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Gender and Racial/Ethnic Differences in the Association Between Alcohol Drinking Patterns and Body Mass Index—the National Health and Nutrition Examination Survey, 1999–2010

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Abstract

Background Racial/ethnic variations in both alcohol consumption and obesity prevalence are well established. However, previous research indicates that drinking patterns influence the relation of alcohol intake to body mass index (BMI), and information on racial/ethnic differences in the relation of drinking pattern to BMI is lacking.

Methods Multi-year cross-sectional data extracted from the 1999–2010 National Health and Nutrition Examination Survey for adults ≥ 20 years ($N = 25{,}816$) were used. Effects of drinking patterns were analyzed using a linear dose–response model that considered the joint effects of frequency (number of days in the past year that at least one drink was consumed) and dosage (the number of drinks consumed in excess of the first drink on days when more than one drink was consumed).

Results For all racial/ethnic groups except Mexican Americans, current drinkers had a lower mean BMI than non-drinkers. Mean BMI differences were − 0.721 kg/m^2 for non-Hispanic white (white) men and − 1.292 kg/m^2 for white women. Among drinkers, drinking frequency was negatively associated with BMI for all racial/ethnic groups; however, this effect was significantly smaller for Mexican American men and other Hispanic men than white men. Dosage was positively associated with BMI among all racial/ethnic groups except Mexican American women and other Hispanic women; this effect was significantly stronger among black women than white women.

Conclusion Gender and racial/ethnic differences in the relation of drinking patterns to BMI should be taken into consideration when investigating factors that influence the effect of alcohol consumption on BMI.

Keywords Alcohol drinking patterns · Body mass index · Racial/ethnic differences · Gender

Introduction

The prevalence of overweight, defined as a body mass index (BMI) between 25.0 and 29.9 kg/m^2, and obesity, defined as a BMI ≥ 30 kg/m^2, has been steadily increasing over the last three decades among US adults [1, 2]. In 2011–2012, 69.0% of US adults were estimated to be overweight or obese [1]. Overweight and obesity are associated with an array of health complications including type 2 diabetes, cardiovascular disease, depression, and many forms of cancer [3].

In 2011–2012, overweight (obesity included) was most prevalent among Hispanics (77.1%), followed by non-Hispanic blacks (blacks 76.3%) and non-Hispanic whites (whites 68.5%) [1]. With such reported prevalence, factors associated with major racial/ethnic differences in BMI deserve careful consideration. Studies showed that BMI is affected by factors such as physical activity [4], nutrition [5], income [6], and alcohol use [7]. However, the extent to which these factors impact BMI is variable between individuals; efforts are therefore needed to better understand the complexities and differences at the individual level to better tailor future intervention strategies aimed at reducing BMI.

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There is little research on racial/ethnic differences in the relationship between alcohol consumption and BMI, although much is known in each domain: e.g., whites have higher drinking rates [8] and Mexican Americans and blacks have higher overweight and obesity rates [9] when compared to their racial/ethnic counterparts. One of the factors that has hindered investigation of these differences is that alcohol use is a complex behavior, involving many possible patterns of consumption that differ with respect to drinking frequency and quantity, and contributing to contradictions in the literature on the relationship between alcohol use and obesity [10].

Findings from previous analyses of alcohol intake and BMI based on measures such as drinking frequency, quantity, and volume obscure the effects of drinking pattern [7, 11–13], which significantly limits their interpretation. It is reasonable to assume that both physiological and behavioral effects associated with having one drink a day 7 days a week will differ from those associated with having seven drinks on 1 day a week, even though the total alcohol intake per week represented by these two very different drinking patterns is the same. This limitation can be overcome by using data on drinking frequency and quantity to categorize respondents according to their drinking patterns [14, 15]. However, categorical approaches in analyzing drinking patterns also have limitations—the arbitrary nature of definitions used to classify patterns, the large sample sizes required to provide stable estimates for numerous drinking patterns defined, and the lack of statistical methods to evaluate the significance of differences in alcohol effects across population groups. If the definition of 30 drinking patterns is required to investigate the relations of drinking patterns to BMI according to gender [14], extending this analysis to include racial/ethnic differences would require definition of 120 drinking patterns and would not support significance tests of these differences.

We performed a descriptive and exploratory study, employing a recently developed linear alcohol dose–response model [16, 17] to investigate the significance of racial/ethnic differences in the relation of drinking patterns and BMI according to gender. The alcohol dose–response model was derived from a more general model of additive risks related to drinking (i.e., each drink adds to the risks for a problem outcome). The model is based on measures of drinking frequency and quantity routinely assessed in surveys of alcohol intake. It assesses the joint effects of frequency and quantity in terms of frequency and dosage, where frequency represents the number of days when at least one drink of alcohol is consumed and dosage represents the number of drinks in excess of the first drink on days when alcohol is consumed and the effect of having additional drinks above and beyond one on drinking days. The model yields regression coefficients for frequency and dosage that can be readily interpreted in terms of drinking patterns defined by frequency and quantity. In addition, it supports the use of interaction terms to test the significance of differences between population groups in the effects of drinking patterns on health. Based on previous studies [14, 18, 19], the objective of this study was to examine whether gender and race/ethnicity moderate the relationship between drinking patterns and BMI. We hypothesized that drinking frequency would be negatively associated with BMI while dosage would be positively associated with BMI.

