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Title: The impact of childhood obesity on health and health service use

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Abstract:

Objective: To test the impact of obesity on health and healthcare use in children, by the use of various methods to account for reverse causality and omitted variables.

Data sources/study setting: Fifteen rounds of the *Health Survey for England* (1998-2013), which is representative of children and adolescents in England.

Study design: We use three methods to account for reverse causality and omitted variables in the relationship between BMI and health/health service use: regression with individual, parent and household control variables; sibling fixed effects; and, instrumental variables based on genetic variation in weight.

Data collection/extraction methods: We include all children and adolescents aged 4-18 years old.

Principal findings: 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 4-12) and adolescents (aged 13-18). The findings are comparable in each model in both boys and girls.

Conclusions: By using econometric methods we have mitigated several confounding factors affecting the impact of obesity in childhood on health and health service use. Our findings suggest that obesity has severe consequences for health and health service use even among children.

Introduction

Many studies have found that adult obesity accounts for a substantial proportion of health care costs (Bierl et al., 2013). However, an important question is whether obesity in childhood has consequences for health and health care expenditures when the children are still young. Several studies shows significant negative associations between youth obesity and various measures of health (Tsiros et al., 2009). These studies consistently report that, compared with normal weight, obese youths have lower health-related quality of life, lower self-assessed general health (SAH), lower self-esteem and more chronic conditions (Tsiros et al., 2009, Skinner et al., 2008, Griffiths et al., 2010). However, evidence of the impact of childhood obesity on costs while the children are still young is ambiguous (John et al., 2010, John et al., 2012).

Although obesity in adults has been shown to translate into substantial health care utilization and expenditures (Bierl et al., 2013, Cawley and Meyerhoefer, 2012), the same cannot be said for children and adolescents. Findings based on US data are mixed about the impact of childhood obesity on health service use. Two studies found excess overall health care costs in obese children compared with overweight adolescents aged 12-18 (Buescher et al., 2008) and in children/adolescents aged 8-19 (Finkelstein and Trogdon, 2008). Monheit et al. (2009) used data on adolescents aged 12-19 and found significant positive associations between overweight and health care expenditures in girls, but not boys. Significant associations have also been found between child obesity and outpatient visits, prescription drug use and emergency room visits in children aged 6-19 (Trasande and Chatterjee, 2009). Conversely Johnson et al. (2006) looked at children aged 4-17 and did not find significantly increased health care utilization associated with obesity. Skinner et al. (2008) found a negative association between obesity and SAH in children aged 6-17, but did not find any significant

relationship between obesity and expenditures for these children. Evidence from other countries like Canada also suggests no differences in physician costs between normal-weight and overweight/obese young people aged 12-17 years (Janssen et al., 2009). Conversely, evidence from Australia suggests that children who were overweight or obese at age 4 to 5 had higher pharmaceutical and non-hospital medical care costs during the following 5 years compared with normal-weight children (Au, 2012).

The above studies measured the association between BMI and health and health service use. These are less useful for establishing causality, as there might be four reasons why obesity, health, and health service 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 (Juonala et al., 2011, Skinner et al., 2008, Rashid and Roberts, 2000, Wing et al., 2003, Poussa et al., 2003). The impaired health status increases health service use as the obese are more likely to utilize health services to manage these conditions. In addition, obesity may directly increase health care use as obese children might be prescribed lifestyle interventions and appetite-suppressing drugs for weight reduction(National Institute for Health and Clinical Excellence (NICE), 2014).

ii. *Simultaneity*. Health and health care affect obesity, e.g., a number of underlying health conditions in childhood can result in reduced appetite and weight loss (Rabbett et al., 1996, Fryar and Ogden, 2009, Picton, 1998, Soliman et al., 2009, Aukett et al., 1986). Some interventions, e.g., lifestyle interventions, may reduce

weight (Young et al., 2007). Conversely, common childhood diseases like asthma might lead to reduced physical activity and consequently higher BMI. There are drugs that cause weight gain, for example, psychotropic medicines (National Institute of Mental Health, 2008).

iii. *Omitted variables*. There might be other variables that affect obesity, health and health service use. For example, parents are likely to play a large role in deciding the health care use of their children, thus parental characteristics like time preference, which is correlated with BMI (Komlos et al., 2004, Borghans and Golsteyn, 2006, Smith et al., 2005), may influence health service consumption (Jones, 2000, Murphy, 1987, Coffey, 1983).

