Alcohol Consumption and Ambulatory Blood Pressure: A Community-Based Study in an Elderly Cohort

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BACKGROUND

Although heavy alcohol consumption is associated with hypertension, the impact of lighter consumption on blood pressure (BP) is controversial. The protective effect of light alcohol consumption on cardiovascular disease described in previous studies could be, in part, mediated by effects of alcohol on BP. However, only a few studies investigating the association between alcohol and BP included elderly subjects, despite their higher risk of hypertension sequelae. Accordingly, we evaluated the relationship between alcohol consumption and 24-hour ambulatory BP in a community-based elderly cohort.

METHODS

Among the participants in the Cardiac Abnormalities and Brain Lesion study, 553 subjects (mean age = 70.6±9.6 years) who underwent 24-hour ambulatory BP monitoring were examined. Alcohol consumption was categorized as (i) none (reference; <1 drink/month); (ii) very light consumption (1 drink/month to 1 drink/week); (iii) light consumption (2 drinks/week to 1 drink/day); (iv) moderate-to-heavy consumption (>1 drink/day). Former drinkers were excluded.

RESULTS

After adjustment for relevant covariables, mean values of daytime diastolic BP (DBP), nighttime DBP, and 24-hour DBP were significantly higher in moderate-to-heavy drinkers than in the reference group, whereas systolic BP parameters were not significantly different across consumption groups. Daytime systolic BP and DBP variability (SD of the measurements) were significantly lower in very light drinkers than in the reference group, independent of potential confounders.

CONCLUSIONS

Moderate-to-heavy alcohol consumption was associated with higher DBP values. Very light alcohol consumption was associated with reduced daytime BP variability. The latter association may contribute to the known beneficial cardiovascular effects of light alcohol consumption.

Keywords: alcohol; ambulatory blood pressure monitoring; blood pressure; blood pressure variability; hypertension.

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There is general consensus on the detrimental effects of heavy alcohol consumption on blood pressure (BP).[1,2] In fact, it is estimated that at least 5%–7% of hypertension cases are secondary to heavy consumption.[3] Large prospective studies have shown that the risk of developing hypertension increases with increasing alcohol consumption.[4,5] Intervention trials confirmed the relation between alcohol consumption and BP, showing that a reduction in alcohol intake was associated with a substantial decrease in BP values,[6–8] even when measured by 24-hour ambulatory BP monitoring (ABPM).[9]

Although heavy alcohol consumption has been clearly associated with increased BP, the effects of light-to-moderate alcohol consumption on BP are still controversial, with conflicting results obtained in different populations.[10–12] Several studies have strongly suggested a favorable impact of moderate consumption on incident or prevalent coronary artery disease as well as incident stroke or heart failure.[13–15] The mechanisms of this beneficial effect on cardiovascular outcomes are still poorly understood. The established association between alcohol and higher high-density lipoprotein serum levels may be of importance through a protective effect on the development of atherosclerosis,[16] but other mechanisms may exist that are not mediated by an anti-ischemic effect.[15] We recently reported on the protective effect of light-to-moderate alcohol consumption on aortic atherosclerosis[17] and that BP variability, obtained from ABPM, is independently associated with severe aortic atherosclerosis.[18] The study of the effects of alcohol on BP may therefore help elucidate an alternate mechanism to explain

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the beneficial effects of alcohol on atherosclerosis and cardiovascular outcomes. In particular, the possibility that the relation between alcohol consumption and BP may not follow a linear, dose–response pattern, but differ instead on the basis of the amount consumed (i.e., beneficial effect at lower doses, deleterious effect at higher ones), is intriguing because it would mimic the J-shaped relation that is known to exist between alcohol consumption and CV events.[19,20]

The association between alcohol and BP levels has been studied in young and middle-aged populations.[21,22] Little is known about this relation in the elderly, who represent the population subgroup most likely to suffer from sequelae of hypertension such as heart failure and coronary artery disease or stroke. Furthermore, the protective effect of light-to-moderate drinking on cardiovascular diseases appears to be limited to older subjects[23] or those with worse cardiovascular risk profile.[24] The aim of this study was therefore to evaluate the association between alcohol consumption and 24-hour BP and its variability in a cross-sectional study in a community-based elderly population with high cardiovascular risk profile.

