

**Joan Costa-Font and Joan Gil**

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of Child Obesity**

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## **Abstract**

Can the rise in obesity among children be attributed to intergenerationally parental influences? How important is a parent's socioeconomic status in accounting for the emergence of obesity among children? This paper documents evidence of an emerging social gradient of obesity in pre-school children resulting from a combination of income and education effects, as well as less intensive childcare associated with maternal employment, when different forms of intergenerational transmission are controlled for. We also estimate and decompose income related inequalities in child obesity. We take advantage of a uniquely constructed dataset in Spain spanning the years 2003 to 2006, a period in which a significant spike in the growth of child obesity was observed. Our results suggest robust evidence of a socioeconomic and intergenerational gradient. Higher income systematically prevents obesity in children, while inequalities in child obesity have doubled in just three years with a pure income effect accounting for 72-66% of these income inequality estimates, even when intergenerational transmission is accounted for. Although, intergenerational transmission does not appear to be gender specific, when accounted for, mother's labour market participation significantly explains obesity among boys but not among girls.

**Keywords:** child obesity, intergenerational transmission, socio-economic gradient, inequalities in child health.

JEL Classification: I12, I19, D13.

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## 1. Introduction

The prevalence of obesity among children is rising at alarming rates.<sup>1</sup> Latest estimates from the International Association for the Study of Obesity (IASO, 2011) suggest that overweight (including obesity) among children aged 5 to 17 years in Spain<sup>2</sup> is, together with Greece, Italy and the UK, among the highest in Europe.<sup>3</sup> Such estimates are especially concerning given its impact on a child's burden of disease in childhood, adolescence and adulthood (Berenson *et al.*, 1993). Indeed, the association between childhood obesity and mortality and morbidity in adulthood has been well documented (Hoffmans *et al.*, 1988).<sup>4</sup> Gortmaker (1993) found that US women who had been obese in late adolescence were less likely to be married and had lower household incomes seven years later and Sargent and Blanchflower (1994) found that women obese at the age of 16 in the UK earned 7% less seven years later. Adolescents that suffer from obesity are more likely to attempt suicide (Eisenberg *et al.*, 2003) and to perform worse at school (Schwimmer *et al.*, 2003). There is weak evidence that dietary changes are behind the rise in child obesity (Agostini, 2007), hence the underpinning explanations might be found elsewhere.

Unlike obesity in adults, obesity in children is heavily influenced by both parental and the overall child's social environment in addition to genetic transmission (Anderson *et al.*, 2009). However, with few exceptions, intergenerational transmission is not generally accounted for in studies examining the existence of a socioeconomic and educational gradient in child obesity.<sup>5</sup> Currie *et al.* (2007) finds that children of obese parents tend to be obese themselves. An early study by Coate (1983) finds that children with fatter parents are able to produce more adipose tissue, so that the probability of an adolescent (10-16) being obese increases by 20% if either of his parents is obese, while it increases by up to 40% if both parents are obese. However, his results employ a two-stage least square approach with income

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<sup>1</sup> Estimates point towards a prevalence of overweight of 36% (27%) of 9-year-olds in Italy (Spain) threatening an acute health crisis (Gregg and Guralnick, 2007).

<sup>2</sup> A prevalence of 33% among boys and 23% among girls.

<sup>3</sup> Overweight is defined as a *thermodynamic disequilibrium* between calorie intake - especially from the consumption of energy-dense foods, high in saturated fats and sugars - and calorie outtake - primarily, physical activity.

<sup>4</sup> However, some countervailing evidence suggests that individuals at greater risk are those who were thin as children and that become fat as adults (Wright, 2001).

<sup>5</sup> Children lack the knowledge to make informed nutritional decisions, and so parents, as children's guardians, are also their agents in choosing their lifestyles.

and education as instruments, which shed some doubt on its validity. Propper et al. (2007) find that a mother in the top quantile of the pre-pregnancy BMI is 15% more likely to have a child with a BMI distribution in the top 10%. The exact sources affecting the transmission of an individual's health behaviour is the subject of a long-standing debate in the social sciences, and is generally measured by examining the correlation between children's and parents health.<sup>6</sup> If the overall correlation has increased over time, this suggests that something in the common environment (or related to the decisions made by the family) is affecting all family members. On the other hand, if the correlation has decreased over time, then this suggests a larger role for something unique to the environment those children – but not their parents – face, for example something present in child care settings or public schools. Goode et al. (2008) find that the intergenerational transmission of unhealthy eating habits appears to be more intense amongst individuals in low income households.

In addition child obesity appears to be highly influenced by socio-economic factors. Socioeconomic status is often found to be inversely associated with child and adolescent obesity as measured by high levels of BMI (Goodman, 1999; Gordon-Larsen et al., 2003; Wang and Zhang, 2006, Case et al., 2002). In addition, Case et al. (2008), using expanded English and US samples, showed that the income–health gradient for children does indeed increase with age in both countries. Importantly, some evidence suggests an association between child obesity and female employment which is more intense among poorer people (von Hinke Kessler Scholder, 2008)<sup>7</sup>. One potential source might lie in the intergenerational transmission process itself (Baum II and Ruhm, 2007).<sup>8</sup> Currie and Moretti (2007), drawing upon birth records from California, find evidence of a substantial intergenerational correlation as well as an intergenerational transmission of low birth weight, which appears to be stronger for individuals of low socioeconomic status. Anderson et al. (2009) not only report that obesity rates are 25% higher for disadvantaged children but also find that these children gained weight faster. However, importantly, parent-child elasticity and identifiable environmental factors did not exhibit much difference. Propper et al. (2007) and Khanam et al. (2009) found that parental health, particularly the mother's health, plays an intermediary

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<sup>6</sup> In the case of smoking, Duncan et al. (1968) reported a marked increase in smoking amongst teenagers when two or more of their peers smoked, compared to only a moderate increase if only one parent smoked

<sup>7</sup> This is in contrast with the US where it appears to be driven by higher socio-economic groups (Anderson et al., 2003).

<sup>8</sup> Interestingly, they find that an additional year of maternal education reduces child body mass index (BMI) by an average of 0.2 kg/m<sup>2</sup>.

role so that the socioeconomic gradient in child health disappears when parental health is controlled for.

This paper seeks to contribute to the literature by examining the influence of the intergenerational transmission of obesity and its effects on the socioeconomic gradient of obesity. After netting out this influence, we then estimate and decompose the income-related inequalities in child and adolescent obesity. We first examine a reduced health production function that integrates both individual child production factors, parents' agency constraints – working time and income – along with parents obesity that translate into what we define as child to adult obesity, which is taken as a measure of agency failure.<sup>9</sup> Among potential explanations, this paper forwards the hypothesis of parental agency failure as explaining socioeconomic gradients in child obesity. This is hypothetically more acute among children in lower socio-economic households, and hence furthers the income gradient in children's health. We focus on data from one country (Spain) given that together with Italy and Greece it exhibits one of the highest percentages of child obesity in the European Union, and the question has been relatively unexplored. Specifically, we test the effect of differences in parental obesity, affluence (tighter budget restrictions), education<sup>10</sup> and maternal employment as exerting an influence in explaining child and adolescent obesity. It is our contention that this strategy should allow us to disentangle the extent to which the rise in childhood obesity, and its underpinning socioeconomic gradient, result from a combination of these factors.

Secondly, the paper contributes by examining the socioeconomic gradients of obesity and overweight in children. We have estimated the concentration index, the income elasticities and factor decomposition between 2003 and 2006. Specifically, we draw upon the time and individual variation in child obesity, before and after controlling for the transmission of obesity and overweight. Emanuel et al. (1992) find a positive relationship between infant and parental birth weights using the 1958 British Birth Cohort Study. Hence, in our view,

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<sup>9</sup> Agency failure is defined here as the failure of parents to guarantee their children's health, which arguably could result from restrictions that individuals trade off against prioritising their child's health as well as their own cultural and "obesogenic" environment. Thus, the simple existence of obesity in children is itself an indicator of failure, though not necessarily attributable to parental inability to act upon their children. Therefore, the extent to which parents succeed in guaranteeing their children's health is a matter subject to empirical scrutiny.

