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Acute effects of beer on endothelial function and haemodynamics: a single-blind, cross-over study in healthy volunteers

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Abstract

Objective—Moderate consumption of beer is associated with lower cardiovascular (CV) risk. To explore the underlying mechanisms we studied the acute effects of the constituents of beer (alcohol and antioxidants), on established predictors of CV risk: endothelial function, aortic stiffness, pressure wave reflections and aortic pressure.

Research Methods & Proceedures—In a randomized, single – blind, cross - over study 17 healthy, non-smoking, volunteers (28.5±5.2 years and 24.4±2.5 BMI) consumed in 3 separate days, at least one week apart: a) 400 ml of beer & 400 ml water, b) 800 ml of dealcoholized beer (same amount of polyphenols), and c) 67 ml of vodka & 733 ml water (same amount of alcohol). Each time aortic stiffness (pulse wave velocity, pressure wave reflections (Aix), aortic and brachial pressure (Sphygmocor device) and endothelial function (brachial flow mediated dilatation) were assessed at fast and 1 and 2 hours postprandial.

Results—Aortic stiffness was significantly and similarly reduced by all 3 interventions. However, endothelial function was significantly improved only after beer consumption (average of 1.33%, CI 0.15-2.53). Although wave reflections were significantly reduced by all 3 interventions (average of beer: 9.1%, dealcoholized beer: 2.8%, vodka 8.5%, all CI within limits of significance), the reduction was higher after beer consumption compared todealcoholized beer (p=0.018). Pulse pressure amplification (i.e. brachial/aortic) was increased by all 3 test drinks.

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The authors declare that they have no conflict of interest.

Conclusions—Beer improves acutely parameters of arterial function and structure, in healthy non-smokers. This benefit seems to be mediated by the additive or synergistic effects of alcohol and anti-oxidants and merits further investigation.

Keywords

beer; endothelial function; arterial stiffness; aortic pressure; alcohol

Introduction

Considerable interest in the cardiovascular effects of moderate alcohol consumption is evident during the last 30 years [1]. The majority of studies suggest a lower risk of coronary heart disease and all-cause mortality in middle-aged and older adults consuming one to two alcoholic beverages daily [2, 3]. It is suggested that there are several combined, additive or synergistic effects of alcohol and non-alcoholic components (i.e. antioxidants) found in alcoholic drinks (mostly red wine, beer and whisky) covering all phases of the atherosclerotic process; endothelial function, arterial stiffening and atherogenesis as well as coagulation and fibrinolysis [2]. Particularly, the acute and long – term beneficial effects of red wine and its components (alcohol and antioxidants) on endothelial function and haemodynamics have been explored previously in healthy volunteers and patients with coronary artery disease (CAD) [3, 4].

On the other hand, the effect of another widely consumed drink such as beer on arterial function and structure has been inadequately investigated so far. Like red wine, beer contains both ethanol and antioxidant substances [7]. Ethanol concentration is less than in red wine and its antioxidant content is equivalent but varies on specific antioxidants [5]. As with other alcohol beverages, a moderate daily beer consumption appears to be negatively associated with blood pressure and cardiovascular disease but higher daily dose may have detrimental effects, leading to a J- shape or U-shape relation [6]. A few studies have investigated the effect of beer on arterial function in comparison to red wine [12-13]. To our knowledge there are no data regarding the effect of acute beer intake on arterial biomarkers of atherosclerosis (pulse wave velocity (PWV), augmentation index (Aix), flow mediated dilation (FMD) as well as on aortic pressure. The above biomarkers assess non-invasively accelerated atherosclerosis and they are independent predictors of cardiovascular disease risk [7, 8]. Significant improvements of these markers through drug administration or lifestyle changes have a potential beneficial impact on the atherosclerosis process and reduce cardiovascular morbidity/mortality [16-23].

The aim of the present study was to assess the postprandial effects of beer consumption on arterial function/structure and brachial/aortic pressure, as well as, to investigate the role of beer's constituents (alcohol and antioxidants) on these parameters. For that purpose, we compared in a single-blind cross-over study, the effects of regular beer, dealcoholized beer and vodka (containing comparable amount of antioxidants or alcohol) on endothelial function, aortic stiffness, pressure wave reflections and aortic pressure in apparently healthy non-smokers male volunteers.

Materials and Methods

Study population

Apparently healthy, non-smokers, male volunteers were invited to participate in the study though a university advertisement and word of mouth. Exclusion criteria were: medical history of coronary artery disease, diabetes mellitus, liver or endocrine diseases, smoking, alcohol consumption more than the recommended amount (20-30 gr alcohol/day), vigorous exercise, antioxidant vitamin supplementation and dieting at the time of the study. The study complies with the Declaration of Helsinki, and was approved by the Laikon Hospital scientific committee; all subjects gave their informed consent before entering the study.

