ORIGINAL ARTICLE

The Emerging Link Between Alcoholism Risk and Obesity in the United States

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Context: The prevalence of obesity has risen sharply in the United States in the past few decades. Etiologic links between obesity and substance use disorders have been hypothesized.

Objective: To determine whether familial risk of alcohol dependence predicts obesity and whether any such association became stronger between the early 1990s and early 2000s.

Design: We conducted analyses of the repeated cross-sectional National Longitudinal Alcohol Epidemiologic Survey (1991-1992) and National Epidemiologic Survey on Alcohol and Related Conditions (2001-2002).

Setting: The noninstitutionalized US adult population in 1991-1992 and 2001-2002.

Participants: Individuals drawn from population-based, multistage, random samples (N = 39 312 and 39 625).

Main Outcome Measure: Obesity, defined as a body mass index (calculated from self-reported data as weight in kilograms divided by height in meters squared) of 30 or higher and predicted from family history of alcoholism and/or problem drinking.

Results: In 2001-2002, women with a family history of alcoholism (defined as having a biological parent or sibling with a history of alcoholism or alcohol problems) had 49% higher odds of obesity than those without a family history (odds ratio, 1.48; 95% confidence interval, 1.36-1.61; $P < .001$), a highly significant increase ($P < .001$) from the odds ratio of 1.06 (95% confidence interval, 0.97-1.16) estimated for 1991-1992. For men in 2001-2002, the association was significant (odds ratio, 1.26; 95% confidence interval, 1.14-1.38; $P < .001$) but not as strong as for women. The association and the secular trend for women were robust after adjustment for covariates, including sociodemographic variables, smoking status, alcohol use, alcohol or drug dependence, and major depression. Similar trends were observed for men but did not meet statistical significance criteria after adjustment for covariates.

Conclusions: These results provide epidemiologic support for a link between familial alcoholism risk and obesity in women and possibly in men. This link has emerged in recent years and may result from an interaction between a changing food environment and predisposition to alcoholism and related disorders.

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OBESITY, DEFINED BY THE World Health Organization[1] as body mass index (BMI; calculated as weight in kilograms divided by height in meters squared) of 30 or higher, is associated with an increased incidence of hypertension, type 2 diabetes mellitus, coronary heart disease, stroke, gallbladder disease, osteoarthritis, sleep apnea, respiratory problems, and certain cancers.[2,3] Moreover, the prevalence of obesity in the United States has doubled in the past 3 decades, from 15% in 1976-1980 to 33% in 2003-2004.[4] Correspondingly, there has been a marked increase in the risk of premature death due to obesity-related disease, and the relative contribution of obesity-attributable mortality to total US deaths rose substantially between 1990 and 2000.[5,6]

Increases in obesity in the United States are not simply the result of an across-the-board population weight increase. Rather, distributions of BMI across time in American adults are characterized by a gradual shift toward a higher mean BMI but with a marked increase in the higher end of the distribution, indicating that the largest increases have occurred in the highest weight categories.[4,7] The increase in mean BMI may be due to changes in environmental factors that influence all individuals, such as greater availability of high-calorie foods.[8,9] However, the increased number of people at the higher end of the BMI distribution suggests that certain subgroups of individuals are particularly vulnerable to such a changing environment.

Among the factors that might contribute to differential vulnerability to overeating in an obesigenic environment is a deficiency in impulse control, possibly related to individual differences in sensitivity to neurochemical rewards. These character-

istics are hallmarks of substance use disorders,[10,11] and behavioral and neurobiological commonalities between overeating-associated obesity and substance use disorders have been documented in recent years.[12-18] Substance use disorders and overeating-associated obesity are complex and moderately heritable; both are influenced by availability and access to highly reinforcing substances (ie, drugs or palatable foods), both are aggravated by stress, and both lead to dopamine-modulated neurobiological adaptations.[15] Observational and laboratory studies have detected links between impulsive characteristics and overeating,[12,19,20] as well as a preference for highly palatable (eg, sweet, salty, or fatty) foods.[12] Therefore, it is plausible that individuals at risk for substance use disorders have been differentially affected by the obesity epidemic in the United States.

