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Smoking, sex, risk factors and abdominal aortic aneurysms: a prospective study of 18 782 persons aged above 65 years in the Southern Community Cohort Study

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Abstract

Background—Abdominal aortic aneurysm (AAA) is a leading cause of death in the USA. We evaluated the incidence and predictors of AAA in a prospectively followed cohort.

Methods—We calculated age-adjusted AAA incidence rates (IR) among 18 782 participants aged ≥65 years in the Southern Community Cohort Study who received Medicare coverage from 1999–2012, and assessed predictors of AAA using multivariable Cox proportional hazards models, overall and stratified by sex, adjusting for demographic, lifestyle, socioeconomic, medical and other factors. HRs and 95% CIs were calculated for AAA in relation to factors ascertained at enrolment.

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Results—Over a median follow-up of 4.94 years, 281 cases were identified. Annual IR was 153/100 000, 401, 354 and 174 among blacks, whites, men and women, respectively. AAA risk was lower among women (HR 0.48, 95% CI 0.36 to 0.65) and blacks (HR 0.51, 95% CI 0.37 to 0.69). Smoking was the strongest risk factor (former: HR 1.91, 95% CI 1.27 to 2.87; current: HR 5.55, 95% CI 3.67 to 8.40), and pronounced in women (former: HR 3.4, 95% CI 1.83 to 6.31; current: HR 9.17, 95% CI 4.95 to 17). A history of hypertension (HR 1.44, 95% CI 1.04 to 2.01) and myocardial infarction or coronary artery bypass surgery (HR 1.9, 95% CI 1.37 to 2.63) was negatively associated, whereas a body mass index ≥25 kg/m^2 (HR 0.72; 95% CI 0.53 to 0.98) was protective. College education (HR 0.6, 95% CI 0.37 to 0.97) and black race (HR 0.44, 95% CI 0.28 to 0.67) were protective among men.

Conclusions—Smoking is a major risk factor for incident AAA, with a strong and similar association between men and women. Further studies are needed to evaluate benefits of ultrasound screening for AAA among women smokers.

INTRODUCTION

Abdominal aortic aneurysm (AAA) is a focal dilation of the abdominal aorta of at least one and a half times the normal diameter at the level of the renal arteries or an absolute value of 3.0 cm or greater.[1] The overall prevalence is 2% in men above 65 years of age,[2] four times higher in men than women,[3,4] but with a worse prognosis in women than men.[5,6] While the annual incidence and prevalence rates of AAA continue to decline,[7–9] and mortality rates have dropped to just 2% in 2010,[10] death from all aortic aneurysms remains the 16th leading cause of death in the USA among those aged above 65 years.[10] The greatest mortality risk is among those previously undiagnosed who may present with ruptured AAA. These individuals have a 90% mortality rate if rupture of the aorta occurs outside the hospital.[11] Thus, screening is an important component in the management of AAA with evidence in certain populations that the mortality benefit of screening among men aged 65–74 years is maintained for at least a decade and the cost-effectiveness remaining more favourable over time.[12] The known risk factors for AAA include male sex, smoking, hypertension and a family history of AAA in a first-degree relative.[13]

In an effort to increase early diagnosis with the expectation of improved outcomes via optimal medical management and timing of surgical intervention, the USA Preventative Services Task Force recommends a one-time screening abdominal ultrasonography for men between the ages of 65 and 75 years who have a history of smoking.[14] The USA Preventative Services Task Force specifically recommends against screening for AAA in women,[14] though these guidelines are not universally accepted. For instance, the Society for Vascular Surgery recommends screening for women[15] and Medicare provides screening coverage for women with any family history of AAA.[16] In this context, a better understanding of the predictors of incident AAA may inform the improvement of current screening guidelines and facilitate consensus among providers. We have examined the incidence and predictors of clinically detected AAA among participants aged ≥65 years at the time of diagnosis of AAA in the Southern Community Cohort Study (SCCS), a prospective epidemiological cohort study designed to examine racial differences in cancer and other chronic diseases within the southeastern region of the USA.[17]