<sup>10</sup> There is a large and positive correlation between health and education given that children's decisions depend upon the information and ability of their parents (agents), together with that of other agents in the process of education, namely their school teachers and educators.

studies examining the social gradients of obesity in children need to take into account the impact of transmission; otherwise they are likely to suffer from omitted variable bias.

Our results suggest evidence of a socioeconomic gradient in child obesity that is robust to the controls for the intergenerational transmission of life styles, which might have produced omitted variable bias in previous studies. Importantly, when parental obesity is accounted for, a mother's labour market participation significantly explains obesity among boys but not among girls. Patterns of intergenerational transmission are stable over the two samples and periods examined and the effect of the intergenerational transmission tends to concentrate in the extreme quantiles. Finally, we find that income inequalities in child obesity have doubled in just three years with a pure income effect accounting for between 72-66% of these income inequality estimates, even when intergenerational transmission is accounted for.

The rest of the paper is organised as follows. In section 2 we report existing evidence and the theoretical underpinnings for the determinants of child obesity and its social disparities. Section 3 highlights the empirical methods used in our analysis, while section 4 is devoted to presenting our data and variables. Section 5 shows the results and section 6 concludes.

## **2. Intergenerational and Socioeconomic Obesity Gradients**

### *2.1 Intergenerational Gradients*

Parents can influence their children's weight both directly through their genetic influence and environmentally, given that parents' food choices are learned by their children and are so repeated over time. Parents are thus able to influence their children's weight outcomes via a process of "cultural inheritance," for instance, through raw models, learning and interpersonal interactions. There is some evidence in the medical literature of the so-called "child to adult body mass" effects that consider the association between child and parental obesity (Lake et al. 1997). However, only a handful of studies examine the existence of an intergenerational gradient in child obesity.

Ahlburg (1998) examines the intergenerational relationships in health outcomes and diseases and reports estimates of intergenerational correlations for life spans between parents



and children in the range of 0.15–0.3. In making suggestions for further research, he stresses the need to disentangle the causal component. Genetics can have an important effect; for instance, Emanuel et al. (1992) estimate a positive and significant relation between infant and parents' birth weight using the 1958 British Birth Cohort Study.

Some association between mothers and children's weight is found in Classen (2010) who examines the weight status of mothers and their offspring at similar ages using data from the National Longitudinal Survey of Youth 1979 (NLSY79). He finds an intergenerational correlation of BMI between female children and their mothers of 0.38 relative to a BMI correlation of 0.32 between mothers and their sons. The association appears to increase at higher levels of the BMI distribution. However, he finds that having a mother who was overweight significantly increases the likelihood of becoming obese for both white and black females and males, but does not have a statistically significant effect for the likelihood of obesity among Hispanics. Overall though, children of obese mothers are 38% more likely to be overweight or obese.

Whitaker et al. (1997), using observations of BMI during childhood and adolescence combined with data on parental BMI levels, find that children who are obese early in life and have at least one obese parent are three times more likely to become obese adults than non-obese children in households where neither parent is obese. While this effect dissipates slightly as children grow older, they find a very strong correlation with obesity in adolescence. Classen and Hokayem (2005) measure the influence of maternal obesity on the likelihood of child and adolescent obesity with controls for several influential socioeconomic and demographic characteristics. Using data from the NLSY79, they find that children of extremely obese mothers (with BMI greater than 40) are 50% more likely to be obese than their counterparts with mothers having BMI levels in the recommended range of 18.5–25 kg/m<sup>2</sup>.

Anderson et al. (2009) draw on data from repeated cross sections of the National Health and Nutrition Examination Survey (NHANES) to measure the contemporaneous correlation of BMI between women and their children in a given survey year. They find that the intergenerational BMI elasticity between women and their children has increased over time (using observations from 1971 to 2004), but that it does not vary significantly between families of different income levels. They also find similar intergenerational BMI elasticities

for both fathers and mothers in relation to their children. In the most recent NHANES, they find an intergenerational BMI elasticity between women and their children of roughly 0.2, which they attribute to the interaction of common environments and genes between parents and children, with the role of the former becoming increasingly influential over time.

Classen (2010) argues that if high parental BMI levels impinge on family resources then economic success for the subsequent generation may be limited by an increased likelihood of weight problems due both to the genetic predisposition of children as well as to resource constraints. Goode et al. (2008) investigate the possibility of intergenerational transmission of unhealthy eating habits from parents to adult children. Their regressions, which are based on the 2003 Scottish Health Survey, suggest that the paternal history of eating habits has no impact on either sons or daughters while the maternal history influences negatively the eating behaviour of daughters.

Monherit et al. (2009) finds evidence of parental body weight influencing adolescents' body weight. This result contrasts with an earlier study by Coate (1983) showing that while parental obesity does influence child and adolescent weight, diet is unrelated to these outcomes so only genetic transmission matters. By contrast, Anderson et al. (2003) finds that a mother's BMI is strongly related to child overweight.

## *2.2 Socioeconomic Gradients*

A set of studies (Case et al. 2004, Currie et al. 2004, Currie and Stabile, 2003, Currie and Hyson, 1999) investigates the effect of socioeconomic status on child health and finds evidence of a family income gradient. Socioeconomic gradients in health are argued as having their origin in childhood. Case et al. (2008) examine differences in child health resulting from income gradients. Although socioeconomic gradients of wellbeing among children and adults are well known, the interpretation of their specific triggers is controversial (Abernathy et al, 2002). Socially disadvantaged individuals are found to have less autonomy to choose healthy behaviours (Wickrama et al, 1999). There is some evidence that indicates that parent's education and social position influences the likelihood of a child being obese (Power et al. 2003) as well as some evidence of pro-rich socio-economic inequalities in obesity among children (Kinra et al. 2000; Armstrong et al. 2003). Importantly, Anderson et al. (2003) as well as Cawley and Liu (2007) provide evidence that a child might become overweight if his

or her mother works because (a) working mothers face more time constraints than non-working mothers, hence they may have less time to ensure that their children consume a nutritionally balanced diet; (b) working mothers are more prone to serving their families high-fat prepared or fast foods; (c) working mothers' children may spend more time on sedentary activities such as playing computer games or watching TV, and may often choose to sneak high-calorie foods after school.<sup>11</sup>

Some studies claim that unmeasured and often unobserved parental factors have an effect on children's health (Dooley and Stewart, 2007). Other studies find that parents' obesity can predict weight in young adulthood, which again calls for an intergenerational transmission of body weight but, interestingly, those children whose parents tried to control their weight are more likely to be obese. Hence, parental control of adolescent diets does exert an influence on adult obesity (Crossman et al. 2006). Anderson et al. (2003) using the NLSY data find evidence of a significant positive association between the number of hours per week by the mother and child overweight. Similarly, Garcia et al. (2005) provide empirical evidence that is apparently consistent with these effects.

### **3. Methods**

#### *3.1 Measurement of childhood obesity*

We measure childhood obesity by means of parents' self-reported data on height and weight allowing us to define the widely used "body mass index" (BMI) indicator for each child.<sup>12</sup> This index, defined as weight in kilograms divided by the square of height in meters ( $\text{kg}/\text{m}^2$ ), although imperfect enables us to obtain an estimate of the prevalence of obesity. As is well known, the adult cut-off points in widest use for defining overweight is a BMI of  $25 \text{ kg}/\text{m}^2$  while that for defining obesity is  $30 \text{ kg}/\text{m}^2$ , both figures intended to be related to health risks

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<sup>11</sup> While some studies report a protective effect of physical activity against obesity, others find no association and a few studies have suggested that higher activity is related to increased fatness (Goran et al. 1998). Other studies using longitudinal cohort data from England find that the association varies in early adolescence and late adolescence (Parsons et al. 2005). Generally, however, the gender difference in children's physical activity is well established and Hamershmesh (2010) finds that both the amount of time spent eating and the frequency of eating influence individuals' weight. Particularly, those who eat more (regular) meals have lower BMI and report better health status.

