phrenia. Although there is a biological component to psychosis, this finding suggests that the personal and social pressures of belonging to any ethnic minority group in Britain are important determinants of the excess of psychotic disorders found.

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Helicobacter pylori infection in childhood: risk factors and effect on growth

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Abstract

***Objective*—To investigate the current prevalence of *Helicobacter pylori* infection in childhood, the risk factors for infection, and the effect of infection on growth in preadolescent schoolchildren.**

***Design*—Population based sample of 7 year old schoolchildren followed up at age 11; data on risk factors for infection collected at age 7; presence of infection at age 11 determined by measurement of salivary IgG against *H pylori* by a newly developed enzyme linked immunosorbent assay (ELISA). Height was measured at 7 and 11 years of age.**

***Subjects*—554 schoolchildren from Edinburgh.**

***Results*—62 (11%) children had *H pylori* infection. Independent risk factors for infection were single parent families (adjusted odds ratio=2·5; 95% confidence interval 1·1 to 5·7), the 10% most crowded homes (3·1; 1·3 to 7·2), and schools serving predominantly rented housing estates (2·5; 1·0 to 6·5). School catchment area was more important than parental social class or housing tenure. Growth in height between 7 and 11 was diminished in infected children by a mean of 1·1 cm (0·3 to 2·0 cm) over four years. This growth reduction was largely confined to girls (1·6 cm over four years), among whom it correlated with salivary IgG (P=0·015).**

***Conclusion*—Data from salivary assay to investigate the epidemiology of *H pylori* suggest that factors relating to the type of community in which the child lives may now be as important for acquisition of this infection as features of the family home. The greater reduction of growth among infected girls raises the possibility that *H pylori* infection may delay or diminish the pubertal growth spurt.**

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Introduction

Helicobacter pylori is a bacterium that colonises the human stomach and is the cause of type B gastritis.[1] It is strongly associated with peptic ulceration,[2] and more recently a link with gastric cancer has been found.[3] Once acquired, the infection lasts several decades in most cases, with a very low rate of spontaneous eradication.[4] Besides the well documented local effects of *H pylori*, little is known about the distant and perhaps more subtle effects of this chronic, lifelong infection.

In the developing world *H pylori* is acquired early in life; most of the population is infected by the age of 10 years.[5] In the developed world there is a gradual rise in prevalence with age. The most important risk factors in adults relate to socioeconomic deprivation,[5] and to overcrowding and poor hygiene in the childhood home.[6] Currently there are few data on prevalence of infection among children in developed countries, but it is believed to be low. It is not known whether the risk factors identified in adults still apply to children in more recent times, as transmission has greatly decreased.[7]

Viable *H pylori* organisms have never been isolated from the environment, hence interpersonal transmission is most likely. *H pylori* seropositivity is more common in household contacts of infected children,[8] but the family contacts of infected adults do not show

an increased risk of infection.[9] It is therefore uncertain whether the infection is mainly acquired from other family members.

Three studies have suggested that children and adults infected with *H pylori* are shorter than uninfected individuals. One study in the developing world showed the height to age ratio to be significantly lower in children infected with *H pylori* than in uninfected children.[10] A study in Europe indicated that infected adults were shorter than uninfected adults[11]; and a recent report showed that over half the children being investigated for short stature were infected with *H pylori*.[12] Whether these differences in height reflect poverty or may in some way be caused by *H pylori* is unclear.

Most epidemiological studies of *H pylori* have used serological testing. We have recently shown that IgG antibodies to *H pylori* can also be detected in saliva.[13] This method of determining *H pylori* status has practical advantages over other methods for studying the epidemiology of this infection in children. In the current study, stored saliva samples from a longitudinal study of children were used to investigate the current prevalence of *H pylori* infection, the risk factors for infection, and the effect of infection on growth in schoolchildren.

