Relationship between household income and childhood respiratory health: an econometric perspective

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Abstract

Growing empirical evidence on the association between household income and adverse child health outcomes has generated mixed results with some North-American studies showing a significant inverse relationship and some British studies identifying a much weaker association. We use data from the rich UK Millennium Cohort Study data set and check the robustness of these recent findings by focusing on the impact of household income on adverse childhood respiratory outcomes (i.e. asthma and wheezing). We attempt to establish whether the association between household income and child health is causal, which is crucial for guiding and motivating policy makers' decisions aimed at improving child health outcomes. We use the instrumental variable approach, which enables us to draw causal inferences by eliminating biases associated with unobservable factors, measurement errors and simultaneity between income and child health outcomes. Our results show that household income has an independent effect on child health. We argue that our evidence should motivate and support government fiscal policies aimed at reducing health inequalities in childhood.

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1. Introduction

Recent government reports in the United Kingdom (see, for example, HM Budget report 2006) and other countries, such as the United States, Canada and Australia (see Public Health Agency of Canada, 2000; US Department of Health and Human Services, 2004; National Public Health Partnership, 2004), have called for the implementation of policies and programmes to reduce socioeconomic inequalities in adverse child health outcomes. Since 1997, the British Government has supported a series of policy interventions to financially assist low income families with children (see HM Pre Budget Report, 1999). This has been based, in part, on the assumption that income supplements, such as universal benefits, means-tested benefits and tax credits payable to low income families with children, are an effective way to improve these children's health, education and future labour markets outcomes.

A number of observational studies conducted in North America have shown a positive association between household income and child health, with some suggestion of a steepening of the income-health gradient during the later childhood years. (see Case et al., 2002; Currie and Stabile, 2003). This is contrasted with health econometric studies from the UK that point to a much weaker income effect upon health throughout the childhood years (see Burgess et al., 2004; Currie et al., 2007; Propper et al., 2007). A statistical association between income and child health outcomes might not necessarily imply causation. There might, in fact, be a variety of mechanisms underlying the income-child health relationship, and it is important to delineate these in order to inform appropriate responses by policy makers. It is crucial, therefore, for guiding and motivating policy makers' decisions, to try to establish not only the magnitude but also the direction of the association between household income and child health outcomes and to determine the extent to which this might be causal.

The conflicting evidence in this area and the related policy concerns motivate a further investigation into the extent to which the relationship between household income and child health might be spurious and driven by unobserved heterogeneity rather than representing a causal effect. Our study attempts to disentangle this nexus in the UK context using the instrumental variables approach, which will enable us to draw causal inferences by eliminating biases associated with unobservable factors, measurement errors and simultaneity between income and child health outcomes.

Our focus is upon adverse childhood respiratory outcomes, principally preschool asthma and wheezing, and is justified as follows. Firstly, respiratory illnesses are chronic conditions that require long-term care and, consequently, have a significant financial impact not only upon the national health system, but also upon families and upon society as a whole. Secondly, existing evidence on socioeconomic patterning in childhood asthma and wheezing is contradictory and is derived mainly from empirical research outside of economics. Thirdly, to our knowledge, no comprehensive econometric examination of the causal relationship between socioeconomic factors and preschool asthma and wheezing using UK data has been carried out to date.

We anticipate that our results will increase understanding of the mechanisms underlying the relationship between income and child health. A significant inverse relationship between household income and adverse childhood respiratory outcomes should inform government health and fiscal policy in this area and help to identify areas where government efforts on tackling child health inequalities might be targeted.

The paper is organised as follows. Section 2 reviews the existing literature and empirical work in this area. Section 3 describes our data and presents some preliminary statistics. The

estimation strategy is explained in section 4, whilst section 5 summarises and discusses our results. Finally, section 6 draws the conclusions of the paper, providing policy recommendations and suggestions for further research.

2. Previous literature and empirical evidence

The relationship between socioeconomic status and health and well-being has long been of interest to social scientists, particularly economists. A number of empirical studies (see Ettner, 1996; Deaton and Paxson, 1999) have shown that low socioeconomic status may result in poor health outcomes, in turn resulting in calls for policies that eliminate economically-driven health inequalities.

Whilst the existence of a positive association between socioeconomic status and health outcomes in adulthood is well-documented (see, for example, Adler et al., 1994), the underlying mechanisms are not yet completely understood (see Deaton and Paxson, 1998; Chase, 2002). Recently, attention has been given to the early stages of the relationship, because it has been recognised that the income-health gradient in adulthood has clear antecedents in childhood (see Case et al., 2002, for the US; Currie and Stabile, 2003, for Canada; Burgess et al., 2004 and Propper et al., 2007, for the UK). Children with poor health status are more likely to develop into adults with poor health status and experience lower educational attainment and worse labour market outcomes (see Currie and Hyson, 1999; Currie, 2004; Case et al., 2005).

Early economic studies on the relationship between socioeconomic factors and child outcomes (see Becker and Tomes, 1986, for a review) posited the determinants of childhood attainments within the theory of family behaviour. In the work by Becker and Tomes (1986), 'investment theory' relies on the belief that children's outcomes are a consequence of biological and other types of endowments that the parents transmit to their offspring, in combination with the investments that parents make in them. In particular, parents are concerned with their children's future financial wellbeing and adopt utility-maximising behaviours, such as investing in the child's human capital, to achieve this objective. In a context of perfectly efficient capital markets, given that parents can borrow against the future earnings of their children, household income should not have a substantial impact on the offspring's outcomes. In the presence of inefficient capital markets, however, deprived parents would be unable to invest optimally in their child, which implies a causal link between parental income and the child's future attainments.

Alternative pathways whereby household income can influence children's outcomes have been proposed by theories outside of economics, such as, for example, 'parental stress theory' and 'role–model theory', which are two versions of the same 'good-parent theory' (see Mayer, 1997, for an extensive review). The main idea underpinning this group of theories is that low income decreases parents' aptitude to be 'good' parents in the sense that financial strain brings with it a deterioration of the value of their non-monetary investments, such as the quality of their time inputs, interactions with their children or their future expectations of them.

The challenge facing researchers when trying to investigate empirically whether income is a relevant determinant of children's health outcomes is the difficulty of implementing appropriate methodologies that disentangle income effects from other factors, such as genetic and environmental characteristics (see Blow et al., 2005). The scarce but growing economic empirical evidence on the relationship between household income and children's health outcomes can be classified into two main groups. On the one hand, there are a number of

observational studies conducted in North America, which provide consistent empirical support for the view that income is a relevant determinant of children's health and its influence increases as children age (see Case et al., 2002; Currie and Stabile, 2003). On the other hand, there is a growing stream of empirical literature, mainly from the UK, which shows that the income-child health gradient is weak and diminishes with age and, in some cases, seems not to hold at all (see Burgess et al., 2004; Propper et al., 2007; Currie et al., 2007).

A seminal paper belonging to the first stream of empirical literature is the comprehensive US study carried out by Case et al. (2002). Using several large, nationally representative US data sets[1], the authors investigate the relationship between household income and a wide range of child health outcomes, including chronic conditions, and attempt to explain the mechanisms which underlie the relationship. Their findings show a robust positive impact of household income on children's health with the income–health gradient steepening as children age[2]. The authors rule out, with a high degree of confidence, the hypothesis that causation might run from health to income rather than from income to health as, contrary to what happens in adulthood, it is argued that children do not directly[3] contribute to household economic resources. The concern that the relationship between income and health might be spurious is addressed by the inclusion in the models of 'third factors', such as health at birth, parental health, parental child-health behaviours, genetic ties and the presence of health insurance, which might account for part of the gradient between income and health in childhood. The addition of control variables to account for these factors does not significantly change the basic result that lower household income is associated with poorer health throughout childhood.

Similar results are replicated in the Canadian context by Currie and Stabile (2003). These investigators use panel data on Canadian children from the National Longitudinal Survey of Children and Youth (NLSCY)[4] to study the relationship between socioeconomic status (SES), childhood health measures and age and attempt to disentangle the mechanisms that appear to result in a steepening socioeconomic gradient with age. Despite the fact that Canada has universal health insurance coverage, as opposed to the largely private health insurance system in the United States, the authors find that the socioeconomic status-health association holds for Canadian children. They also show that the strengthening of the socioeconomic gradient with age is mainly due to higher exposure of low income children to adverse health shocks rather than to a slower recovery rate from adverse health conditions.

