



Adiposity and the risk of aortic aneurysm: results from the UK Biobank, Japan-Specific Health Check-ups Study and a systematic review and meta-analysis of cohort studies

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Abstract

Overweight and obesity has been associated with increased risk of aortic aneurysm in some cohort studies, however, results have been inconsistent. To clarify this association we analysed data from two large cohort studies with a total of >1.1 million participants, and conducted a systematic review and meta-analysis of published cohort studies. The analytical cohorts included 495,438 participants in UK Biobank and 630,841 participants in the Japan Specific Health Checkups (J-SHC) Study. Multivariable Cox proportional hazards regression models were used to analyse associations between anthropometric factors and risk of aortic aneurysm. PubMed and Embase databases were searched for studies on adiposity and aortic aneurysm up to 18 March 2026, and random effects models were used to estimate summary relative risks (RRs) and 95% confidence intervals (CIs). In the UK Biobank, a total of 3,447 incident cases and 183 deaths from aortic aneurysm occurred during 12.3 years follow-up, and in the J-SHC Study, 51 aortic aneurysm deaths occurred during 4 years follow-up. In UK Biobank, HRs (95% CIs) were 1.18 (1.08–1.29), 1.51 (1.36–1.67), 1.45 (1.23–1.70), and 1.56 (1.20–2.02) for a BMI of 25–<30, 30–<35, 35–<40, and ≥ 40 vs. 18.5–<25.0, respectively. Positive associations were also observed for the highest vs. lowest quintile of waist circumference (1.66, 1.48–1.87), waist-to-hip ratio (1.67, 1.48–1.89), hip circumference (1.37, 1.22–1.53), waist-to-height ratio (1.70, 1.51–1.91), fat mass (1.62, 1.44–1.83) and body fat percentage (1.45, 1.28–1.63). In the J-SHC Study, comparing the highest vs. lowest categories the HRs were 1.79 (0.86–4.00, $p_{\text{trend}}=0.15$) for BMI, 3.12 (1.10–11.11, $p_{\text{trend}}=0.006$) for waist circumference, and 3.18 (1.17–8.64, $p_{\text{trend}}=0.01$) for waist-to-height ratio. In the meta-analysis, the summary RR per 5 kg/m² increment in BMI was 1.17 (1.13–1.21, $I^2=0\%$, $n=6$ studies) for aortic aneurysm, 1.27 (1.04–1.55, $I^2=33\%$, $n=3$ studies) for thoracic aortic aneurysm, and 1.11 (1.05–1.17, $I^2=55\%$, $n=12$ studies) for abdominal aortic aneurysm. The respective summary RRs for total, thoracic, and abdominal aortic aneurysm were 1.29 (0.98–1.68, $I^2=73\%$, $n=2$), 1.29 (0.92–1.82, $I^2=67\%$, $n=2$), and 1.15 (1.07–1.24, $I^2=49\%$, $n=6$) per 10 cm increase in waist circumference, and 1.37 (1.05–1.78, $I^2=45\%$, $n=2$), 1.47 (0.89–2.42, $I^2=62\%$, $n=2$), and 1.33 (1.25–1.42, $I^2=0\%$, $n=2$) per 0.1 unit increase in waist-to-height ratio. These results suggest that higher BMI and abdominal fatness are associated with a dose-related increase in risk of aortic aneurysm overall and across subtypes. Any further studies should further investigate the associations between adiposity measures and subtypes of aortic aneurysm.

Keywords Body mass index · Adiposity · Waist circumference · Aortic aneurysm · Prospective studies · Systematic review · Meta-analysis

Introduction

Aortic aneurysms are dilations of the aorta, which when ruptured have an 80% overall mortality rate, with about one third of individuals affected dying before reaching hospital [1], and 25–50% of cases undergoing surgery being fatal [2–5]. Survival has improved somewhat in more recent years according to some studies [2]. The Global Burden of Disease Project estimated that aortic aneurysms accounted for 167,200 deaths and 3 million disability-adjusted life years (DALYs) in 2017 [6], most of which occur in the abdomen, and only 3% occurring in the thorax [7]. Some of the established or suspected risk factors for aortic aneurysm include age [8], sex [9], family history [10], low education [9], cardiovascular disease [8, 9], hypertension [11, 12], blood lipids [13], low physical activity [14], and smoking [15, 16], while diabetes appears to be associated with a reduced risk [17], possibly related to protective effects from diabetes medications.

Although overweight and obesity, defined as having a body mass index (BMI) of $25 < \text{BMI} < 30$, and $\geq 30 \text{ kg/m}^2$, respectively, are established risk factors for a number of cardiovascular diseases including coronary heart disease [18], stroke [18], heart failure [19], atrial fibrillation [20], sudden cardiac death [21], and venous thromboembolism [22], fewer studies have been published on the association with aortic aneurysm [18, 23–34]. Studies on BMI and aortic aneurysm have shown mixed results [18, 23–33], with some reporting positive associations [18, 25, 27, 29, 33], however, several other studies reported no association [23, 24, 26, 28, 30–32]. Studies on waist circumference and risk of abdominal aortic aneurysm have tended to show positive associations [26, 34], although in two cohort studies the reported estimates were imprecise [26]. Most of these studies have had a moderate sample size and may have had limited statistical power to detect a modest association between adiposity and aortic aneurysm. In addition, some studies adjusted for risk factors that may be on the biological pathway between adiposity and the development of aortic aneurysm, including hypertension, lipids or cardiovascular disease, possibly leading to risk of over-adjustment of risk estimates. Given the relatively few modifiable risk factors that have been established for aortic aneurysm as well as the seriousness of the disease and the increasing prevalence globally of overweight and obesity [35], clarification of the potential influence from excess weight on aortic aneurysm could have important public health implications. We therefore investigated the association between adiposity and aortic aneurysm incidence and mortality in two large cohort studies and conducted a systematic review of the published prospective studies to summarize the available evidence.

Materials and methods

UK Biobank

The UK Biobank is a prospective cohort with 0.5 million people mostly aged 40 to 69 years at recruitment between 2006 and 2010 [25]. Participants were recruited using invitations mailed to 9 million people whose contact information was obtained from NHS central registers [25]. The purpose of the resource is to provide research opportunities which aim to improve prevention, diagnosis and treatment of a wide range of serious illnesses [25].

Anthropometric measurements, biological samples, and detailed touchscreen questionnaire data on diet, smoking, alcohol intake, socio-demographic characteristics, health and medical history have been collected from the participants in 22 assessment centres across the UK [25]. The database includes prolonged follow-up of participants for cause-specific morbidity and mortality in addition to linkages to NHS data [25, 26].

For this analysis, the dataset contained 501,941 participants after excluding subjects who withdrew during the follow-up. We further excluded participants if they had either a self-reported history of aortic aneurysm or a hospital diagnosis of aortic aneurysm at baseline ($n=476$), different self-reported compared to genetic sex ($n=370$), missing anthropometric data for BMI ($n=3,094$), waist circumference ($n=105$), hip circumference ($n=37$), and missing data on smoking status ($n=2421$), leaving 495,438 participants included for these analyses. Additional participants were excluded because of missing data on body fat mass ($n=7,961$) and body fat percentage ($n=7,398$), leaving 487,477 and 488,040 participants included in the respective analyses.

Anthropometric measurements were taken at the baseline visit by trained staff at the UK Biobank recruitment centers using a standard protocol [36]. Height was measured using Seca 240 cm height measure, with participants standing barefoot and head positioned in Frankfort plane. Waist circumference was measured at the narrowest part of the trunk or the level of the umbilicus at the end of normal expiration, and hip circumference was measured at the widest part of the buttocks (both were measured using Seca 200 cm tape). Weight and body composition measures (body fat percentage, fat mass) were estimated using Tanita BC418MA body composition analysers using bio-impedance analysis or standard scales if bio-impedance was not done [25]. Weight was measured in kilograms (kg), while height, waist, and hip measures were measured in centimeters (cm). This study included anthropometric measures of BMI, waist circumference, hip circumference, waist-to-height ratio, waist-to-hip ratio, fat mass and body fat percentage. BMI was calculated

by dividing weight by the square of height (kg/m^2). Waist-to-hip ratio was calculated by dividing waist circumference by hip circumference. Waist-to-height ratio was calculated by dividing waist circumference by height.

Incident cases of aortic aneurysm were identified by linkages to Hospital Episode Statistics for England (HES), Scottish Morbidity Record (SMR), and the Patient Episode Database for Wales (PEDW). In addition, participants who had aortic aneurysm listed as underlying cause of death were identified by linkages to NHS England (for England and Wales) and NHS Central Register, National Records of Scotland. Cases were identified according to the 10th revision of International Statistical Classification of Diseases (ICD-10) diagnostic codes I71.1-I71.9 and ICD-9 codes 441.1-441.9. Aortic aneurysm subtypes were classified as follows: thoracic aortic aneurysm (I71.1-I71.2), abdominal aortic aneurysm (I71.3-I71.4), thoracoabdominal aortic aneurysm (I71.5-I71.6), and unspecified site aortic aneurysm (I71.8-I71.9), ruptured aortic aneurysm (I71.1, I71.3, I71.5, I71.8), and non-ruptured aortic aneurysm (I71.2, I71.4, I71.6, I71.9). For participants with aortic aneurysm listed as underlying cause of death, but no prior hospital diagnosis, we used their date of death as censoring date, while for hospitalizations, we used the date of hospital diagnosis as censoring date. Participants were followed up from the date of recruitment at baseline and were censored at the date of aortic aneurysm diagnosis, death, loss to follow-up, or the last complete follow-up (30 September 2021 for England and Wales and 31 October 2021 for Scotland), whichever came first. The proportion of participants lost to follow-up was low ($<0.3\%$).

