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Child Health and the Income Gradient: Evidence from Australia

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Abstract

The positive relationship between household income and child health is well documented in the child health literature but the precise mechanisms via which income generates better health and whether the income gradient is increasing in child age are not well understood. This paper presents new Australian evidence on the child health-income gradient. We use data from the Longitudinal Study of Australian Children (LSAC), which involved two waves of data collection for children born between March 2003 and February 2004 (B-Cohort: 0-3 years), and between March 1999 and February 2000 (K-Cohort: 4-7 years). This data set allows us to test the robustness of some of the findings of the influential studies of Case et al. (2002) and J.Currie and Stabile (2003), and a recent study by A.Currie et al. (2007). The richness of the LSAC data set also allows us to conduct further exploration of the determinants of child health. Our results reveal an increasing income gradient by child age using similar covariates to Case et al. (2002). However, the income gradient disappears if we include a rich set of controls. Our results indicate that parental health and, in particular, the mother's health plays a significant role, reducing the income coefficient to zero; suggesting an underlying mechanism that can explain the observed relationship between child health and family income. Overall, our results for Australian children are similar to those produced by Propper et al. (2007) on their British child cohort.

Keywords: Child health, Income gradient, Parental health, Nutrition, Panel data, Australia
JEL Classification: I1

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1 Introduction

A growing literature documents a strong positive correlation between household income and child health (see for example, Case et al., 2002; Currie and Stabile, 2003; Propper et al., 2007; Case et al., 2007; Chen et al., 2006; Currie et al., 2007; Dowd, 2007; Case et al., 2008). Two pioneering and influential papers, by Case et al. (2002) and J. Currie and Stabile (2003), using US data and Canadian data respectively, established that the gradient is greater for older than for younger children. This finding of an increasing income-child health gradient is supported by another two recent studies by Condliffe and Link (2008) and Murasko (2008); although these two papers found smaller effects of income on child health than those reported by Case et al. (2002) and J. Currie and Stabile (2003). The studies that have examined the income-child health gradient after those pioneering papers (Case et al., 2002; Currie and Stabile, 2003) have not, however, always produced corroborative evidence of an age-increasing income-(child-)health gradient. For example, although Chen et al. (2006) documented a very significant effect of income on child health using same data set as Case et al. (2002) they did not find that the income-health gradient steepened with child age. Recent studies by A. Currie et al. (2007) and Propper et al. (2007) using the 1997- 2002 Health Surveys of England (HSE) and the Avon Longitudinal Study of Parents and Children (ALSPAC) respectively also found no evidence that the income-health gradient increased with age in their sample of British children. Several other English studies have also documented a relationship between socio-economic status (SES) and health that presents in childhood, but which either flattens or disappears in adolescence, only to reappear in adulthood (see for example, West,1997; and West and Sweeting, 2004).

Notably, Case et al. (2008) recently re-examined the HSE data and compared their findings with those of A.Currie et al. (2007). They established that the apparent differences in the income-health gradients for American and English children are less striking than those presented by A.Currie et al. (2007) when data from the same time period are compared. Case et al. (2008) used an expanded English sample by adding three more years of data from the HSE (1997- 2005), and compared the results with those from American NHIS data for the period 1998-2005. Their results showed that the income-health gradient for children does indeed increase with age in both the US and the UK. The income-health gradient for children was, however, smaller for the English sample than for that of the United States but slightly greater than that which was uncovered by A. Currie et al. (2007) for the UK. Thus, the literature presents mixed results on the hypothesis that the income-child health relationship is increasing in child age. Furthermore, the existing literature suggests that a gradient exists even in countries (e.g., the UK, Canada) with universal health care financing and delivery schemes.

There are many possible mechanisms via which income may affect child health even if health care is essentially "free" at the point of care. Greater income, *ceteris paribus*, creates greater opportunities for households to consume health and non-health inputs. The latter have been shown to be important sources of cross-sectional variation in health status in developed countries, where the marginal product of medical care may approach zero, but the marginal product of investments in education, etc. are still positive. In the only study to consider the effect of marginal health care services with an experimental design, *viz.* the Rand Health Insurance Experiment (Newhouse and the Insurance Experiment Group, 1993), there was little difference in pediatric health status by health plan. In relation to children in particular, rich households may be more efficient at producing child health. This may be due to a correlation between income and education, the latter of which enables greater allocative efficiency in health input selection; and/or the opportunity to buy more (or better) market inputs for the production of health. Alternatively, or additionally, higher incomes may be correlated with healthier environments (e.g., the physical environment, including housing), more nutritious diets, or more active lifestyles. On the other hand, higher incomes may also be correlated with health production "bads". For instance, assuming that the opportunity cost of time is higher for parents from higher-income households, market inputs may be substituted for parental-time inputs to a greater extent and the marginal heatlh product may be lower as a result. Examples may include the substitution of bought meals for home-cooked meals, or market childcare services for parental care. The empirical direction of the influence of such effects, which may be correlated with income are, in large measure, indeterminate *a priori*. Thus, there are good reasons to conduct an empirical investigation of the relationship between household characteristics –many of which may be correlated with income–and child health.

The empirical evidence on the mechanism(s) via which higher incomes produce better child health is also far from settled, although a small number of studies have explored this issue. Case et al. (2002) found that insurance, health at birth, and simple genetics could not explain the association between health and income in their sample, and concluded that the mechanisms underlying the income-child health association required further exploration. A.Currie et al.'s (2007) work answers this call by using the Health Surveys of England (HSE) to examine the effect of child nutrition (as measured, e.g. by fruit and vegetable consumption by children) and family lifestyle (as measured, e.g. by parental exercise) choices on child health. Interestingly, the inclusion of nutrition and family lifestyle in their analyses did not reduce the magnitude of the income-health gradient, suggesting that the roles of nutrition and lifestyle are important, possibly independent, determinants of child health status. Propper et al. (2007) found evidence that parental behaviour, and especially maternal health, also influences child health and, importantly, that the relationship between household income and child health disappeared when controls for parental health were used. Notably, the mother's health, particularly her mental health plays an important role in their models and effectively reduces the estimated effect of income *per se* to zero. In contrast, Dowd (2007) finds no significant mediator of the relationship between household income and child health. Therefore, the mechanisms by which income transmits to better health remain unresolved. This question is important to resolve for several reasons, not least of which is the potentially important role of health in the intergenerational transmission of economic status (J. Currie, 2008).

Thus, in this paper we examine the income-health gradient in young Australian children using two recent waves of data from the Longitudinal Study of Australian Children (LSAC). Of particular interest to us is this question of whether or not the income gradient increases with child age in our sample (i.e., from early- to mid-childhood). We address this question using parent-reported measures of overall health status and parental reports of chronic conditions that are likely to have been physician-diagnosed. We then direct our focus to an examination of the question of whether other child characteristics (e.g. child's diet) and parental attributes (e.g., health states) or behaviours (e.g., diet and exercise) attenuate the income-health relationship for children in our sample.

We contribute to the existing empirical literature in several ways. First, we produce the first econometric estimates of the income-health gradient for Australian children. Second, by using panel data we were able to account for the past investment made into child health (or cumulative effect of health) in the child health production function which have not been used extensively (with the exception of Murasko, 2008) in this literature, to examine the income-child health gradient. In fact, the previous literature on this topic such as Case et al. (2002) and A.Currie et al. (2007) have utilised cross-section data and hence were unable to account for the cumulative effect of health/health care used in the past on child health production. This represents an important addition to the literature, that is consistent with the conventional theoretical model of human capital accumulation Grossman (1972). Third, using the appropriate econometric techniques, we explore the relationship of some further variables that, in theory, could affect child health and examine whether or not these measures moderate the apparent income-health relationship for Australian children. Specifically, we present evidence on the roles of child's nutrition and parental health on health states of children. Thus, we are able to control for some variations in household characteristics that were not observable (i.e., may have constituted unobserved heterogeneity) in other influential studies. Finally, we compare our specifications of the model with those used in work of Case et al. (2002), J. Currie and Stabile (2003) and A.Currie et al. (2007) by estimating analogs of their models for our Australian birth cohorts. Doing so provides an insight into how the Australian results compare with those generated by other influential studies in this field.

In summary, our results represent novel empirical evidence on (i) the income-child health gradient for parental- and physician-reported child health, (ii) the mechanisms via which household income may affect child health status, and (iii) the relative gains that may be produced by applying sampling weight and clusters for robust estimates. This paper makes significant contribution to our understanding about the relationship between child health and family income. For example, the results of this paper show that the income-child health gradient is much smaller in Australia than that of the USA, and even Canada and the UK; the latter two of which, like Australia, have long-standing universal and compulsory health care financing schemes. This result underscores a fundamental point of health production that was originally made by Grossman

(1972): health production is a multivariate production process. By extension, one should not assume that access to health care services that are heavily subsidised or zero-priced nullifies the influence of income on health.

