

ORIGINAL REPORTS: HISPANIC HEALTH

IS ALCOHOL INTAKE ASSOCIATED WITH BREAST CANCER IN HISPANIC WOMEN? THE NEW MEXICO WOMEN'S HEALTH STUDY

Objective: Previous studies have shown an increased breast cancer risk associated with modest or high alcohol intake, however, few of these studies have included Hispanic women. The alcohol/breast cancer association was investigated in a New Mexico (NM) statewide bi-ethnic study.

Design: A population-based, case-control study.

Methods: Incident breast cancer cases ($N = 712$), aged 30–74 years, were ascertained by the New Mexico Tumor Registry (NMTR). Controls ($N = 844$) were identified by random digit dialing and were frequency-matched for ethnicity, age-group, and health planning district. Data were collected via in-person interview, which included questions regarding recent and past alcohol intake and breast cancer risk factors.

Results: The highest level of recent alcohol intake, compared to no intake, was associated with breast cancer risk for postmenopausal Hispanic women (odds ratio [OR] = 2.0 95% confidence interval [CI] 0.8–5.1, 42+ grams/week) and postmenopausal non-Hispanic White women (OR = 2.2, 95% CI 1.0–5.0, 148+ grams/week), although estimates were unstable and statistically non-significant. Lower recent alcohol intake (<148 grams/week) was associated with reduced risk for non-Hispanic Whites (OR = 0.49, 95% CI 0.35–0.69). This pattern was independent of hormone-receptor status and was present for both premenopausal (OR = 0.29, 95% CI 0.15–0.56) and postmenopausal women (OR = 0.56, 95% CI 0.35–0.90). Results for past alcohol intake and breast cancer association did not demonstrate any trends and were non-significant.

Conclusions: Alcohol intake does not appear to have a consistent or significant association with breast cancer in Hispanic women. (*Ethn Dis.* 2002;12:460–469)

Key Words: Breast Cancer, Case-Control Study, Hispanic, Alcohol, Hormone-Receptor Status

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INTRODUCTION

Breast cancer is the most frequently occurring cancer among Hispanic women.¹ Although breast cancer incidence and mortality rates for Hispanic women are lower than those for non-Hispanic White women, they have increased more rapidly among Hispanic women in recent decades.^{2–6} Age-adjusted incidence rates for New Mexican Hispanic women increased from 57/100,000 to 74/100,000 for the time-periods 1983–1987 and 1988–1992, respectively, compared with 96/100,000 to 107/100,000, respectively, for non-Hispanic White women.⁷

Few data are available on breast cancer risk factors for Hispanic women^{2,3,8–11} or on hormone-receptor status

by ethnicity.^{12,13} Studies among non-Hispanic White women have demonstrated increased risk (30%–70%) of breast cancer associated with modest to high alcohol intake among both premenopausal and postmenopausal women.^{14–16} Any consumption of alcohol, whether recent, past, or lifetime, reportedly increases the risk of breast cancer.^{14,16–18} However, recent intake may be most important, possibly acting as a late-stage promoter.^{14,16,19} Biological data support the hypothesis that high alcohol intake may increase breast cancer risk by increasing serum concentrations of estrogens in both premenopausal and postmenopausal women.^{20–23} This alcohol-estrogen association may be due to a change in estrogen metabolism,²³ or to a co-carcinogenic effect.²⁴ Estrogen-receptor (ER) or progesterone-receptor (PR) status is an important biological characteristic of breast tumors, and is associated with response to endocrine therapy and prognosis, and possibly with different etiologic risk factors,²⁵ including alcohol consumption.^{26,27} Stimulation of estrogen receptor activity by ethanol as well as down-regulation of the tumor suppressor gene BRCA1, has been reported in human breast cancer cell lines.²⁸

The 'New Mexico Women's Health Study' (NMWHS), a statewide population-based case-control study, was initiated in 1992 to investigate etiologic risk factors for breast cancer among Hispanic and non-Hispanic White women. In this study, we examined the association of alcohol consumption with breast cancer risk by menopausal status and hor-

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*Biological data support the hypothesis that high alcohol intake may increase breast cancer risk by increasing serum concentrations of estrogens in both premenopausal and postmenopausal women.*²⁰⁻²³

mone-receptor status among Hispanic and non-Hispanic White women.

