

# Body Mass Index and Alcohol Consumption: Family History of Alcoholism as a Moderator

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Recent research suggests that excess food consumption may be conceptualized as an addictive behavior. Much of the evidence comes from neurobiological similarities between drug and food consumption. In addition, an inverse relation between alcohol consumption and body mass index (BMI) has been observed. Previous research has hypothesized that this inverse relation is attributable to competition between food and alcohol for similar neurotransmitter receptors. The current study explored this neurobiological hypothesis further by examining the influence of an indicator of biological risk associated with alcohol problems (family history of alcoholism) on the relationship between alcohol and food intake. Data from 37,259 participants in the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) were included in the study. BMI, family history of alcoholism, gender, and race/ethnicity were assessed as predictors of typical drinking frequency and estimated blood alcohol concentration (BAC). An inverse relationship between alcohol consumption and BMI was demonstrated. An attenuation of family history effects on drinking behavior was evident for obese compared to nonobese participants. The results suggest a neurobiological link between alcohol use and food consumption, consistent with theories characterizing excess food consumption as an addictive behavior.

*Keywords:* alcohol, BMI, family history, addiction, food

Although the “obesity epidemic” has recently received considerable media and research attention, the international rate of obesity continues to grow to epidemic proportions and shows no signs of slowing. Approximately one-third of American adults are obese (Yach, Stucker, & Brownell, 2006), and obesity has now topped alcohol to become the second leading cause of preventable death in the United States, surpassed only by tobacco use (Mokdad, Marks, Stroup, & Gerberding, 2004). The increased rates of obesity are accompanied by an increase in personal and societal spending to combat this problem. Although billions of dollars are spent on weight loss products and countless research studies are conducted to achieve sustainable weight loss, no successful solution has been found (Anderson, Stokholm, Backer, & Quaade, 1988). Recently, similarities between palatable food consumption and addictive substances have led to the theory that excess food consumption may be appropriately conceptualized as an addictive behavior (Gearhardt, Corbin, & Brownell, in press; Kleiner, Gold, Frost-Pineda, Lenz-Brunsmann, Perri, & Jacobs, 2004).

Both biological and behavioral evidence suggest that food may be addictive. First, neuroimaging and animal model research has found that excess food consumption is associated with neurobiological changes in the opiate and dopamine systems that parallel changes caused by drugs of abuse (Hajnal, Smith, & Norgren, 2004; Hoebel, Rada, Mark, & Pothos, 1999; Mark, Smith, Rada, & Hoebel, 1994; Nieto, Wilson, Cupo, Roques, & Noble, 2002;

Volkow et al., 2003). Behaviorally, animals given intermittent access to sugar develop symptoms of tolerance and withdrawal (Hoebel et al., 1999; Nieto et al., 2002), both of which are hallmarks of addiction. Many of the closest connections between food and addictive substances have been drawn between alcohol and high-fat, high-sugar foods. In addition to producing behavioral reinforcement through the same neurobiological pathway (Hajnal et al., 2004; Hoebel et al., 1999; Mark et al., 1994; Nieto et al., 2002) both high-fat sweets and alcohol are frequently used to regulate emotions (Canetti, Bachar, & Berry, 2001; Cooper, Frone, Russell, & Mudar, 1995). Research on human eating habits has also found behavioral evidence that maps onto substance dependence criteria, such as loss of control, continued use despite negative consequences, and an inability to reduce consumption of calorie dense foods (Gearhardt et al., in press; Kleiner et al., 2004).

Although theories of food addiction are relatively recent, researchers have been interested in the relationship between alcohol and weight gain for some time. Alcohol is a commonly consumed, calorie-dense substance, which led early theorists to predict that heavy alcohol consumption would increase the likelihood of being overweight or obese (Gruchow, Sobocinski, Barboriak, & Scheller, 1985). In fact, Gruchow et al. (1985) found that, although consumption of alcohol increases the overall consumption of calories, men and women drinkers do not appear to be at increased risk for obesity (Gruchow et al., 1985). More recent studies have actually found an inverse relationship between alcohol consumption and body mass index (BMI) (Lahti-Koski, Pietinen, Heliovaara, & Vartiainen, 2002; Liu, Serdula, Williamson, Mokdad, & Byers, 1994; Rohrer, Rohland, Denison, & Way, 2005). In other words, as BMI increases, alcohol consumption decreases. Although, this inverse relationship does not support the hypothesis

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that alcohol use contributes to obesity, it is consistent with the theory that food may be addictive. From a food addiction framework, researchers began to consider the possibility that alcohol and food's shared biological pathways might cause competition between the substances. In other words, when a pathway is occupied by one of the behaviors (i.e., food consumption or alcohol consumption), it would block the other (Kleiner et al., 2004).

These results are consistent with studies using pharmacotherapy to block opiate receptors. When opiate blockers, such as naltrexone, are used to block reward pathways, binge eaters reduce their consumption of sweet high-fat foods (Drewnowski, Krahn, Demitrack, Nairn, & Gosnell, 1995), and alcohol dependent participants reduce their consumption of alcohol (O'Malley, Krishnan-Sarin, Farren, Sinja, & Kreek, 2002). Working from this conceptual model, Kleiner et al. (2004) evaluated the relationship between BMI and alcohol use among individuals awaiting bariatric weight reduction surgery. They found that, in a female overweight and obese population, BMI was inversely associated with alcohol consumption, with 35.4% of extremely obese participants consuming alcohol in the last year compared with 62.5% of participants who were not obese.