Our results show that the child health-income gradient is sensitive to the omission of confounders and controls, and the choice of age break. Furthermore, when we include a richer set of controls, including parental health, we find no evidence of an income-child health gradient at all. Our results indicate that parental health and, in particular, the mother's health play a significant role in this regard, reducing the income coefficient to zero when we account for it. Thus one important contribution of this paper is to show that parental health, particularly the mother's physical and mental health are factors that explain the univariate (and restricted multivariate) result of a positive relationship between child health and income in Australia.

2 Household Production of Child Health

Our theoretical model for the analysis of child health production derives from household production theory, which originated in the work of Becker (1965) and Becker and Lewis (1973), and was adapted by Grossman (1972) to analyse the accumulation and depreciation of health capital. The health production model, in which health capital is conceived as the output of a multivariate production process (Grossman, 1972; Behrman and Deolalikar, 1988; Liebowitz and Friedman, 1979; and Strauss and Thomas, 1994), provides the basis for our empirical modelling. Briefly, in this model it is assumed that the individual inherits an initial stock of health that depreciates over time, but also that the individual may positively influence the stock of health capital via gross investments. Gross investments in health capital can be made via combinations of the individual's own time and market goods such as medical care, diet, housing, exercise and lifestyle. The level of education of the producer also affects how efficiently he or she can produce health and is analogous to the technology of production or stock of knowledge in production theory more generally. Exogenous shocks thus may also affect a consumer's demand for health and the production of gross investments in health. Jacobson (2000) extended the model of Grossman (1972) by taking the family as the production unit. In her model, every individual in the family is both the producer of his or her own own health as well as the health of other family members. In this framework, the income of all family members is used in the production of the health capital of each member of the family. Thus, in one of her models, Jacobson (2000) considers a family unit that consists of a father, a mother and a child. In this model, the child is a passive participant in the production of its own health. She assumes that parents get utility from the good health of their child and can use total time available for market and non-market activities. Therefore, parents use inputs of market goods and their own time and resources to produce child health. This model may be regarded as an extension of Grossman's conception of the determinants of individual demands for health *viz.* as a consumption argument that enters the utility function directly (since sick days produce disutility), and as a derived demand, since sickness/wellness affects the total time available for market and non-market (production-) consumption activities.

Following these extensions, and in the vein of Rosenzweig and Schultz (1982, 1983), Rosenzweig and Wolpin (1988) and Jacobson (2000), suppose that the utility function for a family at time t can be written as

$$U_t = U(H_t, X_t, Y_t, L_{lt}; Z_{ut}, \varepsilon_{ut}) \tag{1}$$

where H_t is the health of a child , X_t is a set of goods that affects child health (e.g., food, toys and housing), Y_t represents other commodities consumed by the household, (L_{lt}) is the leisure time, Z_{ut} and ε_{ut} are exogenous observable and unobservable factors respectively that influence U_t.

Following the specification of the accumulation of health stock introduced in Grossman (1972; 2000), the production of child health is described as

$$H_t = H(H_{t-1}, X_t, L_{ht}; Z_{ht}, \varepsilon_{ht}) \tag{2}$$

where L_{ht} is the amount of time used in the production of child health, Z_{ht} and ε_{ht} are respectively exogenous observable and unobservable variables affecting H_t. In our study, since the LSAC data set consists of data for only one child per family, ε_{ht} may also pick up unobservable fixed family characteristics. To accommodate these fixed effects, and the likelihood that H is path-dependent (i.e., it may partially depend on the health state or health care consumption in a preceding period), a lagged value of H may be included in our empirical models.

The budget constraint of the household is

$$I_t = w_t L_{wt} = P_{xt} X_t + P_{yt} Y_t \tag{3}$$

where I_t is family income, L_{wt} is the time spend to earn wage income, w_t, P_{xt} and P_{yt} are respectively the wage rate, prices of X_t and Y_t.

The household also faces a time constraint

$$L = L_{lt} + L_{Ht} + L_{Wt} \tag{4}$$

where L is the total fixed amount of time available (e.g., 24 hours per day).

The household will maximise its intertemporal utility with the discount rate σ, i.e.,

$$\underset{H_t, X_t, Y_t, L_{lt}, L_{wt}, L_{ht}}{Max} \sum_{t}^{T} (1+\sigma)^{-t} U_t \tag{5}$$

subject to the budget and time constraints above, plus the condition of positive initial stock of child health ($H_0 > 0$).

Taking the first derivatives of the Lagrangian function with respect to child health, and taking its lag repeatedly until the initial condition is met, produces the Marshallian demand function for child health:

$$H_t^* = H(H_0,\, \omega_k;\, Z_{ht},\, Z_{ut},\, \varepsilon_{ht},\, \varepsilon_{ut}) \tag{6}$$

where $\omega = \{H,\, X,\, Y,\, L_l,\, L_w,\, L_h\}$ and $k = 1,\, 2,\, ...,\, t-1$.[1]

Equation (6) above shows that the optimal level of child health is determined by the allocation of parental time between income-generated work, household chores and leisure, the consumption of child-health related goods and other goods and services.

3 Data

3.1 Data Sources

This study utilises the data from the first two waves of the nationally representative Longitudinal Study of Australian Children (LSAC) (Australian Institute of Family Studies, 2007). The LSAC has so far involved two waves of data collection for more than ten thousand children. The LSAC collects data on these children every two years and will follow them until 2010 or beyond. The LSAC was conducted using both face-to-face interviews and survey instruments that were sent and retrieved via mail. The main topics covered include demographics, health status, education, the relationship history of parents, parenting practices, financial factors, lifestyle, housing and neighbourhood attributes.[2] The data were collected using a two-stage clustered sampling design with postcodes were used as the primary sampling unit (PSU). To ensure proportional geographic representation, postcodes were selected as a stratified sample by state of residence, and urban and rural geographical status. The sampling frame for the second stage consisted of all children born in the selected PSUs between March 2003 and February 2004 (B-Cohort, infants aged 0-1 years in 2004), and between March 1999 and February 2000 (K-Cohort, children aged 4-5 years in 2004) who were enrolled on the Health Insurance Commission's Medicare database. The Australian Medicare scheme is universal and compulsory; thus the sample constructed for the LSAC is generally representative of Australian children in these age cohorts, although children living in remote areas were not sampled.

The LSAC approach results in a sample frame that contains approximately 5000 children in each cohort, with an average of 20 children per cohort per postcode. The final respondent samples consist of 5107 and 4983 children in cohorts B and K, respectively, in Wave 1 (conducted in 2004). The numbers of children surveyed in Wave 2 (conducted in 2006) of the respective cohorts is slightly lower, primarily as a result of attrition, with 4606 and 4464 children retained in

[1] See, for example, J.Currie (2008) for a similar derivation of both the Frisch and Marshallian demand functions for child health.

[2] For a more comprehensive account of the LSAC sampling frame of the LSAC see Soloff et al. (2005).

cohorts B (aged 2-3 years in 2006) and K (aged 6-7 years in 2006), respectively. The attrition rates are therefore 9.8 and 10.4 per cent for B and K cohorts, respectively. The logistic regressions conducted by Mission and Sipthorp (2007, Tables 1-2) reveal that attrition occurred mostly at random in the LSAC. However, attrition was slightly more likely if Parent 1 (primary caregiver) is a young male, the household was living in a rented home, or in an areas with a lower socio-economic status index. For the B-cohort, attrition was also more likely to occur among households in areas where fewer people in the postcode speak only English in the home.

In order to take the advantage of the survey's design characteristics , all analyses presented in this paper apply the sampling weights of the LSAC. These are computed as the inverse of the probability of a child being selected for inclusion in the LSAC sample. For example, if the probability of a child is being sampled is 0.20, the weight given to that child's response is 5.0. In addition, cluster information are used to produce correct variances of the estimates as there is less variations among variables within a cluster (i.e., postcode). This approach also corrects for the fact that the variance is reduced in a finite population with non-replacement sampling (i.e., in non-replacement samples, the population being sampled is reduced as the sampling progresses; and the variance is thereby reduced).