Subjects and methods

All children aged 6·5 to 7·5 years attending a random sample of 30 primary schools in Edinburgh, Scotland, were invited to take part in a survey of respiratory disease.[14] The parents of 1012 of 1095 children responded to a questionnaire (93% response rate) and 720 (71%) of these children were re-examined at age 11 years as part of a longitudinal study of lung function. At the second examination, each child provided a saliva specimen by spitting into a clean plastic container. The specimens were stored at −20°C within eight hours of collection and were thawed once for cotinine estimation (the original purpose of saliva collection). Sufficient saliva for measurement of IgG against *H pylori* was available for 554 children (77% of those examined; 54% of those originally recruited).

The children's schools were divided into four well defined areas characterised by the proportion of rented to owned accommodation in each school catchment area. These areas could generally be classed as affluent suburbs, central tenements, mixed housing, and local authority housing estates, the proportion of rented accommodation was less than 10%, 10-24%, 25-74%, and 75-100% respectively. The questionnaire at age 7 inquired about living conditions and social background. Standing heights were measured at ages 7 and 11 to the nearest centimetre with shoes removed.

TABLE I—*Prevalence of H pylori by sex and selected indicators of socioeconomic status and family structure*

Category	No recruited at age 7	No tested at 11	No positive	% Prevalence of *H pylori*	χ^2 test for trend	P value
All children:	1012	554	62	11·2		
Boys	505	280	25	8·9	2·17	0·12
Girls	507	274	37	13·5		
Adults in home at age 7:						
1	115	56	12	21·4	6·16	0·013
2	774	437	44	10·1		
>3	97	45	3	6·7		
Children in home at age 7:						
1	136	75	12	16·0	0·17	0·68
2	537	294	24	8·2		
3	238	128	17	13·3		
>4	86	49	7	14·3		
Persons per room at age 7:						
<0·5	339	183	18	9·8	3·98	0·046
0·5-1·0	500	286	27	9·4		
>1·0	126	56	13	23·2		
Social class of head of household:						
I+II	371	202	16	7·9	1·24*	0·27
III	412	234	32	13·7		
IV+V	115	70	7	10·0		
Unclassified	115	48	7	14·6		
Housing tenure at 7:						
Owned	705	390	36	9·2	4·08	0·043
Rented	302	160	25	15·6		
% of housing rented in school catchment area (age 7):						
<10	394	224	17	7·6	10·04	0·0015
10-24	243	118	12	10·2		
25-75	197	104	11	10·6		
75-100	178	108	22	20·4		

*Children of unclassified social class excluded from the test for trend across social groups.

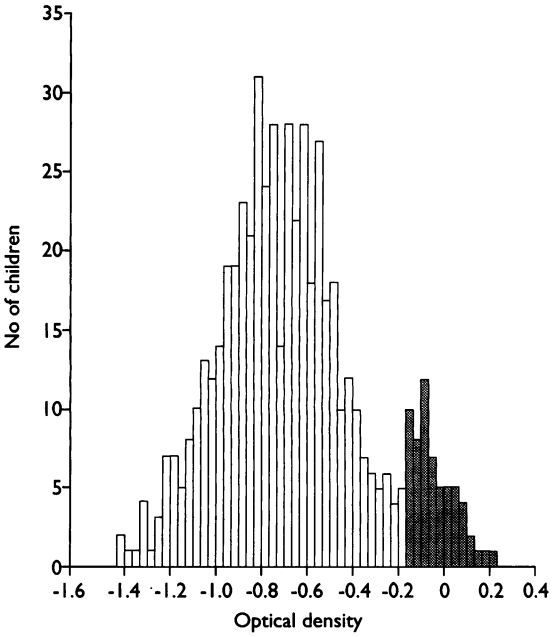

FIG 1—*Frequency distribution of log_{10} standardised optical density readings in 554 children. Positive results above cut off are shaded*

SALIVARY ELISA

H pylori status at age 11 was determined by measuring IgG specific to *H pylori* in saliva by an indirect enzyme linked immunosorbent assay (ELISA). Undiluted saliva was tested in duplicate by ELISA using an in house antigen, which is 98% sensitive and 94% specific for the presence of infection when validated on serum in adults.[6] The steps included incubation of saliva in duplicate with IgG standards in a microtitre ELISA system. Thereafter, salivary antibodies were measured by using a conjugate of anti-human IgG peroxidase followed by substrate and stopping solution. The wells were washed with PBS Tween buffer between each stage and the plate was read at 450 nm on a multispectrophotometer (Anthos Labtec, Austria) after the final step.