Multivariable Cox proportional hazards regression models were used to estimate hazard ratios (HRs) and 95% confidence intervals for the association between different measures of adiposity and risk of aortic aneurysm and its subtypes and mortality in UK Biobank. There was no violation of the proportional hazards assumption using Schoenfeld's test ($p=0.11$). Analyses were conducted by WHO categories of BMI, with cut-offs at <18.5 , 18.5 - <25.0 , 25.0 - <30.0 , 30.0 - <35.0 , 35.0 - <40.0 , ≥ 40.0 and per $5 \text{ kg}/\text{m}^2$ increase. The remaining adiposity measures were categorized by sex-specific quintiles and also modelled continuously for a relevant increment in each measure. Tests for linear trends were conducted by replacing the category number with the median value within each category and by including this variable as a continuous variable in the models. Sensitivity analyses were conducted excluding the first 5 years of follow-up to take into account potential reverse causation. Subgroup analyses were conducted stratified by sex, smoking status, and aortic aneurysm subtype, and interactions tested by incorporating cross-product terms between

each adiposity measure and sex or smoking and by using a Wald test.

Based on prior subject knowledge the main multivariable models included adjustment for age, sex, ethnicity (Caucasian or non-Caucasian, missing), Townsend deprivation index (quintiles, missing), education (college or university degree, other professional qualifications, national examination at ages 17–18 years, national examination at age 16 years, missing), a smoking variable combining smoking status, time since quitting smoking, and number of cigarettes per day (never, former & quit for ≥ 30 years, former & quit for 20 - <30 years, former & quit for 10 - <20 years, former & quit for <10 years, former & missing years since quitting, current & <10 cigarettes/day, current & 10 - <20 cigarettes/day, current & ≥ 20 cigarettes/day, current & missing number of cigarettes/day), alcohol consumption (never, special occasions only, 1–3 times/month, 1–2 times/week, 3–4 times/week, almost daily-daily), height (sex-specific quintiles), leisure-time physical activity (quintiles, missing). We did not adjust the analyses for variables (e.g. blood pressure/hypertension, blood lipids) that could be on the causal pathway between adiposity and aortic aneurysm to avoid over-adjustment. We conducted a further sensitivity analysis adjusting only for age, sex, region, smoking and alcohol consumption in the analyses to mirror the analysis in the Japan-Specific Health Check-ups Study. We also conducted a sensitivity analysis using complete case analyses.

Japan-Specific Health Check-ups Study

The Japan-Specific Health Check-ups (J-SHC) Study is based on health check-ups for all inhabitants in Japan aged 40–74 years old and is covered by the Japanese national health insurance system. The aim is to maintain a healthy life and prevent chronic lifestyle-related diseases. Persons who have been hospitalized for >6 months, living in a support facility or nursing home are excluded. Permission was obtained from the Ministry of Health, Labor and Welfare to link the participants in the study to a database of death certificates that records all deaths that occur in Japan. All participants were prospectively followed until they reached 75 years of age.

The current dataset included 664,933 men and women who participated in health check-ups from 2008 to 2015 in eastern and western Japan. Participants with missing data on BMI ($n=4,262$), waist circumference ($n=8,070$), smoking ($n=1,343$), and alcohol ($n=20,417$) were excluded, leaving 630,841 participants included in the statistical analysis. The outcome was death from aortic aneurysm, which was determined from a review of death certificates and classified according to ICD-10 codes I71.1-I71.9 (the same definitions were used as in UK Biobank for aortic aneurysm subsites

and subtypes). Participants were followed up from January 2008 to date of death, age 75 years, or April 1, 2015, whichever occurred first.

Statistical analyses were performed with the JMP software program, version 12 (SAS Institute, Cary, NC), and R 3.0.2 with additional packages, including Rcmdr, Epi, pROC, and PredictABEL. Multivariable Cox proportional hazards models were used to estimate HRs (95% CIs) for the association between BMI, waist circumference, and waist-to-height ratio and risk of aortic aneurysm death, adjusting for age, sex, region, smoking status (ever vs. never) and alcohol consumption (ever vs. never). P-values for linear trend were calculated using the Wald test. Subgroup analyses were conducted stratified by sex, smoking status, and aortic aneurysm subtype and interactions by sex and smoking status were tested by using cross-product terms and a Wald test.

Meta-analysis

We searched Pubmed and Embase databases for relevant studies published up to 9 March 2025 using the search strategy provided in the Supplementary Text. PRISMA criteria were followed for reporting meta-analyses [37]. We further searched the reference lists of the included publications for additional potentially relevant studies. Published retrospective cohort studies, prospective cohort studies and nested case-control studies within cohort studies reporting adjusted relative risk (RR) estimates and 95% confidence intervals (CIs) for the association between any measure of adiposity (BMI, waist circumference, hip circumference, waist-to-hip ratio, waist-to-height ratio, body fat mass or percentage) and the incidence or mortality of aortic aneurysm were included. Two studies that included cases of aortic dissection together with that of aortic aneurysm were excluded from the primary analysis [38, 39], but included in a sensitivity analysis. A list of studies excluded from the meta-analysis and the corresponding exclusion reasons is found in Supplementary Table 1. The literature screening was conducted manually and in duplicate by DA and YMS.

We extracted from each study the following information: The first author's last name, year of publication, country, study name, study period and duration of follow-up, sample size, sex, number of aortic aneurysm cases and/or deaths, adiposity measure, subgroup, RRs and 95% CIs and confounders adjusted for in the analysis. DA conducted the data extraction and it was checked for accuracy by YMS and any discrepancies were resolved by discussion between the authors.

We used random effects models to calculate summary RRs (95% CIs) of aortic aneurysm incidence or mortality per 5 kg/m² increment in BMI, per 10 cm increment in waist

circumference, and per 0.1 unit increment in waist-to-height ratio (i.e., the three adiposity exposures with two or more studies available) [40]. The average of the natural logarithm of the RRs was estimated and the RR from each study was weighted using random effects weights. The method of Greenland and Longnecker [41] was used to estimate linear trends across categories of BMI, waist circumference, and waist-to-height ratio. For studies that reported the median or mean values per category of each adiposity measure, these were used directly, while for studies only reporting ranges of adiposity measures, we estimated the midpoint for each category as the average of the lower and upper cut-off point. When extreme categories were open-ended, we estimated an upper and lower cut-off value using the width of the adjacent category, however, when the WHO categories of BMI were used, we used 18.5 as a lower cut-off if the upper cut-off was < 25, and we used 15 as the lower cut-off if the upper cut-off was < 18.5. We investigated a potential non-linear association between BMI, waist circumference, and waist-to-height ratio and risk of aortic aneurysm using fractional polynomial models [42, 43], and determined the best fitting second order fractional polynomial regression model, defined as the one with the lowest deviance. We used a likelihood ratio test to test for nonlinearity [42].

Heterogeneity between studies was evaluated using Q and I² statistics [44]. I² is a measure of how much of the heterogeneity that is due to between study variation rather than chance. We analysed all studies combined and investigated sources of heterogeneity by conducting analyses stratified by study characteristics such as sex, duration of follow-up, geographic location, number of cases, study quality and by adjustment for confounding factors for analyses with at least six studies included. Study quality was assessed using a modified version of the Newcastle Ottawa scale which rates studies according to selection, comparability and outcome assessment [45], and the modifications have been described previously [46]. We calculated E-values for selected values from the nonlinear dose-response analyses to obtain an estimate of at least how strong an unmeasured confounder would have to be associated with both adiposity and aortic aneurysm to explain away the observed associations [47]. Publication bias was assessed using Egger's test [48] and by inspection of funnel plots. The statistical analyses were conducted using the software package Stata, version 16.0 software (StataCorp, Texas, US).

We used World Cancer Research Fund criteria to assess the likelihood of causality [49]. These criteria take into account the strength and precision of the association, number of studies, heterogeneity, dose-response relationship, quality of the studies, data from different study types, biological mechanisms and experimental data. The evidence is graded as substantial effect on risk unlikely, limited-no

conclusion, limited-suggestive, probable, and convincing evidence of a causal relationship [49].

Results

UK Biobank

A total of 495,438 participants were included in the analysis. More women than men and non-whites than whites were in the highest BMI categories (Table 1). Participants with a high BMI tended to have a higher level of deprivation, lower education, were less likely to be current smokers, and less likely to consume alcohol daily or almost daily, but more likely to be former smokers, and had a lower level of physical activity and a slightly lower height than those with a normal BMI. There was little difference in age across BMI categories, except for a slightly younger age in the underweight and grade 3 obesity categories than in the intermediate categories (Table 1).