<sup>12</sup> Using self-reported data in child obesity is less problematic than in adults given that parents weigh their own children and keep control of their weight through compulsory annual health examinations at school.

(WHO, 1998). However, these thresholds cannot be used for measuring (overweight) obesity in children/adolescents since their BMI is lower than that of adults. Moreover, the BMI changes significantly during childhood and differs between boys and girls. Hence, this means that age and gender specific reference cut-off points are necessary to interpret the measurement of child obesity (Reilly et al. 2002).

Given the controversy among specialists as regards the best BMI tables and reference threshold points to apply,<sup>13</sup> in this study we have opted to follow two alternative methods to measure childhood obesity. In both cases, child obesity is obtained by comparing individual BMI calculated from the data against age and gender-specific reference values for obesity. First of all, we rely on national data and use the BMI cut-off points corresponding to the 97<sup>th</sup> (85<sup>th</sup>) centile for obesity (overweight) of Sobradillo et al. (2002). These are known as the new ‘Orbegozo Foundation’ tables, and have a long tradition of clinical use in Spain.<sup>14</sup> For instance, the well-known ‘EnKid’ study of the prevalence of childhood and adolescent obesity in Spain used the same 97<sup>th</sup> centile reference point and the previous edition of the ‘Orbegozo F.’ tables (Serra et al. 2003). Second, we also employ as a reference the “international” cut-off points calculated by Cole et al. (2000) for global comparisons of childhood obesity prevalence. In line with recommendations of the International Obesity Task Force (IOTF), these authors, by pooling cross-sectional data on BMI for children from six countries (Brazil, Great Britain, Hong Kong, the Netherlands, Singapore and the United States) and by using the centile based method (ensuring that at the age of 18 they matched the adult cut-off of 30 kg/m<sup>2</sup>), were able to calculate BMI cut-off points for obesity for children aged 2-18 years. In the rest of this study, this scale will be referred to as the IOTF definition or reference tables.

### *3.2 Empirical methods: determinants of childhood obesity*

We first examine the prevalence of childhood obesity for a representative sample of Spanish children by estimating a probit regression model. We are interested in the probability of a child becoming obese ( $y^*$ ) satisfying the linear model:

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<sup>13</sup> Different countries have developed their own centiles for these cut-offs. For example, in the USA obesity is defined as a BMI exceeding the 95<sup>th</sup> centile of the US CDC 2000 reference table, while in the UK the definition uses the 98<sup>th</sup> centile of the British 1990 reference.

<sup>14</sup> Specifically, we employ the cross-sectional data based on a sample of 6,443 individuals aged 0-18 years gathered between Nov. 2000 and Oct. 2001 in the province of Biskaia.

$$y^* = X' \beta + u \quad (1)$$

where  $y^*$  is the underlying unobserved (or latent) continuous variable,  $X$  is a set of regressors,  $\beta$  is a vector of unknown parameters and  $u$  the error term. Yet, what we observe and estimate is the outcome variable,  $y$ , taking one of two values:

$$y = \begin{cases} 1 & \text{if } y^* > 0 \\ 0 & \text{if } y^* \leq 0 \end{cases} \quad (2)$$

Thus the conditional probability of child obesity takes the form:

$$\Pr(y = 1|X) = \Pr(y^* > 0) = \Pr(X' \beta + u > 0) = \Pr(-u < X' \beta) = \Phi(X' \beta) \quad (3)$$

where  $\Phi(X' \beta | X_k = 1)$  is the standard normal cumulative distribution function for the error term  $u \approx N(0,1)$ , giving rise to the probit model. The maximum likelihood (ML) estimator is used to estimate the set of  $\beta$  coefficients. As these probit coefficients cannot be given a direct quantitative interpretation we need to compute marginal (average) effects for the continuous (binary) explanatory variables.<sup>15</sup> As a robustness check we further estimated through OLS and quantile regression the influence of the same set of explanatory variables on the (continuous) child body mass index, so as to measure whether the effects of the main set of covariates remained valid.

Up to this point, the above estimations seek to analyse the empirical association between childhood and parental obesity but they overlook the key issue of endogeneity of parental obesity status. However, as is well known, probit ML estimators are inconsistent if any of the regressors are endogenous. To overcome this problem we adjusted a generalised version of the bivariate probit model in order i) to allow for the correlation between the error terms of the child and parental obesity equations and to recognise the existence of unobservable individual characteristics affecting both outcomes and ii) to accommodate the endogenous effect of the parental obesity covariate. Specifically, we specify a joint model for

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<sup>15</sup> The marginal effect of an explanatory variable ( $X_k$ ) is calculated as  $\partial \Pr(y = 1 | X) / \partial X_k = \beta_k \phi(X' \beta)$ , where  $\phi(\cdot)$  denotes the standard normal density function. While the average effect of a binary variable is computed as:  $\Pr(y = 1 | X_k = 1) - \Pr(y = 1 | X_k = 0) = \Phi(X' \beta | X_k = 1) - \Phi(X' \beta | X_k = 0)$ .

two unobserved latent variables ( $y_1^*$ : childhood obesity;  $y_2^*$ : parental obesity) that may be correlated and depend linearly on a set of explanatory variables as,

$$\begin{aligned} y_1^* &= \delta_1 y_2^* + X_1' \beta_1 + \varepsilon_1 \\ y_2^* &= X_1' \pi_1 + Z_2' \pi_2 + \varepsilon_2 \end{aligned} \quad (4)$$

where the first equation is the structural equation of interest and the second equation is the reduced form of the endogenous discrete regressor  $y_2$  (parental obesity). Here the errors  $\varepsilon_1$  and  $\varepsilon_2$  are jointly normally distributed with means of 0 and variances of 1, and correlations of  $\rho$ . In this model the two observed binary outcomes are,

$$y_1 = \begin{cases} 1 & \text{if } y_1^* > 0 \\ 0 & \text{if } y_1^* \leq 0 \end{cases} \quad \text{and} \quad y_2 = \begin{cases} 1 & \text{if } y_2^* > 0 \\ 0 & \text{if } y_2^* \leq 0 \end{cases} \quad (5)$$

the model collapses to two separate probit models for  $y_1$  and  $y_2$  if  $\rho=0$ . Interestingly, notice that in equation (4)  $X_1$  is a vector of exogenous regressors and  $Z_2$  is a vector of additional instruments that affect  $y_2$  or parental obesity but that they can be excluded from the structural equation as they do not directly affect childhood obesity ( $y_1$ ). The reduced equation serves as a source of identifying instruments and these excluded instruments are essential for identifying the parameters of the structural equation.

### 3.3 A note on the instruments selected

We assume parental obesity to be an endogenous regressor. The intuitive justification lies in the fact that there would appear to be various mechanisms, both cultural and genetic, that might explain the transmission of obesity. Bearing in mind the difficulty of deriving good instruments, we constructed three potential instruments for parental obesity intended to reflect the actual living conditions of the child's father/mother during his/her childhood and more generally of Spanish society at that time. Thus, the goal was to capture a wealth effect when the mother/father was 10 years old. Hence, the *lpmilk10* instrument is the log of the annual production of milk (in litres per capita) in the year in which the mother was aged 10, the *lgdppc10* instrument is measured as the log of the GDP per capita (in real 1995 PTA) when

the mother was aged 10 and, finally, *lpbeef10* instrument is the log of the annual production of beef when the mother was aged 10. These instruments are likely to be relevant since each is expected to present a significant negative correlation with parental obesity ( $y_2$ ). Generally speaking, the better the economic conditions (proxied by these instruments) enjoyed by the population, the lower is the expected obesity prevalence rate. In addition, to be valid, the instruments must satisfy  $E(\varepsilon_1 | Z_2) = 0$ . In practice, we opted to estimate an exactly identified model where the number of exogenous regressors equals the number of instruments.

