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The health care costs of childhood obesity in Australia: an instrumental variables approach

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ABSTRACT

The effect of childhood obesity on medical costs incurred by the Australian Government is estimated using five waves of panel data from the Longitudinal Study of Australian Children, which is linked to public health insurance administrative records from Medicare Australia. Instrumental variables estimators are used to address concerns about measurement error and selection bias. The additional annual medical costs due to overweight and obesity among 6 to 13 year olds is about $43 million (in 2015 AUD). This is driven by a higher utilisation of general practitioner and specialist doctors. The results suggest that the economic consequences of childhood obesity are much larger than previously estimated.

Keywords: Medical costs; children; overweight; obesity; BMI; instrumental variable

JEL codes: I1, I12, I18, C26

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1. Introduction

Childhood obesity is considered one of the most serious public health challenges of this century. One in four Australian children are overweight or obese, with many high income countries experiencing similarly high prevalence rates (OECD 2014). Childhood obesity is concerning not only because of the high risk of persistence into adulthood, but also the elevated risk of serious health conditions during childhood. These conditions include asthma, sleep apnea, bone and joint problems, hypertension, high cholesterol, type 2 diabetes and psychological problems (Reilly et al. 2003; Daniels 2006). Given a majority of health expenses in Australia are funded by the public purse (AIHW 2016), Australian policy makers are particularly interested in the medical costs attributable to obesity. Information on such costs not only allow health insurers to predict future health care expenses, but they also provide evidence that is needed for assessing the short-term value to governments and the wider society of childhood obesity prevention and treatment programs.

Over the past decade, several studies have examined the health care costs associated with childhood and adolescent overweight and obesity (e.g. Johnson et al. 2006; Monheit et al. 2009; Breitfelder et al. 2011; Au 2012; Wenig 2012; Turer et al. 2013; Batscheider et al. 2014; Wright and Prosser 2014; Clifford et al. 2015). A majority of studies find obesity in childhood or adolescence to be correlated with higher health care expenses.[1] While informative, one limitation of previous studies is that they are unable to infer causality; that is, to determine the costs that are attributable to obesity, and not merely associated with obesity.

There are three potential reasons why ordinary least squares (OLS) estimates of the costs of obesity may suffer from bias: i) omitted variables; ii) reverse causality; and iii) measurement error. The direction of bias due to omitted variables (or unobserved confounders)

[1] An insignificant correlation has been reported by a few studies using the Medical Expenditure Panel Survey (MEPS) in the United States (Skinner et al. 2008; Turer et al. 2013; Wright and Prosser 2014)

is *a priori* unclear. For example, unobserved factors (such as poor parental health literacy or geographically remote neighbourhoods) may be correlated with both a higher likelihood of obesity and with lower utilisation or access to health care services among children. This would imply a negative selection bias and an underestimate of medical costs caused by obesity. Similarly, unobserved medical conditions such as cancer or autoimmune diseases (e.g. celiac disease) could explain both a lower likelihood of obesity (Collins et al. 2000; Fasano and Catassi 2005) and a greater utilisation of health care. On the other hand, there may be a positive bias if other medical conditions (such as depression or anxiety) lead to both an increase in weight (e.g. through emotional eating or medication side-effects) and health care utilisation.

Reverse causality (or simultaneity bias) may occur if a visit to the doctor results in a treatment program or drug that affects the child's weight. The direction of bias resulting from this could go in either direction. Finally, measurement error, which can arise if a child's adiposity is measured imprecisely, is concerning because it can lead to an underestimate of the costs caused by obesity. Previous studies examining the consequences of obesity have traditionally recognised that using body mass index (BMI) that is derived from self- or parent-reported height and weight may suffer from measurement error. This is not a concern in our study as we use BMI derived from clinically measured height and weight. However, we raise another source of measurement error in BMI, which arises due to the normal changes in weight relative to height as children grow and physically mature (Horlick 2001). Such short-term fluctuations imply that BMI measured at any one point in time may not accurately reflect a child's adiposity; increases in BMI may be due to both increases in fat mass and fat-free mass (Lindsay et al. 2001; Maynard et al. 2001; Freedman et al. 2004).

This study aims to address these potential biases by employing an instrumental variables (IV) estimator to determine the effect of childhood obesity on health care costs incurred by the Australian Government. We build on recent studies that have used an IV

approach to determine the impact of obesity on health care utilisation (Cawley and Meyerhoefer 2012; Biener et al. 2017; Doherty et al. 2017; Kinge and Morris 2017). Cawley and Meyerhoefer (2012) estimate the medical costs caused by adult obesity in the United States using the weight of a biological relative as the instrumental variable. They show that the estimated effect of adult obesity on medical costs is much greater than previous non-IV estimates suggested. Using the BMI of biological parents as instruments for child's BMI, Kinge and Morris (2017) find that childhood obesity increases the probability of a doctor visit and medications usage in England. Doherty et al. (2017) using the biological mother's BMI as an instrument, find that in Ireland, obesity increases the probability of a GP visit and hospital stay among adolescents. Biener et al (2017) also instrument for child's BMI using the biological mother's BMI and find that obesity increases medical costs in the United States to a much larger extent than previously estimated.

Our study uses a similar approach to investigate the causal effects of childhood obesity on publically-funded health care costs in Australia. We use the body mass index (BMI) of the child's biological parents to instrument for the child's BMI. A key strength of this study is the use of panel data on a representative sample of Australian children, with measured height and weight of each child linked to government administrative records on the child's health care utilisation and costs. We ensure that health care costs are incurred after weight is measured to reduce reverse causality concerns, and utilise the richness of the dataset to control for a wide range of child and parental characteristics that may influence health care seeking behaviour. In addition, we provide new insight into the longer-term costs attributable to childhood obesity by investigating how a higher BMI at age 6/7 affects health care costs over the next eight years that follow.

We find that a heavier BMI significantly increases the total health care costs incurred, and the effect is considerably higher than the estimated association using ordinary least squares.

Falsification tests using BMI of step-parents support the validity of the instrumental variables, and robustness checks using information on a range of health conditions provides additional support for our instruments. We show that the increase in health care costs is largely driven by higher general practitioner and specialist visits, and is not due to mental health, dental, pathology, diagnostic or other services. Our estimates suggest that overweight and obesity in children aged 6 to 13 cost the Australian government approximately $43.2 million annually (in 2015 AUD) over and above the costs for children of normal weight for non-hospital health care services. This indicates that the short-term economic consequences of childhood obesity arising from health problems alone is considerable and a failure to take this into account in economic evaluations of obesity reduction programs may lead to a substantial underestimate of the economic returns from investing in such programs.

2. Data

2.1 The Longitudinal Study of Australian Children

The Longitudinal Study of Australian Children (LSAC) is a biennial representative panel survey of Australian children, which began in 2004 (see Soloff et al., 2005, for a detailed description of the study design). Briefly, postcodes were stratified by Australian State/Territory and by metropolitan and nonmetropolitan area. Postcodes from each strata were randomly selected, and within each postcode, children in the required age cohort were randomly selected from the Medicare enrolment database. Only one child per family was eligible for inclusion in the sample. This study uses data from the first five waves (2004 to 2012) of children from Cohort K, who were aged 4-5 years in the first wave. Of the original Wave 1 sample, the response rate for Waves 2, 3, 4 and 5 was 90%, 87%, 84% and 79% respectively, which is similar to other longitudinal studies of children (such as the Millennium Cohort Study in the

U.K.). Data on the child and their family's social circumstances were collected through a face-to face interview with the child's primary carer. More sensitive information from the parents was collected using self-completion questionnaires.

Physical measurements of the child's height and weight were taken during the interview using digital scales and a stadiometer. These were used to calculate the child's BMI $(kg/m)^2$. In the main analyses, BMI z-scores, based on CDC growth charts (Kuczmarski et al. 2002) are used. BMI z-scores are more flexible than binary indicators of BMI categories, and still allow us to make health care cost predictions in terms of children who are overweight or obese. We test for non-linearity in the relationship between BMI z-scores and health care costs by including squared terms. For the descriptive analyses, children are categorised into underweight, normal weight, overweight and obese using international age- and gender-specific cut-points (Cole et al. 2000).

Because our approach utilises the height and weight of both biological parents in wave 1, the main sample is restricted to 3,458 children who have biological parents with height and weight information at wave 1 (69% of total sample).[2]

2.2. Medicare Records

Medicare is Australia's publicly funded universal health insurance system. It provides free or subsidised health care to all citizens and permanent residents. The LSAC data is linked to Medicare records, which includes complete data on medical services funded under the Medicare Benefits Schedule (MBS) and pharmaceutical subsidies funded under the Pharmaceutical Benefits Scheme (PBS).

Under the MBS, predetermined Medicare benefits are paid by the Australian Government for clinically relevant professional services. These include (but are not limited to)

[2] Wave 1 contains the most complete information on the BMI of biological parents, so the use of wave 1 BMI allows us to maximise sample size. In a sensitivity analysis, we use only the biological mother's BMI from wave 1, which allows us to further increase the sample size to 3,955 children.

