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# Differences between infants and adults in the social aetiology of wheeze

Deborah Baker, John Henderson, and the ALSPAC Study Team

**Abstract**

***Objectives*—To compare the relation between relative deprivation, its associated social risk factors and the prevalence of wheeze in infancy and in adulthood.**

***Design*—A cross sectional population study.**

***Setting*—The three District Health Authorities of Bristol.**

***Subjects*—A random sample of 1954 women stratified by age and housing tenure to be representative of women with children <1 in Great Britain and selected from the Avon Longitudinal Study of Pregnancy and Childhood (ALSPAC).**

***Main outcome measures*—The prevalence of wheeze for infants at six months after birth and for their mothers and fathers at eight months postpartum. Potential mediators of the relation between relative deprivation and wheeze measured were overcrowded living conditions, number of other siblings in the household, damp or mouldy housing conditions, maternal and paternal smoking behaviour, and infant feeding practice.**

***Results*—63.4% (1239) of the sample lived in owner occupied/mortgaged accommodation (relatively affluent) and 36.6% (715) lived in council house/rented accommodation (relatively deprived). Wheeze was significantly more likely for infants living in council house/rented accommodation ($\chi^2$ = 15.93, df=1, p<0.0001), their mothers ($\chi^2$ = 9.28, df=1, p <0.001) and their fathers ($\chi^2$ = 7.41, df=1, p<0.01). For those living in council house/rented accommodation backward stepwise logistic regression analyses showed that infants with other siblings in the household were significantly more likely to wheeze ( OR = 1.83, 95% CI = 1.27, 2.65), as were infants whose mothers smoked (OR = 1.82, 95% CI = 1.30, 2.55) and those who were breast fed for less than three months (OR = 0.66, 95% CI = 0.44, 0.98). Mothers with a partner who smoked were significantly more likely to report wheeze (OR = 1.73, 95% CI = 1.05, 2.85). There was no independent association between the social factors included in the analysis and the likelihood of wheeze for fathers.**

***Conclusions*—This study identified differences in the social factors associated with a higher prevalence of wheeze in infancy and in adulthood; results suggested that this symptom was commonly linked to infection in infancy, but not in adulthood. While environmental tobacco smoke was associated with a higher prevalence of wheeze in infancy and in adulthood, this does not necessarily indicate a common underlying mechanism; possible explanations are discussed.**

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**National Primary Care Research and Development Centre, 5th Floor, Williamson Building, University of Manchester, Oxford Road, Manchester**

Correspondence to:
Dr D Baker.

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Lower respiratory illnesses (LRI) have in the past been closely associated with poverty in infancy, in childhood and in adulthood. But for wheeze, the most common symptom of LRI that is increasingly prevalent in developed economies,[1 2] there is less consistency in the relation between prevalence and deprivation at different points in the life course. Wheeze is more commonly reported for infants in relatively deprived families[3–5] but for school age children it is the severity of symptoms rather than prevalence that is most consistently associated with deprivation.[6–8] In adolescence and adulthood studies of the relation between socioeconomic status and the prevalence of wheeze have either shown a higher prevalence for adults with higher socioeconomic status,[9] or a higher prevalence for those with lower socioeconomic status[10] or find no association at all.[11 12]

One plausible explanation for this lack of consistency would be the growing body of evidence suggesting that the pathogenesis of wheeze changes from infancy through childhood, occurring primarily in conjunction with viral respiratory infections in infancy and in older children and young adults, as an atopic response to airborne allergens. Such differences could underlie variation in patterns of association between social risk factors that mediate the effect of relative deprivation on wheeze in infancy and in adulthood. If this were to be the case it would have theoretical implications for current models of life course epidemiology, suggesting that the same symptom can have different social origins in childhood and in adulthood. Studies of discrete populations of infants and adults provide some evidence to support this assumption. For example, wheeze in infancy is closely associated with social factors that are known to increase the risk of infection such as overcrowded living conditions, large family size and a lower likelihood of breast feeding.[3 4 13–17] Maternal smoking is an additional risk factor for wheeze in infancy[18 19]; this association has been attributed to diminished lung function occurring as the consequence of smaller airways for low birthweight babies of women who smoke.[20–22] Evidence of a relation between LRI and paternal smoking, albeit less consistent, indicates that the effect of smoking could

also be attributable to environmental exposure to tobacco smoke in the home.[23–25]