The purpose of this article is to investigate whether the subset of the US population at elevated risk for alcohol use disorders, as indicated by a family history of alcoholism (FHA), has experienced greater increases in obesity than the subset of the population with no FHA. We accomplish this by examining data from repeated cross-sectional samples of the noninstitutionalized adult population of the United States in the National Longitudinal Alcohol Epidemiologic Survey (NLAES; 1991-1992) and the National Epidemiologic Survey on Alcoholism and Related Conditions (NESARC; 2001-2002). With more than 40 000 participants in each survey, we were able to investigate whether increases in obesity have been more prominent in individuals with a FHA or, in other words, whether the association between FHA and obesity has increased across time. We also investigated whether any such changes could be attributed to confounding sociodemographic characteristics, smoking status, alcohol use, or depression. A clearer understanding of the relationship between familial risk of addiction, obesity, and the changing environment may help inform prevention and treatment efforts for the subpopulation of obese individuals who are prone to addictive behaviors.

METHODS

SAMPLE

The NLAES and NESARC surveys focused on alcohol and drug use, *DSM-IV* substance use disorders, and associated impairment in samples representative of the civilian, noninstitutionalized adult population of the United States. There were many methodological similarities between the 2 surveys, including the sampling universe and instrumentation used to assess alcohol dependence and related risk factors, such as FHA, major depression, and other disorders. Blacks were oversampled in both surveys, and Hispanics were oversampled in the NESARC. Face-to-face interviews were administered by experienced lay interviewers from the US Census Bureau. Respondents were informed about measures taken to ensure the confidentiality of the information they provided. Ethical review and approval of all procedures were conducted by the US Census Bureau and US Office of Management and Budget; all participants provided informed consent. The NESARC sample consisted of 43 093 persons; the overall raw response rate was 81%. The NLAES sample consisted of 42 862 persons, with a response rate of 90%. The analytical samples excluded people with missing height or weight measurements or incomplete or indeterminate family history information, pregnant women, and underweight individuals (BMI $\leq$18.5). This resulted in final sample sizes of 39 312 for the NLAES and 39 625 for the NESARC, or 91.7% and 92.0% of the total samples, respectively. Further details on both surveys and comparative descriptions of methods are available elsewhere.[21-24]

ASSESSMENT

Psychiatric diagnoses, alcohol consumption, and smoking status were assessed in both surveys with the Alcohol Use Disorder and Associated Disabilities Interview Schedule—*DSM-IV* Version (AUDADIS-IV),[25] which covers *DSM-IV* substance use syndromes for the past year and during the lifetime. The psychiatric assessments in both surveys included major depression, alcohol use disorders, and drug use disorders.

Measures of smoking history and past-year drinking were also administered in the NLAES and NESARC versions of the AUDADIS-IV. Questions about height and weight were included in both surveys. The AUDADIS-IV queries FHA by asking whether a relative has "been an alcoholic or problem drinker at *any* time in his/her life." This probe is repeated for each relative type: mother, father, brother, sister, half-sibling, and children.

VARIABLES AND COVARIATES

Main Outcome and Predictor Variables

The BMI classification was chosen as the primary outcome variable; BMI was calculated from self-reported data. The BMI scores were used to classify participants as obese, defined as BMI of 30 or higher, and nonobese, defined as BMI of less than 30.[1] This dichotomous classification was used instead of BMI as a continuous variable because of the previously mentioned change in the shape of the BMI distribution across time. Moreover, relationships between BMI and morbidity or mortality may be nonlinear,[26-28] suggesting that changes in obesity are more relevant for public health considerations than the overall change in mean BMI. The primary predictor variable, FHA, was defined as having either a biological parent or full biological sibling with a history of problem drinking or alcoholism based on the AUDADIS-IV family history assessment.

Participants who were not raised with biological relatives or who reported "unknown" alcoholism or problem-drinking status for all parents and siblings were excluded from the analysis. In addition, pregnant women and underweight individuals were excluded (underweight may be indicative of severe illness). The NLAES queried hospitalization owing to pregnancy in the past year, whereas the NESARC asked whether women were currently pregnant. Hence, the NLAES exclusion was slightly broader.