METHODS

Study population

The study population was derived from the Cardiac Abnormalities and Brain Lesion (CABL) study, a National Institute of Neurological Disorders and Stroke–sponsored study. The CABL participants were derived from the stroke-free cohort of the Northern Manhattan Study (NOMAS), a community-based prospective study designed to evaluate the risk factors for stroke. Details of enrollment have been previously published.[25] Among the 1,004 subjects enrolled in CABL, 855 agreed to undergo ABPM and completed the 24-hour evaluation. Reliable data regarding ABPM or alcohol consumption during the CABL follow-up was available in 706 participants. Among those, 153 participants were former drinkers and were excluded from main analysis, leaving a study population of 553 subjects. Informed consent was obtained by all participants. The study was approved by the Institutional Review Board of Columbia University Medical Center.

Risk factors assessments

Baseline demographics and risk factors were collected through standardized interviews. Office systolic BP (SBP) and diastolic BP (DBP) (mean of 2 measures in sitting position) were measured by trained research assistants, after a period of rest with a mercury sphygmomanometer. Hypertension was defined as office SBP ≥140 mm Hg or DBP ≥90 mm Hg, or self-report of hypertension, or antihypertensive medications use. Blood samples were obtained at enrollment. Diabetes mellitus was defined as fasting blood glucose ≥126 mg/dl, or self-reported diabetes, or diabetes medications use. Hypercholesterolemia was defined as total serum cholesterol >240 mg/dl, or self-reports of hypercholesterolemia, or use of lipid-lowering treatment. Race/ethnicity was determined by self-report using a questionnaire modeled after the US Census Bureau and classified in 3 groups: non-Hispanic whites, non-Hispanic blacks, and Hispanics. Level of education (high school graduation and higher), smoking history, body mass index, and data on antihypertensive therapies (beta-blockers, diuretics, vasodilators, calcium antagonists, angiotensin-converting inhibitors) were collected. Obesity was defined as a body mass index ≥30 kg/m^2.

Adequate social support was defined as a positive response to the question, "Do you have someone you can trust and confide in?"; social isolation was defined as a negative response to the question, "Do you see your relatives and friends as often as you want to?"

Assessment of alcohol consumption

Alcohol consumption was assessed by research assistants during an in-person interview at enrollment. The reliability and validity of this assessment, adapted from the National Cancer Institute Food Frequency questionnaire, has been previously validated in this population.[25] Each subject was followed-up by phone yearly, and the consumption was again assessed at these interviews (mean follow-up time = 5.3 ± 3.5 years). The frequency and the type of beverage over the previous year were recorded. The predefined response allowed 9 possibilities ranging from never drinking to ≥7 drinks/day. A standard drink was defined as 120 ml or 4 ounces of wine, 360 ml or 12 ounces of beer, or 45 ml or 1.5 ounces of liquor. The frequency of consumption was evaluated by the average number of drinks consumed per day and week. Participants were then categorized into 4 groups: (i) reference group: abstainers and very infrequent drinkers (<1 drink/month); (ii) very light consumption: 1 drink/month to 1 drink/week; (iii) light consumption: 2 drinks/week to 1 drink/day; and (iv) moderate-to-heavy consumption: >1 drink/day. The alcohol consumption used in the analysis was derived from the initial report at baseline enrollment and had to be confirmed as unchanged throughout the follow-up until the nearest point to the performance of ABPM. Participants were excluded from the analysis if (i) they could not provide an estimation of their consumption, (ii) they reported a change in consumption between different interviews during follow-up (to avoid the presence of "sick quitters" from drinking because of an incident health condition), or (iii) if they were former drinkers (to avoid residual interference of past drinking on BP).

Ambulatory blood pressure monitoring

ABPM was performed using a BP monitor (SpaceLabs Ultralite 90217; Williams Medical Supplies, Rhymney, UK), previously validated by the Association for the Advancement of Medical Instrumentation and calibrated against a reference mercury sphygmomanometer. ABPM methods have been previously published.[18] Briefly, patients were asked to keep their usual daily activities during monitoring and to note them in a diary together with the waking and the sleeping time. ABPM was performed with a BP cuff appropriately sized on the nondominant arm. BP was recorded automatically every 15 minutes during waking hours and every 30 minutes during sleeping hours for 24 hours. The following

variables were obtained: (i) 24-hour SBP and DBP (average of the 24 hours); (ii) daytime SBP and DBP (average of awake measurements); (iii) nighttime SBP and DBP (average of sleeping measurements); (iv) daytime and nighttime variability (SD of BP readings during day and night); and (v) night-to-day-ratio (night-time SBP/daytime SBP). Mean heart rates during waking and sleeping hours were also recorded.