METHODS

Subject Recruitment

Women newly diagnosed with an invasive or *in situ* breast carcinoma were eligible for inclusion in the NMWHS based on the following criteria: aged 30–74 years, diagnosed between 1992–1994, and residing in New Mexico at the time of diagnosis. Cases were rapidly ascertained through the New Mexico Tumor Registry (NMTR), a member of the National Cancer Institute's Surveillance, Epidemiology and End Results Program (SEER). New Mexico has the largest percentage of Hispanics per capita (40%) in the United States,²⁹ accounting for the second largest (10%) sector of Hispanic SEER coverage.⁶ All eligible Hispanic cases were included, and a 33% random sample of non-Hispanic White cases was identified for inclusion. The sampling fraction for non-Hispanic White cases was chosen to give a distribution similar to the age-groups (30–39, 40–64, and 65–74 years), and geographic distribution (7 state health planning districts) of Hispanic cases ascertained by the NMTR during the period 1988–1990. Of these, 491 eligible Hispanic breast cancer cases were ascertained. Random selection of non-His-

panic Whites resulted in 493 cases. Of the eligible cases, 332 Hispanic women (68%) and 380 non-Hispanic White women (77%) completed in-person interviews.

Controls were frequency-matched on the basis of Hispanic and non-Hispanic White ethnicity, age-group, and health planning district. Women were recruited by using a modified approach to the Waksberg random digit dialing method.³⁰ Details on these procedures and results have been previously reported.¹¹ Of the 8,147 working telephone numbers that were contacted, 4,459 were residential. There were a total of 1,039 eligible controls ascertained from 3,400 respondents who completed the telephone screening interview to ascertain eligibility: 511 Hispanic and 528 non-Hispanic White women. Of these, 388 (76%) Hispanic and 456 (86%) non-Hispanic White women completed in-person interviews.

Data Collection

Information on recent dietary and alcohol intake was collected at the beginning of the interview using a modified version of a quantitative food frequency questionnaire designed by the staff of the Human Nutrition Center at the University of Texas-Houston, School of Public Health. This questionnaire was developed using standard protocols and included 140 items.^{31,32} Modifications, based on an analysis of food intake recalls of 100 women, were made by one of the authors (RSM) to add foods that were important sources of nutrients among New Mexico women. All subjects were asked to recall usual food intake for a 4-week period, 6 months prior to the interview. Women were asked to consider whether the foods eaten during this month reflected how they usually ate and, if not, whether this was due to a hospitalization or a major illness lasting more than 4 days, due to breast cancer treatment, or due to some other reason. Using a calendar, a time-period prior to the 6-month win-

dow was established, and women were asked to select the month during which their diet was typical. Frequency of use information included consumption on a per month (28 day), per week, or per day basis. Two-dimensional food models were used to aid in the determination of portion size consumed. Frequency of consumption and portion size data were entered into an analysis program containing gram weight and nutrient data used to calculate nutrient estimates per food per day, and total nutrient intake per day.^{33,34} Pilot study data were used to assess the validity and reproducibility of dietary intake as measured by the food frequency questionnaire during the past month, and intake for the same month, as recalled 6 months later.³⁵

A risk factor questionnaire was used to collect data on demographic characteristics and breast cancer risk factors using a calendar to record major life events as an assist to recall. Hormone receptor status was abstracted from hospital records. The NMWHS was approved by the University of New Mexico's Human Research Review Committee. In-person interviews were conducted in Spanish or English according to the participant's preference.

Statistical Analysis

The alcohol exposure variables investigated included recent intake collected on the food frequency questionnaire, and history of alcohol consumption as collected on the risk factor questionnaire. Recent alcohol intake, as measured by the food frequency questionnaire, was expressed as the average daily consumption of wine, beer, and liquor, and converted to a weekly intake for analysis. The ethanol content for each type of beverage was based on the amount reported in the US Department of Agriculture (USDA) Nutrient Database for Individual Intake Surveys: 8.1 grams(g)/alcohol for one 3½-ounce glass of wine; 12.6 g/alcohol for one serving of beer; and 21.2 g/alcohol for one liquor drink.³⁴ Alcohol abstinence was

defined as an intake of 0 g/day. Past alcohol intake included ever vs never used, status of alcohol consumption at time of interview (non-drinker, current drinker, former drinker), age at first use, years since last consumption, years of drinking, frequency of drinking (daily, weekly, monthly, yearly), gram intake per week at ages 25, 35, and 50, and average lifetime intake based on total consumption for the latter 3 ages. The number of drinks per week for subjects reporting consumption on a monthly or yearly basis was estimated based on the frequency midpoint divided by the number of weeks per time interval. The ethanol content in grams was multiplied by the number of weekly drinks per beverage type to estimate gram intake per week. Specific beverage type was not analyzed, because there has not been consistent evidence to suggest an effect independent of ethanol content.¹⁹

Covariates in previous studies of alcohol consumption and breast cancer risk were included as potential confounders.^{14,16-18,26,36-40} These included: education; age at menarche; age at first full-term birth for pregnancies lasting 6 months or longer, regardless of pregnancy outcome; number of full-term births lasting 6 months or longer (single birth, multiple birth, stillbirth); cumulative months of lactation for all children; cumulative years of oral contraceptive use; menopausal status; history of fibrocystic disease; breast cancer in mother, sister, or daughter; history of cigarette smoking lasting for more than 6 months; usual adult BMI; physical activity level; energy intake; and energy-adjusted total fat intake.

