Title

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Risk Factors for Incident Coronary Artery Calcium in Younger (Age 32–45 Years) Versus Intermediate (46–65 Years) versus (65–84 Years) Older Persons

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Abstract

The prognostic value of traditional atherosclerotic cardiovascular disease (ASCVD) risk factors may decrease with age. We sought to determine whether the association between traditional ASCVD risk factors and incident coronary artery calcium (CAC) differs for younger versus older persons. We included 5,108 participants with baseline CAC=0. Repeat CAC scoring occurred over 3 to 11 years follow-up. Multivariable Cox proportional hazards regression assessed the association between traditional risk factors and incident CAC in young (32–45 years), middle-aged (46–64 years) and older adults (65–84 years). A total of 61% of participants were women and 37% were black. The proportion with incident CAC ranged from 22% among young adults, 34% for middle-aged adults, and 45% for older adults. Among young adults, traditional risk factors were significantly associated with incident CAC except for diastolic blood pressure and HDL-cholesterol, whereas only total cholesterol/HDL-cholesterol ≥3.5 (p=0.04) was significantly

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associated with incident CAC in older persons. Non-HDL cholesterol (p-interaction=0.02) was more strongly associated with incident CAC in young (HR=1.20, 95% CI: 1.09–1.31) and middle age (HR=1.14, 95% CI: 1.07–1.23) compared to older adults (HR=1.11, 95% CI: 0.99, 1.23). When added to demographics, traditional risk factors provided a greater C-statistic improvement for incident CAC prediction in young (0.752,+0.070, p<0.001) versus middle-aged (0.645,+0.054, p<0.001) and older adults (0.597,+0.025, p=0.08). In conclusion, traditional risk factors more strongly predict incident CAC in young compared to older adults, underlining the importance of primordial prevention through middle-age while identifying the challenges of ASCVD risk assessment in older persons.

Keywords

age; coronary artery calcium; life course; risk factors; atherosclerosis

Introduction

Coronary artery calcium (CAC), measured by non-contrast computed tomography (CT), directly measures subclinical atherosclerotic burden and is strongly predictive of long-term atherosclerotic cardiovascular disease (ASCVD) risk [1]. While traditional ASCVD risk factors are the cornerstone of ASCVD risk prediction, the strength of the association between traditional ASCVD risk factors and ASCVD events is weaker with increasing age [2–5]. For example the relative association between higher total cholesterol/HDL ratio and coronary artery disease mortality is approximately twice as strong for persons aged 40–49 compared to persons >70 years old [5]. However, the extent to which age modifies the relationship between traditional ASCVD risk factors and incident CAC remains uncertain. It is important to have a better understanding of whether there are differences in the risk factors for initiation of CAC for younger versus older persons, because the initiation of CAC is a significant milestone in the atherosclerotic process that is associated with a substantially increased risk for ASCVD [6]. An age-specific investigation of risk factors associated with the development of incident CAC may provide insight into whether certain traditional risk factors are more or less strongly associated with the atherosclerotic process at different stages of the life course[7]. However, the utility of traditional ASCVD risk markers across age groups for predicting incident CAC is unknown[8].

Methods

The Multi-Ethnic Study of Atherosclerosis (MESA) and Coronary Artery Risk Development in Young Adults (CARDIA) studies are both community-based prospective cohort studies and their details have been previously described [9,10]. Briefly, MESA enrolled 6,814 adults aged 45–84 years old who were free of clinical ASCVD, including White, African American, Hispanic, and Chinese participants. All MESA participants had the first CAC scan performed at the baseline visit (2000–02). CAC scoring was performed at subsequent follow-up periods, including MESA Visit 2 (2002–04), 3 (2004–05), 4 (2005–07), and 5 (2010–11). As a part of the study design, not all MESA participants had follow-up CAC scans at each visit. Among persons with CAC=0 at Visit 1, one-half received a follow-up

scan at Visit 2, and the other one-half at Visit 3. Persons who did not have a follow-up CAC scan were prioritized to undergo repeat scanning at Visit 4. Visit 5 preferentially included one-half of participants who had CAC=0 on prior visits, including Visits 3 and 4. The CARDIA study enrolled 5,115 adults aged 18–30 years old without clinical ASCVD with a first examination occurring in 1985. The first CAC scan for the CARDIA study was performed at the Year 15 Visit (2000) with repeat CAC scoring performed 10 years later at the Y25 Visit (2010). Accordingly, MESA Visit 1 and CARDIA Y15 Visit were used as the baseline examinations for this study, which both started in the year 2000. A more detailed description of the design for these community-based prospective cohort studies is available elsewhere [9,10].