### 3.4 The income-related inequalities in child obesity

To estimate the income-related inequalities in child obesity we followed the traditional procedures based on the calculation of concentration indices. Specifically, this index is calculated using the “convenient regression” approach proposed by Kakwani et al. (1997). We further decomposed these concentration indices, along the lines suggested by Wagstaff et al. (2003), according to the individual factors that contribute to income-related inequalities in child obesity. For any linear additive regression model of child obesity ( $y$ ) on a set of regressor ( $x$ ) such as,

$$y = x'\beta + \varepsilon \quad (6)$$

the concentration index for  $y$  (CI) can be decomposed as follows,

$$CI = \sum_k (\beta_k \bar{x}_k / \mu) C_k + GC_\varepsilon / \mu \quad (7)$$

where  $\mu$  is the mean of  $y$ ,  $\bar{x}_k$  is the mean of  $x_k$ ,  $C_k$  is the concentration index of the regressor  $k$  and  $GC_\varepsilon$  is the generalised concentration index for the error term. Equation 7 highlights that the CI of child obesity is equal to a weighted sum of the concentration indices of the set of regressors, where the weight for  $x_k$  is the elasticity of  $y$  respect to  $x_k$  ( $\eta_k = \beta_k \frac{\bar{x}_k}{\mu}$ ), plus a residual component.<sup>16</sup>

## 4. Data

We used pooled data from the 2003-04 and 2006-07 editions of the Spanish National Health Survey (SNHS). This is a biannual and nationwide cross-section survey collecting

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<sup>16</sup> See O'Donnell et al. (2008) for a useful guide for calculating and decomposing concentration indices.

information on the self-perceived health status of the population, primary and specialised health care utilisation, consumption of medicines, perceived mortality, life habits, conducts related to risk factors, anthropometrical characteristics, preventive practices and also socioeconomic characteristics of individuals. Both surveys contain separate adult (16+) and child samples in addition to a household questionnaire. This study is based on the child sample and where required we match information gathered from the adult and household samples. Yet, as two generations are available it is possible to match child health and other characteristics with that of their parents, including a parent's socio-economic status and education.<sup>17</sup>

The original pooled sample contained 15,231 observations of children aged 0-15 from all Spanish regions. Notice the dataset has a non-negligible share of missing information on children's weight and height measurements. Fortunately, rather than deleting these observations (concentrated mainly at the younger ages), we took advantage of a survey question (with almost no missing information) intended to elicit the relationship between a child's height and weight.<sup>18</sup> This allowed us to impute average weight and height based on non-missing cases for this subset of children. Abnormally high and low BMI values were excluded from the estimations. In addition, some observations were excluded owing to missing parental information (i.e. household income, weight and height) and individuals being below the age of two. As a result, our pooled sample contained 13,358 observations of children aged 2-15 (5,483 for the sample 2003-04; 7,875 for the sample 2006-007).

Table 1 presents the definition of the variables used in the regressions. In the case of the dependent variables, we measured child overweight and obesity as binary indicators taking into account both the Orbegozo Foundation and the IOTF cut-off points. Moreover, we also used a continuous covariate to measure child BMI. As for the independent variables, we classified the covariates in four groups: a) children's variables including information on

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<sup>17</sup> The surveys adopt a stratified multi-stage sampling procedure where the primary strata are the Autonomous Communities (Spanish regions). Sub-strata are defined according to population size of area of residence. Within sub-strata, municipalities and sections (primary and secondary sampling units, respectively) are selected following a proportional random sampling scheme. Finally, individuals are randomly selected from the sections. Both surveys provide weighting factors to elevate estimations to the national level.

<sup>18</sup> The question, with four responses, measures whether child's weight to height is considered to be quite a lot higher than what is normal (1); somewhat higher than normal (2); somewhat lower than normal (3) or quite a lot lower than normal (4).



gender, age, physical activity in leisure time, Mediterranean diet,<sup>19</sup> watching TV on a daily basis and number of hours slept per day; b) parents' socio-economic controls including income and education and mother's labour participation; c) key controls for parents such as the adult obesity rate or adult BMI;<sup>20</sup> and d) parents' civil status and sample year.

As the survey collects household income in intervals (with up to eight responses) an interval regression was run to estimate an income level for each household.<sup>21</sup> As an additional step, we imputed a (log) net household income in equivalent terms to each child dividing the income figure by the number of household members powered to 0.5. As for the parental level of education we distinguished up to three levels of completed education: primary or less than primary education, secondary education (the omitted category) and university education. A covariate measuring the number of completed years of education was also analysed.

To verify empirically the veracity of our hypothesis regarding the vertical transmission of obesity, we analysed the influence of the adult obesity rate (BMI) as a key determinant of parental lifestyles and, consequently, as a problem of agency failure.<sup>22</sup> We expected to find a positive effect of this key control on the children's obesity (BMI) equation. As discussed above, parent's increasing income and education are expected to reduce the probability of childhood obesity (BMI). To test the possible association between the mother's labour participation and the prevalence of childhood obesity (BMI) we defined a binary variable measuring whether the mother participates actively in the labour market (i.e., being employed or unemployed). In principle, a positive relationship is expected between the number of hours devoted by the mother to work (resulting in less intensive childcare) and child obesity or BMI.<sup>23</sup>

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<sup>19</sup> We calculated this covariate selecting a subset of foods that are typical of a Mediterranean diet and which are highly correlated to a child's body mass (i.e., fresh fruits, vegetables, fish and sweets). We then assumed that they are consumed at a high frequency rate (between three times per week to a daily basis). A negative association with child obesity/BMI was expected.

<sup>20</sup> Given the type of information collected in the adult questionnaire, here adult obesity or BMI refers to the presence of such condition in the father or in the mother.

<sup>21</sup> As regressors we considered gender, age, level of education, economic activity status and regional variables of the head of the household.

<sup>22</sup> A BMI cut-off point of 30 kg/m<sup>2</sup> was employed to define both the adult obesity prevalence rate as well as the corresponding relative degree of obesity.

<sup>23</sup> In fact, Garcia et al. (2005) found a positive association between female labour market participation and childhood obesity in Spain using the same database.

**Table 1. Definition of Variables. Pooled Sample of Children (SNHS 2003-04 & 2006-07)**

Variable	Description	Mean	Min.	Max.
<i>(N=13,358 obs).</i>				
<i>Dependent variables</i>				
Child overweight (IOTF)	Overweight defined according to IOTF BMI reference tables	0.286	0	1
Child overweight (FO)	Overweight defined according to F. Orbegozo BMI reference tables (percentile 85)	0.233	0	1
Child obesity (IOTF)	Obesity defined according to IOTF BMI reference tables	0.095	0	1
Child obesity (FO)	Obesity defined according to F. Orbegozo BMI reference tables (percentile 97)	0.126	0	1
Child BMI	Child BMI (calculated as weight in Kg divided by square height in m.)	18.22	6.57	55.36
<i>Key explanatory variables</i>				
Income	Log of total net equivalent income	6.568	4.262	8.371
Mother's low education	1 if mother has primary or less than primary education	0.311	0	1
Mother's high education	1 if mother has university education	0.204	0	1
Father's low education	1 if father has primary or less than primary education	0.312	0	1
Father's high education	1 if father has university education	0.185	0	1
Mother's labour activity	1 if mother participates in the labour market	0.611	0	1
<i>Children's characteristics</i>				
Male	1 if male	0.516	0	1
Age	Child's age	8.828	2	15
Age2	Square of child's age	94.09	4	225
Mediterranean diet	1 if follows a Mediterranean diet *	0.053	0	1
Physical activity	1 if performs physical activity in leisure time	0.783	0	1
Watch TV daily	1 if watch TV on daily basis	0.904	0	1
Sleeping hours	Number of hours slept per day	9.440	1	19
<i>Key controls for parents</i>				
Parent's obesity	1 if the household adult parent (father or mother) is obese	0.124	0	1
Parent's BMI	BMI of the household adult parent (father or mother)	25.26	12.77	58.96
<i>Other controls for parents</i>				
Married parents	1 if parents are married	0.829	0	1
Sep./divorced parents	1 if parents are separated or divorced	0.054	0	1

Note: Means are computed taking into consideration sampling weights.