A study from the UK that presents results consistent with the North American evidence is that of Case et al. (2005). Despite the fact that the main focus of the work is on quantifying the enduring consequences of child health and economic conditions on health, employment and socioeconomic status in adulthood, the general findings reinforce the view that the

[1] The data sets are: the 1986-1995 National Health Interview Survey (NHIS), which is an US annual cross-sectional survey; the 1988 child health supplement to the NHIS (NHIS-CH); the Panel Study of Income Dynamics with its associated 1997 Child Development Supplement (PSID-CDS) and, finally, the Third National Health and Nutrition Examination Survey (NHANES).

[2] Chen et al. (2006), using the same datasets, arrive at different conclusions about the relationship between household income and child health, as they find that the gradient does not change with age. See also the two commentaries on the issue written by Case et al. (2007) and Chen et al. (2007).

[3] Case et al. (2002) consider the possibility that chronic illnesses during childhood could indirectly affect household income by reducing parental labour supply, which in turn could explain the strong correlation between low income and poor health during childhood. Their additional analyses in this direction, however, do not provide support for the latter interpretation.

[4] The authors restrict their analyses to the sample of children, aged 0-11, who were surveyed in the three waves of 1994, 1996 and 1998.

socioeconomic gradient in child health increases with age. The causal mechanisms underlying the association, however, remain uncertain.

The study by Case et al. (2005) emerges as an exception within the UK literature as the bulk of the empirical evidence from the UK reveals a weak effect of income on child health. A recent study by Currie et al. (2007), for example, which analysed data on a sample of 13,000 children and their parents drawn from the pooled 1997-2002 Health Surveys for England, found that although a positive association between household income and child health exists for subjective general health measures, the gradient vanishes when objective measure of health, obtained by qualified nurses, are used. Furthermore, the slope of the income-health gradient, whenever it is present, is in general small and does not increase as children age. The authors argue that this proves that the NHS in England has attained its objective of assuring equal access to health care for every child. On the whole, the findings of this study indicate that household economic resources are not significant determinants of the health of English children. Rather, nutrition and family lifestyle choices are identified as important channels for targeting policy interventions for improving children's health.

Another recent British study by Propper et al. (2007) (see also Burgess et al. 2004) identifies a contemporaneous association between household income and child health among a UK geographically-defined cohort of children[5], but identifies no evidence of a strengthening of the income gradient as children age. The authors note that permanent income, measured as average income over the available time span, matters in the relationship, rather than the level of current income. Furthermore, when the authors investigate the potential mechanisms through which the income-child health nexus operates, and control for possible factors that might explain the income-health association in a sample of children aged 7, they find no association at all. In other words, it seems that income does not have an independent effect on the child health outcomes that were analysed[6]. On the contrary, maternal mental health appears to play an important role in explaining child health and in lowering the estimated coefficient for income to zero. The policy implications of this study point towards interventions for improving maternal health as a means of reducing discrepancies in health between children. The authors also acknowledge that their findings might be influenced by unobserved heterogeneity that they were unable to adequately address as a result of the nature of their data.

Finally, the relationship between parental socioeconomic background and child health was investigated by Doyle et al. (2005) using a sample of pooled data from the 1997-2002 Health Surveys of England. Taking into account the endogeneity of both household income and parental education through an instrumental variable approach[7], the authors conclude that the observed association between income and child health is spurious and not driven by a causal effect of financial resources on the health status of children. They therefore throw further doubt on the North American empirical studies in the area[8].

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[5] The authors use data drawn from the Avon Longitudinal Study of Parents and Children (ALSPAC), which is a cohort study of children born in the former county of Avon in England in the early 1990s.

[6] The only exception is body mass index (BMI) which always has a significant and inverse relationship with permanent income independent from the inclusion or exclusion of other factors.

[7] Parental education is instrumented by using the raising of school leaving age (RoSLA), entered as a dummy variable, parental birth month dummies and grandparental smoking dummies. Predicted parental education coefficients from the first stage schooling equations are used as suitable instruments for household income.

[8] In the context of the relationship between household income and child behavioural and cognitive outcomes, it is worth noting the work carried out by Dooley and Stewart (2007) from Canada, and Blau (1999) from the United

3. Data sources and variable definitions

We use data from the first and second surveys of the Millennium Cohort Study (MCS). The MCS is the fourth of the UK's national longitudinal birth cohort studies that are following large samples of individuals through the course of their lives. Its primary objective was to reflect on the circumstances of children in the UK at the start of the new millennium. Its first sweep was carried out between September 2000 and January 2002[9]. The sample design allowed for disproportionate representation of families living in areas of child poverty, in the smaller countries of the UK and in areas with high ethnic minority populations in England. Parents of the babies were recruited from child benefit records after being given a chance to opt out by post, telephone or on the doorstep. The survey included 18,553 families, which after allowance for 246 sets of twins and 10 sets of triplets, amounted to 18,819 children in the cohort. A total of 18,553 main interviews were given, almost entirely by mothers, when the children were aged nine months (survey 1). The first survey (MCS1) recorded the circumstances of pregnancy and birth, as well as those of the all-important early months of life, and the social and economic background of the family into which the children have been born. Further details about the MCS, its origins, objectives, sampling procedures, content, methodology and response rates, are reported elsewhere (Plewis, 2004; Plewis and Ketende, 2006; Hansen, 2006). The second survey (MCS2) was carried out over the period 2003-2005[10] when the children were aged 3. Due to attrition, the size of the sample in the second survey dropped by approximately 16% to 15,808[11].

The sample used in our analyses was restricted by a number of factors. Firstly, in order to make use of the panel dimension of the data, we consider only those families who participated in both surveys. This reduced the size of the sample to 14,898 households and 14,898 cohort children, excluding the twins and triplets in the sample. Secondly, we restricted our analyses to those families in which the main respondent to the interviews was the natural (biological) mother of the cohort child and the respondent to the partner's interviews was the natural (biological) father. This accounted for a further reduction to 11,094 households and 11,094 cohort children. Finally, when we conducted our estimations, we dropped any observation for which data was missing on any variable of interest, resulting in a final sample of 7,659 cohort households and 7,659 cohort children. The latter procedure is usually referred to as 'listwise deletion'. We also conducted a regression-based data imputation that allowed us to run the analysis on the whole sample of 11,094 cohort children and to check, in this way, the robustness of the results obtained in the restricted sample of 7,659 cohort children[12].

Table 1 defines the variables used in the analyses and describes the summary statistics for the sample of 7,659 cohort families and children. Our measures of adverse child health outcomes are based on the answers given by the main respondent[13] to specific health questions included in

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States. Both studies, after analysing the relevant evidence, express scepticism on the use of increasing cash transfers as a key mechanism for substantially improving childhood outcomes.

[9] More specifically, in England and Wales children where eligible if they were born between 1 September 2000 and 31 August 2001, while in Scotland and Northern Ireland they had to be born between 23 November 2000 and 11 January 2002.

[10] Data were collected between September 2003 and April 2005 for England and Wales and between December 2003 and April 2005 for Scotland and Northern Ireland.

[11] Follow-up interviews have been carried out at age 5 (data available in 2008) and are currently taking place for children aged 7. The next follow-up is likely to be when children are aged 10/11.

[12] Results are available from the authors on request.

[13] In our sample the main respondent was the natural (biological) mother of the child.

the second survey[14]. We generated dichotomous variables that took the value of one if the child suffered from the specific respiratory disease of interest (asthma or wheezing) and zero otherwise. 839 children (10%) were reported suffering from asthma and 2,167 (28%) were reported suffering from wheezing[15].