We included the 5,108 participants who had CAC=0 at baseline (MESA Visit 1, CARDIA Year 15) and a subsequent follow-up CAC scan (MESA Visits 2–5, CARDIA Year 25). At study baseline, there were 2,139 young adults aged 32–45 years old included from the CARDIA study, and 2,154 middle-aged (46–64 years) and 815 older adults (65–84 years) adults included from MESA (Supplemental Figure 1).

Half of the MESA and CARDIA field centers used electron beam computed tomography (EBCT) (MESA: Chicago, Los Angeles, New York; CARDIA: Chicago, Oakland), while the other half used multidetector computed tomography (MDCT) (MESA: Baltimore, Forsyth County, St. Paul; CARDIA: Birmingham, Minneapolis) to measure CAC [11–13]. CAC scores derived from EBCT and MDCT scanners have excellent agreement (interobserver κ=0.93, and intraobserver κ=0.90 [1,14]. Calcium scores were quantified using the Agatston method. Each MESA (≥2 scans) and CARDIA (2 scans) study participant underwent multiple CAC scans and the mean Agatston CAC score was used in all analyses.

Both MESA and CARDIA collected demographic and clinical information, including sex, race/ethnicity, education status (post high school education versus high school education or less), income (≥$50,000 versus <$50,000 per year), smoking status, and medication use history using standardized survey methods [9,10]. Smoking status was defined as current versus non-current smoking. Blood pressure was measured in triplicate while participants were in a seated resting position, and the average of the second and third readings were recorded for both studies. Hypertension was defined as a systolic blood pressure (SBP) ≥130 mmHg, diastolic blood pressure (DBP) ≥80 mmHg or the use of antihypertensive medication[15]. A balanced beam scale and vertical ruler were used to measure height and weight, respectively, while wearing light clothing and no shoes. Body mass index was calculated as weight (kg) divided by height^2 (meters^2).

Fasting blood glucose was measured using a hexokinase/glucose-6-phosphate dehydrogenase method [16,17]. Type 2 diabetes was defined as a fasting blood glucose concentration ≥126 mg/dL or the use glucose-lowering medications. Total cholesterol and high-density lipoprotein-cholesterol (HDL-C) were measured enzymatically [16], and low-density lipoprotein-cholesterol (LDL-C) values were calculated using the Friedewald equation[18]. An elevated lipoprotein ratio was defined as a total cholesterol/HDL-C ≥3.5 or the use of lipid-lowering medications[19]. Non-HDL-C was calculated as the difference

between total cholesterol and HDL-C. Elevated non-HDL-C was defined as a value ≥160 mg/dL[20]. Obesity was defined as a BMI ≥30 kg/m^2 [21].

All analyses were conducted separately for younger versus middle-aged versus older adults. Study sample characteristics are presented as mean ± standard deviation (SD) for continuous variables, and categorical variables were presented as percentages. Continuous variables that were not normally distributed were presented as median (Q1, Q3). Differences between normally and non-normally distributed variables were assessed through the Student's t-test and Wilcoxon signed-rank test, respectively. Differences between categorical variables were evaluated through the chi-square test.

We calculated the absolute rate of CAC incidence across age groups by dividing the crude number of incident CAC events by their respective follow-up times (per 1,000 person-years). The association of traditional ASCVD risk factors and incident CAC was assessed through multivariable Cox proportional hazards regression. Traditional ASCVD risk factors were assessed both continuously (per SD change) and categorically. The primary ASCVD risk factor model adjusted for sex, race/ethnicity, education, income, and current cigarette smoking along with age, SBP, DBP, total cholesterol, HDL-C, fasting blood glucose as continuous risk factors. In subsequent ASCVD risk factor models, we evaluated total cholesterol/HDL-C in replacement for total cholesterol and HDL-C and non-HDL-C in replacement for total cholesterol as continuous variables. The primary categorical traditional ASCVD risk model adjusted for age, sex, race, education, income, current cigarette smoking, hypertension, total cholesterol/HDL-C ≥3.5, and type 2 diabetes mellitus.