During an average of 12.3 years of follow-up (6,086,459 person-years for incidence and 6,099,662 person-years for mortality), 3,447 aortic aneurysm cases and 183 aortic aneurysm deaths occurred. The multivariable HRs (95% CIs) were 1.18 (1.08–1.29), 1.51 (1.36–1.67), 1.45 (1.23–1.70), and 1.56 (1.20–2.02), $p_{\text{trend}} < 0.001$ for BMI categories of 25–<30, 30–<35, 35–<40, and 40 vs. 18.5–<25 kg/m², and 1.16 (1.12–1.21) per 5 kg/m² (Table 2). The association was similar when excluding the first 5 years of follow-up (Supplementary Table 2). The association was more apparent among men than in women (1.21, 1.16–1.27 per 5 kg/m² in men vs. 1.03, 0.96–1.11 in women, $p_{\text{interaction}} < 0.0001$), and for never (1.18, 1.10–1.27) and former (1.22, 1.16–1.29) smokers than in current smokers (1.05, 0.97–1.13, $p_{\text{interaction}} = 0.04$, Supplementary Table 2). When stratified by subtype, positive associations were observed for thoracic (1.17, 1.08–1.26 per 5 kg/m² increment in BMI), abdominal (1.18, 1.13–1.24), and unspecified site (1.14, 1.04–1.26, Table 2), ruptured (1.20, 1.04–1.40) and non-ruptured (1.16, 1.12–1.21) aortic aneurysm (Supplementary Table 2). A positive association was also observed for aortic aneurysm mortality (1.18, 1.01–1.38 per 5 kg/m², Supplementary Table 2).

The HR for increasing quintiles of waist circumference were 1.00 (ref.), 1.11 (0.98–1.26), 1.29 (1.14–1.46), 1.37 (1.21–1.54), and 1.66 (1.48–1.87), $p_{\text{trend}} < 0.001$ and per 10 cm was 1.16 (1.13–1.19) (Table 3). The association remained similar when excluding the first five years of follow-up (Supplementary Table 3). The association was more pronounced in men than in women (HR per 10 cm: 1.19, 1.15–1.23 in men vs. 1.07, 1.00–1.13 in women, $p_{\text{interaction}} < 0.0001$), and among never (1.17, 1.11–1.24) and

Table 1 Body mass index and participant characteristics in the UK Biobank

	Participants (n)	BMI (kg/m ²)					
		<18.5	18.5-<25.0	25.0-<30.0	30.0-<35.0	35.0-<40.0	≥40.0
Age (years), median		2609	161,346	210,483	86,690	24,710	9600
Sex (%)		56	57	58	58	58	56
	Men	20.8	34.9	52.9	51.1	40.1	31.4
	Women	79.2	65.1	47.1	48.9	59.9	68.6
Ethnicity (%)		93.1	94.8	94.4	93.8	93.5	92.5
	White						
	Non-white	6.3	4.9	5.2	5.8	6.2	7.0
	Missing	0.6	0.3	0.4	0.4	0.3	0.5
Townsend Deprivation Index, highest (%)		27.4	17.8	18.2	22.9	28.2	34.9
Education, highest (%)		40.5	39.4	31.2	25.1	22.8	21.5
Smoking status (%)		56.3	59.1	53.6	50.8	51.3	53.1
	Never						
	Former	20.9	29.6	36.1	39.3	39.3	37.6
	Current	22.8	11.3	10.3	9.9	9.4	9.3
Alcohol consumption, daily/almost daily (%)		22.2	22.6	21.4	17.2	11.9	7.5
Leisure-time physical activity, frequency/week (median)		12	12	11	11	10	9
Height (cm), women/men (median)		164/175	163/176	162/175.6	161/175	161/175	161/174.5

Table 2 BMI and aortic aneurysm in the UK Biobank

	BMI, kg/m ²					P _{trend}		Per 5 kg/m ²
Cut-offs	<18.5	18.5-25.0	25.0-<30.0	30.0-<35.0	35.0-<40.0	≥40.0		
Median	17.88	23.14	27.20	31.78	36.75	42.65		
Participants	2609	161,346	210,483	86,690	24,710	9600		495,438
Person-years	31032.7	1991815.4	258847.4	1058747.6	300181.2	115834.5		6086458.8
Cases, AA	14	747	1590	840	194	62		3447
HR (95% CI)	1.32 (0.78-2.24)	1.00	1.18 (1.08-1.29)	1.51 (1.36-1.67)	1.45 (1.23-1.70)	1.56 (1.20-2.02)	<0.001	1.16 (1.12-1.21)
Cases, TAA	-	199	349	172	38	-		795
HR (95% CI)	1.53 (0.57-4.12)	1.00	1.14 (0.96-1.36)	1.48 (1.21-1.83)	1.46 (1.04-2.04)	1.76 (1.05-2.96)	<0.001	1.17 (1.08-1.26)
Cases, AAA	9	410	985	528	126	38		2096
HR (95% CI)	1.49 (0.77-2.89)	1.00	1.25 (1.11-1.40)	1.56 (1.37-1.79)	1.57 (1.28-1.92)	1.66 (1.19-2.32)	<0.001	1.18 (1.13-1.24)
Cases, UAA	-	127	235	134	25	-		529
HR (95% CI)	0.58 (0.08-4.16)	1.00	1.09 (0.88-1.36)	1.54 (1.20-1.97)	1.18 (0.76-1.82)	1.07 (0.50-2.31)	0.014	1.14 (1.04-1.26)

AA aortic aneurysm, AAA abdominal aortic aneurysm, CI confidence interval, HR hazard ratio, TAA thoracic aortic aneurysm, UAA unspecified site aortic aneurysm. The number of cases has

Adjusted for age, sex, ethnicity, Townsend deprivation index, education, smoking status, time since quitting smoking and cigarettes/day, alcohol consumption, frequency of physical activity, height

former (1.21, 1.16–1.26) smokers than in current smokers (1.07, 1.01–1.13, $p_{\text{interaction}}=0.08$, Supplementary Table 3). Results were similar across subtypes including thoracic (HR per 10 cm: 1.14, 1.07–1.21), abdominal (1.19, 1.15–1.23), and unspecified site (1.11, 1.03–1.19) (Table 3), ruptured (1.29, 1.15–1.44), and non-ruptured (1.15, 1.12–1.19) aortic aneurysm (Supplementary Table 3). A positive association was also observed for aortic aneurysm mortality (1.25, 1.11–1.41 per 10 cm, Supplementary Table 3).

Similarly, positive associations were observed between most other adiposity measures including hip circumference, waist-to-hip ratio, waist-to-height ratio, fat mass and fat percentage and risk of aortic aneurysm and most subtypes, as well as aortic aneurysm mortality (Table 3, Supplementary Tables 4–8). Similar interactions were observed by sex and smoking status for most of these adiposity measures as for BMI and waist circumference (Supplementary Tables 4–8). When adjusting for the same variables as in the J-SHC Study (age, sex, region, alcohol and smoking), the observed associations largely remained similar or were slightly stronger (Supplementary Tables 2–8). The results remained similar in complete case analyses (Supplementary Tables 2–8).

J-SHC Study

A total of 630,841 participants were included in the analysis. Participants with higher BMI had a higher prevalence of ever alcohol drinking and dyslipidemia and higher systolic and diastolic blood pressure, but there was little difference in age and smoking status (Table 4). When compared to the UK Biobank participants, the J-SHC participants had a lower median BMI (23.1 vs. 26.7 kg/m²), higher prevalence of never smokers (84% vs. 55%), and higher prevalence of never alcohol drinkers (57% vs. 8%).