In adulthood wheeze as an atopic response has been associated with damp or mouldy housing conditions in some studies, but not in others.[26–30] These housing conditions are thought to provide a conducive environment for airborne allergens such as the house dust mite and the airborne spores of fungal moulds.[31 32] Active smoking has been associated with wheeze for atopic adults and with adult onset wheeze for non-atopic adults.[10 12 33 34]

There is however little research that has compared the influence of this range of social risk factors on the prevalence of wheeze in which infants and adults have been drawn from the same population and social conditions are held constant. The conventional method has been to follow the same population cohort from infancy into adulthood. Change over time in the meaning of proxy measures of deprivation such as social class,[35] as well as social bias in attrition are rarely documented in cohort studies, and yet they are likely to affect the robustness of conclusions drawn about the relation between social factors and health at different historical time points.

In this study the prevalence of wheeze and its associated social and behavioural risk factors were examined for infants, their mothers and their fathers drawn from the same population cohort, the Avon Longitudinal Study of Pregnancy and Childhood (ALSPAC) and thus exposed to the same social conditions. The aim of the study was to test the hypotheses that (a) there will be variation in the social risk factors that mediate the relation between relative deprivation and wheeze in infancy and adulthood and (b) that these patterns of association will be indicative of a different aetiology for this symptom at these life stages.

## Methods

### SAMPLE SELECTION

The sample was selected from the ALSPAC, for which all women having a baby between April 1991 and December 1992 in the three health districts of Bristol were approached to invite their participation. For this study data from self completion questionnaires about the infant's health at six months postpartum and both parents health at eight months postpartum were used. A requirement for inclusion in the sample for this study was that questionnaire data on respiratory health were complete for the mother, her partner and their 6 month old infant. This requirement meant that the sample available for analysis was 5701 out of a possible 11 534, because of the much lower response rate of partners and the exclusion of mothers who were not in contact with the father of their child. This "complete cases" sampling reduced the high level of missing values that would otherwise have been included in the analysis, but such a procedure is likely to produce a socially biased sample.[36] It was important for the purposes of this study to be as certain as possible that any identified differences in the social aetiology of wheeze between infants, their mothers and their fathers were not the consequence of biases in the social distribution of responses available for analysis. To avoid biases arising from such selective procedures a random sample was drawn from the sample available for analysis that was representative of those women with children under 1 in Great Britain in the 1991 census. Representativeness was established using age (under 30 or 30 and over) and housing tenure (owner occupied/mortgaged accommodation or council house/rented accommodation) as stratifying variables so that the random sample had the same proportion of mothers in these categories as the 1991 census: 35.8% were under 30 and lived in owner occupied/mortgaged accommodation, 27.6% were over 30 and lived in owner occupied/mortgaged accommodation, 28.6% were under 30 and lived in council house/rented accommodation and 8% were over 30 and lived in council house/rented accommodation. This stratified random sample consisted of 1954 women, with responses available about their own health and social circumstances and those of their partners and children.

### MEASURES USED

The prevalence of wheeze for infants at six months postpartum and for their parents at eight months postpartum was recorded for mothers and fathers by asking "have you had wheeze since the baby was born"; in addition mothers were asked on a separate questionnaire "has your baby had wheeze since his or her birth".

Relative deprivation was measured using housing tenure; this has been shown to be a robust proxy measure for family income[37] and in the selected sample was closely associated with other measures of deprivation such as use of car ($\chi^2$ = 286.9, df=1, p<.0001) and partner's unemployment ($\chi^2$ = 235.3, df=1, p<.0001). Possible mediators of the relation between relative deprivation and a higher prevalence of wheeze in infancy and in adulthood were also measured. These were overcrowded living conditions as measured by maternal report of number of persons per room, number of other siblings in the family at eight months postpartum and the extent of mould or damp in the house as measured by maternal report at eight months postpartum. Maternal and paternal smoking behaviour was recorded at eight months postpartum by asking the mother and the father about whether they smoked and if so, the number of cigarettes smoked per day. Infant feeding practice was measured at six months postpartum by maternal responses to questions as to whether their baby had been breast fed and if so, for how long.