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. To achieve this we apply three strategies, where each method has strengths and weaknesses, and compare the results. First, we account for omitted variables in regressions by including a number of covariates. An issue with this method is that there are a number of unobserved variables that might affect both health and BMI, which we may not be able to control for in regressions. Second, we use sibling-fixed effects (FE) to account for the influence of all factors that are shared between siblings. Although this strategy might account for omitted variables, it does not eliminate reverse causality. Third, we use an instrumental variables framework that has the potential to remove both reverse causality and omitted variables. However, this depends on the validity of the instrument, which is discussed in detail below.

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 (Currie and Stabile, 2003, Currie et al., 2007, Case et al., 2002, Skinner et al., 2008). SAH is essentially subjective, however it has been shown to be a strong predictor of future functional mobility, mortality and health service use (Van Doorslaer and Gerdtham, 2003, Idler and Kasl, 1995, Nielsen, 2013). To measure health service use we use measures of doctor utilization and medication use, as these services are likely to account for the majority of contacts in children (Buescher et al., 2008).

Data and variables

Data source

The analysis is based on data from fifteen rounds (1998-2013) of the *Health Survey for England* (HSE) (National Centre for Social Research and Department of Epidemiology and Public Health University College London (UCL), 1997 - 2013). The HSE is a repeated cross-sectional survey, which draws a different sample of nationally representative individuals living in England each year.

All adults (16+) within the household (up to a maximum of 10) are eligible for interview, plus up to 2 children (0-15). The interviewer randomly selects the children to interview in a household with more than 2 children. For children aged 0-12, parents answer on behalf of the child and the child is present.

Dependent variables

In each round of the HSE the interviewer asked parents about the health of their children (or asks the adolescents themselves in the case of 13-18 years olds): "*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%). We follow Currie & Stabile (2003) and define poor health in children as the bottom three categories of the SAH variable (good health; 1=yes, 0 otherwise).

This study used the WHO recommended version of SAH (from 'very good' to 'very bad'), which differs from the US version ('excellent' to 'poor'). Reinhold and Jürges (2012), who also use SAH to represent child health, suggests that although health levels are not directly comparable across the two versions, both are different categorizations of the same latent continuous variable (Reinhold and Jürges, 2012, Jurges et al., 2008). One might want to model the SAH using the full scale. Hence, we follow Case et al. (2002) and Currie et al. (2007) re-estimate the analyses using four categories of SAH and an ordered probit model instead of the probit model.

Five rounds (1998-2002) of the HSE included 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 at all?*" We created a binary variable describing whether 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. Contacts could be either face to face or phone calls and fewer than 5% were phone calls.

The HSE has information on type of prescribed medication use each year between 1998-2013, 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. We created a binary variable describing whether the respondent was taking any prescribed medication (1=yes, 0 otherwise).

BMI and obesity measures

BMI is computed from measured height and weight values measured by the interviewer. 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 based on BMI and are age- and gender-specific, defined according to WHO guidelines for children aged 5-19 years (World Health Organization, 2011) and 2-5 years (WHO Multicentre Growth Reference Study Group, 2006). In the following analysis, we use both BMI (continuous) and obesity (binary) as independent variables of interest.

Covariates

We include the following covariates in each regression: age (quadratic function); highest education of the mother (four categories) and father (four categories)[1]; ethnicity (white/non-white); Government Office Region (GOR) of residence (nine categories); survey year (sixteen categories); and, equivalised (McClements equivalence scale) household income (five income groups; the sample is equally split into quintiles each year). Eleven percent of our sample has missing household income; hence, we include a separate category with these individuals: missing household income. In addition, we include the following covariates of the parents: age of the parents (linear function); if the parents are married (binary); if the parents are current smokers (binary); SAH of the mother (binary) and the father (binary); and, whether or not the mother (binary) or the father (binary) has any longstanding illnesses.

