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The intergenerational transmission of body mass index across countries

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The Intergenerational Transmission of Body Mass Index across Countries

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Abstract

There is a worldwide epidemic of obesity. We are just beginning to understand its consequences for child obesity. This paper addresses one important component of the crisis; namely the extent to which adiposity, or more specifically, BMI, is passed down from one generation to the next. We find that the intergenerational elasticity of BMI is very similar across countries and relatively constant – at 0.2 per parent. Our substantive finding is that this elasticity is very comparable across time and countries – even if these countries are at very different stages of economic development. Quantile analysis suggests that this intergenerational transmission mechanism is substantively different across the distribution of children's BMI; more than double for the most obese children what it is for the thinnest children. These findings have important consequences for the health of the world's children[1].

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1. Introduction

The epidemic of obesity has become one of the foremost major public health problems in most countries. We examine one important component of the crisis; namely the extent to which adiposity, or more specifically, Body Mass Index (BMI), is intergenerational; i.e. the degree to which it is passed down from one generation to the next. Explicitly we use data on the heights and weights of approximately 100,000 children and their parents, measured by health care professionals from across 6 countries: the UK, USA, China, Indonesia, Spain and Mexico. Our analysis applies to all ages of children up to 18 years and in all countries, from the most to the least developed, and with the most (USA) to least (Indonesia) obese population. We find that the elasticity of intergenerational transmission of BMI is approximately constant – at around 0.2 per parent.

In 2013, the US spent $190 billion on obesity-related health expenses. The US is by no means alone in experiencing this epidemic. Countries like Mexico, the UK and other European countries are all alarmed by the upward trend in obesity from the epidemiological evidence. It is also the case that many developing countries are experiencing a huge rise in the fraction of children who are becoming obese, inside literally, one generation. Countries like China and Indonesia are our relevant comparators. We are only slowly beginning to understand the causes and consequences of childhood obesity. This paper addresses the intergenerational transmission component of this process by examining how childhood BMI is related to the BMI of their parents.

Hence, our central underlying concern is to examine one of the principal mechanisms behind rising childhood obesity. BMI is a result of both the biological process of genetic inheritance and a consequence of decisions made inside families – loosely termed the 'family environment'. Most clearly, the family decisions relating to what to eat, how much to eat, how much exercise to take, how to spend family time, and other key lifestyle choices will all have a bearing on the anthropometric outcomes of individuals in the family. But, to what extent is an individual's BMI passed down to them through their parents and their genetic legacy? This is our central concern.

Our second focus is to pose the question of whether the process of intergenerational transmission of BMI is the same across countries, irrespective of their stage of development, degree of industrialisation, or type of economy. The motivation here is to understand the extent to which the process driving intergenerational transmission is related to the type of economy

and society under consideration. To this end we sought to examine data from literally all the countries from which we could retrieve a reasonably sized sample with the appropriate information. This is a considerable undertaking as there are not many datasets in the world where we have both children's and parents heights and weights; preferably on more than one occasion, which are medically measured rather than self-reported. We were able to obtain data from diverse countries; from one of the most obese populations, the USA, to some of the least obese countries in the world, namely China and Indonesia. Importantly, our paper presents rare findings on how obesity is transmitted across generations in both developed and developing countries.

Our third line of investigation is to explore the heterogeneity of the relationship between a parental and child BMI at different points in the distribution of a child's BMI. In other words, to what extent is the intergenerational mechanism the same for obese children and thin children? One could easily hypothesise that the relationship varies different at different points in the distribution. Specifically, if we see societies getting more obese, then we need to know whether the more obese children are more likely to have obese parents or not, and to what degree these sudden changes in rates of obesity may be driven by 'within generation' experiences. To what extent do decisions taken by this young generation, as they are growing up, relate to their parents. Our findings show how the effect of parents' BMI on their children's BMI depends on what the BMI of the child is. Consistently, across all populations studied, we find it to be lowest for the thinnest children and highest for the most obese. The intergenerational elasticity of BMI (henceforth IBE[2]) transmission for the former is 0.1 per parent and in the latter, 0.3 per parent. As a consequence, we can say that the children of obese parents are much more likely to be obese themselves when they grow up. These findings could have far reaching consequences for the health of the world's children.

To understand the process of obesity it is crucial to understand the intergenerational transmission mechanism behind it. Evidence suggests that BMI is affected by both environmental and genetic factors (Wardle et al., 2008). Clearly, the intergenerational transmission mechanism we are studying operates through both these two channels. So it is transmitted through family environmental factors, which directly relates to the intra-household mechanism (how the resources are allocated within the family), and it is also affected by genetic

[2] In this paper IIE refers to the intergenerational elasticity of income; IEE refers to the intergenerational elasticity of education and IBE refers to the intergenerational elasticity of BMI.

factors through a direct channel. Therefore, through exploring the elasticity of BMI across generations in different countries, we attempt to reveal the underlying intergenerational relationship in anthropometric characteristics.

In order to provide some basic perspective of the underlying relationship between parents and child's BMI, we first of all present some basic non-parametric graphs of the aggregate data, with a kernel plot based on the raw data. Figure 1a below is the local weighted scatter (Lowess plot) smoothing of the log of father's BMI variable against the log of their child's BMI variable. The height of the line is consistent with the most developed countries being at the top and the least developed countries being at the bottom of the figure. This is naturally because the Western countries, whose populations typically have larger, more obese body types, are above the less developed countries whose populations have thinner, smaller frames. This is unsurprising and what we would, of course, expect. Figure 1b is the corresponding figure for the relationship between the child and their mother. The other thing we would expect is that some of the country profiles start much further along the x-axis than others – for example, Indonesia and China – simply because there are relatively few obese children with low BMIs in these countries.

But the most important thing to notice is our central finding in this research; namely that the lines for each country are, for the most part, parallel. This suggests that the elasticity – here the slope of the line in log-log space – is essentially a very similar number in each country, as shown by the similar gradients on these lines. This is a quite striking result; which is the main motivation of our research. This finding suggests that the intergenerational elasticity is relatively high and approximately constant across countries, i.e. that the underlying gradient of the relationship between adiposity across generations is relatively constant and that the stage of development of the country only shifts up the intercept; with the least developed countries having the lowest intercepts and the most developed countries having the highest intercepts. In simple terms, this research presents, the substantive, hitherto unreported finding, that the proportionate increase in a child's BMI which is associated with their parent's BMI, is approximately constant at around .2 across countries and populations which are substantively different in epidemiological terms. This suggests that literally a unit increase in an adult's BMI will have an overall, 20% effect, on their child at the mean. Also this impact is, in practice, nearly doubled when we consider the effect of both parents.

Figure 1a Lowess Plot of Log (Father's BMI) and Log (Child's BMI)[3]

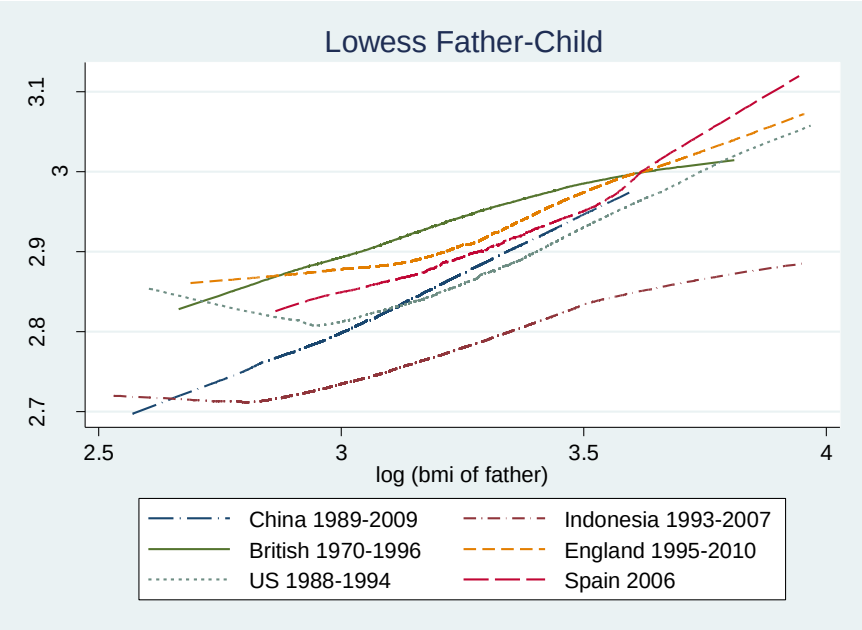
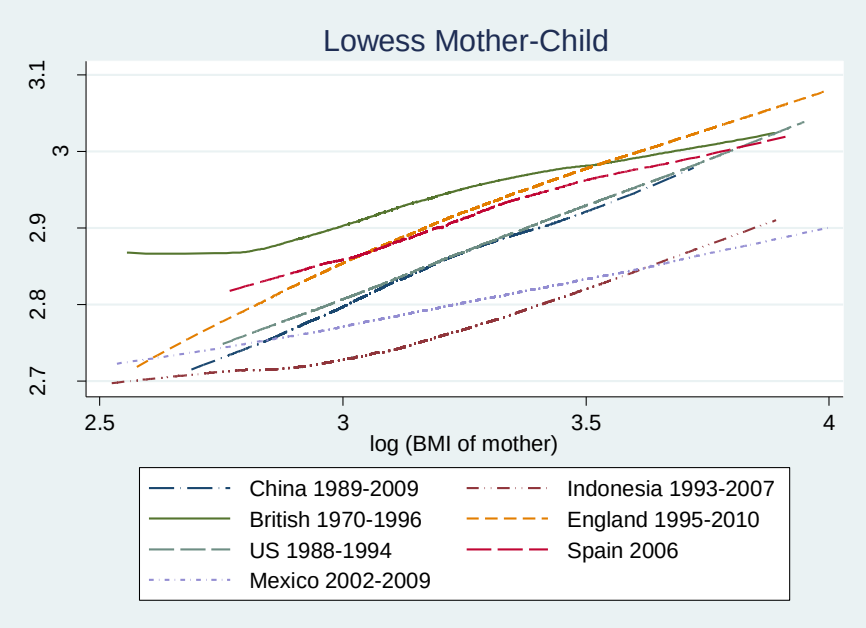


Figure 1b Lowess Plot of Log (Mother's BMI) and Log (Child's BMI)



[3] We drop the observations with the log of BMI less than 2.5.

