Body Height Is Associated With Decreased Long-Term Stroke but Not Coronary Heart Disease Mortality?

Uri Goldbourt, PhD; David Tanne, MD

Background and Purpose—There is disagreement whether shorter persons suffer increased rates of coronary heart disease (CHD) or stroke. Potential mechanisms for such associations are not well understood. We used findings from a cohort study, in which 10 000 Israeli men were followed up, to examine the associations between stature and fatal CHD as well as fatal stroke.

Methods—The cohort was composed of 10 059 men aged ≥40 years who were tenured civil servants or municipal employees. They were followed up for mortality over 23 years (1963 to 1986), for a total of 203 452 person-years of follow-up. We divided men by their height, as measured in the baseline (1963) examinations, into quartiles (≤162 cm, 163 to 167 cm, 168 to 171 cm, and ≥172 cm).

Results—During the follow-up period, 1098 men died of CHD, and 364 men died of stroke. Height and weight had been measured for 10 034 men, including all but 1 of the deceased. In contrast to the finding of little variation of CHD death rates between different quartiles of body height, a clear significant pattern of declining stroke mortality (slightly reduced by age adjustment) was observed with increasing body height, with rates of 46, 36, 33, and 29 per 1000 men with increasing height quartiles, respectively (P<0.002 for linear trend). The estimated age-adjusted hazard risk of stroke mortality associated with a 5-cm decrement in height was 1.13 (95% CI 1.04 to 1.22). The respective risk associated with being at the shortest quartile versus the tallest one was 1.54 (95% CI 1.13 to 2.10). Adjustment for socioeconomic status, a predictor of stroke in this cohort, for antihypertensive therapy and for established predictors of stroke (blood pressure, smoking, and diabetes) did not alter these findings.

Conclusions—Height, a potential strong indicator of nutritional status, may be inversely associated with the long-term incidence of fatal stroke in a way that remains to be elucidated. (Stroke. 2002;33:743-748.)

Key Words: body height ■ coronary heart disease ■ mortality ■ prospective studies ■ stroke

Coronary heart disease (CHD) and stroke are greatly increased in persons with high blood pressure and/or diabetes mellitus and in those who smoke cigarettes. CHD is often also anteceded by a compromised blood lipid profile. However, major ethnic differences in fatal CHD and stroke that do not lend themselves to explanation by corresponding differences in the distributions of variables identified as risk factors for these diseases have also been identified. Genetic and nutritional factors may play a role, as yet only partly identified, in determining the above risks of stroke.

Body height, a variable determined through interaction between genetic endowment, nutrition, and perhaps other unknown factors, is easy to measure. Several investigators have proposed that shorter persons suffered increased rates of CHD (in one instance, the study was of physicians). Others have not concurred. There has been even less research involving stature and the risk of stroke. To determine any association between stature and fatal CHD and between stature and fatal stroke, we used a study in which 10 000 men were recruited, underwent extensive appraisal of health and behavioral patterns, and were followed up for mortality over a long period of time. These men were from different continents; 86% of them had immigrated to the current territory of Israel. For this group, a clear ethnic-geographic diversity in the rates of fatal stroke has been shown.

Subjects and Methods

We followed up 10 059 Israeli civil servants and municipal employees, engaged in diverse occupations, aged ≥40 years (mean age at entry 49.2 years) between 1963 and 1986, for a total of 203 452 person-years (PY) of follow-up. Follow-up after 1968 was only for mortality, and incidence data are not available. Among these men, only 14% had been born in the territory that later became Israel. All others had emigrated from European, Asian, and North African countries. Body height (without shoes) was measured (in all but 25 of the study subjects) to the nearest centimeter; the mean height was 167 cm. With the subjects wearing trousers only, weight was...
TABLE 1. Height, Other Risk Factors, and Stroke Mortality by Area of Birth

