# Adult Height, Stroke, and Coronary Heart Disease

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An inverse relation between adult height and risk of coronary heart disease (CHD) has been reported in many studies, but the association between adult height and stroke remains uncertain. The authors examined the relation between adult height and risk of stroke and CHD in a prospective study of 7,735 men drawn from general medical practices in 24 towns in England, Wales, and Scotland. The men were followed up for an average of 16.8 years (range, 15.5–18.0 years) between 1978 and 1995. During this period, there were 351 major stroke events (63 fatal, 288 nonfatal) and 1,093 major CHD events (465 fatal, 628 nonfatal). The mean height of the men was 173.3 cm. Total stroke risk was increased only in the men who fell into the lowest quintile of the height distribution (<167.7 cm), with little difference being seen between the other groups. When data were examined separately for fatal and nonfatal events, no relation was seen with nonfatal stroke. An apparent inverse association was seen with fatal stroke, even after adjustment for a wide range of confounding variables, but the number of deaths was small and the trend was not statistically significant ($p = 0.17$). By contrast, a significant inverse relation was seen between height and risk of major CHD events: Risk decreased progressively with increasing height, even after full adjustment (highest quintile vs. lowest: relative risk (RR) = 0.74, 95% confidence interval (CI) 0.59–0.91; test for trend: $p < 0.001$). A stronger inverse association was seen with nonfatal CHD events (RR = 0.64, 95% CI 0.49–0.84) than with fatal CHD events (RR = 0.82, 95% CI 0.60–1.11). This study confirms the finding of an inverse association between height and CHD. The inverse association seen for fatal stroke but not nonfatal stroke suggests that height may be related to specific subtypes of stroke. There are different patterns of association between height and stroke and height and CHD. If the apparent association between short stature and increased risk of fatal stroke is confirmed in other prospective studies, this would suggest that different mechanisms underlie the effects of height on stroke and CHD. *Am J Epidemiol* 1998;148:1069–76.

body height; cerebrovascular disorders; coronary disease; lung

There is considerable interest in the possibility that early life factors influence the risk of coronary heart disease (CHD) (1, 2). The findings of a strong relation between short adult stature (a marker of an adverse environment in the early years of life) and increased risk of CHD and cardiovascular disease incidence or mortality (3–11) are consistent with the hypothesis that adverse environmental factors in early life have an effect on risk of cardiovascular disease (1, 2). There is evidence from ecologic (12, 13) and cohort (14) studies that early life factors may play a role in the development of stroke. Early life factors have also been implicated in the development of adult blood pressure (15), a strong and consistent risk factor for stroke. It might therefore be expected that short stature could also influence risk of stroke. However, in those few prospective studies that have examined the association between height and stroke risk, the results have been inconsistent. In two of the studies, no association was observed between height and stroke in men (7) or in women (10). Conversely, in a recent prospective study conducted in Norway, a significant inverse association was seen between height and stroke in both men and women (16). Because of the limited amount of data relating height to risk of stroke and because of the interest in early life influences on cardiovascular disease, we have examined the relation between height and subsequent risk of stroke and CHD in 7,735 middle-aged British men followed for an average period of 16.8 years.

## MATERIALS AND METHODS

The British Regional Heart Study is a large prospective study of cardiovascular disease comprising 7,735 men aged 40–59 years. The men were selected from the age-sex registers of one group general practice in

each of 24 towns in England, Wales, and Scotland (response rate = 78 percent). The criteria for selecting the towns, the general practices, and the subjects, as well as the methods of data collection used, have been reported previously (17). The medical practice selected in each town had a social class distribution which was representative of men in that town, and the overall social class distribution of the cohort closely resembled that of middle-aged men throughout Great Britain.

