Adult Height and Prevalence of Coronary Artery Calcium
The National Heart, Lung, and Blood Institute Family Heart Study

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Background—Adult height has been hypothesized to be inversely associated with coronary heart disease; however, studies have produced conflicting results. We sought to examine the relationship between adult height and the prevalence of coronary artery calcium (CAC), a direct measure of subclinical atherosclerosis and surrogate marker of coronary heart disease.

Methods and Results—We evaluated the relationship between adult height and CAC in 2703 participants from the National Heart, Lung, and Blood Institute Family Heart Study who underwent cardiac computed tomography. We used generalized estimating equations to calculate the prevalence odds ratios for the presence of CAC (CAC>0) across sex-specific quartiles of height. The mean age of the sample was 54.8 years, and 60.2% of participants were female. There was an inverse association between adult height and CAC. After adjusting for age, race, field center, waist circumference, smoking, alcohol, physical activity, systolic blood pressure, antihypertensive medications, diabetes mellitus, diabietic medications, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, lipid-lowering medications, and income, individuals in the tallest quartile had 30% lower odds of having prevalent CAC. The odds ratios (95% confidence intervals) for the presence of CAC across consecutive sex-specific quartiles of height were 1.0 (reference), 1.15 (0.86–1.53), 0.95 (0.73–1.22), and 0.70 (0.53–0.93), and P for trend <0.01. There was no evidence of effect modification for the relationship between adult height and CAC by age or socioeconomic status.

Conclusions—The results of our study suggest an inverse, independent association between adult height and CAC.

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Key Words: diagnostic imaging ■ epidemiology ■ risk factors

The relationship between adult height and cardiovascular disease is unclear. Studies have shown an inverse association between adult height and several cardiovascular risk factors, as well as coronary heart disease (CHD) and cardiovascular mortality.1–6 However, other analyses reported no association between adult height and cardiovascular disease, especially those studies evaluating cardiovascular outcomes in nonwhite populations.7–12

Clinical Perspective on p 57

Several potential mechanisms could lead to an inverse association between adult height and CHD. Height is largely determined by genetic predisposition and environmental factors, such as nutrition, social networks, and physical environment. Childhood socioeconomic status (SES) heavily influences these environmental factors and is also a strong predictor of CHD.13,14 Loss of height in adulthood has been shown to predict cardiovascular mortality, potentially via a decrease in lung function.15,16 Finally, gravity is known to influence the cardiovascular system, and its effect varies according to height, with a decrease in cardiovascular afterload, hypertension, and the incidence of heart failure in taller individuals.4,17,18

As a marker of subclinical disease, coronary artery calcium (CAC) is an excellent marker of atherosclerotic plaque burden and has a high predictive value for the development of CHD, with a ≈10-fold increase in the risk of CHD events in patients with substantially elevated CAC.19 In addition to the increased risk seen with elevated CAC, a CAC score of zero has been shown to be a powerful predictor of low CHD risk, even in the presence of traditional risk factors.20,21 No previous study has examined the relationship of adult height and CAC in a large population. The aim of this study was to determine whether adult height is inversely associated with CAC.
Methods

Study Population
Our hypothesis was tested using data from participants of the National Heart, Lung, and Blood Institute (NHLBI) Family Heart Study who had undergone cardiac-gated multidetector computed tomodography (CT). The rationale and design of the NHLBI Family Heart Study has been previously published.12 Briefly, the goal of this study was to evaluate genetic and nongenetic predictors of cardiovascular risk factors, subclinical atherosclerosis, and CHD in families using a multicenter, population-based study. Probands were recruited from 4 previously established population-based cohorts (Framingham Heart Study, Atherosclerosis in Communities cohorts in Minneapolis and North Carolina, and participants of the Family Tree Health Study at the University of Utah). Later, a fifth center in Birmingham, AL, was added to increase the number of black participants.