<table>
<thead>
<tr>
<th>Area of Birth</th>
<th>n</th>
<th>Mean</th>
<th>SD</th>
<th>SBP, mm Hg</th>
<th>DBP, mm Hg</th>
<th>Diabetes, %</th>
<th>Smoking, %</th>
<th>Age-Adjusted Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Israel</td>
<td>1430</td>
<td>167.2</td>
<td>7.0</td>
<td>134.8</td>
<td>84.1</td>
<td>6.1</td>
<td>52.4</td>
<td>15.9</td>
</tr>
<tr>
<td>Mideast</td>
<td>2367</td>
<td>166.1</td>
<td>6.4</td>
<td>133.7</td>
<td>82.9</td>
<td>5.1</td>
<td>56.2</td>
<td>17.1</td>
</tr>
<tr>
<td>North Africa</td>
<td>1217</td>
<td>167.7</td>
<td>6.5</td>
<td>132.8</td>
<td>83.1</td>
<td>4.8</td>
<td>60.3</td>
<td>19.0</td>
</tr>
<tr>
<td>Eastern Europe</td>
<td>1918</td>
<td>166.3</td>
<td>6.6</td>
<td>136.8</td>
<td>84.3</td>
<td>4.5</td>
<td>44.0</td>
<td>12.4</td>
</tr>
<tr>
<td>Central Europe</td>
<td>1371</td>
<td>168.3</td>
<td>6.5</td>
<td>136.6</td>
<td>84.7</td>
<td>3.8</td>
<td>41.6</td>
<td>11.6</td>
</tr>
<tr>
<td>Southern Europe</td>
<td>1731</td>
<td>168.0</td>
<td>6.6</td>
<td>136.9</td>
<td>84.6</td>
<td>4.2</td>
<td>48.2</td>
<td>11.3</td>
</tr>
</tbody>
</table>

SBP indicates systolic blood pressure; DBP, diastolic blood pressure; diabetes, percentage diagnosed in 1963 (previously known and newly recognized); and smoking percentage of men reporting cigarette smoking in 1963. Age adjustment was by the direct method to the overall study aged. Rates are given per 10,000 PY of follow-up. 

Results

During the follow-up period, 1098 men died of CHD, and 364 men died of stroke. The mean age at death of CHD was 66.6 (SD 8.4) years, whereas the mean age at stroke death was 68.7 (SD 7.9) years. The latter decedents had started the study at 54.1 ± 6.5 years of age (eg, 5 years older than their counterparts who survived or died of other causes). Height and weight had been measured for all but 1 of the deceased. Significant differences were found according to birthplace. CHD was highest among the European-born men and lowest in men born in Asia and Africa. Conversely, fatal stroke rates for European-born men were 62% of the fatal stroke rates for North African–born men.

Table 1 shows the mean and SD height values, other stroke risk factors, and the stroke mortality rates in the various area-of-birth groups. Despite a significant ANOVA (P < 0.05), differences in mean body height were small. Age adjustment had a negligible effect on this finding. The age-adjusted mortality rates were high in the non–European-born men and lower in the European-born men.

The mean body height of study participants was 167.1 ± 6.6 cm. It was progressively lower with older age, from 168.4 cm at age 40 to 44 years to 165.0 cm at age ≥60 years (data not shown). The respective mean ± SD heights in the 4 quartiles (defined in Subjects and Methods) were as follows: 158.6 ± 3.5, 165.1 ± 1.4, 169.5 ± 1.1, and 175.6 ± 3.4 cm, respectively.

The distribution of other coronary or cerebrovascular risk factors in quartiles of body heights is shown in Tables 2 (for continuous variables) and 3 (for attributes).
Mortality by Height

There was almost no variation of the 23-year CHD death rates between different quartiles of body height (Table 4). The results did show a clear significant pattern of declining stroke mortality with increasing body height. Adjustment for age somewhat attenuated the association but did not eliminate it. The age-adjusted rates declined from 46 per 1000 men from the shortest quartile of men to 29 per 1000 among the tallest. With the fatal stroke risk set at 1.00 for the quartile of shortest men, the respective risk calculated by the Mantel-Haenszel method, pooling over 3 age groups (40 to 49, 50 to 59, and ⩾60 years), were as follows: 0.78 (95% CI 0.60 to 1.07), 0.72 (95% CI 0.54 to 0.98), and 0.62 (95% CI 0.45 to 0.85) for the second, third, and top quartile, respectively. The extended Mantel-Haenszel trend test yielded a \( \chi^2 \) (1 df) value of 9.27 \((P=0.002)\).

