

## Research Article

# Alcohol Intake and Growth Rate of Abdominal Aortic Aneurysms in Men – Results of a Prospective Population - Based Cohort Study of a Randomized Screening Trial

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**Abstract**

**Objective:** To investigate the association between alcohol intake and the progression of abdominal aortic aneurysms (AAAs) in large Danish population-based randomised screening trial. We hypothesised that moderate amounts of alcohol decreased the growth rate of AAAs.

**Design:** Cohort study of a population-based prospective randomised screening trial.

**Methods:** Standardised ultrasound scans measurement of maximum antero-posterior infrarenal aortic diameter. If an AAA was found at baseline, men were invited to annual ultrasound surveillance for up to five years. Simple and multiple linear regressions with potential confounders based on an automated empirical procedure. Post hoc power calculation.

**Materials:** 25 083 men were assigned for baseline, triple screening which included evaluation of AAAs, peripheral arterial disease (PAD) and hypertension. Questionnaires provided information regarding alcohol intake, co-morbidity and use of drugs.

**Results:** The prevalence of AAA was 3.3%. One unit increase in alcohol intake increased the growth rate by 4.3% (CI -0.9 to 9.8) in crude analysis and by 3.1% (CI -1.64 to 8.05) in adjusted analysis. Increase in one unit of alcohol changed the hazard ratio for the AAA need for repair by 1.03 (CI 0.91-1.16). Post hoc power calculation revealed coefficient R at 80% power, 5% significant level and a sample size of 417 is 0.134.

**Conclusion:** Our study did not find the protective effect of alcohol. The risk of an AAA progressing increased with every additional unit of alcohol, although, no statistically significant associations were exposed.

**ABBREVIATIONS**

**AAA:** Abdominal Aortic Aneurysm; **CVD:** Cardiovascular Disease; **PAD:** Peripheral Artery Disease

**INTRODUCTION**

Abdominal aortic aneurysm (AAA) is a disease with a pathological dilatation of the aorta with a prevalence of up to 2% among people above 50 years [1-3].

If the AAA is less than 5.0 cm at the time of diagnosis, people are referred to ultrasound surveillance, every three years for aneurysms 3–3.9 cm in diameter, annually for aneurysms 4.0–4.9 cm, and every 3–6 month for aneurysms  $\geq$  5.0 cm every 6-12 months. Surgical repair is offered to people when the AAA exceeds 5.5 cm in men and may be considered for women when AAA exceeds 5.0 cm [4].

Over the last 20 years, studies have been trying to identify risk factors for AAA and found that age, smoking, male sex, family

history of AAA, atherosclerotic disease, and hypertension are risk factors for developing AAAs [5-8]. It has been consistently suggested that moderate consumption of alcohol has a protective effect on coronary artery disease [9,10]. Although, AAA is no longer considered solely an atherosclerotic disease of the aorta but rather as inflammation and proteolytic degradation of connective tissue [11], there have been attempts to analyse the protective effect of alcohol on AAAs. A study by Stackelberg et al., suggested that moderate consumption of alcohol was associated with a lower risk of developing AAA, extending alcohol's protective effect to AAA [12].

Studies have described light to moderate amounts of alcohol being able to decrease the major pathophysiological components of AAA such as systematic inflammation and oxidative stress [13,14]. Studies also suggest that alcohol has a protective effect on endothelial function, elevation of high-density lipoprotein (HDL), cholesterol [15,16].

Even though, alcohol has been studied for protective effect against development of AAAs, the effect of alcohol on growth of AAAs remains unknown.

The aim of this study was to investigate the association between alcohol intake and the growth rate of AAAs. Our objectives were to estimate the association between the growth rate of AAA and alcohol intake in men between 65-74 years. Secondly, to estimate the association between the need for later AAA repair and alcohol intake in the same population. Our primary hypothesis was that moderate amounts of alcohol decrease the growth rate of AAAs.

## MATERIALS AND METHODS

This cohort study was based on data from VIVA (Viborg Vascular) trial, a population-based randomized screening trial of 50 170 men aged 65-74 in the Central Denmark Region enrolling from October 2008 until January 2011 [17,18]. Study participants were randomly assigned 1:1 to triple screening or no systematic screening. 25 083 men were assigned for baseline, triple screening which included evaluation of AAAs, hypertension and peripheral arterial disease (PAD). Specially trained nurses performed ultrasound scans of the infrarenal abdominal aorta. Using the cinematic function, the maximal systolic inner-to-inner anterior-posterior diameter was measured [17]. If an AAA was found at baseline, men were invited to annual ultrasound surveillance in up to five years, if an AAA exceeded 50 mm, the patient was not included in the study and were referred to a vascular surgeon.