STUDY DESIGN AND SETTING

Study population

The SCCS is an ongoing prospective cohort study that enrolled nearly 86 000 adults, age 40–79 years, residing in 12 states in the southeastern USA from 2002 until 2009. Approximately 85% were recruited at participating community health centres, institutions that provide primary health and preventive services in medically underserved populations,[18] and the remainder through general population sampling. The SCCS study design and methods have previously been described in detail.[17] This report focuses on those black and white participants who enrolled in the SCCS who were aged 65 years or older on or before 31 December 2008. Thus, participants may have been younger than 65 years on cohort enrolment (age 40–79 years on enrolment) into the SCCS cohort, but must have had their 65th birthday and at least one Medicare claim by 31 December 2012 to be included in this analysis. The age restriction ensured that participants had similar medical insurance coverage under Medicare, through which incident AAA diagnoses were ascertained, while targeting participants who were of the age for recommended screening.

Data collection

On entry into the SCCS, participants were administered a baseline computer-assisted personal interview at the community health centre, while general population participants completed the same self-administered mailed questionnaire (available at http://www.southerncommunitystudy.org). The questionnaire ascertained demographic and socioeconomic characteristics, personal and family medical history, height, weight, tobacco and alcohol use history, and additional variables. Questions regarding medical history (hypertension, diabetes mellitus (DM), high cholesterol, etc) asked participants to mark if 'yes' and state the age at first diagnosis. Many of the questions on the SCCS questionnaire were adapted from questionnaires used and validated in other settings, and a series of validation studies have demonstrated the high reliability of the questionnaire within the SCCS population.[17]

Outcome ascertainment

Using methods previously described, diagnoses of incident AAA among cohort members were ascertained by linking Social Security Number, date of birth and sex of the cohort participants with national Centers for Medicare and Medicaid Services (CMS) Research Identifiable Files (RIFs) from 1 January 1999 until 21 December 2012.[19] Cases of incident AAA were defined as Medicare beneficiaries aged ≥65 years with at least one new medical claim with an International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9 CM) diagnosis code of 441.3 (ruptured AAA) or 441.4 (AAA without rupture) within the Medicare institutional (Medicare Provider Analysis and Review, MEDPAR), Part-B carrier or outpatient base claims files during this period. Thus, in this study incident, AAA is defined as a new diagnosis made in the clinical setting through physician-initiated screening.

We excluded all individuals with a diagnosis of AAA recorded in Medicare prior to enrolment into the SCCS (132 prevalent cases). A comparison population of SCCS

participants was those aged ≥65 years who had at least one Medicare claim during the same time period but did not meet criteria for incident AAA. Mortality was ascertained by linkage of the cohort with the Social Security Administration vital status service for epidemiological researchers and the National Death Index until 31 December 2012.

Participants in the SCCS provided written informed consent; the study protocols were reviewed and approved by Institutional Review Boards of the Vanderbilt University Medical Center and Meharry Medical College and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments.

Statistical analysis

Person-years of follow-up for ascertainment of AAA were calculated from date of entry into the SCCS or the first day of the month of their 65th birthday, whichever came later, until the date of diagnosis of AAA, date of death, or 31 December 2012, whichever occurred first. Age-adjusted (to the US 2000 standard population) incidence rates (IR) of AAA, or the number of new AAA cases per person-year observation accounting for the difference in age distribution, were calculated overall and by race and sex. We assessed the predictors of incident AAA in the overall population and stratified by sex using Cox proportional hazards models, with age as the time scale, adjusting for demographic, lifestyle, socioeconomic, medical and other factors. HRs and corresponding 95% CIs for incident AAA were calculated in relation to the following baseline covariates: sex; race (black, white); education (less than high school, high/vocational school, some college or more); annual household income (<$15 000, ≥$15 000); body mass index (BMI; <25, ≥25 kg/m^2); height (<68, 68–72, >72 inches for men; <63, 63–66, >66 inches for women); cigarette smoking status (never, former, current); self-reported history (yes, no) of diagnosed myocardial infarction (MI)/coronary artery bypass graft (CABG), stroke, hypertension, high cholesterol, DM (type I and type II) and, for women, ever-use of hormone replacement therapy. Covariates were chosen based on known and suspected risk factors for AAA. Health insurance status was not included in the Cox modelling as all participants had similar coverage through Medicare. Tests of interaction with sex or race were performed by using Wald χ^2 test to determine the modifying effect of sex or race on the HRs for baseline covariates. Analyses were conducted using SAS software, V.9.3 (SAS Institute Inc, Cary, North Carolina, USA).