In our dataset we have information about smoking and alcohol consumption among children over the age of eight years. In the regressions we control for a three category variable for smoking (0=never smoked a cigarette, 1=have smoked a cigarette, 2=smoking information missing) and a three category variable for alcohol (0=have never had a proper alcoholic drink, 1=have had a proper alcoholic drink, 2=alcohol information missing). By this we hope to capture children's risk related behavior, which might be related to BMI. A discussion of these covariates can be found in the discussion of the validity of the instruments below.

We stratify the analysis by gender and age (4-12 and 13-18 years), as parents answer on behalf of children below the age of 13.

Instruments

We follow previous studies and use the BMI of a biological relative as an instrument for BMI and obesity (Cawley, 2004, Cawley and Meyerhoefer, 2012, Lindeboom et al., 2010, Kline and Tobias, 2008, Trogdon et al., 2008). The instrument was constructed by matching parents BMI with each child with a valid height and weight measurement. From this we produce a variable for mothers BMI and/or fathers BMI for each child in the dataset. We include a single instrument based mainly on mothers BMI (N=27 928) as a continuous variable, except when mothers BMI is missing we use fathers BMI (N=2 030) as a continuous variable. 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. We also include a control variable for whether or not the instrument is based on the father or the mother.

We obtained similar results using different combinations of the mother and the fathers BMI separately. However, we prefer our instrument as it allows us to include those with missing values for either the mother or the father.

Validity of the instrument

An instrument must be highly correlated with variables being instrumented conditional on the other variables in the model. The BMI of a biological relative is a powerful instrument because roughly half of the variation in weight across people is of genetic origin (Comuzzie and Allison, 1998). To test this we run F-tests. Our instruments exceeds the benchmark value of F=10 (Staiger and Stock, 1997) by more than a tenfold in all of our models. Hence, we do not report these values in the following.

An instrument must also not be correlated with the error term in the outcome equation conditional on the other covariates in the model. This will not be fulfilled if both the parent and the child's BMI are affected by common household environments that are also directly correlated with the child's health/health service use. It is difficult to prove no such effect, however a large number of studies do not find any evidence of this (Cawley and Meyerhoefer, 2012, Sørensen et al., 1992, Vogler et al., 1995, Maes et al., 1997). This has further support by findings from the UK (Wardle et al., 2008). For example, adoption studies find that the correlation between child and *biological* parents BMI is the same for adoptees as natural children (Vogler et al., 1995), which suggests that variation in weight cannot be attributed to shared household environments.

In our dataset we have a subsample of adopted children. If environmental effects are important, we would expect to see an association between the BMI of adopted children and

their adoptive parents (Lindeboom et al., 2010). Hence, we conduct a falsification test to explore whether BMI of unrelated adopted children are associated with their non-biological parent BMI. We gather a sample of adopted children and their non-biological parents (N=364) and regress adopted children's BMI against their adopted parent's BMI, controlling for age (quadratic function), gender, adopted parents education (7 categories), adopted parents age and adopted parents marital status. The coefficient of the adopted parent's BMI are close to zero, negative (we would expect positive coefficient if there were shared household effects) and insignificant (p=0.427). This provides further support for the validity of our instrument. We also have a sample of children who have step-parents (i.e. they are the child of the partner of the adult) (N=1876). 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 concern is that genes that affect obesity may affect other behaviors (Norton and Han, 2008, Cawley 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 behaviors (Norton and Han, 2008, Cawley et al., 2011, Muramatsu and Higuchi, 1995, Guo et al., 2010). If 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. To explore this we conduct a second falsification test to examine whether parental BMI is correlated with observable behavior that is believed to have an effect on health service use. This is not a definitive test, but if the observable behavior is correlated with our instrument it will cast doubt on the instrument validity. In our dataset we have information about smoking and alcohol consumption among children over the age of eight years, which we regress on our set of control variables and parent BMI. We found significant

and positive associations between parent BMI and the 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 behaviors. As a result of this we control for these variables in our regressions. Although, these variables might not capture all risky health behavior in children they might serve as a proxy for other risky health behaviors.