Calculated per 10 000 PY (data not tabulated), the age-adjusted rates declined from 23 to 14 per 10 000 PY between men in the shortest and tallest quartiles.

Multivariate Analysis

The estimated crude hazard ratio (HR, by Cox regression) of fatal CHD, associated with a decrement of 5-cm body height (Table 4) was 1.09. To account for the fact that the older men were shorter, further adjustment was made for age, and the excess risk was eliminated entirely (Table 4). For stroke mortality, the unadjusted HR of 1.23 was reduced to 1.13 by similar age adjustment. By use of the Mantel-Haenszel estimate, a nearly identical result for the rate ratio, controlling for time and age, was obtained. By Cox regression, the adjusted HR was 1.12 (95% CI 1.04 to 1.21), and it remained unchanged at 1.12 (95% CI 1.03 to 1.21) after additional adjustment for area of birth, whereas the HR for CHD death remained near unity, with HR 1.02 (95% CI 0.98 to 1.07). Testing the assumption of proportional hazards by use of the Schoenfeld method yielded a \( \chi^2 \) (2 df) value of 0.05 \((P=0.98)\); thus, neither height nor age violated the assumption.

For the shortest men in this cohort (≤162 cm), a relative HR of 2.15 (95% CI 1.59 to 2.91) for dying of stroke was reduced by age adjustment to 1.54, observed vis-à-vis the tallest men (≥172 cm). In contrast, men at the shortest and tallest height quartiles in 1963 showed no difference in their long-term risk of fatal CHD.

Body height at age ≥40 years is unlikely to be associated with disease occurrence through the confounding of diabetes, blood pressure, serum cholesterol levels, or cigarette smoking and even less to be influenced by them. Nevertheless, adjusting for these variables, we reran the Cox regression. The adjusted HR associated with a 5-cm decrement, given such additional adjustment, was marginally altered to 1.09 (95% CI 1.02 to 1.18). When we further adjusted for an index of socioeconomic status, on the basis of years of formal education and on type of employment (for which “professional” was considered the highest status, and “laborer” was considered the lowest), the estimated hazards of stroke mortality were not altered. No adjustment for baseline dietary habits was undertaken, because these almost certainly do not reflect the diet to which these men had been exposed in childhood (the men were mostly born overseas in unrelated environmental and economic conditions), nor were any of the nutrients assessed in the present study predictive of fatal stroke.

Because antihypertensive therapy may affect the value of blood pressure and the long-term rates of cerebrovascular disease, differences in implementation of such therapy, if associated with height or with a sociodemographic parameter that had prevailed since childhood and may have influenced height, could confound the results. Information on antihypertensive therapy in the study sample was available only for the 9492 men reexamined in 1968 who had had their height measured. Only 380 men (4.0%) reported being treated. Those men were marginally shorter (mean height 166.5±6.6 cm) than the untreated men (mean height 167.2±6.6 cm). The reporting of antihypertensive therapy was strongly predictive of stroke (age- and height-adjusted HR 3.80, 95% CI 0.96 to

<table>
<thead>
<tr>
<th>TABLE 2. Risk Factors for Stroke by Quartiles of Body Height</th>
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<tbody>
<tr>
<td>Height, cm</td>
</tr>
<tr>
<td>≤162</td>
</tr>
<tr>
<td>163–167</td>
</tr>
<tr>
<td>168–171</td>
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<tr>
<td>≥172</td>
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</tbody>
</table>

TC indicates total cholesterol; HDL-C, HDL cholesterol; and BMI, body mass index. Values are mean±SD.