Our plot needs careful qualification though as unfortunately the country samples are taken in different years. Specifically the US sample ends in 1994 and the British cohort is sampled in 1996. Whereas, the data from China ends in 2009, that from Indonesia ends in 2007, and the data from the Health Survey of England ends in 2010. Therefore it is possible that the intercepts are partly determined by the timing of data collection as well as genuine inter-country differences.[4]

In public health terms our basic finding, of the approximately constant slope of these plots, is of substantive importance as it suggests that a substantial fraction of BMI transmission; and hence possibly the obesity problem itself, is directly related to the process of intergenerational transmission of health outcomes within families from mother and father to son and daughter. These phenomena deserve closer scrutiny and explanation.

2. Evidence on Intergenerational Transmission Mechanisms

Intergenerational studies originate with Francis Galton (1869). By postulating 'a regression' of the offspring's height on their parents' height, he argued that an individual's characteristics are correlated with those of their parents and at the same time "regress to mediocrity". More specifically, the individual characteristics such as height, are closer to the population mean than those of their parents (Galton, 1877). This finding was the basis of Becker-Tomes model (1986) of intergenerational human capital transmission (Goldberger, 1989; Han & Mulligan, 2001; Mulligan, 1999).

There is a growing literature on the intergenerational transmission of various health outcomes, such as birth weight (Currie and Moretti 2007, Yan 2015), self-rated health (Coneus and Spiess 2012, Thompson 2012), longevity (Trannoy et al. 2010), smoking behaviour (Loureiro et al. 2006) and height. These studies mostly find strong positive correlations across generations. In terms of adiposity and related measures[5], they show how parental health outcomes are strongly correlated with children's. For instance, using data from India, Subramanian et al. (2009) find strong links between maternal and child height. Likewise, based on data from the Washington

4 We may suggest that the line in Fig 1a and 1b for the US would be higher if the sample related to a more recent year than 1994.

5 A large proportion of the studies are published in the medical, biological or epidemiological journals.

State Intergenerational Cohort Study, Emanuel et al. (2004) find that there is a strong association of mother's height with daughters' height.

In terms of BMI, using data in the US, Canada (national sample), Quebec and Norway, Bouchard (1994) reports the parental-child correlations of BMI are 0.23, 0.20, 0.23 and 0.20, respectively. Based on large datasets of sibling births in the US and a within-family design, Yan (2015) finds that there is a strong correlation between maternal preconception overweight, and excessive gestational weight gain and the probability of having a high birth weight baby. Likewise, preconception underweight and inadequate gestational weight gain is significantly associated with a low birth weight. Using data from the National Longitudinal Survey of Youth 1979 (NLSY 1979) and the Young Adults of the NLSY79, Classen (2010) estimates the intergenerational transmission of BMI between children and (only) their mother when both generations are between the age of 16 and 24, he finds the intergenerational correlation is significant and around 0.35. Applying a similar strategy of matching parents and children at a similar life stage, Brown and Roberts (2013) use British data on mothers and their adolescent children aged 11 to 15 years, from the British Household Panel Survey, and find the overall intergenerational correlation of BMI is 0.25. In the context of developing countries, using the China Health and Nutrition longitudinal Survey (CHNS) (1989-2009), Eriksson, Pan, and Qin (2014) estimate the intergenerational transmission of health status, using height z-score and weight z-score as the health measure. They find a strong correlation between parents' health and their children's health after accounting for various parental socioeconomic factors (education and type of occupation), household characteristics (whether the household has a flush toilet) and health-care factors (the distance to the nearest health centre in the community). To correct for the unobserved heterogeneity, they use the age and gender adjusted average parents' BMI in parents' province as the instrument for parental BMI variable. Additionally, using decomposition analysis, they find the urban-rural differential in parental health explains 15-27% of the urban-rural disparity in child's health, in addition to the urban-rural differential in parental education and income, which also plays a major role.

Studies usually include a range of parental socioeconomic conditioning factors in the estimation, arguably this controls for part of the family "environmental" factors. Evidence suggests that socioeconomic factors such as education has a long run effect on obesity (Kim 2016). Based on data from the German Socio-Economic Panel (SOEP), Coneus and Spiess (2008) estimate the intergenerational relationship of both father and mother and children. In

addition to the pooled OLS estimation, they use fixed effects estimation and find that father's BMI has a significantly positive effect on child's BMI (with a coefficient of 0.57, the estimates of mother's BMI effects are not significant), while mother's obesity is strongly associated with child's obesity with a coefficient of 0.26. They claim their fixed effects estimates provide a causal estimate of the intergenerational transmission process, since fixed effects estimation allows them to condition out time-invariant unobserved heterogeneity. However, it is possible that fixed effects estimation mainly captures the effects of rather short term environmental factors shedding little light on the underlying transmission mechanism. In addition, in the German Socio-Economic Panel (SOEP), child's health outcomes are provided by the mother rather than medical professionals, and father and mother's health are self-reported, this might lead to measurement error which induces a bias in the estimates due. Using the National Health and Nutrition Examination Survey (NHANES) for 2003-2010, Cawley *et al*. (2015) find that overweight and obese respondents tend to underreport their weight, while underweight respondents tend to overreport their weight. This reporting error could cause upward bias in coefficient estimates. As Black *et al*. (2005) review, among the studies on intergenerational transmission of health, few have claimed a causal interpretation for their estimates, partly due to unobserved behavioural or environmental factors, which could affect the health outcome of both parents and children simultaneously.

One of the central issues in the inter-generational literature is the relative role, and the interaction of, environmental and genetic forces. Our hypothesis is that in the transmission of BMI, a smaller fraction of the process is open to manipulation (such as the diet changes within the household), and a larger fraction of the mechanism is driven by the "natural process". In other words, in the case of a health outcome such as the BMI, it is more likely to be inherited genetically regardless of any variation in the environment. If this hypothesis is true, our estimation for the IBE may provide a lower bound of the intergenerational correlation of any characteristics, including income and education. It is worth noting that in the extensive literature on the IEE, the lowest values are around .1-.2 in Scandavanian countries. This may imply that the intergenerational transmission mechanism elasticity between parents and child cannot be lower than this threshold for inherently biological reasons. Ths is likely even in the face of changes in either family environment (such as shifting of nutrition patterns) or socioeconomic environment (such as the innovation or marketing campaigns in the food industry).

In addition to "regression to the mean" in the intergenerational transmission of BMI, the degree of this inheritability (IBE) may vary across the child's BMI distribution and this variation may relate to family socioeconomic status. The general conclusion in the literature is: in either developed countries or developing countries, the intergenerational correlation in health measures tends to be stronger at lower SES levels (see, for example, Currie and Moretti, 2007; Bhalotra and Rawlings, 2013). In developing countries, this strong correlation emerges at the lower levels of BMI, whereas in developed countries, such as the US, this occurs at higher levels of BMI (Classen, 2010; Laitinen *et al.*, 2001; Scholder *et al.*, 2012). One possible explanation is that in these countries, as the fast food industry is more developed, these "unhealthy" foods are generally cheaper than "healthy" foods. In this context it is argued that lower income families tend to consume more of these "unhealthy" foods, and it is this mechanism which is an important contributory explanation of obesity.

3. Data and Measurement Issues

We use seven datasets from six countries: the China Health and Nutrition Survey (CHNS) data, the Indonesian Family Life Survey (IFLS) data, the British 1970 Cohort Studies (BCS1970), the Health Survey for England (HSE) data, the National Health and Nutrition Examination Survey (NHANES) data, the Spanish National Health Survey (ENS-2006) and the Survey for the Evaluation of Urban Households (ENCELURB) data in Mexico[6]. The heights and weights are mostly medically measured in these data[7]. Compared to self-reported measures, which are widely used in the literature, these data may help to reduce the bias of our estimates due to measurement error. Although the original sample includes children aged under five years old[8] we restrict our analysis to those aged above 5. [9]

The most widely used measure of body adiposity is the Body Mass Index (BMI) which is calculated using the following formula, $\text{BMI} = \left[\frac{\text{weight(kg)}}{\text{height}^2\text{(cm)}}\right] * 10{,}000$. The majority of intergenerational studies use an elasticity (i.e. the IIE and IEE) as a measure of the intergenerational relationship. To facilitate the comparison of our results on anthropometric

[6] See Data Appendix for a detailed description of these data.
[7] Except the Spanish National Health Survey (ENS-2006).
[8] The descriptive statistics of children's age are reported in Table A1.
[9] The BMI of children aged under five years old, their BMI is likely to be related to their birth weight.

data with other intergenerational results, we also use elasticity as the measure of the intergenerational relationship.

A problem we face is exactly how we correlate a child's BMI with their parent's BMI. A child's BMI is a non-linear function of their age and gender – so a simple correlation of child's BMI against parents BMI could be misleading. One way to examine the intergenerational transmission is to wait until the child is an adult and then correlate the two BMIs. This is what Classen (2010) did. There are two problems with this; firstly, there is very little data relating to a child's height and weight observed when they are adults – as well it being unlikely that we have their parents height and weight measured at the same age. Based on the children aged between 16 and 18 years old, we estimate the intergenerational BMI correlation[10]. The estimates for this correlation are slightly larger than the estimates based on the full sample[11]. The other problem with this is that we are mainly concerned with childhood obesity and so waiting until they are adults does not help us.

To address the potential bias due to the fact that a child's BMI is a function which varies with their age, we include child's age, age squared and the interaction term of child's age with their gender as controlling regressors in our estimation. By doing so we are able to condition out for the non-linear effect of age on gender[12]. We also take a more flexible approach by including child's age dummies and their interactions with child gender, the results are reported in Table A5, they suggest that the estimates are similar to those from the specification we focus on in this study. We use this method as a robustness check on our findings, although since it does not change the findings, we use the first method in each of our country datasets.

