The impact of childhood obesity on health and health service use: an instrumental variable approach

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Abstract

In the following paper we estimate the impact of obesity in childhood on health and health service use in England using instrumental variables. We use data on children and adolescents aged 3-18 years old from fifteen rounds of the *Health Survey for England* (1998-2012), which has measures of self-assessed health, primary care use, prescribed medication use, and nurse-measured height and weight. We use instruments for child obesity using genetic variation in weight. We detect a few potential issues with the validity of the instrument; however further testing does not suggest that this has an effect on our results. We find that obesity has a statistically significant and negative impact on self-rated health and a positive impact on health service use in girls, boys, younger children (aged 3-10) and adolescents (aged 11-18). We detect significant endogeneity, which suggest that previous studies underestimate the impact of childhood obesity on health and health service use. For example, obesity is associated with and increased probability of doctor utilisation of 2%, but the IV results show that obesity increase the probability of use by 10%. This suggests that obesity has consequences for health and health service use when the children are still young.

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1. Introduction

Obesity levels have escalated dramatically over the past 30 years resulting in increased disease burden and major cost consequences (Bierl, Marsh et al. 2013, Ng, Fleming et al. 2014). According to estimations by Ng et al., the prevalence has also increased substantially in children and adolescents in developed countries. In 2013, 24% of boys and 23% of girls were overweight or obese (Ng, Fleming et al. 2014), compared with 17% of boys and 16% of girls in 1980.

Obesity is therefore considered to be a global public health concern and many studies have found that adult obesity accounts for a substantial proportion of health care costs (Bierl et al., 2013). An important question is whether obesity in childhood has consequences for health and health care expenditures when the children are still young. A relatively large literature supports the negative health effects of obesity, as there are significant negative associations between youth obesity and various measures of health (Tsiros, Olds et al. 2009). These studies consistently report that, compared with normal weight, obese youth have lower health-related quality of life, lower self-assessed general health (SAH), lower self-esteem and more chronic conditions (Skinner, Mayer et al. 2008, Tsiros, Olds et al. 2009, Griffiths, Parsons et al. 2010). Recently a number of studies have also looked at adolescents and children to explore whether there are short term cost consequences of youth obesity. However, evidence of the impact of childhood obesity on costs while the children are still young is ambiguous (John, Wenig et al. 2010). In addition, cost-effectiveness of interventions aimed at childhood obesity struggle to reach acceptable values (John, Wenig et al. 2010).

This means that, although health problems associated with obesity in adults has been translated into substantial health care expenditures, the same cannot be said for children and adolescents. Relatively few studies have looked at obesity in childhood and health service use, and all of them have used US data. Two US studies found excess overall health care costs in obese children compared with overweight adolescents aged 12-18 (Buescher, Whitmire et al. 2008) and in children/adolescents aged 8-19 (Finkelstein and Trogdon 2008). Monheit et al. who also used US data on adolescents aged 12-19, found significant positive associations between overweight and health care expenditures in girls, but not boys (Monheit, Vistnes et al. 2009). Significant associations has also been found between child obesity and outpatient visits, prescription drug use and emergency room visits using US survey data including children aged 6-19 (Trasande and Chatterjee 2009). On the other hand Johnson et al., who

looked at US children aged 4-17, did not find significant increased health care utilisation (Johnson, McInnes et al. 2006). A study by Skinner et al. looked at both SAH and health care expenditures. Although, they found a negative association between obesity and SAH in children aged 6-17, they did not find any significant relationship between obesity and expenditures for these children (Skinner, Mayer et al. 2008). These studies mainly use regression-based approaches, regressing health and health service use against an obesity measure plus other variables likely to affect health service use. Few account for the potential endogeneity of obesity, which may lead to biased results.

One earlier study has suggested that by using univariate regression methods one will underestimate the underestimate the impact of obesity (in adults) on health care use. Cawley and Meyerhoefer analysed the impact of obesity on health care expenditure using US data for 2000-2005 in a sample consisting of adults aged 20 to 64 (Cawley and Meyerhoefer 2012). They used an instrumental variable (IV) approach to account for endogeneity. BMI measures of the oldest biological child were used as instruments for individual BMI and obesity. When not controlling for endogeneity, obesity increased annual medical care cost by US$656 per person. After controlling for endogeneity the annual cost increase was US$2741 per person. Hence, controlling for endogeneity produced a considerably larger effect of obesity, and the authors conclude that previous studies that did not account for endogeneity produce biased results.

There are four reasons why obesity and medication use may be correlated:

i. *Causal impact*. Obesity has a negative impact on health and a positive impact on health service use. Obesity may directly impair health as it is an important risk factor for a number of diseases in children including diabetes (high fasting plasma glucose levels), heptic steatosis, sleep apnea, orthopaedic conditions, and hypertension (Rashid and Roberts 2000, Poussa, Schlenzka et al. 2003, Wing, Hui et al. 2003, Skinner, Mayer et al. 2008, Juonala, Magnussen et al. 2011). The impaired health status increase health service use as the obese are more likely to utilise health services to manage these conditions, compared with normal weight. In addition, childhood obesity may directly increase health care use. For example, obese children might be prescribed lifestyle interventions and appetite-suppressing drugs for weight reduction. In England guidelines produced by the National Institute for Health and Clinical Excellence (NICE) state that a number of lifestyle

and behavioural interventions aimed at weight reduction should be considered for treatment in overweight and obese children (National Institute for Health and Clinical Excellence (NICE) 2014).

