Journal of Vascular Medicine & Surgery
ISSN: 2329-6925

Green et al., J Vasc Med Surg 2014, 2:3
DOI: 10.4172/2329-6925.1000140

Systematic Review Open Access

Alcohol Consumption as a Risk Factor for Abdominal Aortic Aneurysm

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Abstract

Introduction: Abdominal aortic aneurysm (AAA) represents a major cause of death in the over 65 age group. Current evidence suggests that AAA development may be due to an immune mediated inflammatory response leading to degradation of the extracellular matrix, increased biomechanical wall stress and resultant aortic dilatation. Modifiable risk factors include hypertension and smoking; however the potential role of alcohol remains unclear.

Methodology: The electronic databases EMBASE, Pubmed, Medline and Web of Science were searched using key word search terms in conjunction with Boolean operators based on the PRISMA recommendations ('Ethanol' OR 'alcohol') AND ("aneurysm" OR 'abdominal aortic aneurysm' OR 'AAA'). Articles considering an association between alcohol and patients with and without AAA were included, based on title, keyword and abstract screen. No limitation was imposed by year, methodology or language. Reference lists of included studies and pertinent journal contents were hand searched for additional suitable studies.

Results: A total of eight articles were identified for inclusion, the majority of which were retrospective and prospective cohort studies. Five of the studies reported a positive association between alcohol and AAA; however one reported a loss of association following adjustment for confounders, including smoking. Three further studies reported no association, although in two Scandinavian studies, alcohol consumption was considerably lower compared with those reporting a positive association.

Conclusion: Existing evidence is limited but may suggest a link between high levels of alcohol consumption and AAA development, whilst moderate consumption may confer some protection. Further epidemiological study is required.

Keywords: Ethanol; Alcohol; Abdominal aortic aneurysm

Introduction

Abdominal Aortic Aneurysm (AAA) is defined as a pathological dilatation of the abdominal aorta to greater than 1.5 times its original diameter [1]. AAA affects 4-8% of adults aged over 65 years, and represents a significant cause of mortality in this age group [2,3]. The underlying pathogenesis is complex and is thought to involve an immune-mediated inflammatory response with macrophage and lymphocyte infiltration. Subsequent degradation of the extracellular matrix by proteases, with vascular smooth muscle cell apoptosis eventually leads to gradual dilatation of the abdominal aorta and formation of AAA [4]. Although there is likely to be a genetic component to AAA [5], aside from family history, the key clinical risk factors of increasing age, hypertension and smoking are the most widely established contributors affecting aneurysm development and growth [6-10]. Notwithstanding, women have an increased risk of AAA rupture [11]. Alcohol consumption is an established cardiovascular risk factor, however, the role of alcohol intake in the development and progression of AAA is less well defined.

Excessive alcohol consumption up-regulates matrix metalloproteinase (MMP) activity [12] in many anatomical sites, including the aorta in a rat model of alcoholism [13]. The complex dose dependent relationship between alcohol intake and cardiovascular disease is well established [14,15]. Whether this paradigm also applies to AAA is unknown. The development of a national AAA screening programme (NAAASP; www.aaa.screening.nhs.uk) in the UK offers an opportunity for targeted secondary prevention in patients with AAA and it is therefore timely to review the current clinical evidence regarding the role of alcohol consumption as a risk factor for AAA.

Methods

Search strategy

The electronic databases EMBASE, Pubmed, Medline and Web of Science were interrogated for relevant articles regarding alcohol and AAAs in April 2013. The following key word search terms were utilised in conjunction with Boolean operators: ('Ethanol' OR 'alcohol') AND ("aneurysm" OR "abdominal aortic aneurysm" OR "AAA"). When applicable, equivalent Medical Subject Headings (MeSH) terms were used when searching Pubmed and Medline. The following pertinent journals were hand searched for any further source material: Atherosclerosis, Circulation, Circulation Research, Journal of Vascular Surgery, Annals of Vascular Surgery, European Journal of Vascular and Endovascular Surgery, and Arteriosclerosis, Thrombosis & Vascular Biology.