Methods

Survey and Study Sample

Multi-year cross-sectional data from the 1999–2010 National Health and Nutrition Examination Survey (NHANES) were used. Beginning in 1999, NHANES became a series of nationally representative sample surveys of the US noninstitutionalized civilian population with data released in 2-year cycles; it combines interviews and physical examinations. Response rates for the interview and for the physical examination were 82% and 76% in 1999–2000, 84% and 80% in 2001–2002, 79% and 76% in 2003–2004, 80% and 77% in 2005–2006, 78% and 75% in 2007–2008, and 79% and 77% in 2009–2010, respectively [20].

The 1999–2010 NHANES data were derived from three data files (demographics, physical examination, and questionnaire). Then, data were combined using respondent sequence numbers. Of 32,645 respondents aged ≥ 20 years who participated, 1475 pregnant women, 998 participants who identified themselves as other race, and 4356 participants whose data on alcohol consumption were missing were excluded from the analysis, reducing the sample size to 25,816 for the present study. The sample size of each survey was 3800 in 1999–2000, 4233 in 2001–2002, 3953 in 2003–2004, 3894 in 2005–2006, 4987 in 2007–2008, and 4949 in 2009–2010, respectively.

Measures

All demographic variables were categorized using the 1999–2010 NHANES analytic guidelines [21]. Race/ethnicity was self-reported and included whites, blacks, Mexican Americans, and other Hispanics. During the physical examination, body weight and height of participants were measured using a digital weight scale and a stadiometer. BMI was computed as kilogram per square meter using measured height in meters and weight information in kilograms and categorized into underweight (BMI < 18.5), normal (18.5 ≤ BMI < 25.0), overweight (25.0 ≤ BMI < 30.0), and obese (BMI ≥ 30.0).

Before participants were interviewed about alcohol use, interviewers defined a drink as a 12 oz beer, a 5 oz glass of wine, or one and half ounces of liquor. Current drinkers were defined as respondents who had consumed at least 12 drinks in their entire life and who had consumed alcohol at least 1 day in the past 12 months; respondents who had not were classified as non-drinkers [14, 15]. Using the definition, 16,875 and 8941 respondents were identified as current drinkers and non-drinkers, respectively. Drinking frequency was assessed by asking: "In the past 12 months, how often did you drink any type of alcoholic beverage?" Drinking quantity was assessed by asking "In the past 12 months, on those days that you drank alcoholic beverages, on the average how many drinks did you have?" Four levels of drinking quantity were adapted from a previous study [11]: abstainers, 1–2, 3–4, and ≥ 5 drinks/drinking day. Frequency and quantity were measured using open-ended questions. Drinking volume in the past year was calculated by multiplying drinking quantity (number of drinks consumed, on average, on drinking days) by drinking frequency. Drinking dosage was computed by subtracting drinking frequency from volume, which provides a measure of the extent to which respondents had more than one drink on days when they drank [17].

Covariates included age, poverty income ratio, education, marital status, recreational physical activity status, current smoking status, survey year, and a set of dummy variables to code white (reference), black, Mexican American, and other Hispanics. Covariates were selected a priori based on their known associations with alcohol use and BMI. Diet behavior and nutrition were not included as covariates because survey contents did not remain constant across surveys [22]. For example, in 1999–2000, diet behavior was assessed by asking: "On average, how many times per week do you eat meals that were prepared in a restaurant?" In 2009–2010, it was measured by asking: "During the past 7 days how many meals did you get that were prepared away from home in places such as restaurants, fast food places, food stands, grocery stores, or from vending machines?"

Recreational physical activity was assessed by: (1) "Do you do any vigorous-intensity sports, fitness, or recreational activities that cause large increases in breathing or heart rate like running or basketball for at least 10 minutes continuously?" and (2) "Do you do any moderate-intensity sports, fitness, or recreational activities that cause a small increase in breathing or heart rate such as brisk walking, bicycling, swimming, or golf for at least 10 minutes continuously?" Four levels of physical activity were adapted from prior research [15]: most active ("yes" to both questions), active ("yes" to the first question and "no" to the second question), somewhat active ("no" to the first question and "yes" to the second question), and not active ("no" to both questions).

Current smoking was measured by: (1) "Have you smoked 100 cigarettes in your entire life?" and (2) "Do you now smoke cigarettes every day, some days, or not at all?" Based on the Centers for Disease Control and Prevention glossary [23], participants were classified into three groups: current smokers (ever smoked 100 cigarettes in entire life and reported current cigarette smoking every day or some days), former smokers (ever smoked 100 cigarettes in entire life and reported no longer smoking), and never smokers (never smoked 100 cigarettes in entire life).

