Association of Lifetime Alcohol Drinking Trajectories with Cardiometabolic Risk

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Context and Objective: Alcohol intakes may vary considerably over a drinker's lifetime. This study was designed to examine whether lifetime drinking trajectories are associated with cardiovascular risk factors that are used to define the metabolic syndrome (MetS).

Design, Setting, Participants, and Outcomes: This is a population-based cross-sectional study. Participants were ever-regular drinkers (n = 2818) selected from healthy controls for the Western New York Health Study (1996–2001) in which lifetime lifestyle was ascertained retrospectively. Prevalence of the MetS and its individual components, including obesity, high triglycerides, low high-density lipoprotein cholesterol, elevated blood pressure, and high fasting glucose, were the main outcomes.

Results: Trajectory analyses were based on estimates of total kilograms of ethanol for each age decade between 10 and 59 yr. Two groups of drinkers with distinct lifetime drinking trajectories were obtained, an early peak and a stable trajectory group. Compared with stable trajectory drinkers, early-peak drinkers were 10 yr younger on average, had earlier onset of regular drinking, drank heavily in late adolescence and early adulthood tapering off in middle age, averaged more drinks per drinking day in lifetime, and were more likely to abstain when interviewed. After controlling for age, sex, and other potential confounders, early-peak trajectories were modestly associated with high odds of the MetS [1.31; 95% confidence interval (CI) 1.00, 1.71] overall, low high-density lipoprotein cholesterol (1.62; 95% CI 1.27, 2.08), abdominal obesity (1.48; 95% CI 1.23, 1.78), and overweight (1.32; 95% CI 1.10, 1.60).

Conclusion: Early initiation of alcohol drinking and heavy drinking in adolescence and early adulthood may be associated with an adverse cardiometabolic profile. **(*J Clin Endocrinol Metab* 93: 154–161, 2008)**

Little is known about the variability of lifetime drinking patterns and the cumulative effects of lifetime drinking patterns on cardiovascular risk. In most studies of alcohol-related chronic health conditions, alcohol intake has been assessed at a single point in time, under the assumption that drinking patterns are fairly stable over the lifetime. In cases in which this assumption is met, current and past drinking patterns are very similar, making it difficult, if not impossible, to disaggregate acute and chronic effects of alcohol on cardiovascular risk. However, recent analyses of lifetime drinking trajectories in a population-based sample of adults revealed considerable variability in alcohol intakes over the lifetime in almost half of those examined (1). Studies based on assessment of alcohol intake at a single point in time cannot adequately characterize lifetime exposure to alcohol in these individuals or differentiate them from subjects who do have stable lifetime drinking patterns. To address this issue, we developed the Cognitive Lifetime Drinking History (CLDH), a computer-assisted personal interview designed to assess drinking patterns retrospectively over the lifetime in studies of chronic conditions related to alcohol use (2). To date, analyses of CLDH

0021-972X/08/$15.00/0

Printed in U.S.A.

doi: 10.1210/jc.2007-1395 Received June 22, 2007. Accepted October 15, 2007.

First Published Online November 20, 2007

Abbreviations: ABO, Abdominal obesity; BMI, body mass index; CHD, coronary heart disease; CI, confidence interval; CLDH, Cognitive Lifetime Drinking History; DM, diabetes mellitus; HBP, elevated blood pressure; HTG, raised triglyceride; IFG, impaired fasting glucose; LHDLC, low high-density lipoprotein cholesterol; MetS, metabolic syndrome; OR, odds ratio; WNYHS, Western New York Health Study.

data have been limited to summary measures estimated from the lifetime patterns (3). However, summary measures do not fully capture the changing patterns of alcohol consumption over the lifetime that are obtained retrospectively using the CLDH. In this study we use the CLDH data to investigate, through an innovative approach, the relation of lifetime drinking trajectories to cardiovascular risk factors.

The clustering of cardiovascular risk factors characterized by central adiposity, hyperglycemia, atherogenic dyslipidemia, and elevated blood pressure (HBP) (4, 5) has become known as the metabolic syndrome (MetS). MetS dramatically increases cardiovascular morbidity and mortality (6, 7). Prior analyses of the association between MetS and summary measures of lifetime drinking patterns revealed that cardiovascular risk was directly associated with lifetime average drinks per drinking day, a proxy measure of drinking intensity (3). The present study was designed to expand these preliminary observations by examining drinking trajectories among ever-regular drinkers. Specifically, we sought to investigate: 1) whether there are distinct drinking trajectory patterns over the lifetime; 2) drinking pattern characteristics in each trajectory group; and 3) whether different trajectory groups may predict the likelihood of the MetS as a whole and of its individual components.

Subjects and Methods

Data source

Data for the present study were obtained from the Western New York Health Study (WNYHS) conducted between 1996 and 2001 (8). A well-characterized, population-based core sample of controls from two counties in Western New York State was established to support case-control studies investigating a variety of chronic diseases. Participants aged 35–64 were randomly sampled from lists of licensed drivers, and those aged 65–80 were randomly selected from lists of the Health Care Financing Association (8–11). Study purposes and procedures were explained to participants before the survey administration and blood draw. An informed consent was obtained and witnessed. A detailed description of sample selection, procedures, measurement of outcome and covariates has been published elsewhere (3).