3.2 Choice of Variables

3.2.1 Child Health

As with the foregoing literature on income and child health (see for example, Case et al., 2002; J. Currie and Stabile, 2003; A. Currie et al., 2007), our measure of child health is constructed from the following question that was asked of the child's primary care-giver (Parent 1)[3]: "*In general, how would you say child's current health is?*". The responses were recorded on a five-point Likert scale upon which 1 is "Excellent" 2 is "Very good"; 3 is "Good"; 4 is "Fair" and 5 is "Poor". The proportion of children inexcellent, very good, good, fair and poor health of the sample are 56.1, 31.1, 10.1, 2.3 and 0.3 per cent, respectively. There a decrease of proportion in "excellent" health whilst the proportion of "very good" health increase slightly between the two waves for both cohorts; other health categories show little variations (see Figure 1). Other researchers have found that there are typically very few respondents in the "Poor" health category: in the LSAC approximately 0.30 per cent of the children sampled fell into this category. Some authors (e.g., A. Currie et al., 2007) have chosen to merge the lowest and second-lowest health state categories as a response to the (relatively) small number of observations in the "Poor" health category. Since there are no shortage of degrees of freedom in our study, we do not compress

[3]In principle, Parent 1 is the person in the family who knows the most about the study child. In most cases this is the child's biological mother but, alternatively may be the biological father, a step-parent, an adoptive parent, a guardian, or someone else who has a parental/guardian relationship with the child.

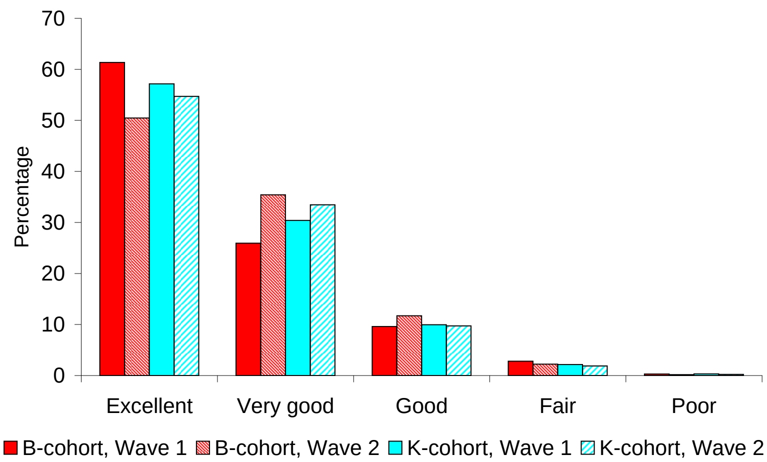

Figure 1: Child health status by waves and cohorts

the "Fair" and "Poor" categories of child health. Thus, our dependent variable for parent-reported overall child health contains the five original categories.[4]

One concern regarding this measure of overall health is that it is subjective and that it may be biased by correlation with some other unobservable variables. For example, there is the possibility that maternal reporting of child's health might be affected by the mother's own health state. Some previous studies (e.g., Dadds et al., 1995; Case et al., 2002) have examined this proposition, but found no empirical support for it. Nevertheless, we also employ other child health measures which should be less prone to this source of bias, if it exists. A good candidate among the measures that are available to us is whether the child is subject to any long-term medical condition. Such conditions are likely to have been diagnosed by a medical practitioner. In the LSAC, Parent 1 was asked whether or not the child had a long-term medical condition, the nature of the condition and whether the child had experienced any developmental delays that were attributable to the problem, compared to children of a similar age. If the answer was yes, the respondent was asked to check up to fourteen chronic conditions. Approximately twenty-three (22.95) per cent of survey children in the LSAC were reported to have at least one such condition, and 6.42 per cent have more than one such condition. Furthermore, the LSAC contains information on whether the child has asthma or bronchiolitis, *as diagnosed by*

[4]Nevertheless, we also conducted analysis with the last two categories recoded and the results show little differences. These estimates are available from the authors upon request.

a health professional.[5] The survey revealed that 19.19 and 13.27 per cent of children, respectively, were reported to have been diagnosed with asthma and bronchiolitis.

3.2.2 Income

In our empirical analysis annual income of the mother and the father (i.e., referred to as "family income" hereinafter) will be used to proxy parents' time spent for earning income, which is L_w in our theoretical model. Following the treatment of income in longitudinal studies of Dowd (2007), Condliffe and Link (2008) and Murasko (2008) we take the average of family income in the two waves, expressed in 2004 (Wave 1) dollar using the Australian national Consumer Price Index (CPI) for the study period (Australian Bureau of Statistics, 2008), as a proxy for permanent income. In the analysis, we use the natural logarithm of average household income to mitigate the well known property (i.e., sharply skewed distribution) of income (see, for example, Mincer, 1958; Petrou et al., 2007).[6]

Child Health and Income: A Raw Sketch of the Gradient

Figure 2 presents a plot of income and parent-reported child's health from the LSAC using the locally weighted polynomial regression (lowess) plot. It shows the expected, positive univariate correlation of child health and household income. However, it is quite obvious that the health-income gradient is increasing in child age (cf, in particular, children in the excellent health status). For other health status, the difference is less distinguishable especially for children in the K-cohort (i.e., age 4-5 and 6-7). This issue is investigated in more depth, and in a multivariate framework, in our econometric analyses.

3.2.3 Other Variables

Based on the availability of data and the analytical model presented in Section 2, other covariates consist of the following groups:

Demographics

We use age and gender of the child , age of parents, the presence of the biological mother and father in the household, parental education and employment, identification as an Aboriginal or Torres-Strait Islander, English speaking household (as a measure of Z_{ht}), household size (Z_{ut} & Z_{ht}), housing condition (X_t),

[5]The survey questions for this variable is "Has a doctor ever told you that you child has: asthma?, bronchiolitis?".

[6]We also re-estimated Specification 4 with income specified as (a) contemporaneous income (i.e., Wave 2 income) and (b) lagged income (Wave 1 income). No changes in statistical significance were recorded although using lagged income reduced the magnitude of the income coefficient by more than half. The coefficients and standard errors on contemporaneous income were not much different to those estimated on our permanent income proxy. We thank an anonymous referee for the suggestion that we explore this issue.

child's birthweight (as a measure of child's initial stock of health, H_0), prior health state of the child (as a measure of child's health stock in the preceding period, H_{t-1}), and breastfeeding (as a measure of postnatal health inputs (Z_{ht}) and mother's time input (L_{ht}) into production of child health). Applying these controls for child characteristics and family characteristics allows us to control for as much of the unobserved child and family fixed effects as possible. We will refer to this set of controls as the "standard background controls" in the rest of the paper.

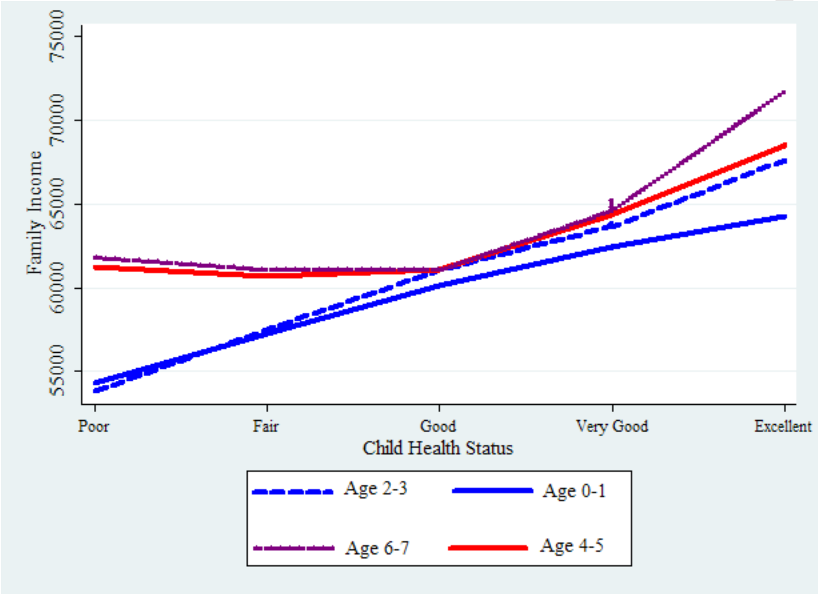

Figure 2: Lowess plot of income and health status by age groups

Source: Computed from the Longitudinal Study of Australian Children (Australian Institute of Family Studies, 2007)

Parents' Physical and Mental Health

Case et al. (2002) argued that "parental health is a "third factor" that accounts for the income gradient in children's health". Following this logic and in line with our theoretical model, we include measures for parental physical health (measured in a 5-point Likert scale, 1= excellent, 5 = poor) and mental health. Our measure of parental mental health is constructed from a variable (in LSAC) which is the mean of the responses of six questions regarding parents' depression scale.[7] Inclusion of parental depression scale in the model enables us to examine the importance of maternal health, which Propper et al. (2007) recently found dominated the effect of household income in their UK sample.

Nutrition

Our theoretical model suggests that the child's diet (a component of X_t) is an important input in the production of child health, as was recently found by A.Currie et al. (2007). We explore this issue using the LSAC which contains even more detailed measures of children's dietary intake than were available to A.Currie et al. (2007). Specifically, we include indicators for the consumption of foods that are high in fat or sugar.