In 1978–1980, research nurses administered to each man a standard questionnaire which included questions on smoking habits, alcohol intake, physical activity, and medical history. Several physical measurements were made, and blood samples (nonfasting) were taken throughout the day between 8:30 a.m. and 6:30 p.m. Details on the measurement of serum lipid concentrations have been provided elsewhere (18). The London School of Hygiene and Tropical Medicine sphygmomanometer was used to measure blood pressure twice in succession, with the subject seated and the arm supported on a cushion. The mean of the two readings was used in the analysis, and all blood pressure readings were adjusted for observer variation within each town (19). Forced expiratory volume in 1 second ($FEV_1$) was measured in the seated position with a Vitalograph spirometer (Vitalograph Medical Instrumentation, Buckingham, England). Height-adjusted $FEV_1$ ($FEV_1(h)$) was obtained by standardizing the values to the average height of the men (1.73 m) in the study.

Details on classification methods for smoking status, alcohol consumption, social class (longest-held occupation), and physical activity have been reported elsewhere (16, 20, 21). The men were classified according to their current cigarette smoking status into six groups: never smokers, former smokers, and four groups of current smokers (1–19, 20, 21–39, and ≥40 cigarettes/day). Heavy alcohol consumption was defined as drinking more than six UK units (1 UK unit = 8–10 g of alcohol) daily or on most days of the week. A physical activity score was derived for each man on the basis of frequency and type of leisure activity, and the men were grouped into six broad activity categories based on their total score: inactive, occasional, light, moderate, moderately vigorous, and vigorous (21). Men who were at least moderately active were defined as "active." Obesity was defined as body mass index (weight (kg)/height (m)$^2$) ≥28, the top quintile of the distribution.

## Preexisting disease

The men were asked whether a doctor had ever told them that they had angina or myocardial infarction

(heart attack, coronary thrombosis), stroke, diabetes mellitus, or any of a number of other disorders. They were also asked for details on any regular medical treatment, including treatment for hypertension. The World Health Organization (Rose) chest pain questionnaire (22) was administered to all men at the initial examination, and a three-orthogonal-lead resting electrocardiogram was taken.

*Previous stroke.* Evidence of a previous stroke was determined by the subject's recall of such a diagnosis' being made by a doctor ($n = 52$).

*Coronary heart disease.* Men with evidence of CHD were defined as those who had had a diagnosis of angina or heart attack made by a doctor, those with angina or possible myocardial infarction indicated on the World Health Organization (Rose) chest pain questionnaire, and those with electrocardiographic evidence of possible or definite myocardial ischemia or myocardial infarction ($n = 1,943$).

*Diabetes mellitus.* History of diabetes was based on recall of a doctor's diagnosis of diabetes ($n = 121$).

## Follow-up

All men were followed up for all-cause mortality and for cardiovascular morbidity, regardless of previous CHD or stroke events (23). All cardiovascular events occurring in the period up to December 1995 were included in the study. Follow-up averaged 16.8 years (range, 15.5–18.0 years), and complete follow-up was achieved for 99 percent of the cohort. Information on death was collected through the established "tagging" procedures provided by the National Health Service registers in Southport (England and Wales) and Edinburgh (Scotland). Fatal stroke episodes were those coded on the death certificate with *International Classification of Diseases*, Ninth Revision (24), codes 430–438. Fatal CHD events were defined as deaths due to CHD (*International Classification of Diseases*, Ninth Revision, codes 410–414). Nonfatal stroke events were those which produced a neurologic deficit that was present for more than 24 hours. Fatal events (stroke and CHD) included in this paper comprise only those deaths which occurred as the *first* event recorded during the course of follow-up, and not deaths which were preceded by a nonfatal event. Nonfatal myocardial infarction was diagnosed according to World Health Organization criteria, which included any report of myocardial infarction accompanied by at least two of the following: a history of severe chest pain, electrocardiographic evidence of myocardial infarction, and cardiac enzyme changes associated with myocardial infarction.

Evidence regarding CHD events and strokes was obtained from reports submitted by general practition-

ers, from biennial reviews of the patients' notes through the end of the study period, and from personal questionnaires administered to surviving subjects at the fifth and 12th years after initial examination. All death certificates on which it appeared that coding of a CHD event or stroke was not appropriate, or on which a CHD event or stroke was not the attributed code when it might have been, were reviewed by correspondence with the certifying doctor and the hospital concerned. No information on type of stroke was available.