Age, defined as age at diagnosis for cases and age at interview for controls, was included in all models to adjust for residual age differences between cases and controls. Premenopausal and postmenopausal categories included women with a history of natural or surgical menopause. Women who could not be classified as pre- or postmenopausal were excluded from the stratified analyses. The criteria

used to classify menopausal status have been described elsewhere in a previous analysis of reproductive factors.¹¹ Categories of menopausal status (premenopausal, postmenopausal, surgical, unknown) were based on self-report of menstrual history, history of hysterectomy, with or without oophorectomy, and use of estrogen replacement therapy. Women who reported a hysterectomy without bilateral oophorectomy within one year of their last menstrual period, and who did not take estrogen within one year of surgery were considered to have an unknown menopausal status. Ethnic-specific distributions of age at menopause among controls were used to assign menopausal status for women with an unknown status; 43 years (<10th percentile) for premenopausal women, and 54 years (>90th percentile) for postmenopausal women. Usual adult body mass index (BMI) was calculated as self-reported weight in kilograms (kg)/height in meters squared (m²). Metabolic equivalents (METs) were calculated for physical activity as kilocalories (kcal)/kg of weight/hour,⁴¹ and multiplied by the mean number of hours per week to compute final METs. Total fat intake was energy-adjusted based on the residual method⁴² in order to remove the correlation with energy intake. Adjustment reduced the Spearman rank correlation coefficient from 0.91 to 0.02. Alcohol intake was not energy-adjusted as it was weakly correlated with total energy intake ($r = 0.15$), as demonstrated in other studies.⁴²

Subjects with an energy intake outside the range of 500–6,000 kcal were excluded. Exclusions were due to 16 subjects with an energy intake >6,000 kcal/day, all but one of whom had a low alcohol intake <10 g/day. Seven subjects were excluded due to incomplete or no food frequency data. Women with an unknown ($N = 84$) menopausal status were coded as unknown for analyses based on all women. Subjects were deleted due to missing data for education ($N = 5$), age at menarche

($N = 6$), cumulative months of lactation ($N = 5$), cumulative years of oral contraceptive use ($N = 10$), and BMI ($N = 15$). Nine controls were dropped from the conditional logistic regression analyses because there were no frequency-matched cases within those specific strata.

Conditional logistic regression was used to determine age-adjusted and multivariate-adjusted ORs and corresponding 95% CIs for alcohol exposure variables.⁴³ Ethnic-specific logistic regression analyses were conditioned on the matching factors of age-group and health planning district. Polytomous logistic regression analysis was used to estimate ORs for the joint classification of hormone-receptor status, when both receptors were known, relative to controls.⁴³ The change-in-estimate method ($\geq 10\%$ change in the OR) was used to identify the most important confounders within each ethnic and menopausal specific model;⁴⁴ however, final models included all covariates to allow comparison of results between ethnic and menopausal status groups and with previous studies. Interaction between menopausal status and alcohol intake was investigated by comparing models, with and without interaction terms, using the log likelihood test statistic.⁴³ Analyses were computed using STATA software.⁴⁵

RESULTS

The mean age of cases at diagnosis was 54 years, compared with 53 years for controls at the time of interview. The median time from diagnosis to interview for cases was 193 days; 201 days for Hispanic women, and 183 days for non-Hispanic White women. Distribution of breast cancer risk factors is shown in Table 1. Compared with the non-Hispanic White women, the Hispanic women were generally younger at their first full-term birth, had a higher parity, higher BMI, less education, and were less likely to report either a history

Table 1. Distribution of breast cancer risk factors, stratified by ethnicity, New Mexico Women's Health Study, 1992-1994

