Patients with small abdominal aortic aneurysm are at significant risk of cardiovascular events and this risk is not addressed sufficiently

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What This Paper Adds

Previous data suggest that patients with small AAA have a high risk of cardiovascular (CV) mortality and morbidity. The recent implementation of the NHS AAA screening programme (NAAASP) and similar programmes elsewhere has led to several individuals being diagnosed with small AAA, yet addressing their CV risk-factors is still not formalised clinical practice within screening programmes. The precise contemporary CV risk-profiles of these patients also remain unknown. Our findings suggest that despite recent advances in CV prevention in high-risk populations, the management of patients with small AAA remains suboptimal. Better CV-protection should be offered and monitored during surveillance.
ABSTRACT

**Background:** Patients with abdominal aortic aneurysm (AAA) are at significant-risk of cardiovascular (CV) events. Recent implementation of AAA-screening means thousands of patients are now diagnosed with small-AAA; however, CV risk-factors are not always addressed. We aim to assess and quantify the CV-characteristics of patients with small AAA following the introduction of screening-programmes.

**Methods:** CV-profiles of 384 men with a small-AAA (<55mm diameter) were assessed through the United-Kingdom Aneurysm Growth Study (UKAGS), a nationwide prospective cohort-study of men with small-AAA. A prospective local cohort of an additional 142 patients with small-AAA with available blood-pressure (BP) and lipid-profiles was also included and followed-up for 1 year.

**Results:** In the UKAGS population, 54% were current and 30% ex-smokers; 58% were hypertensive and 54% hypercholesterolaemic. In the local-group, 54% were current and 40% were ex-smokers, and 94% hypertensive. Patients were not more likely to receive CV medication after entering AAA-surveillance in either group. All local patients were clustered “high-risk” for future CV-events based on the Framingham-score, mean: 21.8% (95% Confidence-Interval: 20.0, 23.6), JBS-2 16.3% (14.7, 17.9) and ASSIGN 25.2% (22.7, 27.7). No change was seen in systolic BP-levels between baseline and one year (140.9mmHg vs. 142.5mmHg, p=0.435). A rise was seen in cholesterol (4.0mmol to 4.2mmol, p<0.0001) values at one year.

**Conclusions:** This study suggests that patients with small AAA are at significant risk for developing CV events and this is not currently addressed, which is evident by the “high-risk” CV risk profiles of these patients despite being in AAA surveillance. Design and implementation of a CV risk reduction programme tailored for this population is necessary.
INTRODUCTION

Ruptured abdominal aortic aneurysm (AAA) is an important cause of cardiovascular (CV) death in the Western World (1). The long latent period between development of AAA and rupture offers an opportunity for screening which has been shown to reduce AAA-related mortality in men by detecting aneurysms prior to rupture and offering elective surgical repair to prevent rupture (2). Screening reduces AAA-related mortality by 50% and has been shown to be cost effective, even at low AAA prevalence rates (2-4). Nationwide AAA screening programmes have subsequently been introduced across the United Kingdom (UK), Sweden, in some regions of Denmark and in the United States (US) Veterans’ Association. However, screening for AAA has little impact on all-cause mortality (1). Interestingly, CV-events, and not AAA rupture, are by far the commonest cause of death in patients with small AAA (<55mm in maximal diameter), regardless of whether they are offered an intervention to treat the aneurysm or not (5) – in the MASS screening trial 41% of deaths in the screening arm were due to a CV cause, of which only 2% were AAA related (2). A recent meta-analysis using data from 10 old retrospective series of patients with a small AAA which had previously reported CV-death rates, showed a 3% per year chance of CV-mortality, as well as a high prevalence (41%) of ischaemic heart disease (IHD) in this population (6). This risk of CV death is similar to patients that have already experienced a major cardiac event. The American Heart Association (AHA), European Society of Cardiology (ESC) and UK National Institute for Health and Care Excellence (NICE) all recommend aggressive CV risk-factor modification for patients after a myocardial infarction (MI) or those with established peripheral arterial disease (PAD); however, current evidence and guidance for those with an AAA is lacking. No relevant guidance is currently available for these patients. In the UK, by 2014, a total of 235,409 men had already undergone screening. A total of 9,031 patients with small AAA had been identified and were entered into surveillance (4). Surveillance involves an assessment of aortic-diameter...
at 6 or 12-monthly intervals but no specific interventions are currently in place to systematically address CV risks. These small AAAs will take years before they require surgical intervention, hence these men are likely to stay in surveillance (measurement of AAA size) for a long period of time (7). Most screening units do not institute specific secondary preventative strategies at the moment. It is unknown if AAA surveillance programmes represent an opportunity to reduce CV morbidity and how this may be implemented, with ongoing international discussions regarding the necessity of such strategies(8).