Over a median follow-up of 4 years (2,339,389 person-years), 51 aortic aneurysm deaths occurred. The HRs for increasing tertiles of BMI were 1.66 (0.79–3.72) and 1.79 (0.86–4.00) compared with the lowest tertile ($p_{\text{trend}}=0.15$, Table 5). A positive association was observed among never smokers with a HR of 1.46 (1.09–1.96) per 5 kg/m², but not among ever smokers (0.59, 0.25–1.38), and there was a suggestive test for interaction by smoking status ($p=0.08$, Supplementary Table 9). The associations between BMI and thoracic and aortic aneurysm mortality were not statistically significant (Table 5). Waist circumference was positively associated with aortic aneurysm mortality for the highest vs. lowest quintile with a HR of 3.12 (1.10–11.11, $p_{\text{trend}}=0.006$, Table 6). This association appeared to be more pronounced in never smokers (HR per 10 cm increment: 1.77, 1.27–2.47) than in ever smokers (1.07, 0.60–1.89), however, the test for interaction was not clear ($p=0.20$, Supplementary Table 10). The highest vs. lowest quintile of waist-to-height ratio

Table 3 Waist, hip and fat mass measures and aortic aneurysm in the UK Biobank

	Waist-to-height ratio					p_{trend}	Per 0.1 unit
Median (all/women/men)	73.0/71.0/83.0	80.0/78.0/91.0	86.0/83.0/96.0	94.0/90.0/102.0	107.0/101.0/111.0		
Participants	102,616	101,267	95,067	96,681	99,807		495,438
Person-years	1269014.7	1251729.7	1171096.0	1185333.7	1209284.7		6086458.8
Cases, AA	423	559	663	792	1010		3447
HR (95% CI)	1.00	1.11 (0.98-1.26)	1.29 (1.14-1.46)	1.37 (1.21-1.54)	1.66 (1.48-1.87)	<0.001	1.16 (1.13-1.19)
Cases, TAA	120	146	161	170	198		795
HR (95% CI)	1.00	1.10 (0.86-1.40)	1.25 (0.99-1.59)	1.25 (0.99-1.59)	1.47 (1.17-1.87)	0.001	1.14 (1.07-1.21)
Cases, AAA	236	308	380	512	660		2096
HR (95% CI)	1.00	1.06 (0.89-1.25)	1.25 (1.06-1.48)	1.46 (1.25-1.70)	1.75 (1.50-2.04)	<0.001	1.19 (1.15-1.23)
Cases, UAA	66	96	114	106	147		529
HR (95% CI)	1.00	1.26 (0.92-1.72)	1.50 (1.11-2.04)	1.27 (0.93-1.73)	1.70 (1.26-2.30)	0.001	1.11 (1.03-1.19)
<i>Hip circumference, cm</i>							
Median (all/women/men)	93/92/95	99/98/100	102/102/103	107/107/107	115/117/113	p_{trend}	Per 10 cm
Participants	103,518	97,894	101,148	98,917	93,961		495,438
Person-years	1264682.6	1204429.7	1248803.9	1218358.8	1150183.8		6086458.8
Cases, AA	609	661	647	749	781		3447
HR (95% CI)	1.00	1.03 (0.92-1.15)	1.12 (1.00-1.25)	1.13 (1.01-1.26)	1.37 (1.22-1.53)	<0.001	1.12 (1.07-1.16)
Cases, TAA	140	147	142	194	172		795
HR (95% CI)	1.00	0.99 (0.78-1.25)	1.00 (0.79-1.26)	1.26 (1.01-1.58)	1.31 (1.04-1.65)	0.005	1.11 (1.02-1.20)
Cases, AAA	381	418	392	426	479		2096
HR (95% CI)	1.00	1.06 (0.92-1.22)	1.13 (0.98-1.31)	1.05 (0.91-1.21)	1.37 (1.19-1.58)	<0.001	1.12 (1.06-1.18)
Cases, UAA	83	91	105	123	127		529
HR (95% CI)	1.00	1.02 (0.76-1.38)	1.27 (0.95-1.70)	1.32 (0.99-1.76)	1.57 (1.17-2.09)	<0.001	1.17 (1.06-1.29)
<i>Waist-to-hip ratio</i>							
Median (all/women/men)	0.753/0.731/0.854	0.792/0.777/0.901	0.829/0.813/0.934	0.872/0.853/0.968	0.981/0.910/1.019	p_{trend}	Per 0.1 unit
Participants	98,129	99,826	100,213	98,261	99,009		495,438
Person-years	1221211.0	1238170.8	1235210.1	1201866.3	1190000.6		6086458.8
Cases, AA	379	509	645	848	1066		3447
HR (95% CI)	1.00	1.11 (0.97-1.27)	1.24 (1.09-1.41)	1.51 (1.34-1.71)	1.67 (1.48-1.89)	<0.001	1.33 (1.26-1.40)
Cases, TAA	129	141	149	179	197		795
HR (95% CI)	1.00	1.01 (0.80-1.28)	1.02 (0.81-1.30)	1.23 (0.98-1.55)	1.32 (1.05-1.66)	0.004	1.22 (1.09-1.35)
Cases, AAA	168	279	394	526	729		2096
HR (95% CI)	1.00	1.29 (1.07-1.57)	1.55 (1.29-1.85)	1.84 (1.54-2.19)	2.14 (1.80-2.53)	<0.001	1.44 (1.35-1.54)
Cases, UAA	80	81	99	135	134		529
HR (95% CI)	1.00	0.89 (0.65-1.21)	1.00 (0.74-1.35)	1.31 (0.99-1.74)	1.20 (0.90-1.60)	0.028	1.11 (0.97-1.27)
<i>Waist-to-height ratio</i>							
Median (all/women/men)	0.445/0.429/0.477	0.487/0.473/0.517	0.529/0.512/0.546	0.569/0.556/0.580	0.633/0.630/0.636	p_{trend}	Per 0.1 unit
Participants	102,173	96,046	98,861	99,299	99,059		495,438
Person-years	1264064.0	1188152.9	1218610.1	1217434.6	1198197.2		6086458.8
Cases, AA	433	529	661	815	1009		3447

Table 3 (continued)

	Waist-to-height ratio					p_{trend}	Per 0.1 unit
HR (95% CI)	1.00	1.18 (1.04-1.34)	1.26 (1.11-1.42)	1.42 (1.26-1.60)	1.70 (1.51-1.91)	<0.001	1.27 (1.21-1.34)
Cases, TAA	130	147	149	180	189		795
HR (95% CI)	1.00	1.19 (0.94-1.50)	1.12 (0.88-1.43)	1.34 (1.06-1.69)	1.48 (1.17-1.87)	0.001	1.23 (1.11-1.36)
Cases, AAA	219	287	401	513	676		2096
HR (95% CI)	1.00	1.22 (1.02-1.45)	1.39 (1.18-1.64)	1.57 (1.33-1.84)	1.92 (1.64-2.24)	<0.001	1.33 (1.24-1.41)
Cases, UAA	83	87	101	119	139		529
HR (95% CI)	1.00	1.06 (0.79-1.44)	1.11 (0.83-1.49)	1.24 (0.93-1.65)	1.45 (1.09-1.92)	0.006	1.20 (1.05-1.36)
	<i>Fat mass, kg</i>						
Median (all/women/men)	14.3/15.8/13.1	19.3/20.9/17.8	23.5/25.2/21.3	28.1/30.5/25.2	37.2/40.1/32.6	p_{trend}	Per 10 kg
Participants	97,160	97,247	96,526	98,205	98,339		487,477
Person-years	1199421.2	1200713.8	1189874.9	1206465.8	1196465.4		5992941.2
Cases, AA	431	587	643	766	925		3352
HR (95% CI)	1.00	1.23 (1.08-1.39)	1.29 (1.14-1.45)	1.37 (1.21-1.54)	1.62 (1.44-1.83)	<0.001	1.16 (1.11-1.21)
Cases, TAA	125	163	134	171	180		773
HR (95% CI)	1.00	1.22 (0.97-1.55)	1.00 (0.78-1.28)	1.21 (0.96-1.53)	1.33 (1.05-1.68)	0.039	1.11 (1.02-1.20)
Cases, AAA	224	335	405	471	601		2036
HR (95% CI)	1.00	1.32 (1.12-1.57)	1.51 (1.28-1.78)	1.51 (1.29-1.78)	1.87 (1.60-2.19)	<0.001	1.20 (1.14-1.26)
Cases, UAA	78	84	95	119	140		516
HR (95% CI)	1.00	0.98 (0.72-1.33)	1.07 (0.79-1.45)	1.23 (0.92-1.64)	1.42 (1.07-1.90)	0.003	1.12 (1.02-1.24)
	<i>Fat percentage, %</i>						
Median (all/women/men)	21.4/27.6/17.8	31.2/33.1/22.5	35.3/36.8/25.4	38.9/40.4/28.3	43.0/45.3/32.5	p_{trend}	Per 10 kg
Participants	97,788	98,861	97,577	97,046	96,768		488,040
Person-years	1209499.8	1222158.9	1202306.2	1191644.0	1173875.9		5999484.9
Cases, AA	424	573	673	747	938		3355
HR (95% CI)	1.00	1.16 (1.02-1.31)	1.24 (1.10-1.41)	1.27 (1.12-1.43)	1.45 (1.28-1.63)	<0.001	1.20 (1.13-1.27)
Cases, TAA	130	174	150	155	164		773
HR (95% CI)	1.00	1.23 (0.98-1.54)	1.04 (0.82-1.32)	1.06 (0.84-1.35)	1.13 (0.89-1.44)	0.72	1.03 (0.91-1.17)
Cases, AAA	208	306	425	472	628		2039
HR (95% CI)	1.00	1.22 (1.02-1.46)	1.49 (1.26-1.76)	1.46 (1.24-1.73)	1.68 (1.43-1.97)	<0.001	1.31 (1.21-1.42)
Cases, UAA	80	89	91	114	142		516
HR (95% CI)	1.00	0.98 (0.73-1.33)	0.95 (0.70-1.29)	1.14 (0.85-1.52)	1.34 (1.01-1.78)	0.02	1.16 (1.00-1.35)

AA aortic aneurysm, AAA abdominal aortic aneurysm, CI confidence interval, HR hazard ratio, TAA thoracic aortic aneurysm, UAA unspecified site aortic aneurysm

