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Light–moderate alcohol consumption and left ventricular function among healthy, middle-aged adults: the HUNT study

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ABSTRACT

Objectives To investigate the association between alcohol consumption and left ventricular (LV) function in a population with low average alcohol intake.

Design, setting and participants A total of 1296 healthy participants, free from cardiovascular diseases, were randomly selected from the third wave of the Norwegian HUNT study (2006–2008) and underwent echocardiography. After validation of the inclusion criteria, 30 participants were excluded due to arrhythmias or myocardial or valvular pathology. Alcohol consumption, sociodemographic and major cardiovascular risk factors were assessed by questionnaires and clinical examination in the HUNT3. General linear models were used to analyse the cross-sectional associations between alcohol intake and LV indices.

Primary and secondary outcome measures LV functional and structural indices were measured with tissue Doppler and speckle tracking echocardiography.

Results We observed no associations between alcohol consumption and multivariable-adjusted LV functional indices. Excluding abstainers who reported regular alcohol consumption 10 years prior to the baseline did not change the results. Alcohol consumption was positively associated with LV mass indices ($p<0.01$ for linear trend of the means); there was no such association among participants with non-risky drinking characteristics ($p=0.67$ for linear trend of the means).

Conclusions We found no clear evidence that light–moderate alcohol consumption is associated with measures of LV function, although our results indicate that consumption, especially when marked by binge drinking, is progressively associated with greater LV mass.

Strengths and limitations of this study

- The HUNT was conducted in a socioeconomically and genetically homogenous population, where due to the strict alcohol policy the majority of the population was non-drinkers or light drinkers which decreased confounding by social factors in this study. This, together with the statistical adjustment for a wide range of cardiovascular risk factors, reduces the possibility of residual confounding in our study.
- Alcohol consumption was also assessed 10 years prior to baseline as part of the HUNT2 study, offering a prospective assessment of former drinking.
- In this study, greyscale speckle tracking and tissue Doppler echocardiography was used, which provides more sensitive left ventricular functional indices than ejection fraction measures.
- The main limitation of our study relates to its cross-sectional design.
- As in many previous studies in this field, alcohol consumption was self-reported.

INTRODUCTION

While excessive alcohol consumption is clearly responsible for alcohol-related cardiomyopathy,[1 2] several studies suggest that frequent but moderate alcohol consumption is associated with lower risk of heart failure.[3] Some studies have examined the associations of light–moderate alcohol consumption with subclinical structural and functional properties of the left ventricle among asymptomatic participants, but the results of these studies have been inconsistent. Although most describe increasing LV mass with increasing level of alcohol consumption,[4–7] the associations with LV function are less consistent. Some found better LV function with increasing alcohol intake,[4] while others reported the opposite.[5 6] Most studies assessed ejection fraction (EF), a potentially less sensitive index of LV functional impairment.[8 9] Speckle tracking and tissue Doppler echocardiography may provide more direct measures of LV contractile function.[8 10–13] Furthermore, the majority of these studies have been subject to several potential limitations. A major methodological problem that limits causal inference in studies regarding the health effects of alcohol is the so called 'sick-quitter' bias.[14 15] Thus the U-shaped or J-shaped association often found for alcohol consumption and many health outcomes[14–16]