Risk Factor	Hispanic				Non-Hispanic White			
	Cases (N=322)		Controls (N=388)		Cases (N=380)		Controls (N=456)	
	N*	%	N	%	N	%	N	%
Education (years)								
<12	104	31	86	22	24	6	29	6
12	129	39	150	39	102	27	111	24
>12	96	29	152	39	253	67	315	69
Age (years) at menarche								
≤12	133	40	170	44	185	49	211	46
13	101	30	109	28	111	29	140	31
≥14	95	29	108	28	84	22	103	23
Age (years) at first full-term birth								
≤18	71	11	89	8	43	16	67	16
19-20	71	21	94	23	60	11	73	15
21-22	50	21	64	24	59	16	64	16
23-26	62	15	68	17	82	16	95	14
≥27	40	19	43	18	76	22	84	21
Nulliparous	38	12	30	11	60	20	73	18
Number of full-term births								
Nulliparous	38	11	30	8	60	16	73	16
1	29	9	35	9	63	17	58	13
2	66	20	98	25	128	34	138	30
3	80	24	72	19	66	17	101	22
≥4	119	36	153	39	63	17	86	19
Cumulative months of lactation								
Nulliparous	38	11	30	8	60	16	73	16
Parous, 1-12	109	33	128	33	167	44	157	34
Parous, >12	52	16	82	21	43	11	99	22
Parous, never	133	40	145	37	110	29	125	27
Cumulative years of oral contraceptive use								
Never used	149	45	146	38	146	38	155	34
<1.5	59	18	82	21	80	21	67	15
1.5-5	54	16	75	19	67	18	114	25
>5	67	20	84	22	83	22	118	26
Menopausal status								
Premenopausal	131	40	154	40	116	31	186	41
Postmenopausal	178	54	219	56	239	63	249	55
Surgical unknown	21	6	14	4	24	6	21	5
History of fibrocystic disease								
No	287	86	348	90	285	75	379	83
Yes	45	14	40	10	95	25	77	17
Breast cancer in mother, sister, daughter								
No	292	88	352	91	317	83	402	88
Yes	40	12	36	9	63	17	54	12
Cigarette smoking								
No	187	56	202	52	195	51	216	47
Yes	145	44	186	48	185	49	240	53
Body mass index (kg/m ²)†								
<21.1	35	11	75	19	119	31	134	29
21.1-<23.0	65	20	73	19	126	33	132	29
23.0-<25.6	95	29	109	28	68	18	103	23
≥25.6	133	40	124	32	65	17	85	19
Vigorous physical activity (METS/week)‡								
None/non-vigorous	148	45	106	27	108	28	87	19
Light, <13	92	28	110	28	95	25	142	31
Moderate, 13-<35	47	14	76	20	104	27	118	26
Heavy, ≥35	45	14	96	25	73	19	109	24

Table 1. Continued

Risk Factor	Hispanic				Non-Hispanic White			
	Cases (N=322)		Controls (N=388)		Cases (N=380)		Controls (N=456)	
	N*	%	N	%	N	%	N	%
Energy intake (kilocalories/day)								
<1608	68	21	87	22	105	28	79	17
1608-<2018	59	18	58	15	86	23	108	24
2019-<2436	72	22	71	18	68	18	95	21
2436-<3032	56	17	79	20	59	16	87	19
≥3032	71	21	84	22	59	16	82	18
Total fat intake (grams/day)§								
<58	75	23	80	21	106	28	86	19
58-<75	64	19	62	16	89	23	104	23
75-<96	63	19	78	20	67	18	88	19
96-<123	57	17	82	21	53	14	84	18
≥123	67	20	77	20	62	16	89	20

* Numbers (No.) may not sum to total for all covariates because of missing data.

† kg/m², kilograms/meters squared.

‡ METS, metabolic equivalents, based on expenditure of kilocalories/kilogram of weight/hour. Physical activities included: walking/hiking, running/jogging, exercise class, biking, dancing, lap swimming, tennis, squash/racquetball, calisthenics/rowing, bowling, golf, softball/baseball, basketball, volleyball, housework, and heavy outside work.

§ Absolute intake.

of fibrocystic disease or a family history of breast cancer. Hispanics reported a lower level of physical activity compared to non-Hispanic Whites. In general, Hispanic women, compared with non-Hispanic White women, reported higher median levels of daily total energy intake (2,257 vs 2,107 kcals/day) and total fat intake (85 vs 80 g/day) (data not shown).

Patterns for age-adjusted ORs for breast cancer risk factors differed by ethnicity. For Hispanic women, a high BMI was the strongest statistically significant risk factor (OR = 2.38, 95% CI 1.46–3.87 for ≥25 kg/m²), and vigorous physical activity was the strongest protective factor (OR = 0.34, 95% CI 0.22–0.54 for ≥35 METS/week) (data not shown). Among non-Hispanic Whites, a positive history of fibrocystic disease (OR = 1.68, 95% CI 1.18–2.39) was the strongest risk factor, whereas 12 months or more of lactation (OR = 0.53, 95% CI 0.34–0.84), and vigorous physical activity (OR = 0.55, 95% CI 0.36–0.84 for ≥35 METS/week) were strong protective factors (data not shown).

A reproducibility analysis, based on the pilot data, showed an overall Spearman correlation coefficient of 0.83 between alcohol intake during the past month and intake for the same month, recalled 6 months later. Results were comparable for cases ($r = 0.82$) and noncases ($r = 0.85$), but results were lower for Hispanics ($r = 0.73$) compared with non-Hispanic Whites ($r = 0.87$). Overall, 42% of cases and 48% of controls reported recent alcohol intake; of these women, 55% of Hispanics and 38% of non-Hispanic Whites reported an intake of no more than one drink per week. A history of past alcohol consumption was reported by 77% of Hispanic women and 88% of non-Hispanic Whites; cases (81%) were similar to controls (85%) for past intake.