<table>
<thead>
<tr>
<th>TABLE 3. Age-Adjusted Frequency of Risk Factors for Stroke by Quartiles of Body Height</th>
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<tbody>
<tr>
<td>Height, cm</td>
</tr>
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<td>≤162</td>
</tr>
<tr>
<td>163–167</td>
</tr>
<tr>
<td>168–171</td>
</tr>
<tr>
<td>≥172</td>
</tr>
</tbody>
</table>
However, its inclusion or exclusion from a multivariate model had a nil effect on the association of height with fatal stroke between 1968 and 1986 (HR 0.881 associated with being 5 cm taller when included, HR 0.886 when excluded). Similar results were obtained for all-cause mortality (data not shown). Finally, we have looked into the possibility that within diverse geographical origins, the height–fatal stroke association varied importantly. In the 6 stratified-sampling areas of birth, the area-specific age-pooled risk ratios associated with a shift of 1 height quartile all fell between 1.04 and 1.29, with the associated CIs showing considerable overlap. Alternatively, inclusion of birth in Europe in the Cox proportional hazards regression or stratification for it in the Mantel-Haenszel analysis had no effect on the height-associated variation in fatal stroke rates.

The Figure shows the patterns of stroke mortality over 23 years as occurring in the 4 body-height quartile groups. Half way into the long-term follow-up of these men, stroke-free survival shows an inverse relationship with the height quartiles, as assessed at the baseline examination.

**Discussion**

An association between body height and mortality from different causes has been reported as an ecological phenomenon and as occurring across groups; this association has also been noted by within-population observations. In the counties of England and Wales during 1968 to 1978, taller populations exhibited lower mortality from chronic bronchitis, rheumatic heart disease, CHD, and stroke (and higher mortalities from several cancers).

Studies in several large samples, including a study of 22,000 male physicians and another study of 121,700 US female nurses followed up for 14 years, consistently demonstrated a protective association of body height, independent of age, and fatal CHD. No association of that nature was found in the Framingham Heart Study, nor was an association found in a representative sample of men and women in the US population (at the National Health and Nutrition Examination Survey [NHANES]) that were followed up for 13 years. Adjustment for lung function in the later study did not alter the previous findings (ie, no association with CHD mortality). The present study, limited to men, agrees with the latter findings. In our cohort, the differences in CHD mortality between men of different heights were entirely due to the inverse association of height with age. The differences in different cohorts are not easy to explain.

Research is more limited when the association of body height with stroke is concerned. The Nurses Health Study looked at this association and identified none for women. In the Finnmark study of 13,652 middle-aged Norwegians (1974 to 1988, involving men and women, aged 35 to 52 years), 241 first events of stroke were registered: 144 among men and 97 among women. In that study, per 5-cm height increase, there was an estimated 18% decrease of stroke incidence in men and a 25% decrease in women. Unlike the present study, there was an important variation of mean body height by origin: Persons of Saami and Saami/Finnish origin were considerably shorter than were men of Finnish and Norse origin. Yet there was no ecological correlation between mean body height and stroke incidence rates. The same holds true for the present study, in which all the groups exhibited similar height distributions yet showed sizable differences in fatal stroke rate, underscoring the important role of other factors that distinguish men from different origins.

In the British Regional Heart Study, 7735 men, aged 40 to 59 years, went on to experience 1093 major CHD and 351 major stroke events over 15.5 to 18.0 years. The highest quintile of height was associated with a 26% decrease in CHD risk. The association with nonfatal stroke risk was less pronounced, whereas fatal stroke incidence (although based on only 63 deaths) declined stepwise with successive height quintiles to 0.77, 0.69, 0.69, and 0.54. Parker et al examined the 11-year CHD and stroke events on the basis of medical records of 2826 men and 3741 women from 2 communities in

<table>
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<tr>
<th>Height, cm</th>
<th>Men, n</th>
<th>CHD</th>
<th>Stroke</th>
<th>CHD</th>
<th>Stroke</th>
<th>CHD</th>
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<td>122</td>
<td>117</td>
<td>51</td>
<td>107</td>
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<td>163–167</td>
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<td>117</td>
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<td>71</td>
<td>105</td>
<td>32</td>
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<tr>
<td>≥172</td>
<td>2496</td>
<td>233</td>
<td>63</td>
<td>93</td>
<td>25</td>
<td>107</td>
<td>29</td>
</tr>
</tbody>
</table>

P = 0.002 for increasing trend of age-adjusted fatal stroke rate with height.