### Definitions

We used self-reported alcohol consumption derived from a questionnaire, which was given at each surveillance visit and we calculated the average use during the observation period. Alcohol consumption was defined as units per day. One unit was 8 grams of alcohol. Moderate consumption was defined up to one drink (1.75 units) per day for women and two drinks (3.5 units) per day for men [19]. AAA was defined as the infrarenal anterior-posterior (AP) measurement  $\geq 30$  mm. Growth rates were calculated based upon the ultrasound scans from the baseline and annual follow-up visits using individual linear regression between AP diameter

and time. Need for repair of the abdominal aorta was defined as undergoing surgery when the aorta diameter exceeded 50 mm within the time of follow-up.

### Inclusion

Men were included if they had at least two measurements of the abdominal aorta and had answered a questionnaire about lifestyle parameters including alcohol use.

### Analyses

The primary objective was to investigate whether the growth rate of AAAs was associated with alcohol use. The association between AAA and alcohol use was estimated using both simple and multiple linear regressions.

The following pre-specified subanalysis was made:

We estimated the need for later repair of AAA and the association with intake of alcohol using simple and multiple cox regression to calculate hazard ratio (HR), and as a sensitivity analysis, we divided the use of alcohol into three groups.

Normal distribution of data was assessed using graphical methods, and consequences were taken using relevant logarithmic transformation in the analyses. Furthermore, we made several graphical assessments of the data to conclude if we had a linear, quadratic or perhaps cubic association between growth rate and alcohol consumption as to find the best model for the main analysis. The linear trend seemed to have the overall best fit. Group comparisons were made using relevant either parametric or nonparametric tests.  $P < 0.05$  was considered statistically significant. The 95% confidence intervals (CIs) are given in brackets with the corresponding odds ratio. Post hoc power analysis was performed.

### Potential confounders

Potential confounders were selected based on an automated empirical procedure. For each of demographic variables, we calculated the estimated growth rate of AAA in relation to a history of alcohol consumption with or without the potential confounder included. If the estimate changed by more than 5% in either direction by including the potential confounder, we included the variable in our adjusted analysis. Thus, age, smoking, and baseline aorta diameter were included as confounders.

## RESULTS AND DISCUSSION

### Results

The VIVA trial consisted of 25 083 men assigned to a baseline screening, with an attendance rate of 74.7% [20]. At baseline, 619 men were found to have AAAs, corresponding to prevalence of 3.3% in the screened population. 103 men were referred for vascular evaluation due to large aneurysms, 65 were lost to follow-up or did not provide information about alcohol consumption, and thus 451 were included in our cohort study for follow-up and analyses. The median age was 69, 322 men (71.4%) reported daily intake of alcohol and median follow-up was 4.5 years (interquartile range 3.9-5.0). Average daily alcohol intake ranged from 1 to 2 units of alcohol (approximately 8 to 16 grams of pure alcohol) with a median intake of one unit. See Table 1 for demographics.

**Table 1:** Demographics.

	AAA	Alcohol intake	No alcohol intake	P-value
<b>Total</b>	(n=451)	(n=322)	(n=129)	
<b>Age</b>	69 (67-72)	69 (67-72)	69 (67-72)	0.894
<b>Family history of AAA</b>	31 (6.9%)	22 (6.8%)	9 (7.0%)	1.000
<b>Daily consumption of alcohol, units</b>	1 (0-2)	1 (1-2)	0 (0-0)	
<b>Smoking</b>				0.762
Current	187 (41.5%)	129 (40.1%)	58 (45.0%)	
Former	221 (49.0%)	160 (49.7%)	61 (47.3%)	
Never	35 (7.8%)	26 (8.1%)	9 (7.0%)	
<b>Body mass index</b>	27 (24-29)	26 (24-28)	27 (25-30)	0.006
<b>Diastolic blood pressure, mm Hg*</b>	86 (79-94)	86 (78-93)	88 (80-95)	0.182
<b>AAA measure, mm*</b>	36 (32-41)	35 (32-40)	37 (33-42)	0.222
<b>Self-reported comorbidity</b>				
Hypertension	241 (53.4%)	175 (54.3%)	66 (51.2%)	0.463
AMI	28 (6.2%)	17 (5.3%)	11 (8.5%)	0.204
COPD	16 (3.5%)	12 (3.7%)	(n<5)	1.000
PAD	132 (29.3%)	89 (27.6%)	43 (33.3%)	0.358
Diabetes	42 (9.3%)	25 (7.8%)	17 (13.2%)	0.106
<b>Current use of medication</b>				
Antidiabetics	29 (6.4%)	16 (5.0%)	13 (10.1%)	0.058
Statins	238 (52.8%)	173 (53.7%)	65 (50.4%)	0.399
ACE inhibitor	113 (25.1%)	77 (23.9%)	36 (27.9%)	0.473
Angiotensin II antagonists	50 (11.1%)	39 (12.1%)	11 (8.5%)	0.320
Beta blockers	116 (25.7%)	86 (26.7%)	30 (23.3%)	0.473
Acetylsalicylic acid	216 (47.9%)	155 (48.1%)	61 (47.3%)	0.916
NSAIDs	18 (4.0%)	11 (3.4%)	7 (5.4%)	0.424

