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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 Survey of Australian (LSAC), which involved two waves of data collection for children born between March 2003 and February 2004 (B-Cohort), and between March 1999 and February 2000 (K-Cohort). 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) , using a sample of Australian children. 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. Thus, our results for Australian children are similar to those produced by Propper et al. (2007) on their British child cohort. We also find some evidence that higher incomes have a protective effect when health shocks do arise: for several chronic conditions, children from higher-income households are less likely to be reported as being in poor health than children from lower-income households who have the same chronic conditions. The latter result is similar to some recent findings by Condliffe and Link (2008) on a sample of US children.

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

1 Introduction

A growing literature documents a strong positive correlation between household income and child health (see for example, Case, Lubotsky, and Paxson, 2002; Currie and Stabile, 2003; Propper, Rigg, and Burgess 2007; Case, Paxson, and Vogl, 2007; Chen, Martin, and Matthews, 2006; Currie, Shields, and Price, 2007; Dowd, 2007; Case, Lee, and Paxson, 2008). Two 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. Subsequent studies that have examined the income-child health gradient 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., they did not find that the income-health gradient steepened with child age. Case et al. (2007) have argued that the divergence in the conclusions of Chen et al. and Case et al. (2002) can be explained primarily by (i) the inappropriate inclusion of 17- and 18-year olds, who tend to live more independently and (ii) the use of the current incomes of these individuals, rather than the incomes of the households in which they were raised. Furthermore, Chen et al. use one year of data and a categorical measure of income, rather than a continuous measure of income as did Case et al. (2002).

However, a recent study by A. Currie et al. (2007), using data from the 1997-2002 Health Surveys of England (HSE), also did not produce evidence that the income-health gradient increased with age in their sample of British children. The authors did find a positive association between family income and child health using a parent-assessed Likert-measure of the child's health state. Interestingly, though, when objective measures (e.g., physiological measurements and blood samples obtained by qualified nurses) were used, no such income-health association was evident. 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) also argue that A.Currie et al. (2007) incorrectly coded the chronic conditions measures included in the HSE, leading to both erroneous estimates and conclusions for the specifications that use those measures. 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).

Using a regional UK birth cohort data Propper et al. (2007) have also found the expected, positive, association between income and child health for early-to-mid-childhood. However, these authors also did not find any age-related increase in the income-child health gradient. Specifically, Propper et al. (2007) examined a number of mother-reported measures of child health that were reported at regular intervals during the first seven years of the child's life. They found evidence of a strong relationship between household's experience of financial difficulties and poor child health but they did not find any compelling evidence that this relationship was increasing in child age. A very recent study by Condliffe and Link (2008) , using Medical Expenditures Panel Survey and the Panel Study of Income Dynamics on the USA, found that the child health and income gradient become more pronounced as the children grow older. These findings are similar to those of Case et al (2002). Thus, the literature presents mixed results on the hypothesis that the income-child health relationship is increasing in child age.

The mechanism(s) via which higher incomes produce better child health is also far from settled, although a number of studies have explored this issue. Case et al. (2002) examined a wide variety of factors that may explain the observed relationship between child health and family income. They 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 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. More generally, Case et al. (2002) and A.Currie et al. (2007) did not find any significant mediators of the relationship between family income and child health.

Propper et al. (2007) also explored the pathways via which household income may affect child health using an array of additional control variables. They 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. Dowd (2007) also uses a broad set of controls (e.g., mother's health status during pregnancy, maternal health behaviour during pregnancy, and early childhood health exposures of the child) to examine the income-health relationship for children. In contrast to the results of Propper et al. (2007), but in line with those of Case et al. (2002) and A.Currie et al. (2007), Dowd (2007) also 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 parental attributes (e.g., health states) or behaviours (e.g., diet and exercise) attenuate the income-health relationship for children in our sample. Finally, we explore whether or not the children from low-SES are hit harder by a particular health shock.