Nevertheless, we cannot rule out the possibility that parental BMI picks up unobserved family characteristics and preferences that also affects children's health outcomes. Hence, we additionally supplement our analysis with sibling-FE models, where essentially all unobserved and observed factors that relate to health, health service use and BMI operating at the sibling level are neutralized.

Analysis and estimation

We model health and health service use for individual *i* as:

$$Y_i = c_0 + c_1 B_i + X_i \gamma + u_i \quad (1)$$

where Y is a 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 probit models for each of the outcomes. Hosmer-Lemeshow goodness of fit tests indicates that the probit function is appropriate. We also use ordered probit models in some of our regressions. Estimations of Eq. (1) will produce unbiased estimates of c provided there are no endogeneity issues.

Although obesity might affect health and health service use, we cannot rule out omitted variables bias. Both early life conditions and parental background can jointly affect BMI and health. Hence, we apply the following family-fixed effects specification:

$$Y_{ij} = c_0 + c_1 B_{ij} + X_{ij}\,\gamma + \varepsilon_j + u_{ij} \qquad (2)$$

where ij denotes individual i in family j, and ε represents a family fixed effect. We include only siblings in each family, and X is a vector of control variables that are not shared between siblings: a dummy variable for being the oldest sibling, as birth order has a known effect on health (Donovan and Susser, 2011); maternal age at delivery (as a categorical variable) as both high and low maternal age is associated with reduced health in the child (Fall et al., 2015); and, the child's age as a quadratic function. We use Chamberlains conditional logit model for each outcome with sibling-fixed effects (Chamberlain, 1980).

All unobserved and observed factors that relate to health, health service use and BMI operating at the sibling level will be neutralized in this model. In addition, as siblings share roughly 50% of their genes, a part of the genetic influence might be neutralised. As measurement error is intensified in sibling-FE models, it is beneficial that our height and weight are measured and not self-reported.

One important drawback of the sibling-FE model is that it cannot account for reverse causality. Hence, we also use two IV regression methods based on a two-stage residual inclusion (2SRI) estimator, which is a consistent nonlinear extension of the traditional IV method (Terza et al., 2008a, Wooldridge, 2015). The first stage is estimated by an exponential

regression model where we include BMI as a continuous variable and by a probit model where we include BMI as a binary obesity variable:

$$B_i = a_0 + a_1 Z_i + X_i\alpha + u_{1i} \quad (3)$$

where B is BMI/obesity, and Z are instruments that are correlated with B but not u_1. Based on this model we predict the response residuals ($\hat{u}_{1i}$) which we include as a regressor in the second stage (ordered) probit model:

$$Y_i = b_0 + b_1 B_i + X_i\beta + b_2\hat{u}_{1i} + u_{2i} \quad (4)$$

Assuming that our instrument is valid; this model no longer has endogeneity problems and we test this using Wald tests of exogeneity ($b_2=0$) after each regression. We used 500 bootstrap samples to calculate the standard errors for the coefficients.

Given that B is a binary variable, we present marginal effects (MEs) of being obese on the probability of poor health/health service use. This is the sample average of changes in the marginal predicted probability of being in poor health/using health services with discrete changes in B keeping all other variables X at their observed values. When B is a continuous variable (BMI) the ME reflects the instantaneous rate of change[2].

In the 2SRI models the MEs are calculated as for the probit models, however Imbens and Angrist (1994) show that in a linear model the b_1 from (4) is the local average treatment effect (LATE). In the case of 2SRI it has been argued that the MEs can be interpreted as average treatment effects (ATE) (Terza et al., 2008a, Terza et al., 2008b).

The conditional logit model does not estimate the fixed effects, hence the MEs cannot be consistently estimated (Kitazawa, 2012, Silva and Kemp, 2016). We provide an estimate of the MEs assuming that the fixed effects are zero. We also provide the average semi elasticity, which can be consistently estimated following (Kitazawa, 2012, Silva and Kemp, 2016).

We apply survey weights reported in the HSE to each observation. In the analysis of SAH and doctor use we apply individual survey weights, which are generated separately for adults and children. 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 nurse visit weights to take account of non-response to the nurse section of the survey. Both sets of weights adjust for the fact that different observations have different probabilities of selection and participation in the survey and 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. 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).

