

Data from the National Health and Nutrition Examination Survey, conducted from 1971 through 1974, and the National Health and Nutrition Examination Survey Follow-up, conducted from 1982 through 1984, were used to test for a beneficial effect of moderate alcohol consumption on coronary heart disease mortality among White men and women. Evidence of such an effect was found for White men; accelerated time-tofailure models showed 3% to 4% longer life spans for moderate drinkers than for nondrinkers or light drinkers. (Am J Public Health. 1993;83:888-890)

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Moderate Drinking and Coronary Heart Disease Mortality: Evidence from NHANES I and the NHANES I Follow-up

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Introduction

In this paper I use data from the National Health and Nutrition Examination Survey (NHANES I), conducted from 1971 through 1974, and the National Health and Nutrition Examination Survey Follow-up, conducted from 1982 through 1984, to examine the relationship between mortality from coronary heart disease and alcoholic beverage consumption. A large number of autopsy, ecologic, case-control, and cohort studies have shown that alcoholic beverage consumption on the order of one to four drinks per day is associated with lower rates of mortality from coronary heart disease than are lower and higher levels of consumption. Two reviews of the case-control and cohort studies show the relative risk of coronary heart disease from moderate alcohol consumption, compared with no consumption, to be concentrated in the .4 to .8 range.1,2 Both of these reviews suggest that a positive association between alcohol consumption and high-density lipoprotein cholesterol concentrations may be the biological pathway by which moderate drinking provides protection from coronary heart disease. There is some concern that the finding of seemingly beneficial effects of moderate alcohol consumption in the major cohort studies may be due to a failure to control for nondrinkers in poor health at baseline.3 However, it has been argued that this is not a likely explanation of the findings because of the care taken in many of the longitudinal studies to exclude subjects with cardiovascular disease at baseline and because the mortality advantage of moderate drinkers does not appear to wear off over time.2

Methods

NHANES I is a stratified probability sample of the US population containing data on about 20 000 individuals collected in the period from 1971 through 1974.⁴ The NHANES I Follow-up contains data on about 11 000 individuals who were examined as part of NHANES I and who were 25 through 74 years of age at the time of the NHANES I examination.5 These individuals were surveyed for the Follow-up in the period 1982 through 1984, or approximately 10 years after their NHANES I examination. The Follow-up effort successfully traced about 95% of the eligible NHANES I participants and had a survey completion rate of 85%. Approximately 2000 members of the NHANES I cohort had died by the time of the Follow-up. Causes of death were determined from diagnostic information in hospital and mortality records.

Summary statistics from NHANES I and the Follow-up for White men are consistent with a protective effect of alcoholic beverage consumption on mortality from coronary heart disease. The age-adjusted proportions of subjects who died during the approximately 10-year period between NHANES I and the Follow-up from myocardial infarction or ischemic heart disease are .10, .05, and .04 for nondrinkers, moderate drinkers (up to two drinks per

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day), and heavy drinkers (more than two drinks per day), respectively. The drinking data are from NHANES I and refer to drinks per day of the subject's favorite alcoholic beverage.

To statistically examine the relationship between alcoholic beverage consumption and coronary heart disease with these data. I estimated accelerated timeto-failure models for White men and women in NHANES I and the Follow-up. Accelerated time-to-failure models are from the class of models known as survivor models; they are functionally related to hazard models. These models are required when duration data are censored. In the context of mortality models, sample observations are right-censored when subjects live through the sample frame. The models are estimated by maximizing a likelihood function that depends largely on the assumed distribution of the hazard rate over time to failure. In mortality studies it is usually assumed that hazard rates increase with age, and different distributions reflecting this assumption are tested in accelerated time-to-failure models to see which best fit the data.6 Explanatory variables rescale time until failure in these models, and their coefficients can be given a partial regression coefficient interpretation.7

In the accelerated time-to-failure model presented below, age at death (time until failure) is assumed to depend on a high blood pressure binary variable; a cigarette smoking binary variable; family income; education; and alcoholic beverage consumption variables adjusted for body weight. These explanatory variables are defined in Table 1. Alcoholic beverage consumption is measured with none/light, moderate, and heavy drinking binary variables to capture possible nonlinear effects of alcohol consumption on mortality and to contend with the underreporting that may characterize survey data when subjects are interviewed about their drinking choices.^{8,9} If the underreporting is concentrated among heavy drinkers, the dummy variable representation of alcoholic beverage consumption may provide estimates of alcoholic beverage consumption effects on mortality that are less subject to misinterpretation than continuous measures. In essence, the heavy drinking dummy accurately captures heavy drinking.

Results

Estimates of the accelerated time-tofailure model with a Weibull baseline du-

TABLE 1—Definitions and Summary Statistics for Explanatory Variables				
None/light, moderate, and heavy drinking	Dummy variables designating subjects in the 0–19th, 20th–89th, and 90th–100th percentiles of the distribution for White men of weekly number of drinks of favorite alcoholic beverage divided by body weight; from the National Health and Nutrition Examination Survey (NHANES I). Mean number of drinks per week of favorite alcoholic beverage, 6.5; SD, 13.9.			
Age	Age in years at time of NHANES I interview. Mean, 50.8; SD, 15.1.			
Smoker	Dummy; 1 = active smoker or active smoker at time of death; from Follow-up. Mean, .31; SD, .46.			
HighBP	Dummy; 1 = diastolic blood pressure above 95 mm Hg; from NHANES I. Mean, .16; SD, .35.			
Education	Years of schooling; from NHANES I. Mean, 11.1; SD, 3.7.			
Income	Family income divided by 10 000; from NHANES I. Mean, 1.12; SD, 7.27.			

