Understanding socio-economic inequalities in childhood respiratory health

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Abstract

Asthma is the most common chronic disease of childhood. Recent evidence has shown a socio-economic gradient in its distribution. This paper examines whether a number of factors argued to have led to a rise in the incidence of asthma might also explain the social gradient. Several of these have been the object of policy intervention, though not necessarily with the aim of lowering childhood respiratory conditions. Using a large cohort study (the Avon Longitudinal Study of Parents and Children) we find significant inequalities in three respiratory conditions in middle childhood. We investigate eight potential mediating factors: exposure to other children in infancy, child's diet, poor housing conditions, maternal smoking, parental history of asthma, poor child health at birth, maternal age at child's birth and local deprivation. We find that each of these alone typically explains a relatively modest part of each respiratory inequality, with child's diet, local deprivation and maternal smoking generally the most important. But taken together, the mediating factors account for a substantial part of the respiratory inequalities. So the socio-economic gradient appears to operate through a number of inter-correlated pathways, some of which may be amenable to policy intervention.

Key words: Asthma, wheeze, socio-economic inequalities, mediating
JEL number: I1

1. Introduction

Asthma is the most common chronic disease of childhood in western societies. Its prevalence in the UK is amongst the very highest throughout the world (ISAAC, 1998B) and the economic burden of asthma and associated allergic diseases alone to the UK National Health Service is estimated to be £1 billion per year (Gupta et al., 2004). Several recent studies have argued that there appears to be a socio-economic gradient in the prevalence of childhood asthma, especially in severe asthma. Childhood asthma and associated respiratory conditions are also linked to poorer subsequent respiratory outcomes (Ulrik, 1999) so that inequalities in childhood will translate into inequalities in adults.

A reduction in inequality in childhood asthma is not currently an explicit part of government policy. But tackling health inequalities in the UK is a cornerstone of the government's public health strategy. Early in its period of office the Labour Government appointed the Acheson Committee to take forward the work initiated by the Black committee in 1980 (DHSS, 1980; DoH, 1998). It produced a raft of proposals and in Labour's 2001 election manifesto contained the pledge to 'close the health gap'.

A recent review of national policy documents since the Labour government's election in 1997 observed that: "A narrow concern with promoting population health is giving way to a broader vision of the goals of policy. The broader vision combines a focus on health gain with a commitment to reducing inequalities in its social distribution" (Graham, 2004: 115). In 2001 two health inequality targets were established, focused on socioeconomic inequalities in infant mortality and on area inequalities in life expectancy (Department of Health, 2001b; ONS, 2002a). However, 'closing the gap' has proved difficult. Progress has been slow and patchy as the government's own review has made clear (DoH, 2005). Not only do the causes of health inequalities lie in the province of many departments but we know too little about the precise mediating factors that link social deprivation and poor health outcomes. It is in this context that we have sought to explore such mediating factors in one specific case where poor health and social deprivation are closely linked. And, whilst not designed with the objective of reducing inequalities in childhood asthma, the factors that are correlated with asthma indicate that many recent government policies might be expected to impact on inequalities in childhood asthma. For instance, motivated by the harmful consequences of living in cold and damp housing (which include an elevated risk of respiratory conditions in children), the government published its UK Fuel Poverty Strategy in 2001 with the aim of ending fuel poverty among families with children and other vulnerable households by 2010 (DETR, 2001). Fruit intake has a protective

effect against the onset of asthma (Gilliland et al., 2003; Woods et al., 2003) and fruit consumption is lower amongst poor than better off children. Thus recent initiatives to promote fruit consumption amongst all children, such as the National Schools Fruit Scheme, may have an influence on the social distribution of asthma and wheeze amongst children. Policies aimed at reducing smoking and traffic pollution are yet further examples of government policy that might be expected to affect the social distribution of childhood respiratory conditions.

So the government's general objective to reduce health inequalities, the widespread nature of childhood asthma and its social gradient mean that an understanding of the socio-economic determinants of inequalities in childhood asthma is of considerable public, and potentially policy, interest. This paper aims to shed light on these issues by first examining whether there is a socio-economic gradient in middle childhood respiratory conditions in the UK and then examining the extent to which several factors that have been argued to be associated with childhood asthma might be underpinning this socio-economic gradient. Our analysis is based on a rich birth cohort study of over 12,000 births in the Avon area of England in 1991/2 that contains linked information on respiratory symptoms in children and on the socio-economic status and behaviours of their mothers and partners of the mothers. In our identification of the mediating factors through which socio-economic status may be linked to inequalities in respiratory health across children, we draw heavily on the medical evidence.

Our analysis first establishes that there is a significant negative gradient between socio-economic status (SES) and three different respiratory outcomes in children in mid-childhood[1] and this gradient is largest for the most persistent respiratory condition. We then investigate the extent to which this social gradient can be explained by eight mediating factors, all of which have been identified as significant correlates of asthma or wheeze in childhood. These factors are: exposure to other children in infancy, child's diet, poor housing conditions, maternal smoking, parental history of asthma, poor child health at birth, maternal age at child's birth and local deprivation. Some of these factors are amenable to change through public policy intervention, for example, childhood diet and parental smoking, both of which are the focus of current public health policies. Other factors are more fixed (e.g. parental history of asthma, a child's initial health at birth, local deprivation) and so cannot be easily altered, or may be the outcome of a large set of forces, meaning that a single policy is unlikely to have much impact on their incidence. Nevertheless,

[1] The outcomes, described in Section 4.1, refer to the related but different respiratory phenomena of transient wheeze, persistent wheeze and asthma, the latter two representing more-severe respiratory conditions than the former.

even for these more immutable factors, it is of interest as to whether the observed SES gradient operates through these factors or through other channels.

The paper is organized as follows. Section 2 reviews recent empirical literature on the association between socio-economic status and childhood asthma, including asthma severity. Section 3 examines those factors that have been identified as determinants or correlates of childhood asthma or wheeze and so may constitute possible paths by which SES and respiratory conditions are associated (i.e are possible mediating factors). Section 4 describes the data including detail on the eight possible mediating factors that we explore. The results are reported in Section 5. A summary of the findings and concluding remarks are contained in the final section.

