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Difference in risk factor profile for abdominal aortic aneurysm and thoracic aortic aneurysm

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ABSTRACT

Objective: Previous studies suggest partly different risk factor profiles of thoracic aortic aneurysms (TAAs) and abdominal aortic aneurysms (AAAs), but prospective data are scarce. The purpose of this prospective population-based case-control study was to investigate differences in risk factor profile between TAAs and AAAs.

Methods: Participants in two prospective population-based studies, the Västerbotten Intervention Project (VIP) and the Monitoring of Trends and Determinants in Cardiovascular Disease (MONICA) study, between 1986 and 2010, underwent cardiovascular risk assessments, including blood samples, oral glucose tolerance test, blood pressure readings, and a self-reported health questionnaire. All individuals who were later diagnosed with TAAs or AAAs were identified. Age, sex, and time-matched controls were selected from the same cohorts, aiming at four controls/case. Adjusted odds ratios (aORs) for potential risk factors for later diagnosis of TAAs and AAAs, respectively, were estimated by multivariate conditional logistic regression analyses.

Results: From a total of 96,196 individuals with prospectively collected data in the VIP/MONICA cohort, a total of 236 individuals with AAAs (181 men and 55 women) and 935 matched controls, and 168 individuals with TAAs (115 men and 53 women) and 662 controls were included. The average age at baseline examination was 57.0 ± 5.7 years for AAA cases and controls, and 52.1 ± 8.8 years for TAA cases and controls. Mean time between baseline examination and diagnosis of AAAs/TAAs was 12.1 and 11.7 years, respectively. There was a clear difference in risk factor profile between AAAs and TAAs. Smoking, hypertension, and coronary artery disease were significantly associated with later diagnosis of AAAs, with highest aORs for a history of smoking (aOR, 10.3; 95% confidence interval [CI], 6.3-16.8). For TAAs, hypertension was the only positive risk factor (aOR, 1.7; 95% CI, 1.1-2.7), whereas smoking was not associated. Diabetes was not associated with either AAAs or TAAs; neither was self-reported physical activity.

Conclusions: In this prospective, population-based, case-control study, risk factor profile differed between AAAs and TAAs. This suggests a partially different etiology for TAAs and AAAs. (J Vasc Surg 2025;81:335-41.)

Keywords: Abdominal aortic aneurysm; Risk factor; Thoracic aortic aneurysm

Abdominal aortic aneurysm (AAA) is a common disease and accounts for significant morbidity and mortality worldwide.[1] Established risk factors are male sex, increasing age, heredity, smoking, hypertension, and hypercholesterolemia.[2] The past 20 years has seen a changed epidemiology, with reduced prevalence in several countries.[3] This has been attributed to reduced smoking[4] and more comprehensive cardiovascular prevention with blood pressure control and statins.[5]

Thoracic aortic aneurysm (TAA) is less common and less explored.[6] A large Danish cross-sectional population screening study where over 14,000 individuals were examined with computed tomography (CT) showed a low prevalence of TAAs compared with AAAs.[7] Previous retrospective data indicate differences in risk factor profile relative to AAAs.[8] However, there is a lack of prospective data on factors associated with TAA development.

In this prospective population-based study, two ongoing cohorts concerning cardiovascular disease were explored. In the beginning of the 1980s, the mortality from cardiovascular disease was higher in the northern part of Sweden than in the rest of the country. With the aim of reducing cardiovascular morbidity and mortality in Västerbotten County (Fig 1), the Västerbotten Intervention Project (VIP) was initiated in 1985. Since then, all men and women aged 40, 50, and 60 years are invited to an individual health survey, which includes a questionnaire on cardiovascular risk factors, and length,

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weight, and blood pressure are measured, blood lipids are analyzed, and an oral glucose tolerance test is performed. Furthermore, blood is saved to be stored in a biobank (The Medical Biobank North).[9]

The MONICA study is a study initiated by the World Health Organization. The purpose is to monitor trends in cardiovascular diseases and risk factors. The counties of Västerbotten and Norrbotten joined the study in 1985 (Fig 1). The basic part of the MONICA study consists of recurring population surveys in Västerbotten and Norrbotten approximately every 5 years. In these surveys, 2500 randomly selected men and women between the ages of 25 and 74 are invited to participate in a health survey. The survey includes a similar form of questionnaire and collection of anthropometric as in VIP.[10]

VHU/MONICA includes prospectively collected data from the years 1986 through 2010 on more than 96,196 unique individuals, and constitutes a unique opportunity to study risk factors in a population-based prospective manner in a well-defined population.

The aim of this population-based prospective case-control study was to investigate differences in risk factor profile between individuals later developing AAAs and TAAs.

ARTICLE HIGHLIGHTS

- **Type of Research:** Population-based prospective case-control study
- **Key Findings:** There was a clear difference in risk factor profile between abdominal aortic aneurysm and thoracic aortic aneurysm. Smoking, hypertension, and coronary artery disease were associated with later diagnosis of abdominal aortic aneurysm, whereas hypertension was associated with later diagnosis of thoracic aortic aneurysm.
- **Take Home Message:** The different risk factor profile supports a partially different etiology for aneurysms in the abdominal and thoracic aorta, respectively.

METHODS

Among all patients in the geographic region of Västerbotten and Norbotten that participated in the prospective survey programs VIP/MONICA 1986 through 2010 and subsequently have been diagnosed with an AAA or a TAA were identified. An AAA was defined as having a maximal anteroposterior diameter of at least 3 cm of the abdominal aorta (below the diaphragm), measured by any of the modalities of ultrasound, CT, or magnetic resonance imaging (MRI). A TAA was defined as having an aneurysm of at least 4.5 cm in the ascending aorta, aortic arch, or descending aorta (above the diaphragm) measured by CT or MRI. All patients with TAAs and AAAs included in the study had a complete examination of the entire aorta with CT or MRI. Individuals with aortic dissections, thoracoabdominal aortic aneurysms (TAAAs), or concomitant AAAs and TAAs were excluded, as were subjects with known connective tissue disease or aortic infections.

It should be noted that all patients with AAAs included in this study were incidentally detected, before population screening for AAAs was introduced.

