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ORIGINAL CONTRIBUTIONS

Height in Young Adulthood and Risk of Death from Cardiorespiratory Disease: A Prospective Study of Male Former Students of Glasgow University, Scotland

Peter McCarron,[1] Mona Okasha,[2] James McEwen,[3] and George Davey Smith[2]

To investigate the association between height in young, socially homogeneous males and cause-specific mortality, the authors conducted a prospective study of 8,361 male former students who underwent medical examinations while attending Glasgow University, Scotland, from 1948 to 1968. The mean age at examination was 20.5 (range, 16.1–30.0) years. The median follow-up time was 41.3 years. There were 863 deaths. In Cox proportional hazards modeling, there was no association between height and all-cause mortality with age-adjusted hazard ratios per 10-cm increase in height (hazard ratio = 0.92, 95% confidence interval: 0.83, 1.02). Height was inversely associated with all cardiovascular disease and coronary heart disease mortality, with hazard ratios per 10-cm increase in height of 0.78 (95% confidence interval: 0.66, 0.93) and 0.76 (95% confidence interval: 0.62, 0.93), respectively. Sizeable inverse associations with stroke and respiratory disease were also found, although these did not reach conventional levels of significance. There was no association with cancer or noncardiorespiratory disease mortality. There was a positive, although nonsignificant, association between height and mortality from aortic aneurysm. Controlling for confounding variables had little effect on these results. The findings suggest that factors operating in early life, and which influence height, also influence future cardiovascular health in men. *Am J Epidemiol* 2002;155:683–7.

cardiovascular diseases; cohort studies; coronary disease; mortality; neoplasms

Editor's note: *An invited commentary on this article appears on page 688, and the authors' response appears on page 690.*

Several studies have examined the association between adult height and cardiovascular disease, with many (1–10), but not all (11), reporting inverse associations. Height-cancer associations are less consistent (2, 4, 12). Potential explanations include the following. 1) Adult height reflects growth, nutrition, infections, and socioeconomic circumstances in early life, and inverse associations between height and cardiovascular disease mortality are consistent with the positive associations between such early life factors and cardiovascular disease risk (13–17). Conversely, the positive associations between height and cancer suggest that high childhood calorie intake increases cancer risk (18, 19). 2) Height is positively correlated with socioeconomic position in adulthood, suggesting that socially patterned exposures in adulthood, which include health behaviors, and psychosocial and environmental factors, along with their physiologic consequences, could account for the associations between height and cardiovascular disease. 3) The early stages of disease could lead to reductions in height, thus generating the inverse association with cardiovascular disease (4).

It is important to establish which of these hypotheses is most plausible. Using height data collected in a young, relatively socially homogeneous population, we can exclude or at least cast doubt on the second and third of these hypotheses. Any associations uncovered would therefore point more convincingly to the importance of early life factors in determining both height and later disease risk.

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[1] Surveillance Research Program, Division of Cancer Control and Population Sciences, National Cancer Institute, Bethesda, MD.
[2] Department of Social Medicine, University of Bristol, Bristol, United Kingdom.
[3] Department of Public Health, University of Glasgow, Glasgow, United Kingdom.
Correspondence to Dr. Peter McCarron, N. Ireland Cancer Registry, Department of Epidemiology and Public Health, The Queen's University of Belfast, Mulhouse Building, Grosvenor Road, Belfast BT12 6BJ, United Kingdom (e-mail: mccarrop@mail.nih.gov).

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TABLE 1. Distribution of risk factors* according to height in 8,361 males attending Glasgow University, Scotland, 1948–1968

	No. of males	Age at screening (years)	Systolic blood pressure (mmHg)	Diastolic blood pressure (mmHg)	Body mass index (kg/m²)	Nonsmokers (%)	Social classes I and II (%)
Height (cm)							
≤170	2,076	20.5	130.1	77.0	21.8	68.4	51.7
171–173	1,359	20.6	130.9	77.5	21.6	66.9	54.6
174–176	1,689	20.6	130.9	77.2	21.5	65.5	55.0
177–180	1,805	20.4	131.4	77.3	21.4	66.0	57.7
>180	1,432	20.5	131.9	77.4	21.5	65.2	62.0
Correlation coefficient		0.004	0.04	0.02	−0.08	−0.03†	0.07†
p value‡		0.75	<0.0001	0.097	<0.0001	0.039	<0.0001

* Values are means unless otherwise stated.
† For correlation between height and being a nonsmoker or being in social class I or II.
‡ Using height as a continuous variable.