As part of the main interview conducted during the first MCS survey, main respondents were asked a series of questions about their personal and household income from employment, from government sources and from other sources. They were then asked to locate total take-home household income from all these earnings and sources after tax and other deductions on one of two sets of eighteen annual household income bands; one for couple households and one for lone parent households. The two sets of income bands were constructed to show approximately equivalent purchasing power when reading from one scale to another; couple household bands approximating at 1.6 times the equivalent band for a lone parent household. The midpoint for the selected income band was calculated and then equivalised for each household using the formula recommended by Gravelle and Sutton (2003) to reflect household composition (equivalised income = household income / $\sqrt{}$ (adults + 0.5 x children)). We then took the logarithm of the equivalised household income. Household income, without equivalisation, averaged £ 29,000 in our sample.

Other indicators of socio-economic status were maternal and paternal education, the age of the mother at the time of birth of the cohort child, lone parent status and housing tenure. The latter can be regarded not only as a measure of material deprivation, since living in a rented accommodation is considered an indicator of low income, but also as an indicator of environmental exposures of children to dumps, house dust mites or insects that might be associated with respiratory diseases (see Baker et al., 1998; Hancox, et al., 2004).

Additional control variables were grouped into the following categories: child characteristics, parental health risk factors, mother's child health-related behaviours and environmental exposures. All of them had been identified by previous epidemiological and health economic analyses (see Oddy et al., 1999; Sherriff at al., 2001; Hancox, et al., 2004; Eder et al. 2006) as significantly associated with asthma and wheezing in children. Child characteristics included birthweight, gestational age at birth, gender, ethnicity and whether the cohort child had older siblings. The latter was taken as indicative of the parity of the mother. Maternal and paternal histories of asthma represented the parental risk factors expected to strongly impact on the child's respiratory health. Behaviours of the mother that were associated with the health of the child included maternal smoking during pregnancy, which is a measure of prenatal exposure to tobacco smoke, and the duration of breast feeding. Both measures were modelled as categorical variables. Finally, two potential environmental risk factors were identified in the form of ownership of domestic pets throughout the child's life up to 3 years of age and postnatal exposures to tobacco smoke, measured by the presence of people smoking in the same room as the child both at age 9 months and at 3 years.

[14] The second survey took place when the child was approximately three years of age but the questions also referred to past health conditions.

[15] We keep the two health outcomes, namely asthma and wheezing, separate as various studies have shown that there are considerable differences among doctors, countries and areas of residence and also over time in the extent to which the term asthma has been used for indicating wheezing illnesses and, in fact, not all wheezing is asthma-related (see Hancox et al., 2004, and ONS, 2004, and the papers quoted therein for a discussion of these medical definitions).

As discussed earlier, the impact of income on adverse child health outcomes might be plagued by endogeneity problems. We deal with the potential endogeneity of income by using grandparental income support and paternal grandfather's socioeconomic status as instruments. Good instruments have to be correlated to the endogenous variable and uncorrelated to the outcome. In the spirit of the work by Ettner (1996), the rationale behind our choice of instruments lies in the consideration that household income is influenced by factors that affect the respondents' wage rate and non-earnings income and these, in turn, are likely not to have a direct link with child health outcomes, but only an indirect one through the income channel. Non-earnings household income might be received as income support from grandparents. Grandparental income support was measured in the form of essentials bought for the child (i.e. food, clothes, nappies), payments for other household costs, gifts and extras bought for the baby, payment for childcare[16] and so on. Given the limits imposed by the information available in the MCS datasets, we also identified paternal grandfather's socioeconomic status as a suitable proxy for grandparents' education, which can be considered as a factor affecting household income[17] through the trans-generational transmission of education and social status, which has, in turn, an effect on the respondents' wage rate. It was considered that these variables affect childhood respiratory outcomes through the income channel. If the impact of household income remains significant, although possibly different in magnitude, after controlling for all these factors, we can conclude, with a certain degree of confidence, that we have found support for an independent income effect on child health.

4. Estimation strategy

For comparison purposes, the relationship between household income and child respiratory health is first analysed by assuming the exogeneity of income. Our general model specification for the generic household *i* is of the following form:

$$H_i^c = \alpha + \beta Y_i + \boldsymbol{\delta}\mathbf{X}_i + \varepsilon_i^c \qquad (1)$$

The dependent variable H^c is a dichotomous variable defining child health status, namely whether the child suffers from asthma or whether the child suffers from wheezing illnesses. Equation (1) is in fact estimated separately for each of the two adverse health outcomes that are

[16] An indirect way of influencing non-earnings income might also be given by the monetary savings that the family can accumulate if grandparents offer to take care of the child for free. The counter-argument, in this case, might be that children cared for by members of the family might have a better health status for a range of health outcomes, as they are better nurtured. This would violate the orthogonality condition required for instruments. At this stage we ignore this variable, but it could be an interesting line of investigation to follow, especially in the presence of more informative data in this regard. Analogously, for families whose children are cared for by private or public nurseries, a potential instrument for income could be the average price of child care. Once again, it could be argued that the higher the price the better is the way in which children are looked after and this might have an influence on their health status. Unfortunately the MCS dataset only included information about the costs of childcare in the second survey and, additionally, does not contain information which allows generating an accurate proxy for the average price of childcare itself.

[17] Parents' educational status and socioeconomic status were also considered as potential instruments. Although they were strongly correlated with household income, they did not satisfy the property of orthogonality to child health outcomes (see section 5 for an exhaustive explanation of the statistical requirements that have to be satisfied by instrumental variables).

the focus of our analyses. The model is estimated as an ordinary probit, with child health considered as a function of the logarithm of equivalised household income, Y_i , and a vector of other relevant controls, $\mathbf{X}_i$, which includes all of the explanatory variables described in section 4, namely child characteristics, parental health risk factors, mother's child-health-related behaviours and environmental exposures. ε_i^c is defined as the error term.

Throughout the paper we present results for three different model specifications for each of the two adverse respiratory outcomes (see Tables 2 to 4). Model specification 1 includes only household income and generates an estimate of the crude correlation between the logarithm of equivalised household income and child respiratory health (unadjusted model). Model specification 2 is the estimation emerging from an accurate model selection[18] and therefore represents the preferred model (restricted or partially adjusted model). Model specification 3 is the general model specification that includes household income as well as all of the other covariates described above (completely adjusted model). The regression analyses incorporated sampling weights that reflected the probability of a household being selected within one of the MCS's primary sampling units (398 UK electoral wards).

The main coefficient of interest is β, which expresses the impact of household income on the probability that the child suffers from the specific respiratory disease. The probit model provides unbiased estimates of β and the other coefficients in model (1) as long as the error term ε_i^c is not correlated with the independent variables. There are various reasons that imply a violation of the above condition and this general problem is referred to as endogeneity. We are primarily concerned with the endogeneity of income, which might arise because of reverse causality and unobserved heterogeneity. In the child health context, reverse causality might be an issue to the extent to which a sick child might affect household income by reducing parents' labour supply. Since asthma and wheezing are chronic health conditions that can constrain the child's daily activities, a reduction of mothers' labour supply, especially in the early years of life, seems a plausible hypothesis to test. We partially address this issue, however, from the outset by using past income (as estimated in MCS1) as opposed to current income in our estimation strategy[19]. More importantly, endogeneity might be due to unobservable factors, e.g. unobservable parental characteristics, which are contained in the disturbance term ε_i^c and simultaneously affect both household income and child health outcomes. The omission of these variables will result in spurious correlation and biased estimates of the causal effect of income on child health. Both causes of endogeneity are expected to generate upward biased estimates of the income effect on child health.

[18] Starting with the general model in specification 3, we reduce, using the appropriate diagnostics, the number of regressors in order to obtain a model that includes only the 'important' variables, as informed by economic and epidemiologic theory ('general to specific' or 'backward deletion' strategy). In particular we use the criterion suggested by Rothman and Green (1998) to select model confounders. Rothman and Green (1998) note that testing procedures will perform well if the tests have a high power that enable detecting any relevant confounder effects. They recommend, therefore, to raise the alpha-level for rejecting the null hypothesis of no confounding to 0.20 or more, rather than using the traditional 0.05 level (see also Dales and Ury, 1978).

[19] Case et al., 2002, show that the association of income with children's health is stronger whenever long-run average household income (permanent income) is used as a measure of household economic resources. Such findings are confirmed by subsequent empirical studies (see, for example, Burgess et al. 2004 and Propper et al., 2007). Given the short nature of our panel (two time periods) the choice of past income can be considered a reasonable measure of household income.