Inclusion criteria

Articles were initially screened by title, keywords and abstract to

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Received May 02, 2014; **Accepted** June 09, 2014; **Published** June 11, 2014

Citation: Green BL, Bailey MA, Bridge KI, Griffin KJ, Scott JA (2014) Alcohol Consumption as a Risk Factor for Abdominal Aortic Aneurysm. J Vasc Med Surg 2: 140. doi: 10.4172/2329-6925.1000140

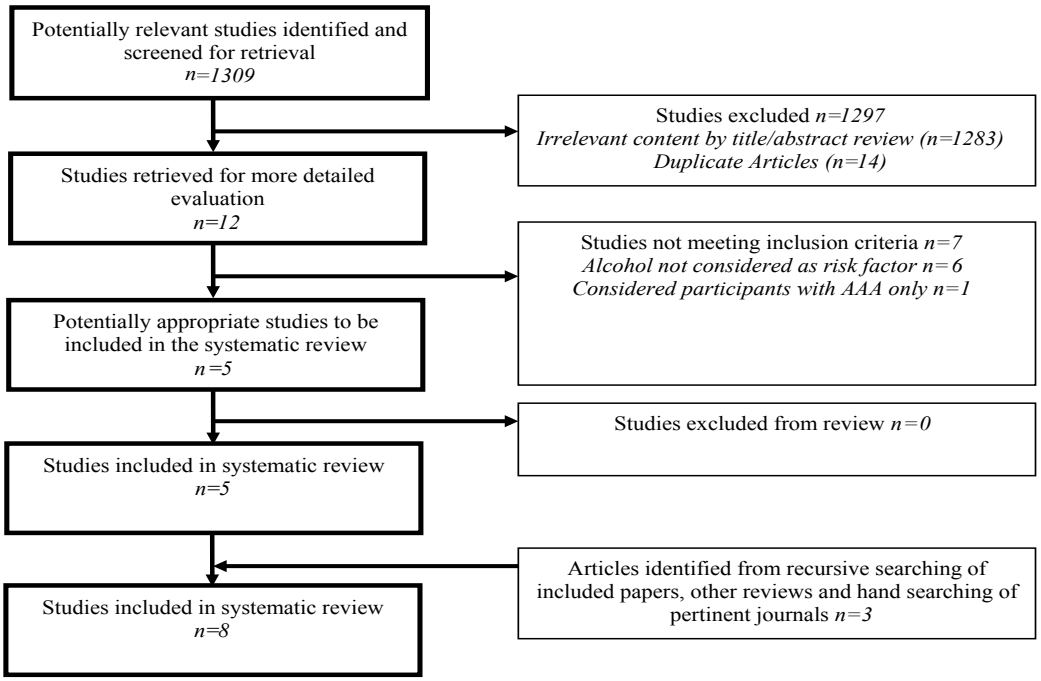

Figure 1: PRISMA diagram of the search strategy employed for the systematic literature review. Numbers presented are pooled across all databases searched.

determine their relevance and suitability for inclusion by two authors (BG, MB), any disagreements were openly discussed and a combined decision made regarding inclusion in the review. Papers were considered suitable for inclusion if they considered an association between alcohol and patients with and without AAA. No limitation was imposed by year of publication, study methodology, or publication language. No limits were placed on methodological quality due to the small number of articles reporting on the topic. In all cases, full text versions of articles were acquired and data extracted for location of study, study size, follow up period, age, sex distribution, alcohol intake, the risk of AAA with high alcohol intake as reported by the study and the type of statistical analysis undertaken by the study authors. Reference lists of all articles were recursively searched for further relevant citations, which were subject to the above inclusion criteria. The full search strategy as guided by the Preferred Reporting Items for Systemic Reviews and Meta-Analysis (PRISMA) recommendations are provided in Figure 1. All authors were involved in the evaluation of the included papers.

Statistical analysis

The current study was assessed by the MRC Biostatistics Unit in Cambridge regarding suitability for meta-analysis. Due to the highly heterogenic nature of the data, regarding both reporting of alcohol intake, grouping based on alcohol intake, and also possible bias of the control groups secondary to other lifestyle and risk factors, it was not considered suitable for further analysis.

Results

The search strategy identified 8 papers for inclusion in the systematic review [8,16-22]. No randomised evidence or review articles were identified. The majority of included studies were retrospective or prospective cohort studies. None of the studies provided a direct comparison of alcohol consumption and AAA, but instead analysed alcohol as part of a series of known and potential clinical risk factors for aneurysmogenesis. Included studies utilised diverse statistical methodologies and therefore reported variable measures of risk. There was a mixture of uni-variable and multi variable statistical analysis techniques employed, but no studies utilised mixed effects or random effects modelling. Due to a relative paucity of available evidence, all relevant studies were included for review: comparable studies are summarised in Table 1.