MATERIALS AND METHODS

Full details of the study methods are presented elsewhere (20). Briefly, students attending Glasgow University from 1948 to 1968 were invited to participate in annual medical examinations. The data collected included height, weight, blood pressure, sociodemographic data, and details of health behaviors. Childhood socioeconomic

TABLE 2. Hazard ratios for cause of death by height by 8,361 in males attending Glasgow University, Scotland, 1948–1968

	No. of deaths	Age adjusted		Fully adjusted*	
		Hazard ratio	95% confidence interval	Hazard ratio	95% confidence interval
		All causes			
Mean height (m)					
1.668	224	1		1	
1.721	139	0.91	0.73, 1.12	0.91	0.74, 1.13
1.749	198	1.00	0.83, 1.22	1.02	0.84, 1.24
1.786	164	0.85	0.69, 1.04	0.84	0.68, 1.02
1.840	138	0.88	0.71, 1.09	0.89	0.72, 1.11
Hazard ratio per 10-cm increase in height		0.92	0.83, 1.02	0.93	0.83, 1.03
p for trend†		0.14		0.16	
		Cardiovascular disease‡			
Mean height (m)					
1.668	95	1		1	
1.721	63	0.96	0.70, 1.33	0.98	0.71, 1.34
1.749	75	0.89	0.66, 1.21	0.93	0.68, 1.26
1.786	62	0.75	0.55, 1.04	0.76	0.55, 1.05
1.840	44	0.66	0.46, 0.95	0.69	0.48, 0.99
Hazard ratio per 10-cm increase in height		0.77	0.65, 0.91	0.78	0.66, 0.93
p for trend†		0.002		0.005	
		Coronary heart disease§			
Mean height (m)					
1.668	67	1		1	
1.721	51	1.11	0.77, 1.60	1.13	0.78, 1.62
1.749	53	0.90	0.63, 1.29	0.94	0.65, 1.34
1.786	42	0.72	0.49, 1.06	0.72	0.49, 1.06
1.840	29	0.62	0.40, 0.95	0.64	0.41, 0.99
Hazard ratio per 10-cm increase in height		0.75	0.62, 0.91	0.76	0.62, 0.93
p for trend†		0.004		0.008	

Table continues

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position was assigned by coding the father's occupation into social class, a five-point scale from I (most affluent) to V (least affluent), using the Registrar General's classification (21, 22). Participants were traced through the National Health Service Central Register of the United Kingdom. Because there were few female deaths, analyses are confined to males and include deaths up to December 31, 1998. Cox proportional hazards models were used to estimate the association between height measured at baseline and death from all causes, cardiovascular disease, coronary heart disease, stroke, respiratory disease, and all causes other than cardiovascular or respiratory disease (noncardiorespiratory disease). The association between height and mortality from ruptured aortic aneurysm was also investigated. In multivariable analyses we adjusted for smoking (yes, no), systolic blood pressure (mmHg), body mass index (kg/m^2), father's social class (categorical variables I–V), and quintile of year of birth to control for any cohort effect. Analyses were performed on Stata 6.0 software (23).

RESULTS

A total of 11,755 male students (almost 50 percent of the male student population from 1948 to 1968) who stated their nationality to be British and gave a home address in the United Kingdom participated in the health surveys. Of these, 9,887 (84.1 percent) have been traced. The median follow-up was 41.3 years. The mean age at the time of examination was 20.5 (range, 16.1–30.0) years, and the mean height was 174.8 (standard deviation, 6.33) cm. Because age-adjusted results were similar for the full cohort

TABLE 2. Continued

	No. of deaths	Age adjusted		Fully adjusted	
		Hazard ratio	95% confidence interval	Hazard ratio	95% confidence interval
		Stroke¶			
Mean height (m)					
1.668	21	1		1	
1.721	8	0.55	0.24, 1.24	0.56	0.25, 1.26
1.749	14	0.75	0.38, 1.47	0.78	0.40, 1.54
1.786	13	0.73	0.36, 1.45	0.76	0.38, 1.53
1.840	11	0.77	0.37, 1.59	0.81	0.39, 1.68
Hazard ratio per 10-cm increase in height		0.76	0.52, 1.11	0.79	0.54, 1.15
p for trend†		0.16		0.22	
		Respiratory disease#			
Mean height (m)					
1.668	17	1		1	
1.721	8	0.69	0.30, 1.60	0.63	0.27, 1.48
1.749	4	0.26	0.08, 0.76	0.24	0.08, 0.73
1.786	10	0.70	0.32, 1.55	0.63	0.29, 1.38
1.840	6	0.50	0.20, 1.31	0.49	0.19, 1.24
Hazard ratio per 10-cm increase in height		0.68	0.43, 1.07	0.64	0.40, 1.03
p for trend†		0.095		0.068	
		Noncardiorespiratory causes			
Mean height (m)					
1.668	112	1		1	
1.721	68	0.89	0.66, 1.20	0.89	0.66, 1.21
1.749	119	1.22	0.94, 1.58	1.23	0.95, 1.59
1.786	92	0.95	0.72, 1.25	0.93	0.71, 1.23
1.840	88	1.12	0.85, 1.48	1.12	0.85, 1.49
Hazard ratio per 10-cm increase in height		1.08	0.94, 1.25	1.08	0.94, 1.25
p for trend†		0.26		0.27	

* Adjusted for systolic blood pressure, body mass index, smoking, father's social class, and year of birth quintile.
† Trend for height as a continuous variable.
‡ *International Classification of Diseases*, Ninth Revision (ICD-9), codes 390–459.
§ ICD-9 codes 410–414.
¶ ICD-9 codes 430–438.
# ICD-9 codes 460–519.