RESULTS

Table 1 presents baseline demographic and other characteristics for the study cohort separately for those with and without AAA. From 2002 to 2012, participants aged ≥65 years contributed a total of 92 103 person-years of follow-up (average follow-up of 4.98 years, median follow-up of 4.94 years), during which we identified 281 incident cases of AAA among SCCS Medicare recipients. Of these, 273 were identified using ICD-9 CM 441.4 (AAA without rupture), while the remaining 8 were identified using ICD-9 CM 441.3 (ruptured AAA). In this study population, the overall mean age at enrolment was 64.5 years (median age of 64 years). Women comprised 63.9% of the study population, 59.9% were blacks, 21.2% were current smokers and 36.9% were former smokers. The overall baseline

prevalence of MI/CABG was 12.1%, stroke 9.8%, hypertension 71.2%, high cholesterol 50.4% and DM 31%. Table 2 presents the distribution and age-adjusted IR of AAA in relation to selected baseline characteristics of the 281 SCCS participants with incident AAA overall and by race and sex. Data on all covariates are available in online supplementary 1. Among incident cases of AAA, 123 (44%) were women and 124 (44%) were black, with an age-adjusted IR (per 100 000) of 153 among blacks, 401 among whites, 354 among men and 175 among women.

Smoking was prevalent among participants with incident AAA, with approximately 40% being current smokers and another 40% being former smokers; the highest prevalence (44.5%) of current smoking was among women. (table 2) The IR for AAA among current smokers (IR=783) was over twofold higher compared to former smokers (IR=294) and much higher relative to never smokers (IR=81); a similar pattern was consistently observed in all race and sex groups, with the IR being highest among current white smokers (IR=1849). The most pronounced effect of smoking was seen among women, among whom current smokers had a 16-fold higher IR than never-smokers (IR=843 vs 51, respectively). The IR of 843 among current female smokers exceeded that of current male smokers (IR=563), former smokers (IR=369) or ever smokers (IR=432).

A history of high-blood pressure was present in over 70% of incident cases regardless of sex, while a prior history of MI/CABG was more prevalent in men than in women (34% and 18%, respectively). The IR for AAA was similar among those with and without a history of high-blood pressure (IR=256 vs 189, respectively) but 2.7-fold higher among those without a history of MI/CABG than those with (IR=73 vs 27, respectively) and higher among men (IR=714). Men with a college-level education comprised 28% of the male population and had an IR of 319.

Table 3 presents the multivariate HRs and 95% CIs for the significant associations between incident AAA and patient characteristics, overall and stratified by sex. Data on all covariates are available in online supplementary table S2. Black race (HR 0.51, 95% CI 0.37 to 0.69) and female sex (HR 0.48, 95% CI 0.37 to 0.69) were inversely associated with incident AAA. Overall, former smoking (HR 1.91, 95% CI 1.27 to 2.87) and current smoking (HR 5.55, 95% CI 3.67 to 8.40) were significantly associated with increased risk of AAA. In analyses stratified by sex, former smoking was significantly associated with incident AAA among women (HR 3.4, 95% CI 1.83 to 6.31) but not men (HR 1.1, 95% CI 0.65 to 1.86; p for interaction between sex and former smoking was 0.28). Furthermore, the highest HR observed in our data was for current female smokers (HR 9.17, 95% CI 4.95 to 17), as compared to an HR of 3.4 (95% CI 1.96 to 5.9) among their male counterparts, though the effect of gender was not significant (p interaction=0.29). Examination among women of the interaction between current smoking and race also showed no significant modification by race of the effect of current smoking (p interaction=0.22).