Bengtsson et al. [16] first reported an association between alcohol intake and AAA in a prospective cohort study of 375 men born in Malmö, Sweden in 1914. Of the 39 (10.7%) patients diagnosed with an AAA, mean alcohol consumption was found to be 131 ± 175 g per week, compared to 86 ± 106 g per week for the control population. The authors found alcohol consumption to be significantly associated with the presence of an AAA; however, this association did not remain statistically significant following stratification for smoking status.

Simoni et al. [17] similarly reported increased prevalence of alcohol use in patients with AAA compared to patients with normal aortic diameter (41% vs. 65%, $p<0.001$) in their population screening study of 1,601 patients in Genoa, Italy. However, only univariable analysis was employed, without correction for multiple testing. Further, the details of the methods used to subcategorise patients based on their alcohol use at the time of recruitment remain unclear.

Wong et al. [8] report risk factors identified for AAA from the Health Professionals Follow Up Study, an American prospective cohort study including 39,352 men followed-up from 1986-2002, of whom 376 had confirmed AAA. Alcohol intake was determined from a self-reported semi-quantitative food frequency questionnaire, administered at study inception and again every four years. The questionnaire was validated against food and drink diaries in a subset of patients. The

Study	Location	AAA	Duration of follow up	n		Age (mean ± SD)	% Male	Alcohol intake	Risk of AAA with high alcohol intake	Analysis
				Ctrl	AAA					
Bengtsson et al. [16]	Sweden	>35 mm	Cross-sectional	375		Not stated	100	AAA (131 ± 175 g) Normal (86 ± 106 g)	Positive correlation (p<0.05)	Univariable*
				299	39					
Simoni et al. [17]	Italy	>29 mm	Ongoing	1,601		69 ± 3	46.3	Not quantified	RR 1.58	Univariable
				1531	70					
Wong et al. [18]	USA	>30 mm	16 years	39,352		53	100	13.7% of total population drinking >30 g/day	RR 1.65 (1.2 with low intake)	Cox Analysis & Multivariable models
				38976	376					
Wang et al. [18]	China	>30 mm	Cross-sectional	395		66.6 ± 10.3	60.3	21% of total population drinking >30 g/day	Positive correlation (p=0.049)	Multivariable
				393	2					
Laughlin et al. [19]	USA	Not defined	Cross-sectional	1,926		62.1 ± 9.8	50.4	Not quantified	Positive correlation (p<0.01)	Univariable*
				1904	22					
Tornwall et al. [20]	Finland	Not defined	5.8 years	27,111		Median 57 (IQR 53-61)	100	11 (Inter-quartile-range 3-26) g per day	RR 0.93 (0.7 with low intake)	Cox Analysis & Multivariable models
				26909	202					
Forsdahl et al. [22]	Norway	>30 mm	7 years	4,345		59.5 ± 9.5	47	Male (3.2 ± 4.6x/month) Female (1.6 ± 3.0x/month)	No correlation	Multivariable
				4226	119					

* Significance of positive correlation was lost on multivariable analysis. All relative risks presented are in comparison to non-drinking groups.

Table 1: Summary of extracted data from comparable studies included in the systematic literature review..

authors reported a strong positive correlation between alcohol intake (by quintile) at baseline and risk of AAA using multivariable Cox analysis. The relative risk of AAA was 1.09 in the lowest intake group (0.1-4.9 g/day) and 2.52 in the highest intake group (>30 g/day) of alcohol consumption after controlling for age alone (p<0.0001). After controlling for age, smoking status, quintiles of body mass index, physical activity (determined by metabolic equivalents), hypertension, diabetes and hypercholesterolaemia the relative risk of AAA in those drinking >30 g/day at baseline fell to 1.21 (p=0.03); however, this increased to 1.65 when including the linear trend in drinking behaviour over the duration of the study (p=0.02). Subgroup analysis of beverage type revealed "spirits" to have the strongest correlation with AAA (p=0.009), however, the authors urge caution in interpretation due to the small numbers in the subgroups.