Statistical Analysis

All analyses used sample weights for each 2-year survey cycle to take into account the features of the survey including survey non-response, over-sampling, post-stratification, and sampling error [24]. All variables had 10% or less of missing data and were analyzed without further evaluation or adjustment based on NHANES analytic guidelines [25]. Two weight variables (WTMEC2YR and WTMEC4YR) that the NHANES demographic file contained were used to create a 12-year weight variable for 12 years of data from 1999 to 2010. Constructing weights for the 1999–2010 survey cycles were based on formulas provided by the National Center for Health Statistics [26].

The linear alcohol dose–response model employed in this study was derived from a more general model of additive risks related to drinking (i.e., each drink adds to the risks for a problem outcome). The influence of alcohol use on BMI is assumed to be related to the additive effects of exposure to different "doses" of alcohol, assessed in terms of drinks per drinking day, as follows:

$$C = \alpha E_0 + (\alpha + \beta_1)E_1 + (\alpha + \beta_2)E_2$$
$$+ (\alpha + \beta_l)E_l \ \ (\alpha + \beta_n)E_n$$

where C represents BMI and E_i represent exposures to drinking at each dose (e.g., the number of days drinking at dosage levels $i = 1,2,3,\ldots,\ n$ drinks). In this equation, background risks are given by α (i.e., the background level of BMI on drinking and non-drinking days). The contributions of drinking to BMI are represented by the parameters β_i. Different assumptions about the relationships of these parameters to dosage levels enable different assessments of dose–response relationships. For example, if it is assumed that all β_i are equal to one another, but different from zero, then β represents the contribution of drinking regardless of drinking level to BMI (a constant effect). If it is assumed that BMI is linearly related to greater drinking quantities then $\beta_i = \beta + \delta(i-1)$, where δ is the slope of the linear dose–response function, then β represents the change associated with consuming one drink, and δ represents linear changes associated with having increasing numbers of drinks (a "linear dose–response" model). As demonstrated elsewhere [17], this model can be reduced algebraically to a form which can be estimated using measures of alcohol intake available in NHANES, drinking frequency, and total volume (drinking frequency × quantity):

$$C = \alpha T + \beta F + \delta(V - F)$$

where T represents the time frame of measurement (i.e., $T = E_0 + E_1 + E_2 + E_3 \ldots + E_n$), F is the frequency of alcohol use [i.e., number of drinking days, ($F = E_1 + E_2 + E_3 \ldots + E_n$)], and V is the total volume of alcohol consumed ($V = E_1 + 2E_2 + 3E_3 \ldots + nE_n$). $V - F$ represents the number of drinks consumed beyond the first drink over time, T, and δ is the estimated increase in dose–response over greater drinking quantities. Here, α represents background changes in BMI that are not related to drinking, β indicates the linear contribution of drinking per se (days when at least one drink was consumed) to changes in BMI assessed from the model, and δ estimates the linear contribution of drinking levels to BMI, the extent to which BMI changes with increasing drinks per drinking day (drinks exceeding the first drink on days when alcohol was consumed).

Chi-square tests and one-way analyses of variance were conducted to examine racial/ethnic differences in demographics and health-related factors (e.g., current smoking status and alcohol use). Multiple linear regression models were developed to examine BMI differences related to race/ethnicity and to determine the influence of drinking status and alcohol drinking patterns (i.e., frequency and dosage) on BMI among men and women, controlling for the covariates (age, poverty income ratio, education, marital status, recreational physical activity status, current smoking status, and survey year). Models showed no departure from normality or multicollinearity problems. In addition, racial/ethnic differences in the relationship between alcohol use and BMI were investigated by including interaction terms (e.g., frequency × racial/ethnic group) in the regression models. Regression analyses incorporated strata, primary sampling units, sampling weights, and Taylor series linearization based on NHANES analytic guidelines [21]. All analyses were conducted with STATA version 13 (STATA Press, College Station, TX).

Results

Demographic and Health Behavior Differences Among Men and Women by Race/Ethnicity

Compared to white men, blacks, Mexican Americans and other Hispanics were younger, were more likely to live below the federal poverty level, and were less likely to have a high school degree or above ($p < 0.0001$). Blacks and other Hispanics were less likely to be married or living with a partner than whites and Mexican Americans ($p < 0.0001$) (Table 1). Similar racial/ethnic demographic variations were found among women ($p < 0.0001$) (Table 2).

Among men, blacks, compared to other racial/ethnic groups, were more likely to be obese and to currently abstain from alcohol consumption ($p < 0.0001$). White men drank more frequently than other racial/ethnic counterparts ($p < 0.0001$). Mexican Americans, followed by blacks, had a greater dosage of alcohol compared to whites and other Hispanics ($p < 0.0001$) (Table 1).

Among women, Mexican Americans and other Hispanics were less likely to report being current smokers ($p < 0.0001$). Compared to the other racial/ethnic groups, whites reported the most recreational physical activity, had a higher prevalence of normal body weight, and were the least likely to abstain from current alcohol consumption ($p < 0.0001$). White women drank with the most frequency ($p < 0.0001$). Blacks, followed by whites, had higher mean dosages of alcohol than Mexican American women and other Hispanic women ($p < 0.0001$) (Table 2).