Statistical analysis

Continuous variables were expressed as means ± SDs, and categorical data were expressed as proportions. For continuous variables, pairwise comparisons between two groups were assessed by *t* test, and overall differences among ≥3 groups were examined by 1-way analysis of variance. For categorical variables, comparison among groups was carried out by χ^2 test. Linear models were used to evaluate the association between different levels of consumption and BP variables in univariable and multivariable analyses. Covariables known to affect BP or the association between alcohol and BP (age, sex, race-ethnicity, education, body mass index, diabetes, smoking history, antihypertensive medication, social support, and social isolation) were considered and included in the multivariable models when significantly different among the alcohol consumption groups. Adjusted means were obtained for each ABPM variable in the 4 consumption groups. All tests were 2-sided, and the level of significance was set at $P = 0.05$. All statistical analyses were performed using SAS software version 9.2 (SAS institute, Cary, NC).

RESULTS

Study population

The characteristics of the study sample by consumption categories are summarized in Table 1. The mean age was 70.6 ± 9.6 years. In the reference group, 104 persons were occasional drinkers (<1 drink/month), and 138 persons were abstainers. Significant differences in demographics and cardiovascular risk factors were present among the drinking categories (Table 1).

BP and alcohol consumption

As shown in Table 2, office SBP, daytime SBP, nighttime SBP, and 24-hour SBP were significantly lower in very light alcohol drinkers than in the reference group (all $P < 0.05$). Office SBP was significantly lower in light drinkers compared with the reference group ($P < 0.01$). In multivariable analysis, differences in SBP values among the groups were no longer statistically significant. In Table 3, diastolic ABPM variables by alcohol consumption groups are shown. In univariable analysis, daytime DBP and 24-hour DBP were higher in light and moderate-to-heavy

Table 1. Characteristics of the study population (n = 553) in different alcohol consumption groups

Characteristic	Reference (n = 242)	Very light consumption (n = 138)	Light consumption (n = 120)	Moderate-to-heavy consumption (n = 53)	*P* value[a]
Age, y, mean ± SD	72.2 ± 9.4	69.4 ± 9.3**	69.2 ± 9.9**	69.3 ± 9.4*	<0.01
Men, no. (%)	33 (13.6)	46 (33.3)**	69 (57.5)**	39 (73.6)**	<0.01
Race/ethnicity, no. (%)					
Black	35 (14.5)	24 (17.4)*	16 (13.3)**	10 (18.9)**	
Hispanic	189 (78.1)	93 (67.4)	75 (62.5)	29 (54.7)	
White	18 (7.4)	21 (15.2)	29 (24.2)	14 (26.4)	<0.01
Higher education, no. (%)	90 (37.2)	68 (49.3)*	70 (58.3)**	34 (64.2)**	<0.01
BMI, kg/m^2, mean (SD)	29.1 (5.1)	27.7 (4.7)*	28.3 (4.7)	26.9 (3.9)**	<0.01
Obesity, no. (%)	97 (40.3)	38 (27.5)*	37 (30.8)	10 (18.9)**	<0.01
Diabetes, no. (%)	86 (35.5)	34 (24.6)*	29 (24.2)*	12 (22.6)	<0.05
Hypertension, no. (%)	202 (83.5)	98 (71.0)**	85 (70.8)**	41 (77.4)	<0.05
Cigarette smoking, no. (%)	91 (37.6)	68 (49.3)*	68 (56.7)**	40 (75.5)**	<0. 01
Hypercholesterolemia, no. (%)	161 (66.8)	79 (57.3)	79 (57.3)	27 (50.9)*	0.07
Serum HDL, mg/dl, mean (SD)	53.0 (15.7)	53.3 (16.8)	52.8 (16.3)	55.7 (19.0)	0.72
Antihypertensive medication, no. (%)	191 (79.6)	91 (68.9)*	77 (67.0)**	31 (63.3)*	<0.05
CAD, no. (%)	17 (7.0)	12 (8.7)	3 (2.5)	4 (7.6)	0.22
Social support, no. (%)	135 (55.8)	97 (70.3)**	66 (55)	37 (69.8)	<0.05
Social isolation, no. (%)	124 (51.2)	82 (59.4)	80 (66.7)**	34 (64.2)	<0.05

Higher education was defined as high school or higher degree.
Abbreviations: BMI, body mass index; CAD, coronary artery disease; HDL, high-density lipoprotein.
[a]*P* value for the overall test.
*$P < 0.05$ and **$P < 0.01$ for the comparison vs. reference group.