Sociodemographic Covariates

Racial and ethnic categorization included non-Hispanic white, non-Hispanic black, Hispanic, Asian/Pacific Islander, and "other." The "other" category included groups that were too small for independent analysis, such as indigenous Americans and non-Hispanic multiracial individuals. Race/ethnicity was determined by self-report, and categories were drawn from more detailed categories, defined by the survey administrators, that followed the 1990 and 2000 census conventions for the NLAES and NESARC, respectively. Race/ethnicity was assessed in these surveys as a demographic variable and for statistical weighting purposes.

Age was categorized into 6 groups: 18-27, 28-37, 38-47, 48-57, 58-67, and 68 years or older. With respect to educational

level, individuals were categorized into 1 of 4 groups: no high school diploma or General Educational Development certificate, high school diploma or General Educational Development certificate only, some college or other postsecondary education but not a bachelor's degree, and bachelor's degree or higher. Participants from each survey were grouped into quartiles defined by total annual household income. Quartiles were defined separately for each survey, and cutoffs were determined by the ranking of unweighted data.

Behavioral Covariates

On the basis of responses to smoking-history questions, individuals were categorized as current smokers, former smokers, or nonsmokers. Former smokers were those who had smoked 100 or more cigarettes in their lives but none in the past 12 months, whereas current smokers were defined as those who had crossed the 100-cigarette threshold and had smoked in the past 12 months. Nonsmokers were those who had never crossed the 100-cigarette threshold. The estimated number of drinks per month during the previous 12 months was computed from retrospective self-report by multiplying the typical frequency of drinking a given beverage by the number of drinks of that beverage consumed on a typical occasion. This value was included as a continuous variable in several analyses. Participants were also categorized according to their *DSM-IV* alcohol dependence status, with "current" alcohol-dependent participants meeting the criteria for a past-year diagnosis and "former" alcohol-dependent participants meeting the criteria for alcohol dependence before, but not during, the past 12 months. The same approach was taken for drug dependence, with all separate types of drug dependence assessed in the NLAES and NESARC categorized as a single diagnosis of current or former drug dependence. The *DSM-III-R* diagnoses of major depression on a past-year and lifetime basis are available in both the NLAES and NESARC and were used as covariates in the multivariate analyses.

Statistical Procedures

All descriptive statistics and associated standard errors, as well as regression models, were calculated using the SUDAAN statistical software package.[29] Variance estimation used a Taylor linearization method appropriate for the multistage design of the surveys. Significance of between-survey differences in odds ratios (ORs) was assessed using 2-sample z tests applied to the β-coefficients (log OR). This is the large-sample equivalent of a 2-sample t test; z is the between-sample difference in effect size divided by the pooled standard error.

RESULTS

Table 1 describes participants with complete data on FHA and BMI, after exclusions for current (or recent) pregnancy and underweight status (see the "Sample" subsection in the "Methods" section), and includes cross-tabulations of family history by sociodemographic and psychiatric/behavioral covariates. For the NLAES, 880 participants were excluded owing to pregnancy, 785 because of missing BMI, 630 because of missing family history, and 1255 owing to underweight status. In the NESARC, 453 participants were excluded owing to pregnancy, 1423 because of missing BMI, 762 because of missing family history, and 830 owing to underweight status. This resulted in final sample sizes of 39 312 for the 1991-1992 NLAES and 39 625 for the NESARC, conducted 10 years later in 2001-2002. In the NLAES, individuals with missing BMI were slightly more likely than those in the analysis samples to report a FHA (OR, 1.11; $P=.03$), and this effect was more pronounced in the NESARC (OR, 1.33; $P<.001$). There were no differences in obesity between the analysis sample and individuals with missing family history in the NLAES or the NESARC.

The prevalence of obesity was 14.9% (95% confidence interval [CI], 14.2%-15.6%) in the NLAES sample and 23.0% (95% CI, 22.2%-23.8%) in the NESARC sample. Mean (SD) BMI in the NLAES was 25.4 (5.2) for women and 26.0 (4.1) for men. In the NESARC, it was 27.0 (6.1) for women and 27.3 (4.8) for men.

In the NLAES, 32.2% of the sample reported a FHA (95% CI, 30.6%-33.8%) with a very similar percentage reporting a FHA in the NESARC (32.4%; 95% CI, 31.3%-33.5%). The likelihood of having a FHA was similar in whites, blacks, and Hispanics but was much lower among Asians. Women were slightly more likely to report a FHA than men, a result that was consistent across types of relatives (eg, mother, father, brother, or sister) and surveys. Individuals between the ages of 28 and 57 years reported higher rates of FHA than participants who were 18 to 27 years old or 58 years or older.