Overall, prior MI/CABG (HR 1.9, 95% CI 1.37 to 2.63) and a history of high-blood pressure (HR 1.44, 95% CI 1.04 to 2.01) were significantly and adversely associated with AAA. Overall, a BMI of 25 or greater was protective for AAA (HR 0.72, 95% CI 0.53 to 0.98). Prior MI/CABG was significantly associated with incident AAA among men (HR 2.12, 95%

CI 1.41 to 3.17; p for sex interaction=0.35). Among women, ever-use of hormone replacement therapy, while not significantly associated with AAA, showed a marginal association towards a protective effect (HR 0.67, 95% CI 0.41 to 1.07).

Overall, sociodemographic variables including recruitment method, education and income were not associated with incident AAA. However, college-level education was protective, but only in men (HR 0.6, 95% CI 0.37 to 0.97; p inter-action=0.98). An inverse association was observed between DM and AAA, albeit not statistically significant (HR 0.75, 95% CI 0.53 to 1.05). A history of high cholesterol was modestly but not significantly associated with incident AAA (HR 1.19, 95% CI 0.88 to 1.6).

DISCUSSION

In this study within the SCCS cohort, we have evaluated the incidence and predictors of AAA among those aged 65 years and older, a group known to have the highest burden of AAA. The overall incidence of AAA in this cohort was 238 per 100 000 per year, with the rate being highest among white men (498/100 000) and lowest among black women (114/100 000). This is lower than the rate of 350–650 per 100 000 person-years found in screening studies in the USA and the UK.[2021] Most interesting among our findings is the profound risk associated with smoking among women. This provides added support to the growing body of evidence showing that smoking increases the risk in women.[322–25]

Smoking is the most important modifiable risk factor for AAA.[38222326–28] Current smoking appears to have the greatest effect. Duration of smoking and daily cigarette number are also associated with a higher risk of AAA, with the association having a dose-dependent association.[82729] One large systematic review of studies evaluating smoking and aortic aneurysm places the relative risk for aortic aneurysm-related events in current smokers between 3 and 6, consistent with our findings.[30] Others have demonstrated a negative association present with smoking cessation.[27] We did not observe such negative association, but showed that former smokers have a lower risk of AAA compared to current smokers and that, among men, former smoking was not associated with increased risk of AAA. Smoking contributes significantly to the prevalence of AAA and may account for 75% of all AAA of 4 cm or larger.[28]

In the stratified HR analysis by sex, the association between current smoking and risk of incident AAA was more pronounced among women than men, albeit not significantly different. The higher HR among women smokers than men smokers resulted in the absolute risk of AAA being higher among current female smokers than among male ever smokers, the group currently targeted for AAA screening. Previous studies, such as the Women‘s Health Initiative and the Rotterdam study, have demonstrated a strong association between AAA and smoking in women.[2324] Our study, however, is the first to describe a 2.5-fold higher risk of AAA among current female smokers compared to current male smokers (HR 9.17, 95% CI 4.95 to 17 and 3.4, 95% CI 1.96 to 5.9, respectively), though the interaction between smoking and gender was non-significant. This is much higher than the findings of the Rotterdam study, in which the OR for ever smoking was 3.8 (95% CI 1.57 to 9.20) in women compared to 3.3 (95% CI 3.04 to 3.67) in men.[23] Our findings may be in part due to

a selection bias where female smokers were more likely to be screened for AAA by their physician then their non-smoking counterparts. This selection bias also favours screening men, thus indicating that the findings reported here among women might indeed be stronger than we observed. Despite the lower prevalence of AAA among women when compared to men demonstrated in studies such as The Cardiovascular Health Study,[13] the similar risk among women demonstrated here is a matter of concern when considering the current smoking habits among women, which are now more similar to men.[31] Despite a declining prevalence of current female smokers over time, women are starting to smoke at a younger age and smoking for more years.[31] Additionally, the number of former female smokers has increased significantly over the past 50 years. All of these attributes may lead to increasing rates of AAA in the future.