### ANALYSIS

The prevalence of wheeze for infants, their mothers and their fathers was calculated for the whole sample. Each of these populations was then divided into two groups, those living in owner occupied/mortgaged accommodation (relatively affluent) and those living in council house/rented accommodation (relatively

*Table 1 Relation between social and behavioural variables and the prevalence of wheeze for those living in mortgaged and in rented accommodation*

| | | *Infant* | | | |
|---|---|---|---|---|---|
| *Risk factors for wheeze* | | *mortgaged* n† % | $\chi^2$ | *rented* n† % | $\chi^2$ |
| *Risk factors associated with infection* | | | | | |
| Crowding | ⩽1 per room | 217 (19.0) | 0.70 | 112 (24.2) | 5.04* |
| | >1 per room | 12 (15.2) | | 75 (32.2) | |
| Presence of other children in household | yes | 141 (23.5) | 17.20**** | 131 (31.2) | 11.80*** |
| | no | 91 (14.3) | | 58 (19.7) | |
| Breast fed infant | Yes | 175 (17.5) | 4.70* | 113 (25.3) | 0.71 |
| | No | 55 (23.7) | | 73 (28.2) | |
| Breast fed infant | never/<3 months | 123 (20.0) | 0.00 | 142 (28.3) | 4.51* |
| | ⩾3 months | 105 (17.5) | | 42 (20.5) | |
| *Poor housing conditions* | | | | | |
| Damp reported | Yes | 120 (18.7) | 0.00 | 110 (26.7) | 0.02 |
| | No | 109 (18.6) | | 77 (26.2) | |
| Mould reported | Yes | 62 (21.2) | 1.60 | 110 (28.6) | 0.98 |
| | No | 167 (17.9) | | 77 (25.2) | |
| *Parental smoking behaviour* | | | | | |
| Mother smokes | Yes | 36 (18.9) | 0.01 | 98 (33.4) | 12.16*** |
| | No | 194 (18.7) | | 91 (21.7) | |
| Number smoked per day | 1–9 | 11 (14.7) | 4.66 | 26 (31.3) | 0.49 |
| | 10–19 | 17 (18.5) | | 53 (35.3) | |
| | 20+ | 8 (34.8) | | 19 (31.7) | |
| Father smokes | Yes | 60 (22.9) | 4.03* | 105 (30.3) | 5.98* |
| | No | 169 (17.5) | | 82 (22.7) | |
| Number smoked per day | 1–9 | 21 (25.3) | 0.44 | 13 (29.5) | 0.75 |
| | 10–19 | 20 (22.5) | | 57 (32.4) | |
| | 20+ | 19 (21.1) | | 35 (27.8) | |

†Number reporting wheeze. The small discrepancies in the total number reporting wheeze between social and behavioural variables are because of missing values for these variables.
*=p<0.05; **=p<0.01; ***= p<0.001; **** =p<0.0001.

deprived). Initial analyses identified significant differences between these groups in the prevalence of wheeze, in housing conditions and in health related behaviours using $\chi^2$ tests of independence. These analyses were then repeated for infants, for fathers and for mothers living in council house/rented accommodation and in owner occupied/mortgaged accommodation. Variables associated with a higher prevalence of wheeze ($p < 0.10$) at the univariate level were then entered into a stepwise logistic regression analysis to test for the independence of their association with this health outcome. Constructing logistic regression models for this purpose within groups divided into relatively deprived and relative affluent by housing tenure was preferred to building a model into which housing tenure was entered simultaneously with the other social risk factors for wheeze. If these factors do function as mediators of the relation between deprivation and wheeze, including them in the same logistic regression model could explain the effect of housing tenure, underestimating the strength of its association with wheeze. A final analysis estimated the extent to which the social risk factors identified as significant in the stepwise regression models mediated the effect of housing tenure on wheeze (see note 1, table 3). A detailed rationale for this procedure is well described by Baron and Kenny.[38]