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, we use panel data, which have not been used extensively in this literature, to examine the income-child health gradient, using econometric techniques that are appropriate to characteristics of the data. We invoke econometric techniques to account for both the cluster sampling and panel structure of the data that may improve our confidence in the specification of the panel model. These methods also provide a basis for comparing the cross-sectional and panel estimates that we produce. Third, we explore the relationship of some further variables, 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, parental health and health related behaviours (e.g., smoking and drinking) and other lifestyle measures (e.g., parental exercise) on health states of children. 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. 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 using panel data and other econometric innovations. Our results show that the income-child health gradient is much smaller in Australian than that of the USA, Canada and UK. In our relatively young child age groups we find no evidence that the income-health relationship has an increasing gradient, even when use a small set of background controls. Furthermore, when we include a richer set of controls, including parental health, we find no evidence of an income-child health gradient at all.

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; 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 in health capital. 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 (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 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) AIFS (2007). 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), and between March 1999 and February 2000 (K-Cohort, children aged 4-5 years) 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. The numbers of children surveyed in Wave 2 of the respective cohorts is slightly lower, primarily as a result of loss-to-follow-up, with 4606 and 4464 children retained in cohorts B and K, respectively.

The LSAC was conducted using both face-to-face interviews and survey instruments that were sent and retrieved via mail. The main topics covered

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

include demographics, health status, education, the relationship history of parents, parenting practices, financial factors, lifestyle, housing and neighbourhood attributes.[2]

In order to take the advantage of the survey's comprehensive design, 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. 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". 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 fall 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 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 variable. For example, there is the possibility that maternal reporting of child's health might be affected by her own health state. Some previous studies (e.g., Dadds, Stein, and Silver, 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

[2]For a more comprehensive account of the LSAC sampling frame of the LSAC see Soloff, Lawrence, and Johnstone (2005)

[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.

[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.

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

3.2.2 Income

In our empirical analysis income will be used to proxy parents' time spent for earning income, which is L_w in our theoretical model. In order to measure permanent income, which is believed to have stronger effect on health than transitory variations in income, we take the average annual income of each family in Wave 1 and Wave 2. The income estimates were adjusted using the Australian national Consumer Price Index (CPI) for the study period, using the CPI at Wave 1 as the base (ABS, 2008). We use the natural logarithm of average CPI-adjusted income as a proxy for permanent income.

Child Health and Income: A Raw Sketch of the Gradient

Figure 1 presents a plot of income and parent-reported child's health from the LSAC. It shows the expected, positive univariate correlation of child health and household income. However, it is not obvious that the health-income gradient is increasing in child age (cf, in particular, the B-Cohort of Wave 2 and the K-Cohort of Wave 1). It is, of course, possible that the age difference between these two groups is too small to generate any observable difference in the income-health gradient even if it exists. This issue is investigated in more depth, and in a multivariate framework, in our econometric work.

3.2.3 Other Variables

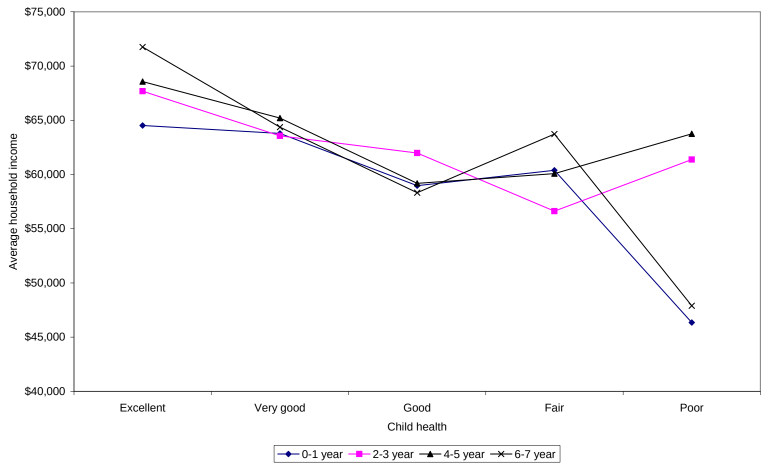
Based on the availability of data and the analytical model presented in Section 2, other covariates consist of the following groups:

Demographics

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

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

Figure 1: Income and health status



Source: Computed from the Longitudinal Study of Australian Children (AIFS, 2007).

production of child health). Applying these controls for child characteristics and family characteristics allows us to controls 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.