ii. *Simultaneity*. Health affects obesity, e.g., a number of underlying health conditions in childhood can result in reduced appetite and weight loss (Rabbett, Elbadri et al. 1996, Picton 1998, Fryar and Ogden 2009). Health care affects obesity, e.g., there are drugs that cause weight gain, for example, psychotropic medicines (National Institute of Mental Health 2008). On the other hand lifestyle interventions, provided by the health service, may reduce weight, and thus previously obese children with health problems might not be obese at the time of measurement when participating in a health survey.

iii. *Omitted variables*. There might be other variables that affect obesity, health and health service use. One example is time preference, which is correlated with BMI (Komlos, Smith et al. 2004, Smith, Bogin et al. 2005, Borghans and Golsteyn 2006) and may influence health service consumption (Coffey 1983, Murphy 1987, Jones 2000). Another example is socioeconomic status, which has a direct effect on the use of medical care after controlling for need (Morris, Sutton et al. 2005) and also affects obesity (Wardle, Waller et al. 2002). Some of the variables that affect health/health service use and obesity are likely to be observed directly in household survey data (e.g., socioeconomic status) and so can be controlled for in empirical studies; others are unlikely to be observed directly (e.g., time preference).

iv. *Measurement error*. Obesity may be measured systematically with bias due to unobserved factors that are correlated with health or health service use.

The aim of this study is to measure the first effect – the impact of obesity on health and health service use – and to produce unbiased estimates that are not contaminated by the other effects.

To measure the health of children we use SAH. This measure is commonly applied in economic research on child health, but also in other fields (Case, Lubotsky et al. 2002, Currie and Stabile 2003, Currie, Shields et al. 2007, Skinner, Mayer et al. 2008). This measure is essentially subjective, however it has been shown to be a strong predictor of future functional mobility, mortality and health service use (Idler and Kasl 1995, Van Doorslaer and Gerdtham

2003, Nielsen 2013). To measure health service use we use measures of doctor utilisation and medication use, as these measures where available in our dataset.

2. Data and variables

Data source

The analysis is based on data from fifteen rounds (1998-2012) of the *Health Survey for England* (HSE)[1] (National Centre for Social Research and Department of Epidemiology and Public Health University College London (UCL) 1997 - 2012); 2012 is the most recent year of data available.

All adults (16+) within the household (up to a maximum of 10) are eligible for interview and up to 2 children (0-15) are eligible. The interviewer had a child selection label to use to randomly select the children to interview in a household with more than 2 children. For children aged 0-12, parents answer on behalf of the child but the child should be present to help with the interview if possible.

Dependent variables

As a measure of health in children previous research (Case, Lubotsky et al. 2002, Currie and Stabile 2003, Currie, Shields et al. 2007) have used self-assessed general health (SAH), which usually results from a question asked to the parents about general health (or to the children themselves in the case of 13-18 years olds). We follow previous research as in each round of the HSE from 1998-2012 the interviewer has asked the question: "*How is your health in general?"* The possible responses are on the following ordinal scale with percentage of children in each response category: Very good (56.2%), Good (37.3%), Fair (5.7%), Bad (0.7%) and Very bad (0.1%). Few children had less than good SAH and we created a binary variable describing whether or not the respondent has very good SAH (1=yes, 0 otherwise).

Five rounds (1998-2002) of the HSE include information on doctor visits. The interviewer asks the question: "*During the two weeks ending yesterday, apart from any visit to a hospital, did you or any other member of the household talk to a doctor on your behalf for any reason*

[1] The HSE is a repeated cross-sectional survey which draws a different sample of nationally representative individuals living in England each year. The sample is selected using a multi-stage stratified probability sampling design with postcode sectors selected at the first stage and household addresses within postcode sectors selected at the second stage. Stratification is based on geographical areas and not on individual characteristics.

at all?" We created a binary variable describing whether or not the respondent had talked to a doctor the last two weeks (1=yes, 0 otherwise). We included visits to private and public doctors, however 98% of the consultations were public. We also have information about the number of visits and less than 2% had more than one visit during the two-week period. Phone calls could count as contact with a doctor, however this accounted for less than 5% of the consultations.

The HSE has information on type of medication use each year between 1998-2012, which is collected by a trained nurse. The nurse asks the question: "*Are you taking or using any medicines, pills, syrups, ointments, puffers or injections prescribed for you by a doctor?"* If yes, then the nurse asks to see the container with the prescribed medicines in order to accurately record the details of the medication usage. The nurse records up to 22 medicines currently being taken for each respondent. We created a binary variable describing whether or not the respondent was taking any medication (1=yes, 0 otherwise).

BMI and obesity measures

BMI is computed from measured height and weight values obtained by a nurse. One useful feature of the HSE is that the BMI values are not based on self-reported height and weight, which reduces the likelihood of measurement error. We measure obesity as a binary variable taking the value one if a child is obese and zero otherwise. The obesity cut-off values are age and gender specific and defined according to WHO guidelines for children aged 5-19 (World Health Organization 2011) and for children aged 2-5 (WHO Multicentre Growth Reference Study Group 2006).