Although suggestive of a shared biological pathway, the results of the Kleiner et al. study (2004) do not speak directly to the issue of biological vulnerability. Demonstrating a relation between biological risk for one behavior (e.g. alcohol use) and risk for excessive engagement in the other (e.g. overeating) would provide a more compelling argument for a shared biological vulnerability. There is, in fact, some evidence to suggest that such a relation may exist. For example, a personal or family history of alcohol problems can affect one's food choices. Alcoholics and those with a positive family history demonstrate a greater preference for higher concentrations of sweeteners than nonalcoholics or negative family history participants (Kampov-Polevoy, Garbutt, & Janowsky, 1999). These results suggest that risk for alcohol use may increase the hedonic value of foods that are implicated in obesity (Drewnowski, Kurth, & Rahaim, 1991). However, no studies to date have examined family history of alcoholism with respect to the relation between BMI and alcohol use.

In the current study, we hoped to fill this gap in the literature by using data from a large nationally representative data set (the National Epidemiologic Survey on Alcohol and Related Conditions; NESARC). By using a nationally representative sample, we were able to explore the risk factor of a family history of alcohol problems as a possible moderator of the relationship between alcohol consumption and body mass index. We hypothesized that measures of alcohol consumption would be significantly lower among obese and severely obese individuals relative to nonobese individuals (underweight, normal weight, and overweight). In addition, individuals with a positive family history of alcohol problems were expected to consume alcohol more frequently, and at higher typical levels (Merikangas et al., 1998). Finally, consistent with the idea that consumption of one substance can block reward pathways for the other, we expected the increased risk for elevated levels of alcohol consumption associated with a family history of alcoholism to attenuate at high levels of BMI (obese and morbidly obese).

In addition to the primary study hypotheses, we hoped to explore the impact of other potentially important moderators, including gender and race/ethnicity. Previous studies of the relationship

between alcohol consumption and BMI have found different patterns of results for men and women. Among women, an inverse relation between BMI and alcohol consumption has been found for both frequency and quantity of alcohol consumption (Lahti-Koski et al., 2002; Liu et al., 1994; Rohrer, Rohland, Denison, & Way, 2005). For men, studies of the association between BMI and alcohol use have not shown a consistent relationship in either direction. Frequency of alcohol use has not been associated with BMI, and although average alcohol consumption has been found to relate positively to BMI in some studies (Lahti-Koski et al., 2002), others have found no association (Colditz et al., 1991; Liu et al., 1994; Williamson et al., 1987).

The null findings in men may be attributable to aspects of the study designs used in prior research. Previous studies have often failed to report participants' ranges of body mass indices (Colditz et al., 1991; Gruchow et al., 1985; Liu et al., 1994; Williamson et al., 1987) or have had very few obese participants in the study samples. To the extent that decreased alcohol use is associated specifically with obesity (as opposed to overweight), restricted range of weights may have limited the ability to detect effects. Gender differences in weight may also have contributed to a lack of effects within samples of men. Drinking quantity is likely to differ by BMI simply because heavier individuals need to consume more alcohol to reach comparable blood alcohol concentrations (BACs), and this may be especially true for men who typically have higher average weights. Thus, it is important to control for the impact of weight when examining the quantity of alcohol consumed. For this reason, estimated BACs may provide the best index of drinking quantity for studies of the relation between alcohol use and BMI. In the current data set, we were able to explore the relation between BMI and BAC for all weight classifications and therefore hypothesized that the inverse relationship would be present for both male and female participants.

Although there is a lack of prior research on other factors that may moderate the relation between BMI and alcohol use, other important variables known to impact both alcohol consumption and risk for obesity should be considered. For example, racial/ethnic differences in rates of alcohol use/abuse and obesity are well documented. Although Caucasians often have higher rates of alcohol consumption (Higuchi, Parrish, Dufour, Towle, & Harford, 1993), Hispanic and African American populations typically have higher rates of obesity (Kumanyika, 1993). Although there is sufficient reason to suspect that race/ethnicity might serve as a moderator (based on known associations with both alcohol use and BMI), there is no prior empirical work to guide hypotheses about the nature of possible interactions. Thus, relative to gender, analyses of race/ethnicity as a possible moderator of the relation between BMI and alcohol use were more exploratory in nature. We also controlled for socioeconomic status (SES) and age in all analyses. It is possible that racial/ethnic group differences in alcohol use and food consumption are at least partially attributable to differences in SES. Thus, to accurately assess the role of race/ethnicity in the relation between alcohol use and BMI, it is necessary to control for the impact of SES. Age is another important characteristic associated with both alcohol use and obesity. As participants get older they are less likely to drink or to drink heavily (Johnston, O'Malley, Bachman & Schulenberg, 2006), but are more likely to be overweight or obese (Romeis, Grant, Knopik, Pedersen, & Heath, 2004). Thus, the inverse relation between BMI

and alcohol consumption could alternatively be explained by the aging process.

## Method

### Participants

The NESARC, which was designed and sponsored by NIAAA, is a longitudinal survey that began in 2001–2002. The target population of the NESARC is the civilian noninstitutionalized population, 18 years and older, residing in the United States and the District of Columbia. Data were collected from 43,093 Americans in the first wave of the survey. (Grant, Kaplan, Shepard, & Moore, 2003). A total of 37,259 participants had complete data on the variables of interest for the current study.