Recent Alcohol Intake

The age-adjusted OR for recent alcohol intake was 1.14 (95% CI 0.56–2.29) for Hispanic women consuming ≥85 g/week (5+ drinks/week), and 1.42 (95% CI 0.82–2.46) for non-Hispanic White women consuming ≥148 g/week (8+ drinks/week), as compared

to non-drinkers (Table 2). Multivariate adjustment increased these ORs to 1.35, and 1.56, respectively (Table 2), primarily due to education and BMI among Hispanic women and cigarette smoking among non-Hispanic White women. A low level of recent alcohol intake (<8 drinks/week) was consistently associated with a reduced risk of approximately 50% in the multivariate full model for non-Hispanic White women and, overall, there was no evidence of a significant alcohol effect on breast cancer risk in Hispanic women (Table 2).

There was an increased risk of breast cancer associated with the highest alcohol intake category among postmenopausal women, but the estimate was unstable and statistically non-significant (Table 3). Among non-Hispanic White women, there was a statistically significant reduced risk for breast cancer among women reporting a low alcohol intake (8 drinks/week) compared to nondrinkers which was present for both premenopausal and postmenopausal groups (Table 3).

Table 2. Odds ratios (OR)* and 95% confidence intervals (CI)† for age-adjusted models‡, and multivariate-adjusted full models§ for breast cancer risk associated with recent alcohol intake, based on a food frequency questionnaire, stratified by ethnicity, New Mexico Women’s Health Study, 1992–1994

Recent Alcohol Intake (grams/week) ,¶,#	Hispanic						Non-Hispanic White					
			Age-Adjusted		Multivariate-Adjusted				Age-Adjusted		Multivariate-Adjusted	
	Cases N	Controls N	OR	95% CI	OR	95% CI	Cases N	Controls N	OR	95% CI	OR	95% CI
Nondrinker**	212	236	1.00		1.00		189	188	1.00		1.00	
<8	33	43	0.89	0.54–1.47	1.21	0.68–2.15	34	47	0.71	0.43–1.16	0.49	0.28–0.85
8–<21 (1 drink)	28	38	0.77	0.45–1.32	1.00	0.54–1.85	33	57	0.57	0.35–0.93	0.46	0.27–0.79
21–<42 (2 drinks)	22	29	0.80	0.44–1.47	0.75	0.37–1.53	31	54	0.60	0.36–0.99	0.44	0.25–0.77
42–<85 (3–4 drinks)	13	15	0.95	0.43–2.06	1.24	0.52–2.93	35	49	0.71	0.43–1.16	0.60	0.35–1.05
85–<148 (5–7 drinks)	18	18	1.14	0.56–2.29	1.35	0.63–2.93	17	29	0.58	0.30–1.12	0.49	0.24–1.00
≥148 (8+ drinks)							38	27	1.42	0.82–2.46	1.56	0.85–2.86

* Odds ratio.

† 95% confidence interval.

‡ Conditional logistic regression models matched for age-group and health planning district, and adjusted additionally for age.

§ Conditional logistic regression models matched for age-group, health planning district, and adjusted for age, education, age at menarche, menopausal status, age at first full-term birth, number of full-term births, cumulative months of lactation, cumulative years of oral contraceptive use, history of fibrocystic disease, breast cancer in mother, sister, daughter, cigarette smoking, body mass index, physical activity, energy intake, and energy-adjusted total fat intake.

|| Absolute intake.

¶ Categories for 5–7 drinks and 8+ drinks combined for Hispanic women, due to low level of alcohol intake for Hispanic women.

Recent alcohol intake data missing or excluded for 9 cases and 14 controls.

** No intake in 4-week period, 6 months in past.

Table 3. Multivariate-adjusted (OR)* and 95% confidence intervals (CI)† for breast cancer risk associated with recent alcohol intake, collapsed into fewer categories, based on a food frequency questionnaire stratified by ethnicity and menopausal status, New Mexico Women’s Health Study, 1992–1994

Recent Alcohol Intake	Low		Medium		High	
	OR	95% CI	OR	95% CI	OR	95% CI
Hispanic‡						
Nondrinker	1.00		1.00		1.00	
All	1.21	0.68–2.15	0.88	0.54–1.45	1.31	0.72–2.38
Premenopausal	1.69	0.67–4.30	0.70	0.32–1.51	0.96	0.36–2.60
Postmenopausal	0.89	0.37–2.14	0.96	0.42–2.18	2.03	0.81–5.09
Non-Hispanic White§						
Nondrinker	1.00				1.00	
All	0.49	0.35–0.69			1.55	0.84–2.83
Premenopausal	0.29	0.15–0.56			1.08	0.32–3.64
Postmenopausal	0.56	0.35–0.90			2.23	0.99–5.03

* Odds ratios estimated from conditional logistic regression models matched for age-group, health planning district, and adjusted for age, education, age at menarche, age at first full-term birth, number of full-term births, cumulative months of lactation, cumulative years of oral contraceptive use, history of fibrocystic disease, breast cancer in mother, sister, daughter, cigarette smoking, body mass index, physical activity, energy intake, and energy-adjusted total fat intake.