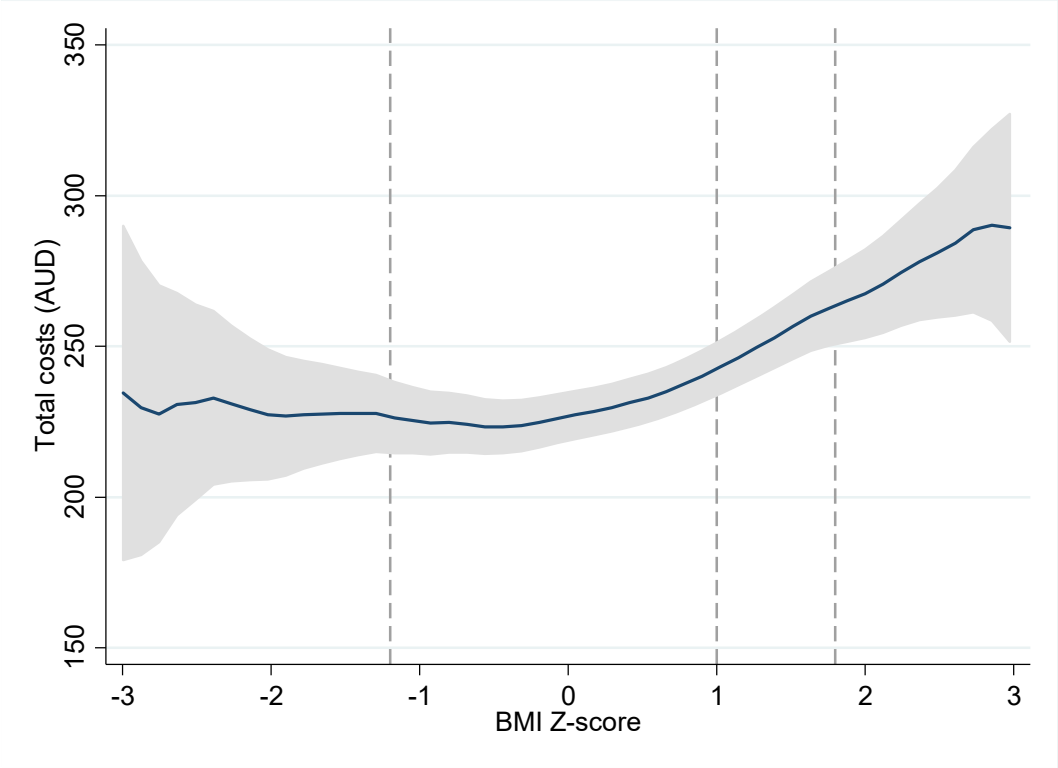
consultations by general practitioners and specialists, diagnostic and imaging, pathology, dental services and some allied health services. The benefit amount is applied nationwide. In a majority of cases (about 80% of all services), health practitioners charge exactly the benefit amount (known as bulk billing), and patients receive free health care at the point of service delivery. Where doctors choose to charge a fee above the Medicare benefit, patients will pay the difference out-of-pocket. Bulk billing incentives are also financed through the MBS. These provide additional payments to practitioners for bulk billing primary care services to children under 16 and concession card holders. The MBS also covers hospital services that are provided in private hospitals, however, because public hospital costs are not included in MBS records, we exclude all hospital services from this study. Inpatient services are relatively uncommon for this age group (about 4% of children reported staying in a hospital in a 12 month period). To provide some understanding of the effect of obesity on hospital services, we provide supporting evidence on the probability of a hospital stay (see Section 4.6).

The PBS covers eligible prescription drugs for most medical conditions. It excludes over-the-counter medicines and in-hospital prescription drugs. The amount paid by the Government under the PBS equals the difference between a fixed patient copayment and the listed drug price (which are both indexed annually and applied nationwide).

A sample of 4,534 LSAC children (93%) were successfully linked to Medicare records in Wave 1. A total Medicare cost is calculated by adding the annual costs for non-hospital services and pharmaceuticals incurred by each child under the MBS and PBS. Costs are adjusted to 2015 AUD. We exploit the longitudinal nature of the data to mitigate reverse causality by taking all costs from the 12-month window following the child's height and weight measurement at each wave. In any given year, approximately 81% of children aged 6 to 13 incur Medicare costs. This largely reflects utilisation of medical services (81% incur MBS costs

while only 19% incur PBS costs). A majority (91%) of Medicare expenses are through MBS in our data.

Figure 1. Nonparametric regression of total costs on BMI



Note: Total costs comprises of MBS and PBS expenses for the year following BMI measurement. Costs are adjusted for inflation and are in 2015 Australian dollars. Data is for the pooled sample (age 6 to 13), N=11,506. The dashed lines indicate the approximate cut-points for the BMI categories using age and gender specific cut-offs from Cole et al.(2000): Underweight is to the left of the first line, normal weight is between the first two lines, overweight is between the second and third lines, obesity is to the right of the third line.

Estimated nonparametric regressions between BMI z-scores and total costs are shown in Figure 1. The vertical dashed lines indicate the approximate cut-offs for BMI categories: underweight, normal overweight and obese. The figure shows that there is a shallow U-shaped relationship between BMI and total Medicare costs, with average costs reaching the lowest level at around BMI z-score of 0 (the mean BMI for normal weight), and increasing as children move away from the normal weight range. The positive association between costs and BMI is particularly pronounced as BMI z-scores increase above 1 (overweight and obese

categories). There is much greater variation in costs for BMI z-scores below -1 (underweight category). Due to this large variation among children in the underweight category and the potential for underweight children to have health problems, we exclude underweight children (6% of our analysis sample). This allows the study to focus on the objective of estimating the excess medical costs due overweight and obesity.

The mean total Medicare costs per year by BMI category and age are shown in Table 1. Column (1) shows that on average, across ages 6 to 13, total costs are greater for children in heavier BMI categories. Children with obesity incur $299 per year in non-hospital Medicare costs, which is $61 (26%) more than children of normal weight. Overweight children incur $29 (12%) more than normal weight children. Columns (2) to (5) show that on average, the inflation-adjusted costs for each BMI category increase as children get older. The difference in mean costs of obesity (or overweight) compared with normal weight are statistically significant at all ages except age 12/13 (column 5).

Table 1. Mean total cost by BMI group and age

	(1) Pooled (Waves 2-5)	(2) Age 6/7 (Wave 2)	(3) Age 8/9 (Wave 3)	(4) Age 10/11 (Wave 4)	(5) Age 12/13 (Wave 5)
Normal	237.8	189.3	230.3	254.7	290.1
	(4.43)	(5.72)	(8.38)	(10.90)	(10.66)
Overweight	267.1	219.7	261.2	266.2	309.0
	(9.37)	(14.38)	(17.35)	(18.60)	(21.43)
Obese	298.7	229.4	309.3	327.7	319.3
	(20.37)	(29.89)	(47.77)	(36.02)	(42.80)
N	10804	2858	2804	2633	2509

Note: Robust standard errors in parentheses. Total cost comprises of MBS and PBS expenses for the year following BMI measurement. Costs are adjusted for inflation and are in 2015 Australian dollars. Sample includes children who incurred zero costs. Underweight children are dropped from the sample. Survey weights are used for mean calculations.

3. Empirical Strategy

3.1 Instrumental Variables Approach

The main challenge with estimating the effect of childhood obesity on health care costs is the possibility of unobserved confounders such as other health conditions and illness, access to health care services, family environment and parents' attitudes and behaviour. The presence of such unobserved confounders means that estimates from OLS models may be biased. In the absence of an experiment which randomises children into differing levels of BMI (or obesity status), we must rely on observational data and methods that allow us to emulate randomisation of children's BMI. The instrumental variables approach, and the use of body weight of biological relatives as instruments has been used previously to estimate the labour market consequences of obesity (e.g. Cawley 2004; Lindeboom et al. 2010), and more recently, to estimate the effect of obesity on medical costs (Cawley and Meyerhoefer 2012). The use of a biological relative's weight takes advantage of the genetic variation in the propensity for obesity. Specifically, it aims to exploit 'Mendelian randomisation', which refers to the random assignment of an individual's genotype at conception. An important feature of Mendelian randomisation is that the genetic variation in the propensity for obesity captures differences in obesity that are persistent throughout the individual's life and therefore can be used to reduce measurement error bias that arises due to short-term fluctuations in BMI (Davey Smith and Ebrahim 2005).

Following the approach of previous studies, we use the BMI of biological parents as instruments for the child's BMI. The use of parents' BMI as instruments, instead of siblings, has the advantage of not limiting our estimation to a sample of children who have siblings. Specifically, we take the BMI of the child's biological mother and biological father, which are calculated from self-reported height and weight, from the first wave of LSAC in 2004. By taking the height and weight observed at Wave 1 and using the child's BMI observed from

Wave 2 onwards, we reduce the possibility that the relationship between the parents' BMI and child's BMI is due to contemporaneous shocks or operating in the direction from child to parent. Wave 1 contains the most complete information on the BMI of biological parents, so the use of parental BMI from wave 1 (instead of contemporaneous waves) also allows us to maximise sample size. This measure from wave 1 is used even if parents separate in later waves and therefore does not restrict the estimation sample to households with two biological parents. Our preferred model includes BMI of both biological parents because 50% of a child's chromosomes are inherited from each parent. In sensitivity analyses, we estimate the IV models using only the biological mother's BMI from wave 1 (this allows a slightly larger sample due to more complete data on mother's BMI), and we further estimate IV models using either biological mother or father's BMI as a single instrument for our main estimation sample.

In order for the chosen instruments to be suitable, they must strongly predict the child's BMI. That roughly 45 to 75% of the variation in bodyweight across individuals has been attributed to genetics (Farooqi and O'Rahilly 2007), suggests that the BMI of biological parents is likely to be powerful predictor of the child's weight. One limitation is that parental BMI is derived from self-reported height and weight and may therefore be measured with error.[3] Many studies demonstrate that BMI from self-reported height and weight is highly correlated with measured BMI (e.g. Stunkard and Albaum 1981; Spencer et al. 2002) and thus is still likely to be a strong predictor of child's BMI. The large F-statistic in our first-stage regression (shown in Section 4.1, Table 2), which surpasses the conventional minimum of 10 (Stock et al. 2002) supports the strength of our instruments. Chalak (2017) suggests a mismeasured instrument can be used to provide consistent IV estimates, provided the exclusion restriction is satisfied (as is required in any IV model).

[3] Studies have shown that the error is non-classical; there is a tendency for self-reported height and weight to result in an underestimation of BMI (Cawley et al. 2015).