Parents' Health Related Behaviour and Lifestyle Measures

The existing evidence (e.g., Case and Paxson, 2002) suggests that socioeconomic status affects parental lifestyle decisions and child health. Parents from high SES backgrounds are more likely to have healthy lifestyles. The lifestyle factors selected in this study include exercise (which is L_l of our theoretical model and measured by the number of days per week in which at least 30 minutes of rigorous physical activity was undertaken), dietary habits (measured by the number of servings consumed per day of fresh fruits and vegetables, which reflect Y_t of our theoretical model), the consumption of cigarettes (which reflects X_t and measured by a dummy variable =1 if the respondent is a smoker), and alcohol (which also reflects X_t and measured by dummy variable=1 if the respondent consumes alcohol several times per week to daily). In general, parental lifestyle factors are also used to proxy Z_{ut} and Z_{ht} in our theoretical model, which we expect will minimise much of unobserved factors in the family.

The descriptive statistics for the main variables are presented in Table 1. It is noteworthy that the mean estimates, using the survey design adjustment, produce much smaller standard deviations than those that one would estimate by assuming that the data are collected using simple random sampling.[8] This owes to the "design effect", whereby the variance of individuals within a cluster is less than that expected from a simple random sample (Kerry and Bland, 1998; Connelly, 2003). Also note that, by applying the survey clustering adjustment, the computed sample means may be interpreted as approximates of the population means for Australia.

[7]The depression scale is measured using six questions asked of the mother and father of the study child, *viz.*: (1) In the past 4 weeks about how often... Did you feel - nervous? (2) hopeless? (3) restless or fidgety? (4) that everything was an effort? (5) so sad that nothing could cheer you up? (6) worthless ? The responses are recoded in 5 point scale:1= depressed all the time, 5= not depressed at all. The final mental health variable, which is constructed from the mean of these question, takes values between 1 to 5.

[8]For brevity, standard deviations estimated by assuming that the data were collected by simple random sampling were not reported but can be made available from the authors upon request.

Table 1: Descriptive statistics

	Both-Cohorts		B-Cohort (0-3yr)		K-Cohort (4-7 yr)		Test B=K
Descriptions	Mean	Std.	Mean	Std.	Mean	Std.	(p-value)
Previous health state of a child	1.53	0.01	1.52	0.02	1.55	0.02	0.00
Log of average household income	11.07	0.01	11.05	0.02	11.10	0.02	0.03
Child's age (months)	57.47	0.33	33.81	0.06	81.90	0.07	0.00
Child's gender (1=male)	0.52	0.01	0.52	0.01	0.52	0.01	1.00
Aboriginal or Torres-Strait Islander (1=yes)	0.02	0.00	0.02	0.00	0.01	0.00	0.01
English speaking household (1=yes)	0.90	0.01	0.92	0.01	0.89	0.01	0.01
Birth weight<2500 gram	0.05	0.00	0.05	0.00	0.06	0.00	0.03
The child is breastfed (1=yes)	0.94	0.00	0.94	0.01	0.94	0.01	0.00
Log of household size	1.47	0.00	1.43	0.00	1.51	0.00	0.00
Mother's age	35.80	0.10	34.04	0.12	37.60	0.12	0.00
Father's age	38.09	0.11	36.26	0.13	39.97	0.13	0.00
Housing condition (1= all rooms are uncluttered)	0.95	0.00	0.95	0.00	0.95	0.01	0.63
Mother completed Year 12	0.63	0.01	0.68	0.01	0.58	0.01	0.00
Mother has undergraduate qualification	0.27	0.01	0.29	0.01	0.25	0.01	0.00
Mother has postgraduate qualification	0.08	0.00	0.09	0.01	0.06	0.01	0.00
Father completed Year 12	0.57	0.01	0.61	0.01	0.54	0.01	0.00
Father has graduate qualification	0.23	0.01	0.24	0.01	0.22	0.01	0.35
Father has postgraduate qualification	0.09	0.01	0.09	0.01	0.10	0.01	0.00
Mother is employed (1=yes)	0.65	0.01	0.61	0.01	0.69	0.01	0.00
Father is employed (1=yes)	0.96	0.00	0.96	0.00	0.96	0.00	0.77

Continued over...

Table 1: Continued

	Both-Cohorts		B-Cohort (0-3)		K-Cohort (4-7)		Test B=K
Descriptions	Mean	Std.	Mean	Std.	Mean	Std.	(p-value)
Parents' Physical and Mental Health							
Mother's health (1=excellent/very good, 0=good, fair and poor)	0.68	0.01	0.69	0.01	0.66	0.01	0.00
Father's health (as above)	0.64	0.01	0.65	0.01	0.63	0.01	0.01
Mother's depression scale (1=very depressed, 5=not depressed)	4.55	0.01	4.57	0.01	4.54	0.01	0.00
Father's depression scale (as above)	4.49	0.01	4.49	0.01	4.48	0.01	0.22
Child's Nutrition							
Fruit & vegetable (serves of fruit and veg in last 24 hours)	3.16	0.02	3.18	0.03	3.13	0.03	0.00
Dairy product (full cream and skim milk in last 24 hours)	1.64	0.01	1.69	0.02	1.59	0.02	0.00
Sugary drink (soft drink or cordial in last 24 hours)	0.49	0.01	0.40	0.02	0.59	0.02	0.00
High fat food (serves of high fat food in last 24 hours)	1.19	0.01	1.13	0.02	1.24	0.02	0.00
Parents' Lifestyle							
Mother's fruit & vegetable intake (serves/day)	3.76	0.03	3.74	0.04	3.79	0.04	0.28
Father's fruit & vegetable intake (serves/day)	3.36	0.03	3.27	0.04	3.46	0.05	0.00
Mother's exercise (active days/week)	2.79	0.03	2.65	0.04	2.93	0.04	0.00
Father's exercise (active days/week)	3.19	0.03	3.20	0.04	3.18	0.05	0.21
Father smokes (1=yes)	0.19	0.01	0.19	0.01	0.14	0.01	0.03
Mother smokes(1=yes)	0.13	0.01	0.13	0.01	0.14	0.01	0.07
Father drinks(1=yes)	0.76	0.01	0.77	0.01	0.75	0.01	0.02
Mother drinks(1=yes)	0.57	0.01	0.55	0.01	0.59	0.01	0.00

Notes: (i) Variances are estimated using the survey design adjustment, which invokes the Taylor linearisation method (Kish, 1995; Chambers and Skinner, 2003). (ii) Tests for the differences of mean/median between the B and K-Cohorts are t-tests for continuous variables and χ^2 tests for categorical variables.

Source: Computed from the Longitudinal Study of Australian Children (Australian Institute of Family Studies, 2007)

4 Econometric model

An empirical formulation of the dynamic health demand function, equation (6) can be written as

$$H_{it} = \alpha H_{i(t-1)} + \beta I + \gamma Z_{it} + \eta_{it} \quad (i = 1, ...n;\ t = 1, 2) \tag{7}$$

where H_{it} is the stock of health of child i in period t (in this case, the LSAC Wave 2), $H_{i(t-1)}$ is the stock of health of child i in period $t-1$ (in this case, the LSAC data Wave 1), I represents average income of the family (i.e., log of average CPI-adjusted family income in Wave 1 and Wave 2) and is our proxy for permanent income, Z_{it} is a set of exogenous variables that affects child health and η_{it} represents unobservable determinants of H. The error term in equation (7) has two components

$$\eta_{it} = u_i + e_{it} \tag{8}$$

where $u_i \sim i.i.d.(0, \sigma_u^2)$, is a child-specific component that captures time-invariant unobserved factors. The $e_{it} \sim N(0, \sigma_e^2)$ is a child-specific time varying component of the error term, which captures the effects of other unobserved factors that affect child health. It is assumed that e_{it} is exogenous and serially uncorrelated.