Wang et al. [18] report on factors significantly associated with infra-renal aortic diameter in a cohort of 395 patients. Alcohol intake was determined by clinical questioning at recruitment and high alcohol intake defined as >30 g/day. The authors report a significant positive correlation between alcohol intake and aortic diameter using linear regression analysis (r=0.265, p<0.00001). Multivariable analysis confirmed that statistical significance remained when controlling for age, sex, height, blood pressure, lipoproteins, chronic obstructive pulmonary disease, diabetes, metabolic syndrome and smoking (p=0.049); however, the small number of cases (n=2) limits the significance of the results.

Laughlin et al. [19] also reported risk factors correlating with aortic diameter. They used Osiris software to determine aortic diameter at the aortic bifurcation and at 5 cm proximal to this, in 1,926 randomly selected patients from the Multi Ethnic Study of Atherosclerosis recruited from five centres in the USA between 2002 and 2005. Alcohol intake was determined by standardised health questionnaire completed at recruitment. Although the authors describe a positive linear correlation between aortic diameter and quartiles of alcohol intake (p<0.01), the significance was lost both at the bifurcation (p=0.11) and 5 cm above (p=0.79) when included in a linear regression analysis along with age, sex, ethnicity, body surface area, diabetes, hypertension, lipid lowering medication, smoking, physical activity, CRP, IL-6, D-dimer, homocysteine and recruitment centre.

Tornwall et al. [20] analysed risk factors associated with a clinical diagnosis of ruptured or non-ruptured AAA in 27,111 Finnish male smokers from the Alpha-Tocopherol, Beta Carotene Cancer Prevention Study, recruited between 1985 and 1993. Alcohol intake was determined at baseline as part of a patient self-administered diet history questionnaire covering the twelve months prior to recruitment. Using a Cox proportional hazard ratio with multivariable adjustment the authors found no clear trend between moderate alcohol intake (16-30 g/day) and the relative risk of AAA; relative risk 0.70 (95 per cent confidence interval (CI) 0.41-1.19).

In 2001 Jamrozik et al. [21] conducted an epidemiological study in Western Australia to determine if country of origin and dietary habits had an influence on aortic diameter. The authors undertook ultrasound screening of 11,745 migrant males assessing dietary and drinking habits with a health questionnaire at recruitment. The authors found the prevalence of AAA was lower in migrants of Mediterranean origin, who, on average consumed more alcohol than other nationalities studied. Notwithstanding, the level of alcohol intake was moderate at <20 g/day. The authors linked this potentially protective Mediterranean bias to the well-known "French Paradox" in ischaemic heart disease.

Forsdahl et al. [22] reported data on risk factors for ultrasound-detected AAA between the Tromsø IV (1994-1995) and Tromsø V study (2001), in 4,345 male and female patients in Norway. Alcohol intake was recorded (as number of times per month) in 1994-1995 using a structured questionnaire. The authors found no relationship between alcohol consumption and the development of AAA, in support of Tornwall and colleagues. However, the authors themselves point out that in both Scandinavian studies the overall level of alcohol consumption is considerably less than other populations (only 2.3 times per month in the Tromsø study) and specifically much lower than the heavy drinking category of >30 g/day used in the Wong et al. [8] and Wang et al. [18] studies.

Discussion

Advancing age, male sex, hypertension and smoking are clear clinical risk factors for AAA [7,9]. The establishment of a national UK AAA screening programme provides a clear opportunity to instigate secondary prevention strategies to prevent the progression of AAA. Alcohol appears to have a dose dependent relationship to ischaemic heart disease, whereby it is protective in moderate doses but harmful

in large doses [23-25]. The link between alcohol intake and AAA is, however, much less widely reported. Only eight studies presenting any data linking alcohol intake and AAA were identified in the contemporary literature, including a total of 86,850 patients with 830 confirmed AAA cases. There were no studies that looked directly to compare alcohol intake with AAA, and thus results may be biased by the authors need to balance study groups based on other risk factors. Of the eight studies identified, there was disparity in overall findings, with five studies suggesting varying degrees of association between alcohol intake and AAA, whilst 3 studies failed to demonstrate any significant relationship or even suggested a slight protective effect. Further, there was variability in the calculation of alcohol intake and a risk of misclassification bias across all studies as with the exception of a single study (Wong et al.), alcohol intake was only quantified at a single time period, and thus previous heavy drinkers who were abstinent from alcohol at the time of study would be classified as 'non-drinkers'.