The subset of participants used in this study had an average age of 46.55 years ( $SD = 18.24$ ) and an average family income of \$35,000 to \$39,999. The mean weekly alcohol consumption was 3.11 drinks per week ( $SD = 9.24$ ), with the average participant drinking 0.99 days per week ( $SD = 1.81$ ). Mean quantity per drinking episode was 1.59 drinks ( $SD = 2.22$ ) with participants reaching an average BAC of .03 g% ( $SD = .04$ ). Not including the 35.78% of participants who did not drink, mean weekly alcohol consumption was 4.88 drinks per week ( $SD = 11.19$ ), with an average of 1.55 drinking days a week ( $SD = 2.05$ ) and 2.49 drinks ( $SD = 2.34$ ) per drinking episode. Drinkers reported reaching an average BAC of .04 g% ( $SD = .05$ ).

With respect to BMI, the majority of participants were in the normal weight category (40.4%). The largest racial/ethnic group was Caucasian (60.4%), although there were also relatively large numbers of African American (19.2%) and Hispanic (20.4%) participants because of oversampling of these groups. The gender breakdown was relatively even, although there were more women (56.5%) than men in the sample (43.5%). With respect to family history status, 77.0% reported no family history of alcohol prob-

lems, and 23.0% reported a family history of alcohol problems. Table 1 provides detailed demographics for the study sample.

### Procedures

The Census Supplementary Survey (C2SS), in combination with the Census 2000 Group Quarters Inventory, formed the sampling frame for the NESARC. One sample adult was selected for interview in each household (Grant et al., 2003). Data were collected in face-to-face computer-assisted personal interviews that were conducted in respondents' homes. The NESARC oversampled African American and Hispanic participants at the design phase of the survey and oversampled adults 18 to 24 years of age at the household level.

### Measures

BMI for each participant was computed by using the standard formula:  $[(\text{weight (lb)})/(\text{height (in)}^2)] \times 703$ . Participants were then classified by BMI into the following weight categories: underweight  $< 17.99$ , normal weight = 18–24.9, overweight = 25–29.9, obese = 30–39.9, and severely obese  $\geq 40$ .

A family member's history of alcohol problems was defined as that person having the following: physical or emotional problems because of drinking; problems with a spouse, family, or friends because of drinking; problems at work or school; legal problems (e.g. drunk driving arrests); or having to spend a lot of time drinking or being hungover. Participants were given this definition and were asked to evaluate whether each of their first-degree relatives was an alcoholic or problem drinker. In the current study, a positive family history of alcohol problems was indicated by either a paternal or maternal alcohol problem using this criterion. A negative family history of alcohol problems was indicated by neither parent meeting the criteria for an alcohol problem. The use of parental history of alcohol problems as an index of family

Table 1  
Alcohol Consumption by Weight Class, Family History Status, Gender, and Race/Ethnicity

Predictor variables	Weekly frequency of drinking days			Typical BAC			Average quantity of alcohol consumed		
	<i>n</i>	<i>M</i>	<i>SD</i>	<i>n</i>	<i>M</i>	<i>SD</i>	<i>n</i>	<i>M</i>	<i>SD</i>
Weight class									
Underweight (average BMI = 16.93)	463	.84	1.77	245	.07	.07	245	2.48	3.53
Normal weight (average BMI = 22.29)	14,703	1.09	1.90	9,669	.05	.05	9,669	2.39	2.11
Overweight (average BMI = 27.23)	13,385	1.08	1.86	8,690	.04	.05	8,690	2.55	2.27
Obese (average BMI = 33.38)	7,832	.76	1.56	4,710	.03	.04	4,710	2.56	2.42
Severely obese (average BMI = 44.65)	1,146	.47	1.24	614	.02	.04	614	2.74	4.58
Family history									
Negative	28,886	.96	1.78	17,986	.04	.04	17,986	2.37	2.19
Positive	8,643	1.10	1.88	5,942	.05	.06	5,942	2.86	2.73
Gender									
Female	21,214	.67	1.49	12,298	.04	.04	12,298	1.99	1.79
Male	16,315	1.41	2.08	11,630	.04	.05	11,630	3.02	2.71
Race/ethnicity									
African American	7,215	.79	1.66	3,737	.04	.04	3,737	2.28	1.99
Caucasian	22,668	1.16	1.93	15,627	.04	.05	15,627	2.42	2.15
Hispanic	7,646	.70	1.47	4,564	.05	.06	4,564	2.92	3.08

Note. BMI = body mass index.

history status is a common approach and one that has been used in prior studies from the NESARC data set (Grant et al., 2006; Stonenberg, Mudd, Blow, & Hill, 1998). Excluding those with alcohol-dependent mothers had little effect on outcomes, suggesting that fetal alcohol effects were not responsible for the observed results.

Participants were asked to respond to separate questions regarding the frequency (number of drinking days in the past year) and quantity (drinks per drinking day) of alcohol use. Participants were shown a series of flashcards with life-sized picture representations of different alcoholic beverages and the corresponding number of ounces to assist them in estimating standard drink size (Grant et al., 2003). The number of drinking days the participant reported was used to indicate frequency of alcohol consumption, whereas quantity was used to compute typical blood alcohol levels. Typical blood alcohol levels were computed using an empirically validated calculation (Hustad & Carey, 2005) for estimating BACs from naturally occurring drinking episodes [Women; (drinks per drinking day)/2] × [9/(weight in pounds)] – [.017/number of hours over which drinking occurred]; Men; [(drinks per a drinking day)/2] × [7.5/(weight in pounds)] – [.017/number of hours over which drinking occurred]. No information was available in the data set regarding the time over which alcohol consumption took place. Thus, three different time periods were initially examined; one hour, two hours, and three hours. Because the same pattern of results was found for each time frame, we report results only for the one-hour time frame as it produced the best distributional properties for BAC. Because the formula can occasionally return negative values (e.g. a 250-lb male consumes one drink over a one-hour period), any negative values were recoded as zero values.