## Statistical methods

The Cox proportional hazards model was used to assess the independent contribution of height to the risks of stroke and CHD and to obtain relative risks adjusted for age and the other risk factors (25). Age, systolic blood pressure, cholesterol, $FEV_1$, and body mass index were fitted as continuous variables. Smoking (six levels), physical activity (six levels), social class (seven levels), diabetes (yes/no), preexisting stroke (yes/no), antihypertensive treatment (yes/no), and preexisting CHD on the questionnaire/electrocardiogram (three levels) were fitted as categorical variables. Direct standardization was employed to obtain age-adjusted rates per 1,000 person-years of observation by 5-year age interval, using the study population as the standard.

## RESULTS

During the mean follow-up period of 16.8 years, there were 351 major stroke events and 1,093 major CHD events in the 7,733 men for whom we had data on height. The mean height of the men in the study was 173.3 cm (standard deviation 6.6; range, 144.6–199.6 cm). Age-adjusted mean height was significantly lower in CHD cases than in non-CHD cases (172.2 cm (standard deviation 0.2) vs. 173.4 cm (standard deviation 0.1); $p < 0.0001$), but there was little difference between stroke cases and nonstroke cases (172.9 cm (standard deviation 0.4) vs. 173.3 cm (standard deviation 0.1); $p = 0.32$).

## Cardiovascular disease risk factors

The men were divided into quintiles of the ranked height distribution. Table 1 shows the age-adjusted prevalence of and mean values for baseline cardiovascular disease risk factors in the five height groups. Shorter men were older and more likely to be manual workers. They had higher prevalences of smoking, obesity, heavy drinking, nondrinking, and preexisting CHD, and were less likely to be physically active. They had the highest mean total cholesterol and high density lipoprotein cholesterol levels and the lowest mean height-adjusted lung function. No association was seen with systolic or diastolic blood pressure or the prevalence of diabetes. The shorter men did not

**TABLE 1.** Age-adjusted prevalence of and mean values for cardiovascular risk factors at baseline, by quintile of the height distribution: British Regional Heart Study, 1978–1995

| | Quintile of height (cm) | | | | | |
|---|---|---|---|---|---|---|
| | <167.7 ($n$ = 1,533) | 167.7–171.5 ($n$ = 1,559) | 171.6–174.8 ($n$ = 1,555) | 174.9–178.8 ($n$ = 1,548) | ≥178.9 ($n$ = 1,538) | $p$ for trend |
| Mean age (years) | 51.4 | 50.8 | 50.1 | 50.0 | 49.0 | <0.0001 |
| Manual worker (%) | 73.0 | 63.5 | 58.0 | 50.6 | 41.7 | <0.0001 |
| Cigarette smoker (%) | 48.3 | 41.6 | 42.1 | 38.3 | 35.5 | <0.0001 |
| Obese* (%) | 20.9 | 19.9 | 19.3 | 19.4 | 17.1 | <0.01 |
| Physically active (%) | 31.2 | 34.0 | 37.7 | 38.6 | 42.0 | <0.0001 |
| Heavy alcohol drinker† (%) | 13.1 | 10.0 | 10.8 | 11.4 | 8.6 | <0.01 |
| Nondrinker (%) | 6.6 | 6.4 | 6.3 | 5.6 | 5.3 | <0.05 |
| Preexisting CHD‡ (%) | 28.6 | 24.9 | 26.8 | 22.7 | 22.5 | <0.0001 |
| Diabetes mellitus (%) | 1.3 | 1.4 | 1.4 | 1.6 | 2.0 | NS‡ |
| Antihypertensive treatment (%) | 5.3 | 5.4 | 5.0 | 3.8 | 4.8 | NS |
| Stroke (%) | 0.7 | 0.5 | 0.8 | 0.5 | 0.9 | NS |
| Mean systolic blood pressure (mmHg) | 145.1 | 145.6 | 145.7 | 144.8 | 144.7 | NS |
| Mean diastolic blood pressure (mmHg) | 81.7 | 82.2 | 82.4 | 82.4 | 82.6 | NS |
| Mean serum cholesterol (mmol/liter) | 6.35 | 6.34 | 6.30 | 6.29 | 6.21 | <0.0001 |
| Mean HDL‡ cholesterol (mmol/liter) | 1.17 | 1.15 | 1.14 | 1.14 | 1.13 | <0.0001 |
| Mean height-adjusted $FEV_1$‡ (liters) | 3.24 | 3.31 | 3.33 | 3.36 | 3.34 | <0.05 |
| Mean unadjusted $FEV_1$ (liters) | 2.91 | 3.18 | 3.32 | 3.49 | 3.72 | <0.0001 |