A case-control study design was set up with the ratio 1:4 of patients with AAAs/TAAs and healthy controls matched by study (VIP/MONICA), age, sex, and the time point of data collection.

A ratio of 1:4 is the default when one wishes to match cases with controls for studies within the VIP/MONICA cohorts. Data were collected from the two studies: smoking status (current or former smokers), body mass index, hypertension (defined as diagnosed with hypertension, on antihypertensive treatment, or a single measured systolic blood pressure ≥160 mmHg or diastolic blood pressure ≥100 mmHg), diabetes mellitus (defined as diagnosed with diabetes, on antidiabetic treatment, or a fasting blood p-glucose ≥7.0 mmol/L or 2-hour oral glucose tolerance test ≥12.2 mmol/L), coronary heart disease (defined as diagnosed with myocardial infarction or on anti-anginal medical treatment), and Cambridge index for physical activity.[11] The medical records of all individuals with TAAs were reviewed for the presence of bicuspid aortic valve, which is associated with dilatation of the ascending aorta.[12]

Statistics. Missing data were handled by multiple imputation under the assumption that the data were missed at random using the Multiple Imputation by Chained Equations (MICE) package (R).[13] The average of missing data was 11.2%, and the distribution of missing data is presented in Supplementary Table I (online only). All covariates and the two dependent variables AAA and TAA were used for imputation. Continuous variables were imputated with Bayesian linear regression, binary variables were imputated with logistic regression, and unordered/ordered categorical variables were imputated with polytomous logistic regression and proportional odds logistic regression, respectively. The number of imputations used was 50, and statistics were pooled with Rubin's rules.[14] A post imputation sensitivity analysis of the assumption that data missing at random[15] was made for all variables included in the multivariate conditional logistic regression with more than 10 percent missing values (see Supplementary Table I, online only). The analysis, tested for local deviations from the missed at random assumption, showed stable odds ratios (ORs) with variation rates less than 10 percent (Supplementary Table III and Supplementary Table IV, online only), suggesting robust imputations.

Risk factors for AAAs and TAAs, respectively, were tested in a univariate analysis. Those with a *P* value ≤ .2 or deemed clinically important were included in a

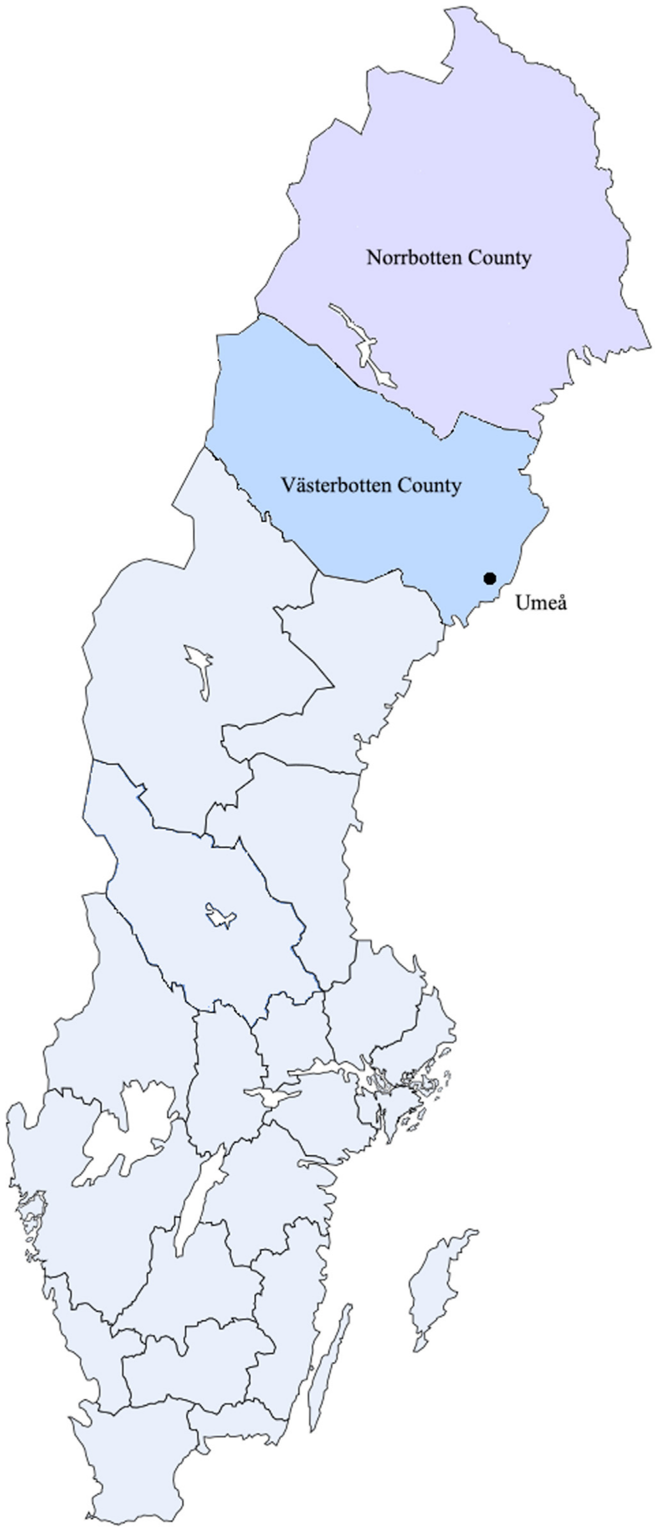

Fig 1. Sweden with Västerbotten County and Norrbotten County in the north.

conditional logistic regression analyzes. The aim was to compare conditional logistic regression with the same covariates for both AAAs and TAAs as the dependent variable and not to find the fittest model possible in the respective analysis. Linearity between logit for the dependent variable and each continuous covariate was tested (see Supplementary Figs 1-6, online only). When none of the continual variables showed acceptable linearity properties, they were divided into quartiles before the multivariate analysis. To evaluate whether significant differences were due to differences in power or actual differences in effect size, diagnosis (AAA vs TAA) as a variable and its interaction with each variable was analyzed in separate univariate logistic regression models.