Results

The total number of respondents in the HSE in 1998-2013 was 233,001. Of these, 29,958 were aged 4-18 and had valid height and weight measurement, and of these 20,294 had a nurse visit where their medication use was reported. Doctor utilization was reported in 11,445

children in the five rounds of the HSE from 1998-2002. The sibling samples have comparable age, though the obesity prevalence is about 0.5 percentage points lower (Table 1).

The MEs of BMI and obesity on SAH are significant and negative in boys, girls, younger children and older children in the probit models, 2SRI-probit models and in the sibling-FE models (Table 2). The MEs in the 2SRI-probit models, of the impact of BMI and obesity on SAH, are comparable with the sibling-FE results. The MEs in the 2SRI-probit models are significantly larger than for the probit models in each group for both BMI and obesity. The results of the ordered probit models (appendix) supports the probit models. The predicted probabilities in the ordered probit models show that obesity in children reduce the probability of very good health and increase the probability of bad health.

The MEs of BMI on doctor utilization are not significant in any of the probit models (Table 3). The ME of obesity is weakly significant and positive in the total sample and significant and positive in boys and younger children. As above, the MEs in the IV models, of the impact of BMI and obesity on doctor utilization, are larger, compared with the probit model results. The impacts of BMI and obesity on doctor utilization in the 2SRI-probit models are weakly significant and positive in the total sample. We also observe that the impact of BMI and obesity on doctor utilization in younger children is significant in the 2SRI-probit models. In sibling-FE models the impact of obesity on doctor utilization is significant for the full population, boys and girls and the MEs are comparable in size to the 2SRI-probit MEs.

The MEs of BMI and obesity on medication use are significant and positive in the total sample, girls, younger children and older children in the probit and the 2SRI-probit models (Table 4). The sibling-FE models show significant impacts of BMI and obesity on medication

use in the full sample. The MEs in the 2SRI models, of the impact of BMI and obesity on medication use, are larger, compared with the probit model results.

The predicted percentage of boys who are not obese who report good SAH was higher in each specification, compared with obese boys (Table 5). Conversely, obese boys have more doctor visits and use more medication than boys who are not obese. Similar findings are obtained for girls. Hence, obesity reduces health and increase health service use in children.

Discussion

Our main finding was that obesity in childhood has a negative impact on SAH and positive impact doctor utilization and medication use. The findings were comparable in each model. In the IV models, which used genetic variation in BMI to remove endogeneity, the marginal effect of obesity on SAH, doctor utilization and medication use were higher than the effect found in the probit models.

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 (Juonala et al., 2011, Skinner et al., 2008, Rashid and Roberts, 2000, Wing et al., 2003, Poussa et al., 2003). In addition, obesity in itself might reduce general health perception, self-esteem and health-related quality of life (Griffiths et al., 2010, Tsiros et al., 2009). However, to the best of our knowledge this is the first study to use alternative methods like sibling-FE and instrumental variables; and use these to suggest a causal impact of obesity on health in children.

Earlier studies report odds-ratios or total expenditures, which precludes direct comparison with our study. 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, where e.g. the association between obesity and doctor visits was significant in boys but not in girls. However, our sibling-FE specification found that this association was significant in both boys and girls.

Our IV-results broadly mirror findings by Cawley & Meyerhoefer on the impact of obesity on health service use in adults. Cawley & Meyerhoefer (2012) suggest two reasons why IV-models lead to larger marginal effects of obesity. First, that reporting error with respect to height and weight may cause attenuation bias. Second, non-IV models may suffer from omitted variable bias. The last reason may be the primary explanation for our findings, as a child who suffers from various illnesses may also have reduced appetite and weight loss as a result of these illnesses (Rabbett et al., 1996, Picton, 1998, Fryar and Ogden, 2009, Schaible and Stefan, 2007). For example, cancer, various infectious diseases or anemia are causes of morbidity in children and may also lead to weight loss (Picton, 1998, Schaible and Stefan, 2007, Aukett et al., 1986, Soliman et al., 2009). Also in the case of diabetes type 2, obesity may be masked by significant weight loss in the months or year before diagnosis (American Diabetes Association, 2000). Another explanation might be simultaneity, as health care may have an impact on obesity. In England guidelines produced by the National Institute for Health and Care Excellence (NICE) state that a number of lifestyle and behavioral interventions aimed at weight reduction should be considered for treatment in overweight and obese children (National Institute for Health and Clinical Excellence (NICE), 2014). GPs should take action when there are concerns about the child's weight and decide whether referral to a lifestyle weight management program is appropriate. The family should be

involved in the plan for care as this improves the child's weight loss treatment (Young et al., 2007). Hence, the health service may have reduced the BMI of the child at the time of measurement in the HSE.