Adjusted for age, sex, ethnicity, Townsend deprivation index, education, smoking status, time since quitting smoking and cigarettes/day, BMI categories, frequency of physical activity, height

was positively associated with aortic aneurysm mortality (HR=3.18, 1.17–8.64, p_{trend} =0.01, Table 6), and the association appeared to be more pronounced in never smokers (HR per 0.1 unit increment: 2.13, 1.27–3.57) compared to ever smokers (0.98, 0.37–2.59), but the test for interaction

was not clear (p =0.20, Supplementary Table 11). Across all analyses there was no indication of a statistical interaction by sex ($p \geq 0.74$ for all comparisons). Both waist circumference and waist-to-height ratio were positively associated

Table 4 BMI and study participant characteristics in the Japan-Specific Health Check-ups Study

		BMI (kg/m ²)		
Cut-offs (tertiles)		<21.8	21.8–<24.5	≥24.5
Median (all/women/men)		20.2/20.5/20.1	23.1/23.1/23.0	26.4/26.3/26.5
Participants (n)		205,551	209,178	216,112
Age, years (median)		64	65	65
Sex, %	Men	30.8	46.5	50.7
	Women	69.2	53.5	49.3
Smoking, %	Never	84.0	84.8	84.5
	Ever	16.0	15.2	15.5
Alcohol, %	Never	59.4	54.7	57.5
	Ever	40.6	45.3	42.5
Dyslipidemia, %	No	57.6	42.5	32.3
	Yes	42.4	57.5	67.7
Systolic blood pressure, mmHg, median		124	129	132
Diastolic blood pressure, mmHg, median		73	77	80

with thoracic aortic aneurysm, but not clearly associated with abdominal aortic aneurysm (Table 6).

Meta-analysis

Data from 16 cohort studies were included in the meta-analysis, including 14 published cohort studies (14 publications, 14 risk estimates) [18, 23–27, 29–34, 50] and the UK Biobank and J-SHC study (Fig. 1, Supplementary Table 12). Two of the studies [18, 29] were identified by separate searches from other projects. One publication reported results for two different cohort studies (Cohort of Swedish Men and Swedish Mammography Cohort) [26]. Six studies included men, one included women and nine studies included both men and women. Eleven studies reported results for aortic aneurysm incidence, four studies reported results for mortality, and one study (UK Biobank, herein) reported on both aortic aneurysm incidence and mortality.

Nine studies were from Europe, four studies were from the US, and three studies were from Asia (Supplementary Table 12).

Six studies (including four published studies [18, 23, 31, 51], J-SHC study and UK Biobank) with 8,185 cases and 4,961,358 participants were included in the meta-analysis on BMI and aortic aneurysm, three studies (including one published study [31], J-SHC study and UK Biobank) with 869 cases and 1,230,251 participants were included for BMI and thoracic aortic aneurysm, and twelve studies (including ten published studies/nine publications [24–27, 29–33], J-SHC, and UK Biobank) with 5,580 cases and 1,491,750 participants were included for BMI and abdominal aortic aneurysm. In the meta-analysis, the summary RR per 5 kg/m² increment in BMI (95% CI) was 1.17 (1.13–1.21, I² = 0%) for aortic aneurysm (Fig. 2a), 1.27 (1.04–1.55, I² = 33%) for thoracic aortic aneurysm (Fig. 2c), and 1.11 (1.05–1.17, I² = 55%) for abdominal aortic aneurysm (Fig. 2e). In the analysis of BMI and aortic aneurysm, the summary RR per 5 kg/m² increment ranged from 1.16 (1.13–1.20) when excluding the study by Takada et al. [31], to 1.19 (1.13–1.25) when excluding the UK Biobank (Supplementary Fig. 1). In the analysis of BMI and abdominal aortic aneurysm, the summary RR per 5 kg/m² increment ranged from 1.09 (1.03–1.16) when excluding the UK Biobank, to 1.12 (1.07–1.18) when excluding the Cohort of Swedish Men [26] (Supplementary Fig. 2). There was no indication of publication bias for aortic aneurysm ($p = 0.62$) or abdominal aortic aneurysm ($p = 0.39$) with Egger's test or by inspection of the funnel plots (Supplementary Figs. 3 and 4). There was indication of nonlinearity for BMI and aortic aneurysm ($p_{\text{nonlinearity}} = 0.04$) (Fig. 2b, Supplementary Table 13), thoracic aortic aneurysm ($p_{\text{nonlinearity}} = 0.11$) (Fig. 2d, Supplementary Table 13), and abdominal aortic aneurysm ($p_{\text{nonlinearity}} = 0.02$) (Fig. 2f, Supplementary Table 13). The summary RR was 1.21 (1.05–1.39, I² = 0%, $n = 2$) per 5 kg/m² for ruptured aortic aneurysm and the test for nonlinearity was not significant ($p = 0.25$) (Supplementary Fig. 5). Separate analyses of aortic aneurysm incidence and mortality gave summary RR of 1.16 (1.12–1.21, $n =$

Table 5 BMI and aortic aneurysm mortality in the Japan-Specific Health Check-ups Study

		BMI (kg/m ²)			P _{trend}	Per 5 kg/m ²
Cut-offs		<21.8	21.8–<24.5	≥24.5		
Median (all/women/men)		20.2/20.5/20.1	23.1/23.1/23.0	26.4/26.3/26.5	-	-
Participants		205,551	209,178	216,112	-	630,841
Person-years		774,582	786,111	778,696	-	2,339,389
Deaths, AA		10	20	21	-	51
HR (95% CI)		1.00	1.66 (0.79–3.72)	1.79 (0.86–4.00)	0.15	1.24 (0.84–1.85)
Deaths, TAA		5	8	10	-	23
HR (95% CI)		1.00	1.32 (0.44–4.40)	1.66 (0.58–5.38)	0.35	1.33 (0.79–2.27)
Deaths, AAA		4	9	8	-	21
HR (95% CI)		1.00	1.96 (0.63–7.27)	1.87 (0.58–7.05)	0.36	1.14 (0.59–2.19)

AA aortic aneurysm, AAA abdominal aortic aneurysm, CI confidence interval, HR hazard ratio, TAA thoracic aortic aneurysm
Adjusted for age, sex, smoking, alcohol, region

Table 6 Waist circumference and waist-to-height ratio and aortic aneurysm mortality in the Japan-Specific Health Check-ups Study

<i>Waist circumference (cm)</i>							
Median (all/women/men)	72/75/70	79/81/77	84/85/82	88.4/89.4/87.5	95.5/96/99.5	P _{trend}	Per 10 cm
Participants	123,576	120,381	129,191	126,242	131,451	-	630,841
Person-years	462,596	453,303	485,614	469,277	468,599	-	2,339,389
Deaths, AA	4	4	14	16	13	-	51
HR (95% CI)	1.00	0.99 (0.23-4.19)	3.31 (1.18-11.67)	4.05 (1.48-14.14)	3.12 (1.10-11.11)	0.006	1.54 (1.15-2.05)
Deaths, TAA	2	1	5	9	6	-	23
HR (95% CI)	1.00	0.49 (0.02-5.11)	2.32 (0.50-16.23)	4.47 (1.15-29.40)	2.83 (0.65-19.34)	0.04	1.65 (1.09-2.50)
Deaths, AAA	2	2	7	4	6	-	21
HR (95% CI)	1.00	1.01 (0.12-8.42)	3.41 (0.82-22.94)	2.08 (0.41-15.07)	3.05 (0.70-20.83)	0.12	1.45 (0.92-2.29)
<i>Waist-to-height ratio</i>							
Median (all/women/men)	0.457/0.456/0.458	0.500/0.494/0.507	0.530/0.519/0.542	0.564/0.545/0.578	0.616/0.586/0.633	P _{trend}	Per 0.1 unit
Participants	126,101	126,203	125,869	126,326	126,342	-	630,841
Person-years	473,097	478,437	473,728	468,688	445,439	-	2,339,389
Deaths, AA	5	6	14	8	18	-	51
HR (95% CI)	1.00	1.10 (0.33-3.60)	2.46 (0.88-6.83)	1.36 (0.44-4.16)	3.18 (1.17-8.64)	0.01	1.75 (1.10-2.79)
Deaths, TAA	2	2	7	2	10	-	23
HR (95% CI)	1.00	0.91 (0.13-6.45)	3.03 (0.63-14.68)	0.84 (0.12-6.00)	4.39 (0.95-20.24)	0.03	2.13 (1.11-4.10)
Deaths, AAA	3	3	6	3	6	-	21
HR (95% CI)	1.00	0.93 (0.19-4.61)	1.80 (0.45-7.25)	0.88 (0.17-4.40)	1.86 (0.46-7.55)	0.40	1.51 (0.72-3.15)

AA aortic aneurysm, AAA abdominal aortic aneurysm, CI confidence interval, HR hazard ratio, TAA thoracic aortic aneurysm

Adjusted for age, sex, smoking, alcohol, region

2) for aortic aneurysm incidence (Supplementary Fig. 6a) and 1.19 (1.13–1.25, $n = 5$) for aortic aneurysm mortality (Supplementary Fig. 6c), and there was indication of a non-linear positive dose-response relationship both for incidence ($p_{\text{nonlinearity}} < 0.001$) and for mortality ($p_{\text{nonlinearity}} = 0.06$), with a flattening of the dose-response curve at higher BMI levels for incidence (Supplementary Fig. 6b), but a monotonic positive association from a BMI around 20 for mortality (Supplementary Fig. 6d).