In the course of doing this research we considered if there was an alternative way of retrieving the IBE. We use the WHO standard to generate z scores or percentiles. Naturally, the estimation of the BMI elasticity is sensitive to any possible transformation of its scale. – i.e., to z scores or percentiles. So keeping the analysis simple has many virtues. It turns out that estimating the model in the log of BMI or the BMI itself does not make much difference – the marginal effect is slightly smaller when estimated without logging and so the IBE is routinely less than the IBC. But since taking logs allows – albeit crudely – for general non-linearity in

[10] We report the IBE regression results for children over 16 in Table A6.
[11] Another approach to obtain this correlation of "long-term" BMI might be to use the average of the observations in the data as the "long-term" BMI, but in that case we will lose a large number of observations.
[12] The weakness of this method is that we have to assume that we can net out for the whole non-linear process of the child's BMI rising as they age.

the data and has the nice property that it preserves the constant elasticity across the range of values of the BMI, we adopt it here. This means also that it forces the elasticity to be a constant; which has the virtue that its first derivative (and hence the elasticity) is constant across the whole range of the BMI[13]. We explore these issues more fully in Appendix B making clear the differences between these alternative specifications. We also show they make a limited (predictable) difference to the size of the metric in the results.

Before estimation, we plot the kernel density of child's BMI, father's BMI, mother's BMI across countries in Figures 2, 3 and 4, respectively. They show that in both generations, the distributions of BMI tend to shift rightwards as the development level of these countries increases, with Indonesian cohorts being the leanest and the UK cohorts (children in British 1970 cohorts and father in the Health Survey for England) being the most obese[14]. This is as expected as the nutrition status of population varies with the development of the nation (Floud et al., 2011). In addition, we see the distribution of child's BMI is more concentrated than the distribution of father and mother's BMI. This is consistent with the maturation process. Notwithstanding, there is considerable cross country heterogeneity. A case in poont is Mexico, where noticeably the mother's BMI distribution is predominantly to the right of other countries. This is consistent with the rise of obesity prevalence in Mexico during the survey period (Raymond, et al. 2006).

A further question prompted by these plots is the extent to which height, weight, and, as a result BMI, is influenced by ethnic type. Undoubtedly the answer to this question is – yes it is – but it is unclear what, if anything, we should do about standardising for ethnic or physiological 'body type' in computing BMI. This problem is not solved, and possibly exacerbated by the use of categorical labels like: underweight, normal, overweight and obese. In common with the rest of the literature we acknowledge this problem but suggest there is little we can do about it. One justification for this stance is that in many countries there is huge ethnic diversity anyway. The default position on this is to use WHO definitions which, for adults, are invariant across the world.

[13] We naturally relax this assumption in Section 5.3 when we consider the quantile regression allowing the elasticity to vary across the range of the child's BMI.

[14] Figure 4 suggests that Mexico has the largest fraction of obese mothers, this is consistent with the rise of obesity prevalence in Mexico during the survey period (Raymond, et al. 2006).

Figure 2: The kernel density of child's BMI

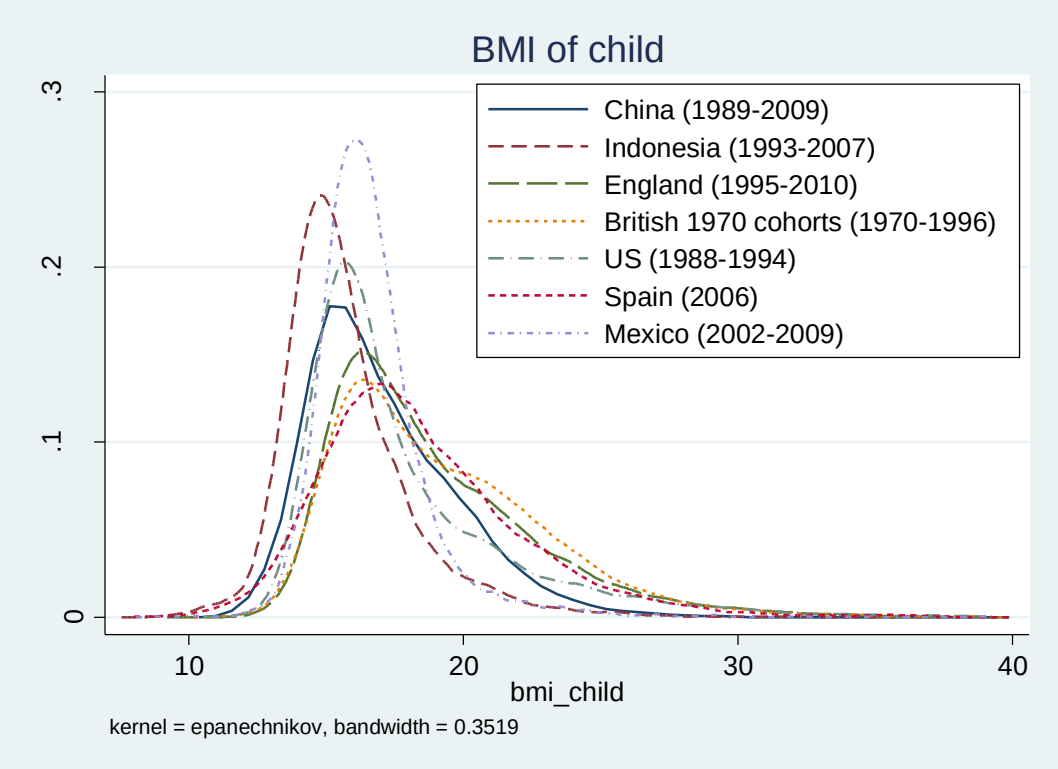


Figure 3: The kernel density of father's BMI

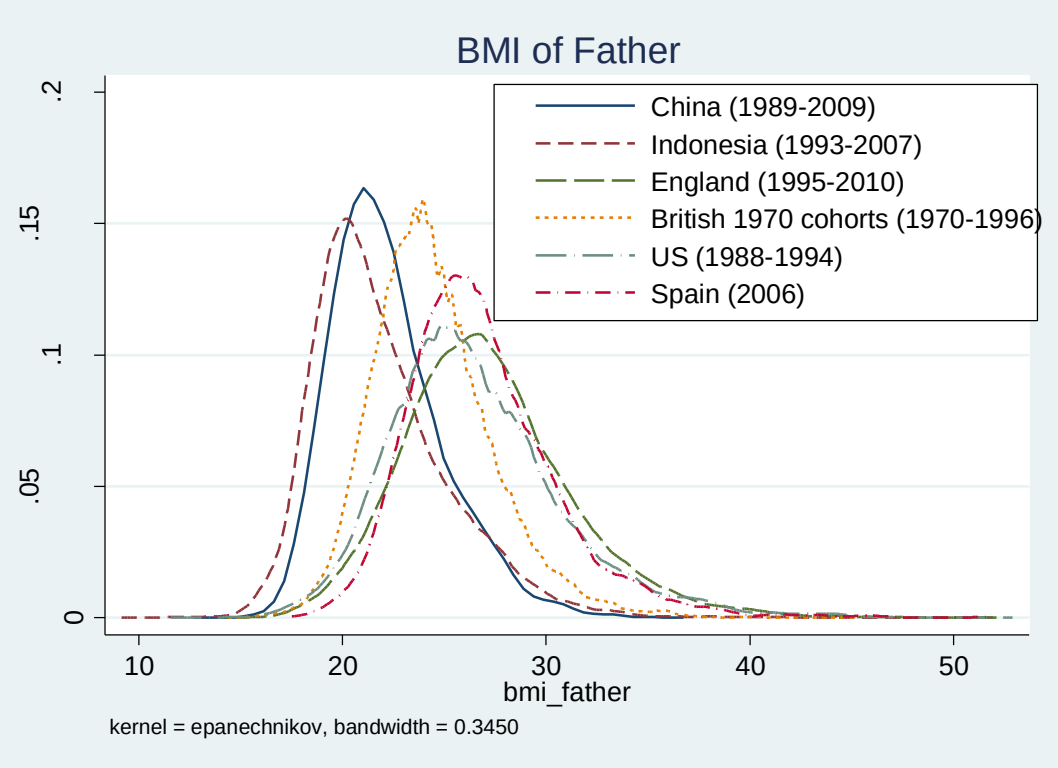
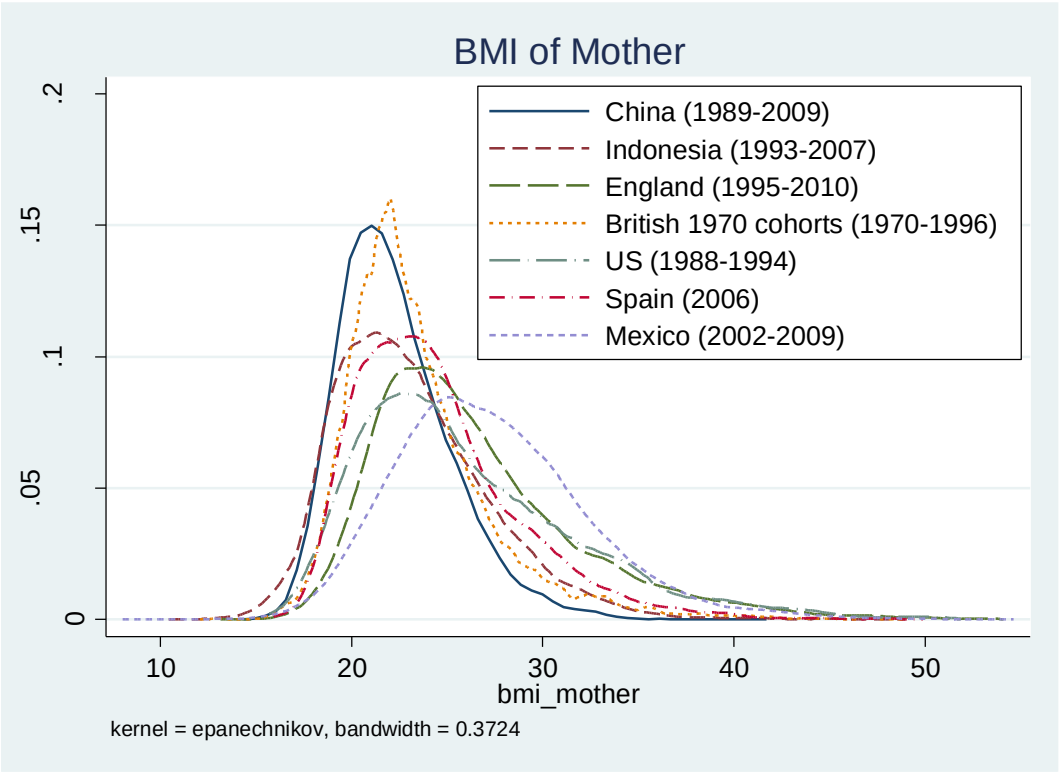


Figure 4: The kernel density of mother's BMI



For children the position is different. The standard approach is to take the child's BMI when they are young and use the WHO's program to compute the child's BMI *z score* which explicitly allows for both the child's age and gender. Once we have this *z-score* we can then ask what their BMI would be with such a *z score* when they are adults. The assumption that we have to make here is that the child would remain in the same position in the distribution when they are adult as when they are a child. Note that by doing this we are not, *de facto*, assuming that this is what will happen to that child when they are an adult – but rather simply getting an estimate of what adult BMI is consistent with a given *z score* for the child. Although this is a strong assumption there is only one other way to proceed. This would be to simply use the child's BMI (as it is- even if they are very young) as the dependent variable in a regression on the parent's BMI on the assumption that if we control for the child's age, gender, age squared and an interaction of the child with that of their gender then we will be able to condition out for the non-linear effect of age on gender[15]. We use both these methods as a robustness check. Fortunately they do not differ much in their estimated findings; with the

[15] The weakness of this method is that we have to assume that we can net out for the whole non-linear process of the child's BMI rising as they age. For some evidence from China on this see Dolton and Xiao (2015).

latter method giving lower variance in the tails than the former variable. We will therefore use the second method in each of our country datasets.