## Results

Wheeze was reported for 21.5% (421) infants at six months after birth; 7.8% (158) of their mothers reported wheeze at eight months postpartum, as did 9.0% (176) of their fathers. 63.4% (1239) of the sample lived in owner occupied/mortgaged accommodation and 36.6% (715) lived in council house/rented accommodation. Wheeze was significantly more likely to be reported for infants living in council house/rented accommodation ($\chi^2$ = 15.93, df=1, p<0.0001), their mothers ($\chi^2$ = 9.28, df=1, p <0.001) and their fathers ($\chi^2$ = 7.41, df=1, p<0.01). Those living in council house/rented accommodation were significantly more likely to report overcrowded housing ($\chi^2$ = 237.8, df=1, p<0.0001), damp ($\chi^2$ = 20.30, df=1, p<0.0001) or mouldy ($\chi^2$ = 44.35, df=1, p<0.0001) housing conditions, having other children in the household ($\chi^2$ = 19.03, df=1, p<0.0001), maternal smoking ($\chi^2$ = 158.68, df=1, p<0.0001), paternal smoking ($\chi^2$ = 159.24, df=1, p<0.0001), and artificial infant feeding ($\chi^2$ = 75.28, df=1, p<0.0001), or breast feeding for less than three months ($\chi^2$ = 69.54, df=2, p<0.0001).

### RISK FACTORS FOR WHEEZE FOR THOSE LIVING IN OWNER OCCUPIED/MORTGAGED ACCOMMODATION (TABLES 1 AND 2A)

*Social factors associated with respiratory infection*

Table 1 shows that for infants living in owner occupied/mortgaged accommodation a higher prevalence of wheeze was associated with the presence of other children in the household ($\chi^2$ = 17.2, df=1, p <0.0003) and not being breast fed ($\chi^2$ = 4.7, df=1, p<0.0299). Backward stepwise regression analysis entering these two variables (table 2A) showed that the presence of other children in the household was the only variable independently associated with the higher prevalence of wheeze (odds ratio (OR)= 1.84, 95% confidence intervals (CI) = 1.37, 2.47). There were no significant univariate associations between crowding or the presence of other children in the family and the prevalence of wheeze for either mothers or fathers living in owner occupied/mortgaged accommodation.

*Table 1 continued*

| *Mother* | | | | *Father* | | | |
|---|---|---|---|---|---|---|---|
| *mortgaged n† %* | $\chi^2$ | *rented n† %* | $\chi^2$ | *mortgaged n† %* | $\chi^2$ | *rented n† %* | $\chi^2$ |
| 74 (5.1) | 0.25 | 41 (13.3) | 3.30 | 89 (7.8) | 0.00 | 54 (11.7) | 0.00 |
| 4 (6.5) | | 31 (8.9) | | 6 (7.6) | | 27 (11.6) | |
| 44 (7.3) | 1.70 | 44 (10.5) | 0.07 | 53 (8.8) | 2.20 | 43 (10.2) | 1.21 |
| 35 (5.5) | | 29 (9.8) | | 42 (6.6) | | 38 (12.9) | |
| 45 (7.7) | 3.32 + | 47 (11.4) | 1.22 | 50 (8.5) | 1.22 | 49 (11.9) | 0.49 |
| 33 (5.1) | | 26 (8.8) | | 44 (6.9) | | 30 (10.2) | |
| 26 (8.9) | 4.12* | 33 (12.3) | 2.00 | 27 (9.2) | | 30 (11.2) | 0.00 |
| 52 (5.6) | | 39 (8.9) | | 67 (7.2) | | 49 (11.2) | |
| 16 (8.4) | 1.60 | 35 (11.9) | 1.55 | 14 (7.4) | 0.27 | 38 (13.0) | 1.25 |
| 62 (6.0) | | 38 (9.1) | | 80 (7.7) | | 43 (10.3) | |
| 3 (4.0) | 4.52 | 10 (12.0) | 0.01 | 7 (9.3) | 2.26 | 11 (13.3) | 0.03 |
| 9 (9.8) | | 18 (12.0) | | 7 (7.6) | | 19 (12.7) | |
| 4 (17.4) | | 7 (11.7) | | 0 (0.0) | | 8 (13.3) | |
| 21 (8.0) | 1.57 | 44 (12.7) | 4.75* | 19 (7.3) | 0.10 | 47 (13.6) | 3.47 |
| 57 (5.9) | | 28 (7.8) | | 76 (7.9) | | 33 (9.1) | |
| 5 (6.0) | 3.46 | 6 (13.6) | 1.86 | 5 (6.0) | 0.27 | 3 (6.8) | 2.01 |
| 11 (12.4) | | 26 (14.8) | | 7 (7.9) | | 25 (14.2) | |
| 5 (5.6) | | 12 (9.5) | | 7 (7.8) | | 19 (16.1) | |