Table 2. Systolic ambulatory blood pressure monitoring variables by alcohol consumption

	Univariable models, mean (SD)				
Variable	Reference	Very light consumption	Light consumption	Moderate-to-heavy consumption	*P* value[a]
Office SBP, mm Hg	138.7 (20.2)	134.5 (17.3)*	132.8 (15.2)**	133.9 (14.7)	<0.05
Daytime SBP, mm Hg	128.5 (15.6)	125.2 (13.1)*	128.3 (12.3)	132.2 (11.8)	<0.05
Nighttime SBP, mm Hg	120.5 (17.5)	116.6 (15.4)*	117.2 (13.3)	120.1 (14.7)	0.07
24-h SBP, mm Hg	125.6 (15.6)	122.2 (13.2)*	124.4 (11.6)	128.1 (12.3)	<0.05
SBP night-to-day ratio	0.94 (0.07)	0.93 (0.07)	0.92 (0.08)**	0.91 (0.07)**	<0.01
Daytime SBP SD, mm Hg	13.2 (3.8)	11.8 (3.0)**	12.1 (3.0)**	12.6 (3.4)	<0.01
Nighttime SBP SD, mm Hg	10.9 (3.5)	10.3 (3.1)	11.2 (3.4)	10.4 (3.0)	0.15
	Multivariable models, mean (95% CI)				
Variable	Reference	Very light consumption	Light consumption	Moderate-to-Heavy consumption	*P* value[a]
Office SBP, mm Hg	136.5 (133.7–139.3)	135.8 (132.6–139.0)	134.0 (130.5–137.6)	136.1 (131.0–141.2)	0.70
Daytime SBP, mm Hg	128.6 (126.3–130.9)	126.2 (123.6–128.8)	127.7 (124.9–130.6)	131.7 (127.6–135.8)	0.10
Nighttime SBP, mm Hg	120.5 (118.0–123.0)	118.2 (115.3–121.1)	117.1 (114.0–120.3)	120.6 (116.0–125.1)	0.22
24-h SBP, mm Hg	125.9 (123.6–128.1)	123.5 (121.0–126.0)	124.1 (121.3–126.9)	128.0 (123.9–132.0)	0.14
SBP night-to-day ratio	0.94 (0.93–0.95)	0.94 (0.92–0.95)	0.92 (0.90–0.93)*	0.91 (0.89–0.94)	0.08
Daytime SBP SD, mm Hg	13.0 (12.5–13.5)	12.2 (11.6–12.8)*	12.6 (11.9–13.2)	13.2 (12.2–14.1)	0.08
Nighttime SBP SD, mm Hg	10.9 (10.3–11.4)	10.6 (10.0–11.3)	11.3 (10.7–12.0)	10.8 (9.8–11.7)	0.43

Covariables were age, sex, race/ethnicity, education, body mass index, diabetes, smoking history, antihypertensive medication use, social support, and social isolation.

Abbreviations: DBP, diastolic blood pressure; SBP, systolic blood pressure.

[a]*P* value for the overall test.

*$P < 0.05$ and **$P \leq 0.01$ for the comparison vs. reference group.

consumption groups (all $P < 0.05$), and nighttime DBP was higher in the moderate-to-heavy consumption group compared with the reference group ($P < 0.01$). The higher ABPM DBP values associated with moderate-to-heavy drinking remained statistically significant in multivariable analyses (all $P < 0.05$).

No significant differences in heart rate (24-hour, daytime, nighttime, or variability) were observed among the different categories of alcohol consumption.

BP variability and alcohol consumption

Daytime SBP SD was significantly lower in very light and light drinkers (both $P < 0.01$) compared with the reference group (Table 2), whereas SBP night-to-day ratio was lower in light and moderate-to-heavy groups compared with the reference group (both $P < 0.01$). In multivariable analysis, daytime SBP SD remained significantly lower in the very light alcohol consumption group compared with the reference group ($P < 0.05$), and SBP night-to-day ratio remained lower in light drinkers compared with the reference group ($P < 0.05$). Daytime DBP SD was lower in very light and light drinkers compared with the reference group in univariable analysis (both $P < 0.01$), and it remained significantly lower in the very light alcohol consumption group compared with the reference group after adjusting for covariables ($P < 0.01$) (Table 3).