Experimental protocol

We conducted a randomized, single – blind, cross - over study, which comprised of three study days with at least one-week interval between. All vascular and hemodynamic tests were performed at the Cardiovascular Research Laboratory (Laiko Hospital) in the morning hours (at 8:00) and after 10-12 hours fast and absence of alcohol, coffee and caffeinated drinks since noon of the previous day. Each study day, after an 10 min resting period in the supine position in a quiet room with a temperature controlled at 20–25°C, ultrasonography and arterial tonometry for assessing endothelial function, arterial stiffness, pressure wave reflections and aortic blood pressures were performed. Afterwards, all volunteers consumed in a randomized order either a) 400 ml of beer & 400 ml water, b) 800 ml of dealcoholized beer (same amount of polyphenols), or c) 67 ml of vodka & 733 ml water (same amount of alcohol) within 15 minutes; the test drinks were accompanied with a sandwich (2 slices of white bread, 1 slice of turkey and 1 slice of low fat cheese). The above mentioned drinks were matched for their antioxidant and alcohol content, namely 400 ml of beer had the same antioxidant concentration with 800 ml of dealcoholized beer (∼48mg polyphenols) and 400 ml of beer had the same alcohol concentration with 67 ml of vodka (∼ 20 g of ethanol). Addition of water enabled us to have the same quantity of fluids in all test drinks (∼800 ml). All the baseline measurements were repeated at 1 and 2 hours postprandially. The same trained observer who was blinded for the type of intervention performed all measurements. All volunteers were instructed to avoid significant changes in their physical activity and their diet between the 3 visits. They were also advised to consume the same quality and quantity of food one day before the study days. At each study day, 24 hour recalls, for the previous day, were taken in order to ascertain the alignment of the subjects to the instructions concerning their diet.

Arterial and hemodynamic measurements

Brachial blood pressure was assessed with Macrolide BP Office device; the average of three consecutive recordings was used in the statistical analysis and the following pressure waveform calibration process. A B-Mode high-resolution ultrasound imaging (Vivid 7 Pro, GE) was used for assessment of FMD, as previously described [9], in order to evaluate endothelial function. Radial artery and femoral artery tonometry were used to assess aortic stiffness via the carotid to femoral PWV, as suggested by recent guidelines [16], with Sphygmocor System (Actor Medical, Sydney, Australia). The Aix was also assessed, as an index of pressure wave reflections, as previously described [21] and normalized for the heart

rate of 75bpm (AI@75) due to the strong dependence of this index on heart rate. Finally, pulse pressure amplification was assessed as the ratio of brachial/aortic PP, as previously suggested [17].

Statistical analysis

Postprandial differences in mean changes from fasting (0 h) were analyzed using 2-way repeated-measures ANOVA in SPSS (version 17.0; SPSS Inc., Chicago, IL, USA), with meal treatment and time as within-subject factors. To account for day-to-day variability, measurements following the test meals were compared by using the changes from fasting values. Greenhouse-Geisser correction for sphericity was employed. Specific comparisons between treatments were adjusted using the Bonferroni correction factor when there was a significant meal effect or meal × time interaction effect. Differences were considered significant at $P < 0.05$. Values in the results are mean (95% CI), unless otherwise specified.

Results

Seventeen healthy male subjects enrolled and completed the study. Their mean age was 28.5±5.2 years and their body mass index (BMI) 24.4±2.5 kg/m^2 (Table 1). Data from 16 subjects were available for the final analysis due to missing values related to poor quality of vascular recordings in at least one of the visits.

Table 2 shows the changes in PWV, AIx, and FMD as well as in the baseline brachial artery diameter. PWV was significantly (time effect: p<0.001) and similarly (i.e. meal effect was non-significant) reduced by all 3 test drinks, by an average of 0.40 m/sec (95% CI: 0.30, 0.50; $P< 0.001$), from a mean fasting value of 5.7 m/sec (95% CI: 5.3, 6.1). AIx significantly decreased after all 3 test drinks (time effect: p<0.001). Difference between them was observed (meal effect: p=0.011); the reduction was significantly greater following beer consumption compared to dealcoholized beer by 6.3% (95% CI: 1.0, 11.6; $p = 0.018$) and tended to be greater after vodka consumption compared to dealcoholized beer by 5.8% (95% CI: -0.7, 12.3, $P = 0.088$). A significant difference between the 3 test drinks was observed for the postprandial changes in FMD (meal effect: $p = 0.044$). FMD increased after beer over the 2-h postprandial period by 1.3% (95% CI: 0.1, 2.5; $P< 0.05$) and did not change after dealcoholized beer or vodka; multiple comparisons testing between beer and the other test drinks did not reach significance. No interactions between time and meal effect was observed for PWV, Aix and FMD. Baseline brachial artery diameter significantly increased following the test drinks (time effect: p<0.001); differences between them was observed (meal effect: p<0.0010. Beer and vodka ($P< 0.001$) but not after dealcoholized beer (beer *v*. dealcoholized beer, $P < 0.025$; vodka *v*. dealcoholized beer, $P < 0.001$) increased the baseline diameter.