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.[6] 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

[6] 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.

Table 1: Descriptive statistics

	Both-Cohorts		B-Cohort (0-3)		K-Cohort (4-7)		Test B=K
Descriptions	Mean	Std.	Mean	Std.	Mean	Std.	(p-value)
Lag of Health (health state, previous period)	1.53	0.01	1.52	0.02	1.55	0.02	0.00
Log of family 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 (AIFS, 2007).

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 a 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 serves 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}, 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 would be estimated by assuming that the data are collected using simple random sampling. 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.

4 Econometric model

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

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

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 permanent income of the family, $Z_{it}$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. Although there is cause to believe that this particular problem is likely to be controlled well by invoking the multi-stage clustered sampling procedures and by selecting one child per family, we also check for the possible correlation of the lagged dependent variable with the error term, using the method proposed by Wooldridge (2005). This test involves estimating the distribution of unobserved effects on the initial values of the model's exogenous variables.

$$u_i = u_0 + u_1 h_{i1} + u_2 \bar{Z}_i + \epsilon_i$$

where $\bar{Z}_i$ is the average over sample period of observations on exogenous variables and $\epsilon_i \sim N(0, \delta_\epsilon^2)$ and independent of exogenous covariates, initial conditions (u_0) and $\bar{Z}_i$. However, when substituting the above formulation of the individual effect into equations (8) and equation (7), reveals that a panel of at least three serial observations (i.e., t_0, $t-1$ and t) is required for the estimation. However, at the time of this study, the LSAC consisted of only two waves and hence we could not apply this approach to the current data set. Nevertheless we are able to control for a further source of individual heterogeneity using the known cluster sample property of the LSAC.

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 the observed counterpart (or indicator) of it is denoted by H_i, which may be specified as follows:

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

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

$$
\begin{aligned}
&prob(H_i = 1) = \phi(\mu_1 - \alpha Z)\\
&prob(H_i = 2) = \phi((\mu_2 - \alpha Z) - (\mu_1 - \alpha Z))\\
&prob(H_i = 3) = \phi((\mu_3 - \alpha Z) - (\mu_2 - \alpha Z))\\
&prob(H_i = 4) = \phi((\mu_4 - \alpha Z) - (\mu_3 - \alpha Z))\\
&prob(H_i = 5) = 1 - \phi(\mu_4 - \alpha Z)
\end{aligned}
$$

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 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.[7]

5 Results and Discussion

In this section we first estimate specifications that are close analogs of the models invoked by Case, Lubotsky, and Paxson (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 also exploit the panel nature of the data and the survey characteristics (i.e., sampling weights and clustering) to estimate otherwise identical models. We then proceed to estimate a more general model 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. 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.

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

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

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, Harmon, and Walker (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.[8] 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.[9]

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 parental education is included (see Table 2). 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 steeping 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

[8] 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.

[9] 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 $F(25,246)=1.28$.

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)			
	Cross-sectional estimates		Panel estimates	
0-3 years	-0.029		-0.035	
(n=7730)	(0.025)		(0.044) (n=3269)	
4-8 years	*-0.063		*-0.071	
(n=8509)	(0.027)		(0.030) (n=4403)	

*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) (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 (AIFS, 2007).

two waves of the LSAC.

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 ommission of confounders and controls. Therefore, we estimate Specification 3 (by adding controls for low birthweight, breastfeeding, and housing conditions to Specification 2) in both the cross section and panel forms. 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.

