mine whether growth hormones are as important in the aetiology of human cancer as they are in animal experiments.

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Contributors: The Boyd Orr cohort was established by GDS and SF. DJG further developed and refined the cohort. GDS and DJG initiated this reanalysis, and the analyses presented here were performed by DJG. JMPH provided input on the possible role of growth factors in the observed associations. DJG wrote the first draft of the paper and GDS, SF, and JMPH all commented and helped in redrafting the text. DJG, GDS, and SF are guarantors for the study.

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Height and mortality from cancer among men: prospective observational study

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Experiments in animals have shown that energy intake during the growth period is positively associated with the later incidence of cancer.[1] Recently, direct evidence of an association between childhood energy intake and adult mortality from cancer among humans was published in a report from the Boyd Orr cohort study.[2] Childhood energy intake was positively associated with mortality from cancers not related to smoking, whereas there was no association between energy intake and mortality from cancers related to smoking.[2] This is to be expected as the substantial effects of tobacco would mask any effects of childhood diet on cancers related to smoking.

Height has been used in previous studies as a marker for energy intake in childhood,[3] with the limited evidence indicating a positive association for some cancer sites.[3 4 5] In the Boyd Orr study data were not available on smoking behaviour and were limited on adulthood socioeconomic position.[2] We therefore analysed the association between height and mortality from cancer in a large cohort of men for whom detailed data on socioeconomic position in adulthood and on smoking behaviour were available.

Subjects, methods, and results

In the Whitehall study of London civil servants, data on employment grade, height, and smoking behaviour were available for 17 378 men aged 40-64 who were examined between 1967 and 1969.[4] During follow up until 31 January 1995, 2226 of these men died of cancer: 725 from cancers unrelated to smoking and 1501 from cancers related to smoking. To adjust for the potential confounding effects of other variables proportional hazards analyses were carried out with height as a continuous variable and age (in age bands of five years), employment grade (administrative, professional and executive, clerical, other), and smoking behaviour (cigarette smoker, pipe or cigar smoker, ex-cigarette smoker, and number of cigarettes smoked per day for current cigarette smokers) as covariates.

The table shows relative death rates from cancer by height category and for each increment of 6 inches in height (1 inch is about 2.5 cm). For cancers unrelated to smoking the association with height was positive and strengthened by adjustment for socioeconomic position (indexed by employment grade) and smoking behaviour. For cancers related to smoking the association between height and mortality was negative but not significant. Adjustment for socioeconomic position and smoking behaviour reversed the direction of the association, but it remained small and non-significant. As expected, cigarette smoking was strongly associated with cancers classified as smoking related but not with cancers classified as unrelated to smoking. The age adjusted relative rates for height and the smoking unrelated and smoking related cancers were significantly different ($P = 0.002$). Exclusion of mortality occurring during the first five or first ten years of follow up did not materially alter these findings.

Comment

Our findings parallel those relating childhood energy intake to cancer mortality in the Boyd Orr cohort

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Association between height and mortality from cancer in Whitehall study

Height (inches)*	No of men	No of deaths	Rate†	Rate ratio (95% CI)		
				Adjusted for age	Adjusted for age and employment grade	Adjusted for age, employment grade, and smoking
Cancers related to smoking‡						
–66	2268	218	4.38	1.0	1.0	1.0
–69	6619	600	4.34	0.96 (0.82 to 1.13)	1.05 (0.90 to 1.23)	1.05 (0.89 to 1.22)
–72	6375	523	4.14	0.92 (0.79 to 1.08)	1.04 (0.89 to 1.23)	1.05 (0.89 to 1.23)
>72	2116	160	4.01	0.88 (0.72 to 1.08)	1.02 (0.83 to 1.25)	1.06 (0.86 to 1.30)
Height increment (6 inches)				0.93 (0.83 to 1.04)	1.02 (0.91 to 1.15)	1.04 (0.93 to 1.17)
P value for trend				0.20	0.70	0.50
Cancers unrelated to smoking¶						
–66	2268	85	1.74	1.0	1.0	1.0
–69	6619	258	1.85	1.02 (0.80 to 1.30)	1.04 (0.81 to 1.33)	1.04 (0.81 to 1.33)
–72	6375	283	2.19	1.20 (0.94 to 1.54)	1.24 (0.97 to 1.59)	1.25 (0.97 to 1.59)
>72	2116	99	2.44	1.29 (0.96 to 1.72)	1.34 (1.00 to 1.80)	1.36 (1.01 to 1.82)
Height increment (6 inches)				1.28 (1.08 to 1.51)	1.32 (1.11 to 1.56)	1.33 (1.12 to 1.57)
P value for trend				0.0042	0.0016	0.0011

*1 inch is about 2.5 cm.
†Age standardised rates per 1000 person years.
‡Lip (international classification of diseases, ninth revision (ICD-9) code 140); tongue (141); mouth and pharynx (143-9); oesophagus (150); pancreas (157); respiratory tract (160-163); and urinary tract (188-189).
¶ICD-9 codes 140-208, excluding cancers related to smoking above.

study, in which the positive association between childhood energy intake and subsequent risk of cancer was also confined to cancers unrelated to smoking.[2] Most previous studies have either grouped all cancers together or looked only at individual cancers. However, consistent with our results, the physicians health study found a positive association of height with all malignant neoplasms but not with lung cancer.[5] In line with extensive animal experimental evidence,[1] therefore, our data and those from the Boyd Orr study[2] suggest that energy intake during growth may be an important determinant of later risk of developing cancer. Since height serves as only an indirect and comparatively weak proxy measure of dietary intake in childhood, the size of the association found in this study may reflect a much stronger underlying association with directly measured childhood energy intake.

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Initiating angiotensin converting enzyme inhibitors in mild to moderate heart failure in general practice: randomised, placebo controlled trial

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Less than 50% of patients with heart failure in community practice receive an angiotensin converting enzyme inhibitor and then only usually at the instigation of or when prescribed by a hospital doctor.[1–3] Fear of side effects seems to be a barrier to starting treatment with angiotensin converting enzyme inhibitors,[3 4] reflecting the lack of substantial studies to show the safety of giving them in primary care.

Methods and results

General practitioners in 47 practices in the United Kingdom recruited patients with mild to moderate heart failure who had been receiving chronic diuretic treatment. Exclusion criteria were age >80 years, frusemide dose >100 mg/day, systolic arterial pressure <100 mm Hg, serum creatinine concentration >250 μmol/l, and sodium concentration <135 mmol/l.