2. Inequalities in childhood respiratory conditions

2.1 The prevalence of asthma and wheeze

The prevalence of childhood asthma and wheeze has increased sharply in several industrialised countries over the last thirty to forty years (Von Mutius, 2000). Results from self-reported questionnaires by 13 and 14 year-olds as part of the International Study of Allergies and Asthma in Childhood (ISAAC) project (ISAAC, 1998A, 1998B), suggest that there are more cases of asthma in more westernised, affluent countries (Smyth, 2002) – a "disease of affluence" according to Hancox et al (2004) – prompting suggestions that the risk for asthma may be related to lifestyle and/or environmental factors associated with a modern, Western way of life.

Around one in five school age children in the UK have a medical practitioner-diagnosed asthma (National Asthma Campaign, 2001). This amounts to a rise of two to threefold compared to the early 1970s. However, rates of childhood asthma appear to have peaked in the UK in the mid 1990s and there is evidence of a decline thereafter (Sunderland and Fleming, 2004; Akinbami and Schoendorf, 2002). Results from the ISAAC project revealed that the rate of asthma is higher amongst children in the UK than any of the other 55 countries in the study.

2.2 Inequalities in respiratory conditions

Several recent studies have drawn attention to the existence of inequalities in childhood respiratory conditions. This recent empirical consensus holds for a variety of measures of socio-economic status, across a range of countries and for different respiratory outcomes. Most studies have used measures other than income to indicate a child's SES, such as father's education or occupation. For example, Almqvist et al. (2005) and Cesaroni et al. (2003) found a steep

gradient in the prevalence of asthma by father's occupational status for children in Stockholm and Rome respectively. These results hold after controlling for potentially confounding factors such as heredity for allergic diseases and maternal smoking. Using income as the measure of SES, Case et al (2002) show that the prevalence of asthma was inversely related to income in US children under nine years of age, though this association was insignificant for older children. These results were net of controls for family size and ethnicity, amongst others. Currie et al. (2004) find similar associations for England using the same analytical framework as Case et al. (2002).

Although most recent studies find a social gradient in the prevalence of childhood respiratory conditions, this finding is not universal. Koopman et al. (2002) found weak evidence on the link between socio-economic status and respiratory conditions in early childhood for a sample of children in the Netherlands in 1996/7. Hancox et al. (2004) found no convincing evidence of an association between income or parental occupation and childhood asthma at any age from childhood to early adulthood in a New Zealand cohort of children borne in 1972/3. Mielck et al. (1996) reviewed twenty-two studies undertaken between 1973 and 1994 and found little consistency in the relationship between SES and childhood asthma (see also the overview by Rona, 2000). It is possible that inequalities in childhood respiratory conditions may have become more apparent over the last twenty-five years, such that an inverse relationship between the prevalence of asthma and socio-economic status is more of a contemporary phenomenon (Rona et al., 1999).[2]

2.3 Inequalities in severity of respiratory conditions

Whilst the evidence on inequalities in the overall prevalence of asthma and wheeze is to some degree mixed, there is much more compelling support for the existence of inequalities for more-severe forms of respiratory conditions, such as severe asthma. Case et al. (2002) found that amongst US children with asthma, poor children were more likely to have severe asthma and that this effect was slightly larger for older children. In other words, whilst the authors reported that the income gradient in the prevalence of asthma decreased with age, the income gradient in asthma severity increased.

Mielck et al (1996) found that poverty and severe asthma were positively associated in Germany. Cesaroni et al (2003), who found a positive association

[2] In an analysis based on three surveys using the same design over a seventeen year period, Rona et al. (1999) found that children whose fathers had a semi or unskilled manual occupation had a three percentage point higher prevalence of asthma in the most recent survey in 1993/4 than children whose fathers belonged to other social classes (16 compared to 13 percent). This gap was not evident in the earlier surveys.

between low socio-economic status and asthma, estimated a stronger association for severe asthma and an even stronger association for hospital admission for asthma. In a UK-based cross-sectional sample of children aged 5 to 11, Duran-Tauleria and Rona (1999) found that father's education was not associated with asthma but that it was inversely related to persistent wheeze (a marker for more severe forms of asthma). Sherrif et al. (2001), who use the same English ALSPAC birth cohort as the present study, found that local authority accommodation (which is occupied disproportionately by low-income families) was associated with persistent (but not transient) wheeze in early childhood (see also Baker and Henderson, 1999). Moreover, asthma mortality rates and hospital admission rates, both markers of asthma severity, usually reveal much higher rates amongst low-income groups (Rona, 2000). There is one recent English study (Currie et al. 2004) that found no significant association between asthma severity and income, though this result contrasts to those reported for the US as well as in other English-based studies.

In summary, recent evidence suggests that socio-economic inequalities exist in childhood respiratory conditions, although this association appears to have been less pronounced twenty to thirty years ago. However, there is more compelling evidence – not just of late but over time – that there is a distinct social gradient in the distribution of more-severe respiratory conditions, such as severe asthma.

3. Socio-economic determinants of childhood respiratory conditions

Why might children from low socio-economic status families be more likely to experience asthma? In other words, what are the economic, demographic, lifestyle and environmental routes through which social disadvantage is transmitted into childhood asthma? A wide array of different factors have been suggested in the literature as associated with childhood asthma and other respiratory conditions, though there is currently no single factor which has been identified as leading to poor respiratory outcomes. These factors may operate simultaneously, though they have often been examined in isolation from one another. In addition, some of the factors may be associated with SES and so may potentially explain the link between respiratory conditions and SES. Others may not be socially graded and so, while associated with the incidence or prevalence of respiratory conditions, cannot be part of the reason for a social gradient.

We focus here on a set of potentially mediating factors that are behaviours of mothers (or parents) post the birth of the child, and so may act as the channel by which low SES translates into poorer respiratory outcomes. These are the

factors that may be most susceptible to policy change (though even these may be hard to change very quickly). Some of these may be socially graded, others may not be.

3.1 The hygiene hypothesis

The hygiene hypothesis posits that increased exposure to other children in infancy increases the risk of current infection but enhances the immune system's ability to protect against the onset of subsequent allergic conditions. It is argued that the reduction in microbial burden that has occurred during the past century in the industrialized world may have altered normal postnatal immune system development (Martinez and Holt, 1999). In particular, a reduction in rates of infection in infancy may have lead to deterioration in allergen-specific immune responses. Using number of siblings and attendance at day centres as proxies for exposure to children, many studies have found an inverse relationship between exposure to children in early childhood and subsequent onset of asthma (see, for example, Ball et al., 2000). The relevance of the hygiene hypothesis as a possible source of inequalities for child respiratory conditions arises from differences in family size and type of childcare arrangements that are correlated with socio-economic status.