Before we present the estimation results for our double log transformation model it is important to examine the basic association between the child's and parent's BMI in raw terms. Table 1 shows the simple unconditional correlation of parents with child's BMI across countries. Whilst this correlation coefficient is not the same as an IBE, it does suggest that the magnitude of the intergenerational correlation in BMI is relatively limited in its range constant across countries from 0.122 to .245. These basic statistics suggest that, in terms of basic correlations, mothers appear to exert a larger effect on child's BMI than fathers. Of course these basic correlations do not control for any regressors like: child age and gender or non-linearities or interactions. We will investigate these influences in section 5 in the context of what has become the standard intergenerational model.

Table 1: Unconditional Correlation Coefficient of Father and Mother's BMI with Child's BMI

	China	Indonesia	UK		US
	CHNS (1989-2009)	IFLS (1993-2007)	BCS (1970-1996)	HSE (1995-2010)	NHANES 3 (1988-1994)
BMI of child					
BMI of father	0.232	0.172	0.122	0.187	0.190
BMI of mother	0.235	0.188	0.136	0.245	0.259
Observations	14,081	18,650	22,657	26,476	6,581

A further issue which we cannot address in our data is the extent to which the BMI of children and parents is moving over time. This is important as the elasticity (or marginal effect) might be affected by the possibility that the variance of the BMI of the child's generation might be larger than that of the parent's generation. For one of our datasets, namely the CNHS data we can look at this for the same cohort over time. In Table 2 we also present the variance of child's BMI by year. The reported figures suggest that the variance of father's, mother's and child's BMI all increased over time and broadly in step (at least in China).

Table 2: The variance of parents and child's BMI in China by Year.

	1989	1991	1993	1997	2000	2004	2006	2009
The variance of child's BMI	4.406	6.211	6.473	7.241	8.211	8.623	9.242	10.658
The variance of father's BMI	4.050	5.671	5.527	6.788	8.665	9.546	9.588	11.955
The variance of mother's BMI	5.675	7.177	7.631	8.208	9.637	10.375	9.338	11.622

4. Empirical Evidence of Intergenerational Transmission

4.1. OLS Estimation

The basic equation we seek to estimate relates child BMI, y_c , to parental BMI, y_p.

$$\log y_c = \alpha + \beta \log y_p + u \qquad (1)$$

This relationship is typically estimated in logs and controls for an array of conditioning covariates. This relationship will typically include regressors for both parents and allow for gender and age effects.

We restricted our sample to those aged above five and estimate the both-parents version of the above equation. Our main results by country are presented in Tables 3a to 3c. The results for all countries pooled together are presented in Table A4. They suggest that the estimates for intergenerational IBC appear larger than those for the IBE based on the full sample. Appendix B discusses the sensitivity of the different possible marginal effects estimates to different possible model specifications.

We estimate the IBE using the data sets listed in section 3. We provide more details of these data sets in the Data Appendix. We regress the log of child's BMI on the log of parents BMI controlling for Child's Age, Child's Age Squared, Child's Gender and Child's Age interacted with Child's gender. In each of these datasets we are, of course, able to control for many different family and parental covariates – but the available covariates are different for each country. We did estimate these models – but here we wanted to focus on a directly comparable equation specification which had the same form in each country. This meant that we had to drop various variables which were not in each dataset as we estimated the 'lowest common denominator' model. Our results – in terms of the sign and size of our main estimated parameter – the IBE – did not change appreciably – no matter what specification we adopted in each country separately when additional regressors were available. So here we focus only

on the estimation results we can get for every country – in order that we can directly compare them.

It is clear from all our tables – that most of our additional control variables are all significant with the logical and consistent relative size and signs of the coefficients. This is reassuring and means we can focus our attention on the parameter of interest, the IBE, with some confidence that the underlying relationship we have specified, is a reasonable way to approach this estimation problem. Prior to considering the regression results from each country separately we would like to draw attention to our overall benchmark estimates reported in Table A4 in the Appendix. These estimates, of an IBE of .2 for father-child, and .182 for mother-child are the overall estimates derived from all of our combined cross country data. Since the dummy variables for each country are statistically significant (and the interactions by country are mostly not) then strictly speaking we do not need to estimate our model separately, by country. But we wish to examine the extent to which the IBE may differ by country and how they each, in turn, compare to this benchmark estimate of 0.2. Table 3a reports the results on IBE when the equation controls for father's BMI variable alone. It suggests that the father-child IBE estimates range from 0.164 in Indonesian sample, to 0.247 in Chinese sample, and they do not vary substantially across countries. This finding is in sharp contrast to the previous studies on IIE that we referred to earlier. For the UK, The IBE estimate on BCS sample (0.211) is close to that from HSE sample (0.198)[16]. These results suggest that the responsiveness of child's BMI variable to parents' BMI variable is around 0.20 and the extent of this "inheritability" is relatively constant across countries, and this seems to be regardless of the general state of economic development in the country. In a similar way, Table 3b presents mother-child IBE estimates from these samples, and we see a similar pattern to those in Table 3a which reported the father-child IBE estimates. In addition, comparing Table 3a and 3b, we can see that in general, the father-child IBEs are larger than mother-child IBE estimates.

Next, we incorporate both father and mother's BMI variables ($log(BMI_{fi})$ and $log(BMI_{mi})$) into the equation, and the results are reported in Table 3c. As we expect, once we control for both father and mother's BMI variables, the sizes of paternal and maternal BMI effects shrink significantly compared with Table 3a and Table 3b, with a slight dominance of father's BMI effects – at least in the CNHS in China and the BCS in Britain.

[16] Notice the HSE was collected from 1995 to 2010, and the BCS 1970 survey tracks the cohorts born in 1970 until they reached 26 years (1996).

One important caveat that must be explained for the interpretation of our results is that the data we have available all comes from different time periods in the different countries. Some of the data is fairly recent – so for example from China our last wave of data is from 2009. In contrast our data from the US – from NHANES is fairly old – it is from 1988. This means that in many respects true cross country comparisons should be tempered by this limitation. This aspect of our results should be factored into any relevant assessment. At the same time this feature of our results is also an advantage in demonstrating that our relatively constant estimate of the IBE is applicable not only across countries but also over time.

One matter of concern to us is the extent to which the estimation of the IBE is dependent on the age of the child being modeled. We sought to look at the robustness of our results to children of different ages. Our results are presented in Appendix A, Tables A3 and A6. We tend to find larger estimates of the IBE for children of younger ages. This might be due to a larger fraction of "environmental factors" shared between parents and children when children are aged above five, than for those aged under five, since children aged under five might have a different dietary pattern from their parents. In addition, children aged 16 and above might have already left the household and the decision to leave may itself be related to the health or BMI of the child. Therefore, we restrict the sample to those aged between 5 and 16, and estimate the both-parents version of equation (1). The estimates are presented in Table A3 and they are close to those based on children aged above five (Table A2). This is reassuring since it suggests that our estimates are not very different as a result of reducing the sample to take account of the fact that older children might have left the family.

Table 3a Intergenerational BMI regressions for Father and Child across countries.

	China	Indonesia	UK		US	Spain
	CHNS (1989-2009)	IFLS (1993-2007)	BCS (1970-1996)	HSE (1995-2010)	NHANES 3 (1988-1994)	ENS-2006 (2006)
Dependent variable: Log(BMI of child)						
Log (BMI of father)	0.247***	0.167***	0.211***	0.203***	0.192***	0.212***
	(0.0117)	(0.00769)	(0.00924)	(0.00704)	(0.0125)	(0.0313)
Age of Child	-0.0374***	-0.0377***	-0.0156***	-0.000328	-0.00190	-0.0100*
	(0.00140)	(0.00129)	(0.000782)	(0.00139)	(0.00285)	(0.00538)
$(\text{Age of Child})^2$	0.00286***	0.00342***	0.00173***	0.00137***	0.00183***	0.00135***
	(7.57e-05)	(8.71e-05)	(3.30e-05)	(7.56e-05)	(0.000174)	(0.000312)
Male Child	0.00835	0.0244***	0.0451***	0.0391***	0.0255**	0.00849
	(0.00732)	(0.00588)	(0.00599)	(0.00704)	(0.0113)	(0.0270)
Male*Age of Child	0.00367**	0.00228	-0.0148***	-0.00968***	-0.00624	-0.00560
	(0.00187)	(0.00178)	(0.00106)	(0.00190)	(0.00394)	(0.00744)
$\text{Male*(Age of Child)}^2$	-0.000317***	-0.000546***	0.000776***	0.000316***	0.000132	0.000489
	(0.000101)	(0.000119)	(4.61e-05)	(0.000103)	(0.000245)	(0.000428)
Constant	2.096***	2.267***	2.149***	2.105***	2.126***	2.161***
	(0.0360)	(0.0239)	(0.0298)	(0.0236)	(0.0415)	(0.104)
Observations	14,081	18,650	21,512	26,476	6,581	2,139
R-squared	0.335	0.207	0.540	0.419	0.423	0.142

Robust standard errors in parentheses *** p<0.01, ** p<0.05, * p<0.1

Table 3b Intergenerational BMI regressions for Mother and Child across countries.