Gender-Specific Multiple Linear Regression of BMI on Alcohol Use and Race/Ethnicity

The influence of race/ethnicity and alcohol use on BMI is summarized for men and women in Table 3, controlling for demographics and health characteristics. Compared to white current abstainers, white current drinkers had significantly lower BMIs among men ($b = -0.721$, 95% CI -1.102, -0.339) and among women ($b = -1.292$, 95% CI -1.689, -0.894). This finding differed significantly according to race/ethnicity only for Mexican American men and women whose mean BMIs related to current drinking were higher than those of white men and women.

Gender-Specific Multiple Linear Regression of BMI on Alcohol Use and Race/Ethnicity Among Current Drinkers

The linear alcohol dose–response model revealed a significant negative effect of drinking frequency on BMI among white men ($b = -0.008$, 95% CI -0.009, -0.007) and women ($b = -0.011$, 95% CI -0.014, -0.008) (Table 4). In contrast, the effect of dosage among whites was positive for men ($b = 0.002$, 95% CI 0.001, 0.003) and for women ($b = 0.002$, 95% CI 0.000, 0.004). Significant differences from these findings were observed in several racial/ethnic groups. Compared to white men, the effect of drinking frequency was significantly less positive among Mexican American men and other Hispanic men. Compared to white women, the positive effect of dosage on BMI was significantly higher among blacks, but was negative among Mexican American women and other Hispanic women.

The findings from the linear dose–response model, which controlled for age, poverty income ratio, education, marital status, recreational physical activity status, current smoking status, and survey year, were used to plot BMI according to drinking quantity, holding drinking frequency constant at 100 drinking days per year for men (Fig. 1), and 57 drinking days per year for women (Fig. 2). As drinking quantity increases, the contribution to BMI associated with alcohol consumption increased most rapidly among Mexican American men and

Table 1 Characteristics of men by race/ethnicity ($n = 13{,}065$), NHANES 1999–2010

Demographics and health behaviors	White ($n = 6879$) %[a]	Black ($n = 2628$) %[a]	Mexican American ($n = 2737$) %[a]	Other Hispanic ($n = 821$) %[a]	p value
Age					< 0.0001
20–39	32.0	37.5	50.9	45.1	
40–59	37.1	34.8	30.9	34.7	
≥ 60	31.0	27.7	18.2	20.2	
Above poverty	91.1	82.8	70.8	76.7	< 0.0001
> High school	85.3	69.1	43.9	58.3	< 0.0001
Marital status					< 0.0001
Married/living with partner	71.0	55.0	71.4	63.4	
Widowed/divorced/separated	13.9	19.3	10.7	12.5	
Never married	15.1	25.6	17.9	24.0	
Current smokers	24.5	31.5	25.9	22.9	< 0.0001
Physical activity					< 0.0001
Most active	23.1	22.1	16.9	19.6	
Active	9.7	12.4	13.2	12.9	
Somewhat active	31.2	21.3	17.0	18.1	
Not active	35.9	44.2	52.9	49.3	
BMI[b]					< 0.0001
Underweight	1.3	2.3	0.3	0.3	
Normal weight	26.4	28.0	23.6	24.3	
Overweight	40.1	31.9	46.7	45.2	
Obese	32.3	37.8	29.4	30.2	
Drinking quantity					< 0.0001
Abstainers	24.6	34.0	21.4	25.5	
1–2 drinks	42.8	34.9	24.1	29.7	
3–4 drinks	18.5	19.3	20.4	19.5	
≥ 5 drinks	14.2	11.9	34.1	25.3	
Drinking patterns[c]	White ($n = 4937$) Mean (SD)[a]	Black ($n = 1776$) Mean (SD)[a]	Mexican American ($n = 2068$) Mean (SD)[a]	Other Hispanic ($n = 601$) Mean (SD)[a]	p value
Drinking frequency[d]	112.0 (116.2)	94.7 (105.0)	74.4 (98.7)	78.1 (99.1)	< 0.0001
Drinking dosage[e]	241.9 (454.6)	262.5 (566.0)	304.2 (613.4)	230.9 (526.7)	< 0.0001

The percentages may not add to 100 because of rounding errors. Tukey HSD indicates that all mean differences in frequency and dosage between racial/ethnic groups are significant at the 0.05 level. Only one category for dichotomous variables is presented to eliminate redundancy in the table

[a] Weighted values

[b] Underweight (BMI < 18.5), normal (18.5 ≤ BMI < 25.0), overweight (25.0 ≤ BMI < 30.0), and obese (BMI ≥ 30.0)

[c] Analysis is limited to current drinkers

[d] Drinking days per year

[e] Total drinks per year minus drinking frequency per year

black men and women. Alcohol-related contributions to BMI increased somewhat less rapidly among white men and women, stayed the same among other Hispanic men, and decreased among Mexican American women and other Hispanic women.