3.2 Child diet

The benign effects of longer duration of breastfeeding for current and subsequent wheeze and asthma have been widely documented (see the review in Halken, 2004; for evidence from ALSPAC, see Sherriff et al., 2001, and Baker and Henderson). Fruit consumption has been inversely associated with depleted lung function in children (Gilliland et al., 2003), and, more specifically, apples and pairs have been found to be protective of asthma (Woods et al., 2003). Childhood dietary patterns vary with SES (North et al., 2000), whilst many aspects of diet in early life have been associated with current and subsequent respiratory difficulties. Thus, variation in foodstuffs by income may account for part of the relationship between SES and asthma.

3.3 Pollutants

A variety of air pollutants have been proposed as aggravating factors for asthma, the incidence of each is likely to vary across SES. These include indoor agents (e.g. cigarette smoke, house dust mites, exposure to pets, mould and damp) and outdoor pollution from motor vehicles. One of the more consistent findings relates to the harmful effect of smoking for development of childhood respiratory symptoms. Several studies have shown a significant association between parental (particularly maternal) smoking and increased wheezing and asthma in children (Lewis et al., 2004; Cook et al., 1999; Tager, 1998). Maternal smoking during pregnancy is significantly associated with reduced

respiratory function in early infancy and recurrent wheezing during infancy and early childhood (Halken, 2004).

3.4 Local Deprivation

Local deprivation is a measure of the environmental conditions in which a child lives, and one which is likely to be closely associated with SES, as poor individuals are less able to afford better neighbourhoods. A recent UK study by Duran-Tauleria and Rona (1999) found that persistent wheeze was associated with geographical variation, which was largely explained by variation in deprivation. The link with deprivation was less pronounced for less-severe forms of respiratory symptoms. Similarly, Cesaroni et al. (2003) found that living in an underprivileged area was a strong independent predictor of hospital admission for asthma - but not less-severe respiratory outcomes – in an Italian sample of children.

These four sets of factors, if associated with SES, could be mediators between SES and respiratory outcomes and also could be amenable to change through policy. The literature also draws attention to a number of factors that are less obviously categorised as mediating, in that they either are in place before the child's birth (for example parental asthma, which may be a genetic transmission of inequality) and/or they are very strongly associated with SES and so may be regarded as an aspect of SES rather than a mediating factor. As these factors have been argued to be important correlates of respiratory illness in children, we also include some of them in our empirical analysis, in order to compare their impact with factors that are more amenable to change. Most of these factors essentially fall into the category of human capital; they include maternal age and the early health of the child. Education level and age of mother have been widely documented to be inversely related to asthma. Cesaroni et al. (2003) find a link between asthma and maternal level of education; Sherriff et al. (2001) find a positive link between (lower) maternal age and respiratory conditions. In terms of the child's own human capital, low birth weight and pre-term births are known markers of subsequent poor child health and both occur disproportionately in low-income families (see Burgess et al., 2004, for evidence based on ALSPAC).

4. The data and methods

We use a rich UK data set on a cohort of children born in one region of the UK in the early 1990s. The Avon Longitudinal Study of Parents and Children (ALSPAC; Golding et al., 2001), is a local, population-based study investigating a wide range of socio-economic, environmental and other influences on the health and development of children. Pregnant women resident

in the former Avon Health Authority were invited to participate if their estimated date of delivery was between the 1st of April 1991 and the 31st of December 1992. Approximately 85% of eligible mothers enrolled, resulting in a cohort of 14,893 pregnancies. Our estimation samples are somewhat smaller than this, representing late miscarriages, stillbirths and post-birth sample attrition and non-response to questionnaire items.[3]

Respondents were interviewed at high frequency compared to any of the UK cohort studies.[4] They were given questionnaires pre-birth and then at regular intervals after the birth of their child. Here we use data from some twenty questionnaires covering the dates between 8 weeks gestation and the 85th month of the child.

4.1 The respiratory outcomes

We use four respiratory outcomes in the analysis. All are based on mother responses and are binary variables. When the study child was aged 6, 18, 30, 42 and 81 months, ALSPAC mothers were asked whether their study child had experienced "wheezing with whistling on the chest" in the past year (or since birth when asked at 6 months). In addition, mothers also provided information in the 81 month questionnaire on whether their child had experienced asthma in the last year.[5]

The first respiratory outcome is "ever wheezed", equal to one if ALSPAC mothers reported their child as having wheezed with whistling on the chest at any of the five points in time listed above or if the child had asthma at 81 months. This measure is similar to the 'transient early wheeze' group identified in the influential Tucson Study, Taussig et al. (2003). Asthma at 81 months is

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3 The cross-sectional representation of the ALSPAC sample has been investigated by comparison with the 1991 National Census data of mothers with infants under one year of age who were resident in the county of Avon. In general, the ALSPAC sample performed reasonably well, although mothers who were married or cohabiting, owned their own home, did not belong to any ethnic minority and lived in a car-owning household were slightly over-represented. As these are typically characteristics that are positively associated with income the initial ALSPAC sample is likely to contain a lower number of mothers with low-income than the population. See Golding et al. (2001).

4 For example, the UK National Child Development Study (NCDS) interviewed at birth and then again at 7. The UK Birth Cohort Study (BCS70, first wave was in 1970) has a similar gap.

5 The questions on whistling with wheezing on the chest have been used in other ALSPAC research, see, for example, Sherrif et al. (2001). The maternal responses on asthma at 81 months have been used by Burgess et al. (2004).

the second outcome, taken directly from contemporaneous mother reports. The final two outcomes refer to persistent wheeze. The first of these, persistent wheeze between birth and 42 months, is equal to one if a child had wheezing with whistling on the chest in at least three out of the four observations over this period. The final outcome, labelled persistent wheeze between birth and 81 months, is equal to 1 if "persistent wheeze between birth and 42 months" is equal to one plus the mother reported either wheezing with whistling on the chest at 81 months or asthma at 81 months. As most wheezing conditions in childhood are likely to be multifactorial, these children may have elements of airway structural abnormalities as well as airway inflammation (with or without asthma).