Participants were selected either randomly (588 families, 2673 participants) or because of high-risk cardiovascular features (566 families, 3037 participants). Participants underwent a baseline clinical evaluation (1993–1995 for the initial cohort). During follow-up, approximately two thirds of the cohort was invited to undergo CT scanning from 2002 to 2004, in addition to the Birmingham participants. The study protocol was reviewed and approved by the participating institutions. Each participant gave informed consent for the study.

Of the 3389 individuals who underwent CT scans, 22 were missing CAC data, and 1 participant had missing data for height. There were 385 participants excluded from the analysis because of a previous myocardial infarction, percutaneous transluminal coronary angioplasty, or coronary artery bypass surgery, and 278 individuals had data missing for other relevant covariates. Therefore, 2703 individuals were included in the analysis for this study.

Anthropometric Measurements and Other Variables
All of the participants underwent a thorough evaluation at the baseline and during follow-up examinations. All measurements for this analysis were taken at the time of CAC measurement (2002–2004). Medical and lifestyle history was obtained via interview by centrally trained interviewers. Interviewers underwent periodic certification, and interviews were standardized by means of periodic review of taped interviews as well as distribution of feedback for individual reviewers and individual centers.

Adult height was recorded as standing height without shoes. Additional anthropometrics were collected in participants wearing scrub suits. Resting blood pressure was measured 3 times on seated participants after a 5-minute rest period. All patients were asked to fast for 12 hours before their visit. Lipid levels and fasting glucose levels were obtained for each individual, and low-density lipoprotein levels were calculated using the Friedewald formula. Hypertension was defined as a systolic blood pressure >140 mm Hg, a diastolic blood pressure >90 mm Hg, or an individual reporting that he/she was on antihypertensive medications. Diabetes mellitus was considered present if an individual was taking hypoglycemic agents, reported a previous clinical diagnosis of diabetes mellitus, or had a fasting glucose above the threshold of 7 mmol/L.

Race was based on self-reports including 2 categories: non-Hispanic white and black. Participants categorized into other races were not included because of inadequate numbers for separate analysis. Cigarette smoking was analyzed as number of pack-years (0, 1–15, 16–30, 31+). Alcohol was analyzed as number of drinks per day (0, 1–2, >2) based on reported consumption of alcoholic beverages. Physical activity was recorded based on self-report and analyzed by quartiles of total exercise activity (metabolic equivalents/minute/week). Income was measured as household income in 3 categories (<$25,000, $25,000–$75,000, and >$75,000).

Measurement of CAC
Cardiac CT examinations were performed using devices capable of 4 or 8 slices with different systems at each of the family heart study sites (General Electric Health Systems LightSpeed plus and LightSpeed Ultra, Siemens Volume Zoom, and Philips MX 8000). Studies were performed using the protocol established in the Multi-Ethnic Study of Atherosclerosis of the NHLBI.13 The scans were performed using prospective gating at 50% of the cardiac cycle, 120 kV, 106 mA, 2.5-mm slice collimation, 0.5-second gantry rotation, and a partial scan reconstruction resulting in a temporal resolution of 250 to 300 ms. A standard algorithm was used to reconstruct images into a 35-cm display field of view. A calcium calibration standard within the imaging field was used for all subjects (Image Analysis, Columbia, KY). The scan was repeated twice during the same examination. The average radiation exposure of each coronary scan was 1.5 mSv for men and 1.9 mSv for women.

All images were electronically transmitted to the central CT reading center at Wake Forest University Health Sciences, Salem, NC. Trained readers interpreted the images using dedicated hardware (GE Advantage Windows Workstation) and software (GE Smartscore). The total CAC score was calculated using the Agatston method based on the area and density of the calcified plaques.14 The CAC scores from each of the 2 scans were averaged to produce the final CAC score for each participant.

Statistical Analysis
For the primary analysis, we dichotomized CAC into zero and nonzero scores given the strong predictive value of a CAC score of zero and the increased cardiovascular risk for individuals with even minimal CAC.6 Baseline characteristics were determined according to quartiles of adult height. Baseline characteristics are described as mean (SD) for continuous variables and number (percentage) for categorical variables. Although the prevalence of CAC is known to vary by sex,15 we observed a similar inverse relationship in both men and women; therefore, we have presented data according to sex-specific quartiles.