There is, however, also the possibility that income effect bias works in the opposite direction. Household income data, as it is common practice in most micro surveys, are grouped in bands and this enhances the probability of measurement errors which, in turn, implies an estimated coefficient for income that is biased towards zero. Whether the impact of income on child health is effectively under- or over-estimated is ultimately an empirical issue.

The probit instrumental variable (IV) approach provides a way of estimating our models without measurement error as well as endogeneity bias. The IV method consists of identifying one or more instrumental variables (excluded instruments), denoted $\mathbf{Z}$, for household income. Excluded instruments $\mathbf{Z}$ have to be observable variables that include information that is correlated with the endogenous variable, household income, but not with the unobservables contained in ε_i^c. In other terms, good instruments have to satisfy two properties: *i)* they must be highly correlated with variables being instrumented (relevance of instruments) and *ii)* they must be contemporaneously uncorrelated with (orthogonal to) the error term (overidentifying restriction). The main problem in implementing the IV estimation technique is due to the practical difficulty of finding valid instruments given the limited range of variables available in survey data. Simplifying the approach[20], the first step of the IV estimation amounts to running a first stage regression of the endogenous variable Y_i in (1) on the vector of instruments $\mathbf{Z}$ as well as on the vector of covariates $\mathbf{X}$, which for household *i* takes the following form:

$$Y_i = \alpha_0 + \mathbf{\Phi Z}_i + \boldsymbol{\gamma} \mathbf{X}_i + \xi_i^Y \qquad (2)$$

where ξ_i^Y is the error term. First stage income regression (2) is estimated by least squares and produces a prediction $\hat{Y}_i$ for household income which is no longer correlated with ε_i^c, as instruments $\mathbf{Z}$ have allowed us to isolate the exogenous variation in Y_i.

In the second stage of the IV approach, we use the computed predicted values $\hat{Y}_i$ from estimation (2) to re-estimate equation (1) as:

$$H_i^c = \alpha + \beta \hat{Y}_i + \boldsymbol{\delta} \mathbf{X}_i + \varepsilon_i^c \qquad (3)$$

using a probit model. The IV technique allows us to obtain an unbiased estimate of the impact of household income on child health because the instruments have eliminated any endogenous relationship between income and child health.

The hypothesis that household income is endogenous to child health is tested by including the estimated residuals from the first-stage income regression (2) as covariates in child health outcome equation (1). If they are statistically significant, then we conclude that there is evidence of endogeneity and the IV approach is therefore justified (see Hausman, 1978; Rivers and Vuong, 1988; Wooldridge, 2002).

Since the performance of the IV estimator critically relies on the degree of relevance of the instruments, namely on the correlation between instruments and the endogenous explanatory variable, we assess this property by focusing on the first stage regression (2). The analysis of the explanatory power of the excluded instruments $\mathbf{Z}$, as expressed by the associated coefficients in

[20] A complete exposition of the method can be found in Davidson and MacKinnon, 1993; Wooldridge, 2002 and 2003; Baum et al., 2003, Greene, 2003; Cameron and Trivedi, 2006.

the first stage regression, represents a first test of relevance of the instruments. A statistic commonly used for testing the relevance property is the *F*-test of the joint significance of the **Z** instruments in the first-stage regression (see Baum et al. 2003). As remarked by Hahn and Hausman (2002), if the excluded instruments have little explanatory power (weak instrument problem) they cause bias in the estimated IV coefficients. In the empirical applications of the IV method, when only a single endogenous variable is present, an *F*-statistic that assumes a value smaller than 10, should cause concern as it might indicate that there is a problem of weak instruments (see Staiger and Stock, 1997).

Finally, following Ettner (1996) and Johnson and Skinner (1986), we run a test of the overdentifying restrictions on the child health equation by comparing the performance of the equation with and without the instrumental variables[21]. If the equation is correctly specified, the inclusion of the instrumental variables should not considerably affect the explanatory power of the model. Wald tests are used to asses the explanatory power of the instruments[22].

5. Results

Table 2 reports the child health outcome probit estimations obtained without correcting for the endogeneity of household income. All the estimates are computed as partial effects[23] and can, therefore, be given a quantitative interpretation in terms of units of probability.

In the unadjusted model column (model specification 1) the coefficient on household income is negative and significant at the 1 percent level for both asthma (coef.: -0.035; p-value ≤ 0.01) and wheezing (coef.: -0.031; p-value ≤ 0.01) outcomes. The partial effect of household income on the probability that the child suffers from asthma is to reduce it by 4 percent, while the analogous partial income effect on the wheezing outcome is to reduce the correspondent probability by 3 percent. These findings show that our data reproduce the standard results in the broader literature, namely that there exists a statistically significant inverse relationship between household income and adverse child health outcomes. As we move across the columns in Table 2, we observe that the coefficients for household income remain negative and almost always significant, even if their magnitudes reduce to approximately 2 percent in both the asthma (model specification 2: coef.: -0.017; p-value: 0.005; model specification 3: coef.: -0.015; p-value: 0.025) and wheezing (model specification 2: coef.: -0.017; p-value: 0.053; model specification 3: coef.: -0.014; p-value: 0.170) models. The model for childhood asthma, which includes all explanatory variables (model specification 3), and the model that emerges after the selection procedure (model specification 2), show that, besides household income, other factors are significantly associated with this respiratory disease in childhood. The estimates reveal the importance of maternal behaviours such as smoking during pregnancy[24] and limited

[21] As Johnson and Skinner (1986) stress, whilst there are various standard tests of overidentifying restrictions for linear models, the corresponding tests in a nonlinear context are quite complex (see also Hausman, 1983). The version that Johnson and Skinner (1986) use in their paper and that we have adopted here was suggested to them by Whitney Newey.

[22] Stata 9 was used to perform all the analysis presented in this paper.

[23] Marginal effects are calculated for continuous explanatory variables while average effects are computed for binary explanatory variables.

[24] It has to be noted that our results on maternal smoking during pregnancy (see Tables 2 and 4), in all of the model specifications, seem to contradict the epidemiological evidence that the harmful effects of smoking increase with the number of cigarettes smoked (see Petrou et al., 2005). Our results suggest that the adverse impact on childhood respiratory health is not always significant when the mother smokes more than 10 cigarettes per day, even if the

breastfeeding practice as factors associated with asthma in early childhood. Childhood asthma was more likely to be reported if there was a familial history of asthma, if the child's birthweight was less than 2.5 kg and if the child was of male sex. Asthmatic children were also more likely to live in council rented accommodation, rather than mortgaged or owned properties, and have older siblings[25]. Contact with pets in the child's first year of life was of borderline significance as a risk factor for childhood asthma. Maternal age at birth, parental educational level and ethnicity do not seem to represent significant risk factors for childhood asthma.

Moving on to the results obtained for the wheezing models (model specifications 2 and 3), we can clearly note that the propensity to suffer from wheezing in the children belonging to our sample was essentially associated with the same risk factors as in the asthma models. Differences can be found, however, in the magnitude of the impact exerted by various risk factors on this health outcome measure, as the coefficients are generally larger than in the asthma models, whilst the direction and significance of their impact do not generally differ. Exceptions to this rule were noted for maternal smoking during pregnancy, the presence of older siblings and contact with pets in the first year of the child's life, which all show a stronger association with reported wheezing than for reported asthma. Furthermore, preterm delivery emerges as a significant risk factor for reported wheezing.

As anticipated, the impact of household income on childhood respiratory outcomes may be subject to endogeneity problems. We account for this potential endogeneity by using grandparental income support and paternal grandfather's socioeconomic status to instrument household income. The last row of Table 2 reports the levels of type I error for rejecting the null hypothesis that household income is exogenous[26]. The values generally indicate that the endogeneity of income cannot be rejected. Consequently, the IV method is likely to produce more consistent estimates and should be preferred.