Our instrument builds on previous research, which argues that the association between family members' BMI is of purely genetic origin (Cawley and Meyerhoefer, 2012). Due to convincing empirical evidence in favor of this argument (Sørensen et al., 1992, Vogler et al., 1995, Maes et al., 1997), a number of authors have applied this instrument to study the impact of BMI on health and economic outcomes. Nevertheless, we cannot rule out the presence of unobserved characteristics of the parents affecting both health and BMI. To mitigate this in our IV-models we control for a number of observable characteristics including parent's health status. Nevertheless, we have no way of knowing that the IV-strategy controls for all e.g. personality traits of the parents. Hence, we ran sibling-FE models. The findings from these models also suggest that obesity has serious negative consequences for the children when they are still young.

Our study has limitations. First, our measure of obesity is BMI, which has been criticized, e.g., because it does not incorporate body fat, which is an independent predictor of ill health (Burkhauser and Cawley, 2008). Although we use age and gender specific cut-off values for obesity, caution is necessary when BMI is used as children and adolescents can experience growth in height and weight during brief periods (Troiano and Flegal, 1998). Second, we include BMI as a linear continuous variable, when there is evidence of a non-linear association between BMI and health in adults. 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).

To conclude, this study contributes 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 sibling-FE and instrumental variables. The estimates of the effects of obesity on health and health service use are significant in each specification. Hence, obesity has consequences for health and health service use when the children are still young.

Endnotes

[1] We also include a missing education category if either the mother or the father is missing from the household.

[2] Marginal Effect B (binary) = $Pr(Y_i = 1|X, B = 1) – Pr(Y_i =1|X, B = 0)$

Marginal Effect of B (continuous) = limit $[Pr(Y_i = 1|X, B+\Delta) – Pr(Y_i =1|X, B)] / \Delta$] where Δ gets closer and closer to 0

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Table 1: Summary statistics for each sample based on Health Survey for England 1998-2013

	SAH sample		**Doctor visit sample**		**Medication use sample**	
	Full	*Sibling*	*Full*	*Sibling*	*Full*	*Sibling*
Total (N)	29,958	21,116	11,445	8,203	20,294	14,447
Male (N)	15,103	10,688	5,803	4,055	10,150	7,252
Female (N)	14,855	10,428	5,642	4,148	10,144	7,195
Aged 4-12 (N)	18,767	13,517	7,327	5,361	12,690	9,208
Aged 13-18 (N)	11,191	7,599	4,118	2,842	7,604	5,239
Obese (%)	10.3	9.7	9.6	8.9	10.2	9.5
Age (mean)	10.8	10.8	10.6	10.7	10.8	10.8
Survey year (%)						
1998	11.2	11.6	29.3	29.8	14.5	15.0
1999	5.6	5.8	14.6	14.9	0.9	0.9
2000	5.6	5.9	14.7	15.2	1.1	1.1
2001	10.1	10.0	26.4	25.6	12.4	12.3
2002	5.7	5.6	15.0	14.5	7.2	7.1
2003	9.9	10.1	0.0	0.0	11.6	11.8
2004	4.2	4.0	0.0	0.0	0.7	0.7
2005	4.7	4.6	0.0	0.0	5.2	5.2
2006	8.9	9.0	0.0	0.0	10.2	10.3
2007	4.4	4.3	0.0	0.0	4.9	4.9
2008	8.8	8.8	0.0	0.0	9.7	9.7
2009	2.8	2.8	0.0	0.0	3.1	3.2
2010	4.7	4.5	0.0	0.0	4.8	4.7
2011	4.4	4.0	0.0	0.0	4.4	4.2
2012	4.4	4.4	0.0	0.0	4.2	4.2
2013	4.8	4.5	0.0	0.0	5.1	4.9