R was used for statistical analyses where the univariate analysis was made with univariate logistic regression using the glm function (stats package, R), and the conditional multivariate analysis was made by the clogit function (survival package, R).

Ethics. The Research Ethics Review Board of northern Sweden approved the study (Dnr 2011-336-31M).

RESULTS

A total of 619 individuals who had participated in VIP/MONICA between 1985/1986 and 2010 were subsequently diagnosed with an AAA or TAA. After excluding individuals whose diagnosis was incorrect (n = 73) or fulfilled the exclusion criteria (n = 142), 236 individuals remained with AAAs (181 men and 55 women) and 168 with TAAs (115 men and 53 women) (Fig 2). Mean ages at baseline VIP/MONICA examination were 54.8/57.2 years (range, 29.2-70.0/36.0-74.0 years), and mean time to diagnosis of AAA/TAA was 12.1/11.5 years (standard deviation, 5.4/5.6 years).

Baseline data and univariate analysis between cases and controls for AAAs and TAAs, respectively, are presented in Table I, showing a clear difference in risk factor profile between AAAs and TAAs. In the multivariable analyses, smoking, cholesterol >6.87 mmol/L, hypertension, and coronary artery disease were significantly associated with later diagnosis of AAAs, with highest aOR for a history of smoking (aOR, 10.3; 95% confidence interval [CI], 6.3-16.8). For TAAs, hypertension was the only positive risk factor (aOR, 1.7; 95% CI, 1.1-2.7), whereas smoking was not associated. Diabetes was not associated with either AAAs or TAAs; neither was self-reported physical activity (Table II). Over 90% of individuals with TAAs have undergone echocardiography or had had surgery for their aneurysm. Of these, 24% (27% for aneurysm in the ascending aorta) had a bicuspid aortic valve. A subgroup analysis was made for ascending aortic aneurysms (143 of 168 TAAs). In the analysis, no covariate was significant for later being diagnosed with an aneurysm in the ascending aorta.

Separate univariate logistic regression models showed significant differences in effect size between AAAs and TAAs for coronary artery disease (P = .031), current smoking (P < .001), ever smoked (P < .001),

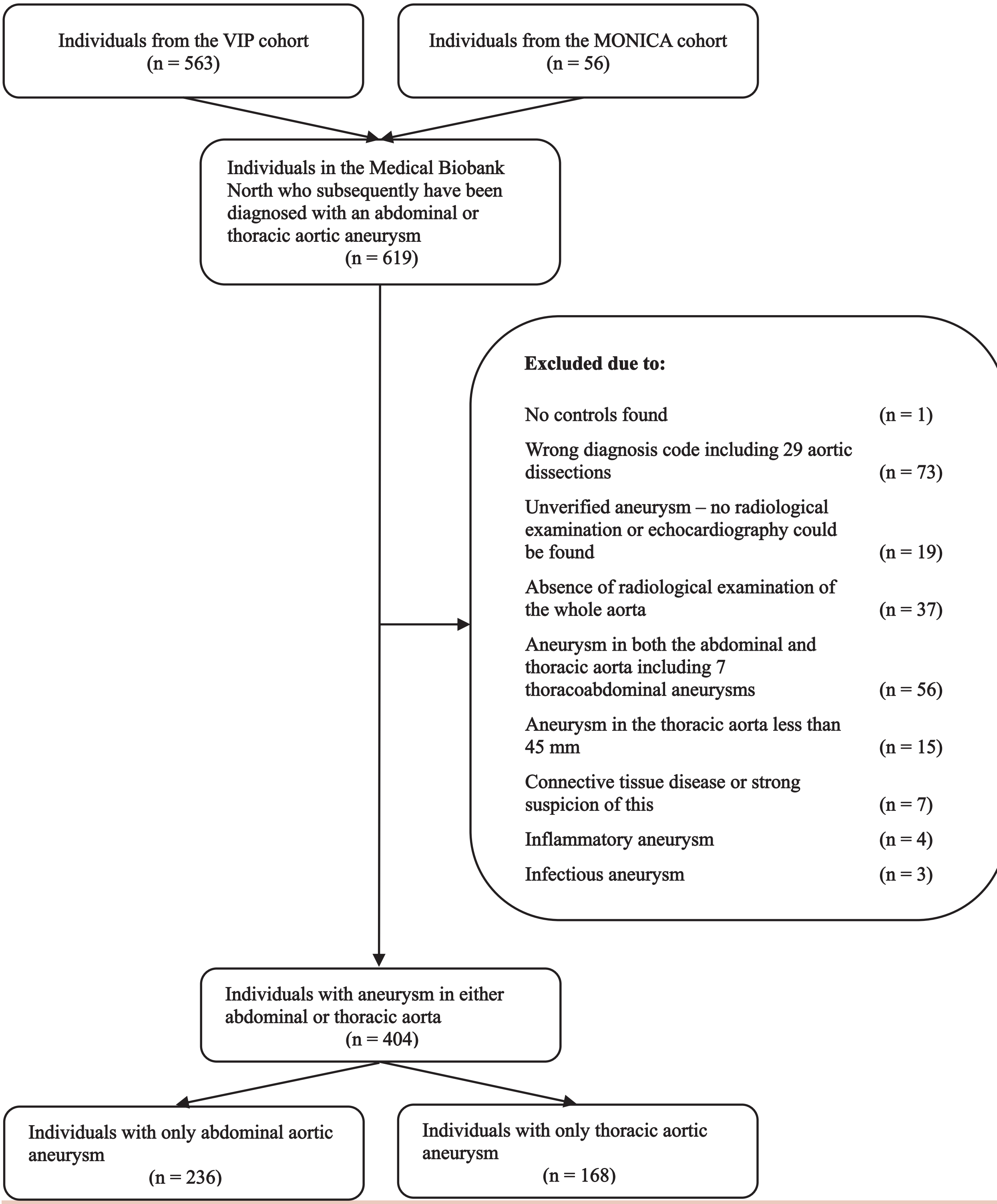

Fig 2. Consort flow diagram for cases. *MONICA*, Monitoring of Trends and Determinants in Cardiovascular Disease; *VIP*, Västerbotten Intervention Project.

and high cholesterol levels ($P < .001$), but not for triglycerides ($P = .605$). This suggests that the observed differences in risk factor profile between AAAs and TAAs were not to any great extent due to lower power in the TAA sample.

