 fl oz	>3.1 fl oz
Protein (g)	100 ± 44*	90 ± 58	72 ± 49
Calcium (mg)	1248 ± 828	1081 ± 965	745 ± 672
Vitamin A (IU)	7241 ± 8890	5708 ± 6821	3215 ± 3520
Vitamin C (mg)	120 ± 167	93 ± 111	47 ± 73
Thiamin (mg)	1.4 ± 0.8	1.2 ± 0.8	0.9 ± 0.8
Iron (mg)	15.4 ± 6.4	15.0 ± 10.5	10.5 ± 8.0
Fiber (g)	3.5 ± 2.6	2.9 ± 2.2	1.9 ± 2.0

* Mean ± SD.

fl oz: fluid ounces.

reported more than 3.1 fl oz per day. Because of the groups from which they were selected, the heavy drinkers may well have imbibed far more than 3.1 fl oz daily. Total energy intake did not vary significantly among the three groups. Thus as the percent of calories in the daily diet obtained from alcohol reached 24% in the heavy drinkers, the percent of calories derived from protein, from carbohydrate, and from fat all decreased significantly. In Table 7 are shown daily nutrient intakes for the three groups. On the average, heavy drinkers consumed 72% as much protein, 60% as much calcium, 44% as much vitamin A, 39% as much vitamin C, 64% as much thiamin, 68% as much iron, and 54% as much fiber as the light drinkers. Intake of niacin, riboflavin, and other nutrients also were reduced, although not at a statistically significant level. When asked about their dietary patterns, 2% of the light drinkers, 14% of the moderate drinkers, and 50% of the heavy drinkers reported skipping meals when drinking.

One limitation of the study just described is that the study sample was not representative of any larger population. In HANES I, the first Health and Nutrition Examination Survey of the United States, conducted during 1971 through 1975, 24-hour dietary recalls and usual adult alcohol intake were collected for a population-based sample of the entire United States.⁴⁰ Even though poverty areas, the elderly, and women of childbearing ages were systematically oversampled,⁴⁰ the 15,000 adults interviewed were reasonably representative. A group from the Medical College of Wisconsin showed that moderate drinkers, defined as those consuming the equivalent of 0.5 to 2.0 fl oz of hard liquor/day, included 22% of the men and 11% of the women and obtained, on the average, 4% to 6% of their daily caloric intake from alcohol.⁴¹ Heavy drinkers, defined as those consuming more than the equivalent of 2 fl oz of hard liquor per day, included 17% of the men and 3% of the women and obtained, on the average, 16% to 17% of their daily caloric intake from

alcohol. Total caloric intake did increase as the level of alcohol consumption rose, but caloric intake from food, excluding caloric intake from alcohol, dropped. Daily nutrient intake in the moderate and heavy drinkers was not calculated.

Reducing nutrient intake is not the only mechanism that has been proposed for the role of alcohol in cancer etiology. The means by which alcohol increases the risk of cancer is not known, and a major reason is that attempts to produce cancer in well-nourished laboratory animals by prolonged ingestion of ethanol have consistently failed.³⁷ Mechanisms for alcohol-associated carcinogenesis include the following. (1) Specific alcoholic beverages may contain naturally occurring constituents or contaminants that are carcinogenic, such as fusel oils, which are produced in the distillation process, polycyclic aromatic hydrocarbons, or nitrosamines. (2) Alcohol may irritate the mucosal lining and increase the number of rapidly dividing, susceptible cells. (3) Alcohol may facilitate the transport of carcinogens; for example, tobacco-associated carcinogens, across the mucosal lining. (4) Alcohol may damage the liver's ability to detoxify certain carcinogens. (5) Alcohol consumption may affect nutritional status by reducing intake and/or absorption of essential nutrients. (6) In conjunction with nutrient deficiencies and liver disease, alcohol may suppress the immune response.

