

Drinking practices and risk of squamous-cell esophageal cancer among Black and White men in the United States

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To evaluate whether the fivefold greater incidence rate of squamous-cell esophageal cancer in Black compared with White men is due to type of alcoholic beverage consumed or to other qualitative differences in alcohol consumption, we conducted a population-based case-control study with 373 males diagnosed with squamous-cell esophageal cancer (124 Whites and 249 Blacks) and 1,364 male controls (750 Whites and 614 Blacks) from three geographic areas in the United States. Included were all histologically confirmed cases newly diagnosed from 1 August 1986 through 30 April 1989, among White and Black men aged 30 to 79 years. Risks varied to some extent according to type of alcohol used, with beer a stronger contributor in Whites, and wine and liquor stronger contributors in Blacks. However, most of the differences in the odds ratios by type of alcohol and race were eliminated after controlling for average weekly amount of total alcohol consumed. Thus, while alcohol use in all forms is an important risk factor for squamous-cell esophageal cancer in Whites and Blacks, type of alcoholic beverage used does not appear to account for the racial differences in incidence. *Cancer Causes and Control* 1997, 8, 605-609

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Introduction

In the United States, incidence rates of squamous-cell esophageal cancer are more than five times higher among Black compared with White men (16.8 cf 3.0 per 100,000 population).¹ To investigate reasons for this large racial

disparity, we conducted a population-based case-control study of esophageal cancer among White and Black men in three areas of the US. A previous analysis² found that the risks were higher among Blacks than Whites at each

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level of drinking and smoking, suggesting racial differences in susceptibility to the carcinogenic effects of alcohol and tobacco. This paper evaluates whether the racial disparity in risk was influenced by the type of alcoholic beverage used or by other qualitative differences in alcohol consumption.

Materials and methods

Methods for case/control selection have been published in detail elsewhere.²In brief, eligible cases were Black and White men aged 30 to 79 years from Atlanta (GA), Detroit (MI), and New Jersey, diagnosed from 1 August 1986 through 30 April 1989 with histologically confirmed esophageal cancer. Controls were selected randomly using random-digit dialing techniques³ for controls aged 30 to 64 and randomized sampling from computerized listings of Medicare recipients provided by the Health Care Financing Administration for controls aged 65 to 79. The controls were selected to be similar to the expected age, race, gender, and area distribution of the cases. In-person interviews were completed for 68 percent of the cases and 76 percent of the controls. Reasons for nonresponse included: death (19 percent of cases, one percent of controls); illness (eight percent of cases, four percent of controls); and refusal (four percent of cases, 16 percent of controls). No proxy interviews with next-of-kin were conducted. The current analysis was restricted to the 124 White and 249 Black cases with squamous-cell esophageal cancer and the 750 White and 614 Black controls.

Data were analyzed using unconditional logistic regression.⁴ Race-specific adjusted odds ratios (OR) and 95 percent confidence intervals (CI) were obtained using the EPICURE programs for personal computers.⁵

Alcohol drinkers were defined as subjects who reported drinking at least one drink of beer, wine, or hard liquor per month for at least six months. All models included the selection factors of age and geographic area, and the potential confounding factors of recent annual family income, number of cigarettes smoked, and number of years of smoking cigarettes. Amount of each kind of alcohol (beer, wine, liquor) was adjusted for the average weekly amount consumed of the other two kinds. In additional analyses, amount of each kind of alcohol, and type and concentration of liquor were adjusted for the average weekly amount of total alcohol consumed from beer, wine, and liquor. To test for linear trend, the categorical alcohol variables were treated as continuous variables in the race-specific logistic models, with each level represented by the median value of that category in the control group. To evaluate whether risks for type of alcohol were significantly different for Blacks and Whites, the race variable and interaction terms combining race and the intensity variables were added to logistic models

containing data for Blacks and Whites combined.

Results

Race-specific adjusted ORs associated with consumption of specific types of alcoholic beverages are presented in Table 1. Use of beer was a heavier contributor to the risk in White men (OR = 4.4, CI = 2.2-9.0) than in Black men (OR = 1.4, CI = 0.9-2.1) (data not shown), with a significant dose gradient present only for Whites. There was a significant interaction by race in the ORs associated with number of beers consumed per week ($P = 0.03$) and a significantly greater percentage of White (10.7 percent) than Black (6.6 percent) controls reported drinking 29 or more beers per week ($P = 0.03$). When risks for beer consumption were adjusted for total alcohol consumption, risks remained slightly elevated for Whites, but not for Blacks (Table 2).