In a subsequent categorical ASCVD risk factor model, we adjusted for a non-HDL-C value ≥160 mg/dL in replacement of a total cholesterol/HDL-C ≥3.5 as a marker of dyslipidemia. To test for interactions for risk factors with age, we assessed the significance of continuous and categorical ASCVD risk markers multiplied by age when added as regression terms to fully adjusted models. To assess the collective predictive ability of traditional ASCVD risk factors, we assessed model discrimination through calculated concordance statistics in multivariable Cox proportional hazards regression models. Differences in concordance statistics between models were assessed through approaches developed by Uno et al [22].

We performed three sensitivity analyses. First, additional hazard ratios for continuous traditional ASCVD risk factors, including age (per 10 years older), SBP and DBP (per 10 mmHg higher), total cholesterol (per 10 mg/dL higher), HDL-C (per 10 mg/dL lower), total cholesterol/HDL-C (per 1-unit higher), non-HDL-C (per 10 mg/dL higher), and fasting blood glucose (per 10 mg/dL higher) were calculated according to clinically relevant increments. Second, we calculated hazard ratios and concordance statistics after excluding participants on blood pressure-lowering, lipid-lowering, and/or glucose-lowering medications. Lastly, we calculated hazard ratios for traditional risk factors among MESA participants who had a follow-up CAC scan at Visit 5. This provided a follow-up time of approximately 10 years between CAC scans, which corresponded to the 10-year follow-period between CAC scans for CARDIA study participants.

Results

A total of 61% of adults were women and 37% were black. The proportion with incident CAC increased from younger (22%) to middle-aged (34%) and older adults (45%) (Figure 1). Regardless of age, most individuals (93%) who developed incident CAC had follow-up CAC scores <100 AU (Figure 2). Assessing middle-aged and older adults in MESA with a CAC scan at each visit separately, the proportion who developed incident CAC was highest for Visit 5, which corresponded to the longest follow-up period (Supplemental Figure 2). Over the CAC scan follow-up periods, younger adults had an incident CAC event rate of 22.4 per 1,000 person-years, compared to incident CAC event rates of 52.5 and 85.6 per 1,000 person-years for middle-aged and older persons, respectively (Supplemental Table 1).

SBP and fasting blood glucose values were higher with increasing age (Table 1). Middle-aged adults had the highest total cholesterol (194.5 mg/dL) and non-HDL-C values (142.7 mg/dL), while the proportion of individuals on lipid-lowering therapy was more than 2-fold higher in older adults compared to middle-aged adults (17.9% versus 8.6%).

In multivariable modeling, a 1-unit SD change in all continuous traditional modifiable ASCVD risk factors was significantly associated with incident CAC for younger and middle-aged adults except for 1) DBP, HDL-C, and BMI in younger adults and 2) DBP and fasting blood glucose in middle-aged adults. There were no significant associations between continuous risk factors and incident CAC observed among older persons (Table 2).

Only SBP and total cholesterol-HDL-C ratio had a significant interaction with age (p-interaction <0.01 for both), which remained significant even after excluding individuals taking blood pressure-lowering and lipid-lowering medications. There were no significant differences in these observed associations after excluding individuals on blood pressure-lowering, lipid-lowering, and/or glucose-lowering medications. Compared to per SD changes in traditional risk factors, evaluating risk factors using clinically relevant incremental changes yielded similar, albeit attenuated associations with incident CAC (Supplemental Table 2).

In categorical multivariable models, male sex was consistently associated with incident CAC regardless of age, whereas current cigarette smoking was associated with a significantly higher risk for incident CAC only among younger persons (*Table 3*). In younger persons, hypertension, a total cholesterol-HDL-C ratio ≥3.5, and non-HDL-C ≥160 mg/dL conferred a 28–37% higher risk for incident CAC. Similar to the continuous risk factor model, age significantly modified the association between non-HDL-C ≥160 and CAC (p-interaction=0.02). Total cholesterol-HDL-C ratio ≥3.5 was also significantly associated with incident CAC in older adults (HR=1.31, 95% CI: 1.01–1.69). Similar strengths of association were observed for continuous and categorical traditional risk factors when a follow-up time of 10 years between CAC scans was used for all age groups (Supplemental Table 3 and Supplemental Table 4).