While we do not observe asthma severity directly and the 'ever' and 'persistent' wheeze between birth and 81 months reflect different respiratory disorders (Baker et al., 1999), they may also be interpreted as proxies for less- and more-severe respiratory conditions respectively (Taussig et al., 2003). The other two outcomes capture different dimensions of child respiratory problems. Mother reported asthma at 81 months is the closest outcome to doctor-diagnosed asthma and we include it as much of the empirical literature relates to asthma. Persistent wheeze between birth and 42 months enables us to investigate whether socio-economic status (and the mediating factors) have a different impact on respiratory conditions during infancy compared to later in childhood. We also take persistent wheeze between birth and 42 months as less severe than persistent wheeze that continues on to age 81 months. The ranking – in terms of severity – of 'asthma at 81 months' and 'persistent wheeze between birth and 81 months' is less clear.

4.2 Socio-economic status

Our primary measure of socio-economic status is based on the study child's father's occupation, taken from mother responses at 32 weeks gestation. We employ a fivefold typology based on the standard occupational classification (Rose and Pevalin, 2003), grouping the study children into one of the following categories: professional, class I (11% of observations); intermediate, class II (35% of observations); skilled non-manual, class III NM (11% of observations); skilled manual, class III M (31% of observations) and partly- and unskilled, classes IV and V (12% of observations).[6] We treat this five category, ordinal variable as a continuous regressor in the multivariate analysis below. Whilst this measure of socio-economic status is not strictly continuous, and its use as a single variable masks possible non-linearities, it provides a reasonable summary

[6] We group classes IV and V together since there were relatively few observations in class V (approximately two and a half percent of the study children). Armed forces were coded as missing, affecting some twenty cases.

measure of socio-economic gradient and is preferred to a binary variable (capturing manual versus non-manual, for example) since it is more in keeping with the current policy focus on health inequalities across the social spectrum and not just the poor health of a socially disadvantaged minority (Graham, 2004).

We repeated all our analysis with an alternative indicator of SES, based on a measure of net family income when the study children are aged approximately three to four years old and report key similarities and differences in our results below.[7] Several considerations motivate our use of father's occupational status as the primary indicator of SES over the income measure. First, unlike the income measure, occupational status pre-dates the timing of all mediating factors, thereby mitigating possible problems of reverse causation (in this sense the income measure is more likely to be endogenous since it may be affected by the level of some of the mediating factors). Second, father's occupational class may represent a better proxy for long-term socio-economic status given the contemporaneous nature of the income measure. Third, a degree of imputation is necessary to construct the income measure and this detracts from its robustness.

4.3 The Mediating Factors

We explore the importance of eight possible sets of mediating factors that might give rise to the inequalities in childhood wheeze and asthma. These correspond to hypotheses advanced in the literature discussed above. The set of factors is not exhaustive but reflects data limitations: we cannot, for example, consider the role of traffic pollution, a potentially important mediating factor, owing to lack of appropriate data. Each factor is typically measured by two or three variables, which capture different aspects of the factor. Sets of dummy variables were used where appropriate to allow for non-linearities between the components of the factor and the respiratory outcomes. Further detail on variable construction as well as descriptive statistics for all variables used in the analysis is in Appendix Table A1.

The first mediating factor, exposure to other children in early childhood, aims to operationalise the hygiene hypothesis. It contains two groups of variables, measuring the number of siblings at 8 months and the type of day care used at 24 months. The second factor is child's diet. We distinguish three separate elements: the duration the child was breast fed, the amount of fruit the child consumed between birth and 38 months and the age the child was introduced to solids. We are not aware of any study to-date that has examined whether early

[7] Full details of the construction of this measure and the full set of results using these measures are available from the authors.

introduction of solids is associated with a greater risk of respiratory problems. Indirect evidence confirming a harmful link with other allergic symptoms (e.g. atopic dermatitis and eczema) suggests that age to weaning solids may play some part in the aetology of asthma.

The next two mediating factors relate to different aspects of air-born pollutants. The first is a measure of poor housing conditions, indicated by whether the study child lived in a house with serious damp, condensation or mould during the first four years of their life. The second factor is the intensity of (separately) pre- and post-natal maternal smoking.

The next three factors are less obviously mediating, in the sense that they represent factors that occur prior to, or at the time of, the child's birth. As explained above, we include these because they have been identified as important correlates of respiratory illness in children. The first of these is parental history of asthma, which is measured by a single variable indicating whether either parent ever had asthma and may capture genetic links. The second of these is poor child health, measured by two variables, the first denoting whether the child was born pre-term, the second, whether the child was born full-term but with low weight. The third is maternal age at the child's birth, which is measured by four dummy variables to allow for non-linearities.

The final factor, which we argue can be regarded as mediating, though it may also be a measure of low SES, is local deprivation. This is measured by the Index of Multiple Deprivation (IMD) in the local electoral ward[8] of the mother at the study child's birth. The IMD is a nationally published index of socio-economic deprivation, based on the income, housing circumstances, education and health of the population resident in the ward. Again, to allow for non-linearities, we divide this index into quartiles.

We do not include one factor that has been found in the literature to be associated with respiratory illness in children. This is parental education. It might be argued that more poorly educated mothers 'produce' child health less well than their more educated counterparts, so transmitting low SES into poorer respiratory outcomes. But for two reasons we not include it. First, education is arguably an alternative measure of SES rather than a mediating factor. And in terms of mediating factors, the link from education to SES and poorer outcomes may run from low education to low SES rather than vice versa, in which case it is not a potentially mediating factor in the way we are considering here.

[8] A ward is around 5000 persons.

4.4 Methods

A prerequisite of any proposed mediating factor is that it is correlated with SES. Thus, we first examine the correlation between each mediating factor and SES to establish whether each factor is in fact significantly associated with SES (and in the anticipated direction). We then examine the relationship between each respiratory outcome and SES using logistic regression. In such a regression, the coefficient on the continuous SES regressor[9] in a regression of a respiratory outcome on SES is an odds ratio. An odds ratio that is not statistically different from 1 indicates that there is no social gradient in the respiratory outcome. The difference between 1 and the coefficient indicates the magnitude of the social gradient. In our analysis, the larger the odds ratio, the more children from poor families suffer poorer health.

Results from a bivariate regression of an outcome on SES represent the unconditional social gradient and provide the benchmark to which we can compare the impact of each mediating factor. We start with this benchmark and then control for each mediating factor separately. The extent to which the odds ratio on the SES regressor moves closer to unity compared to the benchmark case indicates the extent to which the mediating factor accounts for the SES gradient. We can also assess whether each mediating factor has a direct impact (net of SES) on each respiratory outcome from the chi-squared statistic of the joint significance test of the variables used to measure each mediating factor. Finally, we control for all the mediating factors simultaneously, which allows us to take into account the correlation between the factors. The regressor on the SES variable then shows the impact of SES net of all the mediating factors: if it is significantly different from 1 then there is a social gradient that does not operate through the mediating factors.