*Data Analytic Plan*

All analyses were performed using SPSS version 15.0 (2007). Prior to conducting the primary analyses, all variables were examined for missing values. From the original 43,093 participants, 1,439 individuals did not provide sufficient information to calculate BMI and an additional 2,453 did not provide information on their parents' history of alcohol consumption. In regards to the dependent variable, 205 participants did not provide information on drinking frequency and 238 participants did not provide information on drinking quantity. In addition, only Caucasian, African American, and Hispanic participants were included because these

racial/ethnic categories had sufficient sample sizes to provide appropriate power for group comparisons. Thus, an additional 2,033 participants of other ethnic/racial backgrounds were not included in the analyses, resulting in a total of 37,259 participants in analyses testing the primary study hypotheses.

In addition, all variables were examined for outliers and normality. Outliers for the BAC distribution were identified as values that were extreme relative to the distribution (>3 SD above or below the mean) and/or were very unlikely to reflect true values. Because values greater than .30 g% are generally incapacitating and unlikely to occur on average, values above this cutoff were recoded to .30 g%. These values were more than 4 SDs above the mean, and only .07 % of participants had an average BAC above this cutoff. Frequency of alcohol consumption and typical BAC levels (after resetting extreme values) were significantly skewed (values of 5.35 and 2.77 respectively). Log transformation resulted in a skewness value of 1.44 for frequency of alcohol consumption, but transformations made little difference for BAC. Thus, the original BAC values were kept for ease of interpretation. BAC analyses were also conducted using nonparametric tests (i.e. Kruskal-Wallis), and the same pattern of results was found. Separate 2 (Family History Status) × 2 (Gender) × 5 (Weight Classification) × 3 (Race) Analyses of Covariance (ANCOVAs) were conducted for each outcome measure, with age and socioeconomic status included as covariates. The BAC analyses were only conducted for participants who reported drinking alcohol (n = 23,928), whereas analyses for drinking frequency included the full sample (n = 37, 259). To test the study hypotheses, we specifically assessed main effects for each independent variable and interactions between weight classification and each of the other independent variables. All significant interactions were decomposed by using the simple slopes procedures outlined by Aiken & West (1991). Table 2 provides a full correlation matrix of all variables included in the study.

Results

*Frequency of Alcohol Consumption*

Main effects were observed for SES,  $F(1, 37,502) = 485.228, p < .001$ , partial  $\eta^2 = .013$ , age,  $F(1, 37,502) = 60.987, p < .001$ , partial  $\eta^2 = .002$ , family history,  $F(1, 37,502) = 17.399, p < .001$ , partial  $\eta^2 = .000$ , weight class,  $F(4, 37,502) = 34.469, p < .001$ , partial

Table 2  
*Correlations Between the Study Variables*

	Age	Gender	Socioeconomic status	Frequency	Family history	Race	Weight class	Blood alcohol concentration
Age	1							
Gender	.037**	1						
Socioeconomic status	-.124**	-.133**	1					
Frequency	.001	-.223**	.125**	1				
Family history	-.100**	.046**	-.013**	.055**	1			
Race	-.178**	.022**	-.169**	-.139**	-.006	1		
Weight class	.079**	-.081**	-.012*	-.068**	.025**	.094**	1	
Blood alcohol concentration	-.282**	-.092**	.012*	.350**	.115**	-.075**	-.157**	1

Note. Frequency = Frequency of alcohol consumption.  
\* Correlation is significant at the .05 level (2-tailed). \*\* Correlation is significant at the .01 level (2-tailed).

$\eta^2 = .004$ , gender,  $F(1, 37,502) = 193.291, p < .001$ , partial  $\eta^2 = .005$  and race,  $F(2, 37,502) = 16.159, p < .001$ , partial  $\eta^2 = .001$ . The results suggested that higher socioeconomic status and younger age were associated with more frequent drinking. Men and family history positive participants also drank significantly more frequently than women or family history negative participants, respectively. With respect to race, Caucasian participants consumed alcohol more frequently than African American or Hispanic participants, who had relatively similar frequencies of consumption. Finally, obese participants consumed alcohol significantly less frequently than normal and overweight participants, and severely obese participants consumed alcohol significantly less frequently than normal weight, overweight, and obese participants (see Table 1).

Several 2-way interactions relating to the study hypothesis were also observed; family history by weight classification;  $F(4, 37,502) = 6.798, p < .01$ , partial  $\eta^2 = .001$ , gender by weight classification,  $F(4, 37,502) = 7.279, p < .001$ , partial  $\eta^2 = .001$ , and race/ethnicity by weight classification,  $F(8, 37,502) = 14.827, p < .001$ , partial  $\eta^2 = .003$ . Graphic depiction of the significant two-way interactions revealed likely moderation of family history and race effects related to obese/nonobese status. Thus, a 2-level variable consisting of nonobese (underweight, normal weight, and overweight) and obese (obese, morbidly obese) groups was created to simplify interpretation. Consistent with study hypotheses, the family history by weight classification interaction resulted from a significant simple main effect of family history for nonobese participants,  $F(1, 28,544) = 97.260, p < .001, \eta^2 = .003$ , but not for obese participants,  $F(1, 8,971) = 2.577, p = .28, \eta^2 = .000$ . Family history effects for all five weight classifications are depicted in Figure 1.