* Body mass index (weight (kg)/height (m)$^2$) ≥28.
† More than 6 UK units daily or on most days of the week.
‡ CHD, coronary heart disease; NS, not significant; HDL, high density lipoprotein; $FEV_1$, forced expiratory volume in 1 second.

TABLE 2. Age-adjusted rates of stroke per 1,000 person-years and adjusted relative risk of stroke, by quintile of the height distribution: British Regional Heart Study, 1978–1995

| Height (cm) | No. | Cases of stroke | Rate/1,000 person-years | Age-adjusted RR* | 95% CI* | Multivariate-adjusted RR† | 95% CI |
|---|---|---|---|---|---|---|---|
| <167.7 | 1,533 | 88 | 3.5 | 1.00 | | 1.00 | |
| 167.7–171.5 | 1,559 | 72 | 2.9 | 0.82 | 0.60–1.12 | 0.81 | 0.59–1.10 |
| 171.6–174.8 | 1,555 | 62 | 2.7 | 0.75 | 0.54–1.04 | 0.73 | 0.53–1.01 |
| 174.9–178.8 | 1,548 | 68 | 2.9 | 0.82 | 0.59–1.12 | 0.94 | 0.68–1.30 |
| ≥178.9 | 1,538 | 61 | 2.9 | 0.81 | 0.58–1.13 | 0.87 | 0.62–1.22 |
| p for trend | | | | NS* | | NS | |

\* RR, relative risk; CI, confidence interval; NS, not significant.
† Adjusted for age, social class, smoking, preexisting coronary heart disease, stroke, diabetes mellitus, physical activity, alcohol intake, body mass index, cholesterol, antihypertensive treatment, and systolic blood pressure.

have an increased prevalence of recall of stroke or diabetes.

*Height and stroke.* Table 2 shows the age-adjusted rates of stroke per 1,000 person-years and the age-adjusted relative risks of stroke using the lowest quintile of the men as the reference group. Stroke risk was highest among men who fell into the lowest quintile of the height distribution, but there was little difference between the other groups. The men in the lowest quintile were further divided into two deciles. There was little difference between rates in the first and second deciles (age-adjusted rates: 3.6 vs. 3.4/1,000 person-years). The result of a trend test was not significant ($p = 0.21$), and a test for a difference between men in the lowest quintile of the height distribution (<167.7 cm) and the rest of the men (≥167.7 cm)

showed marginal significance (taller men vs. the lowest quintile: age-adjusted relative risk = 0.80, 95 percent confidence interval (CI) 0.63–1.01; $p = 0.07$). Further adjustment for potential confounders for stroke—namely, age, social class, smoking, preexisting CHD, preexisting diabetes, preexisting stroke, physical activity, alcohol intake, body mass index, antihypertensive treatment, and systolic blood pressure—made only a marginal difference in the level of risk (relative risk = 0.83, 95 percent CI 0.65–1.06; $p = 0.13$).

The height-stroke relation was also examined by age group ($n = 99$ cases aged 40–49 years, 92 cases aged 50–54 years, and 160 cases aged 55–59 years). Short men (<167.7 cm) had a higher risk of stroke than taller men in the two younger age groups. No differ-