VALIDATION AND SELECTION OF CUT OFF POINT

The assay was validated for use in children on 26 normal children by the carbon-13 urea breath test. These 26 children were part of a separate study of *H pylori* in families resident in south London.[7] One control child was borderline positive and the rest negative on the test. A cut off point of 0·7 standard optical density units was chosen, being two standard deviations from the mean of the negatives. This point lay between the highest negative and the borderline positive. Optical densities on each plate were standardised to that of the validation plate to provide standardised optical densities for the test children. The standardised optical densities on the 554 test children showed a clearly bimodal distribution (fig 1), with the trough corresponding to our selected cut off point. Furthermore, the assay was 88% specific and 89% sensitive for the presence of infection when validated against histological and urease testing in 102 adults with the same cut off point.[15]

STATISTICAL ANALYSIS

Risk factors for *H pylori* were analysed by multiple

TABLE II—*Prevalence of* H pylori *infection by child's own housing tenure and proportion of rented homes in school catchment area*

% of rented homes in school catchment area (age 7)	Child's own home at age 7	
	% Owned	% Rented
<10	7·2 (15/209)	8·3 (1/12)
10-24	10·8 (11/91)	6·3 (1/16)
25-74	9 (6/67)	13·9 (5/36)
75-100	33·3 (4/12)	18·8 (18/96)

TABLE III—*Differences in height and growth of children with and without* H pylori *infection, before and after adjustment for potential confounding factors*

Difference (positive *v* negative for *H pylori*)	Boys (n=280)		Girls (n=274)		All (n=554)	
	Mean (SE) height (cm)	P value	Mean (SE) height (cm)	P value	Mean (SE) height (cm)	P value
Height at 7						
Unadjusted	0·93 (1·20)	0·45	0·25 (0·92)	0·79	0·53 (0·74)	0·47
Adjusted*	0·30 (1·21)	0·80	0·01 (0·93)	0·99	0·14 (0·85)	0·85
Height at 11						
Unadjusted	1·12 (1·39)	0·42	1·97 (1·21)	0·11	1·62 (0·91)	0·08
Adjusted	0·48 (1·40)	0·73	1·59 (1·23)	0·20	1·24 (0·92)	0·18
Growth 7 to 11						
Unadjusted	0·19 (0·54)	0·73	1·72 (0·62)	0·006	1·09 (0·42)	0·01
Adjusted	0·18 (0·56)	0·74	1·60 (0·62)	0·01	1·10 (0·42)	0·01

*Adjusted by multiple regression for single parenthood, number of siblings, domestic overcrowding, parental social class, housing tenure, and school catchment area. Analyses of both sexes combined were adjusted additionally for sex.

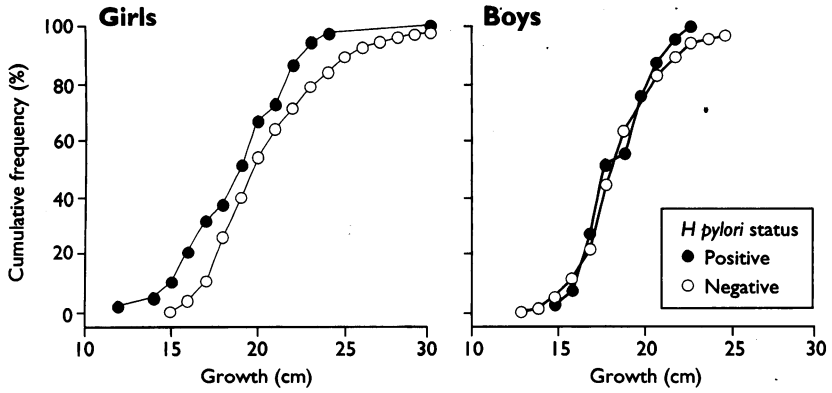

FIG 2—*Cumulative frequency distribution of growth in girls and boys positive and negative for H pylori*

logistic regression using the GLIM statistical package.[16] The number of rooms in the house and the number of children in the home were analysed as continuous variables. Number of adults, number of children, persons per room (crowding index), and parental social class were analysed both as continuous and as categorical variables. The effects of *H pylori* seropositivity on growth were analysed by multiple regression using as outcome variables, in turn, height at age 7; height at age 11; and the increase in height from ages 7 to 11.