The full BMI distribution in the combined-sex NLAES and NESARC samples, stratified by FHA, can be seen in the **Figure**. The Figure shows remarkably little difference in BMI distribution between individuals with and without a FHA in the NLAES. However, in the NESARC, the 2 subpopulations clearly diverge. At BMI values of 30 and higher, the cutoff for obesity, individuals reporting a FHA constitute a higher proportion of membership in any BMI range than those without a FHA.

The core result—the prevalence of obesity stratified by FHA in the NLAES and NESARC—is quantified in **Table 2**. Although there was only a modest association between FHA and obesity in 1991-1992, a highly significant association for both sexes was observed in 2001-2002. The bivariate OR describing the association was significantly higher for women in 2001-2002 compared with women 10 years earlier (OR, 1.48 vs 1.06; cross-survey difference test: $z=5.77$; $P<.001$). There was a similar secular trend for men, but statistical significance was marginal (OR, 1.26 vs 1.08; $z=1.92$; $P=.055$). Although the overall prevalence of obesity for both sexes increased between the NLAES and the NESARC, the increase was significantly stronger among those with a FHA, and this effect was particularly pronounced for women.

We conducted similar comparisons between individuals with and without a FHA that were stratified by major demographic and behavioral variables. Stratification variables included race/ethnicity, age, educational level, total annual household income, smoking status, alcohol dependence status, drug dependence status, and major depression. These results are listed in eTable 1 and eTable 2 (http://www.archgenpsychiatry.com). Women (eTable 1) and men (eTable 2) are presented separately owing to the differences exhibited in the primary comparisons. For almost all the stratified comparisons, the association between FHA and obesity in the NESARC was stronger than in the earlier NLAES, as evidenced by a trend

Table 1. Sample Descriptions

Characteristic	NLAES, 1991-1992 (N = 39 312)[a]			NESARC, 2001-2002 (N = 39 625)[a]		
	No. of Participants	Weighted % (SE)	% With FHA (SE)	No. of Participants	Weighted % (SE)	% With FHA (SE)
Sex						
Male	17 130	50.1 (0.3)	30.5 (0.4)	17 650	49.7 (0.3)	29.9 (0.6)
Female	22 182	50.0 (0.3)	34.0 (0.4)	21 975	50.3 (0.3)	34.8 (0.7)
Race						
Non-Hispanic, white	29 852	76.8 (0.4)	33.1 (0.3)	22 572	71.1 (1.6)	33.1 (0.5)
Non-Hispanic, black	5469	11.2 (0.3)	30.8 (0.8)	7527	11.0 (0.7)	31.9 (1.0)
Hispanic	2589	7.7 (0.3)	30.3 (1.1)	7710	11.6 (1.3)	32.4 (1.1)
Asian/Pacific Islander	865	2.8 (0.1)	13.5 (1.4)	1055	3.8 (0.5)	12.7 (1.3)
Other	537	1.5 (0.1)	43.3 (2.9)	761	2.5 (0.2)	42.5 (2.0)
Age, y						
18-27	6731	19.2 (0.3)	31.3 (0.7)	6403	17.8 (0.3)	28.1 (0.9)
28-37	9233	23.4 (0.3)	37.3 (0.6)	7774	19.1 (0.3)	33.2 (0.8)
38-47	7387	20.0 (0.3)	36.1 (0.7)	8236	21.5 (0.3)	36.9 (0.8)
48-57	4818	13.4 (0.2)	34.2 (0.8)	6384	17.1 (0.3)	36.4 (1.0)
58-67	4643	11.1 (0.2)	28.4 (0.8)	4498	10.9 (0.2)	32.5 (1.0)
≥68	6500	13.0 (0.2)	20.0 (0.6)	6330	13.6 (0.3)	24.4 (0.7)
Educational level[b]						
No high school	8057	19.5 (0.3)	33.7 (0.7)	7146	15.5 (0.5)	37.0 (1.0)
High school only	12 060	31.5 (0.3)	33.3 (0.5)	11 509	29.2 (0.6)	33.5 (0.7)
Some college	9933	26.3 (0.3)	33.8 (0.6)	11 693	30.3 (0.4)	34.4 (0.7)
College degree	8823	22.7 (0.3)	27.9 (0.6)	9277	25.0 (0.6)	25.7 (0.6)
Annual household income						
Lowest quartile	8945	17.9 (0.3)	26.5 (0.6)	9850	19.0 (0.4)	33.6 (0.8)
2nd quartile	10 260	26.8 (0.3)	32.4 (0.6)	9545	21.9 (0.4)	34.0 (0.8)
3rd quartile	9878	26.2 (0.3)	34.4 (0.6)	9806	26.0 (0.3)	32.4 (0.8)
Top quartile	10 229	29.1 (0.3)	33.7 (0.6)	10 424	33.0 (0.8)	30.5 (0.7)
Smoking status[b]						
Never	19 698	49.9 (0.3)	27.1 (0.4)	22 801	55.6 (0.7)	26.7 (0.6)
Current	11 514	29.6 (0.3)	40.2 (0.6)	9169	24.5 (0.5)	42.9 (0.8)
Former	8085	20.5 (0.3)	33.3 (0.6)	7649	19.9 (0.4)	35.3 (0.8)
Alcohol dependence						
Never	34 255	86.5 (0.2)	29.0 (0.3)	35 098	87.1 (0.4)	29.4 (0.5)
Current	1622	4.5 (0.1)	52.5 (1.5)	1401	3.9 (0.1)	49.1 (1.7)
Former	3435	9.0 (0.2)	53.5 (1.0)	3126	9.0 (0.3)	53.6 (1.0)
Drug dependence						
Never	38 190	97.1 (0.1)	31.3 (0.3)	38 396	96.6 (0.2)	31.2 (0.6)
Current	189	0.5 (0.0)	57.4 (4.2)	229	0.6 (0.1)	62.6 (3.9)
Former	933	2.4 (0.1)	66.5 (1.7)	1000	2.8 (0.1)	64.5 (1.8)
Major depression						
No	30 036	77.5 (0.3)	27.8 (0.3)	31 370	79.3 (0.4)	28.5 (0.5)
Yes	9276	22.5 (0.3)	47.5 (0.6)	8255	20.7 (0.4)	47.3 (0.7)

