

Systolic and diastolic arterial blood pressure by standard occupational group at baseline and four years' follow up of healthy, untreated elderly people (mean age at baseline 74.4 years, mean education 11.2 years) compared with those with incident disease (mean age at baseline 75.8 years, mean education 10.9 years) or hypertension only (mean age at baseline 75.6 years, mean education 10.6 years). Values are mean (SD) unless stated otherwise

Group	No of patients	Baseline blood pressure (mm Hg)		Follow up blood pressure (mm Hg)	
		Systolic	Diastolic	Systolic	Diastolic
Unclassified	8	171 (21)	92 (4)	164 (19)	92 (10)
1	34	155 (19)	83 (10)	162 (21)	89 (10)
2	31	152 (26)	82 (11)	157 (24)	83 (9)
3	14	139 (13)	82 (9)	150 (17)	86 (11)
4	40	148 (20)	84 (10)	160 (22)	87 (8)
5	15	157 (12)	85 (6)	163 (17)	88 (10)
6	8	156 (14)	83 (7)	159 (12)	91 (6)
7	26	159 (24)	82 (8)	164 (20)	86 (11)
8	6	138 (23)	83 (11)	137 (10)	80 (4)
9	11	157 (24)	88 (11)	156 (18)	88 (11)
Healthy	195	153 (22)	84 (9)	159 (20)	87 (10)
Diseased	163	160 (23)	85 (10)	159 (22)	86 (11)
Hypertension only	29	181 (18)	96 (12)	169 (18)	90 (10)

Comment

In the elderly, changes in blood pressure relate to health. In those who remained healthy blood pressure continued to rise by 1.5 mm Hg per year but fell in those who developed disease (representing probable heterogeneous effects of different medical conditions). In the healthy group no rise was seen in retired blue collar workers. Possibly, retired manual workers have

more unrecognised disease, consistent with a persistent effect of socioeconomic factors on health status in old age. For healthy individuals the most important predictor of blood pressure at follow up was baseline blood pressure, accounting for 24% of the variance of systolic, but only 6% of the variance of diastolic, pressure. In general, disease affected systolic more than diastolic pressure. Hence the poorer correlation between baseline and follow up diastolic pressure compared with systolic pressure is not easily explained by possible undetected disease in the healthy group and deserves further investigation.

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Conflict of interest: None.

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Short stature and *Helicobacter pylori* infection in Italian children: prospective multicentre hospital based case-control study

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Helicobacter pylori is mainly acquired in childhood,¹ but the diseases associated with such infection remain unknown. Scottish and Italian schoolchildren infected with *H pylori* showed reduced growth in height,^{2,3} and *H pylori* gastritis was found in 55% of French children examined for short stature.⁴ To evaluate the role of *H pylori* and socioeconomic factors on growth we compared children with idiopathic short stature with those of normal height.

Subjects, methods, and results

This study was approved by and conducted within the guidelines of the gastric disease section of the Italian Society for Paediatric Gastroenterology and Hepatology (SIGEP). Between April 1996 and March 1997 we recruited 134 consecutive children aged 5-13 years (median 9.8 years) whose height was below the third centile—that is, two standard deviations below the mean height of their peers—from 26 paediatric gastroenterology and endocrinology units in Italy. We individually matched them with children of the same age and sex from the same region whose height was above the 25th

centile and who had been referred for minor diseases. Obvious medical reasons for short stature (chronic or neoplastic disease with or without genetic abnormalities) were excluded by history and appropriate tests. Information on risk factors was collected by structured questionnaires with questions on socioeconomic status (education, number of cohabiting relatives, and number of rooms at home) and anthropometric data. Weight and height were measured and serum samples collected.

Serum was tested for *H pylori* IgG in a central laboratory by enzyme linked immunosorbent assay (ELISA) (Helori, Eurospital, Italy). The assay was concurrently validated in 127 children of similar age whose *H pylori* status was known from the results of gastric biopsy. Children were considered to be infected when their titre was >12 AU/ml, the cut off point determined from receiver operating characteristic curves (sensitivity 87%, specificity 94%). The Wilcoxon rank sum test was used to evaluate differences in continuous variables. Categorical data analysis was used on matched sets. Conditional logistic regression models were used for multivariate matched analyses.

Cases had a lower birth weight ($P < 0.01$) and their parents were shorter ($P < 0.01$) and had attended school for fewer years ($P < 0.01$) (table). Families of controls had fewer children but the same number of rooms at home. A high crowding index and presence of more than one type of pet was significantly associated with short stature (table). Serology showed *H pylori* infection in 27 cases (20%) and 18 controls (13%) ($P = 0.191$). Prevalence tended to be higher in boys, but the difference was significant only in cases (27% (23/85) in short boys *v* 10% (5/49) in short girls, $P = 0.03$) and tended to be higher in cases with lower growth hormone concentrations (22% (10/46) in those with peak hormone concentrations < 10 pg/l *v* 12% (6/50) in those with concentrations ≥ 10 pg/l; $P = 0.314$). Parental height, lower birth weight, and a crowded home persisted in a multivariate analysis as independent predictors of short stature.