SAH: self-assessed health

Table 2: The impact of BMI and obesity on good self-assessed health

	Full sample					**Sibling sample**				
	Probit (Eq. 1)		2SRI-probit (Eq. 4)			Probit (Eq. 1)		Sibling-FE (Eq. 2)		
	M.E.	*Z*	*M.E.*	*z*	*Endog. test*	*M.E.*	*z*	*M.E.**	*A.S.E*	*z*
BMI (continuous)										
Total	-0.0034	-8.62	-0.0094	-6.92	4.49	-0.0029	-6.83	-0.0135	-0.0034	-3.97
Male	-0.0034	-5.88	-0.0104	-5.15	3.74	-0.0026	-4.22	-0.0124	-0.0033	-1.95
Female	-0.0034	-6.19	-0.0084	-4.50	2.80	-0.0029	-5.04	-0.0159	-0.0043	-2.36
Aged 4-12	-0.0013	-2.25	-0.0100	-5.09	4.53	-0.0014	-2.39	0.0035	0.0008	0.52
Aged 13-18	-0.0053	-9.11	-0.0101	-4.93	2.52	-0.0046	-6.77	-0.0003	-0.0060	-3.43
Obesity (binary)										
Total	-0.0377	-8.89	-0.1255	-6.59	4.64	-0.0336	-7.03	-0.1600	-0.0421	-4.34
Male	-0.0365	-6.22	-0.1319	-4.92	3.80	-0.0296	-4.42	-0.1841	-0.0467	-2.65
Female	-0.0377	-6.29	-0.1165	-4.23	2.99	-0.0365	-5.32	-0.1147	-0.0470	-2.22
Aged 4-12	-0.0149	-3.09	-0.0918	-4.11	3.57	-0.0143	-2.59	-0.0190	-0.0039	-0.29
Aged 13-18	-0.0740	-9.94	-0.1816	-5.31	3.45	-0.0687	-7.63	-0.0022	-0.0824	-3.47

*The MEs for the based on the sibling-FE logit model are calculated setting the FE equal to zero.
A.S.E: average semi elasticity estimated following (Kitazawa, 2012, Silva and Kemp, 2016).
All F-test of the instruments are above the value of F=20.

Covariates in the probit and 2SRI-probit models: age; highest education of the mother; highest education of the father; ethnicity; Government Office Region (GOR) of residence; survey year; equivalised household income; missing household income; age of the parents; marital status of the parents; smoking status of the parents (current smoker, yes/no); ever tried cigarettes; ever consumed alcohol; SAH of the parents; and, longstanding illnesses of the parents.
Covariates in the sibling-FE models: birth order; maternal age at delivery; and, age.

Table 3: The impact of BMI and obesity on doctor utilization

	Full sample					**Sibling sample**				
	Probit (Eq. 1)		2SRI-probit (Eq. 4)			Probit (Eq. 1)		Sibling-FE (Eq. 2)		
	M.E.	*z*	*M.E.*	*z*	*Endog. test*	*M.E.*	*z*	*M.E.**	*A.S.E*	*z*
BMI (continuous)										
Total	0.0011	1.22	0.0054	1.62	-1.33	0.0001	0.05	0.0062	0.0228	1.07
Male	0.0020	1.59	0.0052	1.07	-0.69	0.0009	0.62	0.0114	0.1121	2.15
Female	-0.0002	-0.15	0.0053	1.15	-1.29	-0.0015	-1.03	0.0010	0.0107	0.25
Aged 4-12	0.0016	1.28	0.0099	2.02	-1.82	0.0012	0.86	0.0079	0.0401	0.98
Aged 13-18	0.0004	0.31	-0.0001	-0.01	0.11	-0.0012	-0.85	0.0000	0.0261	0.72
Obesity (binary)										
Total	0.0170	1.81	0.1085	1.91	-1.56	0.0159	1.44	0.1092	0.4334	2.07
Male	0.0354	2.88	0.1066	1.28	-0.77	0.0289	2.00	0.2826	1.1868	2.57
Female	-0.0043	-0.30	0.0845	1.14	-1.28	-0.0028	-0.17	0.1597	1.0688	1.91
Aged 4-12	0.0256	2.34	0.1626	2.24	-1.85	0.0274	2.14	0.0563	0.5095	1.62
Aged 13-18	-0.0077	-0.43	-0.0253	-0.33	0.26	-0.0148	-0.70	0.0003	0.3667	0.81