TABLE 3. Adjusted relative risk of fatal stroke and nonfatal stroke, by quintile of the height distribution: British Regional Heart Study, 1978–1995

| Height (cm) | No. of cases | Age-adjusted RR* | 95% CI* | Multivariate-adjusted RR† | 95% CI |
|---|---|---|---|---|---|
| | | *Fatal stroke (n = 63)* | | | |
| <167.7 | 19 | 1.00 | | 1.00 | |
| 167.7–171.5 | 15 | 0.81 | 0.41–1.59 | 0.77 | 0.39–1.52 |
| 171.6–174.8 | 12 | 0.71 | 0.34–1.45 | 0.69 | 0.33–1.43 |
| 174.9–178.8 | 10 | 0.59 | 0.27–1.26 | 0.69 | 0.32–1.51 |
| ≥178.9 | 7 | 0.48 | 0.20–1.14 | 0.54 | 0.22–1.31 |
| p for trend | | 0.06 | | 0.17 | |
| | | *Nonfatal stroke (n = 288)* | | | |
| <167.7 | 69 | 1.00 | | 1.00 | |
| 167.7–171.5 | 57 | 0.82 | 0.57–1.17 | 0.82 | 0.57–1.16 |
| 171.6–174.8 | 50 | 0.76 | 0.53–1.10 | 0.75 | 0.52–1.07 |
| 174.9–178.8 | 58 | 0.88 | 0.62–1.25 | 1.00 | 0.70–1.43 |
| ≥178.9 | 54 | 0.90 | 0.63–1.29 | 0.95 | 0.66–1.38 |
| p for trend | | 0.61 | | 0.91 | |

\* RR, relative risk; CI, confidence interval.
† Adjusted for age, social class, smoking, preexisting coronary heart disease, stroke, diabetes mellitus, physical activity, alcohol intake, body mass index, cholesterol, antihypertensive treatment, and systolic blood pressure.

TABLE 4. Age-adjusted rates of major coronary heart disease (CHD) events per 1,000 person-years and adjusted relative risk of CHD, by quintile of the height distribution: British Regional Heart Study, 1978–1995

| Height (cm) | No. of CHD cases | Rate/1,000 person-years | Age-adjusted RR* | 95% CI* | Multivariate-adjusted RR† | 95% CI | Additionally adjusted RR‡ | 95% CI |
|---|---|---|---|---|---|---|---|---|
| <167.7 | 278 | 11.7 | 1.00 | | 1.00 | | 1.00 | |
| 167.7–171.5 | 254 | 10.7 | 0.91 | 0.76–1.07 | 0.93 | 0.77–1.10 | 0.97 | 0.81–1.16 |
| 171.6–174.8 | 222 | 9.9 | 0.83 | 0.02–0.99 | 0.86 | 0.72–1.04 | 0.92 | 0.76–1.11 |
| 174.9–178.8 | 183 | 7.9 | 0.67 | 0.56–0.81 | 0.77 | 0.64–0.94 | 0.81 | 0.66–1.00 |
| ≥178.9 | 156 | 7.3 | 0.62 | 0.51–0.75 | 0.74 | 0.59–0.91 | 0.82 | 0.66–1.02 |
| p for trend | | | <0.0001 | | <0.001 | | 0.04 | |

* RR, relative risk; CI, confidence interval.
† Adjusted for age, social class, smoking, preexisting coronary heart disease, stroke, diabetes mellitus, physical activity, alcohol intake, body mass index, cholesterol, antihypertensive treatment, and systolic blood pressure.
‡ Adjusted for all of the above factors and for unadjusted forced expiratory volume in 1 second.

ence was seen in the oldest age group. The adjusted relative risks for the three age groups were 0.79 (95 percent CI 0.48–1.30), 0.66 (95 percent CI 0.41–1.05), and 0.96 (95 percent CI 0.66–1.37), respectively.

*Fatal and nonfatal stroke.* We examined the height-stroke relation for fatal and nonfatal strokes separately (table 3). There were 63 fatal strokes and 288 nonfatal strokes. An inverse relation was seen with fatal stroke, and the trend was of marginal significance ($p = 0.06$). Little association was seen with nonfatal stroke. The inverse trend with fatal stroke remained even after adjustment for potentially confounding factors, although the trend was not significant ($p = 0.17$). Exclusion of men with diagnosed CHD or stroke made little difference in the relations seen.