Following the above, we aimed to assess the contemporary CV risk characteristics of patients with small AAA identified through screening in the UK [through the NHS AAA Screening Programme (NAAASP)] and ascertain the proportion of patients receiving secondary preventative medication after they have been diagnosed with a small AAA. We used data from a nationwide prospective study [United Kingdom Aneurysm Growth Study (UKAGS)], a local study whereby individuals are invited to enter the study dependent on the presence of a small AAA solely from the NAAASP screening, and also utilised a local prospectively assembled cohort of individuals with small AAA identified through screening for which precise blood-pressure and lipid measurements were obtained over a period of 1 year, further to traditional CV risk-factors, in order to quantify their CV risk using validated risk-scores.

**METHODS**

All individuals who participated in a prospective nationwide cohort-study of men (UKAGS) were included in the study (period ending July 2015). All of these patients had been identified as having a small AAA through a UK based screening programme. Precise aortic diameter was obtained directly from NAAASP records (ultrasound based screening) and participants were sent a bespoke questionnaire for completion, collecting demographics, data on smoking
habit, past medical-history, and drug-history. Men with a newly detected AAA and those already under surveillance for an AAA were sent repeat questionnaires each year to coincide with their surveillance appointments. This follow-up was only completed for those with a detected small AAA, continuing only until the patient has reached the threshold for surgical-intervention (55mm), has died, or withdraws from the study. Given the lack of precise blood pressure measurements in the UKAGS population, hypertension was defined as receiving an antihypertensive agent at the point of inclusion in the study.

A second population of patients with a small AAA (local group) was identified prospectively from vascular outpatient departments in 2 tertiary referral centres in the West Midlands (UK). All patients had been referred to a vascular clinic (outpatients) following AAA-detection through NAAASP (none were UKAGS participants). They were all asymptomatic and AAAs had been detected inadvertently on cross-sectional abdominal imaging. Data collected included: AAA-size, medical-history, smoking-habit, blood pressure (BP) levels, lipid profiles (total cholesterol, high and low density lipoproteins) and drug-history at baseline and 1 year. Patients were followed-up 1 year after their 1st appointment. Relevant ethical approvals for both populations had been granted and patients have provided written informed consent for participation in the study.

In the local group, hypercholesterolaemia was defined as total cholesterol >5mmol/L (9) and hypertension as patient taking antihypertensive-medications or blood pressure >=140/90mmHg(10). All CV events in the study were defined as per the American Heart Association (AHA) (11). In this group, BP was measured using an electronic automated oscillometric BP measuring device (eBPM) – the Omron (Omron, Milton Keynes, UK), which has previously been validated for use in clinical trials (12). BP was recorded by a nurse independent to the study and documented in the patient’s notes and entered on an electronic database. A total of 3 blood-pressure measurements were obtained and the mean reading was
recorded, as per previous evidence suggesting that this is an accurate form of measuring BP in this setting(12). Three sequential independent measurements were taken at all visits.

Using the UKAGS data, in order to assess the pharmaceutical CV protection offered to those patients with an AAA detected compared to those without in the NAAASP AAA population, we compared the proportions of each group reporting antiplatelet agents, statins, beta-blockers, and anti-hypertensive agents use, as well as other identifiable CV risk factors. We also compared the same aspects of CV risk in patients with a newly diagnosed AAA to those who had at least one (or more) surveillance appointments.