Diet, Alcohol and Oral-Pharyngeal Cancer

Cancer of the oral cavity and pharynx is another cancer for which the interaction of diet and alcohol has been investigated. Several years ago Dr. Deborah Winn, Dr. William Blot, Gloria Gridley, Dr. Robert Hoover, Dr. Linda Pickle, and I, all at the National Cancer Institute, conducted a case-control study of oral and pharyngeal cancer among women in North Carolina.⁴² Mortality rates for these two cancers are usually high among white women in the Southeast US. A total of 227 cases and 405 controls, or their next of kin, were interviewed. Snuff dipping and cigarette smoking were the predominant risk factors, with the risk among the white women who dipped or smoked or did both being three to four times that among women who did neither.⁴² Fruit and vegetable intake was protective, with women in the lowest quartile of fruit and vegetable consumption having twice the risk of those in the highest quartile, after controlling for tobacco use.⁹ This relationship with diet was shown not to be an indirect result of alcohol use, dental health, or socioeconomic status. Since other food groups were not consistently associated with reduced risk, it seemed that a specific constituent of fruits and vegetables, perhaps carotenoids or vitamin C or fiber, might be involved, rather than generally poor nutrition.

Alcohol intake also was associated with increased risk of oral and pharyngeal cancer, with drinkers having about three times the risk of nondrinkers.⁴² The effect of alcohol was seen clearly only among the cigarette smokers. In Table 8 is shown for the smokers in the study, the interaction of alcohol consumption and fruit and vegetable intake. In general, the protection afforded by fruit and vegetable intake was seen among nondrinkers and drinkers, and the elevated risk associated with alcohol consumption was seen at various levels of fruit and vegetable consumption, which meant the two effects were distinct. Clearly the most elevated risk was the 14-fold increase noted among the relatively heavy drinkers who also consumed low levels of fruit and vegetables.

It is not possible to say on the basis of this study whether alcohol consumption increased the risk of oral and pharyngeal cancer in part by reducing the intake of whatever were the protective factors in fruits and vegetables. Certainly the heavy drinkers among North Carolina women did not consume anything near the levels of alcohol consumed by the heavy drinkers in our study of Washington, DC black men. Only 35% of the women in the North Carolina study had consumed 20 alcoholic beverages in their lifetime, and the median level of consumption among the drinkers was the equivalent of 1.5 fl oz of hard liquor per week. Among the men in the Washington, DC study 80% had consumed more than five drinks a week for more than a month, and the median level among the drinkers was the equivalent of 90 fl oz of hard liquor per week. However, because of their smaller size and lower physical activity levels, adult women generally maintain a lower level of caloric intake than adult men³⁸; and a small quantity of empty calories from alcohol could have a large relative impact. Other mechanisms have been proposed for the role of alcohol in the etiology of oral and pharyngeal cancer and include irritating the mucosal lining of the upper gastrointestinal tract and increasing its permeability to carcinogenic derivatives of tobacco.

Few Epidemiological Studies on Nutrient-Alcohol Interaction

Epidemiologic data have been presented on nutrient-alcohol interaction for only two cancers, esophageal and oral-pharyngeal cancer. At the National Cancer Institute analogous data for laryngeal and lung cancer is now being analyzed. I do not know of any other epidemiologic studies that address the issue of nutrient-alcohol interaction. Since the dietary risk factors are different for the various cancers, it is extremely likely that the quantitative relationships and biological mechanisms for nutrient-alcohol interaction also will differ. This dearth of data results from the fact that only in the last 5 to 10 years have epidemiologists begun to rigorously and systematically assess

TABLE 8. Smoking-Adjusted Relative Risk of Oral and Pharyngeal Cancer Among Cigarette Smokers by Fruit and Vegetable Intake and Ethanol Consumption

Ethanol consumption in hard liquor equivalents	Fruit and vegetable intake in servings/wk		
	≥21	11-20.9	<11
Nondrinker	1.0 (3, 16)*	2.1 (11, 28)	5.2 (10, 10)
<2 fl oz/day	2.4 (7, 19)	4.4 (14, 18)	2.8 (5, 9)
≥2 fl oz/day	4.4 (9, 11)	4.1 (13, 15)	13.6 (23, 9)

* In parentheses are the nos. of cases and controls.
fl oz: fluid ounces.

the role of diet in cancer etiology. However, the quantity and quality of research in this area is clearly improving. By the next American Cancer Society conference on diet, nutrition, and cancer, we will hopefully have more answers.

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