*Height and CHD.* In contrast to the relation between height and stroke, a significant inverse association was seen between height and risk of CHD, with age-adjusted relative risk decreasing progressively with increasing height ($p < 0.0001$) (table 4). The inverse association between height and CHD persisted, with slight attenuation, even after adjustment for a wide range of potentially confounding factors, including age, smoking, physical activity, social class, body mass index, preexisting CHD, diabetes, antihypertensive treatment, systolic blood pressure, and cholesterol level ($p < 0.001$). Further adjustment for height-adjusted $FEV_1$ made little difference in the relation seen. However, further adjustment using unadjusted $FEV_1$ reduced the difference in risk in the taller men, although the inverse trend remained significant ($p = 0.04$).

*Fatal and nonfatal CHD.* We also examined the height-CHD relation for fatal ($n = 465$) and nonfatal ($n = 628$) CHD events separately, adjusting for the factors listed in table 4 (excluding $FEV_1$). The relation was stronger with nonfatal myocardial infarction than with fatal CHD events. The adjusted relative risks for nonfatal myocardial infarction were 1.00 (referent),

0.90 (95 percent CI 0.71–1.13), 0.89 (95 percent CI 0.71–1.13), 0.72 (95 percent CI 0.56–0.93), and 0.64 (95 percent CI 0.49–0.84), respectively, for the five height groups (test for trend: $p = 0.002$), and the corresponding relative risks for fatal CHD events were 1.00 (referent), 0.96 (95 percent CI 0.74–1.25), 0.76 (95 percent CI 0.57–1.01), 0.84 (95 percent CI 0.63–1.12), and 0.82 (95 percent CI 0.60–1.11) (test for trend: $p = 0.03$). Further adjustment for unadjusted $FEV_1$ attenuated the relation with fatal events (test for trend: $p = 0.35$) but made little difference in the relation with nonfatal events (relative risks = 1.00, 0.92, 0.92, 0.73, and 0.66) (test for trend: $p = 0.007$).

## DISCUSSION

In this study of middle-aged British men, there was a strong inverse relation between height and risk of CHD. Shorter men tended to have an adverse coronary risk profile. They were older and more likely to be manual workers. They had higher rates of smoking, obesity, and heavy drinking and were less likely to be physically active, and they had a higher prevalence of preexisting CHD and a higher mean total cholesterol level. However, the increase in risk of CHD with decreasing height persisted even after adjustment for a wide range of cardiovascular disease risk factors, including height-adjusted $FEV_1$. In contrast, total stroke risk was only increased among men in the lowest quintile of the height distribution. When data were examined separately with regard to fatal and nonfatal stroke, an inverse association was seen for fatal stroke but no association was seen with nonfatal stroke.

The inverse relation seen between height and CHD is consistent with results from other prospective studies (3, 11). However, the extent to which the height-CHD relation is accounted for by the influence of lung function, a strong independent determinant of CHD risk, has been the subject of controversy.

## Height, lung function, and CHD

In earlier research from the British Regional Heart Study based on an average of 7.5 years of follow-up, the association between height and risk of major CHD events (fatal and nonfatal) was attenuated after adjustment for $FEV_1$ (not standardized for height), and the investigators concluded that the association was due to short men's having poorer lung function (6). Since the publication of that article, questions have been raised regarding adjustment for unadjusted $FEV_1$ which is correlated with height (10, 26). In the present study, we examined the effects of adjustment using both height-adjusted and unadjusted $FEV_1$. Height-adjusted $FEV_1$ made little difference in the relation seen between height and risk of major CHD events. In this longer follow-up period (mean = 16.8 years), there was a residual effect of height even when unadjusted $FEV_1$ was used. The residual effect may have been due in part to imprecision of measurement over time, as $FEV_1$ was measured at only one point in time and $FEV_1$ varies more over time than does height.

We observed that height showed a stronger relation with nonfatal events than with fatal events. The relation between height and CHD death appears to be associated with lung size per se, as unadjusted $FEV_1$ appeared to explain the relation with fatal events. However, the relation with nonfatal events was independent of $FEV_1$, which suggests that there are other factors operating. The stronger association between height and nonfatal events has also been observed in women (10).