Two studies (J-SHC and UK Biobank) were included in the analysis of waist circumference and aortic aneurysm (3,498 cases, 1,126,279 participants) and thoracic aortic aneurysm (818 cases, 1,126,279 participants), and six studies including four published studies (three publications [26, 34, 50], J-SHC study, and UK Biobank) were included on abdominal aortic aneurysm (3,314 cases, 1,222,439 participants). The summary RR per 10 cm increase in waist circumference was 1.29 (0.98–1.68, $I^2 = 73\%$) for aortic aneurysm

(Supplementary Fig. 7), 1.29 (0.92–1.82, $I^2 = 67\%$) for thoracic aortic aneurysm (Fig. 3a), and 1.15 (1.07–1.24, $I^2 = 49\%$) for abdominal aortic aneurysm (Fig. 3c). The summary RR for abdominal aortic aneurysm ranged from 1.12 (1.03–1.22) when excluding UK Biobank to 1.19 (1.15–1.23) when excluding the Cohort of Swedish Men [26] (Supplementary Fig. 8). There was no indication of publication bias with Egger's test ($p = 0.91$) or by visual inspection of the funnel plot (Supplementary Fig. 9). There was no indication of nonlinearity for the association between waist circumference and thoracic ($p_{\text{nonlinearity}} = 0.54$) or abdominal ($p_{\text{nonlinearity}} = 0.32$) aortic aneurysm (Fig. 3b and d, Supplementary Table 14), however, the nonlinear analysis did not converge for aortic aneurysm. The summary RR per 10 cm increment in waist circumference was 1.33 (1.17–1.52, $I^2 = 15\%$, $n = 2$) for ruptured aortic aneurysm (Supplementary Fig. 10, the nonlinear analysis did not converge). Because the J-SHC study assessed aortic aneurysm mortality and not

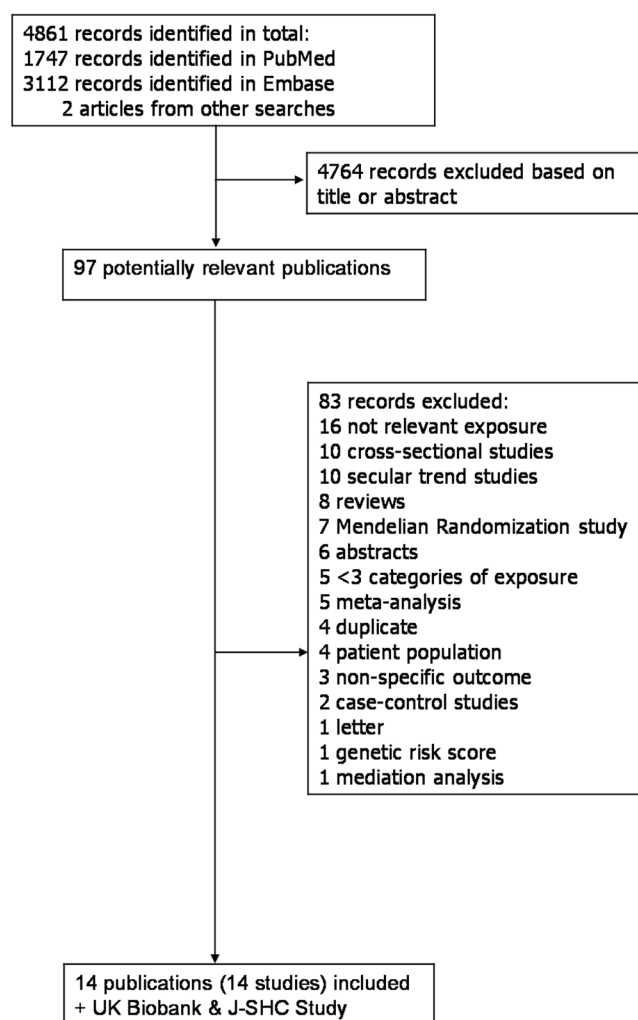


Fig. 1 Flow-chart of study selection

incidence, we repeated the analysis of waist circumference and aortic aneurysm using mortality data in UK Biobank and the summary RR was 1.33 (1.10–1.60, $I^2 = 42\%$, $n = 2$, Supplementary Fig. 11).

Two studies (3,498 aortic, 818 thoracic aortic, and 2,117 abdominal aortic aneurysm cases, 1,126,279 participants) were included in the analysis of waist-to-height ratio and aortic aneurysm overall and for subtypes (J-SHC and UK Biobank). The summary RR per 0.1 unit increment in waist-to-height ratio was 1.37 (1.05–1.78, $I^2 = 45\%$, $n = 2$) for aortic aneurysm (Fig. 4a), 1.47 (0.89–2.42, $I^2 = 62\%$, $n = 2$) for thoracic aortic aneurysm (Fig. 4c), and 1.33 (1.25–1.42, $I^2 = 0\%$, $n = 2$) for abdominal aortic aneurysm (Fig. 4e). There was no indication of nonlinearity for aortic ($p_{\text{nonlinearity}} = 0.99$, Supplementary Table 15, Fig. 4b), thoracic aortic ($p_{\text{nonlinearity}} = 0.99$, Supplementary Table 15, Fig. 4d), or abdominal aortic aneurysm ($p_{\text{nonlinearity}} = 0.51$, Supplementary Table 15, Fig. 4f). The summary RR per 0.1 unit increment in waist-to-height ratio was 1.55 (1.29–1.85,

$I^2 = 0\%$, $n = 2$) for ruptured aortic aneurysm, and there was no indication of nonlinearity ($p = 0.59$) (Supplementary Fig. 12).

We repeated the analysis of waist-to-height ratio and aortic aneurysm using mortality data in UK Biobank and the summary RR was 1.47 (1.21–1.78, $I^2 = 0\%$, $n = 2$) per 0.1 unit increment (Supplementary Fig. 13a) and there was no evidence of nonlinearity ($p_{\text{nonlinearity}} = 0.36$) (Supplementary Fig. 13b).

Subgroup and sensitivity analyses

In subgroup analyses, there was little evidence of heterogeneity between subgroups when analyses were stratified by a range of potentially moderating and confounding factors (Supplementary Table 16). For aortic aneurysm, the positive associations for BMI persisted among studies in men and those in men and women combined, studies with longer follow-up, studies on incidence and mortality, studies from Europe and Asia, studies with a larger number of cases, studies with higher quality, and among studies with adjustment for age, education, alcohol, smoking, and physical activity. Results were similar for abdominal aortic aneurysm (both for BMI and waist circumference) (Supplementary Table 16). There was little indication of heterogeneity between subgroups. Heterogeneity was present only in the subgroup analysis of BMI and abdominal aortic aneurysm stratified by adjustment for waist circumference ($p = 0.03$) and studies that adjusted for waist circumference (likely an over-adjustment) showed a suggestive inverse association, in contrast to studies without such adjustment, which showed a positive association (Supplementary Table 16). In addition, associations were weaker or less clear in most of the subgroups of studies that adjusted for potentially intermediate risk factors including diabetes, hypertension, systolic blood pressure, diastolic blood pressure, serum cholesterol, hypercholesterolemia or dyslipidemia, or cardiovascular disease, coronary heart disease or stroke, although there was no heterogeneity between subgroups (Supplementary Table 16). The mean (median) study quality score was 6.4 (6.6) out of 8.0 (Supplementary Table 17).

When including two additional studies that reported on aortic aneurysm and aortic dissection combined (38, 39) (and were excluded from the main analysis because of reporting on a non-specific outcome), the summary RR was 1.17 (1.09–1.25, $I^2 = 68\%$) per 5 kg/m² increment in BMI for aortic aneurysm overall (Supplementary Fig. 11).

We calculated E-values for the summary RRs from the nonlinear dose-response analysis of BMI and aortic aneurysm at a BMI of 25, 30, 35 and 40 compared to 20 kg/m² and found E-values of 1.67 (lower CI: 1.57), 2.32 (lower CI: 2.21), 3.04 (lower CI: 2.85) and 3.82 (lower CI: 3.41).