Similar comparisons were made for the local group between baseline and the subsequent 1 year appointment; in this instance this included BP measurements, total cholesterol and lipid levels, as well as calculation of the following CV risk scores: Framingham 10-year score (based on age, total and HDL cholesterol, smoking, and systolic BP), Joint British Societies (JBS2, based on age, gender, ethnicity, BMI, total and HDL cholesterol, and systolic BP) CV score(13, 14), and ASSIGN (based on age, gender, history of DM, CVD, or RA, smoking, total and HDL cholesterol, and systolic BP) risk score (15). An online calculator has been used to produce each CV risk-score (http://cvrisk.mvm.ed.ac.uk/) and exhaustive details regarding the precise models used for these validated risk-scores have been published elsewhere (13, 14). BP-levels using the validated method described above (automated oscillometric BP measuring device) and lipid profiles were not available for the UKAGS population.

**Statistical analysis**

Analyses were performed using the SPSS 21.0 (SPSS, Chicago, Ill, USA). Continuous parametric data are presented as mean value ± standard deviation (SD) or 95% Confidence Interval (CI) and categorical data are presented as absolute values and percentages. A Pearson
Chi-Squared test was performed to compare categorical variables and a paired or independent t test was used to compare continuous data. A p value of <0.05 was considered as statistically significant.

RESULTS
Overall, 5,255 men were included from the UKAGS population, 4,871 in the non-aneurysmal group (mean age 69.8, mean aortic diameter 1.8cm) and 384 in the aneurysmal group (mean age: 71.9 years, mean aortic diameter: 3.8cm); at the time 722 patients with an aneurysm had already been contacted by the study group of which 384 had consented and had provided a completed questionnaire; 7,221 patients without aneurysmal disease had been contacted, of which 4,871 had given their consent and information had been collected. Median follow-up in the UKAGS populations was 2.5 years (range: 1-3 years). Patients in the aneurysmal group were 3 times more likely to smoke compared to the non-aneurysmal group (p<0.0001); patients with aneurysmal disease also had a higher prevalence of hypertension (38% in the control group compared to 57.8% in the aneurysmal group, p<0.0001), diabetes mellitus (10% to 17.7%, p<0.0001), and hypercholesterolaemia (32% to 53%, p<0.0001). Overall, 44.2% and 6.2% of participants with an identified small AAA were on aspirin or clopidogrel respectively, whilst 60.9% were on a statin at baseline (1st appointment). Tables 1 and 2 summarise available CV characteristics for the two UKAGS groups.

There were 142 participants in the local group (92.3% males, mean age: 72.3 years), where we sought more in-depth information regarding BP levels and lipid profiles (see Table 3). Overall, 54% were current and 40% were ex-smokers; 94.4% of patients were found to be hypertensive, with 43.1% and 2% receiving aspirin or clopidogrel respectively and 57.7% receiving a statin at baseline (1st appointment).
Within the UKAGS aneurysmal population (findings summarised in Tables 1 and 2), 62.5% of participants had had one or more follow-up surveillance appointment(s); all patients are offered yearly appointments to monitor their aortic size through NAAASP. There were no differences between the proportion of patients with small AAA taking aspirin or a statin at diagnosis or after having been under surveillance for one year or more (aspirin: 44.2% vs. 44.4% at baseline, p=0.997; statin: 61.7% vs. 59.7% at baseline, p=0.746). Similarly, in the local population, patients were not more likely to receive aspirin or a statin one year after diagnosis of the AAA (aspirin: 39% vs. 38% at baseline, p=0.972; statin: 61.7% vs. 57.7% at baseline, p=0.064).

Precise lipid-profiles and BP levels were available for the local group, allowing calculation of 3 distinct CV risk-scores, in order to quantify the CV risk of this group. All patients in the local cohort were identified as “high-risk” for future CV-events based on these scores, as per each relevant definition. Mean values were: Framingham-score: 21.8% (95% CI: 20.0, 23.6); JBS-2: 16.3% (14.7, 17.9); ASSIGN: 25.2 (22.7, 27.7). Also, no change was seen in systolic BP levels between baseline and the appointment after one year (140.9mmHg vs. 142.5mmHg, p=0.435). Interestingly, a rise was seen in both cholesterol (4.0mmol to 4.2mmol, p<0.0001) and high density lipoprotein (HDL) values (1.4mmol to 1.5mmol, p<0.0001) one year after diagnosis of the AAA (sequential lipid profiles available for all patients), despite the fact the proportion of patients receiving statin therapy was not significantly different (61.7% vs. 57.7% at baseline, p=0.064). None of these patients were on ezetimibe or other lipid-lowering agents.