The race/ethnicity by weight classification interaction was also driven by larger effects for nonobese participants, partial  $\eta^2 = .011$ , than for obese participants, partial  $\eta^2 = .001$ , although the simple main effects of race were significant for both groups (see Figure 2 for racial/ethnic groups differences within each of the five weight classes). Caucasian participants drank more frequently than African American or Hispanic participants in both obese and nonobese groups. However, the rank ordering of Hispanic and African Americans for alcohol use differed by weight status.

Hispanic participants had the lowest frequency of alcohol use among the nonobese participants, whereas African Americans had the lowest frequency among the obese participants.

Unlike the 2-way interactions by weight class for family history status and race, the gender by weight classification interaction did not appear to be because of clear differences between obese and nonobese participants. Rather, inspection of the graph of the interaction suggested that it was driven by the two extreme ends of the BMI continuum (the underweight and morbidly obese categories). Additional analyses exploring simple main effects of gender within each weight classification confirmed our impressions based on the graph, with a reduced, although still significant effect of gender for underweight (partial  $\eta^2 = .017$ ) and morbidly obese participants (partial  $\eta^2 = .032$ ). The effect of gender was significant for all other weight classifications and resulted in a partial  $\eta^2 = .059$  for normal weight participants, partial  $\eta^2 = .076$  for overweight participants, and partial  $\eta^2 = .070$  for obese participants (see Figure 3).

#### Average Blood Alcohol Levels on Drinking Occasions

Once again, main effects were observed for socioeconomic status,  $F(1, 23,901) = 611.275, p < .001$ , partial  $\eta^2 = .025$ , age,  $F(1, 23,901) = 1706.301, p < .001$ , partial  $\eta^2 = .067$ , family history,  $F(1, 23,901) = 51.566, p < .001$ , partial  $\eta^2 = .002$ , weight class,  $F(4, 23,901) = 122.734, p < .001$ , partial  $\eta^2 = .020$ , gender,  $F(1, 23,901) = 9.225, p < .01$ , partial  $\eta^2 = .000$ , and race,  $F(2, 23,901) = 10.267, p < .001$ , partial  $\eta^2 = .001$ . The direction of the results was identical to the model for frequency of alcohol use for all variables other than socioeconomic status and race. Results in the BAC analyses suggested that lower socioeconomic status was associated with higher blood alcohol levels. With respect to race, Hispanic participants reached significantly higher blood alcohol levels than African American participants, with intermediate blood alcohol levels among Caucasian participants (see Table 1).

Two-way interactions between the predictor variables were observed for family history by weight classification,  $F(4, 23,901) = 11.314, p < .001$ , partial  $\eta^2 = .002$ , and race/ethnicity by weight

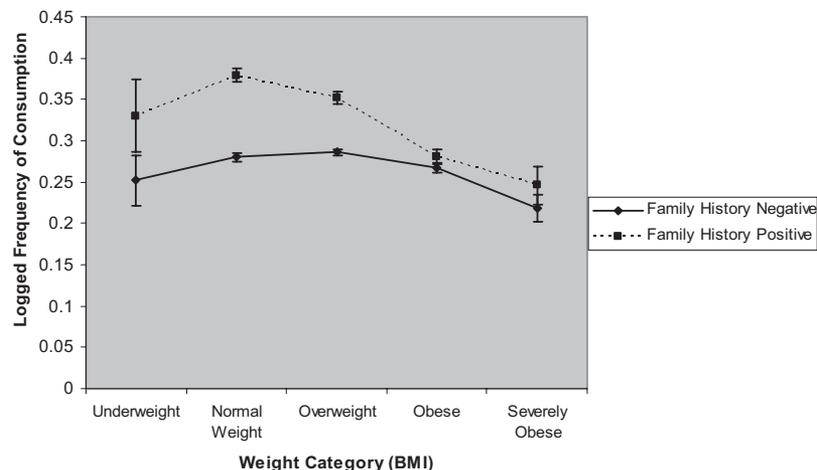


Figure 1. Weight class by family history interaction for frequency of alcohol use.

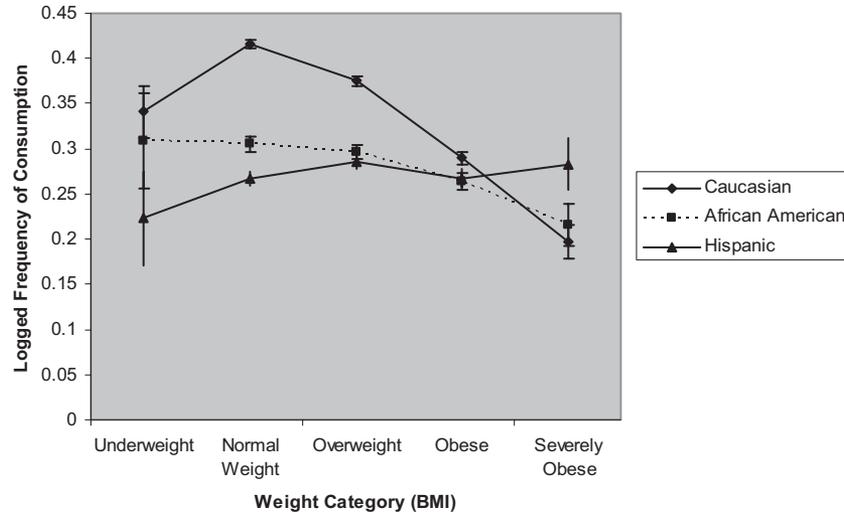


Figure 2. Weight class by race/ethnicity interaction for frequency of alcohol use.