An elevated risk associated with use of wine was seen for Blacks (OR = 1.7, CI = 1.2-2.6) but not for Whites (OR = 0.5, CI = 0.3-0.8) (data not shown). There was a significant dose gradient with increasing wine consumption in Blacks, with the risk reaching 2.5 for the heaviest consumers (Table 1), but dropping to 1.2 after adjustment for total alcohol consumption (Table 2). A significant interaction ($P = 0.01$) was seen between race and amount of wine consumed.

Risks associated with ever use of liquor were greater for Blacks (OR = 4.7, CI = 2.6-8.4) than for Whites (OR = 1.7, CI = 1.0-2.7) (data not shown). ORs for number of drinks per week reached 10.0 and 6.7 in the highest consumption category for Blacks and Whites, respectively (Table 1). The interaction between intensity of drinking and race was of marginal significance ($P = 0.07$). Among liquor drinkers, a significantly greater percentage of Black (16.9 percent) compared with White (5.2 percent) controls were in the highest use category ($P < 0.001$). Risks were somewhat higher for consumption of light liquor (e.g., vodka and gin) than for dark liquor (e.g., whiskey, bourbon, scotch, rye). For both races combined, risk was 70 percent higher for subjects who drank their liquor straight instead of with ice, water, or a mixer (OR = 1.7, CI = 1.2-2.4). When the risks associated with heavy liquor use were adjusted for total alcohol consumption, the ORs were greatly reduced, but a residual risk of around 2.5 remained for both Black and White men (Table 2).

Use of moonshine was reported more often by Black cases (32.9 percent) and controls (21.5 percent) than by White cases (4.8 percent) and controls (4.1 percent) (data not shown). The ORs were not elevated for either race (OR = 0.8, CI = 0.5-1.3 for Blacks and OR = 0.5, CI = 0.2-1.4 for Whites) when controlled for total alcohol consumption (excluding moonshine). Risks associated with moonshine consumption also were not elevated for

Table 1. Risk of squamous-cell esophageal cancer according to type of alcoholic beverage and race

Type of alcohol (servings)	White				Black			
	No. of Cases ^a	No. of Controls ^a	OR ^b	(CI)	No. of Cases ^a	No. of Controls ^a	OR ^b	(CI)
Beer^c								
Never drank	10	274	1.0	—	56	251	1.0	—
< 8 per week	24	252	2.6	(1.1-5.8)	79	196	1.3	(0.8-2.2)
8-14.9 per week	19	95	4.3	(1.8-10.2)	44	78	1.3	(0.7-2.3)
15-28.9 per week	35	71	8.2	(3.6-18.9)	40	53	1.6	(0.8-2.9)
29+ per week	36	50	6.6	(2.8-15.6)	23	23	1.5	(0.6-3.5)
			<i>P</i> < 0.001				<i>P</i> > 0.05	
Wine^c								
Never drank	96	492	1.0	—	152	493	1.0	—
< 8 per week	17	205	0.6	(0.3-1.1)	37	80	1.5	(0.9-2.6)
8-14.9 per week	4	29	0.7	(0.2-2.4)	16	12	2.3	(0.9-6.0)
15+ per week	7	16	1.1	(0.3-3.4)	37	16	2.5	(1.2-5.2)
			<i>P</i> > 0.05				<i>P</i> < 0.001	
Liquor^c								
Never drank	37	342	1.0	—	16	223	1.0	—
< 8 per week	26	253	0.9	(0.5-1.6)	21	142	1.7	(0.8-3.6)
8-14.9 per week	9	76	0.9	(0.4-2.2)	40	94	3.8	(1.9-7.7)
15-28.9 per week	24	50	2.4	(1.2-4.8)	70	78	8.2	(4.2-16.3)
29+ per week	28	21	6.7	(3.0-15.1)	95	64	10.0	(5.0-19.9)
			<i>P</i> < 0.001				<i>P</i> < 0.001	
Type^d								
Dark	63	291	1.0	—	129	231	1.0	—
Light	15	78	1.2	(0.6-2.5)	73	78	1.6	(1.0-2.6)
Concentration^d								
Diluted	48	317	1.0	—	115	273	1.0	—
Straight	35	71	1.7	(0.9-3.1)	97	93	1.6	(1.0-2.5)

^a Excludes subjects with unknown values.

^b OR = odds ratio; CI = 95% confidence interval. All estimates are relative to subjects who never drank that type of alcoholic beverage.

^c Estimates are adjusted for age, area, smoking, and income, and each type of alcoholic beverage is adjusted for amount of the other two.