Fig. 2 Body mass index and aortic, thoracic aortic and abdominal aortic aneurysm, linear and non-linear dose-response analyses

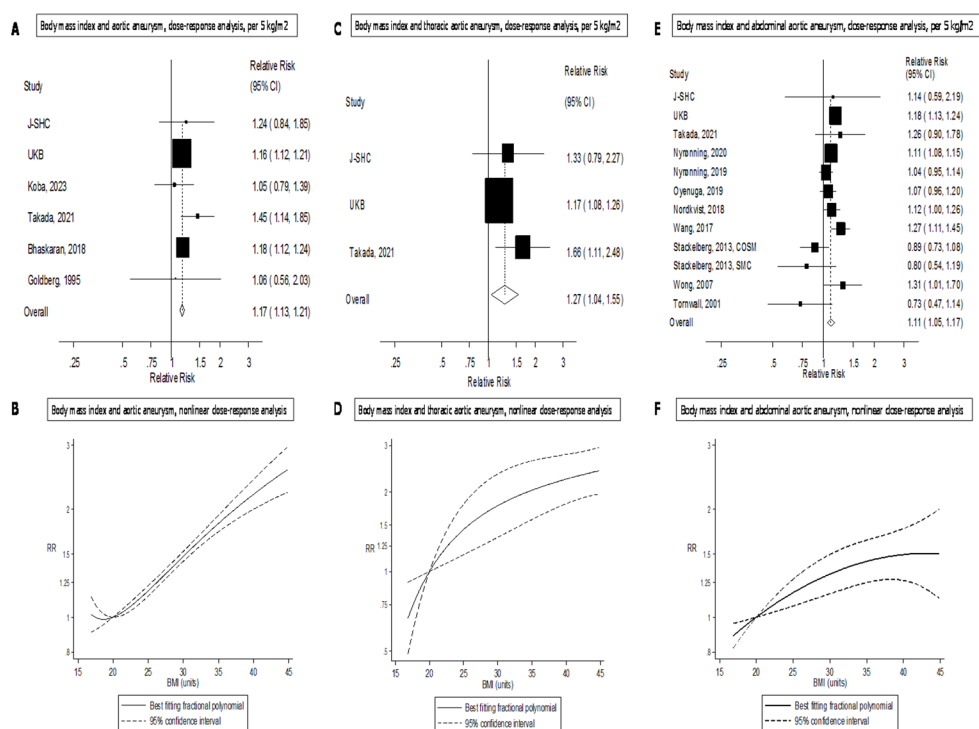


Fig. 3 Waist circumference and thoracic aortic and abdominal aortic aneurysm, linear and non-linear dose-response analyses

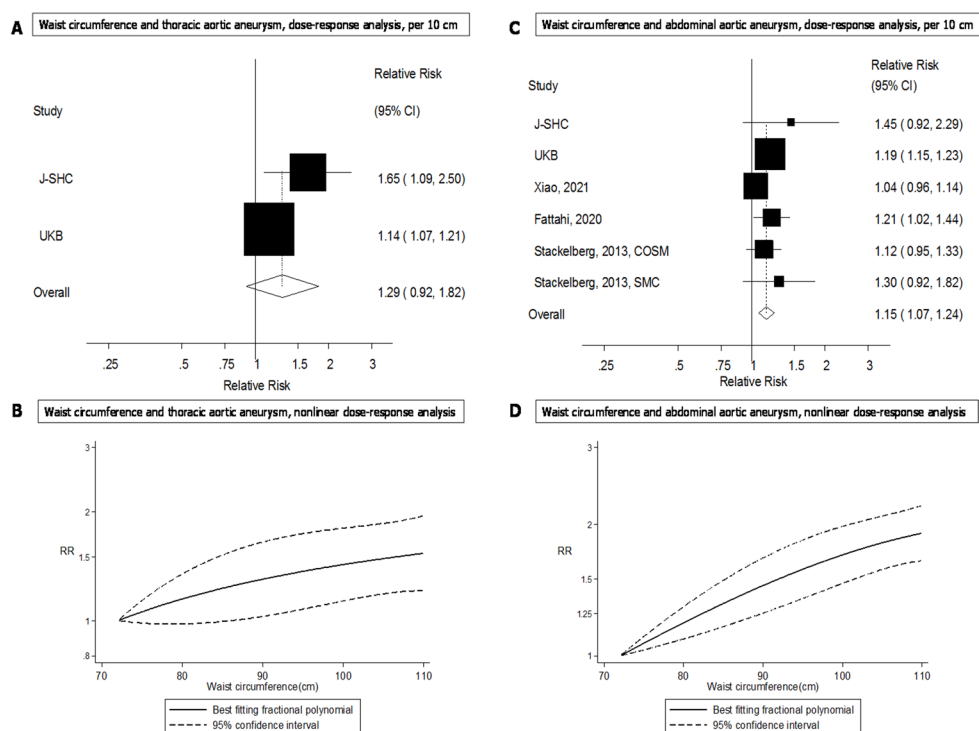
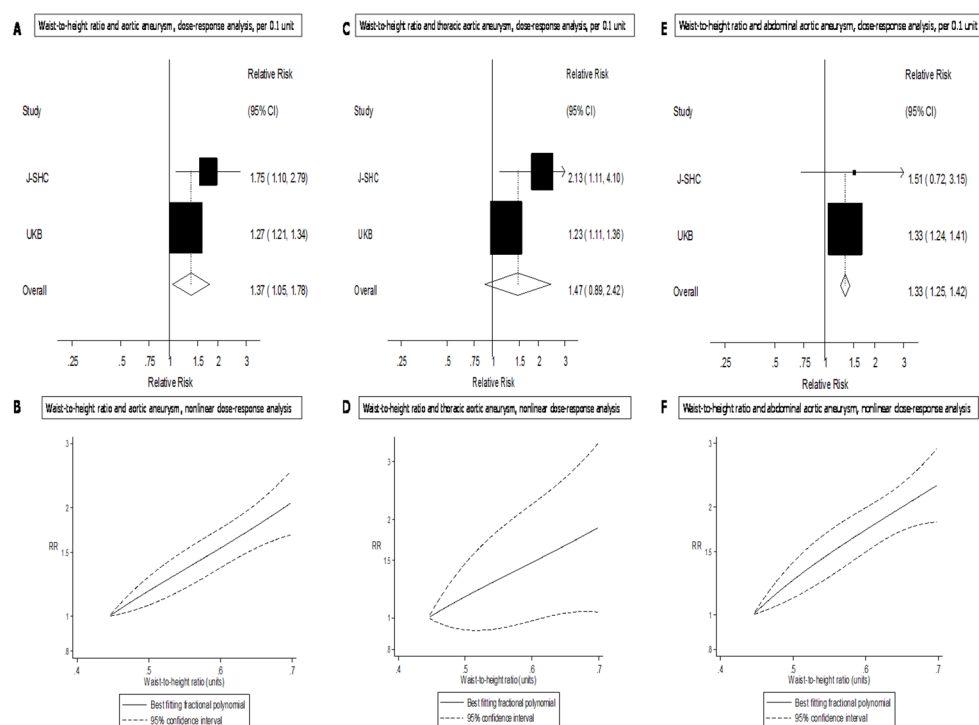


Fig. 4 Waist-to-height ratio and aortic, thoracic aortic and abdominal aortic aneurysm, linear and nonlinear dose-response analyses



Comparing a waist circumference of 80, 90, 100, and 110 vs. 72.1 cm the corresponding E-values were 1.60 (lower CI: 1.40), 2.26 (lower CI: 1.81), 2.81 (lower CI: 2.30), and 3.23 (lower CI: 2.69) for abdominal aortic aneurysm. Similarly, comparing a waist-to-height ratio of 0.50, 0.60, 0.70 vs. 0.44 in relation to aortic aneurysm resulted in E-values of 1.62 (lower CI: 1.34), 2.45 (lower CI: 2.06), and 3.56 (lower CI: 2.75).

Evidence grading

Using World Cancer Research Fund criteria (Supplementary Table 18) to assess the strength of the evidence, we considered the likelihood of causality probable for BMI and aortic aneurysm and for BMI and waist circumference and abdominal aortic aneurysm, while the evidence was limited-suggestive for BMI and thoracic aortic aneurysm, and for waist-to-height ratio and aortic aneurysm and abdominal aortic aneurysm, and limited no conclusion for waist circumference and aortic aneurysm and thoracic aortic aneurysm, and for waist-to-height ratio and thoracic aortic aneurysm (Supplementary Table 19). A justification for these judgments is found in Supplementary Table 20.

Discussion

In the UK Biobank, we found that all adiposity measures were positively associated with risk of aortic aneurysm overall and for most subtypes. The observed associations were more pronounced for men than women, and among never smokers and former smokers than in current smokers. In the J-SHC study, the association between BMI and aortic aneurysm mortality was in the same direction, but less precisely estimated, however, strong positive associations were observed with waist circumference and waist-to-height ratio, and for both measures, stronger associations were observed among never smokers than among ever smokers. In our meta-analysis of prospective cohort studies, we found an 17% higher risk of aortic aneurysm per 5 kg/m² increment in BMI, and a 27% and 11% increase in risk for thoracic and abdominal aortic aneurysm, respectively. For waist circumference, we found a 15% higher risk of abdominal aortic aneurysm per 10 cm increase, while the associations with aortic aneurysm and thoracic aortic aneurysm were less precise. We also found 37% and 33% increases in risk of aortic aneurysm and abdominal aortic aneurysm per 0.1 unit increase in waist-to-height ratio. There was a strong positive dose-response relationship between BMI and aortic aneurysm, with risk increasing even within the normal BMI range, but more substantially in the overweight and obese range. Strong positive dose-response relationships were also observed between waist measures and aortic aneurysm risk. Using World Cancer Research Fund criteria,

we graded the evidence (judging likelihood of a causal association) as probable for BMI and aortic aneurysm and for BMI and waist circumference in relation to abdominal aortic aneurysm, while the remaining associations were graded as limited-suggestive or limited no conclusion.