## 5. Results

### 5.1 Preliminary Evidence

Table 1 shows that the mean BMI of the children aged 2-15 in our pooled data set is 18.22 kg/m<sup>2</sup> ranging from 6.57 to 55.36 kg/m<sup>2</sup>. The mean BMI of boys (girls) increases from 16.58 (15.83) kg/m<sup>2</sup> at age 2 to 20.99 (20.45) kg/m<sup>2</sup> at age 15.<sup>24</sup> According to the new ‘Orbegozo F.’ reference tables the prevalence of overweight for both genders is 23.3% (defined at the 85<sup>th</sup> percentile) while the obesity rate is 12.6% (defined at the 97<sup>th</sup> percentile). Interestingly, a number of differentials are found when the IOTF tables are applied. Thus, childhood overweight is significantly higher (28.6%) while childhood obesity is lower (9.5%).<sup>25</sup> As in other western countries, childhood obesity has accelerated in Spain at all ages in the last two decades. Based on data from the 1993 edition of the SNHS, child obesity has increased between 7-8 percentage points at ages 4 to 5, and between 3-5 percentage points at ages 2 to 3.

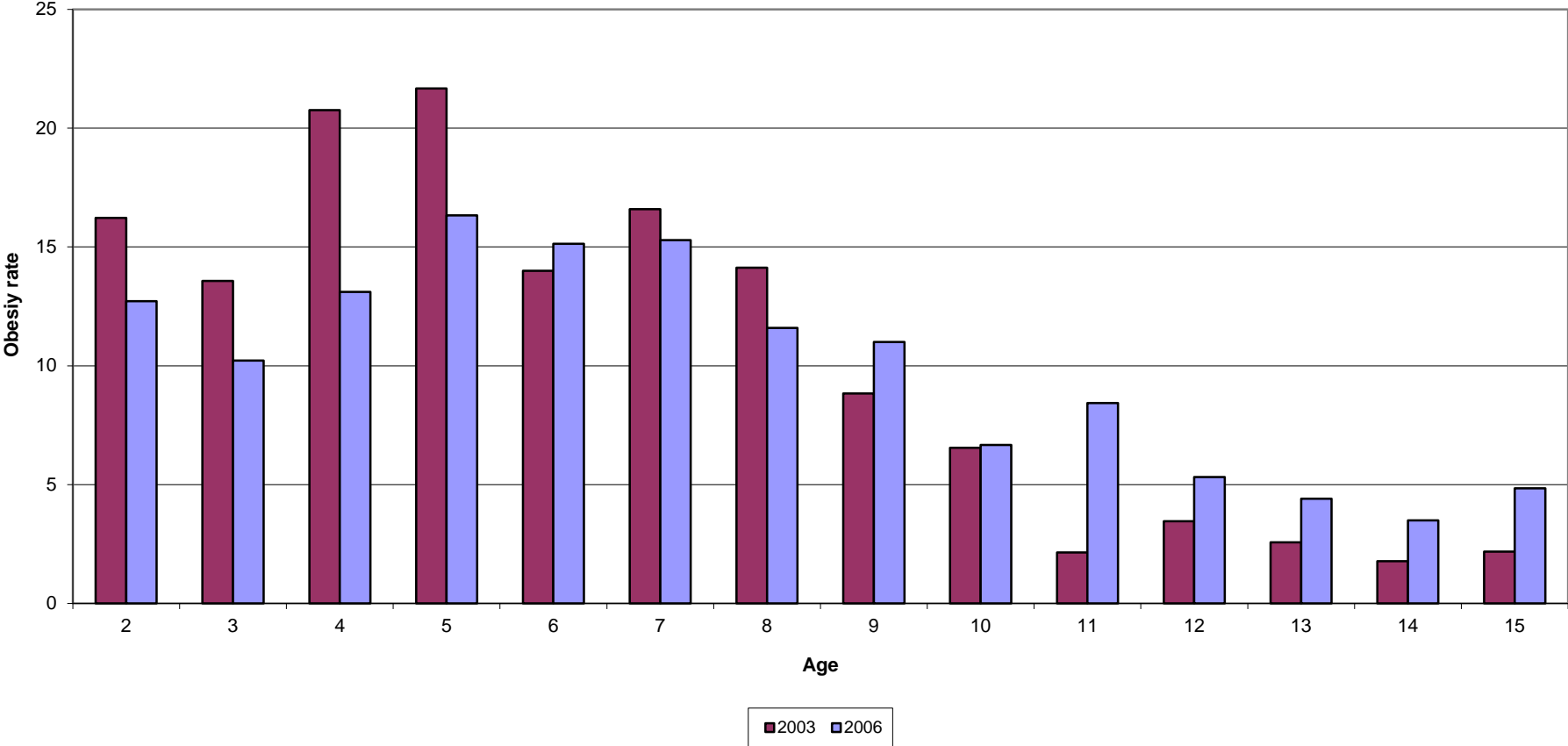
The intuition that the prevalence of childhood obesity is not linearly distributed by age is captured in Figure 1. An inverted U-shape distribution can be seen in both cross-sections indicating that the condition increases up to the age of 5 and then declines monotonically. The figure also documents that child obesity is more accentuated in the 2- to 8-year-olds’ subgroup (averaging 14.8%) than among their older counterparts aged 9 to 15 (5%). This empirical evidence agrees with the fact that it is around the age of 6 or 7 that children start taking part in physical activity at school or in sports clubs and when children spend most of their time at school. Before that age, children are either at home with their parents or carers or/and at the nursery. Clearly, this contrasts sharply with data for the UK. Based on a sample of 2,000 children from the 2004 National Health Survey and considering measured height and weight, it was found that 26.7% (24.2%) of girls (boys) between the ages of 11 and 15 were obese; nearly double the 1995 rate.

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<sup>24</sup> This pattern of mean BMI is roughly similar to that reported by Serra et al. (2003) using another dataset with clinical measurements. These authors report that gender divergences in BMI in Spain occur from age 17 on, progressively increasing in boys and decreasing in girls.

<sup>25</sup> For reasons of space, our main findings are based on the IOTF reference tables (given that researchers are more familiar with them). Results based on the ‘Orbegozo F.’ tables are available upon request.

**Figure 1. Childhood Obesity: IOTF reference tables (SNHS samples 2003-04 & 2006-07)**



Given that food intake in children between the ages of 2 and 7 is fundamentally a parents' responsibility, this preliminary evidence might suggest an "agency failure" underpinning children's negative health outcomes. Previous studies conducted in the adult population show that obesity has risen markedly in Spain, especially among middle-aged married couples (Costa-Font and Gil, 2004). Given that most children today are still being raised by married couples, it might well be that the increase in children's body mass results from the same underlying factors that explain adult obesity. Given that children's food intake and lifestyles are determined by their parents, parents' obesity might well be transmitted to their children via a direct influence (i.e., preparation of meals) and via the cultural transmission of eating habits learned from their parents.