classification,  $F(8, 23,901) = 4.059, p < .001$ , partial  $\eta^2 = .001$ . Once again, graphic depiction of the significant 2-way interactions revealed likely moderation of family history and race effects stemming from obese/nonobese status. Thus, a 2-level variable of obesity status was used to decompose the interactions. Simple main effects of family history were significant for both nonobese and obese participants, although the effect of family history was larger for nonobese, partial  $\eta^2 = .012$ , than for obese participants, partial  $\eta^2 = .001$  (see Figure 4). As in the analyses for drinking frequency, simple main effects of race/ethnicity were significant for both obese,  $F(2, 8,960) = 51.878, p < .001$ , partial  $\eta^2 = .011$ , and nonobese,  $F(2, 28,480) = 186.077, p < .001$ , partial  $\eta^2 = .013$ , participants. African Americans had the lowest blood alcohol levels for both groups, but BACs for Caucasian and Hispanic participants differed by obesity status. In the nonobese group, Caucasian and Hispanic participants had similar blood alcohol

levels, whereas Hispanic participants demonstrated the highest BACs in the obese group (see Figure 5).

### Discussion

Many of the main effects identified in the current study were consistent with the extant literature. Men, younger participants, and those with a family history of alcohol problems drank more frequently and had higher typical BACs than women, older participants, or family history negative participants, respectively (Johnston et al., 2006; Merikangas et al., 1998; Wilsnack, Vogeltanz, Wilsnack, & Harris, 2000). Also consistent with previous literature, Caucasian participants consumed alcohol more frequently than African American or Hispanic participants (Higuchi et al., 1993). In contrast to the findings for frequency, Hispanic participants had higher average BACs than both Caucasian and

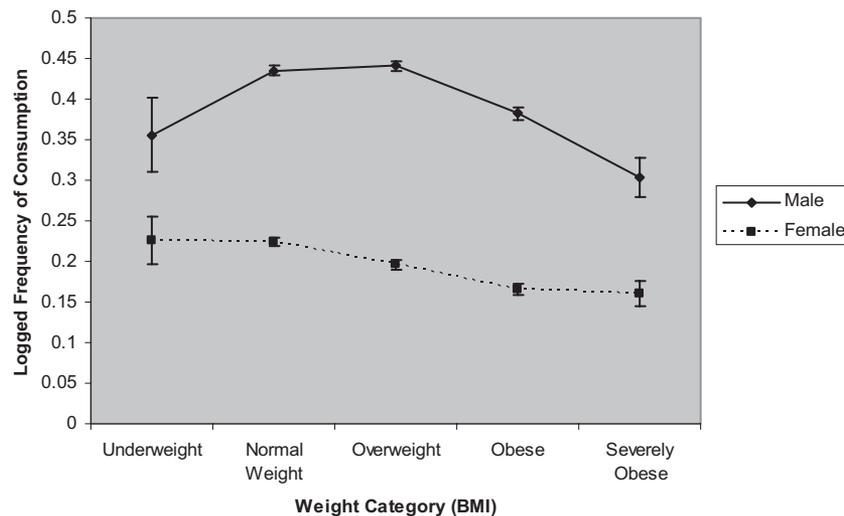


Figure 3. Weight class by gender interaction for frequency of alcohol use.

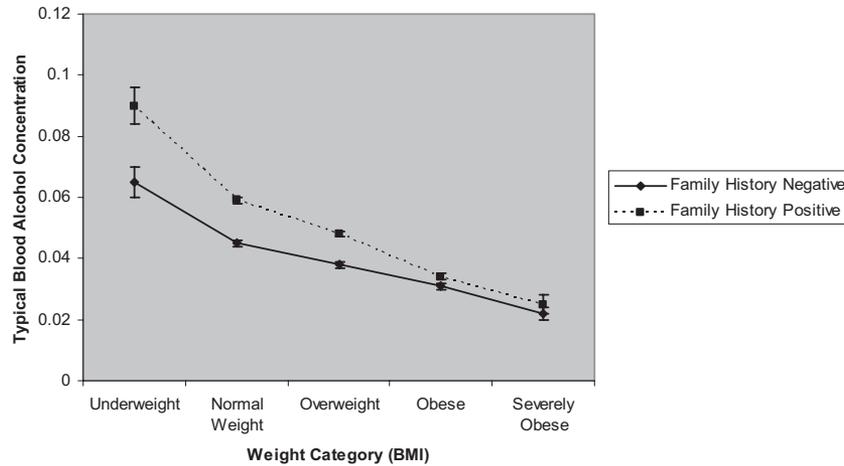


Figure 4. Weight class by family history interaction for typical BAC.

African American participants. Although low SES has generally been considered a risk factor for alcohol-related problems (Van Oers, Bongers, Van De Goor & Garretsen, 1999), more recent research has shown increased risk at very low and very high levels of SES (Hanson & Chen, 2005). The results of the current study were consistent with this more recent literature with higher frequency of consumption among high SES individuals and higher BACs among low SES individuals. The results for both race/ethnicity and SES highlight the importance of disaggregating frequency and quantity of alcohol consumption when evaluating group differences in drinking behavior.