*Poor housing conditions*

Table 1 shows that the number of infants reported as having wheeze did not significantly increase if they lived in damp or mouldy housing conditions; neither were significantly more fathers with wheeze living in such housing conditions. However, an increased prevalence of wheeze for mothers was found for those living in housing where mould was reported ($\chi^2$ = 4.1, df=1, p<0.0424) and there was a trend for this also to be the case when damp was reported ($\chi^2$ =3.32, df=1, p=0.0684). For the stepwise logistic regression analysis neither variable entered the model as an independent predictor of wheeze significant above the 5% level.

*Table 2 Stepwise logistic regression analyses showing the relation between the prevalence of wheeze and social and behavioural variables**

| *(A) Those living in owner occupied/mortgaged accommodation* | | |
|---|---|---|
| *Infants with wheeze* | | *Adjusted odds ratios† (95% CI)* |
| *Risk factors associated with infection* | | |
| Presence of other children in household | no | 1.00 reference |
| | yes | 1.84 (1.37, 2.47) |
| | $\chi^{2}$* p | 17.10 (p<0.0001) |
| Breast fed infant | No | DEM‡ |
| | Yes | |
| *Smoking behaviour* | | |
| Father smokes | No | 1.00 reference |
| | Yes | 1.41 (1.01, 1.97) |
| | $\chi^{2}$* p | 3.93 (p<0.0474) |
| *Mothers with wheeze* | | |
| *Poor housing conditions* | | |
| Mould reported | No | DEM |
| | Yes | |
| *(B) Those living in council house/rented accommodation* | | |
| *Infants with wheeze* | | |
| *Risk factors for infection* | | |
| crowding | ≤1 person per room | DEM‡ |
| | >1 person per room | |
| other children in the household | No | 1.00 reference |
| | Yes | 1.83 (1.27, 2.65) |
| | $\chi^{2}$* p/ | 11.66 (p<0.001) |
| breast feeding | Never/<3 months | 1.00 reference |
| | 3+ months | 0.66 (0.44, 0.98) |
| | $\chi^{2}$* p | 4.32 (p<0.04) |
| *Smoking behaviour* | | |
| Mother smokes | No | 1.00 reference |
| | Yes | 1.82 (1.30, 2.55) |
| | $\chi^{2}$* p | 12.08 (p<0.001) |
| Father smokes | No | DEM |
| | Yes | |
| *Mothers with wheeze* | | |
| *Smoking behaviour* | | |
| Partner smokes | No | 1.00 reference |
| | Yes | 1.73 (1.05, 2.85) |
| | $\chi^{2}$* p | 4.78 (p<0.029) |

*Only those variables reaching the 10% level of significance in the univariate analysis were included in the logistic regression models. †Odds ratios were adjusted only in relation to the other variables within each section presented in the table. ‡DEM = did not enter the model.