The absolute C-statistic values for the prediction of incident CAC were smaller with increasing age group at 0.752 for younger adults, 0.645 for middle-aged adults, and 0.597 or older adults. Traditional risk factors also provided a stepwise smaller incremental

improvement in the C statistic with increasing age and there was no significant change for older adults (0.025, p=0.08) (Table 4). After excluding individuals on blood pressure-lowering, lipid-lowering, and/or glucose-lowering medications, the magnitude improvement in C-Statistics was similar across age groups (Supplemental Table 5).

Discussion

This community-based study is one of the first to provide information regarding risk predictors for the development of incident CAC across the adult life course. We found that almost all traditional risk factors were significantly associated with incident CAC in young adults, whereas among older adults, no single traditional risk factor was consistently associated with the development of incident CAC. Collectively, traditional risk factors significantly improved the prediction of incident CAC when added to demographic information in younger and middle-aged adults, but not among older persons. These results underline the importance of preventive strategies for preventing and treating modifiable risk factors, especially among young and middle-aged persons, and highlight the difficulty of traditional risk factor-based risk prediction approaches among older persons. Further research is needed to examine whether novel risk factors, such as NT-proBNP and troponin[23], may improve incident CAC prediction in adults ≥65 years old.

Among all traditional modifiable risk factors, only a total cholesterol/HDL-C ≥3.5 was significantly associated with incident CAC in older adults. Adults ≥65 years old with a total cholesterol/HDL-C ≥3.5 had a 30% higher risk for incident CAC compared to those with a ratio <3.5, although this observed risk was lower in magnitude compared to middle-aged (48%) and younger (37%) persons. However, nearly all lipid parameters in the current study were either significantly associated (total cholesterol-HDL-C ratio ≥3.5) or had borderline significant confidence intervals of 0.98–0.99 (total cholesterol, HDL-C, non-HDL-C) for their association with incident CAC in older persons.

Possible explanations for the non-significant association among older persons include the presence of resilience factors, a shorter duration of exposure time to elevated lipoprotein levels, higher prevalence of traditional risk factors, and smaller sample size compared to the younger and middle age groups. These findings of no significant difference in the relative risk should not negate the importance of appropriate risk based treatment for traditional risk factors among older persons as this age group as a whole has the highest absolute risk for ASCVD [24]. However, they do suggest that among older persons with CAC=0 a more lenient approach to traditional risk factor treatment and control may be reasonable given that CAC=0 is associated with a low ASCVD event rate regardless of age.

The major strengths of this study include the inclusion of a diverse range of men and women across different stages of the adulthood life course to assess the relationship of traditional risk factors with the onset of CAC. We also had well-defined and precise measurements of upstream ASCVD risk factors, including cholesterol, glucose, and blood pressure. Furthermore, we had repeat CAC scoring on individuals across the adulthood life course, which not that many studies have, and such a design enabled us to assess the initiation of CAC over time in various age groups. Finally, we conducted a robust statistical

assessment of traditional risk factors, assessing both their continuous and clinically relevant thresholds in the development of CAC across various age groups.

Our study should be interpreted in the setting of certain limitations. First, participants with CAC=0 have a low risk for CVD and therefore represent a generally healthier group of individuals compared to the general population, especially for older persons, an age group in which there is a higher prevalence of CAC >0 compared to younger persons. Accordingly, the results from this study examining risk factors for incident CAC are only directly applicable to persons with an absence of CAC. However, at baseline 38% of women age >65 had CAC=0 and there was a high rate of incident CAC among older participants with 45% developing incident CAC within 5 to 10 years follow-up. Similar to all studies including older persons, survival bias is also an important factor consider. Older persons in MESA may have been systematically different from the general population aged ≥65 years old, those who already had prevalent CAC, and/or those who were ill or died from competing risks, such as cancer [25]. In this scenario, the strength of association between traditional risk factors and incident CAC could be misrepresented and perhaps underestimated. We were also unable to measure the duration and/or intensity of exposure to traditional ASCVD risk factors and used a fixed binary definition of risk factors for all age groups. Older persons also generally have a higher prevalence of traditional ASCVD risk factors and inflammation has also been demonstrated to decrease the association between traditional risk factors with coronary heart disease among older persons[26].