Exclusion criteria were self-reported history of cardiovascular disease (prior myocardial infarction, coronary artery bypass graft surgery, angioplasty or diagnosed angina pectoris), history of cancer (n = 117), and a participant with unidentifiable sex (n = 1), yielding a sample size of 3496. In this study we included only those participants who drank at least once a month for a period of at least 6 months during their lifetime, lifetime ever-regular drinkers (n = 2818).

Assessment of lifetime alcohol consumption

Lifetime alcohol use was assessed using the CLDH (2, 3). Lifetime drinking pattern variables (*e.g.* total years of drinking, first and last age of regular drinking, total volume of alcohol consumed, lifetime drinking frequency, lifetime drinking intensity, lifetime frequency of intoxication, lifetime frequency of drinking four or more drinks per drinking day, beverage preference, lifetime percent of drinking without food) have been defined elsewhere (3). Alcohol drinking patterns in the 30 d before the date of interview were assessed using methods comparable to those used in the CLDH; detailed definitions have been published elsewhere (9–11). A standard drink was defined as half an ounce of absolute alcohol, the approximate amount in 12 fluid ounces of beer, 5 fluid ounces of wine, or 1.5 fluid ounces of 80-proof distilled spirits (~12 g alcohol) (12).

Current vs. former drinkers

Current drinkers were those who had at least one drink in the past 30 d. Former drinkers were ever-regular drinkers who did not consume any alcoholic beverage in the past 30 d.

Lifetime drinking trajectories

In this study it is assumed that the population of ever-regular drinkers is composed of a set of relatively distinct groups of individuals with uniquely different trajectories of alcohol consumption over their lifetimes. Lifetime drinking trajectories were identified using PROC TRAJ, a group-based modeling approach for identifying distinctive clusters of individual trajectories within a population and profiling the characteristics of individuals within the clusters (13). The optimal number of groups was guided by the Bayesian Information Criterion. The probability of membership of each individual in each group was given by this procedure. Individuals were classified to groups having the highest predicted probability.

To prepare data for trajectory analysis, total ounces of ethanol for each decade was divided by lifetime average intake, and the ratio was logarithmically transformed. This standardization process is designed to minimize the influence of interindividual lifetime drinking level variability on constructing distinct trajectory groups. Because ethanol consumption in the first, seventh, and eighth decades was quite low and did not vary much in the sample, we only used data for the second through sixth decades (10–59 yr) for the trajectory analysis.

Cardiovascular risk factors and definition of the MetS

Diagnosis of the MetS was based on criteria set by the National Cholesterol Education Program Adult Treatment Panel III (5). Three or more of the following risk determinants warrant a diagnosis of MetS: 1) impaired fasting glucose (IFG) [≥5.6 mmol/liter; new criteria (14)] or diagnosis of diabetes mellitus (DM); 2) raised triglycerides (HTGs) (≥1.7 mmol/liter); 3) low high-density lipoprotein cholesterol (LHDLC) (<1.0 mmol/liter for men and < 1.3 mmol/liter for women); 4) abdominal obesity (ABO) (waist circumference ≥ 102 cm for men and > 88 cm for women); and 5) HBP (systolic/diastolic pressure ≥ 130/85 mm Hg). Whether or not adding medication use for diabetes and hypertension in the definition of relevant components makes any difference in estimates of odds ratios (ORs) was also tested.

Statistical analysis

Data were analyzed using SAS version 9.1 (SAS Institute Inc., Cary, NC). Prevalence and mean (SD) were used to describe sample characteristics and prevalence of MetS and its components by gender and trajectory groups.

General linear modeling was performed to examine differences in drinking patterns between trajectory groups by gender and current drinking status after adjustment for age and race. Variables with skewed distributions were logarithmic transformed.

Multivariate logistic regression was performed to determine whether trajectory group membership is independently associated with the likelihood of MetS and individual MetS components. Interaction analysis revealed no significant sex difference in the association of drinking trajectories with MetS and its components, thus analyses are presented with both sexes combined. Only significant confounders were included in the final regression models. The significance level was set at $P < 0.05$, two-sided.

Results

Trajectory analysis

Trajectory analysis based on standardized ethanol consumption (converted to kilograms) for each decade resulted in two distinct lifetime drinking trajectory groups, one characterized by heavy drinking in the third decade of life (early-peak trajectory) and