*Smoking behaviour*

Table 1 shows that for infants living in owner occupied/mortgaged accommodation father's smoking was significantly associated with an increased prevalence of wheeze ($\chi^2$ = 4.03, df=1, p<0.03); table 2A shows that infants with a father who smoked were 1.41 times as likely to have wheeze than those with a father who did not smoke (95% CI = 1.01,1.97). There was no indication that mothers or fathers who smoked were significantly more likely to report wheeze than non-smokers.

## RISK FACTORS FOR THE PREVALENCE OF WHEEZE FOR THOSE LIVING IN COUNCIL HOUSE/RENTED ACCOMMODATION (TABLES 1 AND 2B)

*Social factors associated with respiratory infection*

Table 1 shows that for infants living in council house/rented accommodation, wheeze was more likely to be reported for those living in crowded accommodation ($\chi^2$ = 5.04, df=1, p<0.0247), those living in a household with other siblings ($\chi^2$ = 11.8, df=1, p<0.0006), and those who had not been breast fed, or had been breast fed for under three months ($\chi^2$ = 4.51, df=1, p<0.0336). Stepwise regression analysis using only these variables (table 2B) showed that the number of siblings in the household and the duration of breast feeding were

*Table 3 Estimated mediating effect of significant social and behavioural variables* on the relation between housing tenure (as a proxy for relative deprivation) and the prevalence of wheeze*

| | *Odds ratio (95% confidence intervals)* | $\chi^2$ | *p value* |
|---|---|---|---|
| *Infants with wheeze* | | | |
| Housing tenure | | | |
| mortgaged | 1.00 reference | | |
| rented | 1.249 (1.12, 1.39) | 15.4 | 0.0001 |
| Housing tenure controlled by number of children per household | | | |
| mortgaged | | | |
| rented | 1.215 (1.09, 1.36) | 11.75 | 0.0006 |
| Housing tenure controlled by duration of breast feeding | | | |
| mortgaged | | | |
| rented | 1.203 (1.07, 1.35) | 10.3 | 0.0015 |
| Housing tenure controlled by maternal smoking | | | |
| mortgaged | | | |
| rented | 1.197 (1.07, 1.34) | 9.28 | 0.0023 |
| Housing tenure controlled by number of children in household, duration of breast feeding, maternal smoking | | | |
| mortgaged | | | |
| rented | 1.123 (1.00, 1.27) | 3.54 | 0.0600 |
| *Mothers with wheeze* | | | |
| Housing tenure | | | |
| mortgaged | 1.00 reference | | |
| rented | 1.249 (1.12, 1.39) | 15.4 | 0.0001 |
| Housing tenure controlled by paternal smoking | | | |
| mortgaged | | | |
| rented | 1.214 (1.02, 1.45) | 4.66 | 0.0308 |

A variable functions as a mediator when (a) variations in levels of the independent variable (housing tenure) significantly account for variations in the presumed mediator (b) when variations in the presumed mediator significantly account for variations in the dependent variable (the prevalence of wheeze) and (c) when (a) and (b) are controlled the relation between the independent and dependent variables is significantly decreased.[38] Conditions (a) and (b) were met for all potential mediating variables included in the table. The effect of housing tenure on the prevalence of wheeze with and without controlling for potential mediating variables is displayed in the table.
*Variables identified as independently associated with the prevalence of wheeze in the stepwise logistic regression analysis displayed in table 2.

independently related to the prevalence of wheeze, but that overcrowding did not enter the model. There was no association between factors associated with infection and the prevalence of wheeze for either the mothers or the fathers of these infants.

*Poor housing conditions*

Univariate analysis found no association between damp or mould in the home and a higher prevalence of wheeze for either infants, their mothers or their fathers.

*Smoking behaviour*

Table 1 shows that a significantly higher percentage of infants with wheeze were likely to have mothers who smoked ($\chi^2$ = 12.16, df=1, p<0.0005) or fathers who smoked, ($\chi^2$ = 5.98, df=1, p<0.0215), although the number of cigarettes smoked was unrelated to the prevalence of wheeze. The stepwise regression model (table 2B) showed only maternal smoking as an independent risk factor for wheeze (OR = 1.82; 95% CI =1.30, 2.55), paternal smoking did not enter the model.