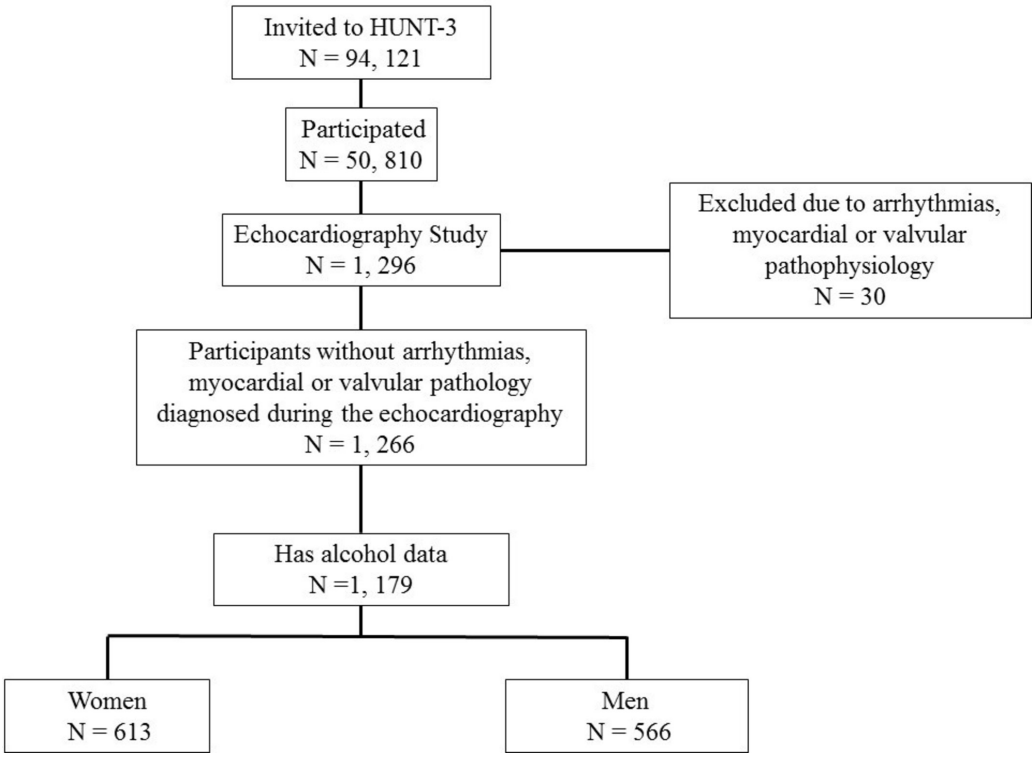

Figure 1 Flowchart for the selection of the study population.

might be an artefact caused by the higher risk of adverse health outcomes among abstainers who quit drinking due to their ill health.[17]

In addition, most previous studies only assessed the quantity of alcohol intake[4–6] and not the drinking pattern (ie, heavy episodic vs regular, low amount of alcohol consumption) at identical quantities of intake, which might potentially modify the association between alcohol consumption and LV function.[6]

In this study, we examined the associations of alcohol intake with LV functional and structural measures in a healthy middle-aged population. We also assessed drinking pattern and quantity of alcohol consumption 10 years prior to baseline. To detect early, subclinical signs of myocardial dysfunction, LV functional indices were measured using highly sensitive tissue Doppler and speckle tracking echocardiography.[13 18]

METHODS

Study population

Between 2006 and 2008, the entire adult population of the Nord-Trøndelag County in Norway, representing 93 210 citizens, was invited to participate in the third wave of Nord-Trøndelag Health Study (HUNT3) (http://www.ntnu.edu/hunt). Altogether, 50 810 individuals (54% of those invited) participated in the study.[19] Data on sociodemographic factors, anthropometrics, overall health status, chronic health conditions and health behaviour were collected by self-reported questionnaires, interviews and clinical examinations.[19]

A subsample of healthy individuals without known cardiovascular diseases (CVDs), diabetes or hypertension (n=1 296) was randomly selected from the HUNT3 cohort to participate in the Echocardiography Study.[13 18] After validation of the inclusion criteria by an experienced physician echocardiographer, 30 participants were excluded from the study due to arrhythmias or myocardial or valvular pathology.[13 18] In addition, for the analyses of the present study, we excluded 87 participants with missing data on alcohol consumption (figure 1).

The Echocardiography study was approved and it was conducted according to the second Helsinki Declaration.

Measures

Alcohol consumption

Alcohol consumption was assessed by self-administered questionnaires. Participants reported the amount and type of alcoholic beverages they consume in a usual 2-week period. The frequency of alcohol consumption was assessed for the previous 12 months. Those who answered that they did not drink any alcohol in the previous 12 months were considered abstainers. Those who reported any alcohol consumption during the previous year but reported no consumption during a usual 2-week period were categorised as rare drinkers. Those who reported at least one standard drink during a usual 2-week period were categorised as having an intake of one of the following: 'at least 0.5 and less than or equal to three drinks/week', 'more than three and less than or equal to seven drinks/week' or 'more than seven drinks/week'. The frequency of drinking was categorised as: consuming

alcoholic beverages 'less than once a month', '1–4 times a month' or 'more than once a week'. Binge drinking was defined as consuming more than five drinks in one sitting. Problem drinking behaviour was assessed by the CAGE questionnaire.[20] Risky drinkers were defined as those who answered positively to at least two questions of the CAGE questionnaire[21] and/or reported binge drinking at least once a month. Overall, 50% of the participants gave information about their alcohol consumption in the second wave of the HUNT Study (HUNT2) conducted approximately 10 years prior to HUNT3.[19] Current non-drinkers were categorised as 'long-term abstainers' or 'former drinkers, but current abstainers' according to their previous alcohol intake.