Several potential mechanisms could explain the positive association between overweight and obesity and risk of aortic aneurysm. Excess weight is a strong risk factor for hypertension [52], which again is strongly positively associated with aortic aneurysm risk [11, 12]. Higher BMI and elevated blood pressure have been positively associated with the size and diameter of the abdominal aorta [53, 54], which is associated with increased risk of abdominal aortic aneurysms [33]. Adiposity is also associated with higher levels of LDL-cholesterol and triglycerides [55] which have been associated with atherosclerosis and increased risk of aortic aneurysm in several studies [23, 24, 56–58], and a recent meta-analysis found positive associations between total and LDL cholesterol and aortic aneurysm risk [13]. Some cases of abdominal aortic aneurysm are known to be due to atherosclerosis in the aortic wall [59]. Experimental animal studies reported that high dietary fat-induced obesity promoted macrophage infiltration and proinflammatory cytokine expression in periaortic adipose tissue and increased the incidence of abdominal aortic aneurysms in mice and in *ob/ob* mice after angiotensin-2 infusion [60], while weight loss was associated with less progression of abdominal aortic aneurysms [61]. Adiposity is associated with low-grade inflammation and with lower levels of adiponectin, a cytokine with anti-inflammatory properties [62]. It has been shown that adiponectin reduced the development of abdominal aortic aneurysm in mice [63]. Obesity has also been reported to be associated with increased rates of wound infection after both endovascular abdominal aortic repair and open repair, which can lead to further complications, longer length of hospital stay, subsequent major operations, and higher rate of graft failure [64], and greater BMI has been associated with increased perioperative mortality after repair of ruptured abdominal aortic aneurysms [65, 66], which is consistent with the positive association observed for aortic aneurysm mortality.

As with any observational studies and meta-analyses based on observational studies, some limitations may have affected the results including potential confounding, heterogeneity between studies, measurement errors in assessment of weight and height, misclassification of outcome, and publication bias. We found that the association between BMI and aortic aneurysm persisted among studies that adjusted for a number of important risk factors including, age, sex, smoking and physical activity. Smoking is among the strongest modifiable risk factor for aortic aneurysm and in the UK Biobank, we observed the association between

adiposity and aortic aneurysm was stronger in never and former smokers than in current smokers, and in the J-SHC study the positive association between adiposity measures and aortic aneurysm mortality was restricted to never smokers, which may indicate negative confounding. This is a consistent finding for health outcomes strongly associated with smoking as smokers tend to have a lower BMI and residual confounding from smoking pushes the risk up at the low end of the BMI range, attenuating the overall association [67, 68]. These observations are also consistent with the strong association observed between smoking and aortic aneurysm [15]. Although we cannot exclude the possibility that other factors may have confounded the observed association, such a factor would have to be relatively strongly associated with both BMI and aortic aneurysm to explain away the increased risk observed in the meta-analysis over and beyond that of the confounders adjusted for in the included studies. Given that the E-values at the high range of BMI and waist measures were in the order of 2–3.8, we consider this possibility less likely. The association between BMI and aortic aneurysm persisted in most subgroup analyses, while for BMI and abdominal aortic aneurysm the subgroup analyses were less consistent, which may have been due to two studies further adjusting for waist circumference [26], which likely is an overadjustment, and exclusion of these two studies from the analysis slightly strengthened the summary estimate. In addition, we observed that studies that adjusted for potential mediating factors such as hypertension or blood pressure, blood cholesterol or hypercholesterolemia, or various cardiovascular diseases showed no association. It will therefore be important for any further studies to present models without adjustment for potential mediators so the full impact of adiposity on aortic aneurysm can be assessed.

Measurement errors in the assessment of weight and height could have affected some of the results. This is unlikely to have been an issue in the UK Biobank, J-SHC Study, and several other studies, where anthropometric measures were measured, however, some of the cohort studies in the meta-analysis used self-reported adiposity measures. Nevertheless, several studies have shown relatively high correlations between measured and self-reported anthropometric variables with correlation coefficients of around 0.95–0.97 suggesting self-report of adiposity is relatively accurate [69–71]. In addition, given the prospective design of the included studies, any measurement errors in the assessment of anthropometric measures would most likely have biased the results toward the null. We found little difference in the size of the summary estimates between studies that used objective vs. self-reported anthropometric measures. Misclassification of the outcome is possible as aortic aneurysms are silent in many cases and diagnosis (and preventive elective surgical repair) could be made due

to hospitalization for other conditions. The cases identified in the UK Biobank therefore likely represent more severe and symptomatic cases. We were not able to further address this issue as routine screening for aortic aneurysm with ultrasound or computed tomography was not done in the UK Biobank. In the J-SHC study, the follow-up was relatively short and the number of aortic aneurysm deaths was low, only aortic aneurysm mortality could be assessed, and we could not make exclusions for prevalent cases because of lack of information at baseline. The small number of aortic aneurysm deaths in J-SHC also hindered us from using more standardized cut-off points for the adiposity measures across the two cohorts. In spite of these limitations, the results were relatively consistent across the two cohort studies. Although publication bias can be an issue for meta-analyses of published studies, we found no evidence of publication bias by statistical testing or by inspection of the funnel plots. Lastly, we had a limited number of studies on other adiposity measures than BMI and further studies are therefore needed for a more comprehensive assessment across different measures of adiposity as well as subtypes of aortic aneurysm.

Strengths of this analysis include the prospective design of the two cohort studies, the large sample sizes, measured adiposity data, and adjustment for a wide range of risk factors in the UK Biobank. Strengths of the meta-analysis include (1) the prospective design of the included studies which avoids problems with recall bias and reduces the potential for selection bias, (2) the detailed subgroup and sensitivity analyses, (3) relatively high study quality, and (4) with up to 8100 cases and 4.9 million participants in the analyses we had sufficient statistical power to detect even a moderate association between BMI and aortic aneurysm. Improved statistical power may to a large extent explain the difference in the results between individual previous studies and the results of this meta-analysis, as most of the included studies reported positive associations, but the associations were often not statistically significant. Our findings have important clinical and public health implications as the number of persons with overweight and obesity has increased considerably in all areas of the world over the last decades [35]. This trend could therefore contribute to additional cases of aortic aneurysm if unabated, but it could potentially be counteracted by the adoption of healthier diets and increased physical activity [72], which in addition to preventing excess weight, also could reduce the risk of aortic aneurysm directly [14, 73]. Given that we in the meta-analysis observed that adiposity was associated with higher risk of aortic aneurysm across geographic locations (America, Europe, Asia), it is likely that the findings may be widely generalisable, however, additional studies are needed across populations and ethnicities for further confirmation.

Conclusion

This analysis of two large cohort studies and a meta-analysis of published studies suggests that higher BMI and abdominal adiposity are associated with an increased risk of aortic aneurysm, and similar results were observed across several additional adiposity measures in the UK Biobank. Further studies are needed to explore the association between different measures of adiposity and risk of aortic aneurysm and its subtypes and to explore the underlying mechanisms.

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Author contributions DA designed the research, conducted the statistical analyses in UK Biobank, conducted the literature search and the statistical analyses in the meta-analysis and wrote the first draft of the paper. YO conducted the statistical analyses in the J-SHC Study. DA and YMS conducted the literature screening. DA, YO, YMS, MH, AKH, ABT, TK, KY, SF, KT, IN, MK, YS, KI, TM, MK, KA, TW, TW, MW, and IJ interpreted the data, revised the subsequent drafts for important intellectual content, read and approved the final manuscript.

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Data availability Data from the UK Biobank are available from <https://www.ukbiobank.ac.uk>.

Declarations

Conflict of interest The authors declare that there is no conflict of interest associated with this manuscript.

Ethical approval The UK Biobank has ethical approval from the North West Multi-Centre Research Ethics Committee, the National Information Governance Board for Health and Social Care in England and Wales, and the Community Health Index Advisory Group in Scotland. In addition, an independent Ethics and Governance Council was formed in 2004 to oversee UK Biobank's continuous adherence to the Ethics and Governance framework, which were developed for the study [25]. All UK Biobank participants provided written informed consent [25]. For the J-SHC study, all procedures performed were done in accordance with the guidelines of the Institutional Ethics Committee of Yamagata University School of Medicine (Yamagata University, 2008, No. 103) or the National Research Committee (the Ethical Guidelines for Medical and Health Research Involving Human Subjects enacted by the Ministry of Health, Labor and Welfare of Japan [74]). In J-SHC, the investigators were not required to obtain informed consent, but information concerning the study was publicized on the web [75] and ensured opportunities for the research participants to refuse the use of their personal information.

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