To correct for the effect of familial clustering, we used generalized estimating equations to calculate the prevalence odds ratios for the presence of CAC across quartiles of height. Model 1 was adjusted for age and race. Model 2 was adjusted for age, race, field center, waist circumference, cigarette pack-years, alcohol, systolic blood pressure, antihypertensive medications, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, lipid-lowering medications, history of diabetes mellitus, diabetic medications, physical activity level, and income. The addition of education did not appreciably change the effect estimates. We evaluated age, race, and income as potential effect modifiers of the relationship between adult height and CAC. We obtained a P value for linear trend by creating a new variable that was assigned the median height value in each quartile and fitting the new variable in each regression model. Finally, adult height was examined as a continuous variable (odds ratio per increase in SD).

As a sensitivity analysis, we calculated the odds ratio per SD using different CAC cut points (CAC>50, CAC>100, and CAC>400). Significance level was set at 0.05. All analyses were performed with the use of SAS version 9.2 (SAS institute Inc, Cary, NC).

Results
Of the 2703 subjects analyzed, 1076 (39.8%) subjects were men, and the average age was 53.6 (SD, 12.7) years for men and 55.7 (SD, 12.5) years for women. The age range was 30 to 89 years. Baseline characteristics of men and women in the study sample are shown in Tables 1 and 2 according to quartiles of adult height. In men, taller participants were younger and had larger waist circumference, lower systolic blood pressure, and lower prevalence of hypertension and diabetes mellitus. Taller women were more likely to be younger and had lower systolic blood pressure, low-density lipoprotein cholesterol, and lower rates of hypertension.

There was evidence of an inverse association between adult height and CAC with 30% lower odds of CAC (95% confidence intervals [7%–47%]) comparing the fourth with the first quartile of adult height in the fully adjusted model (Table 3, P
linear trend < 0.01). Each SD of higher height was associated with 14% lower odds of CAC (95% confidence intervals [0% to 26%]) in the fully adjusted model (Table 3). There was no evidence of modification by sex in the fully adjusted model with a relative risk per SD of 0.86 (0.72–1.02) in men and a relative risk per SD of 0.89 (0.79–1.01) in women (interaction \( P \) value = 0.56). In stratified analysis by race, there did not seem to be an inverse relationship between adult height and CAC in blacks, although there was no conclusive evidence of effect modification (Table 4, \( P \) for race×height interaction = 0.51). There was no evidence for effect modification by age or income (results not shown).

Finally, a sensitivity analysis demonstrated similar results across different CAC thresholds. Odds ratios for CAC per SD higher height were 0.86 (0.72–1.04), 0.87 (0.71–1.07), and 0.81 (0.62–1.06) when we used scores of 50, 100, and 400 as thresholds, respectively, to define prevalent CAC in the fully adjusted model (Table 5).

### Discussion
The results of our study support the hypothesis that adult height is inversely associated with CHD in white populations. We found a significant inverse relationship between adult height and prevalent CAC. Individuals in the tallest quartile had 30% lower odds of prevalent CAC compared with individuals in the shortest quartile. Although the prevalence of CAC differs among men and women, the inverse association between height and CAC was similar in both sexes. To the best of our knowledge, this is the first study to demonstrate an inverse relation of adult height with subclinical atherosclerosis in a large population. Previous reports have focused on the relation of adult height with clinical CHD end points, and the majority of the studies demonstrating an inverse relationship between adult height and CHD have come from white populations. In the Physician’s Health Study, men in the tallest quintile, ≥185.4 cm (6’1”), had a 35% lower risk of myocardial infarction compared with individuals in the lowest quintile, ≤170.2 cm (5’7”).