Results

PREVALENCE OF H PYLORI INFECTION AND ASSOCIATED RISK FACTORS

The standardised optical density of the 554 saliva samples from the Edinburgh children was bimodally distributed (fig 1), and 62 (11·2%) children were above the cut off point selected to indicate *H pylori* infection. Table I shows the prevalence of infection by sex, family characteristics, and selected socioeconomic indicators. Groups at significantly higher risk of *H pylori* infection, on univariate analysis, were children with single parents, crowded households, and rented homes. There was little variation in the prevalence of infection with sibship size or parental social class.

Schools with a high prevalence of infection were widely scattered throughout the city, but all had catchment areas characterised by a high proportion of rented local authority housing. This systematic difference between schools persisted after adjustment for the child's housing (χ^2 for trend=9·1, df=1, P=0·002), whereas after adjustment for school catchment area there was little difference between the prevalence of infection among children from owned or rented homes (χ^2=0·1, df=1, P=0·9) (table II).

All factors in table I were analysed together in a multiple regression model, restricted to 524 children with complete data on all these variables. Children of single parents were at significantly increased risk (adjusted odds ratio=2·5, 95% confidence interval 1·1 to 5·7), but the inverse trend with numbers of adults in the household was not significant at the 5% level (P=0·08). Children from the most crowded homes (more than one person per room) were also at significantly increased risk (3·1, 1·3 to 7·2), although there was no consistent trend across all categories of housing density (P=0·13). The independent effects of sex, sibship size, housing tenure, and parental social class were all non-significant (P>0·1). After adjustment of all these factors, the odds ratio of infection was 2·5 (1·0 to 6·5), comparing a hypothetical school serving a population with 90% rented accommodation with another serving a catchment area with 10% rented accommodation (P=0·07).

RELATION OF H PYLORI INFECTION TO GROWTH

The children had a mean age of 90·7 months at the first examination, but at the second examination the mean age of children positive for *H pylori* was 133·9 months compared with 133·3 months for those negative for *H pylori*. Table III shows the difference in height between children with and without *H pylori* infection, at ages 7 and 11, for boys and girls separately and combined. Results are presented before and after adjustment for school catchment area, parental social class, housing tenure, domestic crowding, and family structure (single parenthood and sibship).

Children infected with *H pylori* were slightly shorter than their peers at age 7, although in neither sex did this difference approach statistical significance. At age 11 there was a greater difference in height between infected and uninfected children, which was of borderline statistical significance. These differences were in part explained by the effects of family circumstances and school catchment area (table III).

The growth between ages 7 and 11 of children infected with *H pylori* was significantly less than growth of uninfected children: 1·1 cm (0·3 to 2·0 cm) difference over four years. This effect of *H pylori* on growth was largely confined to girls: 1·6 cm (0·4 to 2·8 cm) difference over four years. There was little difference in growth between infected and uninfected boys: 0·2 cm (0·9-1·1 cm) difference over four years. These effects were changed very little by adjustment for family circumstances and school catchment area (table III). The cumulative frequency distribution of

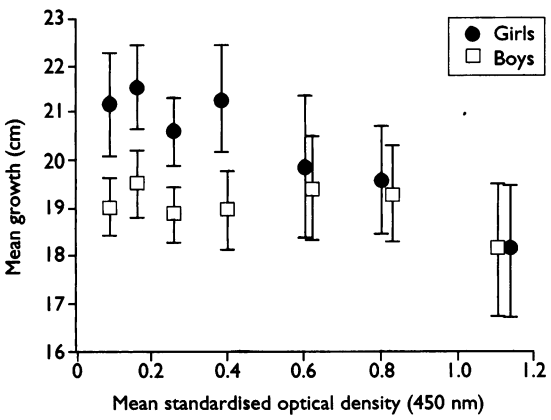

FIG 3—*Mean growth in groups defined by standardised optical density readings in girls and boys; bars show 95% confidence interval*

growth shows diminished growth in girls positive or negative for *H pylori* but no difference for boys positive or negative for *H pylori* (fig 2).