3.2 Instrument validity

The use of parents' BMI as instruments relies on the exclusion restriction assumption; that they are uncorrelated with the error term in the second stage equation after controlling for the child's BMI and other covariates. The validity of the instruments would be questionable if both the parent and child's BMI were influenced by a third factor (such as household environment or parental health habits/preferences) which is also correlated with unobserved determinants of the child's health care utilisation. One might be concerned by the possibility that a parent's health preferences and habits create a household environment that influences body weight for the whole family (e.g. through family meals/snacks, and physical activity habits). These same parental health preferences could conceivably influence decisions around seeking health care for their child.

As discussed in earlier papers (e.g. Cawley 2004; Lindeboom et al. 2010; Cawley and Meyerhoefer 2012), despite widespread belief in the importance of a shared home environment in predicting body weight and obesity, the evidence from studies on adopted children and twins overwhelmingly suggests that the there is no (or very little) relationship between the two (Stunkard et al. 1986; Grilo and Pogue-Geile 1991; Price and Gottesman 1991; Sørensen and Stunkard 1993; Vogler et al. 1995; Maes et al. 1997; Wardle et al. 2008; Haberstick et al. 2010).

For example, Stunkard et al. (1986) found that there was "no relation between the index of adoptive parents and the adoptee weight class" (p.193), and concluded that "genetic influences have an important role in determining human fatness in adults, whereas the family environment alone has no apparent effect" (p.193). The BMI of adoptive parents is assumed to capture the relevant family environment (i.e., non-genetic factors) for determining body weight, which is likely to include parental habits and attitudes to health.

In a review of the environmental influences on weight and obesity, Grilo and Pogue-Geile (1991) determined that "experiences that are shared among family members appear largely irrelevant in determining individual differences in weight and obesity" (p.520). More recent evidence suggests that the same finding applies to preadolescent children for whom the family home is a contemporaneous environment, and who are growing up during a time of dramatic rises in childhood obesity rates (Wardle et al. 2008). This is particularly relevant for the cohort in the current study. The current evidence suggests non-genetic variation in parents' weight is unlikely to influence the child's weight. We are confident that there is enough support for the validity of the BMI of biological parents, especially with the inclusion of a range of parental characteristics that are likely to capture health preferences (i.e., age, education level, employment status, smoking status and depression score) as covariates. In addition, the use of both parent's BMI as instruments allows us to conduct Hansen J tests of overidentifying restrictions; these tests are consistent with the validity of the instrument.

Nevertheless, we conduct falsification tests using BMI from the child's step-father (see Section 4.3) and these results all indicate that there is no correlation between the family environment relevant for weight (proxied by the step-parent's BMI) and the child's BMI, providing further support that the validity assumption holds in our sample.

A further threat to the validity of the instruments lies in the specific functioning of the genes associated with obesity, in particular, the mechanisms through which they affect our outcome of interest (see von Hinke et al. 2016 for a detailed discussion). If the genetic variants related to adiposity are co-inherited with other genetic variants (known as linkage disequilibrium) or affect multiple traits or risk factors (known as pleitropy) that directly affect health care utilisation, then the exclusion restriction assumption of the instruments may not hold (Davey Smith and Ebrahim 2005; von Hinke et al. 2016). Linkage disequilibrium and pleitropy will only lead to biased IV estimates if there is a direct effect on health care utilisation;

if the adiposity-increasing genes are linked with other genes or functions that only influence health care utilisation via its effect on obesity, then the IV estimates will be consistent (Sheehan et al. 2008).

Although knowledge of the genetic architecture of common diseases is increasing, we do not yet have a complete understanding of the physical functions of genes involved in disease risk (Altshuler et al. 2008), and therefore we cannot know with certainty whether linkage disequilibrium or pleitropy are invalidating our instruments. However, in order to provide some evidence on the extent to which these potential threats to validity are influencing our estimates, we estimate falsification tests (in Section 4.3) using information on the child's health conditions. The idea behind the falsification tests is that an effect of child BMI on health conditions that are supposedly unrelated to obesity may signal linkage disequilibrium or pleitropy.

A third biological process, which the Mendelian randomisation literature suggests may lead to biased IV estimates, is 'canalisation'. This refers to the dampening of the effects of genetic variation during development via compensatory processes (Davey Smith and Ebrahim 2003). For example, due to canalisation, an individual who has a genetic variant associated with obesity may not experience adverse health outcomes, despite having obesity. Because the genotype influences health outcomes through alternative channels (not via obesity), it can violate the exclusion restriction assumption. Canalisation can lead to an attenuation of the estimated IV estimates (Dixon et al. 2016). However, testing for this is difficult because a child may still have obesity, but would not experience adverse health outcomes normally caused by obesity.

3.3 Two-part model of health care costs

We estimate a two-part model of health care costs (e.g. Mullahy 1998; Jones 2000), which involves estimating the probability of incurring positive Medicare costs, followed by estimating the amount of health care costs, conditional on incurring any. We specify the first part as a linear probability model (LPM) and the second part as a linear model of log costs.[4] In essence, the predicted probabilities from the first part of the model are multiplied with the predicted costs from the second part to derive unconditional predicted costs.

The distribution of the residuals in the log-linear model for costs are approximately normally distributed (skewness 0.11, kurtosis 2.66) and a conventional Park test on the log-scale residuals indicates there is no heteroscedasticity by BMI or any of the other continuous variables (age, mother's age, SEIFA index). Nevertheless, to transform predictions from the log-linear model into costs, we apply an adjusted Duan smearing estimate (Veazie et al. 2003), which increases the precision of the transformed estimates when the distribution of the error term is non-normal or unknown (Duan 1983; Manning 1998; Manning and Mullahy 2001). The smearing estimate is the predicted variance based on the regression of the squared residuals on the predicted health care costs from the log linear model. After application of the smearing estimate, our mean transformed predicted costs are near identical to mean actual costs.

To determine the additional health care costs of children with overweight or obesity, costs are predicted for children in normal weight, overweight and obese categories by setting the BMI z-score equal to the mean z-score of children in the relevant weight category. For example, for the pooled sample, the mean BMI z-score equalled 0.13, 1.45 and 2.15 for normal,

[4] Specifying the first part as a logit model provides identical estimates to the LPM. Both parts of the two-part model are estimated by linear IV methods using Stata user-written code '*ivreg2*' (Baum 2002). An alternative approach that is commonly used in models of health care costs is a generalised linear model (GLM), however the log linear model produced a better or near-identical fit for our data when compared with GLMs. We compared the model fit of GLMs with gamma and poisson distributions with log, power (0.5), and power (-1) links by examining the skewness of the residuals, the Pearson correlation between the residuals the predicted cost values, Link test, the mean prediction error, mean absolute prediction error, root mean square error and Copas test with v-fold cross validation for out-of-sample performance and over-fitting.

overweight and obese categories respectively. The predicted cost when BMI z-scores were set to 'obese' (or 'overweight') were subtracted from the predicted cost when BMI z-scores were set to 'normal' to give the additional cost attributable to obesity (overweight). Costs were predicted for each child and then averaged over the population of children. Standard errors were calculated using the bootstrap method (500 replications) which takes into account the sampling design.

In all regression models, we include a rich set of control variables. *Demographics*: gender, age (in months), ethnicity proxied by language spoken at home (English, European, Asian or other), and number of older/younger siblings. *Early childhood/in utero environment*: whether birth weight was low (<2500g), whether child was breast fed at 6 months and whether mother smoked while pregnant with respondent. *Socioeconomic background:* whether parent is single, health care concession status, measure of neighbourhood deprivation (SEIFA index), income quintiles, state or territory, geographical area (city, inner regional, remote or rural), mother and father's education level (university, diploma, high school, below high school). Because mother's characteristics can influence the child's health as well as the decision to seek treatment for the child, we include the following *mother's characteristics:* age, employment status (full-time employed, part-time employed, unemployed, not in the labour force), K-6 depression scale score (1-5), current smoker. Wave fixed effects were included for all pooled models. The descriptive statistics of the control variables are shown in the Appendix Table A1. To maintain sample size, missing variable dummies were included for household income, mother's smoking status (while pregnant and current), breast fed status, father's education, mother's employment and mother's depression scale. Respondents with missing values for

these variables were coded as zero and indicators for these observations are included in the models.[5]

We estimate the predicted costs for the pooled sample (from ages 6/7 to 12/13, with standard errors clustered at the individual level), and for each separate age group. The effect of obesity on total Medicare costs are estimated first, followed by a breakdown of costs by medical services (MBS) and Pharmaceuticals (PBS).

4. Results

4.1 Instrumental variables estimates – the effect of obesity on total Medicare costs

The main results for each part of the two-part model of health care costs are shown in Table 2. Columns (1) and (2) show the estimates from the reduced form equations, which regress child's health care costs on the two instruments (mother and father's BMI). These show that the instruments are not significantly associated with the probability that the child incurs any Medicare costs (column 1), but are positively associated with costs among children who incur Medicare costs (column 2). In the latter equation, mother and father's BMI are jointly significant (p=0.01), with father's BMI being a stronger predictor than mother's BMI.[6] The results from the first stage regression of the IV estimator are shown in Panel A of columns (3) and (4). The results show that for both the total sample (3) and the conditional sample (4), the BMI of both parents are highly correlated with the child's BMI – a one unit increase in BMI increases the child's BMI z-score by about 0.04 standard deviations. It is noteworthy that both parents' BMI are strong predictors, with father's BMI a slightly stronger predictor than

[5] Using the sample with complete observations for all covariates (n = 9,055) results in very similar, but slightly larger coefficient estimates.

[6] To allow more comparability with the size of the IV structural equation estimates, the coefficient estimates from the reduced form equation after standardising the mother and father's BMI (mean = 0, SD=1) were 0.025 and 0.033 respectively.

mother's BMI. The partial F-statistic for the instruments ranges from 116 to 134, which suggests that the instruments are powerful predictors of the child's BMI.