One problem with the estimation of the panel ordered probit specified in equation (7) is the possible correlation between the error term and the lag of dependent variable if the assumption on the exogeneity of initial observations does not hold. The main culprit of such correlations, if they exist, is likely to be unobserved effects caused by the heterogeneity of households and individuals. It is noteworthy that, as we are restricted to only two waves of data, it is not feasible to implement standard treatments such as initial condition assumption of Wooldridge (2005), GMM-based estimate of Arellano and Bond (1991), and the fixed-effects ordered logit estimate of Ferrer-i Carbonell and Frijters (2004), to deal with this problem. As T=2 in LSAC data, the dynamic specification can only be treated as a cross-section model, and this rules out panel data specifications of the heterogeneity (e.g., a fixed effects ordered logit model).[9] In particular, the inclusion of the pre-determined variable H_{t-1} in equation (7) leaves us only with observations from Wave 2 (i.e., a lag of Wave 1 is not available, so observations from Wave 1 are excluded from the analyses). Thus the approach adopted in this paper is not a panel approach but an approach that takes advantage of the availability of (only 2 waves of) panel data to improve the specification of the model by including the lagged health state. This is a theoretical improvement because, according to the theory of health production (Grossman, 1972; 2000), health stock in the present period depends upon health stock in the preceding period, plus net investment. Whether it is an

[9]We are grateful to Andrew M. Jones for making this comment.

empirical improvement is open to question given the restricted panel available to us.Although unobserved heterogeneity remains even in cross-sectional data, we mitigate this issue as far as is possible by producing robust-variances for all estimates using sample weight and cluster information from the survey data. Although there may be individual unobserved heterogeneity, for which there is no feasible solution, our adjustment does account for unobserved heterogeneity between the clusters in the sample. In this regard, we are in the same position as Condliffe and Link (2008) and Murasko (2008), both of whom encountered the same problem and hence were unable to adjust for unobserved heterogeneity. Thus, the approach adopted in this paper to deal with panel data is in line with Currie and Stabile (2003), Condliffe and Link (2008) and Murasko (2008). We are not aware of any other method(s) in this literature that can be used to control for unobserved heterogeneity in our dynamic model given the two time periods of LSAC data. An alternative is an instrumental variable approach, however we could not identify a suitable instrument for this purpose and, as Murasko (2008: p.1501) has noted, to do so for health status is notoriously difficult. Thus, unobserved heterogeneity remains a potential source of biasedness in our estimates; but it is a problem that does not have a feasible solution, given the data available.

Given the ordered nature of the Likert parent-reported health states of children, we invoke an ordered probit model to analyse the latent health status of children. For the ith child, assuming that there is an underlying response variable H_{it}^* that is defined by the relationship:

$$H_i^* = \alpha Z_i^* + \eta_i$$

where α is the vector of coefficients, Z_i^* is a vector of explanatory variables (i.e., income, demographics, lifestyles) and η_i is a random error.

In practice H_i^* is a latent dependent variable, and the observed counterpart (or indicator) of it is denoted by H_i, which may be specified as follows:

$$H_i = \begin{cases} 1\, if \; -\infty < H_i^* \leq \mu_1 & (if\, the\, child\, has\, excellent\, health) \\ 2\, if\; \mu_1 \leq H_i^* \leq \mu_2 & (if\, the\, child\, has\, very\, good\, health) \\ 3\, if\; \mu_2 \leq H_i^* \leq \mu_3 & (if\, the\, child\, has\, good\, health) \\ 4\, if\; \mu_3 \leq H_i^* \leq \mu_4 & (if\, the\, child\, has\, fair\, health) \\ 5\, if\; \mu_4 \leq H_i^* < \infty & (if\, the\, child\, has\, poor\, health) \end{cases}$$

where $\mu_1 - \mu_4$ are threshold parameters that denote the cut-points between one health state and another. Under the assumption that the error term is normally distributed, the probability of observing a particular category of the health status of a child from changes in the explanatory variables is

$$prob(H_i = 1) = \phi(\mu_1 - \alpha Z)$$
$$prob(H_i = 2) = \phi((\mu_2 - \alpha Z) - (\mu_1 - \alpha Z))$$
$$prob(H_i = 3) = \phi((\mu_3 - \alpha Z) - (\mu_2 - \alpha Z))$$
$$prob(H_i = 4) = \phi((\mu_4 - \alpha Z) - (\mu_3 - \alpha Z))$$
$$prob(H_i = 5) = 1 - \phi(\mu_4 - \alpha Z)$$

where ϕ is the cumulative normal distribution function, and the sum total of the above probabilities is equal to one. We maximise the log-likelihood function to obtain the estimates of α and μ. The parent-reported general health states and chronic conditions are ordered categorical and binary variables, so ordered probit and probit regressions, respectively are utilised.

In order to utlise the survey characteristics, all estimates (probit and ordered probit) in this study are produced using the pseudo-likelihood techniques, in which parameters' likelihood function is weighted using sample weights while variances of the estimated parameters are estimated using the first-order Taylor series expansion.[10] This econometric measure enable us to take is to account for the cluster sample nature of our data set and produce robust variances of the cluster sample estimates. As mentioned previously, variances were estimated by applying the survey design of LSAC (i.e., two-stage cluster sampling) are smaller than those produced by assuming that the data were collected from simple random sampling.

5 Results and Discussion

In this section we first estimate specifications that are close analogs of the models invoked by Case et al. (2002), J.Currie and Stabile (2003) and A. Currie et al. (2007) to examine income-child health gradient using similar variables as Case et al. and Currie et al., on cross-sectional analyses. We refer to these specifications as "Specification 1" and "Specification 2". In addition to these two specifications, we estimate another specification ("Specification 3") to account for some additional child and family specific factors. We then proceed to estimate a more general model ("Specification 4") that includes additional covariates that are available to us in the LSAC, a model based on the analytical model presented in Section 2. Our motivation for this approach is as follows: we view the existing models as nested, specific, forms of more general formulations that include the latter variables, which also help us to explore the mechanisms that can explain the relationship between child health and income . Our objective in presenting the results of estimates from both the specific and general forms is not simply to present new empirical data on the Australian sample, but to provide estimates of the orders of magnitude of the income-child health gradient that differently-specified econometric models may produce, especially when one is able to exploit panel and other sample properties in the econometric specification.

[10] For more information about the pseudo-likelihood estimate with survey data, see for example, Kish (1995), and Chambers and Skinner (2003).

5.1 Are Household Income and Parental Education Endogenous?

An examination of the household income-child health gradient that does not consider the potential endogeneity of household income is subject to serious criticism. Even if Australian children are unlikely to have a direct effect on household income in Australia (because they are unlikely to be put to work, irrespective of their health status), child health may affect the labour market decisions of parents. Specifically, if poorer child health states reduce parental earnings (e.g., via participation, wages and hours worked) income may still be endogenous with respect to child health. An analogous problem may be associated with parental educational attainment although this source of endogeneity seems *a priori* less likely, because presumably only post-partum education decisions may be affected by child health.

The possibility of income and education endogeneity was examined by Doyle et al. (2007), using an instrumental variables approach. In that study, the effects of parental income and education on health were greater when those variables are treated as endogenous, suggesting that the estimated effect of income and education were downwards-biased when the endogeneity problem was unaddressed. In the LSAC data set we could not identify instruments that would allow us to follow such an approach. However, we did test for endogeneity using the generalised Hausman test.[11] The resulting test statistics suggest that both household income and parental education may safely be treated as exogenous variables for the purposes of this paper.[12]

5.2 The Income Gradient

To see whether the income-child health gradient is increasing in child age, we compare estimates from LSAC data with those of Case et al. (2002), J. Currie and Stabile (2003) and A. Currie et al. (2007), using the same age groups (i.e., 0-3 and 4-8) and similar covariates to those used in the original studies. "Specification 1" includes the dummies for age and gender of the child, log of the household size, a dummy for the survey wave, race (Aboriginal and Torres-Strait Islander status), whether the biological mother and father present in the house, the age of the mother and the father, and the person responding to the survey questions. "Specification 2" includes all controls from Specification 1 plus parents' education and employment. We observe an increasing income-health gradient for children in these two age groups, irrespective of whether or not

[11] The original Hausman test cannot be applied in this study as the assumption that at least one specification is efficient (i.e., asymptotically has minimum variance) is violated in clustered survey data, where variances differ from each cluster (StataCorp., 2005). The generalised Hausman test, in essence, is an adjusted Wald test that compares a model with income as a regressor and a model without income as a regressor. If income is endogenous, the estimates will be biased and hence, the point estimates of common covariates of the two models (i.e., with and without income) will differ.

[12] The test did not reject the null hypothesis that income and education of parents are exogenous. The respective test statistics are F(37,234)= 0.78 and F(25,246)=1.28.

parental education is included (see Table 2).