In summary, there are three statistics of interest in the following analysis: (i) the correlation coefficients between SES and the respiratory outcomes (a significant statistic is a pre-requisite for a mediating factor to be described as such), (ii) the odds ratios from the SES regressor from logistic regressions of each respiratory outcome on SES plus one or all mediating factors (this provides an indication of the size of the social gradient), and (iii) the chi squared statistic from joint significance tests of the mediating factor on each respiratory outcome (an indication of whether the mediating factor(s) has a direct effect on the respiratory outcome, net of SES).

[9] SES is entered as a continuous variable with a value of 5 being the lowest SES.

5. Results

5.1 Socio-economic status and the respiratory outcomes: Is there a gradient?

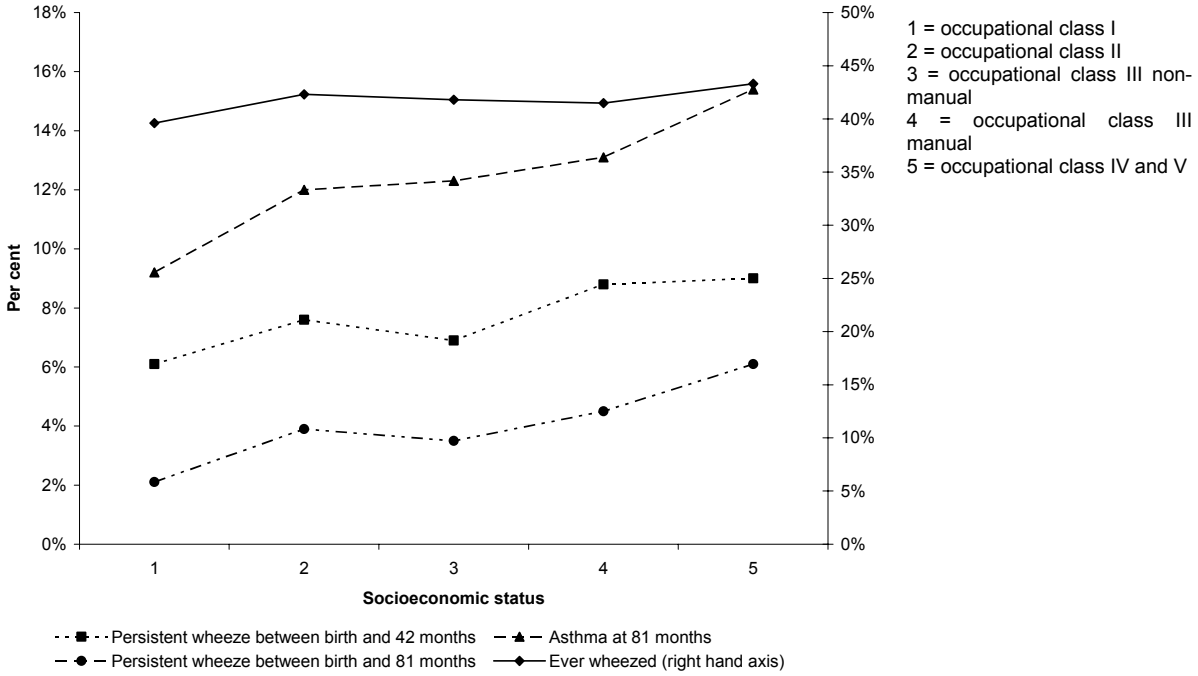
Over two-fifths of children had the mildest of symptoms i.e were reported to have wheezed between birth and 81 months ('ever wheezed'). As expected, the prevalence of the other three, more severe, outcomes are much lower. Just over one in ten children had asthma at 81 months, less than one in ten children had persistent wheeze between birth and 42 months and less than one in twenty children had persistent wheeze from birth to 81 months (see Appendix Table A1 for further detail). The prevalence of each respiratory outcome by occupational class is plotted in Figure 1 (the data are in Table A2). The graph shows that the prevalence of each respiratory condition is, broadly speaking, a decreasing function of occupational class. The prevalence of each outcome is lowest in occupational class 1 and highest in occupational classes 4 and 5. In absolute terms, asthma at 81 months increases most across SES. In relative terms, the gradient in Figure 1 is most pronounced for persistent wheeze between birth and 81 months. Compared to the probability of a child in occupational class 1, a child in occupational classes 4 and 5 is almost ten percent more likely to have ever wheezed, approximately one and a half times as likely to persistently wheeze between birth and 42 months and to have asthma at 81 months, and almost three times more likely to persistently wheeze between birth and 81 months. This indicates the greater social inequality that exists for the more-severe compared to less-severe respiratory conditions.

5.2 How much of the gradient is explained by the mediating factors?

Figure 1 shows the social gradient in childhood respiratory conditions, particularly the more severe ones. Here we examine the extent to which the eight possible mediating factors discussed in Section 4 may account for this gradient. To begin with, for a mediating factor to explain a part of any of the inequalities observed in Figure 1, it must be positively correlated with occupational class. Selected correlation coefficients between occupational class and our measures of the mediating factors are shown in Table 1 (the full list of correlation coefficients are reported in Table A1). Table 1 shows that six of the proposed mediating factors are unambiguously, significantly and positively (in the direction anticipated) correlated with occupational status, providing initial evidence that these factors may account for a part of the observed respiratory inequalities. In particular, occupational class is associated with poor housing conditions, both pre- and post-natal maternal smoking, poor child health (both low-birth weight and pre-term), poor child's diet (in particular, low duration of breastfeeding, including never been breast fed), low fruit in-take and both early

and late weaning to solids),[10] high levels of local deprivation and younger mothers.

Fig 1.The association between SES and respiratory conditions



The relationship between occupational status and exposure to other children is less straightforward. Low-occupation families are more likely to have more children but are more likely to use informal (largely home-based) day care arrangements (as opposed to day care and child minding facilities) where they are likely to be exposed to fewer children. Parental history of asthma is negatively correlated with occupational class suggesting that, if anything, it may help ameliorate rather than exacerbate respiratory inequalities (though the effect is likely to be modest given the small correlation coefficient).