### Table 1. Baseline Characteristics of 1076 Men in the NHLBI Family Heart Study According to Quartiles of Adult Height

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Q1 (1.60–1.72)</th>
<th>Q2 (1.73–1.76)</th>
<th>Q3 (1.77–1.81)</th>
<th>Q4 (1.82–2.03)</th>
<th>( P ) Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>294</td>
<td>219</td>
<td>297</td>
<td>266</td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>56.9±13.2</td>
<td>54.5±13.2</td>
<td>52.3±11.8</td>
<td>50.5±11.6</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>29.2±5.0</td>
<td>29.5±5.4</td>
<td>29.6±4.9</td>
<td>28.9±4.6</td>
<td>0.52</td>
</tr>
<tr>
<td>Race, %, white</td>
<td>82.3</td>
<td>83.1</td>
<td>83.5</td>
<td>84.6</td>
<td>0.47</td>
</tr>
<tr>
<td>Cigarette pack-years, %</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>51.7</td>
<td>51.1</td>
<td>56.9</td>
<td>59.4</td>
<td>0.03</td>
</tr>
<tr>
<td>1–15</td>
<td>16.3</td>
<td>17.8</td>
<td>15.5</td>
<td>22.9</td>
<td>0.10</td>
</tr>
<tr>
<td>16–30</td>
<td>13.6</td>
<td>14.6</td>
<td>12.8</td>
<td>9.0</td>
<td>0.09</td>
</tr>
<tr>
<td>30+</td>
<td>18.4</td>
<td>16.4</td>
<td>14.8</td>
<td>8.7</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Current drinker, %</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never</td>
<td>54.4</td>
<td>60.3</td>
<td>55.9</td>
<td>57.9</td>
<td>0.60</td>
</tr>
<tr>
<td>1–7 drinks/wk</td>
<td>22.8</td>
<td>16.9</td>
<td>22.9</td>
<td>18.8</td>
<td>0.53</td>
</tr>
<tr>
<td>8–14 drinks/wk</td>
<td>11.6</td>
<td>11.9</td>
<td>10.8</td>
<td>11.3</td>
<td>0.82</td>
</tr>
<tr>
<td>&gt;14 drinks/wk</td>
<td>11.2</td>
<td>11.0</td>
<td>10.4</td>
<td>12.0</td>
<td>0.84</td>
</tr>
<tr>
<td>Income, %</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;$25000</td>
<td>19.4</td>
<td>12.3</td>
<td>8.8</td>
<td>9.4</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>≥$25000–$75000</td>
<td>53.4</td>
<td>53.4</td>
<td>55.6</td>
<td>45.1</td>
<td>0.11</td>
</tr>
<tr>
<td>≥$75000</td>
<td>27.2</td>
<td>34.3</td>
<td>35.7</td>
<td>45.5</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Hypertension, %</td>
<td>47.6</td>
<td>40.2</td>
<td>34.7</td>
<td>31.6</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Antihypertensive meds</td>
<td>40.5</td>
<td>32.9</td>
<td>29.6</td>
<td>21.4</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Diabetes mellitus, %</td>
<td>11.6</td>
<td>11.4</td>
<td>10.8</td>
<td>6.4</td>
<td>0.05</td>
</tr>
<tr>
<td>Diabetic meds</td>
<td>8.2</td>
<td>9.1</td>
<td>8.8</td>
<td>5.3</td>
<td>0.24</td>
</tr>
<tr>
<td>Lipid-lowering meds</td>
<td>19.4</td>
<td>17.8</td>
<td>16.8</td>
<td>12.8</td>
<td>0.04</td>
</tr>
<tr>
<td>Waist circ, cm</td>
<td>101±13</td>
<td>104±14</td>
<td>105±13</td>
<td>104±12</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Phys act, met-min/wk</td>
<td>765±1246</td>
<td>1057±1376</td>
<td>827±978</td>
<td>1004±1333</td>
<td>0.14</td>
</tr>
<tr>
<td>Systolic BP, mm/Hg</td>
<td>123±18</td>
<td>124±18</td>
<td>122±16</td>
<td>121±17</td>
<td>0.03</td>
</tr>
<tr>
<td>LDL chol, mg/dL</td>
<td>112±32</td>
<td>117±33</td>
<td>113±31</td>
<td>113±36</td>
<td>0.84</td>
</tr>
<tr>
<td>HDL chol, mg/dL</td>
<td>45±12</td>
<td>44±12</td>
<td>44±12</td>
<td>44±12</td>
<td>0.22</td>
</tr>
</tbody>
</table>