## *5.2 Child obesity and BMI*

The results of the probit estimation of child obesity (measured using the IOTF tables) are reported in Table 2.<sup>26</sup> Several econometric specifications are examined. While the first three columns analyse overall obesity in the sample, the last two differentiate by gender. All estimations include controls for children's characteristics and those for their parents (see Table 1). Notice that parental obesity is controlled for in order to examine the presence of intergenerational transmission, and its effect on the socioeconomic gradient in child obesity. Statistical inference is based on clustered standard errors using the covariate region. All parameter estimates are jointly significant and the estimations show an adequate goodness of fit.<sup>27</sup>

According to the first three columns, the results confirm the existence of a significant positive association between adult obesity and child obesity, confirming our vertical transmission hypothesis. That is, children are more likely to be obese when this condition is also prevalent among their fathers or mothers. The results indicate that the probability of having obese children is between 4.2% and 4.6% higher for obese parents. As expected, income exerts a significant negative influence on child obesity in all specifications. The probability of child obesity is roughly 3% lower as net equivalent income rises. We find that parents' education also affects child obesity. Interestingly, mothers with a relatively high

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<sup>26</sup> Results relative to the estimation of child overweight are available upon request.

<sup>27</sup> Estimates of marginal and average effects are computed at the sample means of the regressors.

education background are between 1.6% and 2.2% less likely to have children exhibiting some level of obesity. As column three shows the positive association between child obesity and a mother and father's low educational attainment disappears once income is included in the model. As expected, our estimations suggest that the mother's labour market participation is positively correlated with child obesity.

However, the last two columns of Table 2 show the emergence of a differential pattern when the pooled sample is split by gender. Interestingly, while the hypothesis of intergenerational transmission is verified for both genders, this correlation is much stronger among boys. The estimations indicate that boys (girls) aged 2-15 are roughly 6% (2.5%) more likely to be obese when this condition is also prevalent among their fathers and mothers.<sup>28</sup> Income and mother's higher education present a significant and negative correlation with child obesity in boys only. Notwithstanding, equivalent income tends to reduce the likelihood of child obesity among girls by 2.8%.<sup>29</sup>

Similarly, in the first three columns of Table 3 we present the OLS estimations of child BMI using the complete set of controls. Here again the findings indicate that parental BMI is significantly and positively correlated with child obesity in both genders, documenting once more a problem of agency failure. Thus, a one unit increase in the father's/mother's BMI is associated with an increase of 0.11kg/m<sup>2</sup> in the mean child BMI. The estimations also provide evidence of a significant negative effect of income on children obesity. The last three columns of Table 3 show the estimates of the quantile regression of child BMI based on the 95<sup>th</sup> percentile, the cut-off point generally used by experts to measure child obesity. As expected, these effects are much stronger for the upper deciles of the body mass distribution. Estimates are in line with previous studies (Ahlburg, 1998, Emanuel et al., 1992 and Classen, 2010).

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<sup>28</sup> We find fairly similar results when we use the Orbegozo F. tables. These tables use the 97<sup>th</sup> percentile as indicative of child obesity.

<sup>29</sup> The estimates in Table 2 are only marginally affected when we exclude the influence of parent obesity. This indicates that our measure of intergenerational transmission is autonomously captured by this covariate.

**Table 2. Probit Estimation of Child Obesity. Pooled Sample of Children (SNHS 2003-04 & 2006-07)**

	Dependent variable: Child Obesity (IOTF tables)				
	Full sample Col. [1]	Full sample Col. [2]	Full sample Col. [3]	Boys Col. [4]	Girls Col. [5]
Income	-0.222*** [-0.032]	-	-0.199*** [-0.029]	-0.180*** [-0.028]	-0.223*** [-0.028]
Mother's low education	-	0.069*** [0.010]	0.044 [0.006]	0.031 [0.005]	0.061 [0.008]
Mother's high education	-	-0.166*** [-0.022]	-0.120** [-0.016]	-0.166** [-0.024]	-0.075 [-0.009]
Father's low education	-	0.103** [0.015]	0.066 [0.010]	0.138* [0.022]	-0.024 [-0.003]
Father's high education	-	0.003 [0.000]	0.047 [0.007]	0.025 [0.004]	0.059 [0.008]
Mother's labour activity	-	0.067*** [0.009]	0.093*** [0.013]	0.075* [0.011]	0.114 [0.014]
Parent's obesity	0.267*** [0.044]	0.282*** [0.046]	0.258*** [0.042]	0.315*** [0.057]	0.178*** [0.025]
Controls for children's charact.	YES	YES	YES	YES	YES
Other controls for parents	YES	YES	YES	YES	YES
Observations	10,273	12,211	10,273	5,231	5,042
Wald test	1,477.7	7,244.5	42,784.8	5,083.5	2,920.5
Pseudo R-squared	0.070	0.073	0.072	0.068	0.087
Obs. Pr.	0.094	0.091	0.094	0.100	0.088
Pred. Pr. (%)	0.077 (82%)	0.074 (81%)	0.077 (82%)	0.084 (84%)	0.066 (75%)

Note: Child obesity is calculated using the International Obesity Task Force tables. Parent's obesity corresponds to the obesity of the father or mother. Standard errors are adjusted for 18 clusters in region. Estimations include a dummy for the sample year. Marginal and average effects are reported in brackets. \*\*\* P < 0.01, \*\* P < 0.05, \* P < 0.1

**Table 3. OLS and Quantile Estimation of Child BMI. Pooled Sample of children (SNHS 2003-04 & 2006-07)**

	Dependent variable: Child BMI						
	OLS			Quantile (95 <sup>th</sup> )			
	Full sample Col. [1]	Boys Col. [2]	Girls Col. [3]	Full sample Col. [1]	Boys Col. [2]	Girls Col. [3]	
Income	-0.330***	-0.302**	-0.359**	-1.111***	-0.938**	-1.155**	-
Mother's low education	0.040	-0.061	0.139	0.760*	0.775	1.403**	-
Mother's high education	-0.211**	-0.261	-0.160	-1.185***	-0.626	-1.609***	-
Father's low education	0.018	-0.014	-0.039	0.483	1.054**	-0.538	-
Father's high education	0.098	0.194	0.001	0.287	0.082	0.534	-
Mother's labour activity	0.158	0.150	0.175	0.172	-0.195	0.880**	-
Parent's BMI	0.115***	0.114***	0.115***	0.206***	0.173***	0.214***	-
Controls for children's charact.	YES	YES	YES	YES	YES	YES	-
Other controls for parents	YES	YES	YES	YES	YES	YES	-
Observations	10,273	5,231	5,042	10,273	5,231	5,042	-
R-squared	0.157	0.158	0.156	-	-	-	-
Pseudo R-squared	-	-	-	0.064	0.085	0.057	-

Note: Parent's BMI corresponds to the BMI of the father or mother. OLS standard errors adjusted for 18 clusters in region. Bootstrapped standard errors in Quantile regression with replications set at 200. The estimations include a dummy for the sample year. \*\*\* P < 0.01, \*\* P < 0.05, \* P < 0.10.



### 5.3 Child obesity: accounting for endogeneity and unobservables

Table 4 reports the results for the generalised bivariate probit model estimation of child obesity and parental obesity. This strategy enables us, first, to control for the presence of unobservable characteristics influencing both outcomes and, second, to accommodate the endogenous effect of the parental obesity epidemic. We instrument parental obesity using the log of the production of milk per capita in Spain in the year in which the mother was ten years old. Again we used clustered standard errors based on 18 regions. For the sake of brevity, the table only presents the coefficients of the structural model.