As was to be expected, there were no randomised trials identified on this subject. Consequently, conclusions must be drawn from a mixture of case-control, cross sectional and cohort studies of varying size and quality which all collected data on alcohol intake via some form of patient questionnaire or clinical interview in a retrospective manner. There are clearly issues surrounding recall bias and social desirability bias, shared by all the included studies. It should additionally be noted that studies by both Wong and Tornwall et al. did not actively screen patients for AAA, they utilised a pragmatic approach, reviewing medical records, which may lead to an underestimation of AAA prevalence in these cohorts.

Furthermore, alcohol intake can change over time and this was only taken into account by Wong et al. [8], who repeated their health questionnaire every four years during their 16 year study. They specifically reported that the strongest trend linking alcohol intake to AAA was identified when changes in alcohol consumption over time were taken into account. This is a striking area of weakness in the methodology of the other included studies which failed to account for changes in drinking behaviour.

The two largest cohort studies included in the review reached opposing conclusions. Wong et al. [8] found a significant association between alcohol consumption and AAA, which remained significant after controlling for significant covariables. In contrast, Tornwall et al. [20] failed to demonstrate any association with alcohol consumption in their Cox Proportional Hazards Multivariable Model. The authors reported increasingly large confidence intervals with rising alcohol consumption as high alcohol intake was rare in not only their own, but both large Scandinavian studies [20,22] and the epidemiological study from Western Australia [21], which collectively failed to demonstrate any correlation between alcohol intake and AAA.

This is itself a valuable observation, and although current evidence is limited, there is some suggestion of a protective effect associated with moderate alcohol intake, as evidenced by Tornwall et al. [20], with increased risk for AAA only appearing with the highest levels of alcohol consumption. This raises the possibility of a dose-dependent effect as observed in ischaemic heart disease, and would additionally account for the lower incidence of AAA as observed by Jamrozik et al. Accounting for known mechanisms, the most likely intermediary process by which alcohol may exert its effect is by promoting the development of hypertension; however, as hypertension may only be a modest risk factor for AAA development [26], it is probable that any mechanism relating alcohol consumption to AAA development will be multi-faceted.

Whilst some studies suggest that alcohol may contribute to AAA development, high alcohol consumption has been shown to be an independent risk factor for smoking [27]. Since smoking is strongly implicated in AAA development, the ability to accurately control for this confounding factor is significantly reduced in those studies utilising univariable analysis only. It is further interesting to speculate as to the potential impact of second-hand smoke exposure in drinkers who themselves do not smoke, although this would be difficult to accurately assess in a clinical setting. These findings are however, based on limited evidence and should be further clarified by ongoing assessment of alcohol usage (both moderate and excessive) in large prospective cohorts of aneurysm patients around the globe.

There are further reports that alcohol may result in lipid profile changes that may contribute to aneurysm development. Wang et al. [18] found an independent relationship between lipoprotein-a (Lp(a)) and infrarenal aortic diameter. Furthermore, the authors found an association between alcohol and Lp(a). Thus, Lp(a) may act as an intermediate mechanism in the development of AAA, although the authors additionally reported a negative association with LDL:HDL cholesterol, which has been shown to be anti-atherogenic. This may be explained by the observation that, until highly advanced, atherosclerosis is primarily a disease of the intima, whilst AAA is principally a disease of the media [27].

It is well established that matrix metalloproteinases (MMPs), in particular MMP-2 and MMP-9, are associated with aortic wall degradation and AAA development. In murine models, alcohol consumption equivalent to that seen in heavy drinking significantly increases MMP-2 in aortic tissue [13]. Given that MMPs are regarded by many to be an essential pathological mechanism in the development of AAA, the finding the alcohol can induce MMP-2 expression and activity in aortic tissue suggests that this may in fact contribute to the multifactorial pathogenesis of AAA development. This is an area of study that clearly needs to be explored further.

More recent evidence [28,29] suggests that enzymes responsible for degrading alcohol, including isoenzymes of alcohol dehydrogenase (ADH) and aldehyde dehydrogenase (ALDH) are significantly reduced in the intima and media of abdominal aortic aneurysms. In particular, class 1 ADH was significantly reduced by 48%. The significance of these findings in aneurysm development is unknown, however, a reduction in enzyme activity has been shown to increase production of advanced glycation end products (AGEs), which are known to be pathological and may cause disruption of normal tissue remodelling in the abdominal aorta.