*Act indicates activity; BMI, body mass index; BP, blood pressure; chol, cholesterol; circ, circumference; HDL, high-density lipoprotein; LDL, low-density lipoprotein; meds, medications; n, number; NHLBI, the National Heart, Lung, and Blood Institute; and phys, physical.

**χ² test used for dichotomous variables; ANOVA used for continuous variables.**
Data from 31,199 men and women in Finland demonstrated ≈10% lower risk in cardiovascular mortality per 5 cm additional height. Conversely, studies from nonwhite populations in Japan, Korea, and Iran have demonstrated no association between adult height and CHD. In our study, odds ratios for blacks do not seem to show an inverse linear association between height and CAC. This finding would support the hypothesis that race is an effect modifier of the relationship between adult height and CHD. However, we lacked adequate statistical power to fully address this issue with only 541 black participants. Additional investigation with adequate statistical power is needed to clarify the relation of adult height and CAC in nonwhite populations. Not all exposures with established associations with CHD have been found to have a similar association with subclinical atherosclerosis. For instance, moderate alcohol consumption has a well-established association with a lower risk of CHD events but is associated with a higher prevalence of CAC, suggesting a nonatherosclerotic mechanism. The results of our study indicate that the relationship between adult height and CHD is mediated through atherosclerosis. Childhood SES influences adult height and has been shown to be predictive of the risk of CHD as an adult. Additionally, the association between height and CHD may vary according to SES. A previous study by Langenberg et al demonstrated that the inverse association between adult height and CHD in male civil servants was limited to participants with high levels of SES because no relationship was seen in male civil servants with lower levels of SES. Our study suggests that the inverse association between adult height and CAC is independent of SES, and we saw no evidence of interaction between income and height, although adjustment for level of income during adulthood may not adequately adjust for childhood levels of SES. Although ethnicity and SES may modify the relationship between height and CHD, these factors do not point to a specific pathophysiologic mechanism. Gravitational forces on the cardiovascular system...
Cardiovascular disease (CVD) is the leading cause of death in the United States. Significant long-term determinants of CVD include, but are not limited to, age, sex, race, socioeconomic status, genetic risk factors, and traditional cardiovascular risk factors. Among the traditional risk factors, height has been shown to have a protective effect against the development of CVD.1–5 A recent study indicates that adult height is inversely associated with the prevalence of coronary artery calcium (CAC).6

**Table 3. Adjusted Odds Ratios (95% CI) for CAC According to Sex-Specific Quartiles of Height and per SD Increment of Height**

<table>
<thead>
<tr>
<th>Sex-Specific Quartiles of Height</th>
<th>CAC Cases**</th>
<th>Model 1</th>
<th>Model 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Q1 (reference)</td>
<td>429/691</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Q2</td>
<td>377/622</td>
<td>1.18 (0.90–1.53)</td>
<td>1.15 (0.86–1.53)</td>
</tr>
<tr>
<td>Q3</td>
<td>365/702</td>
<td>0.96 (0.75–1.22)</td>
<td>0.95 (0.73–1.23)</td>
</tr>
<tr>
<td>Q4</td>
<td>276/688</td>
<td>0.68 (0.53–0.88)</td>
<td>0.70 (0.53–0.93)</td>
</tr>
<tr>
<td>P for linear trend</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td>Odds ratio per SD*</td>
<td>0.83 (0.72–0.96)</td>
<td>0.86 (0.74–1.00)</td>
<td></td>
</tr>
</tbody>
</table>

Model 1 is adjusted for age and race. Model 2 is adjusted for age, race, field center, waist circumference, cigarette pack-years, alcohol, physical activity, systolic blood pressure, antihypertensive medications, diabetes mellitus, diabetic medications, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, lipid-lowering medications, and income. CAC indicates coronary artery calcium; and CI, confidence intervals.