DISCUSSION

In this population-based prospective case-control study with long-term follow-up there was a clear difference in risk factor profile between subjects who, one decade later, were diagnosed with AAAs vs TAAs. Smoking,

Table I. Univariate analysis between cases and controls for abdominal aortic aneurysms (*AAAs*) and thoracic aortic aneurysms (*TAAs*), respectively

Baseline characteristics/risk factors	AAAs (n = 236), (95% CI)	Controls (n = 935), (95% CI)	*P*	TAAs (n = 168), (95% CI)	Controls (n = 662), (95% CI)	*P*
Men, no.	181	716		115	453	
Men, %	76.7	76.6		68.5	68.4	
Mean age, years	57.0	57.0		52.1	52.1	
VIP, no.	221	875		157	618	
VIP, %	93.6	93.6		93.5	93.4	
MONICA, no	15	60		11	44	
Mean BMI, kg/m^2	26.8 (26.3-27.3)	26.2 (25.9-26.4)	.019	26.9 (26.3-27.6)	26.1 (25.8-26.4)	.017
Mean S-cholesterol, mmol/l	6.7 (6.5-6.8)	6.2 (6.1-6.3)	<.001	5.9 (5.8-6.1)	6.1 (6.0-6.2)	.105
Mean S-triglycerides, mmol/l	1.2 (1.1-1.3)	1.1 (1.1-1.1)	.002	1.2 (1.1-1.2)	1.1 (1.1-1.2)	.250
Hypertension, %	36.9 (30.7-43.0)	23.9 (21.1-26.6)	<.001	32.1 (25.1-39.2)	21.5 (18.3-24.6)	.004
Diabetes mellitus, %	7.2 (3.9-10.5)	6.8 (5.2-8.5)	.846	3.6 (0.8-6.4)	5.4 (3.7-7.2)	.327
Coronary artery disease, %	17.3 (11.9-22.8)	6.9 (5.0-8.8)	<.001	8.1 (3.2-13.1)	4.1 (2.4-5.8)	.067
Lipid lowering medication, %	7.5 (3.5-11.6)	3.6 (2.2-5.1)	.031	4.3 (0.7-7.8)	3.0 (1.5-4.4)	.484
Current smoker, %	58.0 (51.5-64.4)	18.1 (15.5-20.6)	<.001	26.1 (19.3-32.8)	22.3 (19.0-25.5)	.300
Ever smoked, %	85.5 (81.0-90.1)	43.4 (40.1-46.6)	<.001	52.3 (44.6-60.0)	44.5 (40.6-48.3)	.075
Ever snuffed, %	32.7 (26.3-39.1)	26.6 (23.6-29.6)	.084	30.2 (23.0-37.5)	26.3 (22.8-29.8)	.327
PA index, %			.404			.471
1	22.4 (16.4-28.4)	17.5 (14.8-20.3)		18.0 (11.8-24.3)	16.7 (13.6-19.8)	
2	34.9 (28.1-41.8)	36.8 (33.5-40.1)		31.7 (24.0-39.3)	38.5 (34.5-42.5)	
3	29.3 (22.7-35.9)	29.7 (26.5-33.0)		29.7 (22.3-37.0)	27.8 (24.1-31.4)	
4	13.3 (8.6-18.1)	16.0 (13.4-18.6)		20.6 (14.0-27.3)	17.1 (14.0-20.2)	

BMI, Body mass index; *CI*, confidence interval; *MONICA*, monitoring of trends and determinants in cardiovascular disease; *PA index*, physical activity-index; *VIP*, västerbotten intervention project.
PA-index: 1, inactive; 2, moderately inactive; 3, moderately active; 4, active (percentage of PA-index).

hypertension, high cholesterol levels, and coronary artery disease were significantly associated with diagnosis of AAA, with the highest aOR for smoking. For TAAs, hypertension was the only positive risk factor, whereas smoking was not significantly associated with TAA development. Diabetes was not associated with either AAAs or TAAs, nor was self-reported physical activity.

Several reports have studied risk factors for AAA development, with smoking consistently being the most important, as observed in the present study.[2,16] Risk factors for the development of TAAs are, however, not as well-studied. In a recent Danish cross-sectional study, with more than 15,000 individuals examined with a CT scan, smoking was associated with abdominal aortic dilation but not with thoracic aortic dilation. Furthermore, hypertension was associated with dilation of both the abdominal and thoracic aorta.[7] These results are consistent with our findings.

Several studies have shown a lower incidence of AAAs in diabetic patients.[16,17] A meta-analysis demonstrated a statistically significant inverse association between diabetes mellitus and TAAs, similar to that of AAAs.[18] In the Danish cross-sectional study, the incidence of thoracic aortic dilation was lower in diabetics.[7] However, another Danish study by the same authors failed to demonstrate a negative association between diabetes and AAAs,[19] which is in line with the present study. The fact that diabetes, which often correlates with atherosclerotic vascular disease, is not associated with aortic aneurysm formation, has triggered a specific interest in studies of aortic aneurysm pathophysiology. Recently, much focus has been put on the fact that metformin, a commonly used drug for treatment of type II diabetes, may be the confounder explaining the negative correlation between diabetes and aneurysm formation. In animal studies of aneurysm formation, metformin acts protectively and reduces aortic wall degradation. Epidemiologic studies suggest that prevalence of aortic aneurysm is reduced in diabetics under metformin treatment, but not in non-metformin-treated diabetic individuals. Currently, randomized trials are under way to assess if metformin treatment can reduce the growth rate of established small AAAs. Although the role of metformin in TAA development merits further studies, the observed

Table II. Multivariate analysis between cases and controls for abdominal aortic aneurysms (*AAAs*) and thoracic aortic aneurysms (*TAAs*), respectively