Thus, it is possible that six of the proposed mediating factors may play some part in explaining the social gradients in Figure 1. Exposure to children may either increase or decrease the gradient, depending on the relative importance of number of siblings versus type of day care. It would appear that parental history of asthma has no potential role in explaining the observed respiratory inequalities given its negative association with occupational status.

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10 There appear to be non-linearities in the association between age of weaning to solids and occupational status with 'medium' values for age of weaning to solids (6 to 11 months) associated with high occupational class.

Table 1: Selected correlation coefficients between socioeconomic status and mediating factors

Variable	Correlation coefficient
Exposure to other children in infancy	
2+ siblings at 8 months	0.04***
Informal day care at 24 months	0.17***
Child's diet	
Never been breast fed	0.23***
Never observed with high fruit diet	0.13***
Age of weaning to solids	
<6	0.29***
6-11	-0.34***
>12	0.16***
Poor housing conditions	
Ever had serious damp, condensation or mould	0.63***
Maternal smoking	
High pre-birth[1]	0.02***
High post-birth[2]	-0.19***
Parental history of asthma	
Either parent ever had a history of asthma	-0.04***
Poor child health at birth	
Low birth weight (children born full term)	0.04
Maternal age at child's birth	
21 years or less	0.17***
Local deprivation	
Highest quartile of index of multiple deprivation at child birth	0.23***

Notes
1. Smoked 10+ cigarettes at 32 weeks gestation per day.
2. Smoked 10+ cigarettes per day

Evidence on the importance of the eight proposed mediating factors in accounting for respiratory inequalities is presented in Table 2. Logistic regressions are estimated for each outcome so the coefficients in the table are odds ratios from the occupational class regressor, treated as a continuous variable. An odds ratio above 1 denotes a negative social gradient. The first row of Table 1 reports the odds ratios for the occupational class variable with no controls. This is a test of the bivariate relationship shown in Figure 1. The remainder of the table shows the odds ratio on the occupational class variable controlling for each of the mediating factors separately, followed by the odds ratio controlling for all the mediating factors together. An odds ratio that is closer to 1 than the bivariate (benchmark) case indicates that the mediating factor in question is, at least in part, driving the social gradient. Chi-squared statistics from joint significance tests of the components of each mediating factor on each outcome are shown below the odds ratios for the occupational class variable.

The bivariate associations in the first row of table 2 confirm the patterns in Figure 1: there is evidence of social inequalities for all four respiratory outcomes as the odds ratios are all greater than 1. However, the SES inequality is not significant for the least severe outcome – the 'ever wheezed' outcome. The analysis below therefore concentrates on the other three outcomes, for which there is evidence of significant social inequality in the distribution of respiratory conditions as the unconditional odds ratio are significantly different from 1 for all these three outcomes. The odds ratio is highest for the persistent wheeze between birth and 81 months indicating that the raw gradient is steeper for the more severe outcome.

The next 8 rows of the table show that each mediating factor alone typically explains a relatively modest part of each respiratory inequality. In general, child's diet, local deprivation and maternal smoking are the mediating factors that tend to exert the greatest impact in reducing the odds ratios and so in explaining the SES gradient. For instance, the odds ratio for persistent wheeze between birth and 81 months decreases from 1.20 to 1.18 after controlling for either child's diet or maternal smoking, and to 1.17 after controlling for local deprivation. Child's diet is the only mediating factor that has an appreciable impact on the odds ratio for asthma at 81 months, which falls from 1.11 without controls to 1.07 after controlling for child's diet.[11] On the other hand, exposure to other children in infancy, parental history of asthma and poor child health at birth have no appreciable impact on the SES gradient for any of the respiratory outcomes. Indeed, as anticipated above, controlling for parental history of

[11] The impact of child's diet on asthma at 81 months is driven exclusively by duration of breast feeding.

asthma increases rather than decreases inequalities for some of the respiratory outcomes (asthma at 81 months and persistent wheeze between birth and 81 months).

Table 2: The association between occupational class and childhood respiratory conditions

	Respiratory outcome			
	Ever wheezed between birth & 81 months	**Persistent wheeze between birth & 42 months**	**Asthma at 81 months**	**Persistent wheeze between birth & 81 months**
No controls	1.02 (0.02)	1.10*** (0.03)	1.11*** (0.03)	1.20***(0.06)
With each mediating factor separately				
Exposure to other children in infancy	1.02 (0.02)	1.10*** (0.03)	1.10*** (0.03)	1.20*** (0.06)
	37.3***	83.6***	5.5	24.0***
Child's diet	1.00 (0.02)	1.06* (0.03)	1.07** (0.03)	1.18*** (0.06)
	13.2	19.4***	21.1***	10.9
Poor Housing Conditions	1.01 (0.02)	1.08** (0.03)	1.10*** (0.03)	1.19*** (0.06)
	4.6**	14.1***	5.3**	7.3***
Maternal Smoking	1.00 (0.02)	1.06* (0.03)	1.10***	1.18***
	22.8***	28.3***	3.7	3.1
Parental History of Asthma	1.01 (0.02)	1.09*** (0.03)	1.11*** (0.03)	1.20*** (0.06)
	2.2	0.0	0.8	0.3
Poor Child Health at Birth	1.01 (0.02)	1.09*** (0.03)	1.11*** (0.03)	1.20*** (0.06)
	6.0*	13.5***	3.2	5.6**
Mother's Age	1.01 (0.02)	1.07** (0.03)	1.10*** (0.03)	1.19*** (0.06)
	3.8	7.4**	4	2
Local Deprivation	1.01 (0.02)	1.07** (0.03)	1.10*** (0.03)	1.17*** (0.06)
	6.6*	6.4*	8.9**	6.5*
With all mediating factors	0.98 (0.02)	1.03 (0.04)	1.06* (0.03)	1.14** (0.06)
	197.2***	256.1***	170.6***	120.7***
Observations	6448	9056	7566	7192

Notes:
Standard errors in parantheses, X^2 statistic for significance of factor below coefficient and standard error.
Significance level *10% ** 5% *** 1%
For variable definitions, means and standard deviations see Table A1.

The final column of Table 2 shows that together the mediating factors account for the majority of the SES inequality in persistent wheeze between birth and 42 months, causing the odds ratio to fall from 1.10 to 1.03 and no longer be statistically different from 1. But they account for less than half of the inequalities for asthma at 81 months and persistent wheeze between birth and 81 months and the odds ratio for both these outcomes remain significant in the specification with all mediating factors.