The first column shows the estimation of the model accounting for the entire sample of both genders. As expected, we find the coefficient of the instrument (*lpmilk10*) to be negative and highly statistically significant. The first stage regression has reasonable explanatory power as indicated by the high value of the Wald test. The correlation coefficient for the two error terms ( $\rho = -0.248$ ) recognises that unobservable factors affecting both child obesity and parental obesity are negatively associated, although the low chi-square test indicates that this parameter is only significantly different from zero at 10%. Interestingly, the results provide evidence of a much stronger, significant positive causal impact of parental fatness on child obesity (0.741 compared to 0.258 in Column 3 of Table 2), indicating a causal impact of parental lifestyles on child obesity. The other hypotheses documented earlier in Table 2 are also evidenced here. Here again, income and maternal higher education exert a similar significant negative influence on child obesity and a mother's labour market participation is positively correlated with child obesity. When we distinguish by gender, we find that the transmission to boys is significantly higher than transmission to girls.<sup>30</sup>

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<sup>30</sup> The same set of results is obtained when using the other instruments.

**Table 4. Bivariate Probit Estimation of Child Obesity: Accounting for Endogeneity. Pooled sample of Children (SNHS 2003-04 & 2006-2007)**

	Dependent variable: Child Obesity (IOTF tables)		
	Full sample Col. [1]	Boys Col. [2]	Girls Col. [3]
Income	-0.174*** [-0.016]	-0.147*** [-0.012]	-0.193*** [-0.013]
Mother's low education	0.030 [0.004]	0.0123 [0.003]	0.044 [0.003]
Mother's high education	-0.121** [-0.002]	-0.164** [-0.010]	-0.077 [-0.005]
Father's low education	0.046 [0.006]	0.106 [0.010]	-0.038 [-0.001]
Father's high education	0.053 [0.003]	0.031 [0.001]	0.067 [0.004]
Mother's labour activity	0.090*** [0.006]	0.068* [0.005]	0.113 [0.005]
Parent's obesity	0.741*** [0.089]	0.933** [0.113]	0.766** [0.072]
Control for children's charact.	YES	YES	YES
Other control for parents	YES	YES	YES
Observations	10,130	5,157	4,973
Instrument "lpmilk10" (1st stage)	-0.262**	-0.349*	-0.205
Wald test (1st. stage)	15,915	425.6	-
Rho	-0.248	-0.318	-0.300
Wald test of exogeneity	3.017 (p=0.082)	2.865 (p=0.090)	2.891 (p=0.089)

Note: Child obesity is calculated using the IOTF tables. Parent's obesity is instrumented by the log of the production of milk per capita in the year in which the mother was ten years-old. Standard errors adjusted for 18 clusters in region. Marginal effects in brackets computed as  $y = \Pr(\text{child obesity}=1 | \text{adult's obesity}=1)$  at the mean values. \*\*\* P < 0.01, \*\* P < 0.05, \* P < 0.10.

The availability of two waves allows us to test whether the parent-child BMI correlation changes over time. Specifically, Table 5 calculates the change in the intergenerational transmission of child obesity and BMI between the 2003-04 and 2006-2007 waves by gender. Interestingly, the estimates show a marked increase in the intergenerational transmission mainly in boys. This finding is somewhat similar to the results reported by Anderson et al. (2009) who documented a substantial increase in the transmission of child obesity since the early 1970s, suggesting that shared common genetic-environmental influences have become more important in determining obesity.

#### *5.4 Social gradients*

To estimate the differential impact of the parental transmission hypothesis by income levels, Table 6 reports the estimates of the intergenerational transmission of child obesity across income quartiles in the two waves. Interestingly, we find the transmission to be higher among extreme quartiles, which suggests different explanations for the development of a socioeconomic gradient in child obesity.

Figure 2 represents graphically the concentration curves of child obesity using both the IOTF tables and the Orbegozo F. tables. As can be seen, the graphs are well above the 45° line and, thus, they clearly indicate that child obesity is unequally distributed to the disadvantage of poorer children. Interestingly, as the concentration curves for the years 2006-2007 lie above those for the years 2003-2004, the figure shows that income-related inequalities in child obesity have increased over time, regardless of the tables used to define the condition. Indeed, the degree of income-related inequality is clearly shown in Table 7, where a set of statistically significant negative CIs of child obesity is derived. Our findings indicate that according to the IOTF (Orbegozo F.) tables the CI is -0.1034 (-0.0901) for the years 2003-04 while the CI is as high as -0.1599 (-0.1369) for 2006-2007.<sup>31</sup> As such, child obesity is pro-poor distributed and has risen by roughly 50% in just three years.

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<sup>31</sup> Following Wagstaff (2005) notice that the upper and lower bounds of the CIs of child obesity (a binary indicator) are 1-mean, mean -1, respectively.

**Table 5. Intergenerational Transmission by Gender and Sample Year (SNHS 2003-04 & 2006-07)**

	<b>Dependent variables: Child's Obesity (IOTF tables) and BMI</b>			
	<b>Years 2003-2004</b>		<b>Years 2006-2007</b>	
	<b>Boys Col. [1]</b>	<b>Girls Col. [2]</b>	<b>Boys Col. [3]</b>	<b>Girls Col. [4]</b>
Parent's BMI (OLS estimation on Child BMI)	0.100***	0.125***	0.123***	0.112***
Parent's obesity (Probit estimation on Child obesity)	0.284*** [0.049]	0.300*** [0.042]	0.327*** [0.060]	0.095 [0.013]
Income & Education	YES	YES	YES	YES
Mother's labour activity	YES	YES	YES	YES
Controls for children's charact.	YES	YES	YES	YES
Other controls for parents	YES	YES	YES	YES

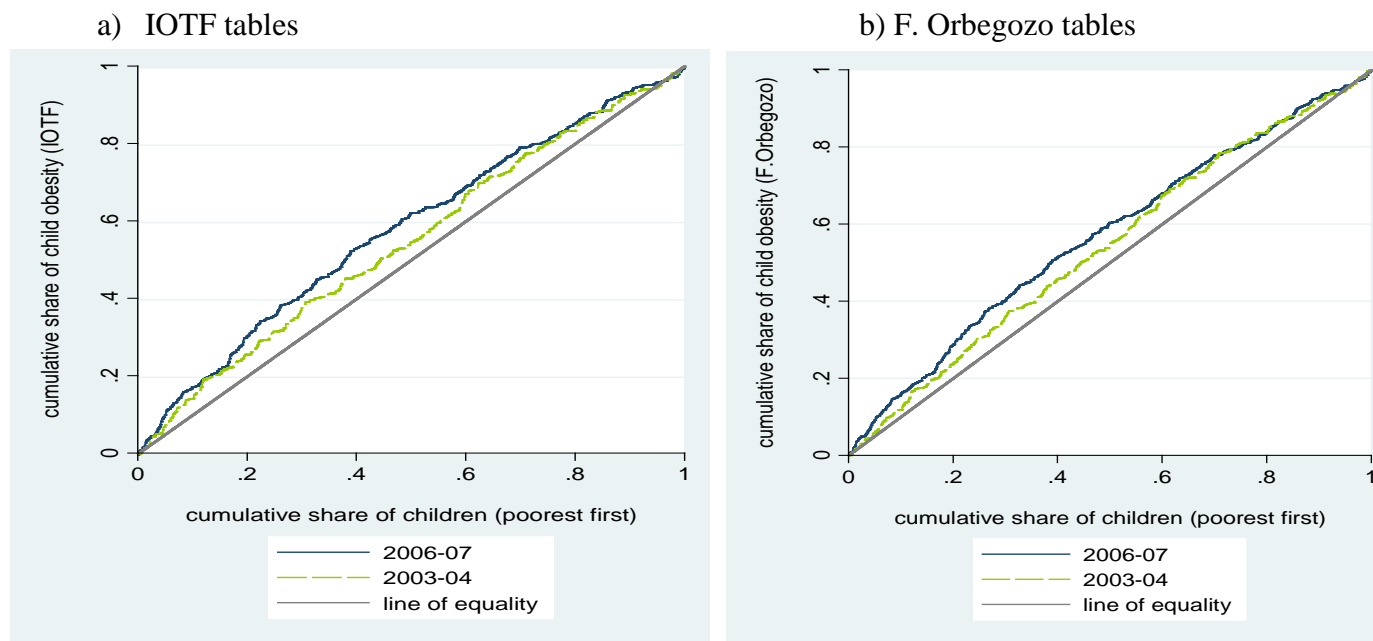
Note: Child obesity is calculated using the IOTF tables. Parent's obesity (BMI) corresponds to the obesity (BMI) of the father or mother. Standard errors are adjusted for 18 clusters in region. Estimations include a dummy for the sample year. Marginal effects are reported in brackets. \*\*\* P < 0.01, \*\* P < 0.05, \* P < 0.