The Figure shows the patterns of stroke mortality over 23 years as occurring in the 4 body-height quartile groups. Half way into the long-term follow-up of these men, stroke-free survival shows an inverse relationship with the height quartiles, as assessed at the baseline examination.
southeastern New England. Men whose height exceeded 69.75 inches (177.1 cm) exhibited an 83% lower CHD risk and 67% lower stroke risk compared with the risk for their counterparts whose height was <65 inches (<165 cm).

In a recent extensive examination of the factors that predicted the incidence of fatal stroke over 20 years in the large Renfrew and Paisley study in Scotland, Hart et al22 found that age-adjusted fatal stroke rates among men declined progressively from 43 to 38, 33, 33, and 26 per 10 000 PY, respectively, with the 4 increasing height quintiles. A 1-SD decrement was associated with a 21% increase in rates. These results are quite similar to ours, even though the authors were able to adjust additionally for forced expiratory volume over 1 second and cardiothoracic ratio. Presumably the forced expiratory volume over 1 second, which is correlated with height, may interact to coaffect mortality.23 Among women, the association between body height and stroke mortality, as assessed in the Renfrew and Paisley study, appeared somewhat weaker than the association in men.

The controversy as to whether increased height is a protective factor against reduced CHD risk, is a marker of reduced CHD risk, or is neither is likely to continue. The present study further confounds the initially attractive hypothesis, ie, that factors affecting growth in childhood later influence the probability of adult CHD mortality. Given the paucity of stroke incidence studies of persons with different heights, it is more interesting in this context to speculate on possible mechanisms that control the association between decreased height and increased fatal stroke late in life. Factors connected with health and nutrition in early life may be related to stroke morbidity and mortality,23 even among nonsmokers.24 Short stature may serve as an indicator of socioeconomic deprivation, interrupting growth in childhood and adolescence. How, exactly, the latter bears on long-term prognosis is not easy to delineate. The Barker hypothesis (ie, infant and fetal undernutrition increases the risk of coronary disease in adulthood)25 has its supporters and detractors. An interesting analysis, which went as far back as a survey of diet and health in prewar England,26 revealed that leg length in childhood was the anthropometric measure most closely related to socioeconomic and dietary exposures. Leg length was also inversely related to CHD mortality later in life as these boys and girls reached middle and old age. The authors concluded that adverse diet and living conditions in childhood influenced late prognosis. To what extent these factors would specifically increase the rate of fatal stroke remains a point of speculation. Adverse socioeconomic conditions in childhood have been shown, in a separate study, to precede an increase adult stroke mortality.27 Our assessment of socioeconomic status, based on years of schooling and type of employment, was performed in men aged ≥40 years. It probably reflected very little of the childhood social environment, because of the major intergenerational changes, resulting from the holocaust and the disruption of Jewish life almost everywhere in Europe and in Mideast and North African Arab countries. Practically all the migrants participating in the present study were from those areas. Although it was associated with fatal stroke, the baseline (1963) socioeconomic status did not alter the height–fatal stroke associa-

tion, nor did antihypertensive therapy (scarce when this sample was examined in the 1960s) confound it. This does not exclude the possibility that other unrecorded socioeconomic parameters were associated with height attained and stroke mortality. We collected no data regarding the physical environment, whether in childhood or later; thus, we cannot relate any such data with the findings.

In conclusion, we have demonstrated a very weak association, or lack of one, between short stature and fatal CHD, whereas for fatal stroke, our results are consistent with stepwise increased risk with decreasing heights. We do not know how this is divided between an association of height with incidence and an association of height with case fatality, and we cannot resolved this from our data, which are limited to fatal cases. Height might represent a strong indicator of nutritional status, especially in a study such as ours, which included many subjects who had lived as persecuted minorities in their childhood. It could also be associated with environmental conditions in childhood and adolescence. Because our cohort included only Jewish men, its findings cannot be extended to women and may not reflect the association in the non-Jewish Israeli men. Further analyses of patients with use of the existing cohort data collected in prospective studies of CHD, stroke, or mortality in general should clarify to what extent other unmeasured risk factors may account for a part of the association between height and stroke mortality.

Acknowledgment

This study was completed while Dr Goldbourt was a visiting professor at the Department of Preventive Medicine and Epidemiology, Loyola University Chicago, Maywood, Ill.

References


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