The choice of age break is not explained in previous studies and it is possible

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

		Spec1	*Spec2*	*Spec3*
B-Cohort	**Cross-sectional estimates**			
	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)
	Panel estimates	*-0.065	-0.061	-0.034
		(0.033)	(0.038)	(0.038)
K-Cohort	**Cross-sectional estimates**			
	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)
	Panel estimates	*-0.178	*-0.134	*-0.081
		(0.028)	(0.031)	(0.032)

Notes: As for Table 2.

Source: As for Table 1.

that the income gradient may be sensitive to changes in choices of age break (Harris, Hollingsworth, Inder, and Maitra, 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 but the coefficient on income is 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. Our panel data regressions produce significant coefficients on income for the B-Cohort only for Specification 1. 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, pp.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".

It can also be seen from Table 3 that the magnitude of the income gradient increases with age in both the cross-sectional and panel settings despite the fact that the estimates are insignificant for the B-Cohort. We now subject this hypothesis to further testing by constructing a full model, taking into account additional factors that may affect the child health and the income gradient.

5.3 Determinants of Child Health: The Main Model

The determinants of child health estimated by the main model are presented in Table 4. The results show that the income is no longer statistically significant in this full 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 cor-

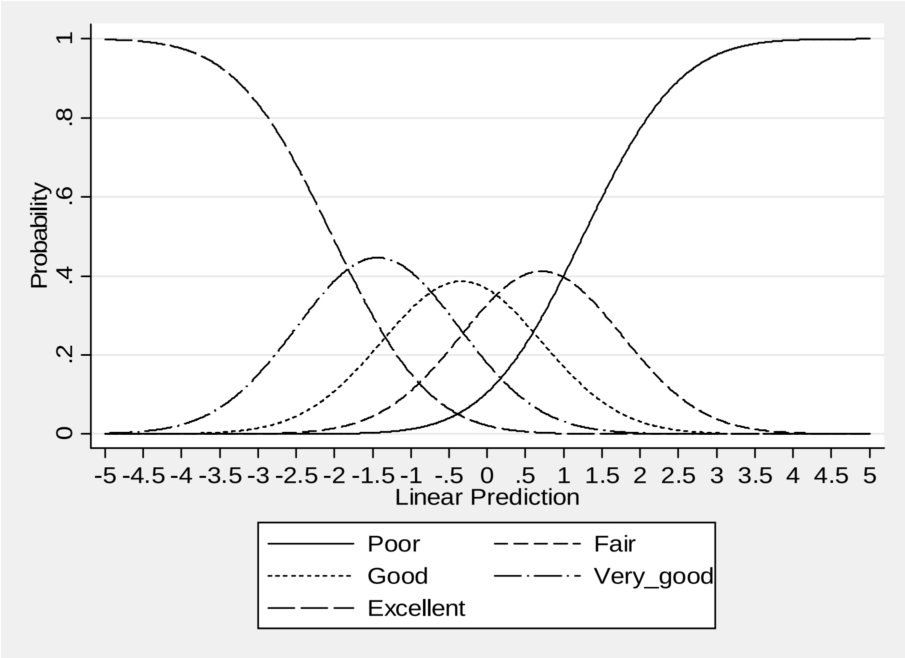
Table 4: Determinants of child health in Australia (ordered probit models)

Variables	Both-Cohorts (n=4590)		B-Cohort (n=2312)		K-Cohort (n=2043)	
	Coef.	Std. Err.	Coef.	Std. Err.	Coef.	Std. Err.
Lag of health state (health state, previous period)	*0.390	0.024	*0.296	0.031	*0.507	0.036
Log of family 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	*-0.040	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.