With respect to study outcome, our study used CT-measured CAC as a measure of subclinical atherosclerotic disease burden. However, coronary computed tomography angiography (CTA) can measure noncalcified plaques, which develop first and therefore may have had a higher sensitivity for persons with a shorter duration of follow-up. Further research which assesses the relationship between traditional risk factors and CTA-measured subclinical atherosclerotic disease burden across different ages may thus be important mechanistically and for ASCVD prediction purposes[27]. Lastly, the repeat CT scan interval was not homogenous across all age strata, which may have influenced our identified hazard ratios for each traditional risk factor with the onset of CAC. We attempted to mitigate this heterogeneity by conducting an additional sensitivity analysis with a standardized interscan interval, and these results were consistent with the main study findings.

Overall, the observed stronger association between traditional ASCVD risk factors and incident CAC in young compared to older adults underscores the importance of primordial prevention and screening for traditional risk factors among young adults.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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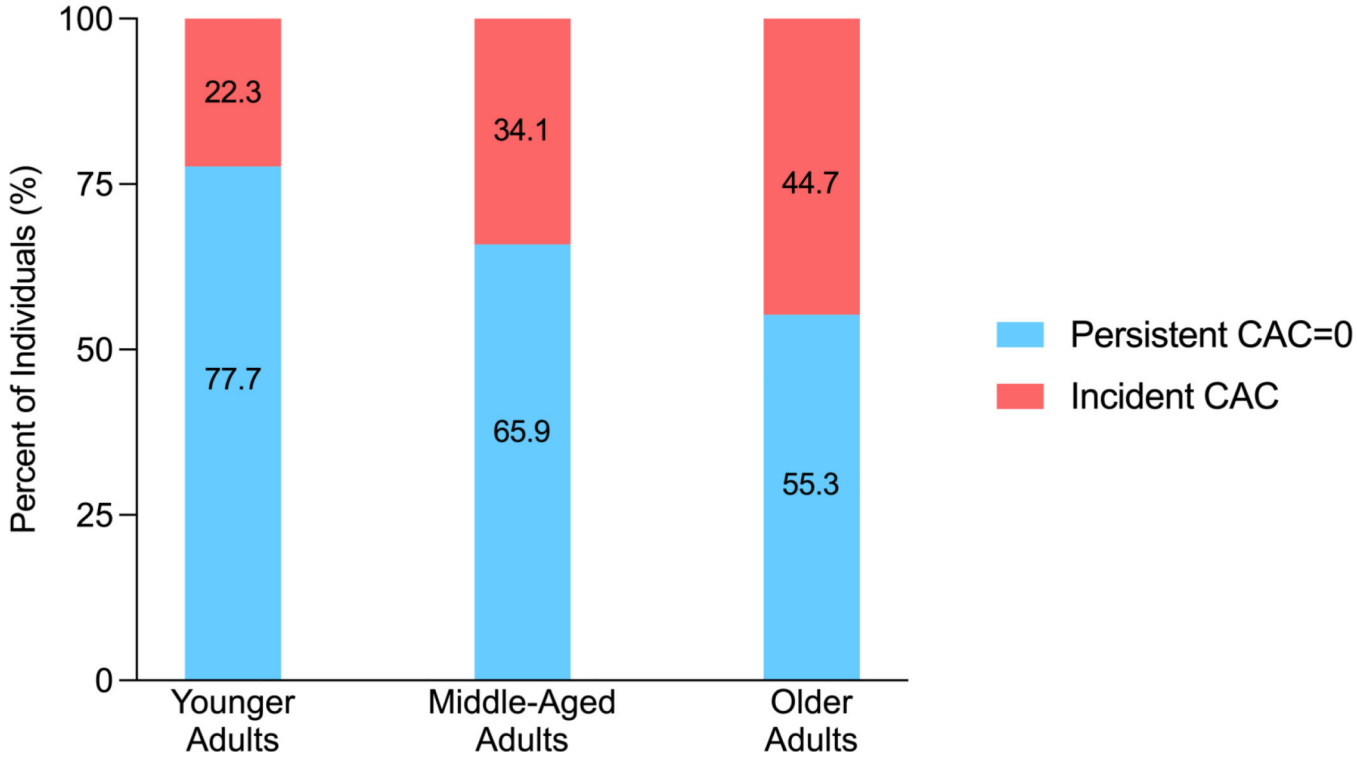

Figure 1.