† 95% confidence interval.

‡ Hispanic, levels of recent alcohol intake (grams/week): low=<8 (<1 drink); medium=8–<42 (1–2 drinks); high=42+ (3+ drinks).

§ Non-Hispanic White, levels of recent alcohol intake (grams/week): low=<148 (<8 drinks); high=148+ (8+ drinks).

|| Menopausal status included in these models.

Hormone-Receptor Status and Recent Alcohol Intake

The distribution for ethnic-specific hormone-receptor status was similar with the exception of ER–/PR– (24% for Hispanic vs 17% for non-Hispanic White). About 40% of whom from both ethnic groups were ER+/PR+; 10% to 12% were ER+/PR–; 3% were ER–/PR+; and 9% to 12% were unknown. In polytomous logistic regression analysis, based on the collapsed categories for recent alcohol intake, the status groups of ER+/PR+ and ER–/PR– were compared simultaneously with the controls (Table 4). Stratification was limited to ethnicity as stratum-specific numbers were too small to additionally stratify by menopausal status. The direction of the ORs was similar for the 2 hormone-receptor status groups (Table 4). The effect estimate for Hispanics was increased by almost 80% for ER+/PR+, but was not statistically significant. Odds ratios for ER+/PR+ status were statistically significant for both low and high alcohol intake for non-Hispanic White women (Table 4). An increased risk for non-Hispanic Whites

Table 4. Multivariate-adjusted (OR)* and 95% confidence intervals (CI)† for breast cancer risk associated with recent alcohol intake, based on a food frequency questionnaire, stratified by ethnicity and joint estrogen/progesterone receptor status, New Mexico Women's Health Study, 1992–1994

Recent Alcohol Intake (grams/week)‡	Controls N§	ER+PR+			ER-PR-		
		N	OR	95% CI	N	OR	95% CI
Hispanic							
Non-drinker	236	80	1.00		50	1.00	
<8	43	10	0.83	0.35–1.98	9	1.04	0.39–2.79
8–<42 (1–2 drinks)	67	20	0.97	0.49–1.91	7	0.39	0.14–1.08
≥42 (3+ drinks)	33	18	1.78	0.86–3.68	9	1.43	0.55–3.74
Non-Hispanic White							
Non-drinker	188	72	1.00		33	1.00	
<148 (<8 drinks)	236	59	0.46	0.28–0.74	27	0.37	0.19–0.73
≥148 (8+ drinks)	27	22	2.13	1.03–4.43	5	1.62	0.51–5.18

* Odds ratio estimated from logistic regression models adjusted for matching variables (age-group, health planning district), and for age, education, age at menarche, menopausal status, age at first full-term birth, number of full-term births, cumulative months of lactation, cumulative years of oral contraceptive use, history of fibrocystic disease, breast cancer in mother, sister, daughter, cigarette smoking, body mass index, physical activity, energy intake, and energy-adjusted total fat intake.

† 95% confidence interval.

‡ Absolute intake.

§ Recent alcohol intake data missing for 14 controls, 5 cases with ER+PR+ status breast cancer, and 4 cases with ER-PR- status breast cancer. The remaining cases, also excluded from analysis, were categorized as: ER+PR- (N=77), ER-PR+ (N=20); hormone-receptor determination not done (N=108); and either results borderline or unknown (N=77).

associated with an intake of 8+ drinks per week was 50% higher for ER+PR+ compared with ER-PR- status, but the difference was not statistically significant (Table 4).

Past Alcohol Intake

Multivariate results for ever vs never consuming alcohol, age at first use, and duration of drinking, did not show a significant association with breast cancer risk (Table 5). Test for interaction between menopausal status and ever vs never consuming alcohol was not statistically significant ($-2 \log$ likelihood test statistic: $\chi^2 = 1.56$, $P=0.21$, data not shown). There was no alcohol-breast cancer association for lifetime average intake or for ages 25, 35, and 50 (data not shown).

A minimal risk for former drinkers was present (Table 5). This was due primarily to 44 cases who reported cessation of drinking within the year of diagnosis; risk decreased as years since last alcohol consumption increased (Table 5). These cases were distributed evenly

by ethnicity and exhibited more severe disease (regional/remote) at diagnosis (58% vs 30%). They were younger (48 vs 55 years), reported the lowest level of average lifetime alcohol intake (32 vs 36 g/week), the lowest intake at previous ages (2.0 vs 2.5 g/week at age 25; 2.3 vs 2.9 g/week at age 35; 2.9 vs 4.1 g/week at age 50), and were younger at the time of first alcohol use (20 vs 22 years) compared with cases who were also former drinkers ($N = 135$), but reported cessation of drinking more than one year prior to diagnosis (data not shown). However, exclusion of this group did not affect estimates, for either past or recent intake, by more than 10% (data not shown).