Abbreviations: FHA, family history of alcoholism; NESARC, National Epidemiologic Survey on Alcohol and Related Conditions; NLAES, National Longitudinal Alcohol Epidemiologic Survey.

[a] After exclusions for missing data on FHA, height, weight, underweight status, or current or recent pregnancy.

[b] Category total does not match sample size because of missing data and/or "Don't Know" responses.

toward higher ORs in the NESARC (although not all differences were statistically significant). The only exceptions to this trend were for Asian/Pacific Islander men and for women who listed "other" as their race/ethnicity. In both cases, these groups comprise relatively small and potentially heterogeneous strata. Still, the association between FHA and obesity met nominal significance criteria ($P<.05$) for 36 of 60 comparisons in the NESARC compared with only 3 of 60 comparisons for NLAES data. These results suggest that the increased influence of FHA across time is independent of several factors indicative of socioeconomic status and social disadvantage and is not a result of confounding by sociodemographic or behavioral factors.

Table 3 lists adjusted ORs from a series of planned logistic regression analyses in which potentially explanatory covariates are entered sequentially into a model predicting obesity from a FHA. For both sexes, the addition of sociodemographic covariates (race/ethnicity, age, educational level, and household income) resulted in a small reduction in the difference in OR between the NLAES and NESARC. For women, the ORs remained highly significant in the NESARC, as did the difference in OR between the NLAES and NESARC. (The addition of state of residence as a demographic variable resulted in essentially no change in ORs and therefore was not included in subsequent analyses.) In the second model listed in Table 3, alcohol dependence, drug depen-