Figure 2: Prediction of child health probability



Notes: As for Table 2.
Source: As for Table 1.

related 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. Parental education appears to be a weak determinant of child health in Australia, as the mother's education is only significant at the 10% level, in the pooled model; the father's education is significant only for the K-Cohort. However, parental education starts to affect child health if the parent has more than graduate qualification. The child's current health is strongly related to its health in the preceding period, which is in line with our theoretical prediction.

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 have a low propensity to consume such products due to their age. So it is not surprising that the variable is statistically significant only in K-Cohort.

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. 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. The most striking 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.

The effects of covariates in the main model on child health are also summarised in Figure 2, which presents the linear predictions of the child health states. The plot shows that, on average (i.e., at the linear prediction of -2.16) the probability that an Australian (LSAC) child has excellent, very good, good, fair, and poor health are 55.2%, 33.0%, 9.8%, 1.8% and 0.1%, respectively. According to the main results presented in Table 4 , the value of the linear prediction of health state is shifted left by advancing child age, and thus the probability of having a excellent health is increasing in the child's age. Similar shifts are experienced for children who have good birthweight, who have parents with good stock of health, and/or who consume healthy food.

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 complete set of controls; hence in this section, we explore the reason for this . 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 and the pooled model. We then use the rich set of variables that are available to us from LSAC data, adding measures of child's nutrition, parents' physical and mental health, and then by adding 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 the full model 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

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

Models	Both-Cohorts		B-Cohort		K-Cohort	
	Coef.	Std. Err.	Coef.	Std. Err.	Coef.	Std. Err.
Model with basic background controls	*-0.060	0.026	-0.033	0.039	*-0.082	0.032
Full model	-0.046	0.036	-0.023	0.049	-0.067	0.047
Excluding only child's nutrition variables	-0.043	0.036	-0.022	0.050	-0.066	0.047
Excluding only parental lifestyle variables	-0.042	0.031	-0.027	0.042	-0.063	0.041
Excluding only parental health variables	*-0.071	0.036	-0.038	0.048	*-0.106	0.049

Notes: As for Table 2.

Source: As for Table 1.

the coefficient on income has changed very little.

We estimate another specification by excluding income from the full model, the coefficients on other variables in this specification are almost identical to the full model and the coefficients on both parents' physical health and mother's mental health are still statistically significant. However, if we exclude both parent's physical health and mather's mental health from the full model, the coefficients on other variables change substantially, and income becomes statistically significant.[10] 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 a strong correlation of parental health and income (i.e., that parents in poor health have lower earnings). However, the correlation of these variables in the LSAC data is actually very weak in the LSAC data.[11] A competing explanation is that income has no protective effect on child health in the presence of poor parental health states.

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). 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

[10]The results of this specification can be obtained from the authors upon request.

[11]The Spearman rank correlation test did not reject the null hypothesis that income and parental health are independent and the correlation coefficients are very small (-0.12 and -0.07 for mother and father, respectively).

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

Chronic conditions	Both Cohorts		B-Cohort		K-Cohort	
	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.*

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		K-Cohort	
	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 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.

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.[12] 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

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

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.[13] 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, Cousens, de Góes Siqueira, Alves, and D'Angel 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.[14]

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 of Table 7 reports the result from the latter regression.) The coefficients (β_2) on the chronic condition binary 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 for the B-Cohort, but no statistically significant result for the K-Cohort 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

[13] 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" RCH (1995, p.70).

[14] Developmental delay and anaemia are available only for B Cohort and attention deficit disorder, frequent headaches and obesity are only for K Cohort.

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 nutritional and lifestyle/health behaviour variables 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, estimated with econometric techniques that exploit the panel and other sample properties of the data set.

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. Secondly, when we specify a more encompassing model of child health production, we find no income gradient in this Australian sample. Finally, we find that parental health, in particular, the mother´s health and the child´s nutritional intake are strongly correlated with the child health. These results are similar to the recent findings of Propper et al. (2007), who found no income gradient, but uncovered an important relationship between mother´s health and the health of UK children.

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