Within the space of one year, 9 patients (6.3%) in the local group developed a non-fatal myocardial infarction, 1 required admission due to heart failure (0.7%) and 2 (1.4%) developed a non-debilitating stroke, amounting to an 8.4% incidence of major CV events. Precise CV events per year were not available for the UKAGS population.
DISCUSSION

This study has attempted to provide a snapshot of the contemporary CV risk profiles of patients diagnosed with a small AAA following the introduction of AAA screening. We used data from two independent sources: a prospective nationwide cohort study (UKAGS) and a local prospectively assembled cohort, for which more detailed risk-profiles were available (BP and lipids); these two groups act complementary to each other. Our data suggests that patients with a small AAA are already at significant risk of CV events when diagnosed with aneurysmal disease. This is evidenced by their comorbidities and confirmed by their classification as “high-risk” using a series of validated CV risk scores. These CV risk-factors are not currently being adequately addressed during AAA surveillance, which represents an important missed opportunity for healthcare providers to reduce the burden of CV events in this population. In fact, there is no specific guidance by bodies such as NICE, AHA, or NAAASP regarding how exactly CV risk-factors should be addressed in these populations undergoing surveillance, which is an important omission, supported by our results showing that at 1 year patients were at similar risk compared to baseline.

Aneurysmal and CV disease share common risk factors (16, 17). It is therefore rational to suggest that patients with AAA will be at increased risk of CV events, even before the AAA merits surgical-intervention. Our recent meta-analysis with retrospective data prior to the nationwide implementation of AAA screening and publication of recent CVD management guidance by NICE and AHA had suggested a high-prevalence of IHD and CV morbidity in patients with small AAA(6). This is confirmed by this more contemporary prospective analysis. More than half of the population were active or ex-heavy smokers and the prevalence of hypertension exceeded 90% in our local cohort, when precise BP measurements were used. As clearly evidenced on Table 1, all CV-related risk-factors were far more prevalent in the small
AAA group compared to the non-aneurysmal group at baseline in the UKAGS group. In the smaller regional group, we calculated CV risk-scores using BP and lipid levels and found that all patients were clustered as “high CV risk”. We have in fact used 3 different scores to validate this conclusion: the Framingham score was one of the first CV risk scores introduced in clinical practice and was developed using data from a North American population. We therefore also employed the JBS-2 and ASSIGN scores which have included more up to date populations at risk for CV events and most importantly, these scores have previously been validated and studies extensively in UK populations(13, 15). Further to our risk-score related findings at baseline, after patients had entered surveillance, they were not more likely to be on an antiplatelet agent or a lipid-lowering agent, even after seeing a vascular surgeon. No change in the BP levels were recorded either in the local group within the one year they had been in surveillance for.

All patients diagnosed with a small AAA via screening-programmes enter annual surveillance. This provides an excellent opportunity to offer systematic supervised forms of protection against future CV events, in the form of medication or lifestyle-modification interventions. Unfortunately, only a small proportion was offered medical prevention in this study. Evidently, there is a missed opportunity following the identification of an AAA to modify CV parameters appropriately as per current protocols. Currently in the UK, the NAAASP standard operating procedures do not propose aggressive CV prevention for patients with small AAA in the form of medication, such as antiplatelets or statins.

Another important element is the period between small AAA diagnosis and possible subsequent intervention. Addressing CV risk may theoretically impact on AAA growth rates, given the shared pathogenetic mechanisms. However, a paucity of evidence still remains in the factors affecting growth rate of such aneurysms(18). Addressing CV risk factors may potentially impact on AAA growth, and potentially have a larger impact on mortality rates than
any prophylactic AAA surgery for this cohort group, but more evidence is definitely required to prove such rationale.