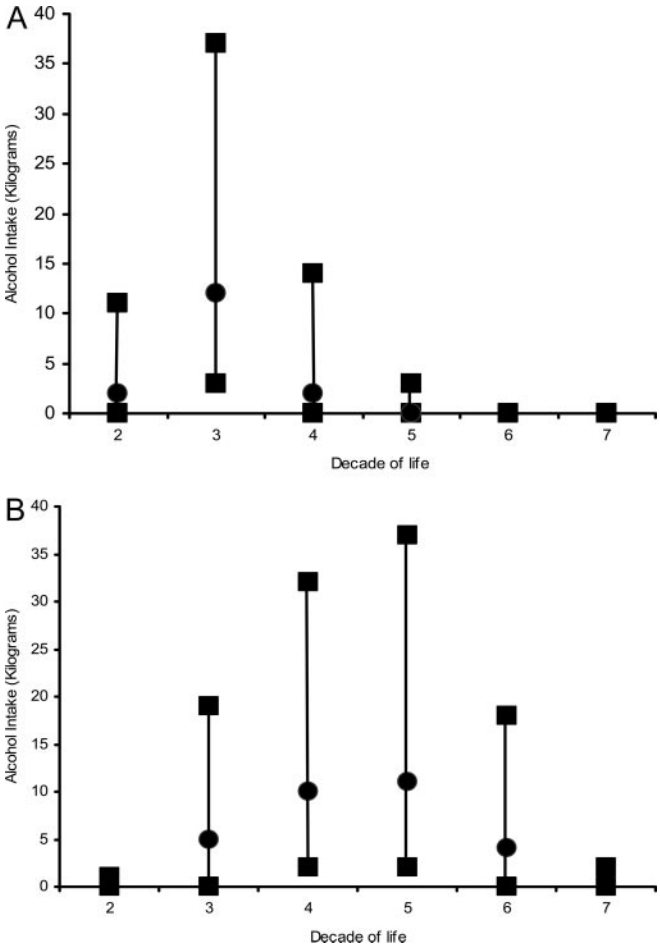

FIG. 1. Intake of kilograms of ethanol per decade of life among women early-peak drinkers (A) and women stable drinkers (B). ●, Median quartile; ■, upper and lower quartiles.

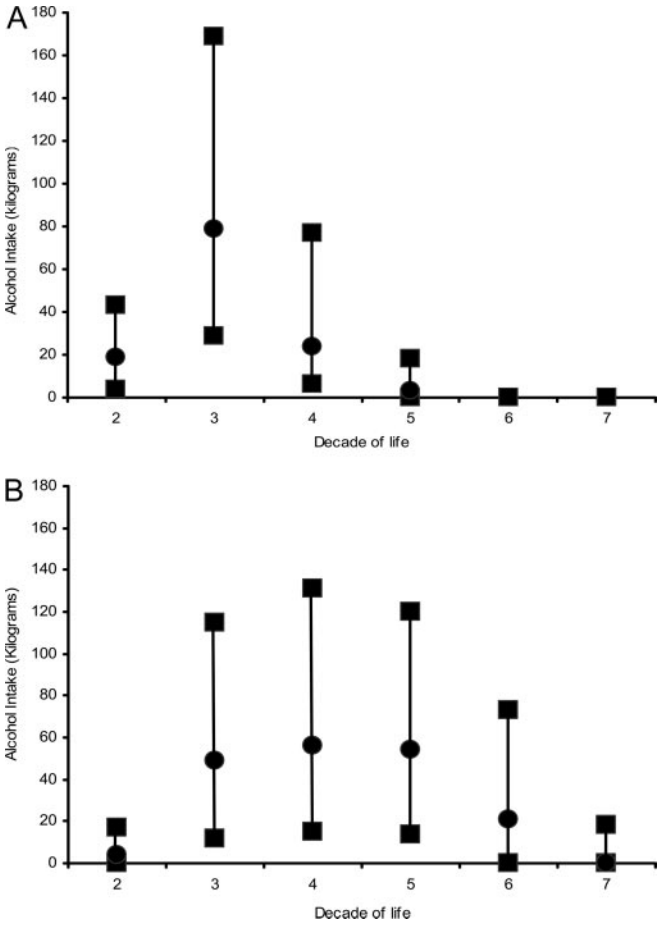

FIG. 2. Intake of kilograms of ethanol per decade of life among men early-peak drinkers (A) and men stable drinkers (B). ●, Median quartile; ■, upper and lower quartiles.

the other by more moderate intakes over a longer period of life (stable trajectory), as shown in Fig. 1 for women and Fig. 2 for men.

Demographic, lifestyle, and metabolic characteristics of the study sample according to drinking trajectory groups are shown in Table 1. Compared with stable trajectory drinkers, early-peak drinkers were 10 yr younger on average, and they were less likely to be drinking at the time of the interview; they were currently consuming a higher percent of total calories from saturated fat and less dietary fiber, and were somewhat more likely to have been physically active during the past week. Male early-peak drinkers smoked far fewer cigarettes in their lifetimes. They were almost as likely to be smoking currently as men with stable drinking trajectories, but they were less likely to have ever started smoking, whereas those with a stable trajectory were more likely to be former smokers. Among women, early-peak drinkers spent fewer hours per week during their lifetime for strenuous physical activity ($P = 0.04$) and had higher body mass index (BMI) than stable drinkers ($P = 0.0005$). There was no appreciable difference between early-peak and stable trajectory drinkers in the distributions of sex, race, family history of coronary heart disease (CHD) and diabetes, and total energy intake. The prevalence of IFG/DM ($P = 0.0002$) and HBP ($P < 0.0001$) was much lower among early-peak drinkers than among stable drinkers, and the prevalence of LHDLC was much higher ($P < 0.0001$). The prevalence of MetS in these two groups was comparable ($P = 0.95$).