The relevance of instruments, namely their correlation with household income, is easily tested by evaluating the significance of the instruments themselves in the first stage regression. The upper part of Table 3 reproduces the coefficient estimates and the *F*-statistics of excluded instruments in the first stage regression, with the aim of investigating their explanatory power. The results show that the chosen instruments are relevant: their coefficient estimates are significant both individually and jointly. In addition, the problem of weak instruments (which can increase the bias in the estimated IV coefficients) does not emerge: the *F*-statistics take values larger than 10, which is not cause for concern according to the rule of thumb put forward by Staiger and Stock (1997).

The second property required of an instrumental variable, namely the orthogonality with the error term, is verified by using the overidentifying restrictions test in the version suggested by

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direction of the effect is positive as expected. Rather, the harmful effects of smoking are confirmed when the mother smokes less than 10 cigarettes per day. A probable explanation of this result might be the loss of efficiency due to the small proportion of mothers smoking ≥10 cigarettes per day during pregnancy, which does not invalidate the general message that fetal exposure to maternal smoking is harmful to the child health.

[25] The positive impact of older siblings (see asthma models, Tables 2 and 4) is in line with the results of epidemiological studies such as Baker et al., 1998, and Sheriff et al., 2001. On the contrary, the negative impact of older siblings (see wheezing models, Tables 2 and 4) appears to support the 'hygiene hypothesis' (see Eder et al., 2006), according to which respiratory disease develops partly as a result of the lack of exposure to infections and microbial products in the early stages of a child's life.

[26] This is particularly true for model specifications 2, both for the asthma and the wheezing outcomes, as they are ultimately the models that best shape the income-child health relationship according to the model selection procedure we used.

Newey (see footnote 23). As it emerges from the lower part of Table 3, the adjusted Wald tests do not provide consistent support that any of the instrumental variables has a strong explanatory power in the models. This suggests that the orthogonality conditions are satisfied.

Table 4 reports the IV probit estimates for the three model specifications for the asthma and wheezing outcomes. Within each model, after taking endogeneity into account, increasing income has a negative, statistically significant and increased effect. Focusing on model specification 2, which is our preferred model, we notice that the partial effect of increased household income on the probability that the child suffers from asthma is to reduce it by 6 percent (coef.: -0.056; *p*-value: 0.039), which is three times the effect that was found with the ordinary probit estimation. The impact of increased income on the probability of the child suffering for wheezing is even larger. It decreases the probability of the outcome by 9 percent (coef.: -0.087; *p*-value: 0.006), which is more than four times the impact exerted by increased income when endogeneity was not accounted for. Income is significant at the 5 percent level in the asthma model, whilst it is significant at the 1 percent level in the wheezing model.

The fact that, after instrumenting, the magnitude of the income effect increases rather than diminishes could seem, at first, a surprising result, as potential bias due to reverse causality and data heterogeneity are expected to overestimate the effect of the endogeneous variable on the outcome. Nevertheless, if we consider that income might be measured with error and that the usual effect of errors-in-variables is to bias ordinary estimates towards zero, then our results can be plausibly justified. The finding that the net effect of accounting for endogeneity is a larger impact of income on respiratory outcomes might simply indicate that, in the ordinary probit estimates, forces that tend to bias coefficients downwards prevailed over those that pushed the estimates upwards. Furthermore, as Ettner (1996) observes[27], the direction of endogeneity bias is theoretically ambiguous whenever the model includes more than one regressor. In fact, there might be second-order effects that work through the correlation of income with other explanatory variables that might change the expected direction of the bias. For this reason, only empirical analysis of the type we have conducted can throw light on these uncertain impacts.

As for the other factors that have an impact on both childhood respiratory outcomes, the IV estimations produce results that are largely unchanged, in terms of magnitude, significance and direction, with respect to those obtained using the ordinary probit approach. They, therefore, confirm the importance of low birthweight, male sex, the presence of older siblings, familial history of asthma, rented housing tenure, maternal smoking and limited breastfeeding as determinants of asthma and wheezing illnesses among pre-school children.

6. Conclusions and policy implications

In this paper we have investigated the empirical relationship between household income and two childhood respiratory outcomes, asthma and wheezing, using a sample of 7,659 families extracted from the UK Millennium Cohort Study survey data. Our analysis is motivated by recent research that has shown that the association between income and child health is strong in the United States and Canada, but seems rather weak in the United Kingdom. While most published studies primarily document the association between parental income and child health outcomes, attempts to address the potential endogeneity of household income with respect to

[27] The empirical analysis conducted by Ettner (1996) investigates the income-health relationship in an adult population. Her findings show that the impact of income on health remains significant and increases in size when the IV approach is applied in place of the OLS method.

child health outcomes remain largely unexplored in the child health literature. We contribute to this body of empirical literature by taking into account the endogeneity of household income on childhood asthma and wheezing by using an IV approach.

For comparison purposes, we start analysing the income-child health relationship by assuming the exogeneity of income. We confirm the findings of earlier studies that lower household income is associated with poorer respiratory health. We then estimate the models with an IV approach that relaxes the assumption of exogeneity for household income. Grandparental income and paternal grandfather's socioeconomic status are identified as valid instruments for parental income. The results generated by the probit models and probit models with IV are similar in most respects. However, the exogeneity test indicates the rejection of the null hypothesis that household income is exogeneous and confirms the superiority of the IV approach, which in turn generates more consistent estimates.

As anticipated, the results from the two approaches are similar. Interestingly, taking into account the endogeneity of income not only maintains the significance and the direction of the income coefficient, but also increases the magnitude of the income effects. The influence of other socioeconomic characteristics and risk factors on childhood respiratory health outcomes does not substantially vary, even in magnitude, between the two approaches. Those children who suffer from asthma and wheezing are more likely to be male, of low birthweight, to have older siblings and to live in rented accommodation. Familial history of asthma and exposure to pets in the early years of life also increase the chance of suffering from these respiratory diseases. Finally, a strong role is played by mother's child-health-related behaviours. The continuation of breast feeding rather than bottle feeding is very likely to have a protective effect on child health, while both asthma and wheezing are more likely to be reported if the mother smoked during pregnancy.

The results also seem to support a substantial stream of research that provides evidence on the persistence of economic status across generations (see, for example, Corcoran *et al*, 1992; Solon, 1992; Zimmerman, 1992). This is shown in the first stage regressions (see Table 3) where the instrumental variables, namely grandparental income support and paternal grandfather's social status, have a significant relationship with household income: the wealthier the grandparents the better off the parents are, in general, and, consequently, the children. This evidence might corroborate the hypothesis of an intergenerational mechanism of socioeconomic status transmission, which might imply an intergenerational mechanism of transmission of health inequalities. While this hypothesis requires additional investigation, and can therefore represent the focus of further research, it might also suggest a mechanism for governments to address the problems of health inequalities. Enhanced cash transfers to low income families with children could be accompanied by alternative strategies that tackle the problem at root and with lasting effects. An enhanced mobility across the social status hierarchy can be encouraged by promoting programmes of early education targeted towards those strata of the population that are at highest risk of deprivation. There is therefore scope for further research aimed at investigating more deeply the mechanisms that underlie the income-child health nexus and which can inspire alternative policy actions.

Finally, given the importance that mothers' child-health-related behaviours have on affecting child health, some public health measures such as encouraging the practice of breast feeding as opposed to early bottle feeding, and a stronger campaign for discouraging smoking, especially during pregnancy, are highly recommended.

Our study has several limitations that should be considered by readers. From a methodological point of view the acceptability of the 'listwise deletion' approach for dealing with missing data could be questioned. We addressed this possible limitation by running a regression-based data imputation (multinomial logit) to fill the gaps left by the missing observations. We then ran our estimates on the imputed data set. Our results confirmed the robustness of the findings based on the reduced sample of 7,659 cohort children and, therefore, confirmed the representativeness of the reduced sample (data available upon request).

More serious drawbacks of the study can be identified in the way in which the main variables of interest, namely household income and measures of adverse child respiratory health outcomes, have been defined in the MCS dataset. Similar to current practice in other survey datasets, information on the level of household income is categorised in income groups. This might introduce an element of inaccuracy not only in the measure itself, but also in the estimates based on the variable. Despite the fact that the instrumental variable approach addresses the errors-in-variables bias, the model estimates would certainly benefit from the use of a more precise measure of income.