Table 3 shows the changes in heart rate and BP (aortic and brachial BP and pulse pressure amplification) at 1 and 2 h following the test drinks compared with fasting values. Heart rate increased (time effect: p=0.035) over the 2-h postprandial period following all test drinks by 1.4 bpm (95% CI: 0.3, 2.5; $P< 0.025$) and with a tendency for drinks to differ in their responses but this did not reach significance (interaction meal × time effect, $P = 0.064$).

Mean arterial pressure similarly fell over the 2-h period following the 3 drinks (time effect: p<0.001) but no differences between the test drinks were observed (meal effect: non-significant). Aortic and brachial pulse pressure did not significantly change after the test drinks. Pulse pressure amplification significantly increased following the 3 test drinks from a mean fasting of 1.5% (95% CI: 1.5, 1.6) by an average of 0.08% (95% CI: 0.06, 0.11; $P < 0.001$); there was a tendency for meal and meal × time interaction effects to reach significance ($P = 0.059$, $P = 0.055$, respectively) for the changes in pulse pressure amplification.

Discussion

In this study we examined the postprandial effects of beer, dealcoholized beer and vodka, on endothelial function (FMD), aortic stiffness (PWV), pressure wave reflections (Aix) and aortic/brachial pressure. In order to test the effects of beer's constituent (alcohol and antioxidants) we matched the quantity of antioxidants in the regular and dealcoholized beer and the quantity of alcohol in the beer and vodka. Beneficial effects were observed in all arterial biomarkers (PWV, Aix, FMD) by all test drinks, however beer improved endothelial function more than the other two test drinks and reduced pressure wave reflections (and by that improve improves/increases pressure amplification) more than the dealcoholized beer. These findings suggest that consumption of regular beer has a measurable acute beneficial effect on these cardiovascular biomarkers that seems to be mediated by the “synergistic” action of both alcohol and antioxidants.

Assessment of arterial stiffness through measurements of PWV is a significant predictor of cardiovascular disease events and mortality [10, 11]. In the present study, all test drinks decreased PWV indicating that the observed findings could potentially be attributed to both ethanol and antioxidants found in beer. The underlying mechanism may be the vasodilation as well as the beneficial effect on endothelial function, both observed in this study. The possible postprandial effects of ethanol and antioxidants found in alcoholic drinks have not been well investigated. In one study including 8 healthy volunteers, red wine (0.8gr/kg body weight) reduced PWV, while dealcoholized red wine had no such effect, suggesting that only ethanol but not red wine antioxidants had a significant beneficial effect on arterial stiffness [12]. To our knowledge there is no previous study investigating the acute effects of antioxidants found in alcoholic drinks, in arterial stiffness. However, in a study of healthy volunteers it was shown that antioxidants found in black tea may have beneficial postprandial effects on PWV [13], supporting the hypothesis that antioxidants in drinks might favorably affect arterial stiffness postprandially.

AIx is complex marker reflecting the augmentation of blood pressure due to returning reflected waves from distal sites of the circulation [14]. All test drinks decreased AIx postprandially, but beer's effect was significantly greater than that of the dealcoholized beer, and almost similar with vodka. Thus, both ethanol and antioxidants seem to reduce pressure wave reflections, but the contribution of ethanol seems to play the leading part in reducing AIx. This is in accordance with previous studies in red wine, which however have shown that both ethanol and antioxidants may reduce AIx postprandially [12, 15-17]. However, beer antioxidants differ from those found in red wine [5], which is a possible explanation for

the divergent findings. AIx depends both on heart rate and the magnitude of the reflected wave [14]. In the present study heart rate increased significantly yet only by 1-2 bpm; this magnitude of change cannot explain the observed change in Aix since an increase by 10bpm induces Aix reduction by 4% [17]. We thus hypothesize that the observed findings in wave reflections are primarily mediated by the recorded peripheral vasodilation i.e. the reduction of the arterial reflection coefficients and the magnitude of wave reflections [17]. The exact arterial site of action cannot be determined by the present study, however, both function of conduit arteries (assessed through brachial diameter increase) as well as the microcirculation (assessed through mean blood pressure reduction/and thus total peripheral resistance decrease) may play part.