	China	Indonesia	UK		US	Spain	Mexico
	CHNS (1989-2009)	IFLS (1993-2007)	BCS (1970-1996)	HSE (1995-2010)	NHANES 3 (1988-1994)	ENS-2006 (2006)	ENCELURB (2002-2009)
Dependent variable: Log(BMI of child)							
Log(BMI of mother)	0.215***	0.155***	0.184***	0.201***	0.177***	0.171***	0.117***
	(0.0110)	(0.00640)	(0.00746)	(0.00586)	(0.00962)	(0.0189)	(0.00734)
Age of Child	-0.0365***	-0.0383***	-0.0156***	-0.000724	-0.00275	-0.00565	-0.0347***
	(0.00139)	(0.00130)	(0.000770)	(0.00137)	(0.00286)	(0.00359)	(0.00273)
(Age of Child)^2	0.00281***	0.00343***	0.00173***	0.00137***	0.00184***	0.00131***	0.00399***
	(7.55e-05)	(8.71e-05)	(3.24e-05)	(7.44e-05)	(0.000175)	(0.000213)	(0.000270)
Male Child	0.00990	0.0272***	0.0490***	0.0407***	0.0243**	0.0287	0.0115*
	(0.00729)	(0.00588)	(0.00598)	(0.00695)	(0.0114)	(0.0179)	(0.00688)
Male*Age of Child	0.00402**	0.00150	-0.0156***	-0.00999***	-0.00589	-0.00212	0.00269
	(0.00187)	(0.00179)	(0.00105)	(0.00188)	(0.00393)	(0.00517)	(0.00386)
Male*(Age of Child)^2	-0.000345***	-0.000507***	0.000805***	0.000332***	0.000110	2.98e-05	-0.000242
	(0.000101)	(0.000119)	(4.55e-05)	(0.000102)	(0.000244)	(0.000309)	(0.000381)
Constant	2.191***	2.299***	2.244***	2.121***	2.186***	2.280***	2.458***
	(0.0342)	(0.0203)	(0.0238)	(0.0198)	(0.0321)	(0.0617)	(0.0244)
Observations	14,081	18,650	22,657	26,476	6,581	3,418	7,405
R-squared	0.329	0.210	0.545	0.435	0.435	0.164	0.099

Note: Spain uses the following, since only have “father-child” or “ mother-child”.

Robust standard errors in parentheses *** p<0.01, ** p<0.05, * p<0.1

Table 3c Intergenerational BMI regressions for Mother and Father and Child across countries.

	China	Indonesia	UK		US
	CHNS (1989-2009)	IFLS (1993-2007)	BCS (1970-1996)	HSE (1995-2010)	NHANES 3 (1988-1994)
Dependent variable: Log(BMI of child)					
Log (BMI of father)	0.211***	0.130***	0.178***	0.164***	0.149***
	(0.0116)	(0.00772)	(0.00914)	(0.00678)	(0.0124)
Log(BMI of mother)	0.176***	0.126***	0.162***	0.179***	0.153***
	(0.0108)	(0.00644)	(0.00764)	(0.00575)	(0.00972)
Age of Child	-0.0377***	-0.0386***	-0.0155***	-0.00100	-0.00306
	(0.00138)	(0.00129)	(0.000789)	(0.00136)	(0.00281)
(Age of Child)^2	0.00284***	0.00345***	0.00173***	0.00138***	0.00185***
	(7.47e-05)	(8.65e-05)	(3.31e-05)	(7.36e-05)	(0.000172)
Male Child	0.00986	0.0256***	0.0445***	0.0410***	0.0260**
	(0.00728)	(0.00589)	(0.00612)	(0.00693)	(0.0112)
Male*Age of Child	0.00369**	0.00190	-0.0149***	-0.0102***	-0.00630
	(0.00185)	(0.00178)	(0.00107)	(0.00186)	(0.00386)
Male*(Age of Child)^2	-0.000321***	-0.000529***	0.000784***	0.000339***	0.000133
	(9.98e-05)	(0.000119)	(4.64e-05)	(0.000100)	(0.000239)
Constant	1.667***	1.990***	1.746***	1.656***	1.778***
	(0.0453)	(0.0276)	(0.0353)	(0.0280)	(0.0462)
Observations	14,081	18,650	21,253	26,476	6,581
R-squared	0.356	0.226	0.556	0.452	0.449

Robust standard errors in parentheses *** p<0.01, ** p<0.05, * p<0.1

A further matter of concern to us is the extent to which we are justified in our assumption that mother's and father's BMI each have an additively separable effect on child's BMI. Specifically it is possible that the effect of the parents is interactive and hence non-linear and multiplicative. The argument here is that there is potential assortative mating (Mare 1991, Kalmijn 1994) between father and mother. Further, the subsequent sharing of a household environment and common nutrition regime may re-enforce the effects of fathers and mothers who have similar BMI status. For instance, having an overweight father and an overweight mother may generate an interaction effect which is greater than the sum of these two terms together. In this case we would expect that an equation which included both parents may have a coefficient on the IBE for either parent which is somewhat attenuated. On the other hand, if the father is overweight whereas the mother is normally weighted or underweighted, the interaction effects may depend on the role of them in this family (such as who is in charge of the food preparation or allocation) and their bargaining power within the household (Pollak 2005). (We report the simple regression of mother's on father's BMI in our data in Table 4.) When we tested this in the data by introducing simple interactions of male and female log BMI, the nature of the nonlinearity of the multiplication of two log values gave understandably strange results. Running the regression without taking logs destroys the elasticity interpretation we seek to use. Hence our solution is to use two dummy variables which relate to having both an underweight father and mother or both an overweight father and mother. We report these results in column 4 of Table A4 for the pooled data. For the most part we do not find large interaction effects when we examine countries individually (not reported but available on request) – although there is a small positive effect of having both an overweight mother and an overweight father on child's BMI in the UK and the US and a small positive effect on child's BMI of having an underweight mother and an underweight father in Indonesia. The former finding is consistent with extreme overweight families in Western countries having an even more overweight child. The latter finding is consistent with regression to the mean in Indonesia. Including an interaction term does not detract from the size or significance of the IBE terms of main interest to us. This indicates that there is some evidence for an independent role for the interaction effect in the intergenerational transmission. However, this "assortative mating" of father and mother, with its " reinforcing" effect on the BMI development of the child, does not detract from the underlying IBE estimate of the effect of each parent on the child.

Table 4: Assortative mating: the association between father and mother's BMI

	China CHNS (1989-2009)	Indonesia IFLS (1993-2007)	UK BCS (1970-1996)	HSE (1995-2010)	US NHANES 3 (1988-1994)
Dependent variable: Log (BMI of father)					
Log (BMI of mother)	0.199***	0.223***	0.132***	0.138***	0.166***
	(0.0112)	(0.00744)	(0.00804)	(0.00660)	(0.0100)
Constant	478***	378***	776***	841***	727***
	(0.0346)	(0.0231)	(0.0252)	(0.0214)	(0.0322)
Observations	14,081	18,650	37,197	26,476	6,581
R-squared	0.044	0.065	0.027	0.030	0.048

Robust standard errors in parentheses, *** p<0.01, ** p<0.05, * p<0.1

The biological literature suggests that health transmits across gender-specific lines (Pembrey *et al.*, 2006). Next we estimate the elasticities by gender, we compare the mother-daughter and father-son relationship, the results are presented in Table 5. These results are somewhat mixed and do not provide conclusive evidence of the mother-daughter and father-son correlations being stronger – nor is it uniformly the case that the mother-child correlation is higher than the father-child correlation.

A further question of interest is whether children keep the same position in their own cohort's distribution of BMI as their parents did in their own BMI distribution. We are concerned with this as children might not "keep" their position in the BMI distribution as they grow up. Also, since the distribution of BMI changes over time, this may be happening differently by generation. To examine this for each child we compute their percentile position in their own country distribution, similarly, we compute father and mother's BMI percentiles in their own country distribution. We then regress child's BMI percentile on both parents' BMI percentile, the results are reported in Table 6. These results suggest that children do keep the position in the distribution that their parents had – but that the correlation effect is not as high as the IBE effect.

Table 5: The intergenerational transmission of BMI by gender

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
VARIABLES	Log(BMI of child)									
	China		Indonesia		British		England		US	
	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls
Log (BMI of father)	0.220***	0.200***	0.128***	0.132***	0.162***	0.193***	0.165***	0.163***	0.157***	0.142***
	(0.017)	(0.016)	(0.011)	(0.011)	(0.012)	(0.014)	(0.009)	(0.010)	(0.017)	(0.018)
Log(BMI of mother)	0.178***	0.173***	0.121***	0.132***	0.126***	0.193***	0.163***	0.195***	0.162***	0.145***
	(0.015)	(0.015)	(0.009)	(0.009)	(0.011)	(0.011)	(0.007)	(0.008)	(0.014)	(0.014)
Age of Child	-0.034***	-0.038***	-0.037***	-0.039***	-0.031***	-0.016***	-0.011***	-0.001	-0.010***	-0.003
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.003)	(0.003)
(Age of Child)^2	0.003***	0.003***	0.003***	0.004***	0.003***	0.002***	0.002***	0.001***	0.002***	0.002***
	(6.60e-05)	(7.46e-05)	(8.13e-05)	(8.64e-05)	(3.24e-05)	(3.32e-05)	(6.89e-05)	(7.36e-05)	(0.000)	(0.000)
Constant	1.640***	1.709***	2.038***	1.966***	1.953***	1.602***	1.743***	1.607***	1.751***	1.828***
	(0.065)	(0.062)	(0.039)	(0.039)	(0.047)	(0.0513)	(0.0381)	(0.0382)	(0.064)	(0.066)
Observations	7,524	6,557	9,582	9,068	10,118	11,135	13,508	12,968	3,198	3,383
R-squared	0.333	0.379	0.182	0.266	0.591	0.521	0.435	0.465	0.430	0.461

Table 6: The intergenerational transmission of BMI percentile (within each country)

	(1)	(2)	(3)	(4)	(5)
	China	Indonesia	England	Britain	US
Variables: Child's BMI percentile					
Father's BMI	0.159***	0.128***	0.124***	0.105***	0.127***
percentile	(0.00842)	(0.00705)	(0.00526)	(0.00543)	(0.00968)
Mother's BMI	0.142***	0.141***	0.158***	0.119***	0.156***
percentile	(0.00832)	(0.00695)	(0.00527)	(0.00551)	(0.00989)
Age of Child	-7.498***	-8.511***	0.475**	-2.317***	-0.454
	(0.258)	(0.245)	(0.214)	(0.133)	(0.422)
(Age of Child)^2	0.564***	0.731***	0.189***	0.264***	0.261***
	(0.0137)	(0.0156)	(0.0109)	(0.00532)	(0.0236)
Male Child	2.949**	6.553***	7.500***	7.315***	4.724**
	(1.396)	(1.140)	(1.201)	(1.067)	(1.937)
Male*Age of Child	0.505	-0.227	-1.945***	-2.418***	-1.074*
	(0.347)	(0.337)	(0.296)	(0.182)	(0.582)
Male*(Age of	-0.0536***	-0.0537**	0.0777***	0.125***	0.0377
Child)^2	(0.0183)	(0.0216)	(0.0150)	(0.00739)	(0.0328)
Constant	42.93***	46.66***	10.67***	21.11***	21.25***
	(1.131)	(0.924)	(0.909)	(0.854)	(1.493)
Observations	14,081	18,650	26,476	21,253	6,581
R-squared	0.373	0.245	0.470	0.565	0.410

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

A large body of the literature looks at the intergenerational transmission of height, to compare the intergenerational correlation of weight, we also examine the correlation between parents and child's height and the results are presented in Table A8.