Covariates

We include the following covariates in each regression: age (quadratic function); age of the parent (linear function); marital status of the parent (two categories married/non-married)[2]; education qualifications of the mother (four categories); education qualifications of the father (four categories); ethnicity (two categories white/non-white); Government Office Region (GOR) of residence (nine categories); survey year (fifteen categories); and, smoking status of the parent (current smoker yes/no)[3]. Finally we control for equivalised[4] household income (five income groups) and missing household income[5] (yes/no).

[2] We include the values for the parent that is used to generate the instrument.
[3] This variable will control for passive smoking and is negatively associated with parent's obesity status.

We stratify the analysis by gender and age (3-10 and 11-18).

Instruments

In this study we follow previous studies and use the BMI of a biological relative as an instrument for BMI and obesity (Cawley 2004, Kline and Tobias 2008, Trogdon, Nonnemaker et al. 2008, Lindeboom, Lundborg et al. 2010, Cawley and Meyerhoefer 2012). The genetic instrument for was constructed by matching parents individual level values of BMI in the HSE sample with each child between aged 3-18, who had a valid height and weight measurement. From this we produce a variable for mothers BMI and fathers BMI for each child in the dataset. We use two different combinations of these instruments. First, we include a single instrument based on mothers BMI, however when mothers BMI is missing we use fathers BMI (N=1964). This was done to include the maximum amount of children in the estimation sample. The studies that have looked at the biological mother and father find that the gender of the parent should not have an impact on our results as 50% of the genes should be transmitted from the mother and the father. For example, Sørensen, Holst & Stunkard found that the average correlation of adoptees with biological mothers was 0.17 (95% confidence limits: 0.03, 0.31) and with biological fathers, 0.16 (0.00, 0.32) (Sørensen, Holst et al. 1992). However, other studies have found that the variation in the association between parental BMI and child's BMI vary between mothers and fathers (Lindeboom, Lundborg et al. 2010). To investigate this further we conduct over-identification tests as described below. We also include a control variable in both the first and second stage regressions for whether or not the instrument is based on the father or the mother.

Second, we use multiple instruments. Following Trogdon et al. we rerun our analysis using the following four instruments: mothers obesity status, fathers obesity status, mothers obesity status (missing) and fathers obesity status (missing) (Trogdon, Nonnemaker et al. 2008). In these models all individuals have at least one BMI value for the mother or the father or both.

The first requirement of an instrument is that it is highly correlated with variables being instrumented conditional on the other variables in the model. The weight of a biological relative is a powerful instrument because roughly half of the variation in weight across people

[4] We use McClements equivalence scale to adjust for household size and composition.
[5] Eleven percent of the population has missing income.

is of genetic origin (Comuzzie and Allison 1998). To test this we run and F-test and our instruments exceeds the benchmark power of F=10 in all of our models. Hence, we do not report these values in the following.

The second requirement of an instrument is that it must not be correlated with the error term conditional on the other covariates in the model. There can be, in principle, various situations where the second requirement is not fulfilled. Firstly, the requirement could be violated if both the parent and the child's BMI are affected by common household environments that are also directly correlated with responding children's health/health service use. It is impossible to prove no effect of shared household environments on weight and some doubt will remain. However, a large number of studies have been conducted on the impact of shared household environment on weight, and they do not find any evidence of this (Sørensen, Holst et al. 1992, Vogler, Sørensen et al. 1995, Maes, Neale et al. 1997, Cawley and Meyerhoefer 2012). This has further support by findings from the UK (Wardle, Carnell et al. 2008). For example, adoption studies find that the correlation between children and *biological* parents BMI (not the parents that raise them) is the same for adoptees as natural children (Vogler, Sørensen et al. 1995), which suggest that no variation in weight can be attributed shared household environments. Finally, in our dataset we have a subsample of adopted children. If environmental effects are important, one would also expect to see an association in BMI between adopted children and their adoptee parents (Lindeboom, Lundborg et al. 2010). Hence, we conduct a falsification test to explore whether BMI of unrelated adopted children are associated with their (non-biological) parent weight. We gather a sample of adopted children and their (non-biological) parents (N=446) and regress parents BMI against adopted children's BMI. The coefficients are close to zero, negative and highly insignificant (p=0.955). This provides further support for the validity of our instrument. We also have a sample of children who has a step parents (i.e. they are the child of the partner of the adult) (N=1815). In this sample we control for the BMI and other characteristics of the biological parent and test whether there is a significant correlation between step-parent and children's BMI. As above the coefficients values are small and insignificant.

Another worry is that the genes that affect obesity may affect other behaviours (Norton and Han 2008, Cawley, Han et al. 2011), which may in turn affect health and health service use. For example, the Dopamine Transporter (DAT1 gene) has an impact on obesity, alcoholism and other risky behaviours (Muramatsu and Higuchi 1995, Norton and Han 2008, Guo, Cai et

al. 2010, Cawley, Han et al. 2011). If the genes that cause obesity also have an effect on health and health service use, through other channels than through obesity, it will violate the second requirement for an instrument, and our analysis will be invalid. To explore this we conduct a second falsification test to examine whether parents BMI is correlated with observable behaviour that is believed to have an effect on health service use. This is not a definitive test, however if the observable behaviour is correlated with our instrument it will cast doubt on the instruments validity. In our dataset we have information about the smoking and alcohol consumption among in children over the age of 8. Whether or not the respondents have smoked a cigarette between the age of 8-15 and whether or not the respondents have had a proper alcoholic drink between the age of 8-15. We regress smoking and alcohol on our set of control variables and parents BMI. We found significant and positive associations between parents BMI and probability of having smoked a cigarette ($p=0.03$), and significant and positive associations between parents BMI and the probability of ever had a proper alcoholic drink ($p=0.01$). Hence, it is likely that our instruments are associated with other risky behaviours that have an impact on health and health service use in children.