Echocardiography

Echocardiograms were recorded in the left-lateral decubitus position by a Vivid 7 high-end scanner (V.BT06, GE Ultrasound, Horten, Norway) with a phased-array transducer (M3S and M4S) assessed by an experienced physician echocardiographer. Analyses were performed in EchoPAC SWO software (GE Ultrasound) if not otherwise specified. The detailed protocol, methodology and validity of the echocardiographic measurement have been published elsewhere.[13 18] Shortly, the mean errors of the LV function indices were 4%–9% (test– retest, two observers analysing separate recordings).

LV function was assessed by well-established echocardiographic indices of the diastolic and systolic longitudinal function. Global longitudinal end-systolic strain (percentage shortening of the left ventricle during systole) and peak global strain rate (the maximal speed of the global longitudinal strain) were measured as the average of the segmental values according to a 16-segment model of the left ventricle [22] using a combined tissue Doppler and greyscale speckle tracking method in a customised software package (GcMat) based on a MatLab platform (MathWorks, Natick, Massachusetts, USA).[13 18] Both LV longitudinal strain and velocity can detect subclinical myocardial dysfunction before EF is materially affected. These indices are also associated with acute myocardial infarction, heart failure and cardiovascular and all-cause mortality.[10 23]

Systolic mitral annular excursion (MAE) was measured by corrected motion mode from greyscale recordings as the average of the total systolic excursion of the mitral annular plane at the inferoseptal, lateral, anterior and inferior positions of the LV wall. Peak systolic (S') and peak early diastolic mitral annular velocity (e') were calculated as the average of peak velocities measured at the same locations by pulsed wave tissue Doppler echocardiography.[13 18]

Tricuspid annular plane systolic excursion (TAPSE) and tricuspid annular peak systolic velocity (RS') were measured close to the tricuspid plane in the free wall of the right ventricle, by similar methodologies as described for the MAE and mitral annular velocities.

Mitral inflow early (E) and late (A) diastolic velocities were recorded by pulse wave Doppler, and the E/A ratio was calculated. E/e' ratio was calculated as the ratio of the peak early diastolic mitral inflow per mitral annular early diastolic velocities.

Conventional LV structural indices (interventricular septum and posterior wall thickness and LV internal dimensions) were assessed in parasternal M-mode. LV mass was estimated according to the Cube formula and indexed for body surface area (BSA). Relative wall thickness was defined as 2xLV posterior wall thickness divided by the LV end-diastolic diameter.

Sociodemographic and lifestyle factors

Highest achieved education was categorised as primary or lower secondary school (<10 years), upper secondary school (10–12 years) or high school/university (>12 years). Marital status was categorised as: (1) never married, (2) married or cohabiting and (3) separated or widowed.

Participants were classified as never, former or current smokers. A validated physical activity index was calculated based on the reported frequency, duration and intensity of the physical activity, as previously described.[24]

Clinical examination

Weight, height and blood pressure were measured by trained nurses. Body mass index (BMI) was calculated by dividing weight in kilogram by height^2 in metres and dichotomised at 25 kg/m^2. BSA was calculated according to the DuBois formula. Information on common chronic disorders were self-reported by the participants (described in detail elsewhere).[19]

Statistical analyses

As the reference values for most of the echocardiographic measurements differ according to sex, we conducted our main analyses for the whole population and separately for men and women.[22 25] To examine the association between alcohol consumption and echocardiographic indices of cardiac structure and function, we performed general linear models. Least square means of cardiac function indices (MAE, global longitudinal strain, global longitudinal strain rate, peak early diastolic and systolic mitral annular velocities (e', S'), E/e', E/A, TAPSE and RS') and LV structural indices (myocardial mass, wall thickness and dimensions) and 95% CIs were calculated across alcohol consumption categories. In light of covariates, we conducted full-case analyses. We adjusted our analyses for age, education, marital status, physical activity, smoking, BMI and sex (if not stratified). In additional analyses, we further included systolic blood pressure, which may be a consequence of alcohol consumption. We tested linear as well as quadratic trends. As estimates did not differ between the abstainer and rare-drinker groups, we combined these two groups (as non-drinkers) for further analyses and present all results accordingly.