The effect modification (statistical interaction) of sex and *H pylori* infection was of borderline significance (test for interaction: P=0·1). However, when the relation between growth and titre of salivary IgG against *H pylori* was analysed in boys and girls separately (fig 3) there was a significant difference between the sexes in the gradient of the regression line relating growth to salivary optical density (test for interaction: P=0·015).

Discussion

Our data show that the prevalence of *H pylori* infection in children in Edinburgh aged 11 is about 11%. This relatively low prevalence of *H pylori* contrasts with a much higher prevalence in children from developing countries.[5] The only other British study showed a prevalence, determined by serological testing, of about 5% in 150 well schoolchildren.[17] Children with abdominal symptoms have a higher prevalence of infection (18%), as would be expected.[12] If, as other studies have suggested, the most important period of acquisition of *H pylori* is in childhood, then the relatively low prevalence of infection in these 11 year old children bodes well for the burden of peptic ulcers and gastric cancer in the future.

Prior validation of our salivary assay was limited because of the low prevalence of infection in children in south London. However, the distribution of optical densities in the Edinburgh study suggests the presence of two different populations. Our study used specimens originally collected for another purpose, and storage for three years at −20°C could have led to reduced antibody levels. On the other hand, gum disease could have led to increased transudation of IgG through the gingival crevice and thus to higher salivary antibody titres. Even with these possible effects, the salivary assay for *H pylori* infection performs well.[15]

RISK FACTORS

Like others, we have shown overcrowding and parental social class as risk factors for *H pylori* infection, but none of these studies have controlled for socioeconomic variables or for the general environment from which index cases were drawn. We found that school catchment area and housing tenure were associated risk factors for infection, but after adjustment for school catchment area there was no social class trend and the influence of housing tenure, family structure, and domestic crowding was small.

Previous studies in adults have suggested that childhood overcrowding and the absence of a fixed hot water supply in the childhood home are strong risk factors for infection.[6] The association of overcrowding with *H pylori* seropositivity would be consistent with interpersonal spread of infection within the home. This is supported by the increased incidence of seropositivity in adults who shared a bed in childhood.[18] However, it is of interest that the effect of overcrowding in the present study is confined to the most crowded 10% of homes, there being little gradient across the other groups. It seems likely that in more recent times acquisition from outside the home has become more important as living conditions have improved. In support of this, our data show that features of the community rather than those of the home are important in determining *H pylori* positivity among children in Edinburgh. Thus children attending schools in poor catchment areas are more likely to become infected, regardless of other risk factors. The high prevalence of infection among children of single parents, who tend to make greater use of child care, is also consistent with acquisition of infection outside the home.

H PYLORI AND GROWTH

The association of *H pylori* with diminished growth on children was independent of other confounding factors and is unlikely to be a chance effect. The effect was largely confined to girls, although results of the test of statistical interaction were not significant. The lack of association between height at age 7 and *H pylori* infection may be explained if the infection was acquired after this age. The greater effect in girls could be explained by earlier acquisition of infection in girls, but this is unlikely. An alternative explanation for these observations could be an effect of *H pylori* infection on the timing or the magnitude of the pubertal growth spurt. In modern times this is first noticeable between the ages of 9 and 10 in girls, but not until after the age of 11 in boys. The expected additional growth in pubertal girls compared with those not undergoing a pubertal growth spurt translates into a difference of about 2 cm by age 11,[19] which is comparable with the difference found in our study between infected and uninfected girls.