To explore whether or not children's risky behaviour would alter our findings we rerun the following analysis in children aged 8-18 with and without control variables for children's smoking status (interacted with age) and alcohol consumption (interacted with age). We found that the association between child smoking and SAH was significant and negative, and that the association between child smoking and doctor visits was significant and positive. No significant results were found for alcohol. Importantly, we did not find that it altered our IV coefficients. The impact of BMI and obesity were almost identical in the models with these controls to models without these controls. In the following we run regressions without controlling for children's risky health behaviour[6].

Essentially the validity of our instrument can be tested by an over-identification test, where we test whether or not the instruments are themselves explanatory variables of the outcome. The key to such a test is to have more than one instrument and to know that at least one of these is valid (Murray 2006). We test this by testing the mother and the fathers BMI and

[6] The main reason for this is that children's risky health behaviour is only measured in children aged 8 and above. Hence, we do not have these values for a large part of our sample. In addition, these variables can be considered to be "bad control variables". Such variables are potentially caused by the independent variables of interest (children's BMI). Consequently, including these independent variables in the regression equations affects the causal interpretation of the coefficient estimate on health/health service use (Angrist, J. D. and J.-S. Pischke (2008). Mostly harmless econometrics: An empiricist's companion, Princeton university press.).

obesity status against each other. However, these instruments build on the same rationale, which reduce the implications of the test for determining the validity of the instruments. However, if the instrument picks up something else than genetic variation one could expect that this differed by mothers and fathers. These test do not suggest that obesity picks up anything else other than pure genetics (see appendix).

To summarise, we use two different combinations of the instruments. First, we include a single instrument based on mothers or fathers BMI, second we use multiple instruments. There is reason to suggest that our instrument is correlated with the error term. However, further testing suggests that this does not explain our findings below.

3. Analysis and estimation

We use both multivariate and IV regression methods to estimate the impact of BMI and obesity on health and health service use. We model health and health service use by individual *i* as:

$$Y_i = c_0 + c_1 B_{ij} + X_i \gamma + u_i \quad (1)$$

where Y is a binary measure of either SAH, doctor visits or medication use; B is a measure of BMI or obesity; and X is a vector of individual and maternal/paternal characteristics. u is an error term and c and γ are coefficients to be estimated. Our primary models are univariate probit models[7] for each of the outcomes. Estimations of Eq. 1 will produce unbiased estimates of c provided there are no endogeneity issues in the relationship between our dependent and independent variables.

To account for endogeneity we use two IV regression methods depending on whether we estimate the impact of BMI as a continuous variable or as a binary obesity variable. To estimate the impact of BMI as a continuous variable we use a control function approach applied using maximum likelihood estimation. The first stage is estimated by OLS:

$$B_i = a_0 + a_1 Z_i + X_i \alpha + u_{1i} \quad (2)$$

[7] We perform Hosmer-Lemeshow goodness of fit tests for each specification. We fail to reject this test in each instance indicating that the probit function is appropriate. We have checked the probit model is robust to alternative specifications by running linear probability models for each outcome. The coefficients are very similar to the predicted marginal effects form the probit models.

where B is BMI , a, b, α and β are coefficients to be estimated, and Z are instruments that are correlated with B but not u_1. Based on this model we predict the residuals ($\hat{u}_{1i)}$ which we include as a regressor in the second stage univariate probit model[8]:

$$Y_i^* = b_0 + b_1 B_i + X_i\beta + b_1\hat{u}_{1i} + u_{2i} \quad (3)$$

Given that our instruments are valid; this model no longer has endogenity problems and we test whether or not the BMI variable is endogenous by Wald chi squared tests of exogeneity after each regression. This model makes few distributional assumptions compared with alternatives and is recommended for dealing with endogneity in health economic research (Terza, Basu et al. 2008). However, a drawback with this model is that the regressor of interest (in this case BMI) must be continuous (Rothe 2009).

To investigate the impact of obesity as a binary variable we use a recursive bivariate probit model[9] of the form:

$$B_i = a_0 + a_1 Z_i + X_i\alpha + \mu_{1i}$$
$$Y_i^* = b_0 + b_{1\ i}\hat{B} + X_i\beta + \mu_{2i} \quad (4)$$

where B is an unobserved latent variable, a, b, α and β are coefficients to be estimated, and Z are instruments that are correlated with B but not μ_1. The coefficient of interest is b_1. The IV regression models use Z to isolate exogenous variation in B and thereby estimate the impact of B on Y. The bivariate probit model can be used when both the dependent variable and the endogenous explanatory variable are binary (Woolridge 2002, Jones 2007); this model has been used by others who investigate the impact of adult obesity (Morris 2007).

In the regressions where we use both the BMI of the mother and the father as instruments we test whether or not the instruments are correlated with the error term conditional on the other covariates in our model. This is done by running linear GMM models and performing Hansen's J statistic chi-squared test of overidentifying restrictions (Hansen 1982).