With respect to the definition of adverse respiratory health, both childhood asthma and wheezing were parent-reported in the MCS surveys. They therefore suffer from all the related limitations that typically affect self-reported measures of health (such as bias due to subjectivity) as opposed to more objective measures that, on the contrary, are clinically diagnosed and validated.

In addition, it can be argued that the analysis overlooked an important risk factor for asthma and wheezing, namely air quality. As the researchers of the Asthma Center of the University of Chicago (http://asthma.bsd.uchicago.edu/AboutAsthma/AACause.html accessed 16 may 2007) point out, there is clear evidence that that air pollution can have a negative effect on the respiratory ability of people that already suffer from asthma. On the other hand, many epidemiological studies have shown that asthma is likely to occur with greater frequency in certain families, suggesting a genetic origin of the disease (see, for example, Sheriff et al. 2001; McCunney, 2005). Furthermore, as Peden (2005) observes, the occurrence of asthma and other allergic disease have increased continuously in the last 40 years in the US and worldwide, even though the general quality of air has improved. This throws additional doubts on whether air quality is really a relevant risk factor for the development and the severity of the asthma and wheezing illnesses. In the light of this, the absence from our analysis of an air pollution component, which is not easily linkable to the individual-level MCS data, should not represent a crucial shortcoming.

In conclusion, our study provides strong evidence of a causal link between household income and child respiratory health. This evidence should motivate and support government fiscal policies aimed at reducing health inequalities in childhood. This will require targeting deprived populations with specific interventions, aimed at health promotion and protection, which are known to be cost-effective. In addition, however, the reduction of childhood respiratory health inequalities will almost certainly require broader macroeconomic initiatives, such as tax and benefit changes, aimed at reducing relative poverty.

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Table 1. Definitions and summary statistics of analysis variables

Variable (dummy variable if not otherwise specified)	Mean[a]	Standard deviation[a]
Child-health outcomes		
Whether the child has ever had asthma	0.102	0.302
Whether the child had wheezing or whistling in the chest in the last 12 months	0.281	0.449
Child characteristics		
Birth weight: ≤ 2.5 kg	0.058	0.234
Gestation: < 37 weeks	0.074	0.261
Child's gender: male	0.504	0.500
Child's ethnicity		
White[*]	0.920	0.271
Black	0.010	0.098
Asian	0.040	0.195
Other	0.030	0.171
Number of older siblings		
No older siblings[*]	0.428	0.495
1 older sibling	0.384	0.486
2 or more siblings	0.187	0.390
Socio-economic factors		
Mother's age at child's birth		
13 to 19	0.028	0.165
20 to 35[8]	0.826	0.379
36 or more	0.146	0.353
Mother's highest educational qualification		
nvq4/nvq5/degree/equivalent[*]	0.110	0.313
higher education below degree	0.068	0.251
nvq3/gce a level equivalent	0.083	0.277
nvq2/gce o level equivalent	0.199	0.399
nvq1/cse other equivalent	0.081	0.273
foreign/other	0.083	0.276
no qualification	0.375	0.484
Father's highest educational qualification		
nvq4/nvq5/degree/equivalent[*]	0.132	0.338
higher education below degree	0.053	0.224
nvq3/gce a level equivalent	0.066	0.249
nvq2/gce o level equivalent	0.199	0.399
nvq1/cse other equivalent	0.032	0.176
foreign/other	0.134	0.341
no qualification	0.384	0.486
Lone parent status	0.012	0.107
Housing tenure at 9 months of age		
mortgaged/owned[8]	0.796	0.403
council rented	0.079	0.271
rented (housing association or private)	0.095	0.293
other	0.029	0.168
Logarithm of equivalised household income (continuous variable)	9.547	0.661
Parental health risk factors		
Maternal history of asthma and hay fever	0.335	0.472
Paternal history of asthma and hay fever	0.339	0.473
Mother's child-health-related behaviours		
Maternal smoking during pregnancy		
Never smoked[*]	0.739	0.439
Stopped smoking during pregnancy	0.119	0.324
Smoked throughout pregnancy less than 10 cigarettes	0.085	0.279
Smoked throughout pregnancy 10 or more than 10 cigarettes	0.057	0.232
Mother breastfed		
never breastfed[*]	0.223	0.417
less than 7 days	0.099	0.299

Table 1 - Continued

Variable (dummy variable if not otherwise specified)	Mean[a]	Standard deviation[a]
Continue… Mother breastfed		
1 week to 3 months (included) *	0.275	0.446
3 to 6 months (included)	0.171	0.377
more than 6 months	0.231	0.422
Environmental exposure		
Presence of pets in child home		
no pets*	0.399	0.490
pets kept at home at present	0.114	0.318
pets kept at home during child's first year of life	0.084	
pets kept at home throughout the years of child's life	0.402	0.278
		0.490
Tobacco exposure (whether anyone smokes in same room as child)	0.146	0.353
INSTRUMENTAL VARIABLES		
Non-earnings income received from the grandparents		
Buying essentials for the child	0.332	0.471
Paying for other household costs	0.103	0.304
Buying gifts & extras for the child	0.883	0.322
Lending money	0.234	0.423
Paying for childcare	0.014	0.118
Other help	0.051	0.220
Relatives Social Status		
Paternal grandfathers' social status		
not working	0.047	0.212
managers and senior officials*	0.148	0.355
professional occupations	0.091	0.288
associate professional and technical occupations	0.098	0.297
administrative and secretarial occupations	0.039	0.194
skilled trades occupations	0.257	0.437
personal service occupations	0.009	0.096
sales and customer service occupations	0.020	0.140
process, plant and machine operatives	0.157	0.364
elementary occupations	0.083	0.276
other job (not classified)	0.005	0.070
can't remember / father died before respondent was 14	0.045	0.208

[a] Means and standard deviations are weighted by the MCS weights by strata for the UK (see Hansen, 2006, for technical details)
Sample size: 7,659
* Reference case

Table 2 – Probit ordinary estimates – partial effects (robust standard errors in parentheses)

	Dependent variable: whether the child has asthma			Dependent variable: whether the child has wheezing		
Explanatory variables	***Model specification 1***	***Model specification 2***	***Model specification 3***	***Model specification 1***	***Model specification 2***	***Model specification 3***
Socio-economic factors						
Logarithm of equivalised household income	-0.035***	-0.017***	-0.015**	-0.031***	-0.017***	-0.014
	(0.006)	(0.006)	(0.007)	(0.008)	(0.009)	(0.010)
Mother's age at child's birth						
13 to 19	--	--	0.022	--	--	-0.020
			(0.025)			(0.032)
36 or more	--	--	0.002	--	--	0.016
			(0.011)			(0.019)
Mother's highest educational qualification						
higher education below degree	--	--	0.003	--	--	-0.028
			(0.016)			(0.025)
nvq3/gce a level equivalent	--	--	0.011	--	--	-0.028
			(0.019)			(0.029)
nvq2/gce o level equivalent	--	--	0.015	--	--	-0.030
			(0.017)			(0.026)
nvq1/cse other equivalent	--	--	0.001	--	--	-0.012
			(0.018)			(0.030)
foreign/other	--	--	0.014	--	--	0.005
			(0.020)			(0.029)
no qualification	--	--	0.002	--	--	-0.029
			(0.014)			(0.023)
Father's highest educational qualification						
higher education below degree	--	--	-0.031*	--	--	-0.025
			(0.016)			(0.033)
nvq3/gce a level equivalent	--	--	-0.022	--	--	0.014
			(0.015)			(0.030)
nvq2/gce o level equivalent	--	--	0.005	--	--	0.022
			(0.016)			(0.026)
nvq1/cse other equivalent	--	--	0.040	--	--	0.071*
			(0.030)			(0.043)
foreign/other	--	--	-0.012	--	--	0.005
			(0.016)			(0.024)
no qualification	--	--	-0.004	--	--	0.026
			(0.013)			(0.021)
Lone parent status	--	--	0.0009	--	--	0.063
			(0.026)			(0.055)
Housing tenure at 9 months of age						
council rented	--	0.038***	0.035**	--	--	0.015
		(0.015)	(0.015)			(0.025)
rented (housing association or private)	--	-0.0006	-0.002	--	--	-0.017
		(0.0140)	(0.014)			(0.024)