We recognise that a limitation of this study is the lack of a meta-analysis of the available data. This was not undertaken as the data extracted from published studies was highly heterogeneous and therefore likely to result in a strongly significant heterogeneity statistic. As such, any attempt to pool data across studies unlikely to produce valid or useful results as compared to a comprehensive systematic review. In order to reach valid conclusions in the future, uniform reporting of alcohol data, repeated over time is required. This poses a challenge as there is no accepted standard for alcohol intake questionnaire that is either comprehensive or consistently implemented. In addition, studies directly primarily at assessing alcohol intake and AAA, rather than including this as one of many risk factors would be of use in forming more solid conclusions.

Conclusion

The current literature hints at a possible relationship between alcohol intake and the development of AAA but there is a high risk of other confounding factors. The scope and quality of the available evidence is limited and therefore, no absolute conclusions can be drawn. Future studies should investigate alcohol consumption on the development and growth of AAA with a sample size large enough to control for potential confounders.

Acknowledgement

The authors are grateful to Daniel Jackson at the MRC Biostatistics Unit, Cambridge for his advice on the suitability of the included study data for synthesis and meta-analysis and Patrick Coughlin at the Vascular Surgery Department, Addenbrooke's Hospital, and Cambridge for his constructive comments on the paper.

Funding

Nil Specific to the study. BG: Leeds University Research Enterprise (LURE), MB, KB, KG: National Institute for Health Research (NIHR) & British Heart Foundation (BHF).

Declarations

No authors have any conflict of interest to declare. No specific funding was received in relation to this article. Ben Green received a Leeds University Research Enterprise Scholarship at The University of Leeds; Mr Bailey, Miss Bridge and Miss Griffin have received support from the British Heart Foundation. This work was presented as a poster at The Associated of Surgeons of Great Britain & Ireland AGM 2013, Glasgow. As a consequence, an abstract of the work may appear in a future edition of the British Journal of Surgery. There has been no previous publication of this work or dual submission of the manuscript.