Table 1 shows that maternal wheeze was more prevalent when the partner smoked ($\chi^2$ =4.75, df=1, p<0.0293); logistic regression analysis when only this variable was entered showed that mothers were 1.73 times more likely to report wheeze if their partner smoked (95%CI = 1.05, 2.85) There was also a trend for the prevalence of wheeze to be higher for fathers in relation to their own smoking ($\chi^2$ =3.47,df=1, p<0.0623), but this did not enter into the regression model as a significant predictor of wheeze for fathers.

THE MEDIATING EFFECT OF SOCIAL AND BEHAVIOURAL VARIABLES IN THE RELATION BETWEEN HOUSING TENURE AND WHEEZE

Table 3 shows the mediating effect of those variables identified as significant in the stepwise regression analysis on the relation between housing tenure and wheeze. In infancy each variable identified reduced the effect of housing tenure on wheeze, and when number of children per household, maternal smoking and duration of breast feeding were all controlled in the same regression model the association between housing tenure and wheeze became non-significant. For mothers paternal smoking reduced the effect of housing tenure on wheeze, but the relation remained significant, suggesting that this variable was not, on its own, a necessary and sufficient condition for the effect to occur.

## Discussion

The findings of this study showed that the prevalence of wheeze was significantly higher for infants, their mothers and their fathers living in relatively deprived circumstances, when this relation was compared between generations in the same socially representative cohort. This is unlikely to have been attributable to reporting bias, as parents of the children completed separate questionnaires about their own health. In addition, the social distribution of other respiratory symptoms showed contrasting patterns to that of wheeze. For example, we repeated our analysis using self report of the common cold and found that this was unrelated to relative deprivation in infancy and that relatively affluent mothers and fathers were more likely to report this symptom. There was some evidence to suggest that wheeze was associated with different social risk factors in infancy and in adulthood that could be linked to underlying differences in the aetiology of wheeze at these life stages. In infancy, for example, wheeze was more likely in infants living in both deprived and affluent households if there were other children living in the household, suggesting sibling illness as a source of respiratory infection[5] in the first year after birth. No such association was found for either mothers or fathers in these families. An additional risk factor for relatively deprived infants was not being breast fed at all or being breast fed for less than three months, suggesting that the probability of respiratory infection is increased in the first six months after birth without the protective effect of breast milk.

Smoking also seemed to have a differential impact in infancy and in adulthood. Maternal smoking at eight months postpartum was an important risk factor for wheeze for infants from relatively deprived families, but there was no significant difference in birth weight for infants with wheeze whose mothers smoked when compared with those whose mothers did not smoke ($t$ = 1.09, df=173, p< 0.278). Diminished lung function occurring as a consequence of smaller airways for low birth weight infants is thus unlikely to account for the relation between maternal smoking and a higher prevalence of wheeze in relatively

deprived families at eight months postpartum. The mechanisms underlying the relation between smoking and wheeze in infancy remain unclear, but the role of environmental tobacco smoke in its pathogenesis was further evidenced by the finding that in relatively affluent families, where only a small proportion of mothers smoked, paternal smoking was a risk factor for wheeze, albeit with a weaker statistical association.

In adulthood active smoking was not associated with a higher prevalence of wheeze. The most plausible explanation for this result is that wheeze is predominantly a symptom of asthma in young adults and such allergy based clinical syndromes are as likely to be prevalent in the population of non-smokers as smokers. Another possibility is that some adults with wheeze had already given up smoking because it exacerbated symptoms. There was some evidence of this for women but not for men. Women living in owner occupied/mortgaged accommodation who reported wheeze were significantly more likely to have given up smoking at some point in the past than those who did not report this symptom ($\chi^2$ =10.83, df=2, p<0.004). This was unlikely to have arisen as a consequence of giving up smoking at conception, as there was no significant difference in the percentage of women with and without wheeze who gave up smoking in early pregnancy. In terms of passive smoking, relatively deprived mothers with partners who smoked were 1.7 times more likely to report wheeze than mothers with a non-smoking partner; no relation was found between environmental tobacco smoke and wheeze for men. This could reflect differences in exposure between men and women; 36.4% of male smokers living in council house/rented accommodation smoked 20 or more cigarettes per day compared with 20.5% of female smokers. The replication of the analysis used in this study at a different time point would be a useful test of the robustness of the results concerning the relation between smoking and wheeze in adulthood. In addition a more detailed analysis could compare the effect of environmental tobacco smoke in families where both partners smoked compared with those in which either the father or the mother smoked.