In Appendix Table A2 we show, using quantile regressions of the first stage equation, that the mother and father's BMI are strong and stable predictors of the child's BMI across the distribution of child's BMI, including in the upper tail associated with childhood obesity. In Appendix Figure A1, we plot the relationship between health care costs and child's BMI, comparing the actual BMI z-scores with fitted values (from the first stage equation) using locally weighted scatterplot smoothing. The figure shows that, compared to actual BMI values, predicted BMI z-scores show a steeper gradient with health care costs, suggesting negative selection bias. It also shows that the relationship between health costs and BMI (both actual and predicted) appears linear, especially over the relevant positive range of BMI z-scores.

The results from the second stage equation are shown in Panel B for the probability of incurring positive costs (column 3) and the log of costs (column 4). For both parts of the model, the large *p*-values of the Hansen J statistic provide support for the assumption that the instruments are valid. The effect of child's BMI on the probability of incurring any Medicare costs is small and insignificant. However, BMI has a large and positive effect on conditional costs ($p<0.01$). It shows that a one standard deviation increase in BMI z-scores leads to approximately a 17% increase in costs.[7]

For comparison, OLS estimates (of the linear probability model for part one and linear model for part two of the health care cost model) are shown in columns (5) and (6) respectively. The effect in part one of the model is similarly small and statistically insignificant. The effect

[7] Sensitivity analyses that use only the biological mother's BMI or only the biological father's BMI as a single instrument give similar results. Using the same sample, the effect of child's BMI on conditional costs is about 14% using mother's BMI and 19% using father's BMI ($p<0.05$). The 95% confidence intervals of these estimates overlap. Using just the biological mother's BMI in a larger sample of children (not restricted to also having biological father's BMI data), the effect of child's BMI on conditional costs is also similar at about 18% ($p<0.01$), suggesting our main effects are not influenced by the smaller sample size used.

in part two is positive and highly significant, but considerably smaller in size to the IV estimates; here a standard deviation increase in BMI z-score is associated with a 6% increase in costs.

For another point of comparison, we present estimates from a within-child fixed effects (FE) estimator in columns (7) and (8). The FE estimates are considerably smaller than the IV (and OLS) estimates and statistically insignificant for both parts of the health care cost model. This estimator essentially measures the relationship between changes in a child's BMI z-scores and changes in their health care costs from one wave to the next. It controls for all time-invariant characteristics of the child and parent, including potential confounders such as personality traits and health preferences. However, it will also sweep out the time-invariant contribution of genes related to obesity. This means, the FE estimator is identified differently to the IV estimator; i.e., from non-genetic variations in a child's BMI over time. We therefore do not emphasise comparisons between the IV and FE estimates. Additionally, if measurement error in BMI is present, then the FE estimator will exacerbate the downward bias due to measurement error. The FE estimator will also produce biased estimates if there are unobserved time-varying factors that affect both the child's BMI and their health care utilisation (e.g. a change in sport participation or injuries).

Table 2. Estimates from reduced-form equation IV, OLS and FE estimators

	Reduced Form		Instrumental variables (IV)		Non-IV (OLS)		Fixed-Effects (FE)	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Part one: Positive costs	Part two: Log costs	Part one: Positive costs	Part two: Log costs	Part one: Positive costs	Part two: Log costs	Part one: Positive costs	Part two: Log costs
A)			**First Stage**					
	Child's Medicare costs		*Child's BMI z-score*					
Mum's BMI in wave 1	0.001	0.005	0.036***	0.035***				
	(0.001)	(0.003)	(0.003)	(0.003)				
Dad's BMI in wave 1	0.001	0.008**	0.038***	0.039***				
	(0.001)	(0.004)	(0.004)	(0.004)				
F-stat of instruments			134.35	116.25				
B)			**Second Stage**					
			Child's Medicare costs					
BMI z-score			0.017	0.165***	0.005	0.057***	-0.000	0.038
			(0.018)	(0.055)	(0.006)	(0.017)	(0.011)	(0.030)
Hansen J *p*-value			0.979	0.424				
Observations	10804	8688	10804	8688	10804	8688	10804	8688

Notes: Clustered standard errors in parentheses, * p < 0.1, ** p < 0.05, *** p < 0.01. Models are pooled over ages 6-13 (i.e., waves 2-5). Child's Medicare costs comprise of MBS and PBS expenses for the year following BMI measurement. All models control for the full set of covariates. Underweight are excluded from regressions. Instruments in IV model are child's biological mother and father's BMI, measured at wave 1.

To test for possible non-linearity in the relationship between child's BMI and health care costs, we add the square of BMI z-scores in both the OLS and IV models. The OLS results are very similar to that in Table 2: the estimated association between a one standard deviation increase in BMI z-scores and health care costs is jointly insignificant for part one and 0.056 (jointly significant, *P*=0.013) for part two. For the IV model, BMI z-score and its square are jointly insignificant for part one and for part two the estimated effect of BMI z-scores is 0.394 (jointly significant at the 10% level, *P*=0.063). BMI squared is not statistically significant in any of the models. Compared with the model including the BMI squared term, our main model performs better on model fit tests, including mean prediction error, mean absolute prediction error, root mean square error and the Copas test with v-fold cross validation. Further checks with the reduced form model indicate that father and mother's BMI squared are not significant predictors of the child's health care costs. Therefore, our preferred model specification does not include BMI squared terms.

Our IV estimates are considerably larger than the OLS estimates. This difference between IV and OLS estimates is in line with previous studies that use an IV estimator to determine the effect of BMI on health care utilisation (Cawley and Meyerhoefer 2012; Biener et al. 2017; Doherty et al. 2017; Kinge and Morris 2017). The impact of measurement error on the OLS estimates is one possible explanation for this difference. By exploiting genetic variation in BMI, the IV models capture the long-term effects of BMI (Davey Smith and Ebrahim 2005). This means they avoid problems of short-term fluctuations in BMI and measurement error, which would downwardly bias OLS estimates (Davey Smith and Ebrahim 2005). Short-term fluctuations in BMI are likely to be particularly relevant in our population due to growth spurts and normal variations in height and weight gain during childhood (He and Karlberg 2001; Maynard et al. 2001).

The larger IV estimates could also imply that there are unobserved confounders that lead to an under-utilisation of health care services for children with obesity, i.e., negative selection bias. It is possible that there are greater barriers to accessing health care services in areas where obesity is more prevalent, for example in rural and remote regions or in low socioeconomic neighbourhoods. Qualitative studies on rural and remote Australian adolescents have shown that a number of difficulties in accessing health care exist, including: limited number of health care providers, high waiting times, longer distances to travel, a lack of reliable transport, higher out of pocket costs and limited choice of providers (Quine et al. 2003; Aisbett et al. 2007). Concerns about a lack of anonymity and social stigma may further contribute to an underutilisation of health services, especially in small communities (Quine et al. 2003; Aisbett et al. 2007).

We test whether poorer access to health care in rural communities is a plausible explanation for the larger IV estimates by comparing the downward bias in OLS estimates in a subsample of urban/city dwellers (N=5835) with non-urban (regional, rural or remote area) dwellers (N=4969). We find that the downward bias in OLS estimates is considerably larger among children who live in non-urban areas; for the conditional costs, the IV estimate (0.16) is 9.7 times the size of OLS estimates, whereas for children in urban areas, the IV estimate (0.17) is only 1.9 times the size of OLS estimate (see Appendix Table A3).

Other possible explanations for the larger IV estimates compared with OLS estimates include health neglect, which would suggest that parents (or children themselves) underinvest in all things health-related (including good nutrition and health care), or the presence of medical conditions (such as cancer or autoimmune diseases) that lead to both higher medical costs and a lower BMI (e.g. through loss of appetite).

4.2 Predicted costs of overweight and obesity

The predicted annual total Medicare costs that are attributable to overweight and obesity for the pooled sample and by age group are shown in Table 3. Estimates from the pooled OLS (non-IV) models, shown for comparison in column (1), suggest that overweight and obesity are associated with about a $21 and $33 higher total Medicare cost respectively, compared with a child who is normal weight. After taking into account unobserved confounders, the IV estimates in column (2) show that on average, compared to a child who is normal weight, an overweight child costs Medicare $63 (28%) more, and an obese child costs $103 (45%) more per year in non-hospital costs. Columns (3) to (6) show that the costs due to overweight are significantly higher across all childhood ages, but for obesity, there is greater variance in the costs and therefore significant differences are only seen in younger ages. The costs attributable to both overweight and obesity are greatest at age 6/7; compared to being normal weight, overweight costs an additional $81 (46%) and obesity an additional $143 (81%) at age 6/7.

Table 3. Predicted annual total Medicare costs per child by BMI category and age

	Non-IV	IV				
	(1) Pooled	(2) Pooled	(3) Age 6/7	(4) Age 8/9	(5) Age 10/11	(6) Age 12/13
Annual cost ($):						
Normal	240.53	226.84	175.52	222.22	237.96	283.97
	(5.273)	(7.525)	(7.516)	(10.985)	(12.543)	(15.437)
Overweight	261.34	289.33	256.13	283.04	305.19	314.65
	(6.666)	(16.906)	(30.005)	(26.506)	(25.655)	(29.564)
Obese	273.17	329.73	318.33	322.40	345.65	331.88
	(9.499)	(33.559)	(58.986)	(47.824)	(53.055)	(51.349)
Cost above normal weight ($):						
Overweight	20.76	62.50	80.61	60.82	67.23	30.68
	(6.065)	(21.930)	(7.805)	(10.374)	(11.414)	(11.828)
Obese	32.65	102.90	142.81	100.19	107.69	47.91
	(9.761)	(38.950)	(64.119)	(55.538)	(62.418)	(62.812)
	10804	10804	2858	2804	2633	2509

Note: Bootstrapped standard errors in parentheses. Estimated from two part models. All models include the full set of covariates. Pooled models are ages 6-13 (waves 2-5). Obese, overweight and normal weight are defined as the average BMI z-score for that BMI category in the relevant year. Adapted Duan smearing factor is applied to the transformed cost estimates. Costs are in 2015 AUD.