	AAA			TAA		
Risk Factor	OR	95% CI	*P*	OR	95% CI	*P*
BMI (15.7-23.7), kg/m^2	1.0					
BMI (23.8-25.9), kg/m^2	0.8	0.5-1.3	.306	1.0	0.6-1.7	.889
BMI (26.0-28.4), kg/m^2	0.8	0.5-1.4	.506	1.0	0.6-1.8	.933
BMI (28.5-57.5), kg/m^2	1.2	0.7-2.0	.478	1.4	0.8-2.4	.179
Cholesterol (1.77-5.43), mmol/l	1.0					
Cholesterol (5.44-6.14), mmol/l	1.2	0.7-2.1	.566	1.2	0.8-1.9	.399
Cholesterol (6.15-6.86), mmol/l	1.6	0.9-2.7	.089	0.7	0.4-1.2	.262
Cholesterol (6.87-12.4), mmol/l	2.4	1.4-4.1	.001	0.7	0.4-1.3	.243
Triglycerides (0.11-1.01), mmol/l	1.0					
Triglycerides (1.02-1.08), mmol/l	1.5	0.8-2.7	.162	0.9	0.5-1.5	.662
Triglycerides (1.09-1.17), mmol/l	1.8	1.0-3.1	.058	0.9	0.5-1.7	.781
Triglycerides (1.18-7.77), mmol/l	2.1	1.2-3.8	.013	1.3	0.7-2.5	.333
Hypertension, %	2.2	1.4-3.3	<.001	1.7	1.1-2.7	.013
Diabetes mellitus, %	0.7	0.3-1.5	.405	0.5	0.2-1.2	.114
Coronary artery disease, %	2.0	1.1-3.5	.024	1.8	0.8-4.3	.173
Current smoker, %	7.9	5.3-11.7	<.001	1.3	0.8-1.9	.286
Ever smoked, %	10.3	6.3-16.8	<.001	1.3	0.9-1.9	.173

BMI, Body mass index; *CI,* confidence interval; *OR,* odds ratio.
Smoking variables were entered separately into the same analysis.

differences between AAAs and TAAs suggests that metformin, and other potential medical treatment options for AAAs, may not necessarily have any effect on TAAs.

Several studies have examined the association between physical activity and AAAs. Some studies reporting a reduced risk, whereas others have found no association.[20] Physical activity and TAAs are less well-studied, and prospective data is lacking. In this study, no association between physical activity and later TAA or AAA development was observed.

Due to the study design with matching for gender and age, it is not possible to analyze these factors relative to AAAs and TAAs, respectively. The same can be said about time to development analysis. The timing of diagnosis differs between AAAs and TAAs, but all AAAs/TAAs were incidentally detected and in different clinical stages at the time for detection, which precludes time to development analysis.

The male/female ratio in the current cohort was 4:1 for AAAs, which is in line with expectations. For TAAs, the ratio is close to 1:1, which is which is a high proportion of women. However, as this cohort only includes incidental detected AAAs and TAAs, it is difficult to compare with cross-sectional screening population data.

The thoracic and the abdominal aorta have different embryology, and their biochemical properties and the origin of their vascular smooth muscle cells differs.[21] Also, the distribution of atherosclerosis differs, and is more widespread peripheral to the ligamentum arteriosum than proximal to it.[22] Approximately 20% of all TAAs or dissections are hereditary with autosomal dominant inheritance pattern.[23] A corresponding heredity is not seen for AAAs. About one-quarter of individuals with TAAs were found to have a bicuspid aortic valve associated with dilatation of the ascending aorta and can probably explain some of the differences found between AAAs and TAAs.

This, together with the observed marked difference in risk factor distribution between AAAs and TAAs, indicate that these are two different diseases. The findings regarding risk factors for AAAs suggest that this is a degenerative aortic disease, which, to a large extent, shares a profile with atherosclerotic arterial disease, whereas TAAs do not share this profile. The relationship between TAAs and, especially, ascending aortic aneurysm and bicuspid valve, as well as lack of correlation with atherosclerotic manifestation, suggests a morphological and physiological mechanism to aneurysm development in these cases.

The observed differential risk between AAAs and TAAs may have same practical significance. Several studies have shown that a reduction in smoking is associated with a reduction in AAA prevalence, with a dose-response relationship suggesting a causal relationship. However, whether better blood pressure control in the population can influence TAA development needs to be proven.

The current study is limited in size and partly based on self-reported data, which has its limitations. Information is missing in the material regarding heredity for aortic aneurysms in first-degree relatives. Another limitation is the predominance of ascendant pathology in the TAA group, which makes it difficult to draw clear conclusions regarding descending TAAs. However, the strength with the study is that it is prospective, population-based, and includes a high-risk population.[24]

CONCLUSION

In this prospective, population-based, case-control study, risk factor profile differed between AAAs and TAAs. This supports a partially different etiology for TAAs and AAAs.

AUTHOR CONTRIBUTIONS

Conception and design: SP, MH, AW
Analysis and interpretation: SP, MH, KM, AW
Data collection: SP
Writing the article: SP, AW
Critical revision of the article: SP, MH, KM, AW
Final approval of the article: SP, MH, KM, AW
Statistical analysis: SP
Obtained funding: SP
Overall responsibility: SP

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DISCLOSURES

None.

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Additional material for this article may be found online at www.jvascsurg.org.

Supplementary Table I (online only). Missing values in the database

Factors	Controls AAA	AAA	Controls TAA	TAA	In total
Number of individuals	935	236	662	168	2001
BMI					
Numbers with missing data	9	1	5	3	18
Percent missed data	1.0	0.4	0.8	1.8	0.9
Cholesterol					
Numbers with missing data	23	7	15	6	51
Percent missed data	2.5	3.0	2.3	3.6	2.5
Triglycerides					
Numbers with missing data	220	53	163	33	469
Percent missed data	23.5	22.5	24.6	19.6	23.4
Hypertension					
Numbers with missing data	0	0	0	0	0
Percent missed data	0.0	0.0	0.0	0.0	0.0
Diabetes mellitus					
Numbers with missing data	1	0	1	0	2
Percent missed data	0.1	0.0	0.2	0.0	0.1
Coronary artery disease					
Numbers with missing data	226	59	120	37	442
Percent missed data	24.2	25.0	18.1	22.0	22.1
Lipidlowering medication					
Numbers with missing data	303	80	157	44	584
Percent missed data	32.4	33.9	23.7	26.2	29.2
Smoking status					
Numbers with missing data	33	8	24	7	72
Percent missed data	3.5	3.4	3.6	4.2	3.6
Snuff status					
Numbers with missing data	110	38	50	16	214
Percent missed data	11.8	16.1	7.6	9.5	10.7
PA index status					
Numbers with missing data	207	57	103	27	394
Percent missed data	22.1	24.2	15.6	16.1	19.7