Proportion of incident coronary artery calcium versus persistent absence of coronary artery calcium among younger, middle-aged, and older adults.

* The proportion with incident CAC increased from younger (22%) to middle-aged (34%) and older adults (45%)

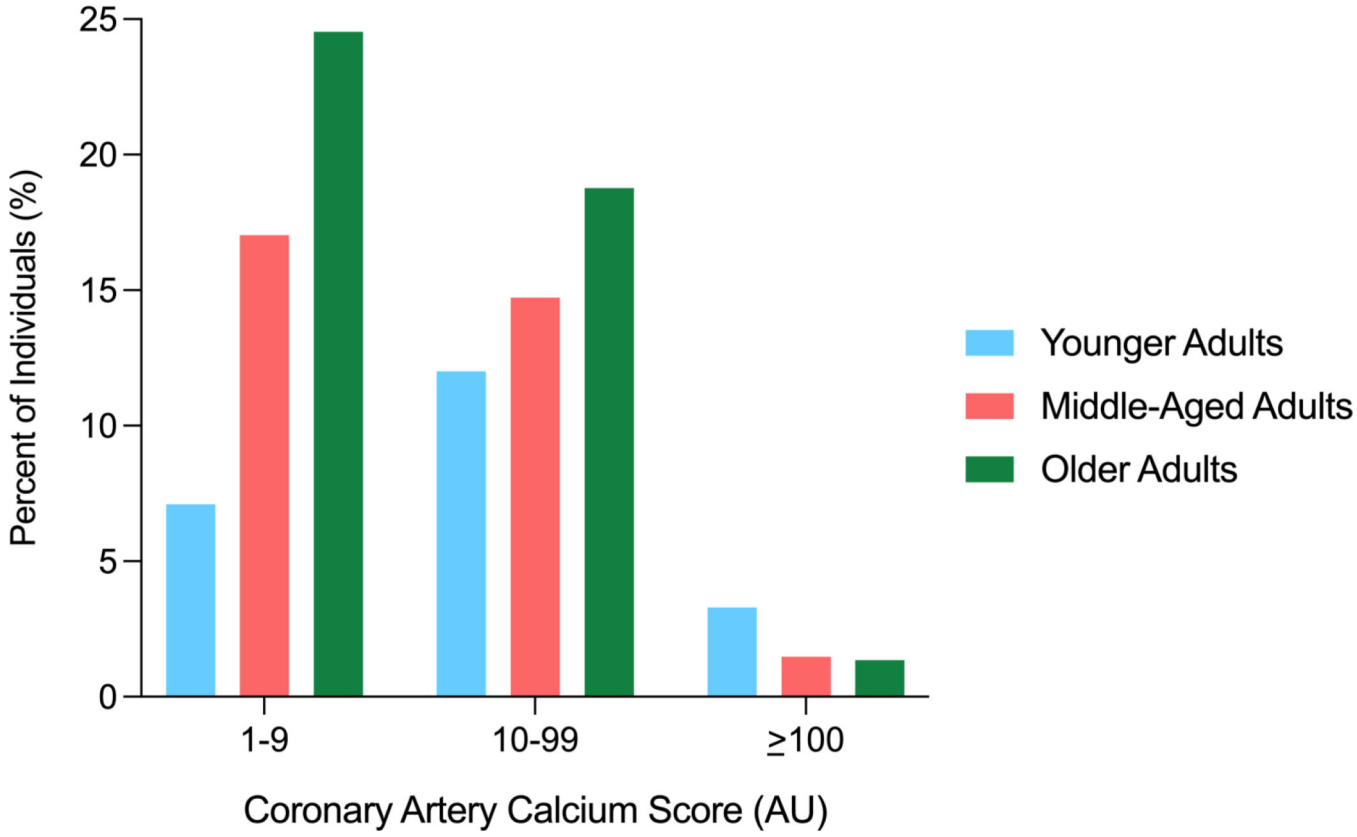

Figure 2.