DISCUSSION

In this community-based elderly cohort, we showed that (i) moderate-to-heavy alcohol consumption was associated with higher BP values (daytime SBP; daytime, nighttime, and 24-hour DBP) independent of demographics and potential confounders; and (ii) very light alcohol consumption was associated with lower daytime BP variability compared with no or very infrequent/occasional consumption.

The former result confirms reports from other studies that showed a detrimental association of moderate-to-heavy consumption on BP levels after taking into account several confounding factors.[12,21] Although the magnitude of the association between alcohol and BP that we observed was smaller compared with some previous reports, on a population perspective, this magnitude of association may still have significant impact on prevalence of hypertension and related incidence of events. Furthermore, the association of alcohol consumption might be different in our predominantly elderly participants compared with younger individuals. Sex composition of the population might have also played a role in our findings compared with previous studies. Our study population was predominantly female, and it is known that women are more sensitive to the effects of alcohol because of lower enzymatic alcohol dehydrogenase hepatic activity.[26]

Table 3. Diastolic ambulatory blood pressure monitoring variables by alcohol consumption

	Univariable models, mean (SD)				
Variable	Reference	Very light consumption	Light consumption	Moderate-to-heavy consumption	*P* value[a]
Office DBP, mm Hg	78.6 (9.8)	77.5 (9.4)	78.5 (9.3)	80.0 (9.3)	0.42
Daytime DBP, mm Hg	72.6 (9.3)	72.8 (8.3)	75.3 (7.7)**	79.8 (7.4)**	<0.01
Nighttime DBP, mm Hg	65.3 (9.4)	65.0 (9.0)	66.4 (8.6)	70.2 (9.4)**	<0.01
24-h DBP, mm Hg	70.0 (8.9)	70.1 (8.0)	72.2 (7.3)*	76.5 (7.6)**	<0.01
DBP night-to-day ratio	0.90 (0.08)	0.89 (0.08)	0.89 (0.08)	0.88 (0.08)	0.13
Daytime DBP SD, mm Hg	9.4 (2.5)	8.5 (2.0)**	8.7 (2.3)**	9.1 (2.6)	<0.01
Nighttime DBP SD, mm Hg	7.90 (2.5)	7.81 (2.4)	8.05 (2.0)	8.60 (2.4)	0.20
	Multivariable models, mean (95% CI)				
Variable	Reference	Very light consumption	Light consumption	Moderate-to-heavy consumption	*P* value[a]
Office DBP, mm Hg	78.8 (77.2–80.3)	78.1 (76.4–79.9)	78.7 (76.7–80.6)	81.0 (78.2–83.7)	0.35
Daytime DBP, mm Hg	74.6 (73.3–76.0)	73.2 (71.6–74.7)	74.4 (72.7–76.1)	78.3 (75.9–80.8)**	<0.01
Nighttime DBP, mm Hg	66.9 (65.4–68.3)	65.3 (63.6–67.0)	65.5 (63.6–67.4)	69.0 (66.3–71.7)*	<0.05
24-h DBP, mm Hg	72.0 (70.7–73.3)	70.5 (69.0–72.0)	71.4 (69.7–73.0)	75.1 (72.8–77.5)*	<0.01
DBP night-to-day ratio	0.90 (0.88–0.91)	0.89 (0.88–0.91)	0.88 (0.86–0.90)	0.88 (0.86–0.91)	0.35
Daytime DBP SD, mm Hg	9.5 (9.2–9.9)	8.8 (8.4–9.2)**	9.2 (8.7–9.7)	9.5 (8.8–10.2)	<0.05
Nighttime DBP SD, mm Hg	8.1 (7.7–8.5)	7.9 (7.5–8.4)	7.9 (7.4–8.4)	8.6 (7.9–9.3)	0.36

Covariables were age, sex, race/ethnicity, education, body mass index, diabetes, smoking history, antihypertensive medication use, social isolation, and social support.

Abbreviations: DBP, diastolic blood pressure; SBP, systolic blood pressure.

[a]*P* value for the overall test.

*$P < 0.05$ and **$P \leq 0.01$ for the comparison vs. reference group.

Therefore, they might experience a greater impact of alcohol on BP than men,[22] but with a sharper dose–effect relationship and higher sensitivity after a threshold is crossed. In a recent report from the Nurses' Health Study, light-to-moderate alcohol consumption was associated with a reduced risk of stroke,[27] but the possible contribution of changes in BP was not evaluated.