**Abbreviations:** \*Numbers are median with interquartile range in brackets.  
AAA: Abdominal Aortic Aneurysm; AMI: Acute Myocardial Infarction;  
COPD: Chronic Obstructive Pulmonary Disease; PAD: Peripheral Arterial Disease; ACE inhibitors: Angiotensin-Converting-Enzyme inhibitors; NSAID: Nonsteroidal Anti-Inflammatory Drugs

Baseline median measurements of the infrarenal aorta were 35 mm versus 37 mm in the alcohol consuming and non-consuming groups respectively,  $p=0.22$ . Analysing correlation between baseline aortic measurements and units of alcohol, we found Spearman's rho of  $-0.03$ ,  $p=0.47$ .

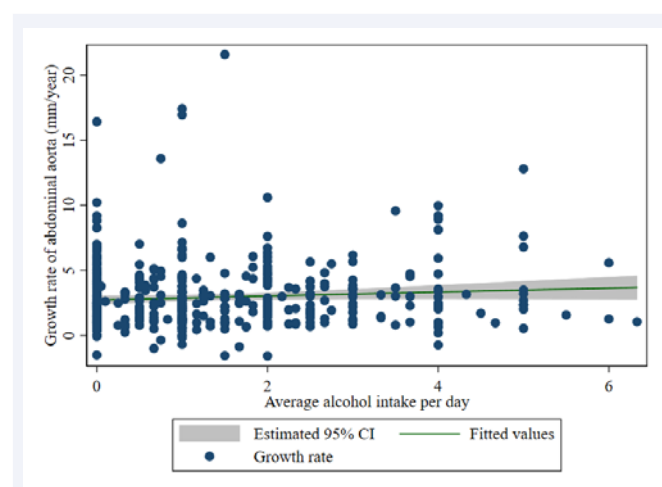
Analysing the association between the aneurysmal growth rate and intake of alcohol, we found in our crude analysis that one unit increase in alcohol intake increased the growth rate by 4.3% (CI  $-0.9$  to 9.8). After adjusting for confounders, we found that a neurysmal growth increased by 3.1% (CI  $-1.64$  to 8.05) for every increase of alcohol unit. Baseline aorta was statistically significant in our adjusted model,  $p<0.000$ , but age and smoking status did not affect the estimate statistically significant. See Figure 1 for scatter plot of the average daily alcohol intake and the growth rate of AAA.

At the end of the follow-up, 191 men had undergone surgical repair due to a large AAA. Analysing the need for repair as a time-to-event analysis, we found in our crude analysis, that one unit of alcohol changed the (HR) by 1.03 (CI 0.91-1.16). Multiple analysis showed us a similar result of one unit of alcohol decreasing the HR by 0.92 (CI 0.82-1.03). Baseline aorta increased HR statistically significant ( $p<0.000$ ). As a sensitivity analysis, we divided the use of alcohol into groups; group 1 with no alcohol intake, group 2 1-2 units per day, and group 3  $\geq 3$  units per day. Interestingly, we found an increase in crude HR 1.02 (CI 0.70-1.59) for group 2 compared with group 1, and a decrease in HR 0.80 (CI 0.46-1.42) for group 3 compared with group 1. In the adjusted analysis, we found a similar decrease comparing group 2 with group 1, HR 0.90 (CI 0.62-1.32) and increase for group 3 compared with group 1, HR 1.09 (CI 0.63-1.85), but this was not statistically significant.

Finally, consumption of alcohol was grouped by consumers versus none-consumers or above and below the 50th percentile. The growth rates were 2.35 mm/year below the 50<sup>th</sup> percentile and 2.46 above,  $p=0.65$ . For the non-consumers vs. consumers, the growth rate was 2.35 mm/year and 2.46 mm/year,  $p=0.67$ .

## Discussion

In this large, prospective population-based cohort study of men aged 65-74, we analysed the association between alcohol intake and growth rate of AAAs. We did not find a statistically significant association and thus we were not able to prove our hypothesis of alcohol having a protective effect against AAA growth. We observed an increased annual growth rate of AAA



**Figure 1** Scatter plot of the growth rate of abdominal aortic aneurysms and the average daily alcohol intake with a fitted line.

with every additional alcohol unit intake by 4.3% (CI -0.9 to 9.8) and 3.1% (CI -1.64 to 8.05) for crude and adjusted analysis, respectively, but this was not statistically significant.