Distribution of scores at incident coronary artery calcium detection among younger, middle-aged, and older adults with incident CAC.

* Regardless of age, the majority of individuals (93%) who developed incident CAC had follow-up CAC scores <100 AU.

Table 1.

Characteristics of 5,108 Participants with Baseline CAC=0 who had a Follow-Up CAC Scan

Variable	Age at the Time of Baseline CAC Scan (years)		
	32–45 (n=2,139)	46–64 (n=2,154)	65–84 (n=815)
Age, (years)	40.1 ± 3.5	54.1 ± 5.3	70.2 ± 4.5
Women	58.2 %	61.7 %	66.9 %
White	54.6 %	35.0 %	30.9 %
Black	45.4 %	29.9 %	35.3 %
Chinese	–	11.3 %	12.4 %
Hispanic	–	23.8 %	21.4 %
Incident CAC	22.3 %	34.1 %	44.7 %
CAC Score at Follow-Up, (AU)	0 (0, 0)	0 (0, 4)	0 (0, 7)
Systolic Blood Pressure, (mmHg)	112.0 ± 13.9	119.4 ± 18.5	131.1 ± 22.2
Diastolic Blood Pressure, (mmHg)	73.8 ± 10.9	71.6 ± 10.1	70.6 ± 10.5
Antihypertensive Medication	5.8 %	21.9 %	38.0 %
Type 2 Diabetes Mellitus	4.7 %	7.6 %	12.8 %
Fasting Blood Glucose, (mg/dL)	92.8 ± 16.6	93.0 ± 27.2	96.6 ± 22.7
Glucose-Lowering Medication	1.8 %	5.7 %	9.8 %
Total Cholesterol, (mg/dL)	183.9 ± 33.5	194.5 ± 35.5	192.7 ± 33.5
HDL-Cholesterol, (mg/dL)	51.1 ± 14.4	51.8 ± 14.8	55.1 ± 15.3
Non-HDL-Cholesterol, (mg/dL)	132.8 ± 35.8	142.7 ± 36.0	137.6 ± 32.8
Lipid-Lowering Medication	1.5 %	8.6 %	17.9 %
Current Smokers	19.3 %	14.8 %	5.8 %
Body mass index, (kg/m^2)	28.4 ± 6.3	28.6 ± 5.8	27.7 ± 5.2

Table 2.

Association of Continuous Traditional ASCVD Risk Factors (per standard deviation change) with Incident CAC, Stratified by Age

Risk Factor [*†]	Age at the Time of Baseline CAC Scan (years) 32–45 (n=2,139) HR (95% CI)	46–64 (n=2,154) HR (95% CI)	65–84 (n=815) HR (95% CI)	Age[*]Risk Factor Interaction P-Value
Systolic Blood Pressure (13.9 mmHg, 18.5 mmHg, 21.8 mmHg)	1.27 (1.11–1.46)	1.12 (0.99, 1.26)	1.11 (0.96, 1.29)	p<0.01
Diastolic Blood Pressure (10.9 mmHg, 10.1 mmHg, 10.3 mmHg)	0.98 (0.85–1.13)	0.99 (0.88, 1.12)	0.98 (0.83–1.15)	p=0.08
Total Cholesterol (33.4 mg/dL, 35.6 mg/dL, 33.6 mg/dL)	1.17 (1.07–1.27)	1.14 (1.07, 1.22)	1.11 (0.99–1.24)	p=0.38
HDL-Cholesterol (14.4 mg/dL, 14.8 mg/dL, 15.2 mg/dL)	1.07 (0.96–1.19)	1.23 (1.12, 1.34)	1.10 (0.97, 1.25)	p=0.24
Total Cholesterol/HDL-Cholesterol [ǂ] (0.5, 1.2, 1.1)	1.17 (1.07–1.27)	1.17 (1.10, 1.26)	1.10 (0.99, 1.23)	p<0.01
Non-HDL-Cholesterol [§] (36.8 mg/dL, 36.0 mg/dL, 32.7 mg/dL)	1.20 (1.09–1.31)	1.14 (1.07–1.23)	1.11 (0.99, 1.23)	p=0.36
Fasting Blood Glucose (16.6 mg/dL, 27.4 mg/dL, 23.1 mg/dL)	1.12 (1.03–1.21)	1.05 (0.98–1.13)	1.06 (0.95, 1.19)	p=0.05
Body mass index (6.3 kg/m^2, 5.8 kg/m^2, 5.1 kg/m^2)	1.08 (0.97–1.20)	1.19 (1.09–1.30)	1.00 (0.87, 1.14)	p=0.33