*The MEs for the based on the sibling-FE logit model are calculated setting the FE equal to zero.
A.S.E: average semi elasticity estimated following (Kitazawa, 2012, Silva and Kemp, 2016).
All F-test of the instruments are above the value of F=20.
Covariates in the probit and 2SRI-probit models: age; highest education of the mother; highest education of the father; ethnicity; Government Office Region (GOR) of residence; survey year; equivalised household income; missing household income; age of the parents; marital status of the parents; smoking status of the parents (current smoker, yes/no); ever tried cigarettes; ever consumed alcohol; SAH of the parents; and, longstanding illnesses of the parents.
Covariates in the sibling-FE models: birth order; maternal age at delivery; and, age.

Table 4: The impact of BMI and obesity on medication use

	Full sample					**Sibling sample**				
	Probit (Eq. 1)		2SRI-probit (Eq. 4)			Probit (Eq. 1)		Sibling-FE (Eq. 2)		
	M.E.	*z*	*M.E.*	*z*	*Endog. test*	*M.E.*	*z*	*M.E.**	*A.S.E*	*z*
BMI (continuous)										
Total	0.0029	3.18	0.0105	3.81	-2.62	0.0020	1.89	0.0055	0.0194	2.00
Male	0.0016	1.22	0.0088	2.21	-1.71	-0.0001	-0.04	0.0054	0.0182	0.84
Female	0.0038	3.09	0.0121	3.27	-2.18	0.0037	2.59	0.0091	0.0305	1.60
Aged 4-12	0.0039	3.01	0.0168	3.90	-2.93	0.0025	1.62	0.0006	0.0032	0.16
Aged 13-18	0.0022	1.71	0.0070	1.75	-1.24	0.0015	0.99	0.0003	0.0166	1.01
Obesity (binary)										
Total	0.0390	3.99	0.1978	3.90	-3.2	0.0332	2.80	0.0493	0.1645	1.63
Male	0.0247	1.83	0.1511	2.32	-1.90	0.0119	0.73	0.0505	0.1826	0.93
Female	0.0532	3.82	0.2663	3.70	-2.81	0.0551	3.28	0.0501	0.1714	0.89
Aged 4-12	0.0368	3.28	0.2113	3.19	-2.64	0.0272	1.99	0.0388	0.2242	1.36
Aged 13-18	0.0418	2.41	0.1872	2.29	-1.97	0.0408	1.92	-0.0001	-0.0057	-0.03

*The MEs for the based on the sibling-FE logit model are calculated setting the FE equal to zero.
A.S.E: average semi elasticity estimated following (Kitazawa, 2012, Silva and Kemp, 2016).
All F-test of the instruments are above the value of F=20.
Covariates in the probit and 2SRI-probit models: age; highest education of the mother; highest education of the father; ethnicity; Government Office Region (GOR) of residence; survey year; equivalised household income; missing household income; age of the parents; marital status of the parents; smoking status of the parents (current smoker, yes/no); ever tried cigarettes; ever consumed alcohol; SAH of the parents; and, longstanding illnesses of the parents.
Covariates in the sibling-FE models: birth order; maternal age at delivery; and, age.

Table 5: Predicted mean (%) of each dependent variable based on each model, setting all other covariates at their mean

	Reporting good SAH		**Doctor utilization**		**Medication use**	
	Not obese	*Obese*	*Not obese*	*Obese*	*Not obese*	*Obese*
Probit (full sample)						
Male	96.3	92.8	7.2	10.6	17.4	19.9
Female	96.4	92.9	8.9	8.5	18.3	24.0
2SRI-regressions (full sample)						
Male	97.0	76.3	6.8	16.2	16.5	31.6
Female	96.9	80.3	8.2	15.7	17.1	43.7

SAH: self-assessed health