While the overall incidence rate of AAA is lower among women, smoking exerts its greatest risk among women, such that the incidence of AAA among current female smokers exceeds that of male ever smokers. Thus, our findings of high IR of AAA among female smokers merit further evaluation in the light of current guidelines that recommend screening for AAA only in men (age 65–75) with a history of smoking. The benefits of screening men have been supported by studies such as the Multicenter Aneurysm Screening Study (MASS), whereas the benefits of screening women remain unclear.[32] One systematic review supported screening women, describing that the incremental cost-effectiveness ratio among women was similar to that found in men.[33] This finding was reflected by the fact that the lower prevalence of AAA among women is balanced by their higher rupture rate.[33] Another study noted that there is a lower prevalence among women with similar rupture rates among those who were screened versus unscreened over 5 and 10 years follow-up, thus indicating no benefit or rationale for screening.[4] Thus, the evidence for screening women remains unclear, and there is no evidence for what size AAA requires repair or the outcomes data from AAA repair in women. Although discussion of the benefits and risks of screening is beyond the scope of this manuscript,[141534] our data suggest that if a one-time AAA screening is appropriate for former and current male smokers aged 65–75 years, then serious consideration should be given to expand recommendations to include current female smokers aged 65–75 years.

In addition to smoking, male sex, prior MI/CABG and high-blood pressure were all strongly associated with incident AAA in our general study population, while high cholesterol was not associated, after adjusting for confounders. Our finding supports prior work that has defined male sex, atherosclerosis, smoking and high-blood pressure as traditional risk factors for AAA.[263536] AAA pathobiology implicates an intricate interplay between mediators of inflammation, immune response and extracellular matrix degradation that precipitate the distortion of the integrity of aortic architecture.[37] The process may have some overlap with atherosclerosis as certain risk factors such as smoking and hypertension, which we have described here, are implicated in both AAA and atherosclerosis[2638]; however, the pathway is unique and has some divergent processes, as large-scale clinical and epidemiological data suggest that DM, a well-established risk factor for atherosclerosis, has an inverse association with AAA development.[212324262829] Similarly, a systematic review provided data to suggest that the relationship between smoking and aortic aneurysm is consistent with a non-atherosclerotic cause for aortic aneurysm.[30]

The results of our study show an inverse trend between DM and a protective association between above normal BMI (>25 m/kg^2) and AAA, despite adjusting for multiple variables including smoking. The mechanisms of these protective effects are not well understood but underlie the complexity of AAA pathology. We have described this finding and further research into the relationship between AAA with both DM and BMI will need to be performed to better characterise these associations. Interestingly, high cholesterol was not associated with incident AAA in this study, despite its known relationship to atherosclerosis.

This study is one of the few to evaluate socioeconomic status as a potential risk factor for AAA. Over 50% of the cohort earned less than $15 000 per year and approximately 40% had less than a high school education. Thus, we were able to effectively evaluate socioeconomic status and its association with incident AAA. While we did not find a significant association between education or income and AAA overall, men with some college education or more had a decreased risk of AAA. This protective effect may be due to educated men seeking more medical care or obtaining follow-up for screening visits. Unfortunately, we are unable to assess for these factors in our current analysis. Interestingly, previous research on socioeconomic status has been inconsistent, with studies such as the Chicago Heart Association Detection Project[36] and a study by Pujades-Rodriquez *et al*[39] demonstrating no association between socioeconomic status and AAA, while other studies such as the Kaiser Multiphasic Health Checkup Cohort Study[29] and a study by Badger *et al*[40] describe an increased risk of AAA with lower socioeconomic status. Thus, questions may remain as to the effect of socioeconomic status on AAA. Also of note is the lower incidence of AAA in blacks than whites overall, mostly due to differences among men. A lower incidence of AAA among blacks has already been reported,[2235] although the mechanism underlying this association remains unclear. Our study suggests that the protective effect of race is not accounted for by socioeconomic status.

The limitations of this study include a lack of information regarding the complete history of AAA among SCCS participants. We identified a first-time diagnosis of AAA among individuals aged ≥65 years via Medicare claims, but did not have data on potential diagnoses at younger ages. Participants might have had a screening examination for AAA prior to enrolment. Thus, the first reported history of AAA in Medicare might not necessarily be equivalent to the detection of incident AAA, but represent the documentation of a preMedicare AAA diagnosis. Furthermore, we did not screen all individuals for AAA, instead relying on physician-initiated screening or the diagnosis of aneurysm rupture for determination of IR. It is possible that those who were found to have AAA during the 5-year follow-up may have had AAA at baseline. Additionally, it is possible that individuals died from ruptured AAA before having an incident claim in Medicare, thus not being included in our analysis. Some participants included as controls may have had AAA prior to Medicare reporting. If this occurred, it would most likely attenuate the associations we observed. Owing to the low number of surgeries and ruptures along with the definition of screening, bias may be introduced into the study. The low rupture rate may be due in part to the ethnic and gender make-up of the cohort.