Table 2: Comparisons of Australian income-child health gradient estimates with existing estimates from Canadian, US and UK samples (ordered probit models)

Child's age	**Australia** (This paper)	**United States** (Case et al. 2002)	**Canada** (J.Currie and Stabile 2003)	**United Kingdom** (A.Currie et al. 2007)
Specification 1				
0-3 years	*-0.050	*-0.183	*-0.151	*-0.146
(n=7879)	(0.024)	(0.008)	(0.026)	(0.040)
4-8 years	*-0.131	*-0.244	*-0.216	*-0.212
(n=8725)	(0.024)	(0.008)	(0.019)	(0.028)
Specification 2				
0-3 years	*-0.059	*-0.114	*-0.132	*-0.142
(n=7865)	(0.026)	(0.008)	(0.027)	(0.045)
4-8 years	*-0.116	*-0.156	*-0.182	*-0.136
(n=8712)	(0.027)	(0.008)	(0.020)	(0.032)
Specification 3	**Australia** (this paper)			
	0-3 years (n=7730)		4-8 years (n=8509)	
	-0.029		*-0.063	
	(0.025)		(0.027)	

*Notes: (i) The dependent variable is an ordered categorisation of the child's general health status (e.g., 1= excellent, 2 = very good, 3 = good, 4= fair and 5= poor) as reported by a parent/guardian. (ii) As the LSAC data are only available for children aged 0-8, we report the results for same age groups from previous studies, though those studies also included children older than 8 years. (iii) Specification 1 includes: age and wave dummies, sex, race of the child, log of household size, the presence and age of biological parents, and dummy for persons response to the survey. (iv) Specification 2 includes the variables in Specification 1 plus parents' education and employment. (v) Specification 3 includes the variables in Specification 2 plus housing conditions, birthweight and breastfeeding . (vi) Standard errors are reported in parentheses. (vii) * Significant at the five per cent level.*

Sources: Case et al. (2002), J.Currie and Stabile (2003), A.Currie et al. (2007). Australian estimates were computed from the Longitudinal Study of Australian Children (Australian Institute of Family Studies, 2007).

Furthermore, we find that the magnitude of the income gradient in our data is smaller than in these studies of US, UK and Canadian children. Indeed, our coefficients are about one third of the magnitude of those produced by previous studies for the 0-3 years age group and approximately one-half of those produced for 4-8 year-old. The smaller income gradient for Australia compared with the UK and Canada (in particular presented in A. Currie et al., 2007 and J. Currie and Stabile, 2003) is noteworthy since all three countries have universal health care financing insurance and relatively generous government support for children from low income families. Although the literature suggests that the steepening income gradient might be flattened or disappear for children older than 8 years of age (A. Currie et al., 2007; West, 1997; West and Sweeting, 2004) this hypothesis cannot be tested using data from the first two waves of the LSAC, in which children only aged up to seven years.

We hypothesise that both Specifications 1 and 2 may suffer from omitted variable bias because of the small set of controls used in these specifications. We suspect that the health-income gradient found in Specification 1 and 2 may be sensitive to the omission of confounders and controls. Therefore, we estimate Specification 3 (by adding controls for low birthweight, breastfeeding, and housing conditions to Specification 2). In this specification, birthweight and breastfeeding are regarded as indicators of the child's initial stock of health and post-natal health inputs, respectively. We believe that accounting for this initial health stock and health inputs flow may substantially improve the estimates of the income-child health relationship. The results indicate an increasing income-child health gradient although estimates of the younger age group (0-3 years old) were statistically insignificant in Specification 3.

The choice of age break is not explained in previous studies and it is possible that the income gradient may be sensitive to changes in choices of age break (Harris et al., 2008). We then examine whether the income gradients that were found in these regressions persist if we use a different choice of (LSAC defined) age breaks (see Table 3). The results also reveal an increasing income gradient (increasing magnitude for income coefficient) but the coefficients on income are insignificant for young age groups (with the exception of Specification 1); significant estimates are only found for children in the 6-7 years age group (i.e., K-Cohort Wave 2) in our cross-sectional analysis. These results indicate that income-child health gradient is sensitive to both the choice of covariates and the selection of age groups. Case et al. (2008, p.7) also note that the differences in such results across countries may be attributable to "different surveys - with different wording of questions, data collection protocols and sample sizes".

Table 3: Income-child health gradient estimates for Australian children with disaggregated age groups (ordered probit models)

		Spec1	*Spec2*	*Spec3*
B-Cohort				
	Wave 1 (0-1 year of age)	-0.041	-0.059	-0.028
		(0.029)	(0.030)	(0.030)
	Wave 2 (2-3 years of age)	*-0.067	-0.065	-0.034
		(0.032)	(0.037)	(0.037)
K-Cohort				
	Wave 1 (4-5 years of age)	*-0.086	*-0.092	-0.052
		(0.027)	(0.031)	(0.032)
	Wave 2 (6-7 years of age)	*-0.195	*-0.151	*-0.083
		(0.031)	(0.034)	(0.033)

Notes: As for Table 2.

Source: As for Table 1.

It can also be seen from Table 3 that the magnitude of the income gradient increases with age despite the fact that the estimates are insignificant for the B-Cohort (0-3 years). We now subject this hypothesis to further testing by constructing a model (Specification 4), taking into account additional factors that may affect the child health and the income gradient.

5.3 Determinants of Child Health

The determinants of child health estimated by "Specification 4" are presented in Table 4. The results show that the income is no longer statistically significant in this model. We explore the reasons for this in following section.

We find the expected results for the English-speaking variable which suggests that children of non-English speaking households may face the cultural barriers, latent educational deficits, or other unobservable effects that are correlated with the difficulty of using the official language. The initial stock of health, proxied by birthweight, significantly increases the probability of having good health, particularly for the B-cohort (0-3 years). Parental education appears to be a weak determinant of child health in Australia, as the mother's education is only significant at the 10 per cent level, in the pooled model; the father's education is significant only for the K-Cohort (4-7 years).[13] However, parental education starts to affect child health if the parent has more than a graduate qualification. The child's current health is strongly related to its reported health state in the preceding period which is consistent with the prediction of our theoretical model, and of Grossman (1972), more generally. As previous studies, such as Case et al (2002), A Currie (2007) and Propper et al (2007), were unable to control for previous the child's existing health stock, this is novel empirical result. It is important inasmuch as the stock of human capital is fundamental in the theory of health capital (Grossman, 1972) and its omission in other studies – due to the unavailability of such measures in the authors' datasets – could result in omitted variable bias.

Now we turn to the discussion of parents' physical and mental health. With the exception of the father's mental health, all remaining measures of parental health affect the child's (parent-rated) health in a statistically significant way, and the coefficients have the expected signs. In particular, a child is more likely to have better health if his/her parents enjoy good health (Table 4); while children of depressed mothers are more likely to have poor health.

The results on our nutrition variables show that indicators of child nutritional intake are significantly associated with the parental-rating of their child's health. The consumption of fruit, vegetables and dairy products in particular appear to contribute to parent-assessed child health. In contrast, the consumption of high fat food is significantly correlated with poorer child health, which is consistent with our theoretical model. It is obvious, though, that the children in the B-Cohort (0-3 years) have a low propensity to consume such products due to their

[13] As we consider only five per cent significance level, so these variables appear as insignificant in Table 3.

Table 4: Determinants of child health in Australia (ordered probit models)

Variables	Both-Cohorts (n=4590)		B-Cohort (0-3 yr) (n=2312)		K-Cohort (4-7 yr) (n=2043)	
	Coef.	Std. Err.	Coef.	Std. Err.	Coef.	Std. Err.
previous health state of a child	*0.390	0.024	*0.296	0.031	*0.507	0.036
Log of average household income	-0.046	0.036	-0.023	0.049	-0.067	0.047
Child's age	*-0.004	0.001	-0.007	0.009	-0.001	0.008
Child's gender	0.028	0.035	0.009	0.049	0.049	0.048
Aboriginal and/or TS Islander	0.182	0.133	0.071	0.166	0.308	0.250
English speaking household	*-0.281	0.065	*-0.311	0.098	*-0.235	0.085
Birthweight <2500gm	*0.257	0.078	*0.411	0.115	0.126	0.100
The child is breastfed	0.064	0.070	-0.001	0.101	0.123	0.103
Log of household size	-0.071	0.088	0.087	0.122	-0.204	0.137
Mother's age	0.006	0.005	0.000	0.006	0.014	0.007
Father's age	0.002	0.004	0.001	0.006	0.001	0.006
Housing condition	-0.051	0.078	-0.014	0.112	-0.137	0.128
Biological father is in the household	-0.217	0.165	-0.443	0.350	-0.193	0.198
Biological mother is in the household	-0.227	0.395	-0.238	0.606	-0.124	0.474
Mother completed year 12	0.022	0.038	0.037	0.056	0.005	0.054
Mother has graduate qualification	0.019	0.040	0.019	0.057	0.041	0.060
Mother has postgraduate qualification	-0.130	0.069	-0.087	0.087	-0.148	0.105
Father year 12	0.023	0.041	-0.025	0.053	0.062	0.055
Father has graduate qualification	-0.057	0.043	-0.009	0.059	-0.121	0.067
Father has postgraduate qualification	-0.010	0.058	-0.029	0.076	0.004	0.090
Mother employed	0.018	0.035	0.075	0.045	-0.054	0.057
Father employed	0.131	0.097	0.234	0.146	0.031	0.136
Parents' Physical and Mental Health						
Mother is in good health	*-0.406	0.037	*-0.397	0.058	*-0.416	0.053
Father is in good health	*-0.104	0.035	*-0.137	0.057	-0.057	0.048
Mother's depression scale	*-0.159	0.038	*-0.148	0.055	*-0.173	0.054
Father's depression scale	-0.034	0.032	-0.040	0.044	-0.045	0.048
Child's Nutrition						
Consumption of fruit & veg	*-0.075	0.014	*-0.112	0.019	*-0.04	0.019
Consumption of dairy product	*-0.098	0.021	*-0.104	0.031	*-0.087	0.031
Consumption of sugary drink	0.027	0.022	0.052	0.033	0.014	0.031
Consumption of high fat food	0.006	0.022	-0.056	0.030	*0.061	0.031
Parents' lifestyle						
Mother's consumption of fruit & veg	*-0.019	0.009	*-0.029	0.013	-0.008	0.014
Father's consumption of fruit & veg	0.007	0.009	0.011	0.012	0.005	0.016
Father's level of exercise	-0.004	0.008	0.004	0.012	-0.012	0.012
Mother's level of exercise	0.004	0.009	0.011	0.013	-0.004	0.013
Father smokes	-0.024	0.048	-0.079	0.069	0.041	0.072
Mother smokes	-0.049	0.057	-0.003	0.078	-0.112	0.091
Father drinks	0.068	0.043	0.111	0.065	0.039	0.056
Mother drinks	*-0.156	0.037	*-0.134	0.051	*-0.178	0.055