[8] As above we perform Hosmer-Lemeshow goodness of fit tests for each specification. We fail to reject this test in each instance indicating that the probit function is appropriate for the control functions estimates.
[9] We perform Hosmer-Lemeshow goodness of fit tests for each specification in the recursive bivariate probit model. We fail to reject this test in each instance indicating that the specification is appropriate.

We apply two sets of survey weights reported in the HSE to each observation. In the analysis of SAH and doctor use we apply the "individual survey weights", which are generated separately for adults and children[10]. The questions regarding medication use are asked by a nurse, and not all respondents participated in this part of the survey. Hence, in the analysis of medication use we have used the "nurse weights" to take account of non-response to the nurse section of the survey. These weights adjust for the fact that different observations have different probabilities of selection and participation in the survey and participating in the nurse visit. It is also possible that, due to the sampling strategy used in the HSE, observations are independent across Primary Sampling Units (PSUs), but not within PSUs. If this is the case then if we use estimators that assume independence within these clusters the standard errors on our regression coefficients will be too small and we will overestimate the statistical significance of the independent variables in our models. We therefore control for clustered sampling within PSUs using unique PSU/year identifiers that produce Huber/White/sandwich robust variance estimators that allow for within-group dependence (Kish and Frankel 1974).

We compute marginal effects (average treatment effects (ATE)) and predicted mean SAH, doctor visits and medication use for each obesity category, fixing the covariates at their whole sample mean values. The difference in the predicted means between the obese and non-obese is the marginal effect of obesity on SAH, doctor visits and medication use. In all analyses *P* values below the 5% level (*z*-scores higher than ≈1.9) are regarded as statistically significant. Values between 5% and 10% (*z*-scores between ≈1.6 and ≈1.9) are regarded as weakly significant.

4. Results

The total number of respondents in the HSE in 1998-2012 was 222,021. Of these 30,266 were aged 3-18 and had valid height and weight measurement, and 20,432 had a nurse visit with their medication use reported. Doctor utilisation was only reported in 12,182 children between 1998-2002 (Table 1). Table 1 also shows that 10% of the children where obese in each sample, the mean age was 10 years old and close to 50% of the sample were female.

[10] For adults (aged 16 or more)**,** the interview weights are a combination of the household weight and a component which adjusts the sample to reduce bias from individual nonresponse within households. For children (aged 0 to 15), the weights are generated from the household weights and the child selection weights – the selection weights correct for only including a maximum of two children in a household. The combined household and child selection weight were adjusted to ensure that the weighted age/sex distribution matched that of all children in co-operating households.

Table 2 reports the first stage of the instrumental variable regressions of the impact of parents BMI on children BMI and obesity status. The BMI regressions (Eq. 2), which display OLS coefficients, are highly significant and very similar across SAH, doctor utilisation and medication use. The coefficients are slightly higher in girls than boys and the impact of parents BMI are larger in older children (aged 10-18) than in younger children (aged 3-10). Very similar trends are shown for the obesity regressions (Eq. 4), which display probit coefficients for the impact of parents BMI on child obesity[11].

Table 3 shows the results of the univariate probit and IV probit models for the impact of BMI and obesity on SAH. The MEs of BMI and obesity on SAH are significant and negative in boys, girls, younger children and older children in the univariate probit models. The MEs are larger in older than in younger children. The MEs in the IV models, of the impact of BMI and obesity on SAH, are larger, compared with the probit model results. The z scores also illustrate that they are significant in boys, girls, younger children and older children. The endogeneity tests show that there is significant endogeneity in each model. Hence, the probit models underestimate the impact of BMI and obesity on SAH. For example, the impact of obesity on the probability of reporting very good SAH across all individuals is -0.1 in the probit models. The same effect is -0.3 in the IV probit models.

Table 4 shows the results of the univariate probit and IV probit models for the impact of BMI and obesity on doctor utilisation. The MEs of BMI on doctor utilisation are positive but not significant in any of the univariate probit models. The ME of obesity is weakly significant and positive in the total population and significant and positive in boys. As above, the MEs in the IV models, of the impact of BMI and obesity on doctor utilisation, are larger, compared with the probit model results. The impacts of BMI and obesity on doctor utilisation in the IV probit models are significant and positive in the total population and the endogeneity tests are weakly significant (for BMI), which indicates that the univariate probit models underestimate the impact of BMI and obesity on doctor utilisation. We also observe that the impact of BMI in girls is weakly significant in the IV probit models and the endogeneity test is weakly significant.

[11] The probit coefficients can not be interpreted as marginal effects and cannot be directly compared with the OLS coefficients. Also the two sets of results in Table 2 are not comparable because one is modelling the impact of the IV on BMI, the other on obesity.

Table 5 shows the results of the univariate probit and IV probit models for the impact of BMI and obesity on medication use. The MEs of BMI on medication use are significant and positive in the total population, girls, younger children and older children (weakly significant) in the univariate probit models. The MEs of obesity on medication use are significant and positive across each of the subsamples and in the total sample in the univariate probit models. The MEs in the IV models, of the impact of BMI and obesity on medication use, are larger, compared with the probit model results. The z scores also illustrate that they are significant and positive in the total sample and in each subsample. The endogeneity tests show that there are significant endogeneity in each model. Hence, the probit models underestimate the impact of BMI and obesity on medication use. For example, the impact of obesity on the probability of medication use across all individuals is 0.04 in the probit models. The same effect is 0.18 in the IV probit models. We also observe that the MEs in the IV probit models are larger in older than in younger children.