**Table 6. Probit Estimation of Child obesity by Income Quartile and Sample Year**

<b>Dependent variable: Child Obesity (IOTF tables). Year 2003-04</b>				
	<b>Quartile 1</b>	<b>Quartile 2</b>	<b>Quartile 3</b>	<b>Quartile 4</b>
	<b>Col. [1]</b>	<b>Col. [2]</b>	<b>Col. [3]</b>	<b>Col. [4]</b>
Parent's obesity	0.240* [0.042]	0.382** [0.065]	0.449*** [0.082]	-0.101 [-0.003]
Income & Education	YES	YES	YES	YES
Mother's labour activity	YES	YES	YES	YES
Controls for children's charact.	YES	YES	YES	YES
Other controls for parents	YES	YES	YES	YES
<b>Dependent variable: Child Obesity (IOTF tables). Year 2006-07</b>				
Parent's obesity	0.281** [0.058]	0.199* [0.033]	-0.033 [-0.004]	0.547*** [0.084]
Income & Education	YES	YES	YES	YES
Mother's labour activity	YES	YES	YES	YES
Controls for children's charact.	YES	YES	YES	YES
Other controls for parents	YES	YES	YES	YES

Note: Child obesity is calculated using the International Obesity Task Force tables. Parent's obesity corresponds to the obesity of the father or mother. Standard errors are adjusted for 18 clusters in region. Estimations include a dummy for the sample year. Marginal and average effects are reported in brackets. \*\*\* P < 0.01, \*\* P < 0.05, \* P < 0.10.

**Figure 2. Concentration Curves of Child Obesity in Spain: SNHS 2003-2004 & 2006-2007**



**Table 7. Concentration Indices for Child Obesity: SNHS 2003-2004 & 2006-2007**

	IOTF tables			F. Orbegozo tables		
	CI	Std. Err.	t-value	CI	Std. Err.	t-value
Year 2003-2004	-0.1034	0.0511	-2.02	-0.0901	0.0470	-1.92
Year 2006-2007	-0.1599	0.0291	-5.49	-0.1369	0.0279	-4.90

Note: Standard errors corrected for sampling weights and regional clusters.

**Table 8. Decomposition of Concentration Indices for Child Obesity in Spain (IOTF tables)**

**Panel A: With Transmission**

	Survey Year 2003-2004				Survey Year 2006-2007			
	Elasticity	Concentration Index	Contr.	Percent	Elasticity	Concentration Indices	Contr.	Percent
Income	-1.3820	0.0493	-0.0681	65.9%	-2.5063	0.0459	-0.1150	72.3%
Mother's low education	0.0606	-0.3295	-0.0200	19.3%	0.0107	-0.3644	-0.0039	2.5%
Mother's high education	-0.0047	0.5266	-0.0025	2.4%	-0.0724	0.4761	-0.0345	21.7%
Father's low education	-0.0061	-0.3090	0.0019	-1.8%	0.0221	-0.3392	-0.0058	3.6%
Father's high education	0.0046	0.5542	-0.0025	2.5%	0.0328	0.5378	0.0181	-11.4%
Mother's labour activity	-0.0280	0.1377	-0.0039	3.7%	0.0664	0.1315	0.0085	-5.4%
Parent's obesity	0.0722	-0.1666	-0.0120	11.6%	0.0565	-0.1665	-0.0094	5.9%
Controls			0.0191	-18.5%			-0.0098	6.2%
"Residual"			-0.0154	14.9%			-0.0074	4.7%
Total inequality			-0.1034				-0.1592	

**Panel B: No Transmission**

	Survey Year 2003-2004				Survey Year 2006-2007			
	Elasticity	Concentration Index	Contr.	Percent	Elasticity	Concentration Index	Contr.	Percent
Income	-1.5046	0.0493	-0.0741	71.7%	-2.6441	0.0459	-0.1214	76.3%
Mother's low education	0.0649	-0.3295	-0.0214	20.7%	0.0144	-0.3644	-0.0052	3.3%
Mother's high education	-0.0053	0.5266	-0.0028	2.7%	-0.0743	0.4761	-0.0354	22.2%
Father's low education	-0.0001	-0.3090	0.0000	-0.03%	0.0221	-0.3392	-0.0075	4.7%
Father's high education	0.0057	0.5542	-0.0032	3.1%	0.0328	0.5378	0.0176	-11.1%
Mother's labour activity	-0.0400	0.1377	-0.0055	5.3%	0.0664	0.1315	0.0087	-5.5%
Controls			0.0190	-18.4%			-0.002	5.8%
"Residual"			-0.0155	15.0%			-0.0069	4.3%
Total inequality			-0.1034				-0.1592	

Finally, in Table 8 we report the decomposition of the inequality indices of child obesity into their explanatory factors (under the IOTF tables). In panel A (B) we include (exclude) parental obesity or our proxy of the intergenerational transmission of life styles. The entries in each column are derived from equation (7) and give the elasticity of child obesity and the concentration index for each regressor and the total and proportional contribution of each factor to the child obesity concentration index. The results provide evidence of the existence of a large pure income effect explaining between 72-66% of these income inequality estimates, even when the intergenerational transmission of life styles is accounted for. Interestingly, parental obesity explains between 6-12% of the income-related inequality.

## **6. Conclusion**

We have examined the existence of a socioeconomic and an intergenerational gradient of child obesity in a country that ranks among the highest in terms of this medical condition and which has experienced a significant increase in child obesity (especially between 2003 and 2006). This rise in obesity has occurred most intensively among pre-school children, though little is known about its potential causes. School meals are increasingly being ruled out as a possible source of intervention, and instead household related variables (e.g., having breakfast) are today deemed more appropriate (Millimet et al. 2008), as attention has become focused on patterns of intergenerational transmission. This paper offers a broad explanation of childhood obesity based on the failure of parents to cater for their children's health either owing to conflicts with work and the opportunity costs of parenting, or because of the intergenerational transmission of attitudes towards food and possibly lifestyles. We find that accounting for parents' transmission of childhood obesity exerts a difference in the development of the condition as well as in the magnitude of the gradient. Furthermore, we contribute to the debate on the impact of maternal employment on child obesity and we add to the discussion on the link between health in early childhood and health, education and earnings later in life (Currie and Madrian 1999, Case et al. 2005).

Our findings are consistent with an intergenerational transmission of child obesity that operates both through genes (nature) and the shared family environment (nurture). This transmission is particularly evident in boys for whom we find an increase in the



intergenerational gradient. Our estimates are robust to different methodologies of measuring obesity in children and account for time, gender and income variability.

Unlike a number of earlier studies, we find that after controlling for intergenerational transmission, a clear socioeconomic gradient appears to drive obesity in children. Furthermore, we find that female labour market participation does not necessarily increase the probability of child obesity; only in the case of women, with a secondary education, whose salary is not sufficient to devote more time to their children or high enough to pay for child care, this effect is significant, but only in relation to the degree of obesity and not to the probability of child obesity. A further important finding that may well account for both a higher propensity and a greater degree of child obesity is that of the mother's age effect. This result would seem to stem from the fact that older mothers have less energy to provide the care and exercise that their children naturally require.

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