An interesting and novel finding of our study was the association between very light chronic alcohol consumption and lower BP variability. Although a detrimental effect of heavy alcohol consumption on circadian BP variability is known,[28] to our knowledge this is the first report of an association between very light alcohol consumption and circadian BP variability. The difference with previous studies that showed an increase in BP variability with alcohol consumption,[28] besides differences in the studied populations, may be linked to the amount of alcohol consumed, which was considerably lower in our study than in previous ones.[28] In fact, an element of novelty of our study is the observation that low alcohol consumption (from 1 drink/month to 1 drink/day) can exert an effect on circadian BP variability, suggesting that the beneficial effect of alcohol may be obtained at lower levels than previously believed. Several studies have shown the association of BP variability with outcomes such as overall mortality or cardiovascular mortality.[29,30] Circadian BP variability was also linked with cardiovascular morbidity, especially for the risk for stroke and coronary artery disease.[31] BP variability can theoretically contribute to atherosclerosis progression through direct mechanical effects, such as shear stress and phasic BP load. The association between BP variability by ABPM and the presence of severe aortic arch atherosclerosis that we reported previously[18] or that was found with target organ damage in other studies[32,33] could be consistent with these deleterious effects. On the other hand, light-to-moderate consumption has been associated with a protective effect on the incidence of coronary artery disease and stroke.[25,34] We previously reported that light-to-moderate alcohol consumption is associated with lower atherosclerotic burden in the aortic arch, a circumstance that could be involved in alcohol's protective effect on stroke risk.[17] Our current findings suggest that lower circadian BP variability might play a role in the beneficial influence of very light alcohol consumption on cardiovascular outcomes, a hypothesis that can only be advanced as such by our cross-sectional study and will require further assessment in longitudinal studies. Interestingly, the general pattern of our data on the effect of alcohol on BP (beneficial for very light consumption, deleterious for moderate-to-heavy consumption) is compatible with the J-shaped relation known to exist between alcohol

and cardiovascular events and stroke.[19,20] It should be noted that visit-to-visit BP variability, which is biologically different from circadian BP variability, has also been shown to have important prognostic implications for a variety of cardiovascular outcomes in ways that differ from variability assessed by ABPM.[35] The assessment of the effect of alcohol consumption on visit-to-visit BP variability, which may also be of interest, was not a part of our study. BP variability is believed to reflect in part the effects of diffuse atherosclerosis. Increased arterial stiffness associated with the atherosclerotic process might decrease the ability of baroreceptors to transduce signals of acute vascular changes,[36] resulting in higher BP variability. Interventional studies have shown a beneficial impact of light-to-moderate alcohol on arterial stiffness, possibly through anti-inflammatory, antioxidant, and antithrombotic properties.[37,38] As a consequence, a beneficial effect exerted by chronic light alcohol consumption on arterial stiffness may also be implicated in its effect on BP variability observed in our study.

Strengths of our study are the relatively large sample size of elderly individuals undergoing ABPM because ABPM has been shown to be superior to clinic BP measurements for the predictors of cardiovascular events.[39] Our cohort was community based and enrolled using random digit dialing, which minimized biases. Also, we only included in the main analysis subjects who confirmed steady alcohol consumption over the 5 years before enrollment, which may have resulted in a more reliable assessment and also helped avoid interference on the results by "sick quitters." Our study also has limitations that need to be highlighted. The cross-sectional design of our study can document associations between variables, and although we accounted for the effect of several confounders in multivariable models, a direct cause–effect relationship of alcohol on BP values cannot be implied. Our cohort had a high cardiovascular risk profile and was largely of Hispanic ethnicity. Therefore, our results might not entirely apply to other cohorts with different demographics and risk profiles. However, our data provide important information on the Hispanic subgroup, which is usually under-represented in epidemiological studies.[40] Because dietary variables and exercise patterns (the latter having, however, a very narrow distribution in this elderly population) were not considered in our analysis, their potential influence on our findings cannot be excluded. The duration of alcohol consumption beyond the years we monitored was not considered in the analysis. The potential effect of a survival bias (i.e., heavy alcohol users not having been included because of premature death) cannot be evaluated in this cross-sectional analysis. Finally, the small sample size of the moderate-to-heavy consumption subgroup may have affected the significance of the results observed for that subgroup.

Our study in an elderly community-based cohort showed that moderate-to-heavy alcohol consumption was associated with higher BP values measured by 24-hour ABPM and that very light consumption was associated with lower BP variability. This association might have a role in the known cardio-protective effects of light-to-moderate alcohol consumption, a hypothesis that requires further investigation.

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DISCLOSURE

The authors declared no conflict of interest.

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