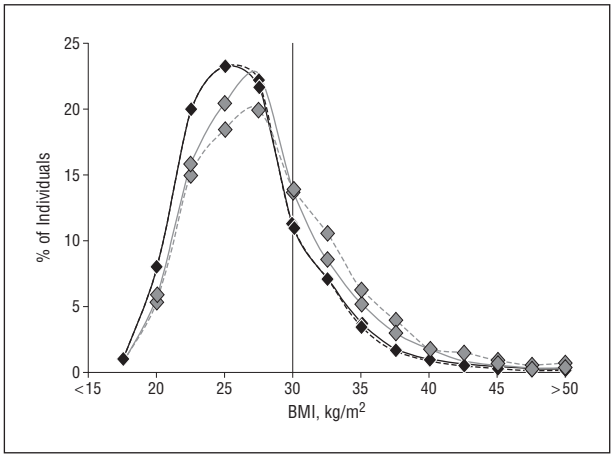

Figure. Body mass index (BMI) distributions of the 1991-1992 National Longitudinal Alcohol Epidemiologic Survey (black line) and 2001-2002 National Epidemiologic Survey on Alcohol and Related Conditions (gray line) weighted samples stratified by family history of alcoholism. Frequencies were calculated in 2.5-kg/m^2 intervals, up to 50 kg/m^2. Individuals with a BMI lower than 18.5 were excluded. Solid lines indicate family history of alcoholism; dashed lines, no family history of alcoholism. Data points are connected via a polynomial smoothed line.

dence, smoking status, and a quantitative measure of alcohol consumption were added. For both sexes, these had a minimal effect on the ORs in both surveys.

Finally, because a FHA is likely to be associated with major depressive disorder and obesity is also a correlate of major depression and/or other mood disorders in the United States,[30,31] lifetime history of major depression was added to the models. In both surveys, this resulted in modest and nonsignificant changes in the association between FHA and obesity for both sexes. In these final models, the adjusted ORs for women were 1.30 (95% CI, 1.19-1.43) in the NESARC and 1.00 (95% CI, 0.91-1.10) in the NLAES (P for difference $<.001$). Hence, the association between FHA and obesity among women remained highly significant in the NESARC ($P<.001$) and the difference in the association between the NLAES and NESARC remained significant for women ($P<.001$). A modest association between FHA and obesity remained for men in the NESARC after adjustment (OR, 1.11; 95% CI, 1.01-1.23; $P=.02$); this was a slight but nonsignificant increase compared with the NLAES (OR, 1.02; 95% CI, 0.94-1.11).

COMMENT

In this study, we used 2 large epidemiologic samples representative of the noninstitutionalized adult population of the United States to examine secular trends in the association between familial risk of alcoholism and obesity. We found a significant association between FHA and obesity among men and women surveyed in 2001-2002. Furthermore, this association was substantially greater in 2001-2002 than in 1991-1992 among women, with a qualitatively similar but less pronounced trend among men ($P=.05$) in bivariate analyses. For men, the association between FHA and obesity in 2001-2002 was smaller than for women but still statistically significant. For women, the association and the secular trend remained significant after adjustment for other sociodemographic variables, smoking status, alcohol consumption, alcohol and drug dependence, and major depression. For men, the secular trend was nonsignificant after adjustment for covariates.

Our findings suggest that a link between FHA and obesity has emerged in recent years, particularly among women. In other words, the interaction between factors related to a FHA and the increasingly obesigenic environment may have resulted in a differential increase in the prevalence of obesity among individuals vulnerable to addiction. This may be specifically the result of a changing food environment and the increased availability of highly palatable foods.[18,32-34] Our finding is consistent with a body of psychological and neurobiological literature describing overeating as an addictive behavior.[12,14,15,17,18,35,36] This point of view postulates that neurocircuitry activated by drugs of abuse overlaps regions of the brain involved in food-related rewards. Behavioral studies also support this idea. For example, some studies have suggested an association between FHA and a preference for sweet foods.[37,38] However, to our knowledge, this is the first documentation of a link between alcoholism and obesity using epidemiologic data and, more important, the first study to suggest that the epidemiologic association between alcoholism risk and obesity has grown across time and perhaps emerged fairly recently.

There was a small albeit nonsignificant reduction in effect size in the association between FHA and obesity among women after adjusting for major depression (the only non–substance-related psychiatric diagnosis assessed in both surveys), and this reduction might have been larger if other psychiatric disorders had been assessed in both surveys. Familial alcoholism may lead to obesity in part through psychiatric comorbidity. Other causes, including a common etiologic mechanism for obesity and other psychiatric disorders that correlate with alcoholism risk, are also plausible.[39-41] Twin models suggest some overlap between the genetic etiologic features of depression and alcoholism; interestingly, evidence for this overlap is stronger in women than in men.[42-44] Regardless of the role of depression and other psychiatric disorders in the causal pathway between familial alcoholism risk and obesity, documentation of this association and its change across time is a significant step in understanding individual differences in vulnerability to the obesity epidemic.