Several other studies have examined the role of lung function in the height-CHD relation, and the results have been inconsistent. The Whitehall Study found a weak relation between height and CHD mortality when height was matched for unadjusted $FEV_1$ (27). By contrast, the Caerphilly and Speedwell heart studies (28) observed a significant inverse relation between height and 5-year risk of CHD after adjustment for $FEV_1$ (not standardized for height). In the East Boston Study (Boston, Massachusetts), no association was seen between height and cardiovascular disease death in elderly men (8). In elderly women, an inverse relation between height and cardiovascular disease death that was not explained by peak expiratory flow rate (height-adjusted or unadjusted) was seen, suggesting that lung function was not implicated. The reasons for the discrepancies between studies are not apparent, and they may relate in part to the differing associations between height, lung function, and fatal and nonfatal events. However, our data suggest that lung function is not the only mechanism underlying the height-CHD relation.

## Height and stroke

Several prospective studies have failed to find any association between height and stroke in men or women, in contrast to the case with CHD (7, 10). However, a recent study in Finnmark, Norway, observed a significant inverse relation between height and total stroke in men and women aged 35–52 years (16). Risk of stroke decreased with increasing height in both men and women. In the present study, we observed a higher risk of total stroke only among men in the lowest quintile of the height distribution, and this was seen only in the younger age groups (40–54 years)—a group similar to that studied in Norway, where an inverse association was found. When the relation was examined for fatal and nonfatal stroke separately, an inverse association was seen with fatal stroke, even after adjustment, although the trend was not statistically significant, possibly because of the small number of fatal stroke events in the study. No associations were seen with nonfatal stroke. In another Norwegian study of Oslo men aged 40–49 years, an inverse but nonsignificant association was also observed with stroke mortality (29).

These findings suggest that height may be associated with those subtypes of stroke most likely to result in a fatal outcome and which account for a particularly high proportion of strokes in younger men (cerebral hemorrhage and subarachnoid hemorrhage (30)). In the Finnmark Study, the strongest association was seen for intracerebral hemorrhage and nonspecified types of stroke. A smaller association was seen with cerebral infarction, and the trend was of marginal significance, but no association was seen for subarachnoid hemorrhage, as might have been anticipated. Although we did not have information on types of stroke, which is a limitation of this study, 85 percent of stroke cases in Great Britain are apparently due to ischemic stroke (30). If height is related only to specific subtypes of stroke—particularly intracerebral hemorrhage, which accounts for a very small proportion of all stroke events but a high proportion of fatal events—this may explain the lack of a relation between height and total stroke incidence.

## Implications for the origins of cardiovascular disease risk

There is now strong evidence that factors operating in the early years of life influence adult risk of CHD (1). The relation between short stature and CHD risk is consistent with the importance of early life factors. However, the relation between adult height and CHD risk may be distinct from the relation reported elsewhere between small size at birth and CHD risk (1, 2).

Although birth weight is related to adult height, the relation is weak, with most of the variance in adult height being accounted for by other factors (31). Moreover, markers of growth in childhood (such as leg length) are themselves related to increased subsequent CHD risk (32). It is not yet clear whether lung function, which is influenced not only by adult height but also by birth weight (33), is a mediator of these relations. Our data suggest that lung function, though it may be important, is not the only factor involved.

The evidence that early life factors influence stroke risk is less convincing. Although ecologic studies have suggested that early life factors might influence stroke risk (12, 13), evidence from cohort studies linking early influences to subsequent stroke risk is weak thus far. In combined follow-up studies of men in Hertfordshire and Sheffield, England, rates of death from stroke tended to fall with rising birth weight, although no adjustments were made for adult risk factors (14). The inconsistent relations between height and stroke seen in earlier studies, the association with only fatal strokes in this study, and the absence of any association between father's social class and stroke (34) all suggest that the effect of early life factors on stroke risk is limited. This is clearly a paradox, because blood pressure is among the factors most consistently related to early life factors in both children and adults (35).

## Conclusion

This study confirms the finding of an inverse relation between height and CHD and indicates a possible relation between short stature and risk of fatal stroke. The inverse relation seen for fatal stroke but not nonfatal stroke suggests that height may only be related to those subtypes of stroke most likely to result in a fatal outcome. The difference in pattern of association between height and CHD and height and stroke suggests that there are different mechanisms underlying the effects of height on stroke and CHD.

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