4.2 Quantile Estimation

Thus far, the estimates for IBE we have reported are at the conditional mean of child's BMI variable. In order to explore the variation of IBE across different quantiles of the child's BMI variable (or whether the association of mother or father's BMI with the child's BMI is constant across the child's BMI distribution), we estimate the quantile elasticities of BMI between father and child at different points in the distribution of child's BMI, using the 'both parents' version of the equation[17].

[17] We estimate only the mother-child version of equation in the Mexican data, as only the pairs of mother and child are identifiable in this data.

The results are displayed for each of our countries in Figure 5. They suggest that the degree of BMI transmission increases throughout child's BMI distribution in all the samples. This means that the father-child IBE tends to be larger at higher levels of child's BMI. In other words, the effects of shared environmental and genetic factors between father and child tend to be larger for more obese children.

The clearest way to understand these results is to consider what they mean at different points in the child's adiposity distribution. Take the case of China, At the 95th percentile of child's adiposity the IBE estimates at the median is .30. The 95th percentile bounds of this estimate are .25-.35. The corresponding estimate at the 5th percentile of children's adiposity at the median is .125 and its 95th percentile confidence interval is .10-.15. This suggests that the strength of the inheritability process is at least double for the most obese children, what it is for the thinnest children.

One possible interpretation of our results is that there is a lower bound to this elasticity of about 0.1 which is more or less a constant at the lower end of the distribution for the thinnest children. This suggests that an IBE of 0.1 could be the lowest feasible value and hence a potential lower bound to what could be measured with a biological transmission mechanism. Any value above 0.1 of this mechanism could be caused by environmental or genetic factors. It is difficult to know what the actual underlying process is here, but it could be a challenge to biologists to conceive of a genetic mechanism which would be higher for obese children than thin children. So – to the extent that a genetically inheritable trait is being measured – then potentially the excess of the IBE over 0.1 for the most obese children could be informative.

One may wish to hypothesize what the mechanisms might be for this underlying relationship – but a formal proof of any of these possible explanations is not going to be possible with this data. Hence – what we wish to do here - is just document and describe this relationship. For China there is limited evidence that the graph turns down slightly for the most obese children – but interestingly for the US the quantile plot turns down quite sharply after the 80th percentile. This indicates that the elasticity is actually falling for the most obese children. This suggests

that maybe, in the US, in the period 1988-94, children who are the most obese become that way more of their own accord.[18]

Looking more closely at each of the individual country figures in Figure 5 we see that the shape of the graph is quite different. For Indonesia the quantile plots rises at an increasing rate as we move from left to right to consider the most obese children. In contrast, the graphs for the UK and Mexico rise monotonically. These figures, taken together, suggest that there is some cross country heterogeneity in the IBE quantile estimates across the distribution of children's adiposity. This may be related to the inherent heterogeneity across countries, or, to some extent, due to the era when the data was collected. Specifically, we should remember that US data is the oldest in that it relates to 1988-94 and the position may have changed somewhat since then. A full explanation of this quantile heterogeneity across countries is again worthy of more thorough investigation when more comprehensive data become available. Specifically it would be interesting, in the future, to see how this quantile regression changed across different generations in the same country over time.

Making sense of the cross country variability of the IBE across the distribution of child's BMI is not totally straightforward. The presence of possible non-linearities, particularly at the extremes of the BMI distribution, require some explanation. But caution is necessary as the BMI of the child at say the 90th percentile in the BMI distribution in Indonesian has a very different BMI than the child at the 90th percentile in the English data. To further investigate this possibility we sought, in effect, to put each of the panels in Figure 5 on the same plot to compare them on one scale. However, the relative position of the same child may vary with country, for instance, an obese child in Indonesian data might not be seen as obese in the US data. This is not completely straightforward as the actual level of the childs' BMI for a given country behind each quantile (the x axis) in each of the separate country panels in Figure 5 is different. Figure 6 is an attempt to do this. It shows how the IBE varies across child's BMI within each country in reference to the pooled country distribution of child's BMI. Therefore, now we pool these data together, calculate the quantiles of child's BMI distribution in these countries, and then obtain the mean of child's BMI in each quantile by country. Next we plot this average of child BMI in each quantile (of child's BMI distribution in these countries) by

[18] The most unusual country is Spain which seems to have a constant IBE across the whole range of children's BMIs. However since the sample size of our data for Spain is small we have large confidence intervals around these estimates. For this reason we omit this graph from Figure 5.

country against the corresponding elasticity estimates. In doing so we are able to see how this elasticity varies with the BMI levels across countries. The results are presented in Figure 6, it suggests that the elasticity of father's BMI with child's BMI in developing countries (China and Indonesia) seems to vary more with BMI levels than that in developed countries (US and UK). The figure also shows a 'fanning-out' at higher BMI levels indicating that the variance of the relationship between father and child BMI gets bigger as the BMI of the child rises. Notwithstanding this finding – we do see – over the largest part of the BMI range a roughly constant slope of the relationship between father and child's BMI at around the child BMI of 20. This is further support for our main proposition that at the median of the child's BMI distribution the elasticity is approximately constant. This analysis, tells us that the variance and heterogeneity at the extremes of the country distributions of BMI is potentially large.

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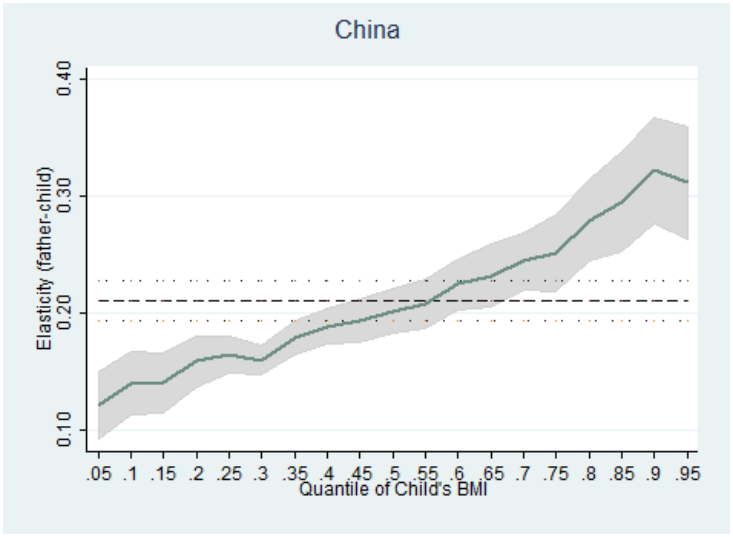
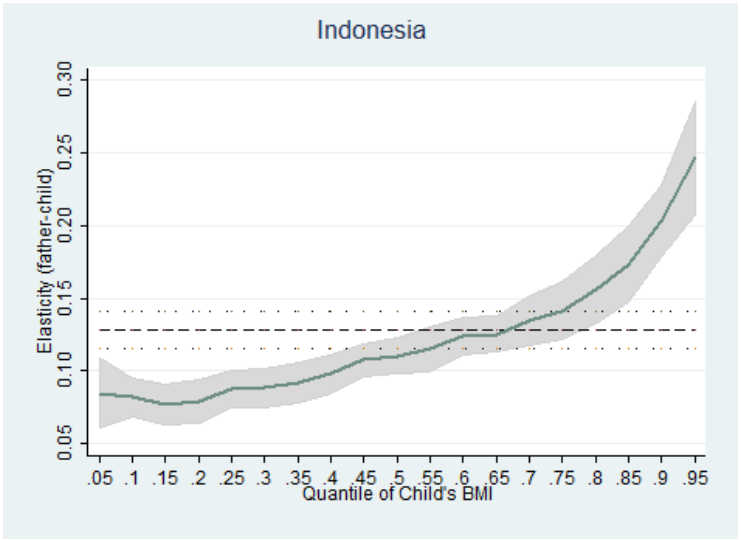
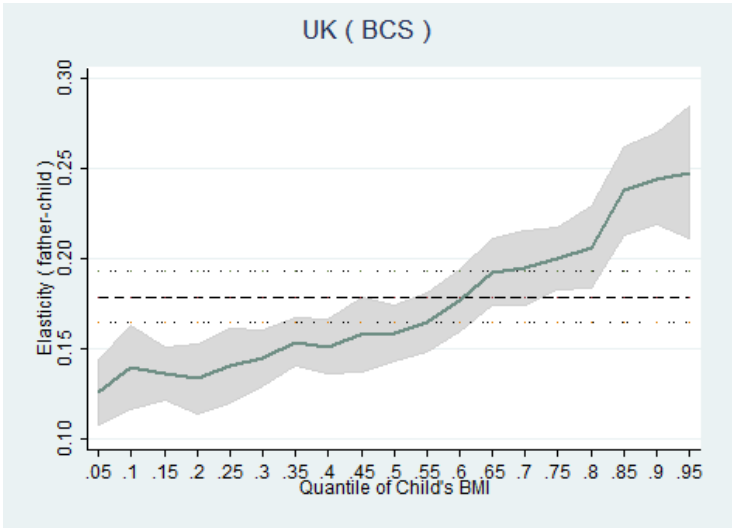
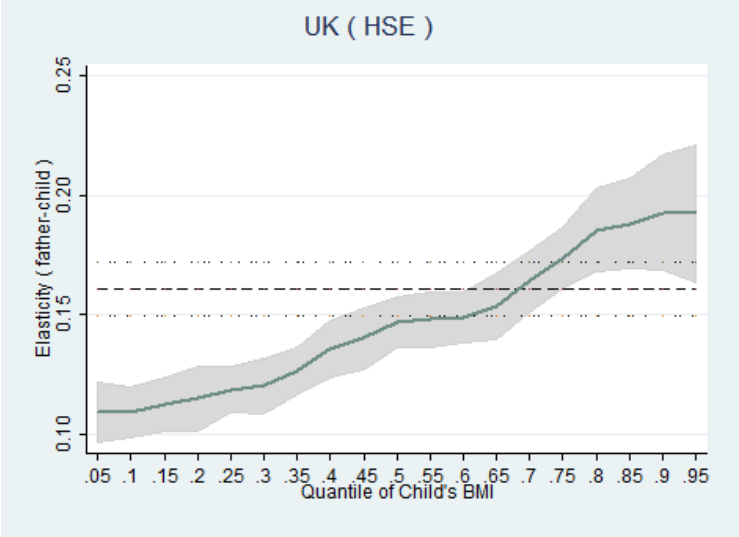
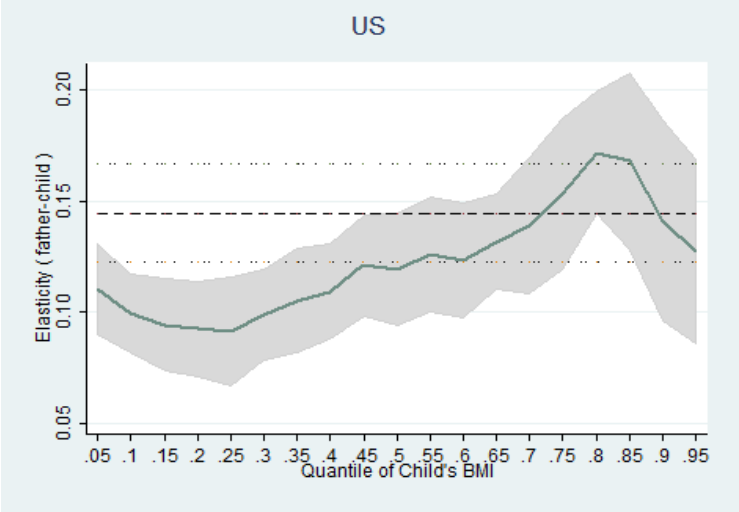
Figure 5: Quantile estimates of IBE relative to OLS elasticity