These results show that while individually the mediating factors do not account for the gradient in a child having early symptoms only, collectively they do. On the other hand, while the mediating factors account for some of the SES gradient in the later (and more continuous) symptoms, the gradient still exists. This may reflect temporal consideration – several of the factors examined here relate to behaviour early in a child's life and their impact may fade over time, but the evidence is also consistent with the view that the factors underpinning the social gradient in respiratory inequalities may change as children age.

Finally, as the combined impact of the mediating factors is typically far greater than the impact of any one mediating factor, this suggests that the way in which SES affects childhood respiratory conditions is complex. There appears to be no single, dominant pathway through which SES has an effect, but a number of potentially important and inter-correlated pathways. Foremost among the mechanisms studied are child's diet, maternal smoking and local deprivation.[12]

5.3 Is there an independent impact of the mediating factors over and above socio-economic status?

The chi squared statistics in Table 2 indicate whether the particular mediating factor in question has a direct association with the respiratory outcome, net of any association that may exist via socio-economic status. The table shows that, in general, the measures of respiratory health taken at a later age – asthma at 81 months and persistent wheeze between birth and 81 months – have fewer significant direct associations with the mediating factors than outcomes occurring earlier in life. This may indicate that the risk factors for respiratory conditions in very early childhood may be different from those for subsequent respiratory conditions. It is also possible that the impact of factors that occur early in the child's life (e.g. poor health at birth) fades over time.

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[12] Exposure to children during infancy slightly increases the gradient for 'ever-wheezed' but reduces the gradient for other outcomes. This pattern of results may reflect temporal considerations in construction of the outcome variables. Investigations (not reported) show that exposure to children during childhood is positively associated with wheeze at 6 months but negatively associated with wheeze at 81 months.

Poor housing conditions, local deprivation and parental history of asthma are the only mediating factors that have a significant direct impact on all four respiratory outcomes (net of their association with occupational class). Local deprivation was also identified as an important route through which socio-economic status had an effect on the respiratory outcomes (as noted above). So local deprivation is both associated with SES, which is associated with poorer respiratory outcomes, and has itself a direct independent effect. This is also true for child's diet, which has an appreciable indirect impact through its association with occupational class and a direct impact for persistent wheeze between birth and 42 months and asthma at 81 months.[13]

5.4 Income as a measure of SES

We repeat the preceding analysis using an alternative indicator of socio-economic status based on family income when the study child is aged approximately three to four years (see Annex 1 for definitions and results). The bivariate results using the income measure point to significant inequalities in all four respiratory outcomes.[14] As above, the income-based measure of SES also reveals the greatest inequalities for persistent wheeze between birth and 81 months, underlining the existence of greatest inequality for the more-severe respiratory condition.

Irrespective of the choice of indicator for socio-economic status, controlling for the mediating factors individually explains a relatively modest part of the respiratory inequalities when income is used as the measure of SES. Maternal smoking and local deprivation once more tend to be relatively important mediating factors as are poor housing conditions. There is less clear evidence for an impact of diet as a mediating factor if income is used as a measure of SES rather than class.

Again, controlling for all mediating factors simultaneously explains much more of the respiratory inequalities than controlling for any of the mediating factors separately. When income is used the odds ratio for the persistent wheeze between birth and 81 months outcome is no longer significant once all mediating factors are controlled for, though the point estimate is still considerably different from 1 (it is imprecisely estimated).

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13 As with the indirect effect of child's diet, the direct effect appears to be dominated by duration of breast feeding. The joint significance of fruit intake and age of weaning to solids is only just statistically significant at conventional levels.

14 This includes the 'ever wheezed outcome', where the odds ratio was insignificant using occupational class.

6. Conclusions

Recent evidence has drawn attention to the existence of socio-economic (SES) inequalities in childhood wheeze and asthma (especially severe asthma). This paper seeks to identify some of the factors that are associated with this social gradient in childhood respiratory conditions. We use data from a very rich birth cohort study, containing data on over 12,000 births in England in 1991/2. The data contains information on child respiratory health up to age 8 as well as parental SES and behaviours pre- and post-birth. Using occupational class as a measure of socio-economic status, we find significant inequalities in three respiratory conditions: persistent wheeze between birth and 42 months, asthma at 81 months and persistent wheeze between birth and 81 months, though not in a fourth, milder, respiratory condition (ever-wheezed between birth and 81 months). Our results thus support earlier findings suggesting that SES is more important for more-severe compared to less-severe respiratory conditions.

We then investigate the extent to which the social gradients in childhood respiratory conditions can be explained by eight potential mediating factors, all of which have been identified as significant determinants of asthma or wheeze in childhood and several of which are potentially alterable by public policy. These are exposure to other children in infancy, child's diet, poor housing conditions, maternal smoking, parental history of asthma, poor child health at birth, maternal age at child's birth and local deprivation.

We find that each of these mediating factors alone typically explains a relatively modest part of each respiratory inequality. In general, child's diet, local deprivation and maternal smoking are the mediating factors that tend to account for most inequality. But taken together, the mediating factors account for the majority of the inequality in persistent wheeze between birth and 42 months, though they explain less than half of the inequalities for asthma at 81 months and persistent wheeze between birth and 81 months. So these factors appear to be more important in explaining SES inequalities in early respiratory conditions, though not in conditions that persist into middle childhood. This may reflect temporal consideration since many of the factors examined are measured early in a child's life and their impact may fade over time. However, the evidence is also consistent with the view that the factors underpinning social gradients in respiratory inequalities may change as children age.

In addition, given that the combined impact of the mediating factors is typically far greater than the impact of any one mediating factor, this suggests that the way in which SES affects childhood respiratory conditions is complex. There appears to be no single, dominant pathway through which SES has an effect, but a number of potentially important and inter-correlated pathways.

Finally, our results suggest that only targeting the factors we have examined here will not eliminate SES inequalities in childhood respiratory health. On the other hand, one consequence of policies to improve the use and duration of breast feeding and to reduce maternal smoking may be to, at least modestly, reduce the extent of respiratory health inequalities in children. these mediating factors could be targeted more sharply in public health programmes. However, the case as a whole illustrates the difficulty of acting in direct ways to reduce health inequalities and the importance of the wider social inequality agenda.