The association between smoking and AAA among women could be influenced by a selection bias in which women who smoke are more likely to have a screening ultrasound

compared to non-smokers. Unfortunately, we do not have the ability to link screening ultrasounds with diagnosis. Additionally, given that AAA screenings for CMS are paid for in men with a prior history of smoking (at least 100 cigarettes), the 'never smoker' category is much less likely to have undergone a screening examination.

The major strengths of our study include the collection of extensive baseline information available and the unique and well-documented racial, sex and socioeconomic characteristics of the SCCS cohort. The large size and follow-up period of the cohort allowed for detection of 281 cases, of which over 40% were among blacks and 40% among women. Thus, we were able to estimate risk of AAA by race and sex for various demographic, anthropometric and clinical characteristics. Our study is one of the few to explore the associations between AAA and socioeconomic status, finding a protective effect of higher-level education among men. Additionally, the large number of women and blacks in the cohort allowed us to provide more definitive data on the risk of AAA among women compared to men and blacks compared to whites, and to demonstrate that the strong association between smoking and AAA among women was similar for black and white women. A major strength is that all participants had similar access to healthcare through Medicare. Therefore, clinicians and participants had equal access to screening for, and diagnosis of, AAA. This makes our finding of the association between smoking and AAA among women a matter of even greater concern, as it is unlikely that this association occurred due to increased imaging among smokers versus non-smokers.

CONCLUSION

While mortality rates have decreased for aortic aneurysms over the past 15 years, AAA remains the 16th leading cause of death in the USA in those aged above 65 years.[10] Therefore, further efforts to improve diagnosis and treatment are needed. This effort should be focused not only on men, who have a higher incidence and prevalence of disease, but also on women who have a worse prognosis despite the lower incidence.[56] This is particularly important considering the high relative and absolute risk of AAA among female smokers aged above 65 years about whom we report here and the national trends towards nearly equal smoking prevalence in men and women.[31] We add to the body of evidence that suggests that women who smoke are at increased risk for AAA.[3] Health economic evaluations should be completed to further understand the potential benefits of ultrasound screening of AAA in this population.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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What is already known on the subject?

Abdominal aortic aneurysms are the 13th leading cause of death and currently screening is only recommended among former male smokers. Women are not recommended for screening despite the poor outcomes reported from aortic aneurysms.

What does this study add?

We describe a high risk of abdominal aortic aneurysms among current female smokers, similar to that among current male smokers (HR 9.17, 95% CI 4.95 to 17 and 3.4, 95% CI 1.96 to 5.9, respectively). Our data add to the body of evidence suggesting that women who have ever smoked are at high risk for development of abdominal aortic aneurysms. Health economic evaluations should be completed to further understand the potential benefits of ultrasound screening of abdominal aortic aneurysms in women.

Table 1

Baseline characteristics of southern community cohort study participants aged >65 years with and without a diagnosis of incident abdominal aortic aneurysm (AAA)