Notes: As for Table 2.

Source: As for Table 1.

age. So it is not surprising that the variable is statistically significant only in K-Cohort (4-7 years). These finding regarding child nutrition are in line with the findings of A. Currie (2007) who found that nutrition was an important determinant of child health in the UK.

Interestingly, the results on parental lifestyle variables suggest that most parental lifestyle factors have no detectable, independent effect on child health. However, the maternal consumption of fruit and vegetables has a protective effect, particularly in the young, B-Cohort (0-3 years). It is also somewhat surprising to see that, compared to the base group of non-smokers and non-drinkers, children from parents who smoke and drink do not have significantly lower parent-rated health states. A puzzling finding is that children from mothers who consume alcohol frequently are more likely to be reported as having good health than children from mothers who consume alcohol less frequently. Errors in variables, due to the sensitivity of respondents to questions about cigarette and alcohol intake, could explain these results. Similarly, systematic differences in parental time preferences, attitudes to risk, perceptions of child health states, and so on could systematically be correlated with the consumption of alcohol and tobacco.

5.4 Understanding the Income Gradient

As we have seen the income gradient that was found in Specifications 1, 2 and 3 disappears if we use a rich set of controls in “Specification 4”; hence in this section, we explore the reason for this. We hope that we will be able to untangle any mechanism via which income translates into better child health. The strategy we follow is to estimate a basic model using a small set of ‘standard’ background controls. The results of this model (see the first row of Table 5) produce a significant coefficient on income for the K-Cohort (4-7 years) and the pooled model. We then report the results of “Specification 4” , where use the measures of child’s nutrition, parents’ physical and mental health, and parental health related behaviour and lifestyle measures. The results of this model (see the second row of Table 5) show that income is no longer statistically significant. In an attempt to understand the income gradient, we re-estimate Specification 4 excluding, alternately: 1) the variables that represent child nutrition; 2) parental lifestyle variables; and 3) parental physical and mental health variables. The results of the first two regressions show that the income coefficient is still statistically insignificant (see the third and fourth row of Table 5). However, the results of the last regression produce a statistically significant income coefficient (see the last row of Table 5). This indicates that, so long as parental health variables are in the model, we do not find a significant relationship between income and child health. Also if we compare the results from this regression with the basic one, we see that the coefficient on income has changed very little.

We estimate another specification by excluding income from “Specification 4”, the coefficients on other variables in this specification are almost identical to the “Specification 4” and the coefficients on both parents’ physical health and

Table 5: Income coefficients from various specifications (ordered probit models)

Models	Both-Cohorts		B-Cohort (0-3 yr)		K-Cohort (4-7 yr)	
	Coef.	Std. Err.	Coef.	Std. Err.	Coef.	Std. Err.
Model with basic background controls[a]	*-0.060	0.026	-0.033	0.039	*-0.082	0.032
Specification 4	-0.046	0.036	-0.023	0.049	-0.067	0.047
Excluding only child's nutrition variables from Specification 4	-0.043	0.036	-0.022	0.050	-0.066	0.047
Excluding only parental lifestyle variables from Specification 4	-0.042	0.031	-0.027	0.042	-0.063	0.041
Excluding only parental health variables from Specification 4	*-0.071	0.036	-0.038	0.048	*-0.106	0.049

*Notes: (i) The dependent variable is an ordered categorisation of the child's general health status (e.g., 1= excellent, 2 = very good, 3 = good, 4= fair and 5= poor) as reported by a parent/guardian. (ii)a−This Specification includes controls for previous health state of a child, age and sex of the child, dummies for aboriginal and/or torres state islander and English speaking household, dummies for low birth weight and breastfeeding, log of household size, the presence and age of biological parents, housing condition, parental education and employment status. (iii) Controls used in Specification 4 are same as Table 4 (iv) * Significant at the five per cent level.*

Source: As for Table 1.

mother's mental health are still statistically significant. However, if we exclude both parent's physical health and mother's mental health from "Specification 4", the coefficients on other variables change substantially, and income becomes statistically significant.[14] So it is parental health, especially maternal physical and mental health that are responsible for reducing the magnitude and the significance of income in our regressions. Assuming that parental health does not skew parental *assessments* of child health, this result has at least two interpretations. One is that the income gradient disappears due to the colinearity between parental health and income (i.e., that parents in poor health have lower earnings) and measurement error in income. The probability of measurement error in income is less likely in this paper, because we use an average income of two periods, which is subject to less measurement error than cross-sectional studies. Also there is no evidence of collinearity among all covariates of Specification 4, including the family income, mother and father's health status, in the LSAC data,[15] which suggests that parental health does not simply reflect parental income in our models. A competing explanation is that income has no protective effect on child health in the presence of poor parental health states. Thus our analysis indicates that parental health, in particular maternal physical and mental health, is the possible mechanism/factor by which low family income translates into poorer child health. There are several possible explanations of

[14]The results of these two Specifications can be obtained from the authors upon request.

[15]The variance inflation factor (VIF) of all covariates in Specification 4 is less than 2, which is much less than the rule of thumb threshold for no serious colinearity that VIF less than 10 (Cohen and Cohen, 2003, p. 425).

this result: children from less healthy parents may be more susceptible to some infections or diseases, subject to a less healthy interuterine environment, receive lower quality of parental own-time inputs in the health production process. Another possible explanation is that parental health and child health might be correlated with a third, unobservable, such as exposure to various environmental hazards, stress, and other factors.

5.5 Chronic Conditions

In this section we first examine whether the income gradient exists for parent-reported chronic health conditions and physician-assessed health measures such as asthma and bronchiolitis (Table 6).

Table 6: The effects of income on the incidence of child chronic condition (binary probit models)

Chronic conditions	Both Cohorts		B-Cohort (0-3 yr)		K-Cohort (4-7 yr)	
	Coef.	Std. Err.	Coef.	Std. Err.	Coef.	Std. Err.
Hearing problems	0.070	0.062	-0.094	0.066	0.153	0.084
Vision problems	-0.060	0.046	-0.111	0.063	-0.027	0.063
Eczema	0.007	0.023	0.029	0.032	-0.011	0.036
Diarrhoea/collitis	0.025	0.064	0.063	0.093	-0.016	0.073
Ear infections	0.006	0.032	0.010	0.048	0.002	0.042
Other infections	*-0.112	0.037	*-0.140	0.057	*-0.101	0.050
Food or digestive allergies	0.004	0.033	0.012	0.037	-0.001	0.048
Other illnesses	0.054	0.030	-0.030	0.041	*0.121	0.041
Other physical disabilities	0.001	0.061	-0.085	0.077	0.072	0.081
Recurrent abdominal pain	0.103	0.059	0.133	0.114	0.093	0.054
Asthma	0.018	0.031	0.011	0.055	0.015	0.036
bronchiolitis	*0.063	0.032	0.083	0.046	0.032	0.042
Developmental delay			0.119	0.111		
Anaemia			*0.483	0.196		
Attention deficit disorder					-0.028	0.078
Frequent headaches					-0.018	0.060
Any chronic conditions	**0.053*	*0.019*	*0.034*	*0.037*	**0.057*	*0.028*

*Notes: (i) Coefficients on log family income from the probits models of each chronic condition are reported. (ii) Other covariates are age, gender, breast feeding, birthweight, age of the parents, the presence of the biological mother and father in the household, parental education and employment, log of household size, housing condition, identification as an Aboriginal or Torres-Strait Islander, English speaking household. (iii) * Statistically significant at the five per cent level.*

Then we follow Case et al. (2002), A.Currie et al. (2007) and J. Currie (2008) to examine the role of chronic conditions in parental reports of poor child health and to test whether any relationship between these is moderated by income (Table 7). The hypothesis underlying our examination of this relationship is that poor children may be more likely to suffer from chronic health conditions because of the lower levels of protection that are afforded by low levels of parental income and education, poorer housing conditions and other unobservable factors. In sum, poorer households have access to fewer resources to devote to the use of market inputs in the production of child health and the technology of health production may also be less health-productive. Thus, poorer households may be susceptible to more frequent health shocks, or to more severe health effects of stochastic shocks to health, or both.