To the best of our knowledge, this is the first study exploring the association between the intake of alcohol and the progression of AAAs. Previous studies have analysed the association of developing AAA and alcohol intake without reaching consistent results. A prospective study by Wong et al. found that prevalence of AAA increased with highest levels of alcohol intake— more than 4 units per day -- with a hazard ratio of 1.65 (CI 1.03- 2.64) [21]. The results of this study are somewhat comparable with our results as the authors did not find a protective effect of alcohol but instead found the potential harms of intake. However, the cohort described by Wong et al. differs as they excluded 4 461 subjects with a history CVD including myocardial infarction, angina, coronary artery bypass grafting surgery, stroke, or transient ischemic attack. Their subjects were as well excluded during the follow-up if they were newly diagnosed with as well as a diagnosis of hypertension, hypercholesterolemia, and diabetes preceding the diagnosis of AAA [21]. Excluding these subjects could lead to a different prevalence of AAA, as most patients diagnosed with AAA have previously been diagnosed with CVD [22]. In contrast, our study was based on a randomized population-based screening trial, allowing us to minimize the risk of selection bias. It is also important that some of the studies reporting on alcohol and risk of developing AAA, based their AAA diagnosis on self-reported diagnosis of AAA or medical records of a participant admitted to hospital during the study period with an aneurysm that was symptomatic or required surgical intervention [12,21,23]. Other population-based randomized control trials have reported AAA prevalence rates of 4–7.2% on ultrasound based screenings [24,25]. These prevalence rates potentially suggest that AAAs due to their asymptomatic nature could be under-reported with self-reported diagnosis. One of the studies basing AAA diagnosis on self-reported diagnosis was carried out by Stackelberg et al., [12]. They found contradicting results to previous studies that moderate consumption of up to 15 units of alcohol per week among men and 7.5 units alcohol per week among women reduced the risk of developing AAA with a hazard ratio of 0.80 (CI 0.68–0.94) and 0.57 (CI 0.40–0.82), respectively [12]. This result is consistent with research on alcohol having a protective effect against other CVDs [13,14]. However, it is worth mentioning that the study by Stackelberg et al. did not include non-drinkers in their dose-response analysis, suggesting that the characteristics of non-drinkers to develop AAAs are different [12]. Furthermore, by only including clinical events of AAA (incident diagnosis, rupture or death resulting from AAA), the prevalence of AAA is underestimated as asymptomatic AAA are not included and the actual association might be different. A recent meta-analysis by Spencer et al. analysed alcohol intake and the risk of developing AAA [26]. The analysis comprised of nine studies analysing 11 unique study populations and found statistically significant non-linear dose response curves with decreased risk of developing AAA with lower levels of intake of up to 1-2 units a day ( $p=0.05$ ) [26]. These results should be taken with some caution since between-study heterogeneity was high due to some studies reporting on a selected population i.e. Törnwall et al., reporting on male smokers [23], Lederle et al., reporting solely in women

[27], and Wong et al., reporting on a cohort without known CVD [21]. Although the under-lining mechanism remains a matter of investigation, male sex and smoking are well-established risk factors for AAA development, influencing the prevalence of AAA across the general population. Studies that include only women or smokers do not have external validity [28]. Finally, all of these studies investigated the relation of alcohol and the risk of developing an AAA, while our study aimed to explore the association between AAA growth and alcohol intake.

Our study has some key strength. Our cohort study is based on a large population-based randomized study with a high attendance rate, which minimizes the risk of selection bias.

Moreover, all men assigned to screening underwent ultrasound measurement of the abdominal aorta by specially trained nurses using a strict standardized method. These measurements previously showed low inter-observer variability thus ensuring us with precise diagnosis of AAA [20]. Questionnaires provided data on lifestyle parameters are not available from national registers such as alcohol intake but also smoking which is important when analysing AAA since it is a known risk factor both in development but also in growth [29,30].

Our main limitation is the self-reported alcohol intake. Studies reporting on alcohol intake and risk of developing AAA also previously mentioned the possibility of under-reporting alcohol intake by the participants. [5,12,21,23]. This could happen due to participants not wanting to disclose their alcohol history. Without the possibility of objective measurements of alcohol intake of a longer period, the most informative source remains self-reported intake. Even though large aneurysms were not

## CONCLUSION

Although no statistically significant associations were exposed, a positive association between alcohol intake and the natural history of AAA was noticed. This study is the first analysing alcohols effect on the growth of AAA. Our results suggest that alcohol could have a different effect on growth than it has on development. In addition, more research to evaluate alcohol's role on AAA growth is warranted to further elaborate on this association and the pathophysiologic mechanisms.

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