Pulse pressure amplification is a novel mechanical biomarker of the cardiovascular system, which is associated with both arterial stiffness and wave reflections, as well as to heart rate and the classical cardiovascular risk factors [17]. It quantifies the fact that brachial and aortic pressure responds differently in drugs and vasoactive substances [17, 30]. Recent data suggest that is also strongly associated with cardiovascular mortality [31]. In the present study it was evidenced that beer consumption may have a beneficial effect on pulse pressure amplification (i.e. to increase). This effect may be mediated by both PWV and AIx reduction, as well as heart rate increase.

Finally, this study showed a differential effect of beer, dealcoholized beer and vodka on endothelial function as assessed by FMD. Although no pairwise comparisons between groups reached significance due to the small sample size of the study, only beer increased FMD (improved endothelial function), whereas the other two test drinks reduced FMD. It has been shown that ethanol and antioxidants found in alcoholic drinks, like red wine, improve endothelial function expressed as increases in FMD [3]. There are no relevant studies concerning the possible acute effects of beer consumption, but in a similar study beer improved endothelial function by increasing reactive hyperemia postprandially [18]. The present data are in line with studies showing that both ethanol and antioxidants (in red wine) modulates NO synthase and promotes NO secretion/production. However, red wine and beer have significant qualitative differences in their antioxidant constituents and thus the extrapolation of these data needs further verifications.

The present study has limitation since no molecular and biochemical mechanisms have been investigated. Moreover, even though this is a cross over design, the sample size of the study is rather small. However we managed to detect significance in arterial properties even after taking into account the baseline (i.e. between the 3 visits) fluctuations of the biomarker, since we analyzed not only the differences but took into account the relative change (from baseline values) between groups. However, our findings cannot be extrapolated to women or subjects with pathological conditions. Further studies are needed to investigate the effects of beer on these populations.

These data suggests that 400ml of beer, acutely improve arterial properties in apparently healthy men and both the alcohol and antioxidant contents may have a significant contribution. Prospective long-term studies in larger samples are needed to confirm these data and evaluate whether these beneficial effects are translated in clinical benefit.

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K.K.: conception and design of the study, analysis and/or interpretation of data, drafting/revision of the manuscript, approval of the final version of the manuscript

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Table 1
Demographics of the 17 healthy male volunteers

	Mean	SD
Age (y)	28.4	5.2
Weight (kg)	77.5	8.0
BMI (kg/m^2)	24.3	2.4
Body fat (%)	15.7	5.0
Waist circumference (cm)	83.2	5.0
Systolic BP (mmHg)	115.4	6.2
Diastolic BP (mmHg)	68.5	5.4

BP: blood pressure, BMI: body mass index.

Table 2

Changes in vascular measurements after beer, dealcoholized beer and vodka consumption from fasting (0 h) at 1h and 2 h

	Beer		Dealc beer		Vodka		Meal effect*	Time effect**	Meal × time effect*
	Mean	**95% CI**	**Mean**	**95% CI**	**Mean**	**95% CI**			
PWV (m/s)									
Fasting (0 h)	5.7	5.3, 6.0	5.8	5.4, 6.1	5.7	5.3, 6.1			
Δ (1 h – 0 h)	-0.4	-0.6, -0.3	-0.2	-0.4, -0.1	-0.4	-0.6, -0.2	*NS*	$P < 0.001$***	*NS*
Δ (2 h - 0 h)	-0.5	-0.7, -0.4	-0.3	-0.4, -0.1	-0.5	-0.7, -0.3			
AIx (%)[a]									
Fasting (0 h)	-2.4	-6.8, 2.1	-5.1	-8.6, -1.6	-4.8	-9.7, 0.1			
Δ (1 h – 0 h)	-9.18	-12.3, -6.1	-3.8	-7.6, 0.1	-8.4	-11.3, -5.4	$P = 0.011$	$P < 0.001$	*NS*
Δ (2 h - 0 h)	-8.94	-13.0, -4.9	-1.7	-4.9, 1.4	-8.7	-13.2, -4.3			
Baseline diameter (mm)[a,b]									
Fasting (0 h)	3.9	3.7, 4.2	3.9	3.7, 4.2	3.9	3.6, 4.2			
Δ (1 h – 0 h)	0.3	0.2, 0.4	0.1	-0.0, 0.1	0.3	02, 0.5	$P < 0.001$	$P < 0.001$	$P = 0.02$
Δ (2 h - 0 h)	0.3	0.1, 0.4	0.0	-0.0, 0.1	0.4	0.3, 0.5†			
FMD (%)[c]									
Fasting (0 h)	2.5	1.4, 3.5	4.7	3.0, 6.3	3.9	2.8, 5.1			
Δ (1 h – 0 h)	1.7	0.3, 3.0	-1.3	-3.3, 0.8	0.1	-1.5, 1.8	$P = 0.044$	*NS*	*NS*
Δ (2 h - 0 h)	1.0	-0.4, 2.4	-0.7	-2.5, 1.1	0.2	-0.7, 1.1			