The predicted mean share of the population reporting very good SAH, doctor utilisation and medication use by obesity category is in Table 6. The predicted probabilities are based on the IV probit models. The share of the population who are non-obese and report very good SAH is 60%, 10% report doctor utilisation and 17% report medication use. Analogous numbers in the obese are 30%, 19% and 35%. Comparable numbers are observed in each subsample. Hence, obesity reduces health and increase health service use in children.

Appendix table 1 display the models where we use four instruments and the results are very similar to the results in the main IV models. We can see that we fail to reject the over-identification tests for each regression, which supports the validity of our instrument. We have also conducted additional over-identification tests using only mother and father obesity status (results not shown). And they all support the validity of our instrument.

5. Discussion

The aim of this study was to estimate the effect of obesity on SAH, doctor utilisation and medication use in children and adolescents. Our main finding is that obesity has a negative impact on SAH and positive impact doctor utilisation and medication use. In the IV models, which use genetic variation in BMI as a natural experiment, the marginal effect of obesity on

SAH, doctor utilisation and medication use are significantly higher than the effect found in models that do not account for endogeneity by instrumental variables.

We found a negative impact of obesity on the probability of reporting very good SAH in the total population. The negative impacts were also significant in girls, boys, younger children (aged 3-10) and older children (aged 11-18). It is unsurprising that obesity has a negative impact on SAH as it has been shown to increase the risk of a number of diseases including diabetes (high fasting plasma glucose levels), heptic steatosis, sleep apnea, orthopaedic conditions, and hypertension (Rashid and Roberts 2000, Poussa, Schlenzka et al. 2003, Wing, Hui et al. 2003, Skinner, Mayer et al. 2008, Juonala, Magnussen et al. 2011). In addition, obesity in itself might reduce general health perception, self-esteem and health-related quality of life (Tsiros, Olds et al. 2009, Griffiths, Parsons et al. 2010).

Our results are consistent with earlier studies, which find an association between obesity and various health measures including SAH children (Skinner, Mayer et al. 2008, Tsiros, Olds et al. 2009, Griffiths, Parsons et al. 2010). However, to the best of our knowledge this is the first study to use instrumental variables and by this illustrating that earlier literature may have underestimated the impact of obesity on health.

We found significant and positive impacts of obesity on the probability of doctor and medication utilisation in the total population. The positive impacts of obesity on medication use were also significant in girls, boys, younger children (aged 3-10) and older children (aged 11-18). The impacts of obesity on doctor utilisation were weakly significant in girls and younger children (aged 3-10). It is difficult to compare our results to earlier studies as they report odds-ratios or total expenditures. However, we note that they have found a mix of significant and insignificant associations between obesity and health service use. This is similar to our non-IV results. We found, for both doctor and medication utilisation, that our endogeneity tests were significant (weakly for doctor utilisation). This suggests that earlier studies have underestimated the impact of obesity on health service use in childhood.

Our findings suggest that future research on childhood obesity should use IV models because the non-IV models may underestimate the impact of obesity on health and health service use where there is a causal impact. These findings broadly mirror findings by Cawley & Meyerhoefer on the impact of obesity on health service use in adults. Cawley & Meyerhoefer

(2012) suggest two reasons why accounting for endogeneity may lead to larger marginal effects of obesity. First, they suggest that reporting error with respect to height and weight may cause attenuation bias. Second, non-IV models may suffer from omitted variable bias. We suspect that the last reason may be the primary explanation for our findings, as a child who suffer from various illnesses also have reduced appetite and weight loss as a result of these illnesses (Rabbett, Elbadri et al. 1996, Picton 1998, Fryar and Ogden 2009). For example, severe weight loss following cancer is a major cause of morbidity in children (Picton 1998). Simultaneity may also be an explanation as health care may have an impact on obesity. For example, in England guidelines produced by the National Institute for Health and Clinical Excellence (NICE) state that a number of lifestyle and behavioural interventions aimed at weight reduction should be considered for treatment in overweight and obese children (National Institute for Health and Clinical Excellence (NICE) 2014).

Our estimates have consequences for health service planning as they demonstrate that obesity in childhood has an impact on health and that it affects resource use. The results can be used in cost-effectiveness analysis and confirms the impact of obesity on conditions which are managed both in primary care and pharmacologically, with major cost implications. Our findings that the causal impacts of obesity on health and health service use are greater than in the non-IV models have direct implications. For example, many costs and cost-effectiveness analyses of obesity interventions are based on associations which might have underestimated the causal effect, see John et al. for a review of the literature. As a result, the cost-effectiveness of these interventions has most likely been underestimated.

Our study has several potential limitations. First, our measure of obesity is BMI, which has been criticised, e.g., because it does not incorporate body fat, which is an independent predictor of ill health (Burkhauser and Cawley 2008). Second, we include BMI as a linear continuous variable, when there is evidence of a non-linear association between BMI and health. Similarly, we compare obese to the non-obese whereas other papers include categories for underweight and overweight (we do not include indicators for these categories as we lack additional instruments for this number of categories) (Kinge and Morris 2010). Third, we use binary outcome variables. However, we do not measure the quantity of doctor utilisation and medications taken, which would have given a more comprehensive results in terms of resource use. Fourth, there might be limitations regarding the validity of the instruments.