Discussion

Past studies [7, 11, 12, 14, 15] have reported the relation of various measures of alcohol consumption (e.g., frequency, quantity, and volume) to BMI, but this is the first time to our knowledge that the association between drinking patterns (frequency and dosage) and BMI has been precisely estimated using a mathematically derived alcohol dose–response model. The model enabled quantification of the competing influences of drinking frequency and dosage on BMI, and facilitated investigation of the moderating influences of gender and race/ethnicity on the relation between drinking patterns and BMI. Overall, we observed that current drinkers had lower mean BMIs compared to non-drinkers for all racial/ethnic

Table 2 Characteristics of women by race/ethnicity ($n = 12{,}751$), NHANES 1999–2010

Demographics and health behaviors	White ($n = 6576$) %[a]	Black ($n = 2633$) %[a]	Mexican American ($n = 2600$) %[a]	Other Hispanic ($n = 942$) %[a]	p value
Age					< 0.0001
20–39	29.2	34.4	44.1	42.8	
40–59	37.2	38.0	33.0	33.9	
≥ 60	33.6	27.6	22.9	23.3	
Above poverty	89.0	75.8	67.6	72.8	< 0.0001
> High school	85.5	70.9	50.1	62.6	< 0.0001
Marital status					< 0.0001
Married/living with partner	62.0	35.8	60.7	50.6	
Widowed/divorced/separated	26.7	36.5	24.0	27.6	
Never married	11.4	27.6	15.2	21.8	
Current smokers	21.7	20.0	12.3	17.9	< 0.0001
Physical activity					< 0.0001
Most active	19.5	13.9	12.2	12.0	
Active	6.8	6.4	7.0	9.3	
Somewhat active	34.7	26.3	23.9	23.2	
Not active	39.0	53.4	56.9	55.6	
BMI[b]					< 0.0001
Underweight	2.6	1.4	0.8	0.9	
Normal weight	36.8	19.1	23.9	29.9	
Overweight	28.5	25.5	33.5	35.5	
Obese	32.0	54.0	41.8	33.7	
Drinking quantity					< 0.0001
Abstainers	32.6	46.7	43.7	38.9	
1–2 drinks	51.2	40.4	36.2	41.3	
3–4 drinks	11.6	9.7	12.7	12.9	
≥ 5 drinks	4.7	3.1	7.4	6.8	
Drinking patterns[c]	White ($n = 4236$) Mean (SD)[a]	Black ($n = 1372$) Mean (SD)[a]	Mexican American ($n = 1349$) Mean (SD)[a]	Other Hispanic ($n = 536$) Mean (SD)[a]	p value
Drinking frequency[d]	70.2 (97.3)	49.3 (80.5)	28.0 (52.1)	31.8 (55.1)	< 0.0001
Drinking dosage[e]	80.0 (203.7)	92.2 (318.8)	56.3 (191.3)	58.9 (174.5)	< 0.0001

The percentages may not add to 100 because of rounding errors. Tukey HSD indicates that all mean differences in frequency and dosage between racial/ethnic groups are significant at the 0.05 level. Only one category for dichotomous variables is presented to eliminate redundancy in the table

[a] Weighted values

[b] Underweight (BMI < 18.5), normal (18.5 ≤ BMI < 25.0), overweight (25.0 ≤ BMI < 30.0), and obese (BMI ≥ 30.0)

[c] Analysis is limited to current drinkers

[d] Drinking days per year

[e] Total drinks per year minus drinking frequency per year

groups except Mexican Americans. Drinking frequency was negatively associated with BMI for all racial/ethnic groups, but the effect was significantly smaller for Mexican American men and other Hispanic men than white men. Dosage was positively associated with BMI among all racial/ethnic groups except Mexican American women and other Hispanic women, with a significantly stronger effect among black women than white women. Mexican American women are more likely than white women to abstain, but if they do drink, they were more likely to report having five or more drinks a day compared to white women. Accordingly, a greater proportion of Mexican American female drinkers may experience negative influences of heavier drinking on food intake than white women, even though overall BMI tends to be higher among Mexican American women who are current drinkers compared to non-drinkers. These findings confirm that high-frequency/low-quantity drinking patterns are associated with lower mean BMIs than low-frequency/high-quantity drinking patterns among men and women [14], and extend them to racial/ethnic subpopulations.

Table 3 Gender-specific multiple linear regression of BMI on alcohol use and race/ethnicity among all participants ($N = 25{,}816$), NHANES 1999–2010