Two groups of patients very similar to those with a small AAA are individuals with asymptomatic carotid artery disease and those with established peripheral arterial disease. Both pathologies are fairly common in the general population (19, 20), especially the elderly, and are associated with risk factors very similar to those leading to cardiovascular disease and AAA formation. Both NICE and AHA suggest statin and antiplatelet therapy for these groups of patients; however, implementing a risk-reduction programme in these two population may prove more challenging compared to patients who are already in AAA screening, given the lack of an established follow-up framework (such as AAA screening).

This analysis has focused on data derived from the UK. However, other areas in Europe and the United States, currently offer AAA screening to the general population. In Sweden, which also has a mature screening network for AAA, a study published in 2011 provided prevalence of CV risk factors for those screened for an AAA – the prevalence of diabetes and hypertension was very similar to our findings (21); however, more individuals were smokers in our UK cohort. Very similar findings, regarding CV risk factors at baseline, have been reported from the Life Line Registry of screened patients in the USA (22). In neither screening programme does an integrated CV risk reduction strategy exist. Regarding smoking, in our cohort, less individuals were active smokers after 1 year of surveillance. Men in the NAAASP surveillance programme are encouraged not to smoke (verbal advice) but they are not all referred to a smoking cessation clinic after AAA diagnosis. Therefore, whether this drop in smoking is simply accidental or due to AAA surveillance is impossible to know.

There are some limitations in our work. The number of participants is relatively small; however, data were prospective and were collected from multiple screening centres,
representative of the general population. Also, for the UKAGS population, we do not have precise lipid profiles or validated BP measurements that would allow calculation of CV risk scores, and this is one of the reasons why there was a difference in the prevalence of hypertension between the 2 groups. Hence we chose to prospectively assemble a cohort of local patients to further quantify CV risk using that type of data. This is also evident in the discrepancies seen in the prevalence of ischaemic heart disease and peripheral arterial disease between the two cohorts, as data were collected in a prospective systematic manner led by a clinician in the regional cohort of patients. Finally, we cannot calculate the precise incidence of CV events per year in the UKAGS cohorts, due to lack of relevant data as part of the study’s protocol.

Overall, this study suggests that patients with small AAA are at significant risk for developing CV events and this is not adequately addressed through the existing framework. Research is therefore required to design and implement a CV risk reduction programme tailored for this population. The established screening and surveillance pathways represent an excellent opportunity in that direction.
CONFLICTS OF INTEREST
Authors have no interests to declare specific to this study.

ACKNOWLEDGEMENTS
None
REFERENCES

Table 1: Baseline characteristics of patients recruited from the United Kingdom Aneurysm Growth Study (UKAGS) and our regional population (second study group)

<table>
<thead>
<tr>
<th>UKAGS population</th>
<th>Non-Aneurysmal Group</th>
<th>Aneurysmal Group</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N of participants (all males)</td>
<td>4871</td>
<td>384</td>
<td>-</td>
</tr>
<tr>
<td>Mean age (95% CI)</td>
<td>69.8 (69.7, 69.9)</td>
<td>71.9 (71.4, 72.5)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Mean aortic diameter, mm (95% CI)</td>
<td>18 (18,18)</td>
<td>38 (37, 39)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Smoking (current)</td>
<td>249/4788 (5.2%)</td>
<td>56/366 (15.3%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Smoking (ever)</td>
<td>2614/4580 (57.1%)</td>
<td>270/321 (84.1%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>HTN</td>
<td>1769/4677 (37.8%)</td>
<td>203/351 (57.8%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>DM</td>
<td>478/4582 (10.4%)</td>
<td>62/350 (17.7%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>IHD</td>
<td>279/4778 (5.8%)</td>
<td>73/366 (20.0%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>CVD</td>
<td>146/4773 (3.1%)</td>
<td>23/363 (6.3%)</td>
<td>0.0007</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>1456/4615 (31.5%)</td>
<td>185/348 (53.2%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Aspirin use</td>
<td>840/4871 (17.2%)</td>
<td>170/384 (44.2%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Clopidogrel use</td>
<td>92/4871 (1.9%)</td>
<td>24/384 (6.2%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Statin use</td>
<td>1722/4871 (35.4%)</td>
<td>234/384 (60.9%)</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Local participants</th>
<th>142</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age (95% CI)</td>
<td>72.3 (71.1, 73.6)</td>
</tr>
<tr>
<td>Male gender</td>
<td>131 (92.3%)</td>
</tr>
<tr>
<td>Mean aortic diameter, mm (95% CI)</td>
<td>39 (34, 41)</td>
</tr>
<tr>
<td>Smoker (current)</td>
<td>77 (54%)</td>
</tr>
<tr>
<td>Smoker (previous)</td>
<td>57 (40%)</td>
</tr>
<tr>
<td>HTN</td>
<td>135 (95.1%)</td>
</tr>
<tr>
<td>DM</td>
<td>30 (21.1%)</td>
</tr>
<tr>
<td>IHD</td>
<td>58 (40.8%)</td>
</tr>
<tr>
<td>PVD</td>
<td>37 (26.1%)</td>
</tr>
<tr>
<td>Hypercholesterolaemia</td>
<td>98 (69%)</td>
</tr>
<tr>
<td>Aspirin use</td>
<td>54 (38%)</td>
</tr>
<tr>
<td>Clopidogrel use</td>
<td>10 (7%)</td>
</tr>
<tr>
<td>Statin use</td>
<td>82 (57.7%)</td>
</tr>
<tr>
<td>Mean Blood Pressure Systolic (95% CI)</td>
<td>141 (137, 144)</td>
</tr>
<tr>
<td>Mean Cholesterol (mmol) (95% CI)</td>
<td>4.0 (3.9, 4.2)</td>
</tr>
<tr>
<td>Mean HDL (mmol) (95% CI)</td>
<td>1.4 (1.3, 1.4)</td>
</tr>
</tbody>
</table>