AAA, Abdominal aortic aneurysm; *BMI,* body mass index; *PA index,* physical activity index; *TAA,* thoracic aortic aneurysm.
There were 820 of 2001 individuals with at least one missing data point.
Sensitivity analysis after the multiple imputation according to Héraud-Bousquet et al: Practical considerations for sensitivity analysis after multiple imputation applied to epidemiological studies with incomplete data. BMC Medical Research Methodology 2012 and Carpenter et al: Sensitivity analysis after multiple imputation under missing at random: a weighting approach. Statistical Methods in Medical Research 2007.

Supplementary Table II (online only). Multivariate logistic regression of factors associated with abdominal aortic aneurysms (*AAAs*) and thoracic aortic aneurysms (*TAAs*) respectively

	Multivariate analysis - AAA		Multivariate analysis - TAA	
Factor	Complete case (n = 1181), aOR (95% CI)	Multiple imputation (n = 2001) aOR (95% CI) M = 50 imputed datasets	Complete case (n = 1181), aOR (95% CI)	Multiple imputation (n = 2001) aOR (95% CI) M = 50 imputed datasets
BMI (15.7-23.7), kg/m^2	1.0	1.0	1.0	1.0
BMI (23.8-25.9), kg/m^2	1.0 (0.5-2.0)	0.8 (0.5-1.3)	1.1 (0.6-2.2)	1.0 (0.6-1.7)
BMI (26.0-28.4), kg/m^2	0.7 (0.3-1.5)	0.8 (0.5-1.4)	0.7 (0.3-1.6)	1.0 (0.6-1.8)
BMI (28.5-57.5), kg/m^2	1.2 (0.6-2.5)	1.2 (0.7-2.0)	1.2 (0.6-2.5)	1.4 (0.8-2.4)
Cholesterol (1.77-5.43), mmol/l	1.0	1.0	1.0	1.0
Cholesterol (5.44-6.14), mmol/l	1.2 (0.6-2.5)	1.2 (0.7-2.1)	1.3 (0.7-2.4)	1.2 (0.8-1.9)
Cholesterol (6.15-6.86), mmol/l	1.3 (0.7-2.6)	1.6 (0.9-2.7)	0.9 (0.5-1.8)	0.7 (0.4-1.2)
Cholesterol (6.87-12.4), mmol/l	1.7 (0.8-3.5)	2.4 (1.4-4.1)	0.9 (0.4-1.9)	0.7 (0.4-1.3)
Triglycerides (0.11-1.01), mmol/l	1.0	1.0	1.0	1.0
Triglycerides (1.02-1.08), mmol/l	1.5 (0.7-3.1)	1.5 (0.8-2.7)	0.7 (0.4-1.5)	0.9 (0.5-1.5)
Triglycerides (1.09-1.17), mmol/l	2.4 (1.2-4.7)	1.8 (1.0-3.1)	0.8 (0.4-1.5)	0.9 (0.5-1.7)
Triglycerides (1.18-7.77), mmol/l	2.1 (1.0-4.5)	2.1 (1.2-3.8)	1.1 (0.5-2.3)	1.3 (0.7-2.5)
Hypertension	2.4 (1.3-4.3)	2.2 (1.4-3.3)	1.9 (1.1-3.2)	1.7 (1.1-2.7)
Diabetes mellitus	0.6 (0.2-1.7)	0.7 (0.3-1.5)	0.8 (0.3-2.4)	0.5 (0.2-1.2)
Coronary artery disease	2.9 (1.4-6.0)	2.0 (1.1-3.5)	2.0 (0.8-5.1)	1.8 (0.8-4.3)
Current smoker	10.4 (5.9-18.3)	7.9 (5.3-11.7)	1.1 (0.6-2.1)	1.3 (0.8-1.9)
Ever smoked	13.1 (6.4-26.8)	10.3 (6.3-16.8)	1.1 (0.6-1.8)	1.3 (0.9-1.9)
	820 observations deleted due to missingness		820 observations deleted due to missingness	

Supplementary Table III (online only). Multivariate analysis for the complete case, multiple imputation and sensitivity analysis, with M = 1000 imputed data sets — coronary artery disease

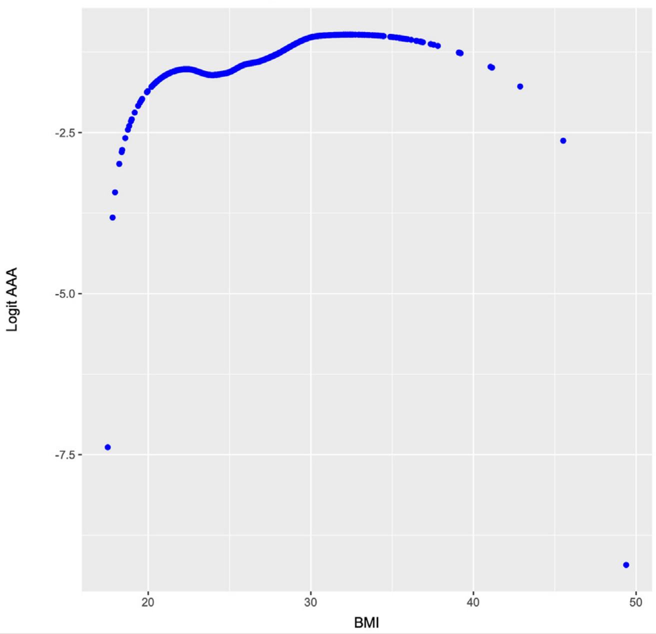
	AAA		TAA	
	Coronary artery disease, %		Coronary artery disease, %	
Missing values %	22.1		22.1	
Complete case (CC)				
aOR	2.92		2.00	
95 % CI	1.41-6.04		0.78-5.09	
SE	0.37		0.48	
CV %	13		24	
Multiple imputation (MI)				
aOR	1.96		1.81	
95 % CI	1.09-3.50		0.77-4.25	
SE	0.29		0.43	
CV %	15		24	
VR_{MI} (MI vs CC) %	−32.9		−9.5	
Sensitivity analysis (SA)				
delta	−0.59	0.86	−0.70	0.70
aOR	1.97	2.06	1.89	1.75
95% CI	1.14-3.40	1.13-3.73	0.86-4.17	0.77-4.00
SE	0.28	0.30	0.40	0.42
CV %	14	15	21	24
VR_{SA} (SA vs MI) %	0.6	5.1	5.7	−2.2