To test for the 'sick quitters' bias,[14 15] we repeated our main analyses after excluding abstainers who reported alcohol intake during the earlier HUNT2. To examine whether risky drinking modified the observed associations, we performed stratified analyses by reported binge and/or problem drinking and tested interaction between alcohol intake and binge and/or problem drinking using Wald tests. We also added the frequency of alcohol intake to model 2 while simultaneously adjusting for the amount of alcohol consumption to determine the relative contributions of quantity and frequency of intake. Beverage specific analyses were conducted in the same fashion. To investigate the effect of a specific type of beverage, for example, wine, we examined the association between alcohol intake and LV functional and structural indices while adjusting for amounts consumed of the other two beverages, that is, beer and spirit in this example.

To assess effect modification, we stratified our analyses by sex, age (dichotomised at 50 years), smoking and BMI (dichotomised at 25 kg/m^2) and also tested for the interaction between alcohol consumption and the effect modifiers. As previous studies usually assessed LV function as EF, we also performed sensitivity analyses with this outcome to improve the comparability of our results with those of previous studies. In other sensitivity analysis, we examined age-adjusted models using alcohol consumption from HUNT2 as exposure and MAE, strain, strain rate, S' and e' and LV mass from HUNT3 as the outcome.

Statistical analyses were performed using SAS Enterprise Guide V.6.0 (SAS Institute) and Stata IC/12.1 for Windows (Stata).

RESULTS

Table 1 shows the characteristics of study participants separately for men and women. The majority of both men and women were light drinkers, consuming less than three alcoholic drinks per week; approximately 4.5% of both men and women reported consumption of more than seven alcoholic drinks per week. For both genders, drinkers were more likely to be smokers and to have a slightly lower BMI than non-drinkers. Men who reported alcohol consumption tended to be younger than abstainers.

Amount of alcohol consumption

Table 2a and b shows the multivariable-adjusted means of LV indices, which were largely similar across alcohol intake categories. Quantity of alcohol intake was associated with higher LV mass, indexed LV mass and LV end-diastolic dimension in both men and women. The results were essentially the same when systolic blood pressure was added to the models. Excluding former drinkers (n=117) did not influence the association. There was no meaningful association between alcohol intake and EF (online supplementary eTable 1).

Drinking pattern and beverage type analyses

In general, drinking frequency did not influence the associations between alcohol intake and LV indices (online supplementary eTable 2). When we stratified our analyses according to risky and non-risky drinking, we found a weak tendency toward higher values on some of the LV indices with higher alcohol intake among participants without risky drinking, and a linear increase in LV mass indices among risky drinkers (table 3). Risky drinking was positively associated with LV mass (P<0.01) but not with the LV functional indices, independently from the amount of alcohol intake. The associations between alcohol consumption and LV indices did not differ according to beverage type nor did we find any consistent evidence for effect modification by age, sex, BMI or smoking status. We found no clear association between alcohol consumption assessed in HUNT2 and LV functional indices and LV mass in HUNT3, but the precision of our estimates was limited by the 573 individuals from our sample participated in both HUNT2 and 3 (see online supplementary eTable 3).

DISCUSSION

We found no clear evidence for a clinically meaningful association between light–moderate alcohol consumption and several sensitive echocardiographic indices for assessment of systolic and diastolic left and right ventricle function in this cohort of healthy individuals. However, LV mass and LV end-diastolic diameter showed positive linear associations with alcohol consumption, and the association with LV mass was particularly strong among participants with a risky drinking pattern. Neither frequency of drinking nor beverage types showed clear associations with the studied echocardiographic indices beyond the quantity of alcohol consumed.

Comparison with previous studies

Our study adds to a still limited literature relating alcohol consumption in the general population and LV function indices. In the present study, LV function was assessed by highly sensitive echocardiographic methods[13 18] in a healthy middle-aged Caucasian population. Previous studies on alcohol consumption and LV function sometimes used less sensitive measurements on LV function such as ejection fraction,[4 26] while others were restricted to elderly[5] or Asian participants.[6] Among the most sensitive indices used to reveal LV diastolic and systolic dysfunction are global longitudinal strain and the peak systolic and early diastolic mitral annular velocities (S' and e'). The indices are sensitive to LV subclinical dysfunction and give prognostic information related to heart failure, cardiovascular death and risk of myocardial infarction.[23 27] Goncalves *et al*[5] found a positive association between alcohol intake and peak early diastolic mitral annular velocity (e') in men.[5] We could not confirm such an association, but e' was slightly higher among women, who reported drinking three to seven alcoholic drinks per week, compared with women who reported less than this amount. On the other hand, alcohol intake

Table 1 Characteristics of the study participants according to the categories of alcohol consumption