4.3 Falsification tests

4.3.1 Testing for non-genetic influences

To assess the credibility of our instrumental variables approach, we first test whether we would get similarly strong first stage estimates if we used the BMI of parents who are not biologically related, but live in the same household. If a large amount of variation in BMI is indeed due to non-genetic variation in parental preferences and the household environment, then we might expect the BMI of a step-parent to be equally as strong as that of a biological parent.

Table 4. Estimates from the first-stage equation – examining the role of stepfather's BMI in determining child's BMI

	(1)	(2)
Father's BMI	0.049***	0.039***
	(0.004)	(0.004)
Step-father	1.106**	0.991**
	(0.485)	(0.470)
Step-father x Father's BMI	-0.044***	-0.039**
	(0.017)	(0.016)
Biological Mother's BMI		0.034***
		(0.003)
Observations	8943	8850

Note: Clustered standard errors in parentheses, * p < 0.1, ** p < 0.05, *** p < 0.01. First stage estimates from instrumental variables model. Dependent variable is child's BMI z-score. All models are pooled over ages 6-13 (waves 2-5) and include the full set of covariates. Father's BMI and Mother's BMI are measured in Wave 1. Step-father is an indicator of whether the father is a step-father (non-biological). Sample includes children with biological and step-fathers.

In Table 4, we present results from the first stage equation when we use the BMI of all fathers (including step-fathers (3% of the sample)) as an instrument for the child's BMI. In column (1), the instrumental variable set is the father's BMI, an indicator for the father being a step-father and an interaction term between step-father and their BMI. In column (2), we add the biological mother's BMI as an additional instrument. These results indicate that a unit increase in the BMI of a biological father is associated with a 0.39 standard deviation increase

in the BMI of the child. However, for a step-father, a unit increase in BMI is associated with 0 change in the BMI of the child (=0.39 + (-0.39*1)). This suggests that a step-father's BMI does not predict the child's BMI.

Appendix Table A4 runs the placebo test another way, using a sub-sample of children who have step-fathers. The first-stage equation estimates for step-father's BMI are shown in column (1), and indicate that step-father's BMI is a poor predictor of child's BMI (the partial F-statistic is only 1.86). The biological mother's BMI, which is added in column (2), is shown to be a strong and statistically significant predictor of child's BMI, but step-father's BMI remains a poor predictor. Although the estimation sample is small, the results provide additional support that the main driver behind the association between parent BMI and child BMI is through genetics rather than the household environment.

4.3.1 Testing for alternative genetic pathways

Health conditions are not included in the main model because they are the main mechanism through which overweight and obesity influences health care costs. In line with previous studies (e.g. Johnson et al. 2006; Cawley and Meyerhoefer 2012; Batscheider et al. 2014), this approach allows the model to capture health care costs of obesity that arise through any health condition. However, one might be concerned with the validity of the instruments if the same genes that affect BMI are associated with other health conditions (either through pleitropy or linkage disequilibrium), and these conditions independently affect health care utilisation. While we cannot rule out pleitropy or linkage disequilibrium, we can control for a wide range of health conditions to understand the extent of bias that this may lead to.

Our main models already include early childhood indicators (low birth weight, breast-fed and mother smoked while pregnant) which are likely to capture effects in utero or during infancy, which may increase health risk independently from obesity. To further account for health conditions, we re-estimate the main IV model including indicators for several common

health conditions that are not recognised as being caused by obesity: attention deficit disorder (ADD), eczema and ear infections. These conditions are reported by the primary parent. The coefficient estimates from the two-part model, shown in Appendix Table A5 (columns 1 and 2), show that while all three conditions are associated with higher medical costs, BMI still has a large significant independent effect in the second part of the model (the conditional effect is 0.151, $p<0.01$).

In columns (3) and (4) we present the estimates when 15 other health limitations or disabilities (reported by the parent) are included in addition to the above three health conditions.[8] The estimated BMI coefficient in the conditional model (column 4) reduces to 0.141, but remains significant ($p<0.01$). The results reassuringly indicate that other associated health conditions are not likely to be driving the main estimated effect.

Another way to test the validity of our model is to determine whether child's BMI impacts those health conditions that are plausibly linked to having a heavier BMI, and does not impact health conditions that are unlikely to be caused by BMI. We select health conditions that had at least 1% of the sample with the condition to minimize imprecision due to small sample size. The conditions are sleep problems, asthma, attention deficit disorder, eczema, ear infections, difficulty learning and other physical problems. We estimate separate IV models using the seven different health conditions as the dependent variable, maintaining the same covariates and instrumental variables as our main model. The estimates from OLS and IV models are shown in Appendix Table A6.[9]

The IV results suggest that a heavier BMI significantly increases the likelihood that the child will have sleep problems and other physical problems. The medical literature indicates

[8] The 15 health limitations and disabilities are the complete list of conditions asked in LSAC at every wave. These are: sight problems, hearing problems, speech problems, blackouts, difficulty learning, limited use of arms or fingers, difficulty gripping, limited use of legs and feet, other physical condition, other disfigurement, difficulty breathing, chronic pain, nervous condition, mental illness, and head injury.
[9] We get almost identical results if we instead use probit and IV probit models.

that obesity in children increases the risk of sleep apnea and abnormal sleep patterns, and several other physical complications, including orthopedic abnormalities, gastroenterological conditions, insulin resistance, type 2 diabetes and hypertension (Daniels 2006), which might be captured under "other physical problems". Our models do not predict an effect of BMI on health conditions that are not recognised as being consequences of an elevated BMI (such as ear infections, ADD and eczema). While the conditions examined here are only a subsample of possible health conditions, the results increase confidence that the IV estimates of higher health care costs caused by obesity are likely to be operating through health conditions that are plausibly caused by a heavier BMI, and not through alternative genetic pathways.

4.4 Aggregate costs attributable to childhood overweight and obesity

In Australia, there are approximately 2.34 million children aged 6 to 13 (Australian Bureau of Statistics 2016), of whom about 7% are obese and 18% are overweight (estimates from LSAC). Extrapolating the results from the pooled IV model to the Australian population indicates that for children aged 6 to 13, compared to children of normal weight the additional non-hospital Medicare cost attributable to obesity is approximately $16.6 million and the further cost attributable to overweight is $26.6 million (in 2015 AUD).[10] These results are shown in Table 5 together with the estimated costs under a non-IV approach. Failing to account for unobserved confounders would lead to an underestimate of the total cost incurred by overweight and obese children by approximately $29 million.

[10] Population weighted means of obesity and overweight were estimated at each wave of LSAC to derive age-specific prevalence rates. These were multiplied by the population of children at the relevant age group using 2016 population estimates (Australian Bureau of Statistics 2016). Annual costs per person, derived from the pooled two part model, were multiplied by the population obese (or overweight) and then averaged across the sample population. Standard errors were calculated using the bootstrap method (500 reps), taking the sampling design into account.

Table 5. Annual Medicare cost attributable to obesity and overweight children aged 6 to 13 (millions of 2015 AUD)

	Non-IV estimates	IV estimates	Population
Obesity	$5.264	$16.586	161,192
	(1.417)	(4.477)	
Overweight	$8.839	$26.614	425,831
	(2.065)	(6.627)	

Note: Bootstrapped standard errors in parentheses. Medicare costs includes all non-hospital MBS and PBS costs. Average cost per child obese and overweight are from pooled two part models used (shown in columns 1 and 2 of Table 3). Population estimates derived by applying rates of overweight and obese from LSAC at each age group to the Australian Demographic Statistics (Australian Bureau of Statistics 2016).

4.5 Heterogeneity by type of Medicare cost

Table 6 presents estimates from the predicted costs (derived from the two-part models) by main source of expenditure; medical services (MBS) or pharmaceuticals (PBS). The estimates from the non-IV estimators are shown in columns (1) and (3) for comparison. Both the IV and non-IV estimates show that a majority of the additional cost attributable to overweight and obesity is through an increase in MBS medical services. The IV estimates in Column (2) show that on average, a child who is overweight incurs an additional $44 and a child who is obese incurs an additional $71 in MBS costs per year compared with a normal weight child. The additional costs incurred through the PBS are smaller and less precisely estimated.

It is useful to gain an understanding of which types of Medical services are being utilised more frequently as a result of obesity. In Appendix Table A7, we show estimates from linear OLS and IV regressions on the effect of BMI on the number of MBS items incurred under the following mutually exclusive and complete list of MBS categories: general practitioner (GP) visits, bulk billing incentive payments, specialist visits, mental health services, diagnostic and imaging, pathology and other.

Table 6. Predicted annual Medicare costs per child by BMI category and split by type of Medicare cost (in 2015 AUD)

	MBS costs (Medical care)		PBS costs (Pharmaceuticals)	
	(1)	(2)	(3)	(4)
	Non-IV	IV	Non-IV	IV
Annual cost ($):				
Normal	217.56	209.45	22.60	19.64
	(4.375)	(6.313)	(1.878)	(2.105)
Overweight	235.57	253.44	24.32	30.76
	(5.664)	(13.660)	(2.552)	(7.406)
Obese	245.73	280.72	25.29	39.00
	(8.155)	(26.072)	(3.481)	(16.203)
Cost above normal weight ($):				
Overweight	17.93	43.99	1.76	11.12
	(5.236)	(17.820)	(2.033)	(8.526)
Obese	28.17	71.27	2.69	19.37
	(8.418)	(30.537)	(2.818)	(11.363)
Observations	10804	10804	10804	10804

Note: Bootstrapped standard errors in parentheses. MBS = Medicare Benefits Schedule. PBS = Pharmaceutical Benefits Scheme. Estimated from two part models. All models are pooled over ages 6-13 (waves 2-5). Obese, overweight and normal weight are defined as the average BMI z-score for that BMI category in the relevant year. Adapted Duan smearing factor is applied to the transformed cost estimates. Costs are in 2015 AUD.