The magnitude of the ORs describing the association between FHA and obesity, even among women, is not exceptionally large. However, our main finding is the change in the magnitude of this association between 1991-1992 and 2001-2002. Notably, the measurement uncertainty inherent in a brief self-reported assessment of a FHA would likely bias effect size estimates downward. The fact that we observed a highly significant change in these ORs for women, and a suggestive trend for men, during the relatively short period of 10 years could have significant implications for understanding obesity in a sizeable subset of the population. If there is a portion of the population whose eating behaviors are more "addiction-like" than others, characterizing such individuals could aid in the individualized treatment of obesity. Pharmacologic and psychosocial interventions modeled on addiction treatment may be indicated in such individuals.

Table 2. Prevalence of Obesity by FHA: 1991-1992 and 2001-2002

	NLAES (1991-1992)				NESARC (2001-2002)			
	No. of Participants	Prevalence, % (SE)		OR (95% CI) (FHA vs No FHA)	No. of Participants	Prevalence, % (SE)		OR (95% CI) (FHA vs No FHA)
		No FHA	FHA			No FHA	FHA	
Women	22 182	15.4 (0.4)	16.1 (0.5)	1.06 (0.97-1.16)	21 975	20.8 (0.5)	28.0 (0.7)	1.48 (1.36-1.61)[a,b]
Men	17 130	13.9 (0.4)	14.9 (0.6)	1.08 (0.97-1.22)	17 650	21.4 (0.6)	25.5 (0.7)	1.26 (1.14-1.38)[a,c]
Total	**39 312**	14.6 (0.3)	15.6 (0.4)	1.08 (1.00-1.15)[d]	**39 625**	21.1 (0.5)	26.9 (0.5)	1.37 (1.28-1.47)[a,b]

Abbreviations: CI, confidence interval; FHA, family history of alcoholism; NESARC, National Epidemiologic Survey on Alcohol and Related Conditions; NLAES, National Longitudinal Alcohol Epidemiologic Survey; OR, odds ratio.
[a] $P<.001$.
[b] NESARC OR differs from NLAES with $P<.001$.
[c] NESARC OR differs from NLAES with $P=.05$.
[d] $P=.05$.

Table 3. Association Between FHA and Obesity Using Various Models: 1991-1992 and 2001-2002

Additional Model Covariates	NLAES (1991-1992) OR (95% CI), FHA vs No FHA	NESARC (2001-2002) OR (95% CI), FHA vs No FHA	Comparison (NESARC vs NLAES)	
			z Score	*P* Value
Women				
Race, age, educational level, and income	1.02 (0.93-1.12)	1.35 (1.23-1.48)[a]	4.30	<.001
Race, age, educational level, income, alcohol consumption, alcohol dependence, drug dependence, and smoking status	1.05 (0.96-1.15)	1.38 (1.25-1.51)[a]	4.09	<.001
Race, age, educational level, income, alcohol use, smoking status, and major depression	1.00 (0.91-1.10)	1.30 (1.19-1.43)[a]	3.97	<.001
Men				
Race, age, educational level, and income	1.00 (0.89-1.13)	1.13 (1.02-1.24)[b]	1.49	.13
Race, age, educational level, income, alcohol consumption, alcohol dependence, drug dependence, and smoking status	1.06 (0.94-1.19)	1.13 (1.03-1.25)[c]	0.88	.38
Race, age, educational level, income, alcohol use, smoking status, and major depression	1.03 (0.91-1.16)	1.11 (1.01-1.23)[d]	0.52	.60

Abbreviations: See Table 2.
[a] $P<.001$.
[b] $P=.01$.
[c] $P=.02$.
[d] $P=.04$.