Study variables	No of participants		Quantity of alcohol consumption							
			<0.5 drinks per week^{-1}*		≥0.5 and ≤3 drinks per week^{-1}		>3 and ≤7 drinks per week^{-1}		>7 drinks per week^{-1}	
	Women	Men	Women	Men	Women	Men	Women	Men	Women	Men
Number of participants	613	566	98	86	364	344	123	110	28	26
Categorical variables (%)										
Education										
Primary–secondary	86 (400)	67 (367)	64 (61)	75 (63)	66 (244)	68 (234)	67 (81)	31 (66)	50 (14)	50 (13)
Tertiary	24 (205)	33 (187)	36 (34)	26 (22)	33 (117)	32 (109)	33 (40)	40 (43)	50 (14)	50 (13)
Smoking										
Never–former	77 (451)	74 (435)	83 (46)	76 (70)	74 (451)	78 (266)	70 (86)	75 (83)	64 (18)	62 (16)
Current	23 (151)	26 (126)	17 (23)	24 (14)	26 (84)	22 (75)	30 (36)	25 (27)	36 (10)	38 (10)
Marital status										
Living alone	41 (249)	36 (203)	47 (46)	36 (47)	39 (142)	33 (115)	40 (49)	39 (43)	43 (13)	54 (14)
Cohabiting	59 (363)	64 (362)	53 (52)	64 (55)	61 (222)	67 (228)	60 (73)	61 (67)	57 (16)	46 (12)
Continuous variables: mean (SD)										
Age (years)	49.3 (13.6)	49.0 (13.5)	49.1 (14.9)	51.4 (15.5)	49.2 (13.7)	49.0 (13.0)	49.6 (13.2)	48.4 (13.1)	49.6 (11.7)	44.0 (12.7)
Body mass index (kg/m^2)	26.3 (4.0)	26.3 (3.7)	27.3 (4.9)	27.3 (4.6)	26.1 (3.9)	26.1 (3.7)	26.2 (3.7)	26.2 (3.3)	25.9 (2.4)	25.9 (2.9)
Physical activity index	2.1 (0.8)	2.2 (0.8)	2.0 (0.8)	2.1 (0.7)	2.2 (0.8)	2.1 (0.8)	2.0 (0.9)	2.2 (0.8)	2.1 (0.8)	2.2 (0.8)
Systolic blood pressure (mm Hg)	127 (17)	133 (14)	127 (17)	132 (15)	127 (17)	133 (14)	126 (16)	133 (14)	130 (25)	133 (12)
Diastolic blood pressure (mm Hg)	71 (10)	77 (10)	70 (9.6)	77 (10)	71 (10)	77 (10)	73 (10)	77 (10)	75 (13)	74(8)
Serum HDL-C (mmol/L)	1.48 (0.34)	1.35 (0.33)	1.47 (0.41)	1.44 (0.36)	1.47 (0.32)	1.39 (0.35)	1.45 (0.29)	1.34 (0.33)	1.49 (0.43)	1.42 (0.34)

*Reported consuming less than one alcoholic drink consumption in a regular 2-week period.
HDL-C, high-density lipoprotein cholesterol.

Table 2a Least square means and 95% CIs for left ventricular functional indices according to categories of alcohol consumption