	Men ($n = 13{,}065$)		Women ($n = 12{,}751$)	
	b	95% CI	*b*	95% CI
Demographics and health characteristics				
Age	− 0.012	− 0.020, − 0.003	− 0.005	− 0.017, 0.007
Poverty income ratio	0.256	0.152, 0.360	− 0.103	− 0.208, 0.002
Education	− 0.038	− 0.177, 0.101	− 0.220	− 0.406, − 0.034
Marital status				
Married/living with partner (ref)				
Widowed/divorced/separated	− 0.336	− 0.685, 0.012	− 0.149	− 0.548, 0.249
Never married	− 1.268	− 1.673, − 0.863	− 0.246	− 0.721, 0.229
Physical activity				
Most active (ref)				
Active	− 0.092	− 0.555, 0.371	0.422	− 0.099, 0.944
Somewhat active	1.188	0.821, 1.555	1.657	1.247, 2.066
Not active	1.479	1.097, 1.862	2.150	1.734, 2.566
Smoking status				
Non-smoker (ref)				
Former smoker	− 0.076	− 0.335, 0.184	0.540	0.186, 0.894
Current smoker	− 1.800	− 2.160, − 1.440	− 0.959	− 1.362, − 0.557
Survey year	0.190	0.099, 0.281	0.100	0.016, 0.183
Race/ethnicity				
White (ref)				
Black	0.382	− 0.203, 0.967	2.556	1.949, 3.163
Mexican American	− 0.599	− 1.222, 0.024	− 0.178	− 0.807, 0.451
Other Hispanic	− 0.363	− 1.205, 0.480	− 0.730	− 1.476, 0.016
Alcohol Use, by race/ethnicity				
Drinker[a]	− 0.721	− 1.102, − 0.339	− 1.292	− 1.689, − 0.894
Drinker × Black	0.334	− 0.337, 1.006	0.596	− 0.192, 1.384
Drinker × Mexican American	0.909	0.248, 1.570	1.677	0.749, 2.606
Drinker × other Hispanic	0.545	− 0.469, 1.559	1.198	0.123, 2.273

All models were weighted and adjusted for age, poverty income ratio, education, marital status, recreational physical activity status, current smoking status, survey year, and three dummy variables (black, Mexican American, and other Hispanic). Only one category for dichotomous variables is presented to eliminate redundancy in the table. *b* unstandardized coefficients, *ref* reference group

[a] Data pertain to the effect of drinking on BMI among whites, who serve as the reference for the effect of drinking on BMI among other racial/ethnic groups

It has been suggested that frequently consuming small amounts of alcohol might contribute to weight gain by adding calories to the diet or stimulating the appetite so that more food would be consumed [27]. However, the consistent negative association of drinking frequency with BMI seen in men and women of all racial/ethnic groups does not support this theory. Analyses of the relation between alcohol drinking patterns and the Healthy Eating Index, a measure of diet quality, revealed that high-frequency/low-quantity drinking patterns were associated with better diets than low-frequency/high-quantity drinking patterns [15]. Thus, it may be that people who drink small amounts frequently, sometimes characterized as a “moderate” drinking pattern, are also moderate in their eating habits. Alternatively, it may be that drinkers who gained weight are now abstaining in order to reduce their body weight.

For a given absolute alcohol consumption, BMI is inversely related to frequency (i.e., average BMI decreases as the frequency of drinking increases). Individuals who frequently drink small amounts of alcohol may have lower average BMIs because they have learned to compensate for the extra alcohol calories, and their moderate drinking habits may carryover to moderation in their intake of other nutrients that provide calories, such as fats and carbohydrates. In contrast, less frequent consumption of larger amounts of alcohol may be associated with celebratory or recreational eating of more than usual and/or high-fat foods, and increases in average BMI. In addition to the influence of

Table 4 Gender-specific multiple linear regression of BMI on alcohol use and race/ethnicity among current drinkers ($n = 16,875$), NHANES 1999–2010

	Men ($n = 9382$)		Women ($n = 7493$)	
	b	95% CI	*b*	95% CI
Demographics and health characteristics				
Age	0.006	− 0.009, 0.022	0.016	0.006, 0.026
Poverty income ratio	0.218	0.082, 0.353	− 0.102	− 0.203, − 0.000
Education	− 0.020	− 0.300, 0.260	− 0.293	− 0.523, − 0.063
Marital status				
Married/living with partner (ref)				
Widowed/divorced/separated	− 0.128	− 0.527, 0.272	0.338	− 0.253, 0.929
Never married	− 1.047	− 1.708, − 0.385	− 0.384	− 0.648, − 0.120
Physical activity				
Most active (ref)				
Active	− 0.108	− 0.383, 0.168	0.525	− 0.580, 1.630
Somewhat active	1.046	0.671, 1.421	1.509	1.050, 1.967
Not active	1.394	0.700, 2.087	2.188	1.438, 2.937
Smoking status				
Non-smoker (ref)				
Former smoker	0.365	0.065, 0.666	0.881	0.464, 1.298
Current smoker	− 1.356	− 2.160, − 0.551	− 0.499	− 1.006, 0.008
Survey year	0.273	0.206, 0.340	0.129	0.086, 0.172
Race/ethnicity				
White (ref)				
Black	0.908	− 0.372, 2.188	2.616	2.282, 2.949
Mexican American	− 0.458	− 0.965, 0.048	0.730	0.145, 1.315
Other Hispanic	− 0.273	− 0.707, 0.160	− 0.034	− 1.586, 1.517
Alcohol Use, by race/ethnicity				
Frequency[a, b]	− 0.008	− 0.009, − 0.007	− 0.011	− 0.014, − 0.008
Dosage[b, c]	0.002	0.001, 0.003	0.002	0.000, 0.004
Frequency × Black	− 0.001	− 0.015, 0.013	− 0.004	− 0.010, 0.001
Frequency × Mexican American	0.004	0.000, 0.007	0.002	− 0.001, 0.006
Frequency × other Hispanic	0.004	0.000, 0.008	0.001	− 0.009, 0.011
Dosage × Black	0.001	− 0.004, 0.004	0.004	0.000, 0.007
Dosage × Mexican American	0.001	− 0.001, 0.002	− 0.003	− 0.010, 0.005
Dosage × other Hispanic	− 0.002	− 0.005, 0.001	− 0.004	− 0.009, 0.000