aOR, Adjusted odds ratio; *CI,* confidence interval; *CV %,* coefficient of variation of the aOR; *delta,* chosen value for delta; *SE,* standard error; *VR_{MI} (MI vs CC) %,* variation rate of the aOR for CC and MI analyses; *VR_{SA} (SA vs MI) %,* variation rate of the aOR for MI and senstitivity analyses.
Sensitivity analysis after the multiple imputation according to Héraud-Bousquet et al: Practical considerations for sensitivity analysis after multiple imputation applied to epidemiological studies with incomplete data. BMC Medical Research Methodology 2012 and Carpenter et al: Sensitivity analysis after multiple imputation under missing at random: a weighting approach. Statistical Methods in Medical Research 2007.

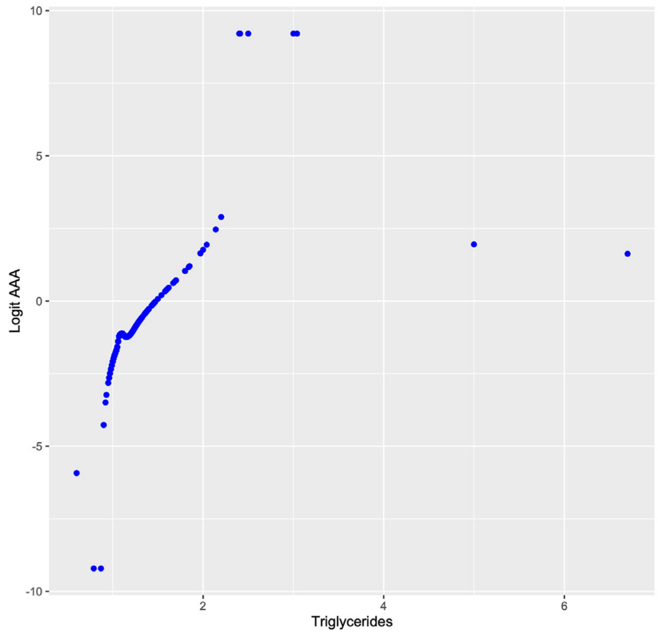
Supplementary Table IV (online only). Multivariate analysis for the complete case, multiple imputation and sensitivity analysis, with M = 1000 imputed data sets − Triglycerides

		AAA						TAA					
	Triglycerides (total)	**Triglycerides (1.02-1.08), %**		**Triglycerides (1.09-1.17), %**		**Triglycerides (1.18-7.77), %**		**Triglycerides (1.02-1.08), %**		**Triglycerides (1.09-1.17), %**		**Triglycerides (1.18-7.77), %**	
Missing values %	23.4												
CC													
aOR		1.49		2.35		2.12		0.74		0.77		1.10	
95 % CI		0.71-3.11		1.17-4.73		0.99-4.53		0.37-1.48		0.39-1.53		0.52-2.33	
SE		0.38		0.36		0.39		0.35		0.35		0.38	
CV %		25		15		18		47		45		35	
MI													
aOR		1.51		1.75		2.10		0.89		0.92		1.34	
95 % CI		0.85-2.66		0.98-3.11		1.18-3.74		0.52-1.52		0.51-1.66		0.74-2.44	
SE		0.29		0.29		0.29		0.28		0.30		0.30	
CV %		19		17		14		31		33		23	
VR_{MI} (MI vs CC) %		1.3		−25.6		−0.8		19.0		19.2		22.6	
SA													
delta		−0.75	0.75	−0.75	0.75	−0.75	0.75	−0.59	0.59	−0.59	0.59	−0.59	0.59
aOR		1.49	1.40	1.74	1.69	2.12	2.23	0.88	0.90	0.94	0.95	1.35	1.39
95 % CI		0.83-2.69	0.83-2.37	0.96-3.16	0.99-2.88	1.16-3.90	1.27-3.92	0.50-1.53	0.54-1.50	0.52-1.71	0.55-1.65	0.73-2.51	0.80-2.40
SE		0.30	0.27	0.30	0.27	0.31	0.29	0.28	0.26	0.31	0.28	0.32	0.28
CV %		20	19	17	16	15	13	32	29	33	30	23	20
VR_{SA} (SA vs MI) %		−0.9	−7.0	−0.5	−3.6	1.1	6.1	−0.9	1.4	2.2	3.3	0.7	3.1

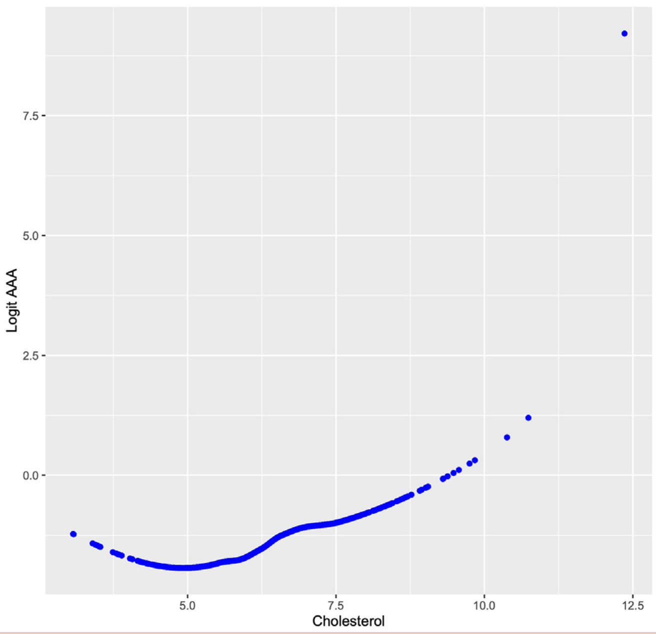
AAA, Abdominal aortic aneurysm; *aOR,* adjusted odds ratio; *CI,* confidence interval; *CC,* complete case; *CV %,* coefficient of variation of the aOR; *delta,* chosen value for delta; *MI,* multiple imputation; *SA,* sensitivity analysis; *SE,* standard error; TAA, thoracic aortic aneurysm; *VR_{MI} (MI vs CC) %,* variation rate of the aOR for CC and MI analyses; *VR_{SA} (SA vs MI) %,* variation rate of the aOR for MI and senstitivity analyses.
Sensitivity analysis after the multiple imputation according to Héraud-Bousquet et al: Practical considerations for sensitivity analysis after multiple imputation applied to epidemiological studies with incomplete data. BMC Medical Research Methodology 2012 and Carpenter et al: Sensitivity analysis after multiple imputation under missing at random: a weighting approach. Statistical Methods in Medical Research 2007.