	Cases n=281	Non-cases n=18 220	p Value (2-sided)
Age at enrolment (years)	67.5±5.3	64.4±5.6	<0.001
Race			
Black	124 (44.1)	10 960 (60.2)	<0.001
White	157 (55.9)	7260 (39.8)	
Sex			
Women	123 (43.8)	11 692 (64.2)	<0.001
Men	158 (56.2)	6528 (35.8)	
Education			
Less than high school	112 (40.3)	6666 (37.1)	0.247
High/vocational school	95 (34.2)	5885 (32.8)	
Some college or more	71 (25.5)	5406 (30.1)	
Annual household income ($)			
Less than $15 000/year	146 (54.1)	9506 (54.1)	0.997
$15 000/year or greater	124 (45.9)	8075 (45.9)	
Health insurance			
No insurance	36 (13.1)	3359 (18.9)	0.011
Any private/CHAMPUS/other	99 (36.1)	6802 (38.3)	
Medicaid/Medicare only	139 (50.8)	7618 (42.8)	
History of smoking			
Never	46 (16.6)	7507 (42.3)	<0.0001
Former	117 (42.2)	6536 (36.8)	
Current	114 (41.2)	3709 (20.9)	
Body mass index (kg/m^2)			
Less than 25	88 (32.1)	3779 (21.3)	<0.001
25 or greater	186 (67.9)	13 959 (78.7)	
Height (inches)	67.8±4.0	66.3±3.9	<0.001
Heart disease first-degree relative	136 (62.4)	7692 (54.1)	0.016
History of alcohol drinking			
None	174 (64.4)	11 019 (62.9)	0.852
Moderate (1–3 drinks/day)	83 (30.7)	5667 (32.4)	
Heavy (>3 drinks/day)	13 (4.9)	827 (4.7)	
History of MI/CABG	74 (26.9)	2124 (11.8)	<0.001
History of stroke	42 (15.3)	1745 (9.7)	0.004
History of high-blood pressure	206 (74.6)	12 773 (71.2)	0.228
History of high cholesterol	155 (56.4)	8997 (50.3)	0.052
History of diabetes	64 (23.2)	5573 (31.1)	0.005

Data presented as n (%) or mean±SD.

Any observations with missing data are not included in the analysis.

CABG, coronary artery bypass graft; CHAMPUS, Civilian Health and Medical Program of the Uniformed Services; MI, myocardial infarction.

Table 2

Age-adjusted (US 2000 standard) incidence rates (per 100 000) of abdominal aortic aneurysm (AAA) among age 65 plus in relation to baseline characteristics of study population overall and by race and sex, 2002–2012

	Overall		Black		White		Men		Women	
Characteristic	**n (%)**	**IR**	**n (%)**	**IR**	**n (%)**	**IR**	**n (%)**	**IR**	**n (%)**	**IR**
Total	281 (100.0)	238.1	124 (44.1)	152.6	157 (55.9)	401.1	158 (56.2)	354.2	123 (43.8)	175.4
Black	124 (44.1)	152.6					61 (38.6)	243.0	63 (51.2)	113.8
White	157 (55.9)	401.1					97 (61.4)	498.3	60 (48.8)	323.7
Women	123 (43.8)	175.4	63 (50.8)	113.8	60 (38.2)	323.7				
Men	158 (56.2)	354.2	61 (49.2)	243.0	97 (61.8)	498.3				
Education										
Less than 12th grade	112 (40.3)	237.0	63 (51.6)	153.8	49 (31.4)	544.0	58 (36.7)	349.8	54 (45.0)	180.8
High/vocational school	95 (34.2)	296.2	33 (27.0)	154.6	62 (39.7)	485.5	51 (32.3)	436.8	44 (36.7)	235.0
Some college or more	71 (25.5)	191.0	26 (21.3)	172.9	45 (28.8)	210.0	49 (31.0)	318.8	22 (18.3)	100.0
Annual household income										
Less than $15 000/year	146 (54.1)	225.2	76 (64.4)	154.4	70 (46.1)	437.6	69 (44.8)	378.6	77 (66.4)	170.7
$15 000/year or greater	124 (45.9)	280.0	42 (35.6)	152.8	82 (53.9)	399.8	85 (55.2)	348.9	39 (33.6)	209.3
History of smoking										
Never	46 (16.6)	81.6	26 (21.5)	69.8	20 (12.8)	104.6	24 (15.2)	198.5	22 (18.5)	50.8
Former	117 (42.2)	294.2	43 (35.5)	178.8	74 (47.4)	457.0	73 (46.2)	369.4	44 (37.0)	225.2
Current	114 (41.2)	782.5	52 (43.0)	357.7	62 (39.7)	1848.9	61 (38.6)	563.3	53 (44.5)	843.4
History of MI/CABG										
No	201 (73.1)	193.5	99 (81.8)	140.9	102 (66.2)	298.0	103 (66.0)	286.0	98 (82.4)	148.7
Yes	74 (26.9)	588.6	22 (18.2)	260.9	52 (33.8)	1029.1	53 (34.0)	714.2	21 (17.6)	400.4
History of high-blood pressure										
No	70 (25.4)	189.1	22 (18.2)	119.8	48 (31.0)	255.2	46 (29.3)	332.2	24 (20.2)	91.1
Yes	206 (74.6)	256.4	99 (81.8)	161.6	107 (69.0)	478.4	111 (70.7)	371.9	95 (79.8)	197.1
History of high cholesterol										
No	120 (43.6)	220.4	56 (46.7)	142.5	64 (41.3)	406.6	72 (46.2)	324.9	48 (40.3)	156.5
Yes	155 (56.4)	258	64 (53.3)	161	91 (58.7)	404	84 (53.8)	395.8	71 (59.7)	192
History of diabetes										