Model includes: baseline age, sex, race/ethnicity, education, income, cigarette smoking, systolic blood pressure, diastolic blood pressure, total cholesterol, HDL-cholesterol, fasting blood glucose, body mass index, and blood pressure-lowering, lipid-lowering, and glucose-lowering medications

[*] Standard deviation in each age group is listed next to each risk factor.

[†] All associations are reported per SD-higher except for HDL-cholesterol, which is reported per SD-lower.

[ǂ] In replace of total cholesterol and HDL-cholesterol in multivariable modeling.

[§] In replace of total cholesterol in multivariable modeling

Table 3.

Association of Categorized Traditional ASCVD Risk Factors with Incident CAC, Stratified by Age

Risk Factor *	Age at the Time of Baseline CAC Scan (years) 32–45 (n=2,139) HR (95% CI)	46–64 (n=2,154) HR (95% CI)	65–84 (n=815) HR (95% CI)	Age*Risk Factor Interaction P-Value
Man	2.07 (1.69–2.55)	1.45 (1.23–1.69)	1.62 (1.27–2.05)	p=0.11
White	1.21 (0.99–1.48)	1.34 (1.10–1.64)	1.29 (0.97–1.73)	p=0.14 ≠
Black	Ref	Ref	Ref	
Chinese	–	0.83 (0.62–1.12)	0.93 (0.63–1.37)	
Hispanic	–	1.09 (0.87–1.35)	0.96 (0.69–1.34)	
Current Cigarette Smoker	1.73 (1.40–2.14)	1.15 (0.93–1.41)	1.13 (0.69–1.83)	p=0.26
Hypertension	1.30 (1.07–1.59)	1.30 (1.07–1.57)	1.02 (0.77–1.35)	p=0.07
Total Cholesterol/HDL-Cholesterol ≥3.5	1.32 (1.06–1.65)	1.43 (1.19–1.72)	1.31 (1.01–1.69)	p=0.64
Non-HDL-Cholesterol ≥160 mg/dL †	1.28 (1.04–1.56)	1.46 (1.24–1.73)	1.11 (0.85, 1.46)	p=0.02
Type 2 Diabetes Mellitus	1.23 (0.82–1.85)	1.83 (1.18–2.85)	0.94 (0.46–1.93)	p=0.74
Body Mass Index ≥30 kg/m^2	1.10 (0.89–1.36)	1.32 (1.13–1.56)	0.90 (0.70–1.15)	p=0.15

*Model includes: baseline age, sex, ethnicity, education, income, cigarette smoking, hypertension, total cholesterol-HDL-cholesterol ratio, type 2 diabetes, body mass index, and blood pressure-lowering, lipid-lowering, and glucose-lowering medications.

†in replace of total cholesterol in multivariable modeling

≠White compared to Black

Table 4.

AUC Analysis for Incident CAC, Stratified by Age

	C-Statistic	Change in C-Statistic	C-Statistic Contrast P-Value
Younger Adults (n=2,139)			
Age, sex, race	0.682	–	–
Age, sex, race + individual traditional risk factors *	0.752	+0.070	<0.001
Middle-Aged Adults (n=2,154)			
Age, sex, race	0.591	–	–
Age, sex, race + individual traditional risk factors *	0.645	+0.054	<0.001
Older Adults (n=815)			
Age, sex, race	0.572	–	–
Age, sex, race + individual traditional risk factors *	0.597	+0.025	0.08

* cigarette smoking, systolic blood pressure, diastolic blood pressure, total cholesterol, HDL-cholesterol, fasting blood glucose, body mass index, antihypertensive medication, lipid-lowering medication, glucose-lowering medication