HTN: hypertension, CI: confidence interval, DM: diabetes mellitus, IHD: ischaemic heart disease, CVD: cardiovascular disease, PVD: Peripheral Vascular Disease, BP: blood pressure, HDL: high-density lipoprotein
**Table 2:** Proportion of the United Kingdom Aneurysm Growth Study (UKAGS) aneurysmal cohort patients that were on cardiovascular (CV) medication after entering screening surveillance

<table>
<thead>
<tr>
<th>Average Time in Surveillance (Months)</th>
<th>14.6 (13.0, 16.2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proportion of patients that had at least 1 surveillance appointment</td>
<td>240/384 (62.5%)</td>
</tr>
<tr>
<td>Proportion receiving CV medication after their 1st surveillance appointment:</td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td>106/240 (p=0.997)</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>17/240 (p=0.759)</td>
</tr>
<tr>
<td>Statin</td>
<td>148/240 (p=0.746)</td>
</tr>
<tr>
<td>Beta-Blocker</td>
<td>29/240 (p=0.993)</td>
</tr>
<tr>
<td>Anti-Hypertensive</td>
<td>95/240 (p=0.459)</td>
</tr>
</tbody>
</table>

*p values refer to comparison with baseline*
Table 3: Changes to cardiovascular parameters in the local group between the baseline and the 1 year surveillance appointment

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Baseline</th>
<th>1 year</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>BP Systolic (mmHg)</td>
<td>140.9 (137.4, 144.3)</td>
<td>142.5 (139.1, 145.8)</td>
<td>0.435</td>
</tr>
<tr>
<td>Cholesterol (mmol)</td>
<td>4.0 (3.9, 4.2)</td>
<td>4.2 (4.0, 4.38)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>HDL (mmol)</td>
<td>1.4 (1.3, 1.4)</td>
<td>1.5 (1.4, 1.6)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Framingham Risk Score</td>
<td>21.8 (20.0, 23.6)</td>
<td>22.7 (21.0, 24.3)</td>
<td>0.114</td>
</tr>
<tr>
<td>JBS2 Risk Score</td>
<td>16.3 (14.7, 17.9)</td>
<td>17.1 (15.4, 18.7)</td>
<td>0.163</td>
</tr>
<tr>
<td>ASSIGN Risk Score</td>
<td>25.2 (22.7, 27.7)</td>
<td>26.7 (24.2, 29.3)</td>
<td>0.009</td>
</tr>
<tr>
<td>Current smoker</td>
<td>77 (54%)</td>
<td>62 (44%)</td>
<td>0.003</td>
</tr>
</tbody>
</table>

Data presented as mean and 95% confidence interval

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