Drinking patterns associated with early-peak and stable drinking trajectories differed markedly. Although there are some differences associated with gender and current drinking status (Table 2), early-peak drinkers generally began drinking earlier than stable drinkers, and they drank fewer years, drank less frequently, and consumed less volume of alcohol over their lifetimes, but averaged more drinks per drinking day and had higher rates of episodic heavy drinking and intoxication during their drinking years. Early-peak drinkers were less likely to choose wine but more likely to choose beer or liquor and drink without any food. In the past 30 d, early-peak drinkers consumed less volume of alcohol but drank less frequently.

Multiple logistic regression analyses were then performed to examine the relation between trajectory membership and the prevalence of MetS and its individual components controlling for important confounders (age, race, years of education, family history of CHD and diabetes, smoking status, smoking pack years, recent and lifetime physical activity, energy intake from saturated fat, and dietary fiber intake) (Table 3). Compared with stable drinking trajectories, early-peak trajectories were modestly more likely to be associated with LHDLC, ABO, and MetS

TABLE 1. Comparison of demographic, lifestyle, and metabolic characteristics between two drinking trajectory groups

	Women			Men		
	Stable trajectory	Early peak trajectory	*P* value for difference	Stable trajectory	Early peak trajectory	*P* value for difference
Mean age (SD)	60.0 yr (10.5)	50.7 yr (10.8)	<0.0001	58.2 yr (9.1)	48.3 yr (8.9)	<0.0001
Mean BMI (SD)	27.6 kg/m^2 (5.6)	28.7 kg/m^2 (7.3)	0.0005	28.2 kg/m^2 (4.5)	28.7 kg/m^2 (5.0)	0.06
Median lifetime no. of cigarette packs smoked (lower and upper quartiles)[a]	256 (0, 6,661)	284 (0, 5,931)	0.41	3285 (0, 10,768)	694 (0, 7,665)	<0.0001
% White	93.0	91.8	0.38	91.7	94.2	0.11
Years of education ≤12 (%)	46.4	42.5	0.12	36.1	30.0	0.033
Smoking status (%)			0.84			0.01
Never smoker	45.0	44.8		34.3	41.3	
Former smoker	38.1	37.2		48.4	39.8	
Current smoker	16.9	17.9		17.3	18.9	
Current drinker (%)	76.1	57.1	<0.0001	81.0	71.7	0.0002
Family history of CHD or diabetes (%)	79.2	79.8	0.76	68.2	70.2	0.48
Average h/wk for lifetime strenuous physical activity <5 h/wk (%)	42.1	47.3	0.039	36.6	37.1	0.85
Total metabolic equivalents (METs) <240 in the past week (%)	47.4	42.9	0.07	36.4	30.4	0.036
Total energy intake ≥1600 kcal/d (%)	33.1	37.1	0.10	67.2	68.4	0.66
Energy intake from saturated fat ≥10% (%)	77.1	82.3	0.011	81.2	90.3	<0.0001
Dietary fiber intake <7 g/1000 kcal (%)	38.2	45.2	0.005	49.6	64.7	<0.0001
IFG/DM (%)	37.1	30.2	0.0049	56.1	49.1	0.02
HTGs (%)	34.9	30.3	0.06	39.6	36.5	0.29
LHDLC (%)	26.7	39.1	<0.0001	30.9	50.0	<0.0001
ABO (%)	38.4	41.3	0.24	32.0	35.3	0.25
HBP (%)	34.1	23.6	<0.0001	44.2	35.6	0.0034
MetS (%)	26.0	25.1	0.76	34.7	34.7	0.99

[a] Median and interquartile ranges were shown for smoking pack years; the group differences were tested by the two-sided nonparametric Wilcoxon test.

overall. Most of these associations persisted when the analysis was restricted to current drinkers only. However, the previous modest association between trajectory membership and overall MetS lost significance for current drinkers. There were no significant associations between trajectory membership and other components of MetS, including IFG/DM, HTGs, and HBP. Further adjustment for current drinking pattern in the past 30 d (*e.g.* total drinks, total drinking days, drinks per drinking day, beverage preference) did not change the results significantly (data not shown). The association between trajectory membership and HDLC was less pronounced but remained significant when overweight was controlled in the model (data not shown), suggesting that the trajectory-HDL association can be partially explained by body adiposity.

Adding medication use for diabetes and hypertension to the definition of relevant MetS components made virtually no difference in estimates of the ORs.

Discussion

This study revealed two distinct lifetime drinking trajectories among ever-regular drinkers, one characterized by heavy drinking in early adulthood followed by a sharply reduced alcohol intake and the other by more moderate intakes over a longer period of life. There was no significant difference in the unadjusted prevalence of MetS related to lifetime drinking trajectories; yet in multiple regression analyses, early-peak drinkers were associated with slightly higher odds of MetS than stable drinkers. The higher odds of MetS associated with early-peak drinkers can be explained by higher odds of LHDLC, obesity, and ABO.