TABLE 2—Maximum Likelihood Estimates of Time-to-Failure Models, Weibuli Baseline Duration Distribution, White Men in NHANES I and the Follow-up^a

	Mortality, All Causes (No. Deaths = 512; No. Survivors = 2801)		Mortality, Coronary Heart Disease (No. Deaths = 171; No. Survivors = 2801) ^b	
	Coefficient (SE)	χ^2	Coefficient (SE)	χ^2
Smoker	106 (.009)	127.5	132 (.017)	56.8
Moderate drinker	.029 (.010)	7.8	.041 (.019)	4.8
Heavy drinker	019 (.016)	1.4	022 (.631)	.6
High blood pressure	014 (.011)	1.6	030 (.019)	2.5
Education	.001 (.000)	.1	.003 (.002)	2.4
Income	001 (.000)	.2	001 (.000)	1.2
Intercept	4.445 (.013)	10446	4.523 (.025)	3328
Scale	.100 (.000)		.106 (.005)	
Log likelihood	-296		-262	

Note. Critical values for chi-square are 3.84 and 2.71 for .05 and .10 significance levels, respectively. NHANES I = National Health and Nutrition Examination Survey.

"Subjects who reported a history of heart attack, heart failure, heart disease, malignant turnor, ulcer, hepatitis, or diabetes at the time of NHANES I were deleted from the samples.

^bSubjects who died from causes other than myocardial infarction or chronic ischemic heart disease deleted from the sample.

ration distribution are presented in Table 2 for White men in NHANES I and the Follow-up. Log normal, log logistic, and exponential hazard formulations yielded lower maximized values of the log likelihood than did the Weibull distribution. To guard against problems of reverse causality caused by nondrinkers in poor health at baseline, a healthy sample at baseline was approximated by deleting from the sample individuals who reported a history of heart attack, heart failure, heart disease, malignant tumor, ulcer, hepatitis, or diabetes at the time of NHANES I (about 15% of the White men in the Follow-up). Two sets of estimates are presented in Table 2. The first models time until death from any cause as a function of the explanatory variables. Statistical evidence of a beneficial effect of moderate drinking in this

model would presumably be due to a protective effect of moderate drinking on coronary heart disease mortality. In the second set of statistical estimates, time until death from coronary heart disease is modeled, with deaths from other causes deleted from the sample. One fourth of all deaths are due to coronary heart disease. Results for the first model show that moderate drinking increases time until death from any cause by about 3%, whereas heavy drinking reduces time until death by 2%, although the latter point estimate is not statistically significant. These percentage difference interpretations are possible because time until death is in natural logs in the accelerated time-to-failure models. Active smoking has a strong negative effect on time until death and the high blood pressure binary variable indicates that this

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condition reduces life span by about 1%. In the second model, smoking, drinking, and high blood pressure effects are larger in absolute value, reflecting the greater importance of these explanatory variables in death from coronary heart disease than in death from all causes. The models fit the data well; the simple correlation between the raw hazard rates and those predicted by using the models' parameters are .87 for the first model and .75 for the second. If the unhealthy subjects are included in the sample or if the light drinking category is confined to nondrinkers, there is little change in the statistical results.

Discussion

These results are consistent with a substantial body of previous research that finds a protective effect of moderate



Data from the Hispanic Health and Nutrition Examination Survey were used to examine the influence of acculturation on alcohol consumption among Puerto Rican, Cuban-American, and Mexican-American women in the United States. Acculturation was found to be positively related to frequency of consumption and probability of being a drinker among all three groups. A positive relationship was also evident for total drinks consumed among Cuban-American women, and volume (drinks per occasion) and total drinks consumed among Mexican-American women. (Am J Public Health. 1993:83:890-893)

drinking on coronary heart disease mortality for White men. Nevertheless, they must be interpreted with caution, because the alcohol consumption variable references drinking during the baseline period only. Also, accelerated time-tofailure model results for White women did not show an important effect for moderate drinking. \Box

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Acculturation and Alcohol Consumption in Puerto Rican, Cuban-American, and Mexican-American Women in the United States

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Introduction

Interest in alcohol consumption among Hispanics in the United States has increased in recent years.^{1,2} In particular, researchers have begun to examine the complex relationship between alcohol consumption and level of acculturation.3,4 Much of this work has been limited to the Mexican-American population, with a particular emphasis on male drinking patterns. Some evidence has accrued, however, that acculturation is more consistently related to the drinking practices of Hispanic women rather than men.5-7 This evidence supports an acculturation model, according to which increased drinking behavior results from the adoption of the norms, practices, and values of the larger society.8

As Hispanic populations are diversifying, it is important to examine how cultural change is influencing their health and health behaviors. For example, are Hispanic women adopting the drinking norms of the dominant culture as they become more acculturated into the larger US society? This question is explored among Puerto Rican, Cuban-American, and Mexican-American women.

Methods

This study used data collected as part of the Adult Sample Person Supplement Questionnaire of the Hispanic Health and Nutrition Examination Survey

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