Table 7: The effect of chronic conditions and income on the chance of a child being in "poor" health (binary probit models)

Chronic conditions	Both-Cohorts		B-Cohort (0-3 yr)		K-Cohort (4-7 yr)	
	β_2	β_3	β_2	β_3	β_2	β_3
Hearing problems	1.733	-0.093	0.229	*-0.900	0.242	0.041
Vision problems	*4.77	*-0.416	5.030	-0.438	*4.703	*-0.411
Eczema	0.493	-0.023	1.042	-0.073	0.050	0.016
Diarrhea/collitis	2.417	-0.136	2.696	-0.178	1.584	-0.035
Ear infections	0.442	0.017	0.611	0.013	-0.212	0.067
Other infections	1.353	-0.048	3.106	-0.194	-0.219	0.085
Food or digestive allergies	0.313	0.019	0.391	0.009	0.513	0.004
Other illnesses	0.515	0.017	1.888	-0.098	-1.773	0.216
Other physical disabilities	1.433	-0.065	1.156	-0.022	1.212	-0.052
Recurrent abdominal pain	-3.556	0.380	-3.823	0.405	-4.161	0.435
Asthma	*1.13	-0.061	0.759	-0.017	1.010	-0.054
Bronchiolitis	0.397	0.004	0.447	0.019	0.115	0.011
Developmental delay			*8.948	*-0.746		
Anaemia			*-40.829	*3.632		
Attention deficit disorder					0.224	0.030
Frequent headaches					-0.970	0.151
Any chronic conditions	*0.527*	*-0.010*	**1.523*	*-0.093*	*0.516*	*-0.002*

*Notes: (i) In the interests of parsimony standard errors are not reported, but are available from the authors upon request. β_2 and β_3 are estimated from the following probit regression: $h = \beta_0 + \beta_1 y + \beta_2 C + \beta_3 C * y + \gamma X + \varepsilon$, where h is the binary variable for poor health, y is the logarithm of average CPI-adjusted family income, C is the binary variable =1 if a chronic condition exists (0 otherwise) and X is a set of standard background controls (age, gender, breast feeding, birthweight and previous stock of health of a child, age of the parents, the presence of the biological mother and father in the household, parental education and employment, household size, housing conditions, identification as an Aboriginal or Torres-Strait Islander, English speaking household). (ii) * Statistically significant at the five per cent level.*

The relationship between income and chronic conditions is examined by estimating probit regressions for each condition and then by including indicators for all conditions in one regression.[16] In this section, we use our "standard background controls" as covariates. The results are reported in Table 6. They show that the income coefficient is not statistically significant for most chronic condition regressions, but there are several exceptions. In the "other infections" category both the pooled and cohort regressions produce statistically significant income coefficients with the expected (negative) sign. However, the bronchiolitis and anaemia regressions also have statistically significant income coefficients and these have an unexpected (positive) sign. This suggests that children in higher income households are more likely to have these conditions. However, if the conditional probability of being *diagnosed* with one of these conditions is a function of income – as it may be, if higher-income individuals have access to more, or higher quality health care – the implication of these findings with respect to prevalence is confounded. It is noteworthy, too that we do not find any significant relationship for the (physician-assessed) health state asthma, but we do for bronchiolitis. The coefficient on bronchiolitis is positive, though, which indicates that children from higher-income households are more likely to have this condition.[17] Once again, perhaps children from high income households are more likely to have been diagnosed with bronchiolitis than children from low-income households. Alternatively, one may interpret this result as being consistent with the so-called "hygiene hypothesis". This hypothesis is that improvements in hygiene and public health may have reduced the stimulation of micro-organisms in the environment and reduced the immunoresponse in children, making them more susceptible to allergic disease (Cardoso et al., 2004). If better hygiene measures were correlated with higher incomes our result could be interpreted as providing some evidence in support of the hygiene hypothesis. Finally, one may speculate as to the correlation between these conditions and maternal age (which may be higher, on average, in higher income groups), or a range of variables that may justifiably be regarded as possible sources of omitted variable bias in these regressions.

Finally, we also estimated the probability that a child would be described as being in "poor" health when a chronic condition was present. Our approach is similar to that of Condliffe and Link (2008): we define our "poor" health state as a state of less than very good health and we estimate our binary variable on the chronic condition, income and an interaction term of income and the binary chronic condition indicator, along with our standard control variables. We estimate this model for each condition separately and for all conditions in one regression. The results are reported in Table 7 (the last row reports the result from the latter regression). The coefficients (β_2) on the chronic condition binary

[16] Case et al (2008) reported that including all conditions will reduce the biases that could arises from co-morbidity.

[17] Acute viral bronchiolitis is defined as "...an acute viral illness in children usually between 2 weeks and 9 months of age, manifested by cough, wheezy breathing, hyperinflated chest, widespread fine crackles and frequently expiratory wheezes on ascultation" (Royal Children's Hospital, 1995, p.70).

indicators are positive and statistically significant in the case of vision problems, developmental delays, and asthma. The presence of any of these conditions increases the probability of having poor health. The negative and statistically significant signs on β_3 for several conditions (hearing problems, vision problems and developmental delay) indicate that, for these conditions, a higher income is protective: richer children with these conditions are less likely to be classified as being in poor health *ceteris paribus*. The positive and statistically significant result on anaemia, on the other hand, is counter-intuitive. We have no plausible explanation for this result. For the presence of any chronic conditions we find expected results for the B-Cohort (0-3 years), but no statistically significant result for the K-Cohort (4-7 years) or the combined cohorts. Finally, note that although we find a statistically significant income coefficient for parents' reported overall health status of children using the standard background controls, there is no convincing evidence for such an effect for the physician-assessed conditions (asthma and bronchiolitis).

6 Conclusions

This paper contributes to an growing literature on the income-child health gradient. This literature is advancing, in part due to the availability of high-quality data and advances in econometric methods. The current paper presents the first Australian econometric evidence on the income-child health gradient and the mechanisms via which child's nutritional and parental health may affect child health, independently of the household's income. It also presents comparisons of the empirical estimates that are derived via applications of the previous specifications and econometric methods that have been used in this literature, estimated on Australian data, and compares the results of applying these with those of expanded specifications.

Three aspects of our findings are particularly noteworthy. Firstly, we find an income-child health gradient in the LSAC data when we use similar covariates to those that were used in the studies of Case et al. (2002), J. Currie and Stabile (2003) and A. Currie et al. (2007), but our income coefficients are uniformly smaller. It is also noteworthy that our income coefficients are even smaller than Condliffe and Link (2008) and Murasko (2008) who, in turn, found smaller income gradients compared to Case et al. (2002) and Currie and Stabile (2003). Nevertheless, the income-child health gradient does appear for Australia when the customary specifications are used. This finding suggests that for Australia, as for Canada and the UK, such a gradient persists despite the existence of a long-standing universal and compulsory health care financing and delivery systems. Secondly, when we specify a more encompassing model of child health production, we find that the income gradient in this Australian sample disappears. We find that parental health–in particular, the mother's health and the child's nutritional intake–is strongly correlated with child health in Australia. Finally, and most importantly, our results suggest that parental health, in particular maternal physical and mental health, is the possible mechanism/factor by which low family income translates into poorer child health suggesting that the effect of income seems to operate through parental health. These results are similar in nature to the recent findings of Propper et al. (2007), for the UK, who found no income gradient, but uncovered an important relationship between mother's health and the health of UK children.

From a policy perspective, it is important to understand the mechanisms that are responsible for the early health disadvantages experienced by low-SES children. One of our findings is that the income/child health gradient is much smaller in Australia compared to many developed countries, and rest of the gradient disappears if parental physical and mental health is considered. Our result suggests that policies which improve parental health, particularly maternal physical and mental health, could have important spill-over effects for children from low-SES households in countries with well-established universal and compulsory health care financing schemes. Policy initiatives of this kind may constitute an important mechanism for breaking the pernicious cycle wherein lower incomes beget poorer health, and poor health begets low incomes.

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