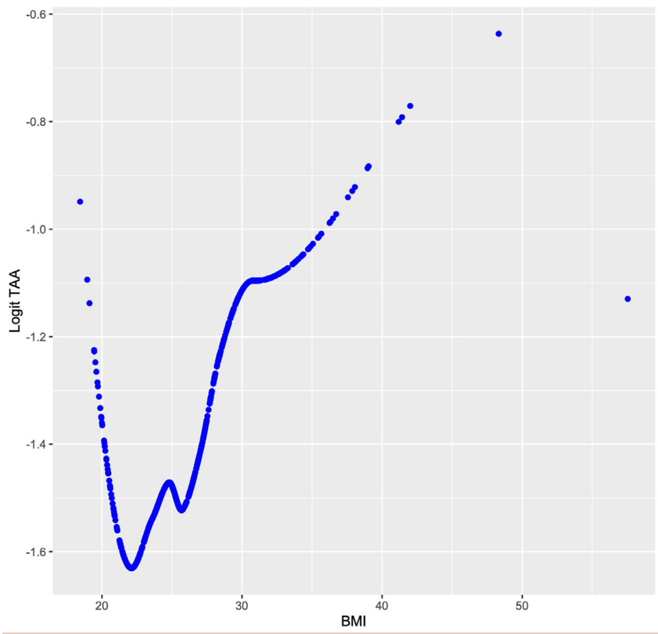
Supplementary Fig 1 (online only). Loess/lowess scatterplot for logit abdominal aortic aneurysm (*AAA*) and body mass index (*BMI*).



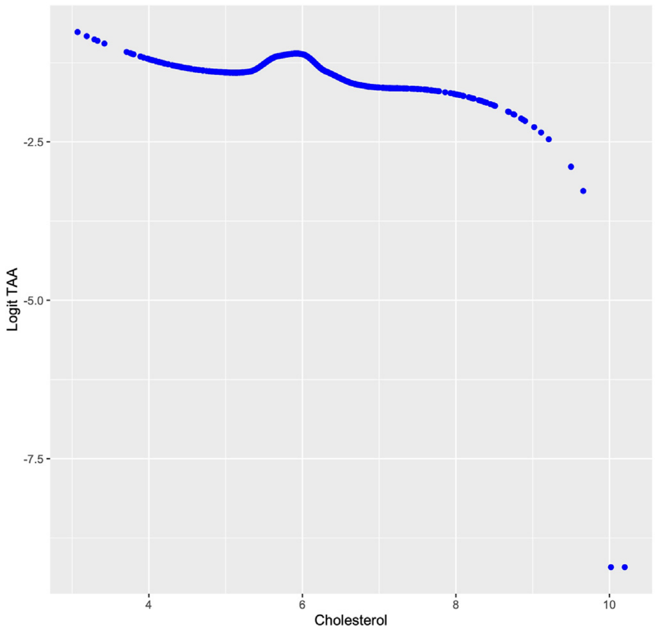
Supplementary Fig 3 (online only). Loess/lowess scatterplot for logit abdominal aortic aneurysm (*AAA*) and triglycerides



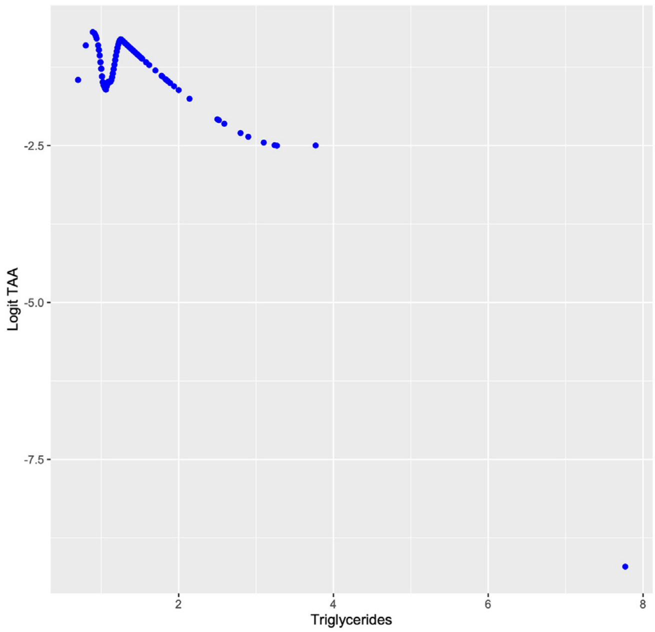
Supplementary Fig 2 (online only). Loess/lowess scatterplot for logit abdominal aortic aneurysm (*AAA*) and cholesterol.



Supplementary Fig 4 (online only). Loess/lowess scatterplot for logit thoracic abdominal aneurysm (*TAA*) and body mass index (*BMI*).



Supplementary Fig 5 (online only). Loess/lowess scatterplot for logit thoracic abdominal aneurysm (*TAA*) and cholesterol.



Supplementary Fig 6 (online only). Loess/lowess scatterplot for logit thoracic abdominal aneurysm (*TAA*) and triglycerides.