The results for the pooled sample (aged 6-13) under both OLS and IV approaches indicate that a higher BMI leads to greater GP utilisation and this in turn is associated with more bulk billing incentive payments.[11] Under the IV estimator, an increase in BMI by one standard deviation increases the number of GP consultations by about half a visit per year, or about 17% at the mean (0.500/2.829). The IV results indicate that specialist doctor consultations also increase as BMI increases (by around 30% at the mean (0.104/0.353) for a one standard deviation increase in BMI). However, there is no significant effect of BMI on the utilisation of mental health, diagnostic, pathology, or other services.

[11] Bulk billing incentive payments are commonly tied to GP consultations to children due to the Government's program to incentivise bulk billing (free medical care at the point of service) to patients under the age of 16 for non-referred services. Non-referred services are typically provided by GPs, but can also include certain pathology or diagnostic imaging services.

4.6. Hospital utilisation

While Medicare costs in our data do not contain hospital costs,[12] we estimate the effect of BMI on the likelihood the child is hospitalised (for an overnight stay for any reason) to gain some insight into how much this omission may affect total Government health care costs. Hospital visits are reported retrospectively for the past 12 month period by the parent at each wave. We use data on hospital stays from the subsequent wave to ensure temporal ordering from BMI to hospital stays. Hospital stays for children of the age group in this study are relatively uncommon. On average, about 4.2% of children aged 6 to 13 reportedly stay at least once in a hospital in a subsequent 12 month period. Appendix Table A8 shows linear IV estimates using the same instruments and covariates as in our main models. The results indicate that BMI does not have a significant effect on the likelihood of staying in a hospital for the pooled sample (age 6 to 13) or at each separate age. While this analysis is on the probability of a (reported) hospital stay and not on health care costs due to hospital stays, it provides some indicative evidence that hospital costs may not differ significantly by the child's BMI. Therefore, the overall impact of obesity on Government health care costs may not be too different from the estimates in this study, despite the omission of hospital costs.

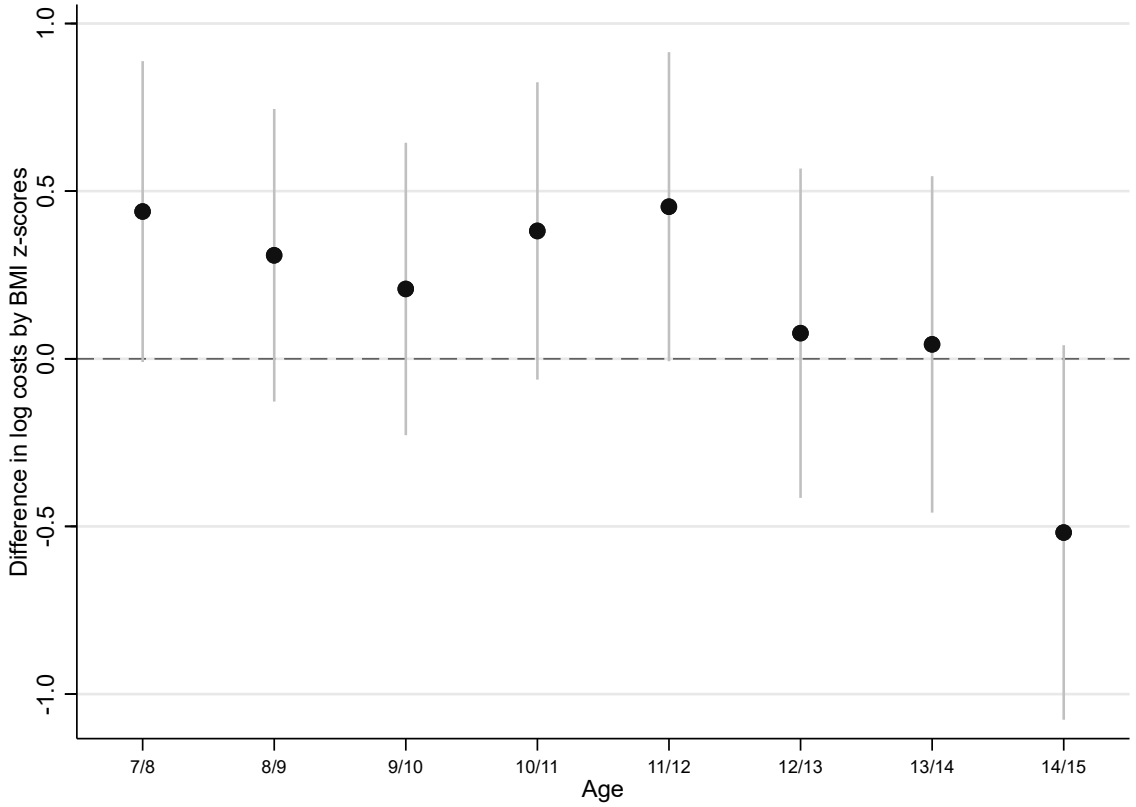
4.7. Longer-term health care costs

While the focus of this study is on short-term costs due to a higher BMI among children, we utilise the panel nature of the data to provide some indication of the longer-term costs. We do this by first estimating the effect of a child's BMI at age 6/7 (wave 2) on the total health care costs over the eight subsequent years, until age 14/15, and second, by estimating the effect of BMI at age 6/7 on annual health care costs for each year until age 14/15. Here, the dependent

[12] Public hospital records are administered separately by each Australian State and Territory, and are not part of the Australian Medicare administrative records.

variable is the inverse hyperbolic sine of total (or annual) costs. This transformation approximates the natural log of costs, but it allows individuals with zero costs to be modelled in one equation (Pence 2006), thereby simplifying the interpretation of the overall effect of BMI.

Figure 2. IV estimates of the effect of BMI z-scores at age 6/7 on annual Medicare costs from age 7/8 to age 14/15.



Notes: Each dot represents the coefficient estimate for BMI z-score at age 6/7 (wave 2) from the IV model. The dependent variable is the inverse hyperbolic sine of total annual costs by age. The models all include the full set of covariates at age 6/7. Underweight children are excluded from regressions. Instrumental variables are the child's biological mother and father's BMI, measured at wave 1.

The coefficient estimates from OLS and IV estimates for the total costs suggest that a child with a one standard deviation higher BMI at age 6/7 incurs health care costs that are about 8% ($p<0.05$) and 10% ($p>0.10$) higher respectively over the subsequent eight-year period. Due

to large standard errors, the IV estimates are statistically insignificant.[13] The IV coefficient estimates for the longer-tem annual health care costs are shown in Figure 2. The results suggest that a higher BMI at age 6/7 has a positive effect on health care costs for the subsequent five years (with stronger and statistically significant effects at age 7/8 and 11/12), but the effect wanes from age 12/13 when the children reach early adolescence.

5. Discussion

In line with global trends, the rate of paediatric obesity in Australia has tripled in recent decades (Wang and Lobstein 2006). While recent studies have shown a positive association between obesity and health care costs during childhood (Au 2012; Batscheider et al. 2014; Clifford et al. 2015), there is little evidence to date on the magnitude of health care consequences caused by childhood obesity. This study contributes to this space by investigating the government-funded health care cost consequences of childhood obesity in Australia. Identification of the effect of childhood obesity is achieved through an instrumental variables approach and the use of BMI of biological parents as instrumental variables. This approach harnesses the large role of genetics in determining obesity. Our main results demonstrate that among children aged 6 to 13, relative to having a BMI in the normal weight range, the non-hospital Medicare costs that are due to being overweight are $63 (28%) more, and that are due to obesity are $103 (45%) more per year per child. Extrapolating these results to the Australian population of children aged 6 to 13 suggests that this amounts to a total cost due to overweight and obesity of approximately $43 million (2015 AUD) per year. This represents about 0.1% of the total Australian government expenditure on Medicare in 2014-15 (AIHW 2016).

[13] However, if we condition on children with positive costs over the eight-year period (99% of the sample), the IV estimates are statistically significant; a one standard deviation higher BMI at age 6/7 increases total costs by 17% ($p<0.10$).

Our IV estimates are considerably larger than non-IV estimates, which could indicate that measurement error in BMI or unobserved factors are biasing OLS estimates downwards. This indicates that previous estimates of health care costs associated with childhood obesity in Australia (Au 2012; Clifford et al. 2015) are likely to underestimate the total cost attributable to obesity. The larger IV estimates compared to non-IV estimates is in line with previous studies that have used an IV approach to estimate the health care utilisation caused by obesity among youths in Ireland (Doherty et al. 2017), England (Kinge and Morris 2017) and the United States (Biener et al. 2017). In the only other study using an IV approach that measures utilisation in costs, Biener et al. (2017) find that obesity raises annual medical costs by US $1,354 per child, which is four times larger than their non-IV estimates of US $310 per annum per child. Our comparably smaller IV estimate for obesity (which converts to approximately US $80 per annum per child) may be due to our focus on government-funded primary care services and medications, while Biener et al. (2017) also include hospital, out of pocket and private health insurance costs. While we are unable to provide estimates on the effect of childhood obesity on hospital costs, our analyses using parent-reported visits to the hospital suggest that the probability of staying overnight in a hospital does not differ by BMI, and therefore the consequences of the omission of hospital data may be small in our context. Other possible explanations for differences in findings include sample age differences (their children were aged 11 to 17, while ours were aged 6 to 13) and broader cross-country differences in health care expenditure (total health spending per capita in the US is double that of Australia (OECD 2018)).

When we disaggregate the costs by age, we find that the costs of having a higher BMI are greatest at ages 6/7 and smallest at age 12/13. This finding is in line with (Kinge and Morris 2017), who also find large positive effects of BMI and obesity on doctor utilisation among younger children (aged 4 to 12), but not older children (aged 13 to 18). However, in contrast,

Doherty et al. (2017) find that the probability of seeing a general practitioner or inpatient stay is only significantly higher for children with obesity at age 13 and not at age 9. The differences in findings may be due to health system contexts or to differences in results when health care costs instead of the probability of a visit is examined.