Most important to the hypotheses of the current study, obese participants consumed alcohol significantly less frequently and had lower typical BACs than normal weight and overweight participants, and severely obese participants consumed alcohol significantly less frequently and had lower typical BACs than normal weight, overweight, and obese participants. In contrast to some previous studies, the inverse relation between alcohol use and BMI was evident for both men and women. The identification of a

consistent inverse relationship between alcohol use and BMI among men in the current study may be related to differences in methodology from previous studies (Colditz et al., 1991; Lahti-Koski et al., 2002; Liu et al., 1994; Williamson et al., 1987). In the current study, we had a sufficient sample size to explore the relation between alcohol consumption and BMI across the full range of BMI, and frequency of alcohol use was disaggregated from quantity. In addition, the measure of drinking quantity (BAC) took into account weight-related differences on the impact of quantity consumed. Although replication of the current results is important, the strengths of the methodology used provide compelling evidence that the inverse relation between alcohol use and BMI is not exclusive to women.

In addition to main effects, there were important interactions between weight classification and the demographic variables assessed. Although there was an interaction between gender and weight classification for frequency of consumption, the same general pattern of results was seen for both men and women. The interaction was driven by a lack of gender differences among

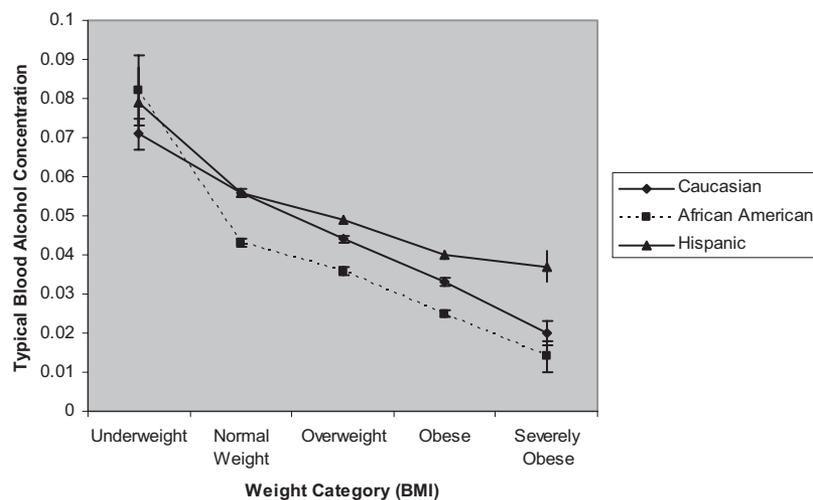


Figure 5. Weight class by race/ethnicity interaction for typical BAC.

underweight and severely obese participants, with robust gender differences in all other weight classes. Perhaps the lack of gender differences in the underweight group relate to higher levels of alcohol consumption associated with certain eating disorders (e.g., bulimia nervosa) that occur disproportionately among women (Dansky, Brewerton, & Kilpatrick, 2000). In regards to the severely obese participants, it is possible that the reduced impact is because of floor effects associated with low frequency of consumption for women.

Race/ethnicity by weight classification interactions were observed for both drinking frequency and BAC. Although the nature of the interaction differed somewhat across the two outcomes, in both cases the interaction was driven by the sample of Hispanic participants. For frequency of consumption, the Hispanic sample was at lowest risk in the nonobese sample and at intermediate risk in the obese sample and for BAC, the Hispanic sample was at intermediate risk in the nonobese sample and at highest risk in the obese sample. In both cases, the result is that obesity status had less of an impact on alcohol use for Hispanic participants than for Caucasian or African American participants. The smaller effect for Hispanic participants was not expected, and it is unclear through what mechanism this effect may be operating. Biological differences in response to alcohol do indeed exist among different racial/ethnic groups and such differences may have important implications for health and well-being (Chan, 1986; Galarza, Diaz, Guzmán, Caballero, & Martínez, 1997). It is also quite possible that racial/ethnic group effects identified in the current study are a result of cultural or environmental influences.

Of central importance to the shared biological vulnerability hypothesis, an interaction between family history status and weight classification was found for both measures of alcohol use. The influence of a family history of alcohol problems was moderated by BMI, such that family history effects on frequency of alcohol consumption were only present among nonobese individuals. Although family history was a significant predictor of BAC for both obese and nonobese participants, the effect was over three times larger for nonobese participants. Although these results do not provide direct evidence for shared neurobiological pathways, they are quite consistent with the hypothesis that food occupies neurobiological pathways related to the reinforcement value of alcohol. Although it is not entirely clear what biological systems might be associated with the increased risk associated with a family history of alcohol problems, the opioid and dopamine systems have been identified as likely candidates (Gianoulakis, 2001; Sander et al., 1995). Thus, the blocking of these neurobiological pathways may be responsible for the attenuation of family history effects on drinking behavior. Although our results are consistent with this conclusion, future studies would benefit from the direct examination of the dopamine and opioid systems to confirm the blocking hypothesis. Regardless of the mechanism, this study supports the burgeoning literature suggesting that excessive food intake may be similar to other addictive behaviors, specifically compulsive alcohol use (Drewnowski et al., 1995; Gearhardt et al., in press; Gold, Frost-Pineda, & Jacobs, 2003; Kleiner et al., 2004).