Comparison with other studies

Several aspects of drinking pattern associated with early-peak drinking trajectories, *i.e.* higher drinking intensity, lower drinking frequency, and drinking more often without any food, have been found in previous analyses from the WNYHS to be associated with higher cardiovascular risk (3, 8–10). For example, Fan *et al.* (3) found a positive relation between lifetime average drinking intensity and the prevalence of MetS. Stranges *et al.* (10) found that drinking most of the time without food during the 30 d before interview was associated with a higher risk of hypertension. Dorn *et al.* (9) found that drinks per drinking day in the 30 d before interview was positively related, and drinking frequency was negatively related to central adiposity, as measured by abdominal height. Finally, Trevisan *et al.* (8) reported

TABLE 2. Comparison of lifetime and current drinking pattern characteristics according to drinking trajectory and drinking status among female (n = 1668) and male (n = 1150) ever-regular drinkers

Characteristics	Former drinkers			Current drinkers		
	Stable trajectories	Early peak trajectories	*P* value	Stable trajectories	Early peak trajectories	*P* value
Women (n)	241	283		768	376	
Age first drinking regularly	24.3 (0.5)	19.2 (0.5)	<0.0001	24.4 (0.7)	18.4 (0.7)	<0.0001
Total years of drinking[a]	27.1 (1.1)	12.5 (1.0)	<0.0001	30.3 (0.9)	22.6 (1.0)	<0.0001
Last age of regular drinking[a]	51.5 (0.9)	32.3 (0.8)	<0.0001	55.2 (0.6)	44.3 (0.7)	<0.0001
Lifetime total drinks[a,b]	2,164 (1,550, 3,020)	684 (510, 919)	<0.0001	2,892 (2,331, 3,588)	1,509 (1,170, 1,947)	<0.0001
Lifetime drinking days[a,b]	906 (688, 1192)	286 (221, 369)	<0.0001	1,555 (1,278, 1,892)	638 (514, 792)	<0.0001
Lifetime drinking intensity	2.4 (2.2, 2.7)	2.6 (2.4, 2.9)	0.26	1.9 (1.8, 2.0)	2.6 (2.4, 2.8)	<0.0001
Lifetime frequency of intoxication[a]	260 (26)	105 (23)	<0.0001	88.8 (11.5)	99.8 (13.0)	0.31
Frequency of intoxication per drinking year	10.7 (1.3)	9.1 (1.2)	0.26	2.4 (0.5)	5.3 (0.5)	<0.0001
Lifetime frequency of drinking 4+[a,b]	23.5 (13.5, 40.7)	9.4 (5.5, 15.6)	0.0049	8.0 (5.1, 12.4)	19.1 (12.6, 28.7)	<0.0001
Frequency of drinking 4+ per drinking year[b]	5.2 (3.6, 7.5)	3.0 (2.0, 4.4)	0.017	2.0 (1.4, 2.7)	3.6 (2.6, 4.8)	<0.0001
% drinking without food	45.2 (4.5)	58.8 (4.1)	0.006	37.5 (3.2)	55.7 (3.5)	<0.0001
% alcohol from wine	27.0 (3.3)	23.7 (3.1)	0.36	33.6 (2.6)	25.7 (2.7)	0.0004
% alcohol from beer or liquor	73.6 (3.3)	74.6 (3.0)	0.77	69.5 (2.5)	77.6 (2.8)	0.0001
Among current drinkers[c]						
Total no. of drinks				8.2 (7.0, 9.6)	4.3 (3.5, 5.2)	<0.0001
Drinking days in a month				9.0 (0.6)	3.2 (0.7)	<0.0001
Drinks per drinking day[b]				1.8 (1.7, 2.0)	1.9 (1.7, 2.0)	0.56
No. of times getting drunk[b]				0.04 (0.02, 0.07)	0.04 (0.01, 0.07)	0.9
Men (n)	129	133		551	337	
Age first drinking regularly	20.9 (0.6)	18.3 (0.6)	0.0001	19.8 (0.5)	17.6 (0.5)	<0.0001
Total years of drinking[a]	27.3 (1.3)	19.3 (1.3)	<0.0001	34.7 (0.7)	31.9 (0.8)	<0.0001
Last age of regular drinking[a]	48.3 (1.1)	38.1 (1.1)	<0.0001	54.9 (0.5)	51.0 (0.5)	<0.0001
Lifetime total drinks[a,b]	5,709 (3,566, 9,139)	3,164 (2,016, 4,967)	0.047	9,798 (7,820, 12,275)	7,382 (5,755, 9,469)	0.009
Lifetime drinking days[a,b]	1,685 (1,184, 2,398)	870 (611, 1,239)	0.0045	3,713 (3,112, 4,430)	2,207 (1,814, 2,686)	<0.0001
Lifetime drinking intensity[b]	3.7 (3.2, 4.3)	4.0 (3.4, 4.6)	0.46	2.7 (2.4, 3.0)	3.7 (3.4, 4.1)	<0.0001
Lifetime frequency of intoxication[a]	338 (42.8)	263 (43)	0.16	303 (26)	289 (28)	0.90
Frequency of intoxication per drinking year[a,b]	11.8 (1.8)	14.6 (1.8)	0.19	7.5 (0.8)	11.4 (0.9)	<0.0001
Lifetime frequency of drinking 4+[a,b]	186 (86, 400)	139 (64, 299)	0.54	205 (121, 349)	383 (216, 676)	0.01
Frequency of drinking 4+ per drinking year[b]	20.8 (12.3, 34.5)	17.5 (10.6, 28.7)	0.61	15.8 (11.3, 22.0)	21.9 (15.4, 30.9)	0.035
% drinking without food	51.8 (4.4)	58.0 (4.6)	0.24	49.6 (2.7)	56.8 (3.0)	0.0013
% alcohol from wine	8.8 (2.1)	12.9 (2.1)	0.09	20.2 (1.8)	15.0 (2.0)	0.0005
% alcohol from beer or liquor	90.3 (1.9)	86.9 (2.0)	0.13	81.4 (1.7)	86.9 (1.9)	0.0001
Among current drinkers[c]						
Total no. of drinks				16.1 (13.3, 19.5)	7.4 (5.9, 9.3)	<0.0001
Drinking days in a month				11.7 (0.7)	4.8 (0.8)	<0.0001
Drinks per drinking day[b]				2.9 (2.6, 3.2)	2.6 (2.3, 2.9)	0.03
No. of times getting drunk[b]				0.14 (0.08, 0.20)	0.13 (0.06, 0.20)	0.60