		LS means* (95% CI) of alcohol consumption categories				P values	
	N	Non-drinkers	≥0.5 and ≤3 drinks per week	>3 and ≤7 drinks per week	>7 drinks per week	Linear	Quadratic
Mitral annular excursion (cm)							
Men	534	1.57 (1.51 to 1.63)	1.57 (1.54 to 1.61)	1.59 (1.54 to 1.64)	1.64 (1.55 to 1.74)	0.18	0.37
Women	544	1.57 (1.52 to 1.63)	1.59 (1.56 to 1.63)	1.61 (1.56 to 1.66)	1.57 (1.47 to 1.67)	0.98	0.34
Global longitudinal strain (%)							
Men	548	−15.9 (−16.4 to −15.3)	−16.0 (−16.2 to −15.7)	−16.0 (−16.2 to −15.5)	−16.2 (−17.1 to −15.2)	0.70	0.59
Women	587	−17.7 (−18.2 to −17.3)	−17.4 (−17.6 to −17.1)	−17.3 (−17.7 to −16.8)	−17.1 (−17.9 to −16.2)	0.34	0.42
Global longitudinal strain rate (s^{-1})							
Men	513	−1.01 (−1.04 to −0.98)	−1.04 (−1.06 to −1.03)	−1.04 (−1.07 to −1.02)	−1.00 (−1.06 to −0.95)	0.74	0.05
Women	548	−1.02 (−1.05 to −0.99)	−1.02 (−1.03 to −1.00)	−1.04 (−1.06 to −1.01)	−1.04 (−1.09 to 0.99)	0.34	0.89
e' (cm/s)							
Men	528	11.1 (10.4 to 11.7)	10.6 (10.3 to 10.9)	11.1 (10.6 to 11.7)	11.6 (10.4 to 12.9)	0.40	0.44
Women	556	11.6 (11.0 to 12.2)	11.6 (11.3 to 11.9)	12.2 (11.6 to 12.7)	11.5 (11.4 to 13.5)	0.10	0.67
S' (cm/s)							
Men	520	8.72 (8.38 to 9.05)	8.63 (8.44 to 8.83)	8.83 (8.54 to 9.11)	9.07 (8.51 to 9.63)	0.30	0.87
Women	556	8.13 (7.86 to 8.40)	8.27 (8.10 to 8.43)	8.20 (7.95 to 8.45)	8.20 (7.71 to 8.69)	0.69	0.85
E/A ratio							
Men	552	1.41 (1.29 to 1.53)	1.38 (1.31 to 1.45)	1.42 (1.32 to 1.53)	1.31 (1.11 to 1.50)	0.48	0.49
Women	586	1.37 (1.28 to 1.46)	1.36 (1.30 to 1.41)	1.34 (1.36 to 1.42)	1.31 (1.14 to 1.47)	0.41	0.90
E/e' ratio							
Men	545	6.92 (6.24 to 7.59)	7.24 (6.86 to 7.63)	6.83 (6.26 to 7.40)	6.81 (5.71 to 7.92)	0.74	0.60
Women	580	6.52 (5.96 to 7.01)	6.67 (6.33 to 7.00)	6.27 (5.76 to 6.79)	6.34 (5.33 to 7.34)	0.61	0.99
RS' (cm/s)							
Men	549	12.9 (11.9 to 12.7)	12.8 (12.3 to 12.8)	13.0 (12.2 to 13.0)	13.0 (11.8 to 13.2)	0.76	0.91
Women	587	12.3 (12.4 to 13.4)	12.5 (12.5 to 13.0)	12.6 (12.6 to 13.4)	12.5 (12.1 to 13.8)	0.57	0.39
TAPSE (cm)							
Men	570	2.80 (2.66 to 2.94)	2.85 (2.78 to 2.92)	2.94 (2.82 to 3.06)	2.84 (2.61 to 3.08)	0.91	0.31
Women	527	2.72 (2.62 to 2.83)	2.81 (2.75 to 2.87)	2.78 (2.69 to 2.88)	2.74 (2.55 to 2.93)	0.59	0.33

*Means are adjusted for age, marital status and education, smoking, physical activity and body mass index.
e', peak early diastolic mitral annular velocity; RS', peak tricuspid annular systolic velocity; S', peak systolic mitral annular velocity; TAPSE, tricuspid annular plane systolic excursion.

Table 2b Least square means and 95% CIsfor left ventricular structural indices according to categories of alcohol consumption

		LS means* (95% CI) of alcohol consumption categories				P values	
	N	Non-drinkers	≥0.5 and ≤3 drinks per week	>3 and ≤7 drinks per week	>7 drinks per week	Linear	Quadratic
LV mass (g)							
Men	551	157.0 (146.8 to 167.2)	158.6 (153.1 to 164.0)	169.5 (160.7 to 178.2)	182.1 (164.6 to 199.7)	<0.01	0.92
Women	578	151.6 (142.0 to 161.2)	159.5 (154.1 to 164.9)	169.3 (160.6 to 177.9)	180.0 (162.7 to 197.3)	<0.01	0.13
LV mass per BSA (g/m^2)							
Men	550	76.8 (71.5 to 82.1)	78.6 (75.8 to 81.4)	83.0 (78.5 to 87.5)	91.4 (82.3 to 100.4)	<0.01	0.67
Women	577	86.1 (80.4 to 91.8)	90.4 (87.2 to 93.6)	95.5 (90.4 to 100.6)	102.6 (92.4 to 112.8)	<0.01	0.02
LV end-diastolic diameter (mm)							
Men	548	50.7 (49.5 to 51.9)	50.9 (50.3 to 51.5)	51.0 (50.0 to 52.0)	52.1 (50.0 to 54.2)	0.03	0.23
Women	577	49.4 (48.3 to 50.5)	50.4 (49.8 to 51.1)	51.4 (50.4 to 52.4)	52.5 (50.5 to 54.6)	0.07	0.31
LV end-systolic diameter (mm)							
Men	548	32.3 (31.2 to 33.4)	32.4 (31.8 to 33.0)	32.4 (31.5 to 33.4)	33.9 (31.9 to 35.8)	0.17	0.29
Women	577	31.1 (30.1 to 32.1)	32.2 (31.6 to 32.8)	32.7 (31.8 to 33.7)	34.6 (32.8 to 36.5)	<0.01	0.48
Relative wall thickness (cm)							
Men	551	0.25 (0.24 to 0.26)	0.26 (0.25 to 0.26)	0.27 (0.27 to 0.27)	0.27 (0.25 to 0.28)	0.07	0.96
Women	578	0.26 (0.25 to 0.27)	0.26 (0.26 to 0.27)	0.26 (0.26 to 0.27)	0.27 (0.26 to 0.28)	0.47	0.31