^d Estimates are adjusted for age, area, smoking, income, and total alcohol.

the heaviest consumers (data not shown). Risk among Blacks varied considerably by area. ORs were 2.1 (CI = 0.8-5.2) in Atlanta, 1.0 (CI = 0.05-1.9) in Detroit, and 0.2 (CI = 0.1-0.5) in New Jersey. The proportion of Black controls who had ever consumed moonshine also showed substantial variation across areas: 31.2 percent in Atlanta, 23.6 percent in Detroit, and 13.8 percent in New Jersey.

Discussion

This population-based case-control study evaluated whether variations in the consumption of different types of alcoholic beverages account for higher incidence rates of squamous-cell esophageal cancer among Black than White men. Risks varied to some extent according to type of alcohol consumed, with beer a stronger contributor in Whites and wine and liquor stronger contributors in

Blacks. However, most of the differences in the ORs and the racial disparity in risks were eliminated after control for average weekly amount of total alcohol consumed. Since it was not possible to use nonalcohol drinkers as the referent (only two White and three Black cases claimed to be nondrinkers), some of the remaining differences in risk could have resulted from the inability to control baseline categories for the other types of alcohol. In agreement with previous reports, we found a 70 percent excess risk for men who drank their liquor straight instead of diluted with water, ice, or a mixer,^{6,7} and no additional risk for consumption of dark instead of light liquor.⁸ These findings extend previous reports suggesting that risk of squamous-cell esophageal cancer is associated with alcohol consumption *per se*, rather than to the presence of substances such as contaminants, flavoring compounds, or additives that may vary among types of

Table 2. Risk of squamous-cell esophageal cancer according to type of alcoholic beverage and race adjusted for total alcohol consumption

Type of alcohol (servings)	White				Black			
	No. of Cases ^a	No. of Controls ^a	OR ^{b,c}	(CI)	No. of Cases ^a	No. of Controls ^a	OR ^{b,c}	(CI)
Beer								
Never drank	10	274	1.0	—	56	251	1.0	—
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15-28.9 per week	35	71	2.5	(0.9-6.6)	40	53	0.7	(0.3-1.3)
29+ per week	36	50	1.4	(0.4-4.2)	23	23	0.4	(0.2-1.0)
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Wine								
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			<i>P</i> > 0.05				<i>P</i> > 0.05	

^a Excludes subjects with unknown values.

^b OR = odds ratio; CI = 95% confidence interval. All estimates are relative to subjects who never drank that type of alcoholic beverage.

^c Estimates are adjusted for age, area, smoking, income, and total alcohol consumption.

alcoholic beverages.⁹

A study of esophageal cancer in the high risk area of coastal South Carolina revealed an elevated risk associated with use of moonshine (home-brewed) whiskey, particularly among Blacks.¹⁰ In the current study, use of moonshine appears to contribute little to the excess risk among Blacks in the overall study population, but the elevated risk for moonshine use in the Atlanta area appears consistent with the previous study in South Carolina. This suggests that regional variation in moonshine use may contribute to the high rates of esophageal cancer among Blacks in Atlanta even though rates of esophageal cancer for Black males in Atlanta do not exceed those in Detroit and New Jersey.

This study adds to the accumulating evidence that alcoholic beverages are carcinogenic to humans and the major contributor to the etiology of esophageal cancer in Western populations.⁹ There was some variability in risks by type of alcoholic beverage used and other qualitative patterns of drinking that may reflect culturally or economically determined drinking habits. The differences were relatively minor, however, and consistent with the notion that alcohol in all forms is a risk factor for esophageal cancer.

In a previous analysis,² we found that while total alcohol was consumed slightly more often by Black than White controls, the difference was too small to account for any meaningful proportion of the excess of squamous-cell carcinoma of the esophagus in Blacks. That analysis did note, however, that the significantly higher risks for Blacks compared with Whites at each level of tobacco and alcohol use could explain a substantial amount of the racial difference in incidence rates.

The results of this analysis suggest that the significant racial interaction between total alcohol consumption and risk of esophageal cancer is not due to differential behavior in drinking habits of Blacks of Whites. Other mechanisms, such as differences in genetic susceptibility to the carcinogenic effects of alcohol as well as tobacco on the esophageal mucosa, and the possible interaction of alcohol and tobacco with nutritional inadequacies need to be investigated. Whatever the mechanisms involved, it is clear that a reduction in use of alcoholic beverages would substantially lower the incidence of this deadly disease in all population groups.

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