[a] Controlled for age and race. All other variables were controlled for race only.

[b] For logarithmic-transformed variables, mean and 95% CIs in original scales are shown.

[c] Current drinking pattern was ascertained for drinking in the past 30 d.

TABLE 3. Multivariate-adjusted ORs for metabolic syndrome and its components in two trajectory groups, early peak *vs.* stable trajectory group

	Ever-regular drinkers (n = 2818) not controlled for current drinking status		Ever-regular drinkers (n = 2818) controlled for current drinking status		Current drinkers only (n = 2032)	
	ORs	95% CI	ORs	95% CI	ORs	95% CI
IFG/DM	1.04	0.86, 1.26	1.02	0.84, 1.24	1.06	0.84, 1.33
IFG/DM or taking diabetes medication	1.04	0.86, 1.25	1.02	0.84, 1.23	1.05	0.84, 1.33
HTGs	0.94	0.78, 1.14	1.04	0.85, 1.25	1.07	0.85, 1.37
LHDLC	1.62	1.27, 2.08	1.41	1.08, 1.84	1.49	1.07, 2.07
ABO	1.48	1.23, 1.78	1.41	1.16, 1.72	1.64	1.28, 2.09
Overweight (BMI $\geq$ 25 kg/m^2)	1.32	1.10, 1.60	1.24	1.02, 1.50	1.25	1.00, 1.57
HBP	1.09	0.90, 1.33	1.12	0.91, 1.36	1.01	0.79, 1.29
HBP or using BP-lowering medication	1.05	0.87, 1.27	1.03	0.85, 1.25	0.90	0.71, 1.14
MetS	1.31	1.00, 1.71	1.32	0.99, 1.77	1.42	0.99, 2.04

The parameters were ORs (95% CI) adjusted for age, sex, family history of CHD or diabetes, years of education (>12 *vs.* $\leq$ 12 yr), smoking status, smoking pack years (quartiles), lifetime and current physical activity (quartiles), energy intake from saturated fat, and dietary fiber intake (quartiles).

that drinking mainly without food during the prior 12–24 months before interview was associated with an increased risk of myocardial infarction.

Previous studies on cardiovascular outcomes comparing different drinking categories without a clear distinction between former drinkers and lifetime abstainers have been criticized on the basis that former drinkers may have stopped drinking because of health problems (15, 16). The WNYHS indicated that early-peak drinkers were less likely to be current drinkers; early-peak drinkers were more likely to have quit drinking because of health problems (data not shown). This analysis showed that early-peak drinkers had mostly higher odds of MetS. There are several possible explanations. First, early-peak drinking trajectories may be associated with unhealthy drinking patterns, as reported in this analysis (*e.g.* higher drinking intensity, drinking without food, higher likelihood of intoxication, and episodic heavy drinking), and the adverse health effects of early unhealthy drinking patterns (*e.g.* obesity) (17) were carried over to later life. Second, early-peak drinkers may differ from stable drinkers in other lifestyle habits that may be detrimental to their cardiometabolic health but were not properly controlled for in our model. However, the modest trajectory-MetS association still held when the analysis was restricted to current drinkers, although the lower boundary of the 95% confidence interval (CI) of adjusted OR changed from 1.00 to 0.99. The modest association may be partly driven by the fact that some adverse effects (*e.g.* increasing blood pressure) (18) offset potential "benefits" of alcohol consumption (*e.g.* increasing HDLC), especially among current drinkers, thus the overall association with MetS was somewhat diluted.

Retrospective approach for drinking pattern ascertainment

A potential limitation of these findings concerns the validity of retrospective measures of lifetime drinking patterns. Prospective ascertainment of alcohol intake poses fewer problems concerning memory than retrospective ascertainment. However, despite these positive attributes of prospective studies, there are also drawbacks. Brody and Mills (19) noted that heavier drinkers may be underrepresented in prospective studies of ensured and general populations. Individuals applying for insurance may minimize reports of drinking on screening questionnaires, and heavier drinkers in the general population may not have the high motivation required to participate successfully in longitudinal studies of health conducted over a number of years. Another potential problem in prospective studies is that drinking patterns assessed at baseline may change over the course of the follow-up period (20). This can be addressed by including periodic assessments, but this adds to the cost of the study, decreasing the likelihood of detailed alcohol assessments. Furthermore, such assessments may induce reactive changes in drinking patterns. By contrast, retrospective assessments of drinking habits may provide several advantages. For example, recall validity studies have reported high correlations between alcohol intakes reported in the distant past and recalled after many years (21–23). Moreover, several studies have found that heavy drinkers report higher alcohol intakes retrospectively than prospectively (24–26), which suggests that people are more comfortable reporting past heavy drinking than current heavy drinking. Thus, retrospectively ascertained drinking histories may have advantages that have not been fully appreciated and may outweigh or offset memory problems. The CLDH used to assess lifetime drinking patterns in this study uses a number of cognitive techniques to maximize memory and has demonstrated high reliability in test-retest studies (2). In addition to its efficiency and economy, a further strength of the retrospective approach is that it allows investigation of selected factors such as drinking pattern, beverage type, and alcohol drinking history in the etiology of alcohol-related health conditions.