Table 2- Continued

	Dependent variable: whether the child has asthma			Dependent variable: whether the child has wheezing		
Explanatory variables	***Model specification 1***	***Model specification 2***	***Model specification 3***	***Model specification 1***	***Model specification 2***	***Model specification 3***
Continue… Housing tenure at 9 months other	--	0.049**	0.045*	--	--	-0.003
		(0.025)	(0.025)			(0.035)
Child characteristics						
Birth weight: ≤ 2.5 kg	--	0.051***	0.045**	--	0.097***	0.098***
		(0.017)	(0.021)		(0.030)	(0.030)
Gestation: < 37 weeks	--	--	0.005	--	0.043*	0.040*
			(0.016)		(0.025)	(0.025)
Child's gender: male	--	0.036***	0.035***	--	0.082***	0.081***
		(0.007)	(0.007)		(0.012)	(0.012)
Child's ethnicity						
Black	--	--	0.051	--	--	0.040
			(0.053)			(0.052)
Asian	--	--	0.009	--	--	-0.006
			(0.019)			(0.030)
Other	--	--	0.021	--	--	-0.035
			(0.025)			(0.036)
Number of older siblings						
1 older sibling	--	0.014*	0.016**	--	0.027**	0.025**
		(0.008)	(0.008)		(0.013)	(0.013)
2 or more siblings	--	0.005	0.008	--	-0.034**	-0.037**
		(0.012)	(0.012)		(0.017)	(0.017)
Parental health risk factors						
Maternal history of asthma and hay fever	--	0.062***	0.061***	--	0.121***	0.121***
		(0.008)	(0.009)		(0.014)	(0.014)
Paternal history of asthma and hay fever	--	0.050***	0.050***	--	0.077***	0.078***
		(0.008)	(0.008)		(0.013)	(0.013)
Mother's child-health-related behaviours						
Maternal smoking during pregnancy						
Stopped smoking during pregnancy	--	-0.013	-0.013	--	-0.0007	0.001
		(0.011)	(0.011)		(0.019)	(0.020)
Smoked throughout pregnancy less than 10 cigarettes	--	0.026*	0.023	--	0.074***	0.073***
		(0.016)	(0.016)		(0.021)	(0.021)
Smoked throughout pregnancy 10 or more than 10 cigarettes	--	0.023	0.022	--	0.052*	0.051*
		(0.017)	(0.017)		(0.029)	(0.031)
Mother breastfed less than 7 days	--	-0.015	-0.016	--	-0.015	-0.013
		(0.012)	(0.012)		(0.021)	(0.021)

Table 2- Continued

	Dependent variable: whether the child has asthma			Dependent variable: whether the child has wheezing		
Explanatory variables	***Model specification 1***	***Model specification 2***	***Model specification 3***	***Model specification 1***	***Model specification 2***	***Model specification 3***
Continue… Mother breastfed						
1 week to 3 months (included)	--	-0.026***	-0.026***	--	-0.040**	-0.040**
		(0.008)	(0.008)		(0.17)	(0.017)
3 to 6 months (included)	--	-0.039***	-0.039***	--	-0.043**	-0.045**
		(0.010)	(0.009)		(0.018)	(0.019)
more than 6 months	--	-0.056***	-0.056***	--	-0.068***	-0.069***
		(0.009)	(0.009)		(0.016)	(0.025)
Environmental exposure						
Presence of pets in child home						
pets kept at home at present	--	--	-0.016	--	-0.017	-0.017
			(0.012)		(0.019)	(0.020)
pets kept at home during child's first year of life	--	--	0.018	--	0.035	0.036
			(0.014)		(0.023)	(0.023)
pets kept at home throughout the years of child's life	--	--	0.009	--	-0.011	-0.012
			(0.008)		(0.014)	(0.014)
Tobacco exposure (whether anyone smokes in same room as child)	--	--	-0.003	--	0.022	0.021
			(0.010)		(0.019)	(0.019)
Efron's pseudo-R^2	0.005	0.041	0.043	0.002	0.043	0.044
Number of observations	7659	7659	7659	7659	7659	7659
Exogeneity test (p-values)	0.000	0.063	0.141	0.000	0.039	0.023

Notes:

1. * Significant at the 10-percent level
   ** Significant at the 5-percent level
   *** Significant at the 1-percent level
2. Robust standard errors are in parentheses.
3. Estimates are partial effects.
4. Efron's pseudo-R^2 is equal to (see Efron, 1978; Veall and Zimmermann, 1996; Hoetker, 2007) :

$$1-\frac{\sum_{i=1}^{N}(y_i-\hat{\pi}_i)^2}{\sum_{i=1}^{N}(y_i-\bar{y}_i)^2}$$ where $\hat{\pi}$ is the predicted probability and N is the sample size.

Table 3 – First stage regressions –Tests of relevance and validity of excluded instruments (robust standard errors in parentheses)

	Dependent variable: log(income) – asthma model			Dependent variable: log(income) – wheezing model		
Explanatory variables	***Model specification 1***	***Model specification 2***	***Model specification 3***	***Model specification 1***	***Model specification 2***	***Model specification 3***
Non-earnings income received from the grandparents						
Buying essentials for the child	-0.097***	-0.104***	-0.086***	-0.101***	-0.113***	-0.087***
	(0.018)	(0.016)	(0.016)	(0.179)	(0.018)	(0.015)
Paying for other household costs	-0.248***	-0.157***	-0.124***	-0.251***	-0.235***	-0.127***
	(0.028)	(0.027)	(0.024)	(0.028)	(0.029)	(0.024)
Buying gifts & extras for the child	0.148***	0.068***	0.057***	0.147***	0.100***	0.056***
	(0.026)	(0.024)	(0.023)	(0.026)	(0.025)	(0.023)
Lending money	-0.245***	-0.148***	-0.114***	-0.248***	-0.173***	-0.116***
	(0.022)	(0.020)	(0.018)	(0.022)	(0.022)	(0.18)
Paying for childcare	0.046	0.035	0.040	0.045	0.048	0.035
	(0.054)	(0.052)	(0.046)	(0.054)	(0.053)	(0.044)
Other help	-0.011	-0.042	-0.080***	-0.007	-0.034	-0.076**
	(0.035)	(0.033)	(0.033)	(0.035)	(0.034)	(0.032)
Relatives Social Status						
not working	-0.356***	-0.190***	-0.122***	-0.350***	-0.269***	-0.118***
	(0.046)	(0.040)	(0.035)	(0.045)	(0.042)	(0.034)
professional occupations	0.190***	0.168***	0.114***	0.189***	0.167***	0.114***
	(0.032)	(0.031)	(0.029)	(0.032)	(0.032)	(0.029)
associate professional and technical occupations	0.023	0.044	0.032	0.028	0.028	0.035
	(0.037)	0.034)	(0.033)	(0.036)	(0.034)	(0.032)
administrative and secretarial occupations	-0.015	-0.012	-0.036	-0.013	-0.011	-0.033
	(0.042)	(0.042)	(0.039)	(0.042)	(0.043)	(0.039)
skilled trades occupations	-0.157***	-0.097***	-0.068***	-0.151***	-0.113***	-0.063***
	(0.026)	(0.024)	(0.022)	(0.026)	(0.025)	(0.022)
personal service occupations	-0.297***	-0.173***	-0.107*	-0.303***	-0.232***	-0.110*
	(0.075)	(0.069)	(0.057)	(0.077)	(0.075)	(0.058)
sales and customer service occupations	-0.133***	-0.155***	-0.089*	-0.129***	-0.129***	-0.087*
	(0.052)	(0.052)	(0.052)	(0.053)	(0.051)	(0.051)
process, plant and machine operatives	-0.200***	-0.116***	-0.081***	-0.193***	-0.135***	-0.076***
	(0.029)	(0.028)	(0.025)	(0.030)	(0.028)	(0.025)
elementary occupations	-0.218***	-0.132***	-0.099***	-0.210***	-0.155***	-0.092***
	(0.036)	(0.033)	(0.029)	(0.036)	(0.034)	(0.029)
other job (not classified)	-0.282***	-0.108	-0.031	-0.260***	-0.175*	-0.013
	(0.100)	(0.091)	(0.087)	(0.106)	(0.107)	(0.092)
can't remember / father died before respondent was 14	-0.235***	-0.115***	-0.079**	-0.227***	-0.172***	-0.072**
	(0.038)	(0.037)	(0.035)	(0.039)	(0.039)	(0.036)