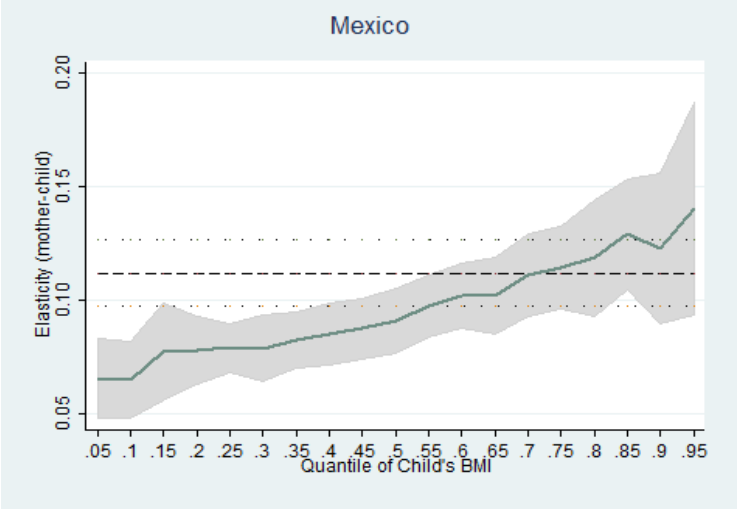






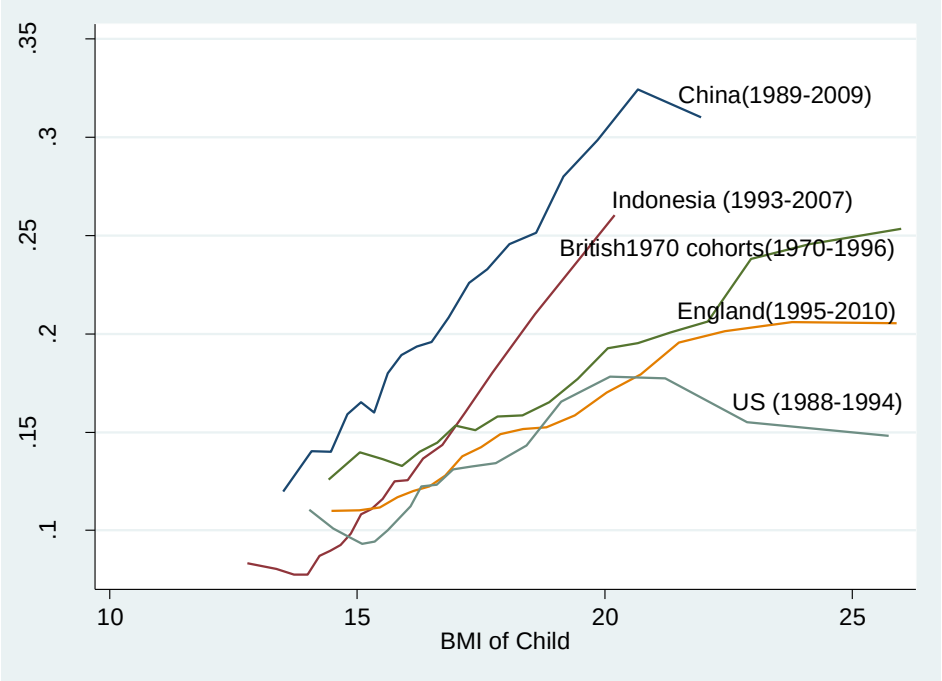


[19]



[19] Note: shaded area are 95% confidence intervals on estimates.

Figure 6: BMI of child and the elasticity of father's BMI with child's BMI across countries



5. Conclusions and Policy Implications

This paper has examined the intergenerational transmission of BMI across generations in six countries across the world. We find that the intergenerational transmission of adiposity is remarkably constant and very comparable across time and countries – even if these countries are at very different stages in their economic development. This suggests that the intergenerational transmission mechanism is both a biological process (which operates via the transmission of both parental genetic inheritance); and also a shared environmental process (within the family, when the child is growing up). These mechanisms determine a significant fraction of the child's likely BMI as an adult. At the mean of the distribution we find that the father and mother, each separately, account for up to 20% of the child's BMI at the mean. Since this effect is linear and additively separable for these two parents then we find that the joint effect of the family and its associated genetic makeup accounts for around 35-40% of the child's likely BMI.

Our second key finding is that this intergenerational transmission mechanism is very different across the distribution of children's BMI. Most specifically, it is up to double for the most obese children what it is for the thinnest children. This could have direct consequences for the health of the world's children. Specifically we find that over 30% of the most obese child's BMI is determined by the mother and 25% by the father. Hence, jointly they account for over 50% of the most obese child's likely BMI.

In contrast, the corresponding (jointly determined) fraction is only around 30% for the thinnest child. Thus, for obese children where both parents are obese, over 50% of the children's tendency to adiposity, on average, is determined by parental factors and therefore less likely to be amenable to dietary or other interventions. For obese parents the possibility of their child not being obese is accordingly lower than average. This is consistent with the common clinical finding that achieving weight reduction in the long term, for an obese individual, is both unlikely and extremely challenging.

To sum up, our evidence from different countries' data suggests that there is a strong consistency in the IBE estimates across countries. This consistency is different from what the previous studies find with respect to the intergenerational transmission of education or earnings. The literature on the transmission of intergenerational elasticity has found that there is a substantial disparity in the IIE and IEE estimates across different countries and different datasets. Ranging from as little as .1 to as much as .6 when they consider the relationship of income of the child with the income of a parent.

An implication of our research is that it puts the emphasis firmly on the family in terms of understanding the considerable fraction of adiposity determination. Specifically, we need to look no further than the simple biological process of genetic inheritance from parents to child; and what happens to the child when they are very young within the family; to explain a sizeable fraction of what they become – as obese or thin adults. We have no way (with the data available to us) of splitting up the IBE into that which is due to genetic inheritance and that which is due to the family environment – but what we do know is that jointly these two influences determine a sizeable faction of what can happen to children. One way of thinking about this process is to suggest that – in the extreme – the thinnest child in the data – still inherits 25% of their BMI from their parents. So that this may be the lowest bound on how much may be due to the process of biological and family inheritance. Some fraction of the difference between their inheritance, and that of the obese child with a (combined) .55 elasticity, may still be due to biology. But it seems likely that this left over residual could be more to do with what goes on inside the family – namely how much exercise is taken; what the family diet is like; whether they use a car for transport; how much TV is watched and generally how active they are[20].

[20] Other influential work – see Campbell et al. (2014) suggests that early childhood interventions can yield substantial health gains.

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Data Appendix

China Health and Nutrition Survey (CHNS)

The Chinese data here uses the longitudinal data from eight waves (1989, 1991, 1993, 1997, 2000, 2004, 2006, and 2009) of the China Health and Nutrition Survey (CHNS). Based on the definition of response rates that those who participated in previous survey rounds remaining in the current survey (Popkin, 2010), the response rates of this data were 88% at individual level and 90% at household level. This data contains detailed information on health outcomes, demographic and anthropometric measures of all members of the sampled households, including height and weight. It is noteworthy that these anthropometric measures are medically measured rather than self-reported which are mostly used in the literature. In addition, it includes information on economic and non-economic indicators such as education, household income and labor market outcomes.

Our sample is restricted to children under 18 years old with information (especially anthropometric information) on both the biological father and mother. We choose 18 as the threshold since age 18 is used to distinguish between adult and child in the CHNS physical exam dataset where the anthropometric information is included. Additionally, children within this age range normally live with their parents and rely on their parents for nutritional intake and health care. As a result, this sample includes 14, 082 person-wave observations made up by 6,045 children with 3,975 fathers and 3,974 mothers. In other words, our sample includes 6,045 sets of father, mother and children.