Potential limitation of the study

Despite potential merits of a retrospective approach, a major limitation of this study remains that the alcohol consumption data used to derive the lifetime trajectories were based on self-report by questionnaire. In addition, persons with heavy drinking history are less likely to be included due to death or illness.

In this report, early-peak drinkers were on average 10 yr younger than stable trajectory drinkers. This large discrepancy makes the trajectory-MetS association more likely to be contaminated by residual confounding of age. Several possible alternative explanations may contribute to the large age gap between the trajectory groups. It may reflect a cohort effect whereby younger individuals tend to have less healthy drinking patterns at younger ages than older individuals, at least in this population. Another explanation may reside in recall bias whereby older individuals tend to underestimate their alcohol consumption at a younger age, but we were unable to verify this. Finally, individuals who begin drinking heavily at a young age may be less likely than moderate drinkers to survive to participate in studies of cardiovascular disease at older ages (27). More studies of lifetime drinking trajectories are needed to clarify some of these questions. Despite being 10 yr younger in age, the early-peak drinkers still manifested a modestly higher likelihood of MetS in adjusted analysis. However, whether the early-peak drinker would also be related to a worse mortality outcome is unknown and needs to be investigated further in a cohort study with a long enough follow-up period.

Public health implications

The possibility that early binge drinking has long-term negative consequences for cardiovascular health is especially significant in view of current trends in alcohol use and episodic heavy drinking (or binge drinking). A report from a U.S. multistate survey indicates that binge drinking in late adolescence and early adulthood, such as that associated with the early-peak lifetime drinking trajectories observed here, may be increasing, especially among 18- to 20-yr-olds (28). Binge drinking has been associated with an increased risk of cardiovascular and other negative health outcomes (15, 16). Others have reported a higher risk of alcoholism associated with early onset of drinking (29). Early initiation of alcohol drinking is associated with unhealthy drinking patterns, *i.e.* more frequently having four or more drinks per drinking day, higher rates of intoxication during drinking years, and drinking without food (3). This report provides additional support for public health messages discouraging early initiation of alcohol drinking in addition to adverse psychosocial, behavioral, and other long-term health outcomes (27, 30, 31).

This trajectory analysis based on lifetime alcohol consumption data collected using the CLDH has important implications. First, typical trajectories representing intraindividual changes of drinking patterns and variability over time can be obtained; second, different trajectory groups manifest significant interindividual differences in drinking pattern characteristics. Finally, trajectory membership is associated with a differential likelihood of health outcomes. Findings suggest that the effect of alcohol consumption on cardiovascular risk may be chronic and cumulative, and that variability in drinking habits over the lifetime may play an important role. More research on the relation of lifetime drinking trajectories to chronic disease morbidity and mortality is needed to guide the formulation of recommendations regarding alcohol consumption and health.

Acknowledgments

We thank all the staff that participated in the design, management, data collection, and analysis of the Western New York Health Study.

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This study was supported by Grants P50-AA09802 and R21-AA013597 from the National Institute on Alcohol Abuse and Alcoholism (to M.R., Principal Investigator).

A.Z.F. initiated the study, conducted the statistical analysis, and wrote up the manuscript. M.R. provided critical input on study design and took responsibility for the integrity of the data and accuracy of the data analysis. S.S. and J.D. offered important comments on results interpretation. M.T. had full access to all of the data in the study and oversaw the whole study.

Disclosure Statement: The authors have nothing to declare.