Table 3 - continued

	Dependent variable: log(income) – asthma model			**Dependent variable: log(income) – wheezing model**		
	Model specification 1	***Model specification 2***	***Model specification 3***	***Model specification 1***	***Model specification 2***	***Model specification 3***
F- test of excluded instruments (*p*-value)	35.04 (0.000)	20.61 (0.000)	15.08 (0.000)	35.04 (0.000)	28.90 (0.000)	15.08 (0.000)
Test of overidentifying restrictions instruments (*p*-value)	--	1.26 (0.218)	1.25 (0.224)	--	1.23 (0.234)	1.33 (0.168)
Number of observations	7659	7659	7659	7659	7659	7659

Notes:
1. * Significant at the 10-percent level
** Significant at the 5-percent level
*** Significant at the 1-percent level
2. Robust standard errors are in parentheses
3. Least squares estimates

Table 4 – IV Probit estimates – partial effects (robust standard errors in parentheses)

	Dependent variable: whether the child has asthma			Dependent variable: whether the child has wheezing		
Explanatory variables	***Model specification 1***	***Model specification 2***	***Model specification 3***	***Model specification 1***	***Model specification 2***	***Model specification 3***
Socio-economic factors						
Logarithm of equivalised household income	-0.090***	-0.056**	-0.058	-0.118***	-0.087***	-0.137***
	(0.020)	(0.027)	(0.038)	(0.026)	(0.032)	(0.055)
Mother's age at child's birth						
13 to 19	--	--	0.006	--	--	-0.056*
			(0.026)			(0.034)
36 or more	--	--	0.008	--	--	0.027
			(0.012)			(0.020)
Mother's highest educational qualification						
higher education below degree	--	--	-0.004	--	--	-0.044*
			(0.016)			(0.027)
nvq3/gce a level equivalent	--	--	0.003	--	--	-0.049*
			(0.020)			(0.029)
nvq2/gce o level equivalent	--	--	0.002	--	--	-0.061**
			(0.020)			(0.030)
nvq1/cse other equivalent	--	--	-0.011	--	--	-0.045
			(0.020)			(0.034)
foreign/other	--	--	0.006	--	--	-0.016
			(0.021)			(0.031)
no qualification	--	--	-0.007	--	--	-0.051*
			(0.016)			(0.027)
Father's highest educational qualification						
higher education below degree	--	--	-0.036**	--	--	-0.043
			(0.016)			(0.032)
nvq3/gce a level equivalent	--	--	-0.032**	--	--	-0.017
			(0.016)			(0.033)
nvq2/gce o level equivalent	--	--	-0.011	--	--	-0.019
			(0.018)			(0.030)
nvq1/cse other equivalent	--	--	0.020	--	--	0.025
			(0.031)			(0.045)
foreign/other	--	--	-0.025*	--	--	-0.030
			(0.015)			(0.028)
no qualification	--	--	-0.018	--	--	-0.009
			(0.016)			(0.025)
Lone parent status	--	--	-0.033	--	--	-0.044
			(0.028)			(0.062)
Housing tenure at 9 months of age						
council rented	--	0.005	0.007	--	--	-0.045
		(0.020)	(0.023)			(0.036)
rented (housing association or private)	--	-0.019	-0.016	--	--	-0.055*
		(0.017)	(0.018)			(0.029)

Table 4 – Continued

	Dependent variable: whether the child has asthma			Dependent variable: whether the child has wheezing		
Explanatory variables	***Model specification 1***	***Model specification 2***	***Model specification 3***	***Model specification 1***	***Model specification 2***	***Model specification 3***
Continue… Housing tenure at 9 months other	--	0.015	0.019	--	--	-0.066*
		(0.029)	(0.030)			(0.043)
Child characteristics						
Birth weight: ≤ 2.5 kg	--	0.049***	0.044**	--	0.093***	0.093***
		(0.017)	(0.021)		(0.030)	(0.031)
Gestation: < 37 weeks	--	--	0.005	--	0.044*	0.038
			(0.017)		(0.025)	(0.025)
Child's gender: male	--	0.036***	0.035***	--	0.082***	0.082***
		(0.007)	(0.007)		(0.012)	(0.012)
Child's ethnicity						
Black	--	--	0.044	--	--	0.046
			(0.054)			(0.052)
Asian	--	--	-0.012	--	--	-0.041
			(0.024)			(0.041)
Other	--	--	0.020	--	--	-0.032
			(0.026)			(0.039)
Number of older siblings						
1 older sibling	--	0.009	0.010	--	0.018	0.009
		(0.008)	(0.009)		(0.013)	(0.015)
2 or more siblings	--	-0.003	-0.001	--	-0.047***	-0.060***
		(0.013)	(0.014)		(0.017)	(0.019)
Parental health risk factors						
Maternal history of asthma and hay fever	--	0.061***	0.060***	--	0.117***	0.115***
		(0.009)	(0.009)		(0.014)	(0.014)
Paternal history of asthma and hay fever	--	0.052***	0.051***	--	0.077***	0.078***
		(0.009)	(0.009)		(0.013)	(0.013)
Mother's child-health-related behaviours						
Maternal smoking during pregnancy						
Stopped smoking during pregnancy	--	-0.016	-0.014	--	-0.009	-0.001
		(0.011)	(0.011)		(0.020)	(0.020)
Smoked throughout pregnancy less than 10 cigarettes	--	0.020	0.021	--	0.060***	0.069***
		(0.016)	(0.016)		(0.022)	(0.022)
Smoked throughout pregnancy 10 or more than 10 cigarettes	--	0.015	0.019	--	0.033	0.043
		(0.018)	(0.018)		(0.030)	(0.031)
Mother breastfed less than 7 days	--	-0.013	-0.016	--	-0.013	-0.013
		(0.013)	(0.013)		(0.021)	(0.022)

Table 4 – Continued

	Dependent variable: whether the child has asthma			Dependent variable: whether the child has wheezing		
Explanatory variables	***Model specification 1***	***Model specification 2***	***Model specification 3***	***Model specification 1***	***Model specification 2***	***Model specification 3***
Continue… Mother breastfed						
1 week to 3 months (included)	--	-0.020**	-0.022**	--	-0.031*	-0.033*
		(0.009)	(0.009)		(0.018)	(0.018)
3 to 6 months (included)	--	-0.028***	-0.031***	--	-0.023	-0.025
		(0.011)	(0.011)		(0.020)	(0.020)
more than 6 months	--	-0.049***	-0.054***	--	-0.057***	-0.063***
		(0.010)	(0.009)		(0.017)	(0.017)
Environmental exposure						
Presence of pets in child home						
pets kept at home at present	--	--	-0.018	--	-0.023	-0.023
			(0.012)		(0.020)	(0.020)
pets kept at home during child's first year of life	--	--	0.015	--	0.029	0.026
			(0.015)		(0.023)	(0.024)
pets kept at home throughout the years of child's life	--	--	0.007	--	-0.017	-0.019
			(0.009)		(0.014)	(0.015)
Tobacco exposure (whether anyone smokes in same room as child)	--	--	-0.010	--	0.005	0.006
			(0.011)		(0.020)	(0.020)
Efron's pseudo-R^2	--	0.032	0.034	--	0.035	0.026
Number of observations	7659	7659	7659	7659	7659	7659

Notes:

1. * Significant at the 10-percent level
   ** Significant at the 5-percent level
   *** Significant at the 1-percent level
2. Robust standard errors are in parentheses.
3. Estimates are partial effects.
4. Efron's pseudo-R^2 is equal to (see Efron, 1978; Veall and Zimmermann, 1996; Hoetker, 2007) :

$$1-\frac{\sum_{i=1}^{N}(y_i-\hat{\pi}_i)^2}{\sum_{i=1}^{N}(y_i-\bar{y}_i)^2}$$ where $\hat{\pi}$ is the predicted probability and N is the sample size.