*Means are adjusted for age, marital status and education, smoking, physical activity and body mass index.
BSA, body surface area; e', peak early diastolic mitral annular velocity; LV, left ventricular; RS', peak tricuspid annular systolic velocity, S', peak systolic mitral annular velocity; TAPSE, tricuspid annular plane systolic excursion.

Table 3 Least square means and 95% of CI for the left ventricular function indices according to problem and/or binge drinking* and quantity of alcohol consumption

	Participants without problem and/or binge drinking		Participants with problem and/or binge drinking	
LV functional and structural indices	**Mean***	**95% CI**	**Mean***	**95% CI**
Mitral annular excursion (cm)	n=717		n=394	
Non-drinker	1.60	1.46 to 1.73	1.60	1.46 to 1.73
≥0.5 and ≤3 drinks/week	1.61	1.57 to 1.64	1.61	1.57 to 1.64
>3 and ≤7 drinks/week	1.62	1.58 to 1.67	1.62	1.58 to 1.67
>7 drinks/week	1.61	1.53 to 1.68	1.61	1.53 to 1.68
P values (linear)	0.97		0.85	
P values (quadratic)	0.87		0.78	
Global longitudinal strain (%)	n=735		n=404	
Non-drinker	−16.6	−17.3 to −16.0	−17.3	−18.6 to 16.0
≥0.5 and ≤3 drinks/week	−16.8	−17.2 to −16.6	−16.6	−17.0 to 16.3
>3 and ≤7 drinks/week	−16.4	−17.0 to −15.9	−16.7	−17.2 to 16.4
>7 drinks/week	−16.8	−18.2 to −15.4	−16.6	−17.3 to 15.9
P values (linear)	0.943		0.41	
P values (quadratic)	0.839		0.59	
Global longitudinal strain rate (/s)	n=727		n=399	
Non-drinker	−0.99	−1.02 to −0.95	−1.03	−1.11 to 0.95
≥0.5 and ≤3 drinks/week	−1.03	−1.04 to −1.02	−1.04	−1.06 to 1.02
>3 and ≤7 drinks/week	−1.04	−1.07 to −1.01	−1.05	−1.07 to 1.02
>7 drinks/week	−1.02	−1.10 to −0.95	−1.02	−1.06 to 0.97
P values (linear)	0.348		0.83	
P values (quadratic)	0.166		0.39	
e' (cm/s)	n=580		n=403	
Non-drinker	11.1	10.4 to 12.0	12.1	10.4 to 13.9
>=0.5 and <=3 drinks/week	11.1	10.8 to 11.4	11.3	11.9 to 11.7
>3 and <=7 drinks/week	11.5	10.8 to 12.2	11.7	11.2 to 12.2
>7 drinks/week	12.5	10.7 to 14.2	11.5	10.5 to 12.4
P values (linear)	0.15		0.60	
P values (quadratic)	0.37		0.56	
S' (cm/s)	n=580		n=403	
Non-drinker	8.49	8.14 to 8.85	8.16	7.41 to 8.91
≥0.5 and ≤3 drinks/week	8.39	8.26 to 8.52	8.43	8.25 to 8.62
>3 and ≤7 drinks/week	8.54	8.25 to 8.82	8.42	8.18 to 8.65
>7 drinks/week	9.32	9.32 to 10.09	8.38	7.96 to 8.81
P values (linear)	0.05		0.63	
P values (quadratic)	0.05		0.52	
LV mass per BSA (gr/m^2)†	n=571		n=403	
Non-drinker	83.6	76.9 to 90.4	87.8	73.7 to 101.8
≥0.5 and ≤3 drinks/week	83.7	81.3 to 86.2	88.8	85.5 to 92.1
>3 and ≤7 drinks/week	82.7	77.4 to 88.1	93.4	89.2 to 97.6
>7 drinks/week	80.4	66.1 to 94.7	100.88	93.3 to 108.4
P values (linear)	0.67		0.07	
P values (quadratic)	0.78		0.46	

Problem drinking was defined as having at least two positive answers on the CAGE questionnaire or reporting more than one binge drinking occasion during the last month.
*Adjusted for age, marital status, education, smoking, body mass index and physical activity.
†The P values for the test for interaction was <0.05.
BSA, body surface area; e', peak early diastolic mitral annular velocity; LV, left ventricular; S', peak systolic mitral annular velocity.