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and for the 8,361 persons with data on confounding variables, we report only the latter here.

The characteristics of the population according to height are presented in table 1. There was no association between height and age at screening. Taller students had slightly higher systolic blood pressure, similar diastolic blood pressure, and slightly lower body mass index compared with shorter men. They were less likely to be nonsmokers and more likely to come from social classes I and II than were shorter students.

There were 863 deaths. Hazard ratios by height quintile and per 10-cm increase in height are shown in table 2. There were strong inverse associations between height and mortality from all cardiovascular disease and coronary heart disease, equivalent to respective mortality reductions of 23 percent and 25 percent per 10-cm increase in height. For stroke and respiratory disease mortality, there were also substantial inverse associations, although these failed to reach conventional levels of statistical significance. Noncardiorespiratory disease mortality showed no association with height. Controlling for potential confounding variables made little difference in these results.

A positive association between height and mortality from ruptured aortic aneurysm was found for the eight men who died from this cause, although this was not significant at conventional levels: the hazard ratio per 10-cm increase in height = 1.63 (95 percent confidence interval: 0.55, 4.84). Controlling for confounding slightly strengthened this association to 1.85 (95 percent confidence interval: 0.58, 6.10).

DISCUSSION

In this study, height measured in young adulthood was inversely associated with later cardiorespiratory disease mortality in males, with evidence of a positive association between height and mortality from ruptured aortic aneurysm. These findings are similar in magnitude to those from studies in which height was measured in middle age (2, 24).

The current study is large and has a long follow-up, and data on several important confounders are available. Participating students were likely to be representative of all male students, and traced students were representative of those who originally participated (20). Although participants were, on average, from more affluent social circumstances than was the general population, the mechanisms responsible for the findings are likely to be generalizable.

Height was measured when cohort members were young and likely to have been free of chronic disease. Although we do not have details on how height was measured, correlations between heights ranged from 0.92 to 0.97 ($p < 0.001$) for those who had 2–4 repeat yearly measurements. Perfect correlation would not be expected because some students had not reached their final height at the time of examination. Errors in measurement are unlikely to have biased the results and would, if anything, attenuate the risks reported here. The generalizabilty of the findings may be limited by the absence of non-White students and because we can report only robust height-mortality associations in men.

Several possible explanations for the results must be considered. It is unlikely that confounding by socially patterned exposures in adulthood could explain the inverse association between height and cardiorespiratory disease mortality, because noncardiorespiratory disease deaths would also have been influenced by the same potential unmeasured confounders. The lack of association between height and smoking-related cancers (25), which are known to be strongly socially patterned, provides further evidence that the effects are not simply confounded by adult socioeconomic position and its attendant exposures. Finally, with less than 5 percent of school leavers at university in Scotland over the period of this study (26) and over 75 percent of graduates from the United Kingdom in social classes I and II in the 1970s (27), differential social patterning of adulthood exposures is itself unlikely, further limiting the possibility of confounding due to socially patterned exposures.

Data on serum lipids were not available, but controlling for lipid levels has been shown to have little effect on the associations between height and cardiovascular disease (28–30). Taller students had higher systolic blood pressure than did shorter students. Blood pressure measured in early adulthood in this cohort is positively associated with later cardiovascular disease mortality (31); therefore, confounding by blood pressure would produce associations with cardiovascular disease mortality in the opposite direction to those observed.

Because height was measured in early adulthood, we can rule out inverse associations due to shrinkage secondary to adult disease, and the need to adjust for the effect of cumulative loss of height with aging (32) does not arise.

Data on fetal growth were not available, precluding investigation of the association among fetal development, adult height, and subsequent disease. However, two other studies have shown that adjustment for birth weight does not significantly modify the height-mortality relation (11, 33). Adult height is, in part, a reflection of growth, nutrition, and exposure to infectious diseases in childhood and, thus, of childhood socioeconomic circumstances. The childhood social circumstances of the current cohort were relatively heterogeneous: 55.9 percent came from social classes I and II, 36.7 percent were from social class III, and 7.4 percent were from the lowest two social classes. Early life social circumstances are inversely associated with cardiovascular disease mortality in this cohort (34), and these associations persist after controlling for adult socioeconomic position (15–17). The associations between fetal development, growth, and exposure to infections in childhood and childhood socioeconomic position may therefore contribute to our findings. It is not surprising that adjustment for father's social class has a limited effect on the height-mortality associations, because father's social class is a crude indicator of the socially patterned factors in childhood that may influence adult health, particularly among this group with a small proportion from lower manual class backgrounds.

The observed association between height and mortality from aortic aneurysm is in agreement with findings from other studies (24, 35, 36). Unlike the incidence of most cardiovascular disease, the incidence of aortic aneurysm has been increasing (37), suggesting that there may be important differences in the pathophysiology of these cardiovascular conditions (37, 38).

In summary, our findings suggest that factors related to growth and development in early life influence achieved adult

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height and also cardiorespiratory disease risk. An understanding of the mechanisms underlying these associations could usefully inform primary prevention strategies.

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