It is unlikely that the association of diminished growth with *H pylori* is confounded by some other factor since the putative confounder would need to be influential in girls but not in boys and in older but not younger children. Nor is it likely that risk factors for diminished growth in girls predispose to *H pylori* infection as there was no height difference at age 7. Social class, family structure, childhood crowding, housing tenure, and school catchment area were controlled for in our analysis, and any other factor would have to be strongly related to the timing of puberty to explain the observed association.

H pylori may merely be a marker for exposure to infectious diseases in general, since the seroprevalence of *H pylori* infection mirrors that of hepatitis A,[20] for instance. However, *H pylori* is a chronic persistent infection, which would be expected of an agent that causes diminished growth. *H pylori* is associated with dyspepsia, which may reduce nutritional intake, and in one study was found to be associated with protein losing enteropathy in children.[21] Our observed effect on growth could be due to malnutrition, but this is probably rare in *H pylori* infection, and the weak association with growth in boys makes this an unlikely explanation.

Diminished height at age 11 in girls may reflect slower growth during puberty. Alternatively, delayed puberty may also diminish growth.[22] Two alternative mechanisms could be postulated whereby *H pylori* could diminish pubertal growth velocity. Firstly, the effect may be non-specific, similar to that seen in chronic diseases like diabetes[23] and inflammatory bowel disease.[24] Secondly, *H pylori* infection could disturb the rise in gonadal oestrogens or growth hormone which naturally occur during puberty. Our data suggest that the degree of immune response to *H pylori* as reflected by salivary IgG levels may be an important part of such a mechanism (fig 3). Release of cytokines (interleukins 1, 6, and 8 and tumour necrosing factor alpha) is induced by *H pylori*.[25 26] Recent evidence suggests that three of these cytokines (interleukin $1_{\beta}6$, and tumour necrosing factor alpha) can disturb endocrine ovarian function.[27] One report has shown that tumour necrosing factor alpha is increased in the circulation of children with relapsing chronic inflammatory bowel disease and inversely correlates with growth velocity.[24] Thus there may be a mechanism of diminished pubertal growth velocity by a chronic low grade effect of systemic inflammatory mediators, either directly at the epiphyseal plate or indirectly through an effect on gonadal sex steroids.

Implications

- Retrospective studies in adults have suggested that early childhood circumstances, relating to the household, are important for acquisition of *Helicobacter pylori* infection
- This study shows that current risk factors for infection in children include single parent families and overcrowded homes
- Attending a school in a socially deprived area is a newly identified risk factor for infection, relating to the type of community serving that area
- *H pylori* infection is associated with diminished growth in preadolescent girls, but not in boys, between 7 and 11 years, suggesting that growth retardation occurs during puberty
- The effects of *H pylori* infection are not confined to the stomach

Data from Europe and America show that since 1900, children with average economic circumstances have increased in height at age 5-7 by about 1-2 cm a decade and at age 10-14 by 2-3 cm a decade.[28] This trend in Europe, partly due in girls to earlier age of menarche, is currently decreasing.[29] During the same period there has been an upward trend in adult height by 1·1 cm a decade in men and 0·3 cm a decade in women.[30] In earlier times final height was not reached in men until 25 years or later, whereas now it is reached at age 18 or 19.[29] Therefore, most of the trend towards greater size in children reflects more rapid maturation. Some of this difference could reflect the decreasing prevalence of *H pylori*, or some other exposure of which it is a marker, as living conditions have improved. We have also found height in adults infected with *H pylori* when compared with uninfected adults is diminished by, on average, 1·9 cm in men and 1·1 cm in women.[31] Therefore the effect of *H pylori* on growth in children may translate into diminished height in adulthood.

CONCLUSIONS

This is the first time that saliva has been used to investigate the epidemiology of *H pylori* infection, and we have shown that infection is relatively uncommon in preadolescent children in Edinburgh. The risk of *H pylori* infection in this population was related to the type of community in which the child lived as well as to features of the family home. We have also identified an effect of *H pylori* infection on growth in childhood relating to the time of puberty in girls, which needs confirmation by studying older boys and by further follow up of girls throughout puberty. If *H pylori* infection is found to be causally associated with growth in childhood and adolescence, it might in future be considered a treatable cause of diminished pubertal growth or delayed maturation.

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