Although the results were consistent with the blocking hypotheses, other possible explanations for the results must also be considered. For example, it may be that obese and severely obese participants drink less because they would need to consume a prohibitively large quantity of alcohol to reach a BAC level that is

reinforcing. Because participants with a family history of alcoholism have been shown to have an innate tolerance to alcohol (Schuckit & Smith, 2000), obese individuals with a positive family history would need to consume even greater quantities of alcohol to achieve reinforcing BACs. Although plausible, there are both conceptual and empirical reasons counter to this potential alternative explanation. Conceptually, the increased quantity of alcohol necessary to achieve an intoxicating BAC does not appear to be substantial enough to prevent obese individuals from drinking to intoxication. For example, during a 2-hour period, an obese 5'6" woman (weighing 210 lbs) would need to consume less than one additional alcoholic beverage to reach a BAC of .05, relative to an overweight 5'6" woman (weighing 170 lbs). The data presented in Figure 4 also argue against this alternate explanation. If the difficulty of consuming enough alcohol were the driving force behind reduced consumption among obese participants, one would expect the effects to be most pronounced among the morbidly obese participants. However, the attenuation of alcohol consumption was seen clearly among both the obese and morbidly obese groups, and attenuation of family history effects was also evident in both groups. Thus, it seems unlikely that difficulty consuming sufficient alcohol to reach reinforcing BACs was responsible for the pattern of results in the current study.

An additional alternative social explanation of the current results is worth considering. Consumption of alcohol frequently takes place in social contexts, such as bars or parties. Obese individuals are often subjected to discrimination based on weight bias (Puhl & Brownell, 2001). The experience of weight-based discrimination could result in reduced participation in social activities, including activities in which alcohol consumption is common. Thus, it is possible that the reduction in alcohol consumption may be due to negative social experiences. Although this is possible, it is less likely that this possibility would explain the reduced impact of a family history of alcohol problems. In fact, one might expect that the negative feelings associated with weight bias would increase alcohol consumption, especially for those with the added risk factor of a positive family history.

Although there were a number of strengths of the research methodology, several important limitations should be considered when interpreting the results. The study was strengthened by the use of a large, nationally representative sample, but the NESARC oversampled African American and Hispanic participants, as well as individuals between the ages of 18 and 24 years. In this study, we addressed this issue by controlling for age in our analyses and by examining the effect of weight on alcohol consumption within each racial/ethnic category. Nonetheless, we cannot say with confidence that the results will generalize to the general U.S. population. In addition, the NESARC depended upon self-report data for all of the variables used in the study. Thus, incorrect reporting of alcohol consumption, body weight, or family history of alcohol problems may have affected the results. This may be especially pertinent, as weight was a variable for which there was significant missing data. Heavier participants may be less likely than normal weight participants to report their weight, such that overweight and obese participants may have been underrepresented in this study. Future studies would benefit from direct assessment of weight and collateral reports of alcohol use to reduce reporting error.

Although the calculation of BACs in the current study was a relative methodological strength, participants were not asked to

report the amount of time over which the alcohol was consumed. Thus, it was necessary to estimate typical time of consumption. Although the pattern of results was consistent across three different possible time periods, future studies would benefit from assessing typical time of consumption. Finally, the effect sizes in the current study were small in magnitude. Small effects are not surprising for behaviors as complex as food and alcohol consumption. It is unlikely that the relationship between obesity and alcohol consumption will explain a large amount of the total variability in these behaviors.

Despite the relatively small effect sizes, we believe there are both public health and theoretical reasons that the findings are important. First, both alcohol use disorders and obesity are major health concerns. Even minor impacts on the consumption of either calorie-dense foods or alcohol use may have major societal implications. Secondly, as Kazdin (2003) points out, small effect sizes in the company of theoretical underpinnings can have a major impact on the understanding of a phenomenon of interest. In the current study, the relationship between alcohol consumption, obesity, and family history is accompanied by a theoretical explanation regarding the addictive properties of food. The finding that a risk factor for excess alcohol consumption can be attenuated by excess food consumption has important implications for the concept of food addiction.

If the characterization of excess food consumption as a possible addictive behavior is accurate, it may have important implications for the prevention and treatment of excessive food consumption. Perhaps the most important implication for food's possible addictive qualities is the potential impact of the "toxic environment" (Brownell, 2004, p. 7) of highly available energy-dense foods. The widespread availability and aggressive advertising of unhealthy foods may play on cue-triggered relapse to derail public health interventions to decrease consumption of these unhealthy foods. Thus, public policy interventions designed to limit exposure to "toxic foods" for both adults and children may prove to be effective in reducing excess food consumption. With respect to treatment, empirically validated approaches for substance dependence and binge eating share important similarities. Both treatments include identification of triggers and other relapse prevention strategies (Agras, 1993; Witkiewitz & Marlatt, 2004). In addition, numerous treatments based on the 12-step Alcoholics Anonymous model, such as Overeaters Anonymous, are currently available. If food is addictive, this would suggest that future obesity and binge eating treatments should continue to explore methods used to treat substance dependence. For example cue-exposure and identification of alternative reinforcers (Monti et al., 1993) may be worth considering in the treatment of obesity and binge eating disorder. Although food's addictive nature is far from established, the results of the current study, in combination with previous research, suggest that further exploration of this possibility is warranted.

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Received May 9, 2008

Revision received October 22, 2008

Accepted December 3, 2008 ■