References

1. Johnston KW, Rutherford RB, Tilson MD, Shah DM, Hollier L, et al. (1991) Suggested standards for reporting on arterial aneurysms. J Vasc Surg 13: 452-458.
2. Lindholt JS, Juul S, Fasting H, Henneberg EW (2005) Screening for abdominal aortic aneurysms: single centre randomised controlled trial. BMJ 330: 750-755.
3. Nordon IM, Hinchliffe RJ, Loftus IM, Thompson MM (2011) Pathophysiology and epidemiology of abdominal aortic aneurysms. Nat Rev Cardiol 8: 92-102.
4. Jagadesham VP, Scott DJ, Carding SR (2008) Abdominal aortic aneurysms: an autoimmune disease? Trends Mol Med 14: 522-529.
5. Bown MJ, Jones GT, Harrison SC, Wright BJ, Bumpstead S, et al. (2011) Abdominal aortic aneurysm is associated with a variant in low-density lipoprotein receptor-related protein 1. Am J Hum Genet 89: 619-627.
6. Chang JB, Stein TA, Liu JP, Dunn ME (1997) Risk factors associated with rapid growth of small abdominal aortic aneurysms. Surgery 121: 117-122.
7. Strachan DP (1991) Predictors of death from aortic aneurysm among middle-aged men: the Whitehall study. Br J Surg 78: 401-404.
8. Wong DR, Willett WC, Rimm EB (2007) Smoking, hypertension, alcohol consumption, and risk of abdominal aortic aneurysm in men. Am J Epidemiol 165: 838-845.
9. Brown LC, Powell JT (1999) Risk factors for aneurysm rupture in patients kept under ultrasound surveillance. UK Small Aneurysm Trial Participants. Ann Surg 230: 289-297.
10. Vardulaki KA, Walker NM, Day NE, Duffy SW, Ashton HA, et al. (2000) Quantifying the risks of hypertension, age, sex and smoking in patients with abdominal aortic aneurysm. Br J Surg 87: 95-200.
11. Sweeting MJ, Thompson SG, Brown LC, Powell JT; RESCAN collaborators (2012) RESCAN collaborators. Meta-analysis of individual patient data to examine factors affecting growth and rupture of small abdominal aortic aneurysms. Br J Surg 99: 655-665.
12. Abdul Muneer PM, Alikunju S, Szlachetka AM, Haorah J (2012) The mechanisms of cerebral vascular dysfunction and neuroinflammation by MMP-mediated degradation of VEGFR-2 in alcohol ingestion. Arterioscler Thromb Vasc Biol 32: 1167-1177.
13. Partridge CR, Sampson HW, Forough R (1999) Long-term alcohol consumption increases matrix metalloproteinase-2 activity in rat aorta. Life Sci 65: 1395-1402.
14. Di Castelnuovo A, Costanzo S, Bagnardi V, Donati MB, Iacoviello L, et al. (2006) Alcohol dosing and total mortality in men and women: an updated meta-analysis of 34 prospective studies. Arch Intern Med 166: 2437-2445.
15. Baglietto L, English DR, Hopper JL, Powles J, Giles GG (2006) Average volume of alcohol consumed, type of beverage, drinking pattern and the risk of death from all causes. Alcohol Alcohol 41: 664-671.
16. Bengtsson H, Bergqvist D, Ekberg O, Janzon L (1991) A population based screening of abdominal aortic aneurysms (AAA). Eur J Vasc Surg 5: 53-57.
17. Simoni G, Pastorino C, Perrone R, Ardia A, Gianrossi R, et al. (1995) Screening for abdominal aortic aneurysms and associated risk factors in a general population. Eur J Vasc Endovasc Surg 10: 207-210.
18. Wang JA, Chen XF, Yu WF, Chen H, Lin XF, et al. (2009) Relationship of heavy drinking, lipoprotein (a) and lipid profile to infrarenal aortic diameter. Vasc Med 14: 323-329.
19. Laughlin GA, Allison MA, Jensky NE, Aboyans V, Wong ND, et al. (2011) Abdominal aortic diameter and vascular atherosclerosis: the Multi-Ethnic Study of Atherosclerosis. Eur J Vasc Endovasc Surg 41: 481-487.
20. Törnwall ME, Virtamo J, Haukka JK, Albanes D, Huttunen JK (2001) Life-style factors and risk for abdominal aortic aneurysm in a cohort of Finnish male smokers. Epidemiology 12: 94-100.
21. Jamrozik K, Spencer CA, Lawrence-Brown MM, Norman PE (2001) Does the Mediterranean paradox extend to abdominal aortic aneurysm? Int J Epidemiol 30: 1071-1075.
22. Forsdahl SH, Singh K, Solberg S, Jacobsen BK (2009) Risk factors for abdominal aortic aneurysms: a 7-year prospective study: the Tromsø Study, 1994-2001. Circulation 119: 2202-2208.
23. Costanzo S, Di Castelnuovo A, Donati MB, Iacoviello L, de Gaetano G (2010) Alcohol consumption and mortality in patients with cardiovascular disease: a meta-analysis. J Am Coll Cardiol 55: 1339-1347.
24. Mukamal KJ, Chiuve SE, Rimm EB (2006) Alcohol consumption and risk for coronary heart disease in men with healthy lifestyles. Arch Intern Med 166: 2145-2150.
25. Roerecke M, Rehm J (2012) The cardioprotective association of average alcohol consumption and ischaemic heart disease: a systematic review and meta-analysis. Addiction 107: 1246-1260.
26. Golledge J, Muller J, Daugherty A, Norman P (2006) Abdominal aortic aneurysm: pathogenesis and implications for management. Arterioscler Thromb Vasc Biol 26: 2605-2613.
27. Dallongeville J, Marécaux N, Fruchart JC, Amouyel P (1998) Cigarette smoking is associated with unhealthy patterns of nutrient intake: a meta-analysis. J Nutr 128: 1450-1457.
28. Daugherty A, Cassis LA (2004) Mouse models of abdominal aneurysms. Arterioscler Thromb Vasc Biol 24: 429–434.
29. Jelski W, Orywal K, Panek B, Gacko M, Mroczko B, et al. (2009) The activity of class I, II, III and IV of alcohol dehydrogenase (ADH) isoenzymes and aldehyde dehydrogenase (ALDH) in the wall of abdominal aortic aneurysms. Exp Mol Pathol 87: 59-62.