References

1. **Gruenewald PJ, Russell M, Light J, Lipton R, Searles J, Johnson F, Trevisan M, Freudenheim J, Muti P, Carosella AM, Nochajski TH** 2002 One drink to a lifetime of drinking: temporal structures of drinking patterns. Alcohol Clin Exp Res 26:916–925
2. **Russell M, Marshall JR, Trevisan M, Freudenheim JL, Chan AW, Markovic N, Vana JE, Priore RL** 1997 Test-retest reliability of the cognitive lifetime drinking history. Am J Epidemiol 146:975–981
3. **Fan AZ, Russell M, Dorn J, Freudenheim JL, Nochajski T, Hovey K, Trevisan M** 2006 Lifetime alcohol drinking pattern is related to the prevalence of metabolic syndrome. The Western New York Health Study (WNYHS). Eur J Epidemiol 21:129–138
4. **Reaven G** 2002 Metabolic syndrome: pathophysiology and implications for management of cardiovascular disease. Circulation 106:286–288
5. **The Expert Panel** 2002 Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. Circulation 106:3143–3421
6. **Isomaa B, Almgren P, Tuomi T, Forsen B, Lahti K, Nissen M, Taskinen MR, Groop L** 2001 Cardiovascular morbidity and mortality associated with the metabolic syndrome. Diabetes Care 24:683–689
7. **Lakka HM, Laaksonen DE, Lakka TA, Niskanen LK, Kumpusalo E, Tuomilehto J, Salonen JT** 2002 The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men. JAMA 288:2709–2716
8. **Trevisan M, Dorn J, Falkner K, Russell M, Ram M, Muti P, Freudenheim JL, Nochajaski T, Hovey K** 2004 Drinking pattern and risk of non-fatal myocardial infarction: a population-based case-control study. Addiction 99:313–322
9. **Dorn JM, Hovey K, Muti P, Freudenheim JL, Russell M, Nochajski TH, Trevisan M** 2003 Alcohol drinking patterns differentially affect central adiposity as measured by abdominal height in women and men. J Nutr 133:2655–2662
10. **Stranges S, Wu T, Dorn JM, Freudenheim JL, Muti P, Farinaro E, Russell M, Nochajski TH, Trevisan M** 2004 Relationship of alcohol drinking pattern to risk of hypertension: a population-based study. Hypertension 44:813–819
11. **Stranges S, Freudenheim JL, Muti P, Farinaro E, Russell M, Nochajski TH, Trevisan M** 2004 Differential effects of alcohol drinking pattern on liver enzymes in men and women. Alcohol Clin Exp Res 28:949–956
12. **Dufour MC** 1999 What is moderate drinking? Defining "drinks" and drinking levels. Alcohol Res Health 23:5–14
13. **Jones BL, Nagin DS, Roeder K** 2001 A SAS procedure based on mixture models for estimating developmental trajectories. Sociol Methods Res 29:374–393
14. **American Diabetes Association** 2004 Diagnosis and classification of diabetes mellitus. Diabetes Care 27(Suppl 1):S5–S10
15. **Rehm J, Gmel G, Sempos CT, Trevisan M** 2003 Alcohol-related morbidity and mortality. Alcohol Res Health 27:39–51
16. **Stranges S, Notaro J, Freudenheim JL, Calogero RM, Muti P, Farinaro E, Russell M, Nochajski TH, Trevisan M** 2006 Alcohol drinking pattern and subjective health in a population-based study. Addiction 101:1265–1276
17. **Arif AA, Rohrer JE** 2005 Patterns of alcohol drinking and its association with obesity: data from the Third National Health and Nutrition Examination Survey, 1988–1994. BMC Public Health 5:126

18. **Klatsky AL** 1996 Alcohol and hypertension. Clin Chim Acta 246:91–105
19. **Brody JA, Mills GS** 1978 On considering alcohol as a risk factor in specific diseases. Am J Epidemiol 107:462–466
20. **Emberson JR, Shaper AG, Wannamethee SG, Morris RW, Whincup PH** 2005 Alcohol intake in middle age and risk of cardiovascular disease and mortality: accounting for intake variation over time. Am J Epidemiol 161:856–863
21. **Dwyer JT, Gardner J, Halvorsen K, Krall EA, Cohen A, Valadian I** 1989 Memory of food intake in the distant past. Am J Epidemiol 130:1033–1046
22. **Thompson FE, Lamphiear DE, Metzner HL, Hawthorne VM, Oh MS** 1987 Reproducibility of reports of frequency of food use in the Tecumseh Diet Methodology Study. Am J Epidemiol 125:658–671
23. **The Atherosclerosis Risk in Communities (ARIC) Study** 1989 design and objectives. The ARIC investigators. Am J Epidemiol 129:687–702
24. **Czarnecki DM, Russell M, Cooper ML, Salter D** 1990 Five-year reliability of self-reported alcohol consumption. J Stud Alcohol 51:68–76
25. **Ernhart CB, Morrow-Tlucak M, Sokol RJ, Martier S** 1988 Underreporting of alcohol use in pregnancy. Alcohol Clin Exp Res 12:506–511
26. **Simpura J, Poikolainen K** 1983 Accuracy of retrospective measurement of individual alcohol consumption in men; a reinterview after 18 years. J Stud Alcohol 44:911–917
27. **Mori M, Shirasaka T** 1995 Early onset of drinking and mortality among male alcoholics. A result of a 10-year prospective follow-up study in Hokkaido, Japan. Arukoru Kenkyuto Yakubutsu Ison 30:426–434
28. **Serdula MK, Brewer RD, Gillespie C, Denny CH, Mokdad A** 2004 Trends in alcohol use and binge drinking, 1985–1999: results of a multi-state survey. Am J Prev Med 26:294–298
29. **Grant BF, Stinson FS, Harford TC** 2001 Age at onset of alcohol use and DSM-IV alcohol abuse and dependence: a 12-year follow-up. J Subst Abuse 13:493–504
30. **Chou SP, Pickering RP** 1992 Early onset of drinking as a risk factor for lifetime alcohol-related problems. Br J Addict 87:1199–1204
31. **Ellickson PL, Tucker JS, Klein DJ** 2003 Ten-year prospective study of public health problems associated with early drinking. Pediatrics 111:949–955