Our study finds that childhood obesity leads to considerably higher medical expenses that are borne by the public. To the extent that this represents a negative externality (and not simply a transfer of costs) (Cawley 2015), our results support the justification for greater government investment in programs to reduce obesity. The findings have implications for economic evaluations of policies and interventions aimed at reducing childhood overweight and obesity. Existing economic evaluations which have used estimates of health care costs associated with childhood obesity (e.g. Gortmaker et al. 2015) or that have not included any health care costs (cost-offsets) incurred during childhood (e.g. Haby et al. 2006), are likely to underestimate the expected returns on investing in effective programs.

We find that the excess costs due to overweight and obesity are driven by medical care expenses through the MBS, specifically, from a higher utilisation of general practitioner and specialist doctor services. A child's BMI does not significantly affect the utilisation of mental health, dental, pathology, diagnostic or other medical services, nor does it have a significant effect on prescription pharmaceutical expenses through the PBS.

Health care costs are measured as the total cost over the one year following BMI measurement. While this period captures short-term health effects due to obesity, it does not capture health conditions that are slower-developing or that do not have any health consequences until adulthood. Our supplementary analyses on the longer-term costs of BMI indicate that a higher BMI at age 6/7 increases health care costs over several subsequent years. This may be partly due to slower-developing conditions, or possibly due to a persistence of BMI. The waning of the effect after age 11/12 suggests a higher BMI at age 6/7 is not causing

irreversible chronic conditions. Further research into the dynamics of BMI and health care costs is needed to understand the role of BMI persistence. As future waves of LSAC become available, it will be possible to understand the health care consequences of childhood obesity beyond early adolescence.

Key strengths of this study are that it uses height and weight that are measured for each child, and this is linked to complete administrative records of non-hospital health care expenses through Australia's Medicare health insurance system. This minimises bias due to reporting error that may be present in studies that rely on parent-reports of height and weight, and/or retrospectively reported utilisation of medical care. An additional strength is that longitudinal data and precise dates of service utilisation enable temporal ordering from obesity measurement to health care utilisation. This minimises potential concerns arising from reverse causality. Furthermore, the rich survey data contained in LSAC allows us to control for a wide range of possible confounders, including socioeconomic background early childhood health conditions and parental characteristics (e.g. such as education level, employment status, smoking status and mental health), which may be associated with both parental BMI and health care seeking behaviour.

However, our paper has several limitations. First, the validity of the instruments. Our identification assumes that the BMI of biological parents is strongly correlated with the child's BMI, but not with the child's health care expenses. As with all other IV approaches, we cannot directly test that the exclusion restriction holds. A large literature indicates that genetics plays a strong role in determining obesity, and that the shared family environment plays little or no detectable role in the similarity in BMI across family members. While we include a rich set of control variables and the results from the overidentification test and falsification tests using step-parent's BMI and health conditions all provide support for the validity of our instruments, we acknowledge the possibility that the genes that affect weight may be related to other traits

that directly affect residual health care costs. It is not obvious what these could be, given the large set of parental and child covariates that we include. Nevertheless, as recognised by earlier studies (e.g. Cawley and Meyerhoefer 2012) this is a possible limitation of the instruments.

Similarly, we recognise that alternative genetic pathways that arise through pleitropy, linkage disequilibrium and canalisation could potentially invalidate our instruments. Our falsification tests suggest that BMI relates to higher health care costs through plausible health conditions, and not through conditions that are supposedly unrelated to obesity. However, we acknowledge it is not possible to rule out alternative genetic pathways.

We also acknowledge the possibility that a parent's BMI may influence the health care utilisation of parents, which may in turn influence the contact that children have with health care providers, irrespective of the child's BMI. However, when we regress the probability of a child incurring health care costs on each parent's BMI as well as the child's BMI and all covariates, the mother and father's BMI are each associated with less than 0.05% of the increase in health care costs and neither are significant (p>0.6). While not definitive, this test provides further evidence to support the validity of the instruments.

Our IV results should be interpreted as a local average treatment effect (LATE). The effect of obesity on health care costs may be heterogeneous and the IV estimates identify the effect on health care costs that are due to a child's BMI that is induced by parents' historical BMI (taken when the child was 5 years old). In other words, the variation in children's BMI comes from variation in the genetic predisposition to a higher BMI, which is expressed in childhood. Encouragingly, quantile regressions of the first stage equation indicate that the instruments influence the child's BMI across the BMI distribution, including the upper tail where children are obese. Because a majority of children are raised by their biological parents, our IV estimates are likely to be generalizable to the wider population. However, our estimated effect may not be generalisable to children who's elevated BMI is entirely due to non-genetic factors.

This raises the question, how useful are our results for inferring the medical cost consequences of childhood obesity reduction policies? After all, policies are unlikely to alter the genetic makeup of children. It is likely that an interaction between genes and the environment explains the current obesity epidemic. As Barness et al. (2007) explain, "The steadily increasing prevalence of obesity among children and adults in many countries suggests that the environment is becoming more permissive to the expression of genetic tendencies toward obesity which may have been advantageous to our ancestors in a more hostile environment." (p.3022). To the extent that policies can reduce the BMI of children who are genetically predisposed to obesity, our estimates suggest that obesity reduction policies will lead to considerable medical cost savings during childhood.

Despite limitations, this study provides unique evidence on the publicly-funded health care costs caused by childhood obesity in Australia. Economic studies that have evaluated the benefits of childhood obesity programs have typically ignored any cost savings due to medical expenses of obesity during childhood, under the assumption that health consequences do not materialise until adulthood (e.g. Haby et al. 2006). This study demonstrates that medical costs of obesity incurred during childhood are substantial and larger than previously estimated. This implies that the economic returns from investing in childhood obesity prevention programs has been considerably underestimated.

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Table A1 Summary statistics of all key variables

	Mean	SD	Min	Max
Health care costs:				
Medicare costs > 0	0.812	(0.391)	0	1
Total Annual Medicare costs ($)	246.796	(372.943)	0	4913
MBS costs > 0	0.808	(0.394)	0	1.000
Annual MBS costs ($)	223.366	(320.109)	0	4913
PBS costs >0	0.188	(0.390)	0	1.000
Annual PBS costs ($)	23.429	(138.976)	0	3955
Covariates:				
BMI z-score	0.490	(0.846)	-1.4	3.0
Male	0.523	(0.499)	0	1
Age (months)	116.924	(27.609)	75	166
English	0.870	(0.336)	0	1
European	0.036	(0.187)	0	1
Asian	0.054	(0.227)	0	1
Other language	0.039	(0.194)	0	1
Birthweight <2500g)	0.062	(0.240)	0	1
Breastfed at 6 months	0.594	(0.491)	0	1
Mother smoked while pregnant	0.127	(0.333)	0	1
No. older siblings	0.828	(0.906)	0	8
No. younger siblings	0.777	(0.876)	0	5
Single mum	0.066	(0.249)	0	1
Health care concession card	0.312	(0.463)	0	1
SEIFA	10.124	(0.735)	6.2	12.1
Income quintile 1	0.175	(0.380)	0	1
Income quintile 2	0.171	(0.376)	0	1
Income quintile 3	0.174	(0.379)	0	1
Income quintile 4	0.176	(0.381)	0	1
Income quintile 5	0.161	(0.368)	0	1
NSW state	0.335	(0.472)	0	1
VIC state	0.245	(0.430)	0	1
QLD state	0.206	(0.405)	0	1
SA state	0.069	(0.253)	0	1
WA state	0.092	(0.288)	0	1
TAS state	0.028	(0.164)	0	1
NT territory	0.007	(0.086)	0	1
ACT territory	0.018	(0.132)	0	1
City	0.556	(0.497)	0	1
Inner regional	0.259	(0.438)	0	1
Remote	0.029	(0.167)	0	1
Mum university degree	0.303	(0.460)	0	1
Mum diploma	0.416	(0.493)	0	1
Mum year 12	0.103	(0.304)	0	1
Mum no high school	0.178	(0.382)	0	1
Dad university degree	0.285	(0.451)	0	1
Dad diploma	0.443	(0.497)	0	1
Dad year 12	0.080	(0.271)	0	1
Dad no high school	0.123	(0.329)	0	1

age mother at birth	31.067	(4.798)	15	48
Mum full time employed	0.249	(0.433)	0	1
Mum part time employed	0.496	(0.500)	0	1
Mum unemployed	0.025	(0.157)	0	1
Mum not in labour force	0.229	(0.420)	0	1
Mum K6 Depression score	4.274	(1.093)	0	5
Mum current smoker	0.141	(0.348)	0	1
Wave 3	0.257	(0.437)	0	1
Wave 4	0.239	(0.426)	0	1
Wave 5	0.238	(0.426)	0	1
N	10804			

Note: Means are calculated using population sampling weights. Underweight children excluded from sample. Also included as covariates in main models but not shown in the table are missing indicators for household income, mother's smoking status (while pregnant and current), breast fed status, father's education, mother's employment and mother's depression scale. All estimates are from pooled data (age 6-13).

Table A2. Estimates of the first stage regression: comparing OLS and Quantile regression models of child's BMI on the instruments

	OLS	Quantile Regression				
	(1)	(2)	(3)	(4)	(5)	(6)
		Q 0.1	Q 0.25	Q 0.5	Q 0.75	Q 0.90
Mother's BMI	0.036***	0.026***	0.036***	0.041***	0.041***	0.037***
	(0.003)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Father's BMI	0.038***	0.031***	0.042***	0.043***	0.041***	0.037***
	(0.004)	(0.003)	(0.003)	(0.003)	(0.003)	(0.002)
Observations	10804	10804	10804	10804	10804	10804

Note: Clustered standard errors in parentheses, * p < 0.1, ** p < 0.05, *** p < 0.01. Dependent variable is child's BMI z-score. Column 1 estimated using OLS. Columns 2-6 estimated using Quantile Regression at selected quantiles. All models are pooled over ages 6 to 13 (waves 2-5). Models include the full set of covariates. Instruments in IV model are child's biological mother and father's BMI taken at wave 1. Underweight children are excluded from the sample.