DISCUSSION

We observed evidence of an increased risk for breast cancer among postmenopausal Hispanic women and non-Hispanic White women at the highest alcohol intake level, and found

that menopausal status may be an effect-modifier in Hispanic women. A protective effect for low to moderate alcohol intake (<8 drinks/week) in non-Hispanic White women was consistently observed; but, results were only statistically significant for recent intake. Age at first use of alcohol and duration of drinking were not associated with risk for breast cancer, but these have not proved to be consistent risk factors in previous studies.^{16,17,39,46,47} We did not find an association between alcohol consumption and joint hormone-receptor status, although a previous study found alcohol intake to be associated with specific hormone-receptor breast cancer.²⁶

Generally, studies have demonstrated a consistent, but modest, increased risk with high alcohol intake, with results differing as to whether the effect is stronger for recent^{14,36,40} or lifetime intake.^{16,18} Results of a recent analysis of the Framingham cohort did not provide any evidence for an increased risk of breast cancer associated with long-term, light to moderate alcohol consumption,⁴⁸ and only a modest effect was associated with moderate consumption among women aged 25–42 years in the 'Nurses Health Study'.⁴⁹ Evidence supporting a dose-response relationship has been present in the majority of epidemiologic studies,¹⁹ and further supported in several meta-analyses.^{15,37,40} Only one study estimated risk at 11% for one daily drink, 24% for 2 daily drinks, and 38% for 3 daily drinks.¹⁵ Longnecker et al¹⁶ reported a monotonic dose-response relationship for all women, with strongest estimates for postmenopausal women. Swanson et al¹⁴ reported a threshold for increased risk at high levels of intake (≥ 14 drinks/week) for premenopausal women. We observed a weak association for a risk threshold, but at a lower level of intake than was previously reported,¹⁴ and only among postmenopausal women. Findings for Hispanic postmenopausal women were similar to those for non-Hispanic White women, with about a 2-fold increased risk, but at few-

Table 5. Multivariate-adjusted (OR)* and 95% confidence intervals (CI)† for breast cancer risk associated with ever vs never alcohol consumption and alcohol usage patterns, stratified by ethnicity, New Mexico Women's Health Study, 1992–1994

Alcohol Usage Patterns	Hispanic				Non-Hispanic White			
	Cases (N=332) N	Controls (N=388) N	OR	95% CI	Cases (N=380) N	Controls (N=456) N	OR	95% CI
History of alcohol consumption								
Never	83	82	1.00		51	46	1.00	
Ever	249	306	0.96	0.61–1.50	329	410	0.72	0.43–1.19
Status of alcohol consumption at interview								
Non-drinker	83	82	1.00		51	46	1.00	
Current drinker	166	230	0.84	0.52–1.36	233	328	0.60	0.36–1.01
Former drinker	83	76	1.21	0.71–2.05	96	82	1.09	0.61–1.95
Years since last alcohol consumption‡								
Non-drinker	83	82	1.00		51	46	1.00	
Stopped within reference year	21	2	12.13	2.51–58.6	23	3	8.04	2.03–31.7
1	7	4	2.77	0.57–13.5	9	3	2.10	0.48–9.23
2–4	11	7	1.86	0.58–5.95	13	10	1.57	0.54–4.55
5–14	18	14	1.65	0.67–4.05	24	32	0.78	0.36–1.66
≥15	25	48	0.53	0.27–1.03	27	34	0.64	0.30–1.34
Current drinker	166	230	0.90	0.56–1.46	233	328	0.62	0.37–1.05
Age (years) at first alcohol use								
Non-drinker	83	82	1.00		51	46	1.00	
≤16	40	63	0.71	0.37–1.36	72	120	0.61	0.33–1.12
17–18	47	70	0.67	0.36–1.23	94	117	0.67	0.39–1.20
19–21	72	88	0.95	0.55–1.65	95	102	0.80	0.45–1.42
≥22	90	85	1.21	0.72–2.03	68	71	0.73	0.40–1.32
Duration (years) of drinking‡,§								
Non-drinker	83	82	1.00		52	46	1.00	
<10	20	32	0.82	0.39–1.70	16	21	0.69	0.29–1.64
10–39	201	224	1.06	0.66–1.73	230	286	0.80	0.47–1.36
≥40	27	49	0.76	0.37–1.57	82	103	0.60	0.33–1.09

* Odds ratios estimated from conditional logistic regression models matched for age-group, health planning district, and adjusted for age, education, age at menarche, menopausal status, age at first full-term birth, number of full-term births, cumulative months of lactation, cumulative years of oral contraceptive use, history of fibrocystic disease, breast cancer in mother, sister, daughter, cigarette smoking, body mass index, physical activity, energy intake, and energy-adjusted total fat intake.