It was also clear from this study that poor quality housing, as measured by damp or mouldy housing conditions and overcrowding, had no independent association with the prevalence of wheeze in infancy or adulthood. In infancy the relation between relative deprivation and a higher prevalence of wheeze was mediated by maternal behaviours such as smoking and a shorter duration of breast feeding and the presence of other children in the household; for mothers paternal smoking partially mediated this relation, but for fathers none of the social risk factors included in the analysis could account for the association between relative deprivation and a higher prevalence of wheeze. This could implicate influences in the outdoor/work environment that were not examined in this study.

KEY POINTS

- Wheeze was associated with different social risk factors in infancy and adulthood; evidence suggested a greater role for respiratory infection in the aetiology of wheeze in infancy.
- Environmental tobacco smoke was associated with wheeze for infants and for mothers, but not for fathers. This could reflect differences in time spent indoors.
- Damp and mouldy housing conditions were not associated with wheeze for either infants, their mothers, or their fathers.
- Life course epidemiology would benefit from the study of the social origins of specific conditions across generations at the same historical point in time.

In this study there were some aspects of the measurement of wheeze that could have affected the estimates of prevalence. Measurement of wheeze for adults was taken over a period of eight months rather than the standardised period prevalence of 12 months,[39] so that it produced a more direct comparison with the measurement of infant wheeze at six months. This could have introduced some bias into the results because of the potential for underestimation of the period prevalence of wheeze for adults. For ease of comparison between generations, the questions "have you had wheeze since the baby was born"/ "has your baby had wheeze since his or her birth" were used to estimate the prevalence of wheeze for infants and adults. For infants the more specific question of "has your baby had wheeze with whistling on the chest since his or her birth" was also asked at six months after birth. Comparison of maternal report of infant wheeze at six months after birth from both questions suggested that the question used in this study produced a higher prevalence (21.5%) when compared with the more specific question (18.5%). This could mean that the prevalence of wheeze for infants was marginally over estimated in this study.

The findings of this study are likely to be generalisable, although the exclusion of lone mothers and their infants could have meant that relatively disadvantaged households were under represented. The representativeness of the random sample was calculated on the basis of the proportions of mothers with children under 1 year falling into one of four categories according to age and relative affluence or deprivation, as measured by housing tenure (see above). These proportions were based on estimates derived from the 1991 census, which included lone mother households. The proportion of relatively deprived households included in the analysis has therefore not been under represented. It could be argued that bias could still have entered the analysis because lone motherhood could be independently associated with poorer respiratory health, after controlling for socioeconomic and demographic factors. This is not the case; previous work examining the mental and physical health

of lone mothers and their infants using the ALSPAC sample[40] has shown that lone mothers who are relatively deprived are no more likely to report wheeze for themselves or their infants than mothers with partners, who live in similarly disadvantaged social circumstances.

### IMPLICATIONS FOR FUTURE RESEARCH

In this study the combination of social risk factors associated with higher prevalence of wheeze was different for infants, their mothers and their fathers drawn from the same cohort. Where complex combinations of economic, cultural and lifestyle factors are likely to influence a health outcome, disentangling their relation with one another and their possible mediating or moderating function in the relation between socioeconomic status and health at different life stages represents an essential task for epidemiology. This is particularly the case if health policy is to be effectively targeted towards the prevention of respiratory illnesses that manifest themselves in wheeze. The findings of this study indicate that, in terms of the social determinants of respiratory health, the child is not in every respect "the father of the man" and that life course epidemiology could profit from studies that compare the social origins of respiratory symptoms across generations at the same historical point in time.

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