Table A3. OLS and IV estimates of the effect of child's BMI z-scores on total Medicare costs by area of residence

	Urban		Non-urban	
	(1)	(2)	(3)	(4)
	Part one: Positive costs	Part two: Log costs	Part one: Positive costs	Part two: Log costs
A) *OLS*				
BMI z-score	0.009	0.092***	-0.000	0.016
	(0.007)	(0.022)	(0.008)	(0.026)
B) *IV*				
BMI z-score	0.012	0.174**	0.021	0.155*
	(0.024)	(0.074)	(0.026)	(0.080)
Observations	5835	4794	4969	3894

Notes: Clustered standard errors in parentheses, * p < 0.1, ** p < 0.05, *** p < 0.01. Urban = subsample of population living in cities. Non-urban = subsample of population living in regional, rural or remote areas (i.e., not in cities). Models are pooled over ages 6-13 (i.e., waves 2-5). Child's Medicare costs comprise of MBS and PBS expenses for the year following BMI measurement. All models control for the full set of covariates. Underweight are excluded from regressions. Instruments in IV model are child's biological mother and father's BMI, measured at wave 1.

Table A4. Estimates from the first-stage equation for sample of children with step-fathers

	(1)	(2)
Stepfather's BMI	0.028	0.024
	(0.021)	(0.017)
Biological Mother's BMI		0.049***
		(0.013)
Observations	273	272
F-statistic	1.858	7.518

Note: Clustered standard errors in parentheses, * p < 0.1, ** p < 0.05, *** p < 0.01. Dependent variable is child's BMI z-score. All models are pooled over ages 6-13 (waves 2-5) and include the full set of covariates. Step-father's BMI & Mother's BMI are measured in Wave 1.

Table A5. IV estimates on the effect of child's BMI z-scores on total Medicare costs over ages 6 to 13, including health conditions

	(1) Part one: Positive costs	(2) Part two: Log costs	(3) Part one: Positive costs	(4) Part two: Log costs
BMI z-score	0.014	0.151***	0.012	0.141***
	(0.018)	(0.055)	(0.018)	(0.053)
Attention deficit disorder	0.138***	0.912***	0.125***	0.862***
	(0.017)	(0.088)	(0.017)	(0.090)
Eczema	0.053***	0.215***	0.053***	0.214***
	(0.012)	(0.041)	(0.012)	(0.041)
Ear infections	0.091***	0.240***	0.087***	0.232***
	(0.020)	(0.074)	(0.020)	(0.073)
Sight problems			0.063*	0.230**
			(0.035)	(0.103)
Hearing problems			0.011	0.014
			(0.040)	(0.108)
Speech problems			0.031	-0.055
			(0.025)	(0.089)
Blackouts			-0.003	0.824***
			(0.064)	(0.212)
Difficulty learning			0.053**	0.208**
			(0.022)	(0.088)
Limited use of arms or fingers			-0.032	0.363
			(0.069)	(0.238)
Difficulty gripping			0.049	-0.153
			(0.039)	(0.186)
Limited use of legs and feet			0.051	0.313
			(0.046)	(0.208)
Other physical condition			-0.011	0.071
			(0.035)	(0.120)
Other disfigurement			0.074	0.552
			(0.053)	(0.367)
Difficulty breathing			0.072**	0.254**
			(0.035)	(0.125)
Chronic pain			0.029	-0.320**
			(0.041)	(0.151)
Nervous condition			0.004	0.035
			(0.047)	(0.182)
Mental illness			-0.000	0.417**
			(0.050)	(0.175)
Head injury			0.087	0.361*
			(0.054)	(0.219)
Observations	10803	8687	10803	8687

Notes: Clustered standard errors in parentheses, * p < 0.1, ** p < 0.05, *** p < 0.01. All models are pooled over ages 6-13 (waves 2-5) and control for the full set of covariates. Underweight are excluded from regressions. Instruments are child's biological mother and father's BMI, measured at wave 1.

Table A6. IV estimates on the effect of child's BMI z-scores on selected health conditions

	(1) OLS	(2) IV	(3) Mean
Sleep problems	0.017**	0.076***	0.307
	(0.007)	(0.024)	
Asthma	0.017*	0.039	0.302
	(0.009)	(0.032)	
Attention deficit disorder	-0.006**	0.008	0.021
	(0.003)	(0.008)	
Eczema	-0.005	0.019	0.111
	(0.005)	(0.020)	
Ear infections	0.003	0.010	0.027
	(0.003)	(0.009)	
Difficulty learning	0.000	0.004	0.026
	(0.002)	(0.008)	
Other physical condition	0.003**	0.016**	0.013
	(0.002)	(0.007)	

Notes: Clustered standard errors in parentheses, * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Each row is a separate regression and shown are the coefficient estimates for child's BMI z-score. Mean of health conditions (dependent variables) in column (3) are calculated using population sampling weights. All models are pooled over ages 6-13 (waves 2-5) and control for the full set of covariates. Sample consists of 10,803 observations. Underweight are excluded from regressions. Instruments are child's biological mother and father's BMI, measured at wave 1.

Table A7. OLS and IV estimates of the effect of BMI on number of MBS service items utilised

	(1) GP visit	(2) Bulk Billing	(3) Specialist visit	(4) Mental Health	(5) Diag-nostic	(6) Path-ology	(7) Other
A) OLS							
BMI z-score	0.216***	0.202***	0.023	-0.010	0.023	0.034	0.029*
	(0.052)	(0.051)	(0.015)	(0.019)	(0.014)	(0.037)	(0.018)
B) IV							
BMI z-score	0.500***	0.628***	0.104**	0.066	0.039	0.191	-0.044
	(0.191)	(0.204)	(0.053)	(0.059)	(0.045)	(0.127)	(0.054)
Outcome mean	2.946	2.370	0.348	0.187	0.519	0.873	0.587
Observations	8638	8638	8638	8638	8638	8638	8638

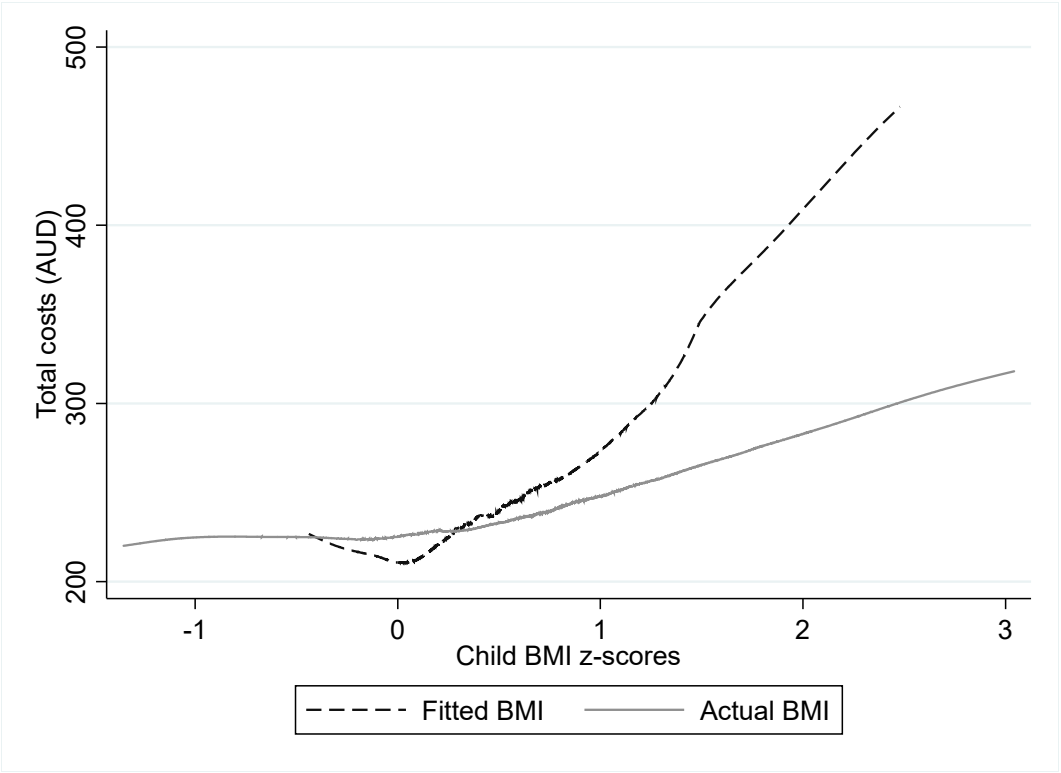
Note: Clustered standard errors in parentheses, * p < 0.1, ** p < 0.05, *** p < 0.01. MBS = Medicare Benefits Schedule. All models are pooled over ages 6-13 (waves 2-5) and control for the full set of covariates. Underweight excluded from regressions.

Table A8. IV estimates of the effect of child's BMI on the probability of having a hospital stay

	(1) Pooled	(2) Age 6/7	(3) Age 8/9	(4) Age 10/11	(5) Age12/13
BMI z-score	0.007	0.031	0.001	-0.015	0.009
	(0.009)	(0.021)	(0.018)	(0.014)	(0.018)
Mean outcome	0.042	0.047	0.039	0.035	0.044
Observations	10193	2769	2687	2471	2266

Note: Clustered standard errors in parentheses, * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Full set of covariates included in all models. Hospital stay is coded as 1 if parent reported in the subsequent wave that their child stayed overnight in a hospital at least once in the last 12 months; therefore indicates a hospital stay one year into the future from when BMI was measured. Underweight excluded from regressions. Instruments in IV model are the BMI of the child's biological mother and father. Estimates from linear IV models (near-identical results are obtained from IV probit models).

Figure A1. Locally weighted scatterplot smoothing: health care costs and BMI z-scores (actual versus fitted)



Notes: pooled over ages 6-13 (waves 2-5). Fitted BMI values are from regression BMI on the instruments (child's biological mother and father's BMI, measured at wave 1) and full set of covariates.