† 95% confidence interval.

‡ Age when alcohol consumption stopped, missing for one case and one control.

§ Does not reflect actual duration of drinking; based on reported age at cessation or reference age minus first age of alcohol use.

er drinks per week (3+ vs 8+); however, these estimates were unstable and not statistically significant.

The present study was unable to evaluate heavy alcohol consumption, especially among Hispanics, because there were so few drinkers with a high intake. The relatively low level of alcohol consumption observed in this study has been reported previously in another New Mexico study.⁵⁰ A low level of alcohol consumption was also seen in our dietary pilot study data, and studies in other regions of the United States have reported a lower average alcohol intake for Hispanics compared with non-Hispanics (3 vs 5 drinks/week).^{51,52} Reproducibility results, based on the pilot

data, were comparable to those reported in previous studies.^{36,53}

It is impossible to determine at this time whether the protective effect observed in non-Hispanic White women for low to moderate alcohol intake is indirect and due to confounding with other unadjusted health-related behaviors, or to an information bias. There was no single strong confounder of alcohol intake, suggesting that both the protective effect, as well as the possible threshold for increased risk observed in non-Hispanic White women, is not due to the confounders included in the analysis. There was evidence that a small group of women stopped drinking at the time of diagnosis, possibly due to

information regarding an alcohol-breast cancer association. This may have led to recall bias by these women if they tended to under-report past or recent intake, as suggested by a comparison of results with other former and current drinkers. However, removal of their data from analyses did not appear to significantly alter estimates.

It should be noted that recent alcohol intake was collected soon after the time of diagnosis for many women, but women were asked to report their usual dietary intake from a month prior to any significant impact. Of those women who were asked whether dietary intake was usual or altered at interview, ($N = 1,363$), 19% of cases reported that their

intake was different compared to a report of 6% by controls. The majority (60%) of these cases reported that their dietary intake was not usual due to breast cancer treatment. Hospitalization or a major illness lasting more than 4 days, and traveling or dieting was reported by 9% and 24%, respectively. Seven percent did not respond. The majority (85%) of controls reported their diet as unusual due to other factors.

If the report of alcohol intake among cases was not reflective of usual pre-diagnosis consumption, it is possible that some women were misclassified, and this may have affected our results. Women who reported that they had stopped drinking alcohol within the reference year showed an increased breast cancer risk (Table 5). This supports the assumption that some women stopped drinking after learning their diagnosis and, if intake was based on the period of time after cessation, the result would be to diminish the estimate. However, of those cases who reported dietary intake as unusual, women who reported recent alcohol consumption (40%) were characterized by a mean of 80 g/week of alcohol. This did not differ statistically ($F = 0.06$, $P = 0.81$) from the 75 g/week reported by recent alcohol consumers (45%) who reported usual dietary intake (data not shown). It seems less likely that the women would increase their alcohol intake following diagnosis; however, if this was the case, the effect would be to reflect an increase for high levels and a reduced effect for lower levels. The percentage of cases who reported recent alcohol intake vs no recent intake also did not vary significantly when stratified by months from diagnosis to interview. Cases who were interviewed prior to 6 months were not found to have a significantly different mean alcohol intake per week (though it was higher) from those women interviewed more than 6 months following diagnosis (31 g/week vs 24 g/week, $F = 2.2$, $P = .14$, $N = 302$, data not shown). Analyses stratified by eth-

Whatever the explanation may be, whether real or spurious, the present study is not the only one to find a potential protective effect for light to moderate alcohol consumption.

nicity did not alter these results (data not shown).

A mechanism by which a low alcohol intake might decrease risk is unknown at this time. The presence of this finding for both premenopausal and postmenopausal non-Hispanic White women seems to argue against an effect mediated by a change in hormone level. Whatever the explanation may be, whether real or spurious, the present study is not the only one to find a potential protective effect for light to moderate alcohol consumption. In their study of alcohol consumption among postmenopausal women, the Longnecker et al¹⁸ study provided evidence for a protective effect associated with low level alcohol intake in the recent past (OR = 0.70, 95% CI 0.51–0.94 for 6–11 g/day). Only a few other studies have suggested a protective effect associated with a low alcohol intake, and this finding has differed by menopausal status.^{46,54}

The results of the present study do not indicate that alcohol intake plays an important part in explaining the increasing incidence of breast cancer in New Mexican Hispanic women. We found no consistent relationship in the low to moderate range observed, and high alcohol intake was rare.

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