A better understanding of the emerging link between FHA and obesity requires further examination of this association in recently ascertained, high-quality, general population samples or carefully designed case-control samples. A more complete psychiatric characterization of participants in such samples could also help refine the pathways through which this association occurs. In addition, it would be desirable to identify specific eating behaviors and psychological characteristics that mediate the link between FHA and obesity. These might include general dietary patterns; preferences for short-term rewards instead of delayed gratification[45,46]; preferences for highly rewarding sweet, salty, or fatty foods[18,37,38]; and binge-eating behaviors.[47-49] To our knowledge, this is the first study to examine the association between FHA and obesity and to study repeated cross-sections of the US population to examine recent secular trends. Gearhardt and Corbin[50] noted an association between FHA and BMI in the NESARC but in the context of understanding drinking behavior in relation to obesity. Barry and Petry[51] noted an association between lifetime alcohol use disorder and obesity in men and an inverse association between past-year alcohol use disorders and obesity among women. The intent of their analysis, however, was not to specifically examine FHA.

The large population-based samples and the analysis of repeated cross-sections of the population constitute considerable strengths. The repeated cross-section approach is particularly well suited for estimating overall change within a population.[52] The use of self-reported height and weight to determine BMI is a limitation and could potentially bias the estimates of the association between FHA and obesity. Self-reported information is known to result in underestimated BMI, with effects that may differ by age, sex, and measured BMI.[53-56] On average, measured BMI is about 0.6 kg/m^2 higher than self-reported BMI, and the discrepancy is larger for individuals with higher BMI.[56] Obesity prevalence estimates based on measurement are approximately 50% higher than those based on self-report.[57] On the other hand, prevalence estimates of obesity obtained in our study are quite close to the self-reported estimates produced by the Behavioral Risk Factor Surveillance System survey,[58] and estimates from that survey have exhibited secular trends and

associations with health outcomes similar to those based on physical measurement.[52,57] The correlation between measured and self-reported BMI ranges from 0.89 to 0.97,[56,59] and the correlation of reporting bias with sociodemographic variables is mitigated in these analyses by the inclusion of numerous sociodemographic covariates in the multivariate models. As demonstrated in eTables 1 and 2, the association between FHA and self-reported obesity is consistent across sociodemographic categories. Considering all these factors, the tendency for individuals with higher BMI to underestimate their BMI to a larger degree, if independent of a FHA, could result in a slight underestimation of the true association between FHA and obesity.

Missing data also may influence effect size estimates. Those with indeterminate family history status were no more or less likely to be obese than others in the NLAES or NESARC, but those with missing BMI data were more likely to report a FHA, an effect that was larger in the NESARC. It is difficult to speculate on how this correlation might affect the results, but if social desirability is a component of missing BMI data (ie, passive refusal to report high weight), this may result in slight underestimates for both the association between FHA and obesity and the secular trend.

Although we tested for several potential confounding relationships, other confounding variables may not have been assessed in both the NLAES and NESARC. In addition, potential explanatory variables such as physical activity, caloric intake, binge-eating behaviors, and other psychiatric disorders were not measured. However, for the secular trend to be attributable to an unmeasured confounding variable, it would be necessary for that variable to be correlated with both FHA and obesity and to have changed across time. This same principle applies to biases inherent in self-reported height, weight, and FHA. In other words, presuming such biases are stable across time, they are unlikely to account for the secular trend in the association between FHA and obesity.

We emphasize that our findings apply only to trends in the United States. There are many environmental contributors to both alcoholism (and by extension, having a family member with alcoholism) and obesity that vary from one cultural context to another. Notably, the United States has much higher rates of obesity than other developed countries[60] but slightly lower rates of alcohol consumption than those countries.[61] Cross-cultural replication of these analyses could provide further insight into the environmental factors that have contributed to these secular trends.

In conclusion, in the decade between the early 1990s and early 2000s, a clear link between familial alcoholism risk and obesity has become apparent in the United States. The link is more prominent among women, for whom it is not explained by potentially confounding sociodemographic variables, smoking status, alcohol consumption, substance dependence, or major depression. These findings provide epidemiologic support for the etiologic links between addiction and overeating or obesity documented in neurobiological studies.[15] Moreover, our results are consistent with the hypothesis that relatively recent environmental changes have contributed to this link. The fields of obesity research and addiction research have a mutual interest in working together to find treatments for obese individuals from high–addiction-risk backgrounds and developing a more detailed understanding of the shared etiologic mechanism between these conditions.

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