Comment

Our results show that *H pylori* is not a risk factor for short stature and that reduced growth is related to genetic determinants such as parental height and to mixed genetic and environmental factors such as birth weight. Low socioeconomic status was relevant. The decreased growth found in Scottish and Italian schoolchildren infected with *H pylori* might be related to the association between lower socioeconomic group and *H pylori* acquisition.^{2,3} The higher prevalence of *H pylori* in disadvantaged children suggests that infection should be considered a marker of low socioeconomic group in studies on growth, with other factors causing the reduced growth. Although genetic factors cannot be modified, more attention should be paid to pregnancy, living conditions, nutrition, infections, and emotional deprivation. The association between short stature and low socioeconomic group seems particularly relevant in view of the reported unsatisfactory long term efficacy of expensive treatment with growth hormone.⁵

Members of the study group were M Baldassarre (Bari), V Benigno (Palermo), E Braggion (Palermo), A Carlucci (Lanciano), G L de'Angelis (Parma), F De Luca (Messina), T Gentile (l'Aquila), G Guariso (Padua), L Iughetti (Florence), G Lauriola (Manfredonia), P Lionetti (Florence), A Liotta (Palermo), F Lizzoli (Pavia), R Longhi (Como), V Lucidi (Rome), A Masciale (Bitonto), M Pastore (San Giovanni Rotondo), A Pavanello (Pordenone), F Rea (Naples), C Romano (Messina), P Roggero (Milan), V Rutigliano (Bari), S Salardi (Bologna), M S Scotta (Varese), M Spina (Catania), A Tozzi (Naples).

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Contributors: GO designed and coordinated the study and wrote the manuscript. DP contributed to the epidemiological aspect of the study design, was responsible for the statistical analysis, and contributed to writing the manuscript. CS was jointly responsible for statistical analysis and interpreting data and revised the final version of the manuscript. EC supervised the collection of clinical data and critically revised the manuscript. GB was jointly responsible for the original study proposal and for coordination. The members of the study group approved the study proposal after extensive discussion, recruited all cases and controls at the 26 participating centres, completed the questionnaires, and collected blood for antibody determination. They also agreed to the adding of their names to the manuscript as members of the study group to show where the recruitment centres were. All approved the final version of the manuscript that was submitted for publication. GO and DP are guarantors for the study.

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Distribution of 134 cases and 134 controls according to selected characteristics and by matched sets

Factor	No of cases	No of controls	Matched sets*	Odds ratio (95% CI)		
				Univariate†	Multivariate‡	Multivariate§
<i>Helicobacter pylori</i> :						
Positive	27	18	225			
Negative	107	116	1691	1.6 (0.8 to 2.9)	0.8 (0.3 to 2.2)	1.3 (0.5 to 3.3)
Father's height (cm):						
<165	54	7	351			
≥ 165	80	127	476	12.7 (4.6 to 35.3)	8.7 (2.8 to 27.6)	
Mother's height (cm):						
<155	69	13	762			
≥ 155	65	121	659	10.3 (4.5 to 23.9)	8.0 (3.0 to 20.9)	
Mean parental height (cm):						
<160	62	5	260			
≥ 160	72	129	369	20.0 (6.3 to 63.8)		28.6 (7.3 to 112)
Parents' education (years):						
<12	28	11	523			
≥ 12	106	123	6100	3.8 (1.6 to 9.4)	2.1 (0.9 to 6.4)	
Birth weight (g):						
<2850	38	17	731			
≥ 2850	96	117	1086	3.1 (1.5 to 6.3)		9.6 (2.4 to 37.9)
Crowding index (subjects/room):						
≥ 1	17	7	215			
<1	117	127	5112	3.0 (1.1 to 8.2)		6.9 (1.0 to 48.7)
Pets (No of types):						
>1	24	7	123			
0-1	110	127	6104	3.8 (1.6 to 9.4)	1.9 (0.6 to 5.9)	

*Distribution of matched sets according to combination of exposure status (positive or negative) for each case and control.

†McNemar's test based on matched sets.

‡Conditional logistic regression model including terms for *H pylori*, father's height, mother's height, parental education, and presence of pets.

§Conditional logistic regression model including terms for *H pylori*, mean parental height, birth weight, and crowding index.

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Correction

Seasonal variation in coronary artery disease mortality in Hawaii: observational study

An authors' error occurred in this paper by Todd B Seto and colleagues (27 June, p 1946-7). The graph of variation in average temperature was incorrect. The correct graph is shown below. Red lines represent monthly averages and blue lines three month moving averages.