PWV: carotid-femoral pulse wave velocity; *NS*: non-significant; AIx: augmentation index; FMD: flow-mediated dilatation.

*Repeated measures ANOVA of the changes at 1 h and 2 h from fasting (0 h).

**Repeated measures ANOVA of the absolute values at 0 h, 1 h and 2 h (for time changes).

***the time effect remained significant after adjustment for MBP

*a,b,c*Bonferroni's correction applied when meal effect was significant:

*a*beer *v*. dealc.beer, $P < 0.025$;

*b*vodka *v*. dealc.Beer, $P < 0.001$;

*c*no pairwise comparisons reached significance for FMD.

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†,‡Bonferroni's correction applied when meal × time effect was significant:

† *v.* dealc.beer, $P < 0.05$;

‡ *v.* beer, $P < 0.05$.

Table 3

Changes in heart rate and arterial pressure after beer, dealcoholized beer and vodka consumption from fasting (0 h) at 1h and 2 h

	Beer		Dealc.beer		Vodka		Meal effect*	Time effect**	Meal × time effect*
	Mean	95% CI	Mean	95% CI	Mean	95% CI			
Mean arterial pressure (mm Hg)									
Fasting (0 h)	83.2	79.8, 86.7	82.1	79.1, 85.1	83.9	80.6, 87.2			
Δ (1 h – 0 h)	-3.8	-7.0, -0.6	-2.0	-4.0, 0.02	-1.7	-3.5, 0.03	*NS*	*P* <0.001	*NS*
Δ (2 h - 0 h)	-3.3	-6.4, -0.3	-3.7	-6.3, -1.1	-5.0	-8.0, -2.1			
Peripheral pulse pressure (mm Hg)									
Fasting (0 h)	45.8	42.4, 49.3	47.7	44.3, 51.1	47.6	44.7, 50.4			
Δ (1 h – 0 h)	3.1	0.4, 5.7	2.75	-0.5, 6.0	0.4	-2.5, 3.4	*NS*	*P* = 0.060	*NS*
Δ (2 h - 0 h)	3.2	0.4, 5.9	0.2	-2.6, 3.0	0.8	-1.9, 3.5			
Central pulse pressure (mm Hg)									
Fasting (0 h)	30.0	27.5, 32.5	30.7	28.5, 33.0	31.0	28.9, 33.1			
Δ (1 h – 0 h)	-0.0	-1.6, 1.5	0.5	-1.6, 2.5	-1.4	-3.4, 0.7	*NS*	*NS*	*NS*
Δ (2 h - 0 h)	-0.3	-2.4, 1.8	-0.5	-2.3, 1.4	-1.4	-3.4, 0.6			
Pulse pressure amplification (%)									
Fasting (0 h)	1.5	1.5, 1.6	1.6	1.5, 1.6	1.5	1.5, 1.6			
Δ (1 h – 0 h)	0.1	0.04, 0.15	0.1	0.03, 0.1	0.08	0.04, 0.12	*P* = 0.059	*P* <0.001	*P* = 0.055
Δ (2 h - 0 h)	0.1	0.1, 0.2	0.03	0.00, 0.06	0.1	0.06, 0.14			
Heart rate (bpm)									
Fasting (0 h)	60.0	55.7, 64.3	57.9	54.7, 61.1	57.3	52.5, 62.1			
Δ (1 h – 0 h)	1.3	-1.2, 3.8	2.0	-0.8, 4.8	1.6	-0.2, 3.3	*NS*	*P* = 0.035	*P* = 0.064
Δ (2 h - 0 h)	2.2	0.5, 3.8	-0.3	-2.3, 1.8	1.6	-0.1, 3.3			

*Repeated measures ANOVA of the changes at 1 h and 2 h from fasting (0 h).

**Repeated measures ANOVA of the absolute values at 0 h, 1 h and 2 h (for time changes).