Although our instruments pass the standard statistical tests for power and over-identification, there is no way to prove beyond doubt the validity of an instrument.

To conclude, this study makes a contribution to the literature by providing estimates of the impact of obesity on health and health service use in children and adolescents accounting for endogeneity bias by instrumental variables. The estimates of the effects of obesity on health and health service use are significantly higher, in the IV models, than the estimates of the effects in non-IV models. Hence, the impact of obesity on health and health service use may be underestimated if no account is made of bias due to endogeneity.

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Table 1: Summary statistics based on Health Survey for England 1998-2012

	SAH sample	Doctor visit sample	Medication use sample
Total (N)	30266	12182	20435
Male (N)	15264	6188	10240
Female (N)	15002	5994	10195
Aged 3-10 (N)	15621	6486	10503
Aged 11-18 (N)	14645	5696	9932
Obese (%)	10.46	9.80	10.32
Age (mean)	10.30	10.15	10.31
Survey year (%)			
1998	11.77	29.24	15.33
1999	5.88	14.62	0.94
2000	5.9	14.66	1.08
2001	10.6	26.34	13.09
2002	6.09	15.14	7.64
2003	10.31	0	12.1
2004	4.33	0	0.76
2005	4.91	0	5.53
2006	9.34	0	10.76
2007	4.55	0	5.15
2008	9.19	0	10.21
2009	2.92	0	3.26
2010	4.98	0	5.1
2011	4.63	0	4.7
2012	4.59	0	4.34

Table 2: the impact of parent BMI on children's BMI/obesity from the first stage regression results

	BMI (Eq. 2) *OLS*		**Obesity (Eq. 4)** *Probit*	
	Coeff.	*z*	*Coeff.*	*z*
Very good SAH				
Total	0.1721	34.46	0.0574	30.43
Male	0.1636	24.54	0.0576	22.86
Female	0.1805	25.81	0.0574	21.72
Aged 3-10	0.1151	21.96	0.0518	19.67
Aged 11-18	0.2263	28.29	0.0641	23.55
Doctor visits				
Total	0.1614	23.73	0.0600	20.23
Male	0.1517	17.96	0.0600	15.18
Female	0.1709	17.2	0.0606	14.55
Aged 3-10	0.1186	15.99	0.0564	14.07
Aged 11-18	0.2100	19.18	0.0659	15.48
Medication use				
Total	0.1782	25.76	0.0595	24.5
Male	0.1703	18.41	0.0613	18.69
Female	0.1868	20.55	0.0578	17.24
Aged 3-10	0.1139	18.74	0.0515	16.09
Aged 11-18	0.2332	21.19	0.0684	19.29

Each regression includes the following covariates: age; age of the responding parent; marital status of the responding parent; education qualifications of the mother; education qualifications of the father; equivalised household income; ethnicity; region; and, survey year.

Table 3: The impact of BMI and obesity on very good self-assessed health

	Probit regressions (Eq. 1)				**IV Probit (Eq. 3)**			**IV Probit (Eq. 4)**		
	BMI		Obesity		BMI			Obesity		
	M.E.	*z*	*M.E.*	*z*	*M.E.*	*z*	*Endog. test*	*M.E.*	*z*	*Endog. test*
Total	-0.0094	-9.91	-0.0942	-9.35	-0.0277	-8.01	p<0.001	-0.2976	-7.32	p<0.001
Male	-0.0085	-6.28	-0.0960	-6.95	-0.0276	-5.81	p<0.001	-0.2412	-4.26	p=0.001
Female	-0.0095	-7.40	-0.0874	-6.14	-0.0275	-6.28	p<0.001	-0.3292	-6.49	p<0.001
Aged 3-10	-0.0048	-2.76	-0.0525	-3.81	-0.0343	-5.01	p<0.001	-0.2594	-4.54	p<0.001
Aged 11-18	-0.0113	-9.98	-0.1431	-9.59	-0.0253	-7.01	p<0.001	-0.3308	-6.72	p<0.001

All F-test of the instruments are above the value of F=10. Each regression includes the following covariates: age; age of the responding parent; marital status of the responding parent; education qualifications of the mother; education qualifications of the father; ethnicity; region; and, survey year.

Table 4: The impact of BMI and obesity on doctor utilisation

	Probit regressions (Eq. 1)				**IV Probit (Eq. 3)**			**IV Probit (Eq. 4)**		
	BMI		Obesity		BMI			Obesity		
	M.E.	*z*	*M.E.*	*z*	*M.E.*	*z*	*Endog. test*	*M.E.*	*z*	*Endog. test*
Total	0.0011	1.24	0.0154	1.65	0.0070	1.99	p=0.089	0.0951	1.98	p=0.103
Male	0.0017	1.43	0.0323	2.65	0.0045	0.87	p=0.575	0.0817	1.15	p=0.533
Female	0.0002	0.13	-0.0032	-0.23	0.0090	1.94	p=0.053	0.1061	1.58	p=0.095
Aged 3-10	0.0004	0.25	0.0130	1.03	0.0093	1.4	p=0.161	0.1396	1.8	p=0.104
Aged 11-18	0.0011	1.08	0.0201	1.48	0.0060	1.67	p=0.166	0.0596	1.08	p=0.494

All F-test of the instruments are above the value of F=10. Each regression includes the following covariates: age; age of the responding parent; marital status of the responding parent; education qualifications of the mother; education qualifications of the father; equivalised household income; ethnicity; region; and, survey year.