(especially in men) showed a positive but weak association with peak systolic mitral annular velocity (S'). However, consistent with previous studies,[5 6] we did not find clinically meaningful differences in most indices between the different alcohol intake categories.

Concerning structural characteristics, both our study and the study by Goncalves and colleagues[5] found marginally higher LV end-diastolic and end-systolic dimensions in higher alcohol consumption categories. We also found a positive association between the quantity of alcohol consumption and LV mass, an established risk factor for CVD.[28] Previous studies have also shown increased LV mass, but only in individuals with alcohol consumption above 14 drinks per week.[4 5 7 29] However, our results provide important information to the relationship between alcohol consumption and structural cardiac changes by, to our knowledge, including for the first time information on risky drinking.

Heavy episodic drinking can lead to myocardial damage[1 30 31] and can be responsible for impaired LV function.[2] It is associated with higher risk for CVD, even if the overall alcohol consumption stays at a moderate level.[16 32–34] Indeed, one binge-drinking episode (ie, consuming more than five drinks in one setting during a short period of time) can cause a pronounced inflammatory response in the myocardium.[35] In our study, we found a clear increase in LV mass with higher reported alcohol intake among individuals with risky drinking patterns, but no such association among those who denied bingeing and problem drinking. The finding of higher LV mass among individuals with risky drinking patterns might be a subtle sign of ongoing cardiac remodelling, without any sign of or preceding subclinical dysfunction. This finding also fits well with the established effects of binge drinking (or 'holiday heart') on atrial arrhythmias.[36]

Strengths and limitations

The HUNT was conducted in a socioeconomically and genetically homogenous population.[19] Furthermore, due to a relatively strict Norwegian alcohol policy, which includes high tax, limited availability of alcoholic beverages only in state-owned alcohol shops and promotion of alcohol-free public places, non-drinking is culturally more accepted than in most other Western countries,[37] where moderate drinking is the cultural norm and both abstainers and heavy drinkers are often socially less active individuals with less social support and more psychological distress.[38 39]

This, together with the statistical adjustment for a wide range of CVD risk factors, reduces the possibility of residual confounding in our study, as it is less likely that unmeasured factors might be strongly associated with both alcohol consumption and LV function and unrelated to the covariates we included.

Many studies in alcohol research may be affected by the sick-quitter bias.[14 15 17] Typically, when former drinking is assessed, it is done so during the baseline measurements and hence may be prone to recall bias. Only one study that examined the association between alcohol consumption and LV function[23] used longitudinal assessment on alcohol consumption, but their results were inconclusive.[5] In our study, alcohol consumption was also assessed 10 years prior to baseline as part of the HUNT2 study, offering a prospective assessment of former drinking. Notably, excluding former drinkers did not change our results.

The main limitation of our study relates to its cross-sectional design. Second, similar to many previous studies in this field, alcohol consumption was self-reported. Study participants, particularly high consumers, may tend to under-report their alcohol intake,[40 41] although it is unlikely that the rank ordering would be markedly influenced. However, alcohol consumption was weakly correlated with high-density lipoprotein cholesterol level in the HUNT3 population. As less than 1% of the participants reported consuming more than seven alcoholic drinks per week, we were not able to examine the possible harmful effect of excessive alcohol intake on LV function over this amount of alcohol consumption. Nonetheless, the relatively low level of alcohol intake reported here does also limit our ability to separately assess other dimensions of drinking, such as beverage type and drinking frequency versus quantity, and it allowed us to examine the specific role of binge drinking in an otherwise light-drinking population.

CONCLUSION

In summary, moderate alcohol consumption was not associated with LV function indices among middle-aged healthy individuals. However, alcohol consumption was positively associated with LV mass among otherwise light consumers, especially among those who report risky drinking behaviours, which highlights the importance of drinking pattern in understanding the cardiovascular effects of alcohol consumption.

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