*Additional sex adjustment.

**CAC defined as CAC>0.

Vary by height because taller individuals have been shown to experience lower levels of afterload, a decrease in pulse pressure, and a lower prevalence of hypertension, which could lead to a decrease in the formation of atherosclerotic plaque.4,17 However, given the increased prevalence of CHD in men and western populations despite significant increased height in these individuals compared with their counterparts, the effect of gravitational forces on the risk for CHD is likely modest at best compared with traditional risk factors. Our study has limitations. Adult height was self-reported. Given the observational nature of our study and the variance in the baseline characteristics across the quartiles, residual confounding may still be present in the multivariable model. Adult loss of height has been shown to be an independent predictor of CHD,15 and the cross-sectional nature of our analysis did not allow us to account for changes in height over time. Shorter individuals in our study may simply represent individuals with greater degrees of height loss during adulthood. However, previous research evaluating men at the time of university enrollment suggests that the relationship between height and CHD is independent of loss of height during adulthood.29 Data were not available on childhood environmental factors that may influence height.26,27 Also, CAC is a surrogate marker of CHD, representative of subclinical atherosclerosis as opposed to actual CHD events. However, CAC is a strong predictor of future CHD events and was recently shown to be the only novel predictor with the capacity to substantially improve discrimination above and beyond traditional risk assessment.30 CAC has been shown to highly correlate with total atherosclerotic plaque burden, even more so than luminal stenosis.31,32 This analysis has several strengths including a large sample, extensive data on traditional cardiovascular risk factors, and a standardized approach to CAC assessment. In conclusion, our study indicates that adult height is inversely associated with the prevalence of CAC.

**Table 4. Adjusted Odds Ratios (95% CI) for CAC per SD of Height Using Different CAC Thresholds**

<table>
<thead>
<tr>
<th>CAC Thresholds</th>
<th>Odds Ratio Per SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>CAC 0</td>
<td>0.86 (0.74–1.00)</td>
</tr>
<tr>
<td>CAC 50</td>
<td>0.86 (0.72–1.04)</td>
</tr>
<tr>
<td>CAC 100</td>
<td>0.87 (0.71–1.07)</td>
</tr>
<tr>
<td>CAC 400</td>
<td>0.81 (0.62–1.06)</td>
</tr>
</tbody>
</table>

Model adjusted for age, race, field center, waist circumference, cigarette pack-years, alcohol, physical activity, systolic blood pressure, antihypertensive medications, diabetes mellitus, diabetic medications, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, lipid-lowering medications, and income.

CAC indicates coronary artery calcium; and CI, confidence intervals.

Acknowledgments

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Disclosures

None.

References


**CLINICAL PERSPECTIVE**

Multiple previous studies have suggested that taller adults have lower rates of cardiovascular disease; however, the mechanism of this relationship is unclear. The results of our study demonstrate an inverse relationship between coronary artery calcium, a direct marker of atherosclerotic plaque, and adult height. Although an atherosclerotic mechanism would be expected for the relationship between adult height and cardiovascular disease, other cardiovascular risk factors have not always shown a similar correlation with coronary artery calcium. For instance, moderate alcohol intake, despite a well-established association with a lower rate of cardiovascular events, has been shown to be associated with a higher rate of coronary artery calcium.

The results of our study suggest that the relationship between adult height and cardiovascular disease is mediated through an atherosclerotic process. Additionally, the association between adult height and cardiovascular disease has mainly been seen in white populations, with studies in other races and ethnicities yielding mixed results. In our study, the inverse relationship between adult height and coronary artery calcium was less evident in black participants compared with white participants, suggesting potential effect modification according to race. However, a limitation of this analysis was relatively few black participants. Research in additional ethnic groups is needed.
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