All models were weighted and adjusted for age, poverty income ratio, education, marital status, recreational physical activity status, current smoking status, survey year, and three dummy variables (black, Mexican American, and other Hispanic). *b* unstandardized coefficients, *ref* reference group

[a] Drinking days per year

[b] Data pertain to the effects of frequency and dosage on BMI among whites, who serve as the reference for effects of frequency and dosage on BMI among other racial/ethnic groups

[c] Total drinks per year minus drinking frequency per year

alcohol calories themselves, other mechanisms associated with heavier drinking may operate to increase BMI. Heavy drinking may have a disinhibitory influence on food consumption, such that people eat more than they plan [28]. When alcohol is consumed with fatty foods, the alcohol is preferentially metabolized, which may stimulate the storage of fat when drinking is heavy [29]. Other factors that may contribute to a negative association between dosage and BMI include alcohol-related chronic disease and/or heavier drinking episodes associated with neglecting meals, being too drunk to eat, or suffering from hangovers [30].

Our identification of significant racial/ethnic differences in the distribution of drinking patterns and in the association between drinking patterns and BMI provides a framework for the development of hypotheses regarding mechanism(s) that may

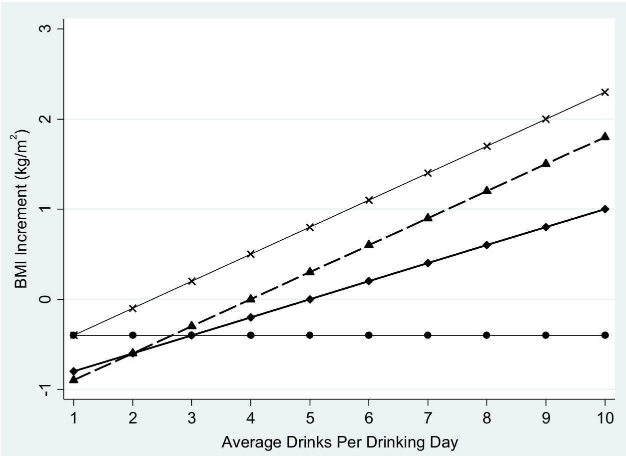

Fig. 1 Racial/ethnic influences of drinking quantity on BMI among US men, NHANES 1999–2010, with drinking frequency equal to 100 drinking days per year (adjusted for age, poverty income ratio, education, marital status, recreational physical activity status, current smoking status, and survey year). Solid line with x symbol, Mexican American. Dashed line with triangle symbol, Black. Thick solid line with diamond symbol, White. Solid line with circle symbol, other Hispanic

explain why race/ethnicity is associated with significant differences in the influence of alcohol consumption on BMI. For example, racial/ethnic genetic differences in ethanol metabolism may interact with drinking patterns and diet in ways that influence BMI. Ethanol is metabolized via two major pathways, alcohol dehydrogenase (ADH) and the hepatic microsomal ethanol oxidizing system (MEOS). Unlike energy from proteins, carbohydrates, or lipids, energy derived from ethanol cannot be stored; therefore, its metabolism takes priority, and the metabolism of lipids is suppressed when there is ethanol in the

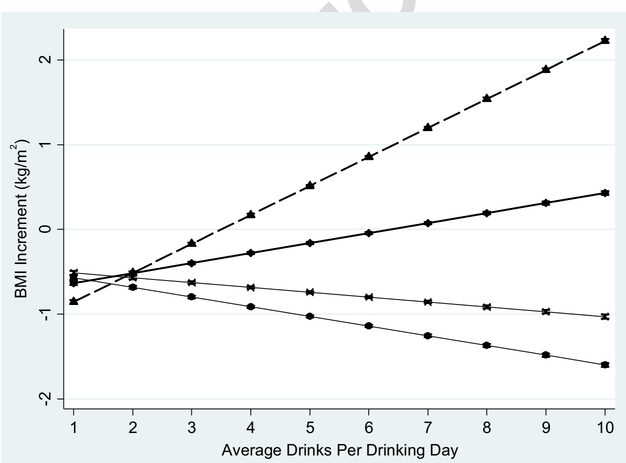

Fig. 2 Racial/ethnic influences of drinking quantity on BMI among US women, NHANES 1999–2010, with drinking frequency equal to 57 drinking days per year (adjusted for age, poverty income ratio, education, marital status, recreational physical activity status, current smoking status, and survey year). Dashed line with triangle symbol, Black. Thick solid line with diamond symbol, White. Solid line with x symbol, Mexican American. Solid line with circle symbol, other Hispanic

bloodstream [29]. Racial/ethnic differences in the ADH pathway among blacks [31] and Mexican Americans [32] are related to slower ethanol metabolism that may affect body weight more strongly, particularly in the presence of a high-fat diet. Increases in alcohol intake are associated with increasing fat intake [33], and drinkers tend to consume more fat on days when they drank compared to non-drinking days [34].