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Annex Tables

Table A1: Descriptive statistics of variables used in the analysis

Variable	Mean	Standard deviation	Correlation coefficient with SES	Number of Observations
Respiratory outcomes				
Ever wheezed between birth & 81 months	0.42	0.49	0.01	6448
Persistent wheeze between birth & 42 months	0.08	0.27	0.03***	9056
Asthma at 81 months	0.12	0.33	0.04***	7566
Persistent wheeze between birth & 81 months	0.04	0.20	0.05***	7192
Socioeconomic status				
Father's occupational status[1]	2.98	1.26	1.00	10453
Exposure to other children in infancy				
Number of siblings at 8 months				
1	0.42	0.49	-0.02***	9581
2	0.38	0.49	-0.01	9581
2+	0.19	0.39	-0.41***	9581
Type of day care at 24 mths				
Day care centre	0.10	0.31	-0.11***	8960
Child minder	0.14	0.34	-0.44***	8960
Other	0.78	0.42	-0.17***	8960
Child's diet				
Duration of breast feeding				
Never	0.24	0.43	0.22***	9412
< 3mths	0.23	0.42	0.09***	9412
3-5 mths	0.17	0.37	-0.04***	9412
>6 mths	0.36	0.48	-0.25***	9412
Number of times observed with high fruit diet				
Never	0.44	0.50	0.13***	6907
1	0.27	0.44	-0.01***	6907
2	0.17	0.38	-0.64***	6907
3+	0.12	0.32	-0.11***	6907
Age of weaning to solids				
<6	0.10	0.30	0.03***	9277
6 to 11	0.83	0.38	-0.03***	9277
>12	0.07	0.25	0.02***	9277

Variable	Mean	Standard deviation	Correlation coefficient with SES	Number of Observations
Poor Housing Conditions				
Never had serious damp, condensation or mould	0.97	0.17	-0.03***	4585
Ever has serious damp, condensation or mould	0.03	0.17	-0.16***	4585
Maternal smoking				
Pre-natal maternal smoking				
Smoked 10+ cigarettes at 32 weeks gestation	0.17	0.38	0.06***	10453
Number of times observed smoking 10+ cigarettes per day[2]				
None	0.77	0.42	0.20***	7563
1-2	0.09	0.28	0.06***	7563
3	0.15	0.35	0.18***	7563
Parental History of Asthma				
Neither parent ever had a history of asthma	0.77	0.42	0.04***	7324
Either parent ever had a history of asthma	0.23	0.42	-0.04***	7324
Poor child health at birth				
Full term and not low birth weight	0.81	0.39	-0.04***	10453
Pre term (less than 37 wks gestation)	0.10	0.30	0.02**	10453
Low birth weight (children born full term)	0.09	0.28	0.04***	10453
Maternal age at Child's birth				
21 or less	0.06	0.24	0.17***	10453
22-25	0.19	0.39	0.18***	10453
26-35	0.67	0.47	0.18***	10453
36 or more	0.08	0.26	-0.09***	10453
Local deprivation				
Index of multiple deprivation at child birth				
Lowest quartile	0.21	0.40	-0.24***	9540
Second lowest quartile	0.19	0.39	-0.08***	9540
Second highest quartile	0.22	0.41	-0.04***	9540
Highest quartile	0.39	0.49	-0.23***	9540

Notes:
1. At 32 weeks gestation
2. Maximum number of observations = 3
Significance level *10% ** 5% *** 1%

Table A2: The association between socioeconomic status and childhood respiratory conditions

	Respiratory outcome (per cent)			
	Ever wheezed	**Persistent wheeze between birth and 42 months**	**Asthma at 81 months**	**Persistent wheeze between birth and 81 months**
Occupational class				
1	39.6	6.1	9.2	2.1
2	42.3	7.6	12.0	3.9
3 non - manual	41.8	6.9	12.3	3.5
3 manual	41.5	8.8	13.1	4.5
4 & 5	43.3	9.0	15.4	6.1
All	41.8	7.8	12.4	4.0
Observations	6448	9056	7566	7192

Using Income instead of class as a measure of SES

Table A3 contains results for the association between socioeconomic status and childhood respiratory conditions, where income is used as the indicator of socioeconomic status. The measure of income used in the analysis is log mean equivalised net family income. This is constructed from banded ALSPAC income data available from separate observations when the study child is aged 33 and 47 months. Exact amounts were imputed using external data - the same as that used to produce the official UK income distribution statistics (HBAI 2005). The income amounts were then equivalised using the modified OECD equivalence scale. The variable used in the analysis was the log of the mean equivalised income averaged from responses at 33 and 47 months.

Table A3: The association between income and respiratory symptoms in children

Variable	Outcome			
	Ever wheezed between birth & 81 months	**Persistent wheeze between birth & 42 months**	**Asthma at 81 months**	**Persistent wheeze between birth & 81 months**
Without controls	0.86***	0.74***	0.75***	0.72***
	0.04	0.05	0.05	0.07
Each mediating factor separately				
Exposure to other children in infancy	0.85*** (0.04)	0.75*** (0.05)	0.77*** (0.05)	0.73*** (0.07)
	33.69***	55.58***	2.64	17.80***
Child's diet	0.87*** (0.04)	0.76*** (0.05)	0.76*** (0.05)	0.73*** (0.07)
	9.35	9.92	4.93	7.70
Poor Housing Conditions	0.88*** (0.04)	0.77*** (0.05)	0.76** (0.05)	0.74** (0.07)
	7.48**	0.78***	0.76***	0.74***
Maternal Smoking	0.90** (0.04)	0.78*** (0.05)	0.76*** (0.05)	0.74*** (0.07)
	15.64***	12.23***	2.39	1.34
Parental History of Asthma	0.88*** (0.04)	0.74*** (0.05)	0.74*** (0.05)	0.72*** (0.07)
	3.30*	0.41	0.70	0.49
Poor child health at birth	0.86*** (0.04)	0.74*** (0.05)	0.75*** (0.05)	0.72*** (0.07)
	7.28**	26.10***	2.01	12.49***
Mother's age	0.87*** (0.04)	0.74*** (0.05)	0.76*** (0.05)	0.72*** (0.07)
	4.54	2.49	1.40	1.25
Local deprivation	0.87*** (0.04)	0.76*** (0.05)	0.76*** (0.05)	0.75*** (0.08)
	4.94*	4.15	7.13*	5.92
With all mediating factors	0.95 (0.05)	0.87* (0.07)	0.81*** (0.06)	0.82 (0.10)
	195.02***	200.87***	135.88***	119.55***

Notes:

Coefficients appear first followed by standard errors and then chi2 statistics.

Significance level *10% ** 5% *** 1%