Table 5: The impact of BMI and obesity on medication use

	Probit regressions (Eq. 1)				**IV Probit (Eq. 3)**			**IV Probit (Eq. 4)**		
	BMI		Obesity		BMI			Obesity		
	M.E.	*z*	*M.E.*	*z*	*M.E.*	*z*	*Endog. test*	*M.E.*	*z*	*Endog. test*
Total	0.0027	2.87	0.0364	3.75	0.0138	4.5	p<0.001	0.1811	4.16	p=0.001
Male	0.0019	1.39	0.0269	2.02	0.0146	3.26	p=0.002	0.1366	2.38	p=0.055
Female	0.0033	2.63	0.0464	3.31	0.0127	3.07	p=0.020	0.2391	3.36	p=0.009
Aged 3-10	0.0045	2.74	0.0371	3.07	0.0222	3.63	p=0.003	0.2554	3.75	p=0.003
Aged 11-18	0.0020	1.74	0.0375	2.56	0.0107	3.12	p=0.008	0.1467	2.58	p=0.055

All F-test of the instruments are above the value of F=10. Each regression includes the following covariates: age; age of the responding parent; marital status of the responding parent; education qualifications of the mother; education qualifications of the father; equivalised household income; ethnicity; region; and, survey year.

Table 6: Predicted mean of each dependent variable based on the IV regression models

	Reporting very good SAH		Doctor utilisation		Medication use	
	Non-obese	*Obese*	*Non-obese*	*Obese*	*Non-obese*	*Obese*
Total	0.60	0.30	0.10	0.19	0.17	0.35
Male	0.60	0.36	0.09	0.18	0.17	0.30
Female	0.59	0.26	0.11	0.21	0.17	0.41
Aged 3-10	0.64	0.38	0.10	0.24	0.15	0.40
Aged 11-18	0.55	0.22	0.09	0.15	0.18	0.33

The figures reports predicted mean of the population reporting very good SAH; who have had a doctor visit; who use medication across BMI categories in each model setting the covariates to their sample mean values. The covariates are: age; age of the responding parent; marital status of the responding parent; education qualifications of the mother; education qualifications of the father; equivalised household income; ethnicity; region; and, survey year.

Appendix table 1: The impact of BMI and obesity on SAH, doctor utilisation and medication use, using four instruments (mother obese, father obese, mother obese (missing), father obese (missing))

	IV Probit (Eq. 3)				**IV Probit (Eq. 3)**			
	BMI				Obesity			
	M.E.	z	*Overid. test*	*Endog. test*	M.E.	z	*Overid. test*	*Endog. test*
Reporting very good SAH								
Total	-0.0273	-6.98	*p=0.447*	*p<0.001*	-0.2788	-5.97	*p=0.639*	*p<0.001*
Male	-0.0264	-4.87	*p=0.669*	*p<0.001*	-0.2126	-3.16	*p=0.759*	*p=0.080*
Female	-0.0278	-5.68	*p=0.297*	*p<0.001*	-0.3190	-5.73	*p=0.489*	*p<0.001*
Aged 3-10	-0.0392	-4.97	*p=0.647*	*p<0.001*	-0.2574	-4.11	*p=0.736*	*p=0.001*
Aged 11-18	-0.0218	-5.25	*p=0.158*	*p=0.007*	-0.2985	-5.00	*p=0.247*	*p=0.007*
Doctor utilisation								
Total	0.0074	1.89	*p=0.720*	*p=0.103*	0.0833	1.63	*p=0.789*	*p=0.196*
Male	0.0065	1.09	*p=0.816*	*p=0.417*	0.0706	0.95	*p=0.831*	*p=0.655*
Female	0.0078	1.62	*p=0.832*	*p=0.104*	0.1342	1.67	*p=0.853*	*p=0.082*
Aged 3-10	0.0161	2.02	*p=0.501*	*p=0.047*	0.2055	2.44	*p=0.542*	*p=0.027*
Aged 11-18	0.0018	0.47	*p=0.340*	*p=0.854*	-0.0129	-0.28	*p=0.357*	*p=0.492*
Medication use								
Total	0.0120	3.41	*p=0.415*	*p=0.007*	0.1784	3.42	*p=0.503*	*p=0.008*
Male	0.0119	2.35	*p=0.655*	*p=0.043*	0.1461	2.11	*p=0.684*	*p=0.089*
Female	0.0120	2.54	*p=0.704*	*p=0.064*	0.2076	2.61	*p=0.738*	*p=0.054*
Aged 3-10	0.0261	3.56	*p=0.270*	*p=0.004*	0.3690	4.69	*p=0.366*	*P<0.001*
Aged 11-18	0.0065	1.68	*p=0.899*	*p=0.233*	0.0721	1.22	*p=0.915*	*p=0.579*

Each regression includes the following covariates: age; age of the responding parent; marital status of the responding parent; education qualifications of the mother; education qualifications of the father; equivalised household income; ethnicity; region; and, survey year.

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