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	Overall		Black		White		Men		Women	
Characteristic	**n (%)**	**IR**	**n (%)**	**IR**	**n (%)**	**IR**	**n (%)**	**IR**	**n (%)**	**IR**
No	212 (76.8)	253.7	89 (73.6)	169.4	123 (79.4)	381.2	125 (79.6)	385.9	87 (73.1)	176.6
Yes	64 (23.2)	210.8	32 (26.4)	121.5	32 (20.6)	542.4	32 (20.4)	285	32 (26.9)	178.1

Data presented as n (%) or incidence rate/100 000.

Table 3

Cox proportional hazards models for the association between selected characteristics and incident AAA in the SCCS

	Overall			Men			Women		
Characteristic	**Observed***	**HR**	**95% CI**	**Observed**	**HR**	**95% CI**	**Observed**	**HR**	**95% CI**
Women†	88	0.48	0.36 to 0.65						
Black race‡	87	0.51	0.37 to 0.69	44	0.44	0.28 to 0.67	43	0.66	0.42 to 1.04
Education§									
Less than high school	87	1.03	0.74 to 1.44	45	0.94	0.60 to 1.48	42	1.13	0.70 to 1.83
Some college or more	49	0.69	0.47 to 1.01	34	0.6	0.37 to 0.97	15	0.75	0.40 to 1.42
History of smoking¶									
Former	80	1.91	1.27 to 2.87	49	1.1	0.65 to 1.86	31	3.4	1.83 to 6.31
Current	93	5.55	3.67 to 8.40	51	3.4	1.96 to 5.90	42	9.17	4.95 to 17.0
BMI ≥25 at Enrolment**	136	0.72	0.53 to 0.98	77	0.72	0.49 to 1.08	59	0.64	0.40 to 1.03
History of MI/CABG††	58	1.9	1.37 to 2.63	43	2.12	1.41 to 3.17	15	1.43	0.80 to 2.54
History of high-blood Pressure‡‡	155	1.44	1.04 to 2.01	84	1.36	0.90 to 2.06	71	1.57	0.90 to 2.06
History of high cholesterol§§	118	1.19	0.88 to 1.60	66	1.23	0.82 to 1.83	52	1.12	0.72 to 1.75
History of diabetes¶¶	45	0.75	0.53 to 1.05	23	0.68	0.43 to 1.10	22	0.81	0.49 to 1.34

In the Cox proportional hazards models, the variables of recruitment population location, annual household income, height, family history of heart disease, history of stroke, history of high cholesterol, history of diabetes or history of hormone replacement therapy use (among women only) were not significantly associated with incident AAA.

Adjustments were made for all other covariates in the model.

* In the Cox hazard models presented in this table, any observations with missing model variables are not included in the analysis. A total of 208 total observed cases are reported here versus 281 in table 2.

† Versus men.

‡ Versus whites.

§ Versus high school/vocational school.

¶ Versus never history of smoking.

** Versus body mass index (BMI) <25 at enrolment.

†† Versus no history of myocardial infarction (MI) or coronary artery bypass graft (CABG).

[##] Versus history of high-blood pressure.

[§§] Versus no history of high cholesterol.

[¶¶] Versus no history of diabetes.

AAA, abdominal aortic aneurysm; BMI, body mad index; CABG, coronary artery bypass graft; MI, myocardial infarction; SCCS, Southern Community Cohort Study.