The MEOS metabolic pathway is induced by frequent intake of large amounts of alcohol, and it is less efficient in capturing energy from ethanol than the ADH pathway, "wasting" some of the calories contributed by drinking [29]. The amount of alcohol required to induce metabolism by the MEOS system seems to be highly variable and may be influenced by genetic factors [29]. If frequent consumption of moderate amounts of alcohol was associated with induction of the MEOS metabolic pathway, this might contribute to the negative association between drinking frequency and BMI.

An association between high-fat diets and heavy drinking may contribute to racial/ethnic differences in the relation of drinking patterns to BMI observed in this study; however, the association is likely to be complex. Ethnic drinking culture in their countries of origin, acculturation, gender, and education all influence drinking patterns of Mexican Americans and other Hispanics [35], and acculturation influences the fat content of diets among Mexican Americans [36].

We are not aware of any studies that have investigated racial/ethnic differences in the influence of alcoholic beverage preferences on the relation between drinking patterns and BMI. However, alcoholic beverage preferences are associated with dietary and other lifestyle characteristics likely to influence BMI. A study of predominately whites found that drinkers preferring wine were better educated, had higher incomes, and tended to have lower fat intakes and consume higher amounts of fruits, vegetables, and grain products, whereas beer and liquor drinkers were less well educated and had lower incomes, higher energy intakes and consumed fewer fruits, vegetables, and grain products [37]. Between 1989 and 2012, there was an increase in the proportion of US adults who drink on any given day, and an increase in calories consumed from alcoholic beverages when drinking occurs; among less-educated drinkers; beer contributed 70% of the latter increase [38]. Beer is the alcoholic beverage preferred by less-educated Mexican Americans [39].

Age could influence drinking patterns and their relation to BMI. There are significant racial/ethnic differences in drinking patterns related to age [40]. Heavy drinking among whites tends to be highest during late adolescence and early adulthood, a period during which weight gain tends to occur [41].

Limitations

This study has several limitations. The findings of the present study may have been confounded by recall bias although probes were used by interviewers to assist participants in recalling the number of drinking days and the number of drinks. The alcohol dose–response model assumes that response is a combination of linear functions of frequency and dosage. However, if BMI increases at lower dosages associated with maintaining or increasing food consumption and decreases at higher dosages associated with chronic disease and nutritional deficiencies, this could produce a curvilinear response. If the relations between drinking patterns and BMI are nonlinear, then our estimates are at best linear approximations and should be interpreted with caution. Dose–response models that are sensitive to curvilinear relationships can be estimated, but require more complete information about drinking distributions (i.e., the numbers of days on which exposures at different drinking levels occur) [16]. Reliable and valid survey methods in assessing the necessary drinking data have been developed in the USA [42], but we are not aware of large-scale epidemiological studies of BMI in which they have been applied. The cross-sectional nature of the data precludes inferences about causality or temporality. Furthermore, diet behavior and nutrition were not included as covariates because survey contents did not remain constant across surveys and therefore were not comparable [22]. It is also important to keep in mind that residual confounding by unmeasured variables is always a possibility in epidemiology (e.g., poor sleeping habits, medication, depression, chronic illness, or genetic aspects). Finally, BMI is only one marker of health, and a higher BMI does not necessarily imply that health is compromised.

Conclusion

The use of a mathematically derived linear alcohol dose–response model to investigate the impact of alcohol consumption on BMI revealed significant racial/ethnic and gender differences in the distribution of drinking patterns and the relation of drinking patterns to BMI. The average frequency was lower but the average dosage was higher among both black males and females compared to white males and females. In addition, dosage contributes significantly more to BMI among black females compared to white females. These racial/ethnic differences may account for racial/ethnic disparities in obesity and obesity-related health problems (e.g., diabetes and hypertension) among blacks. Further research is needed to investigate racial/ethnic and gender differences in factors that influence the effect of alcohol consumption on BMI (e.g., dietary intake, particularly of fat; consumption of alcohol in combination with high-fat foods; alcoholic beverage preferences; rates of alcohol metabolism; induction of the MEOS pathway of alcohol metabolism; country of origin; acculturation; age; health status; and their potential interactions). Such information is needed to design overweight and obesity prevention and treatment programs that differentially target men and women of different racial/ethnic backgrounds.

Compliance with Ethical Standards

Conflicts of Interest The authors declare that they have no conflict of interest.

Research Involving Human Participants All procedures performed in this study involving human participants were in accordance with the ethical standards of the Institutional Review Board and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. The study was exempted from the review of the Institutional Review Board at the authors' institution because of the public availability of the data.

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Q1. References [16] and [42] based on original manuscript we received were identical. Hence, the latter was deleted and reference list and citations were adjusted. Please check if appropriate.