Indonesian Family Life Survey (IFLS)

The Indonesian Family Life Survey (IFLS) is an on-going longitudinal survey data which started in 1993. The sample used here is drawn from 1993, 2000 and 2007 waves of the survey, it is representative of 83% of the Indonesian population and contains over 30,000 individuals living in 13 of the 27 provinces in Indonesia. This survey includes a range of health measures for both parents and children. It is noteworthy that as in CHNS data, the anthropometric outcome in IFLS survey was also measured by trained nurses rather than self-reported. Additionally, the IFLS data also includes information on socioeconomic factors such as education and income. Thus, the IFLS data is similar to CHNS data in terms of the survey design and measure methods, this similarity improves the comparability of results based on these two datasets. The sample is restricted to those aged from 0 to 14 years old in each wave and have both parents and household's information. It is noteworthy that this is different from the CHNS data, where the child sample comprises those aged between 0 and 18 years old.

In addition, in the Indonesian Family Life Survey (IFLS), we also consider step/adopted children as the sample. The adopted or step children account for around 1% of the whole sample in each wave, for these children, the information on their parents use the step parents' rather than biological parents'.

British Cohort Study 1970 (BCS)

The 1970 British Cohort Study is an ongoing follow up study of 17,200 babies born in England, Scotland, Wales and Northern Ireland between 5 and 11 April 1970 who are still living in Britain (excluding Northern Ireland). The survey was conducted when the cohorts at birth, aged 5 (in 1975),10 (in 1980), 16 (in 1986),26 (in 1996), 30 (in 1999-2000),34 (in 2004-2005) and 38 (in 2008-2009). The samples at the age 5 and 10 were augmented since immigrants born in the same week were added in. In this paper we use the cohorts in the first five waves (sweeps).

At the birth, the questionnaires were completed by midwife and the supplementary information was collected from clinical records. As the cohorts got older, the approach of survey changed, parents were interviewed by the health stuff and questionnaires were completed by teachers. In terms of the anthropometric information, the height and weight were measured at the age of 10 and self-reported at the age of 26 (Shaheen, et al., 1999)

Health Survey for England (HSE)

The Health Survey for England is designated to be nationally representative of people of different age, gender, geographic region and socio-demographic circumstances. [21] It was started in 1991 and has been conducted annually since then. The survey combines questionnaire-based answers with physical measurements and the analysis of blood sample. Each year's survey has a particular focus on a disease or condition or population group, but height, weight and general health are covered each year. An interview with household members is followed by a nurse visit. Thus, there are both self-reported and medically-measured height and weight in this data. In the computation of BMI z-score, we use "htval" and "wtval" in the survey which are referred to as the "valid" height and weight.

The National Health and Nutrition Examination Survey III (NHANES) (US)

The National Health and Nutrition Examination Survey (NHANES) is a program of studies designed to assess the health and nutritional status of adults and children in the United States. Four surveys of this type have been conducted since 1970:

1. 1971-75—National Health and Nutrition Examination Survey I (NHANES I);
2. 1976-80—National Health and Nutrition Examination Survey II (NHANES II);
3. 1982-84—Hispanic Health and Nutrition Examination Survey (HHANES); and
4. 1988-94—National Health and Nutrition Examination Survey (NHANES III) and
5. 1999-present--National Health and Nutrition Examination Survey (Continuous NHANES)

[21] "The 1991 and 1992 surveys had a limited population sample of about 3,000 and 4,000 adults respectively. For 1993 to 1996 adult sample was boosted to about 16,000 to enable analysis by socio-economic characteristics and health regions. In 1995 for the first time a sample of about 4,000 children was also introduced. In the 1997 Health Survey the sample was about 7,000 children and 9,000 adults. In 1998 the sample was again about 16,000 adults and 4,000 children. "

Note in NHANES data, there is only a personal identification variable (seqn), there is no household id on the public release file, the relationship of a participant to the household reference person is not publicly released[22]. Thus, we cannot track down the participants' parents via father and mother's id (as in CHNS and Indonesian data), or identify the potential parents via the household id (as in English HSE data). In other words, there is no way to identify the parents by ID. However, in one of these surveys---NHANES III, there is a family background section in the youth file, where limited characteristics of the parents were collected, including mother and father's height and weight.

NHANES III, conducted between 1988 and 1994, included about 40,000 people selected from households in 81 counties across the United States. In NHANES III, black Americans and Mexican Americans were selected in large proportions,each of these groups comprised separately 30 percent of the sample. It was the first survey to include infants as young as 2 months of age and to include adults with no upper age limit. Our sample is obtained by merging the youth data which includes child's age and parents' height weight with examination data which includes child's final (medically measured) height weight. Our final sample includes 6,582 pairs of father, mother and child.

The Spanish National Health Survey (ENS-2006)

The Spanish data used here is from the Spanish National Health Survey (ENS-2006), which is the most recent statistical data collection of its type conducted by the Instituto Nacional de Estadistica (INE). This survey is representative at both the national and autonomous regional level. All the members residing at home are requested to provide information on certain demographic variables, adults answer the adult health questionnaire, and members under 16 answer the child health questionnaire. The survey covers the period between June 2006 and June 2007. The final sample used here includes more than 7,000 individuals, which consists of 2,139 pairs of father-child and 3,420 pairs of mother-child.

The Survey for the Evaluation of Urban Households (ENCELURB) (Mexico)

This survey is a longitudinal data for three years (2002, 2004 and 2009) from the Survey for the Evaluation of Urban Households (ENCELURB). This survey contains comprehensive anthropometric and general health outcomes (such as weight, height, hemoglobin levels, diabetes status, etc), and all the anthropometric measures such as weight and height have been collected by medical personnel, instead of self-reported.

This survey only includes pairs of mother-child and does not contain information on fathers, as the programme was initially designed to help children and their mothers, therefore the anthropometric information collected for children (under four years old at the beginning of the program, 2002) is more specific.

[22] With the exception of dietary data, the relationship of the sample participant to the proxy is not publicly released, either.

The sample used in this study considers 7,413 person-wave observations constituted by 2338 pairs of children and mothers for 2002; 3,459 for 2004; and 1,616 for 2009[23]. Since children are not necessarily observed in all waves, The table shows the number of parent-child pairs that were observed more than once. We see that almost 50 percent of the individuals were observed at least twice in the time horizon being considered, this may allow us to apply individual fixed effects.

Number of times children are observed in Mexican data

Waves (Years)	1	2	3
Observations	3,709	2,936	768

Source: ENCERLUB 2002, 2004 and 2009.

[23] The data relative to the external evaluation for the *Oportunidades* programme for Urban Households is also available for 2003, we omit this wave since the survey did not collect anthropometric measures this year.

Appendix A

Table A1a: the summary of mean BMI by country

	China	Indonesia	UK	US	US
Average BMI of child	19.19833	17.07465	18.88252	15.78037	17.92261
Average BMI of father	24.47539	22.19705	27.11764	21.85945	26.50184
Average BMI of mother	23.43018	22.27842	26.41251	23.08085	26.06565

Table A1b: The age of child by country

	Variable	Obs	Mean	Std. Dev.
British	Age of child	44899	14.91	4.83
China	Age of child	14081	9.085	4.78
England	Age of child	26476	9.84	4.56
Indonesia	Age of child	18650	7.17	4.23
Mexico	Age of child	7405	3.87	2.71
Spain	Age of child	11114	8.05	4.81
US	Age of child	6581	7.24	4.31

Figure A1a: The kernel density of BMI for boys aged 13-16 year (puberty)

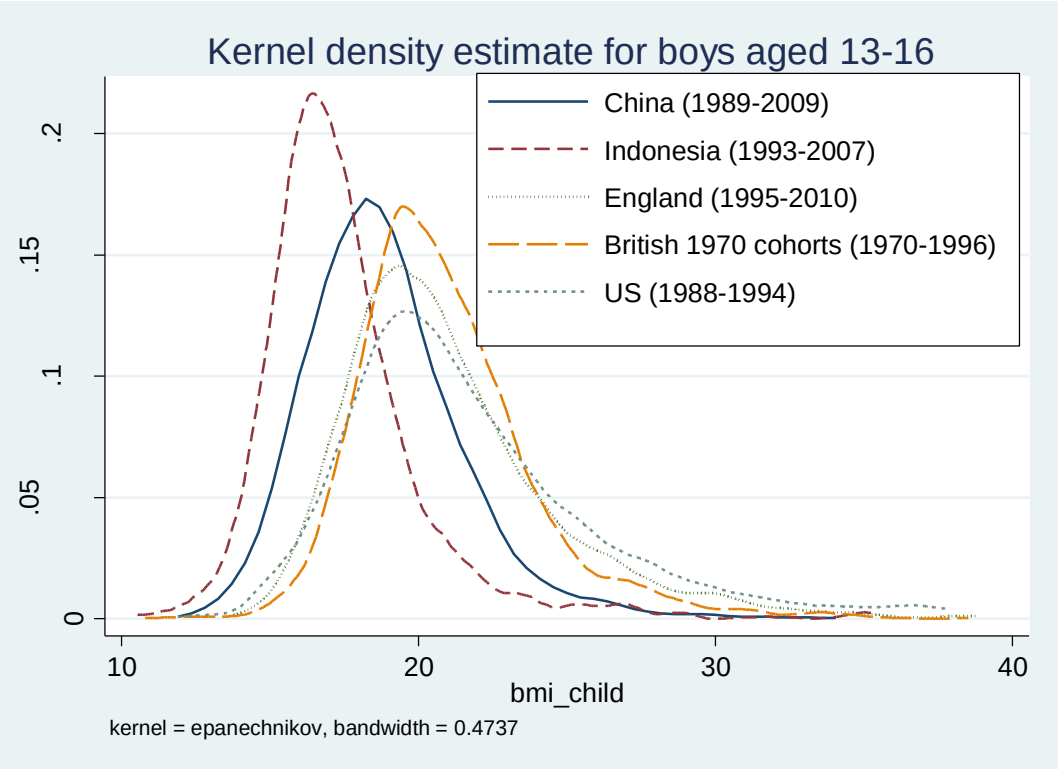

Figure A1b: The kernel density of BMI for girls aged 11-14 year (puberty)

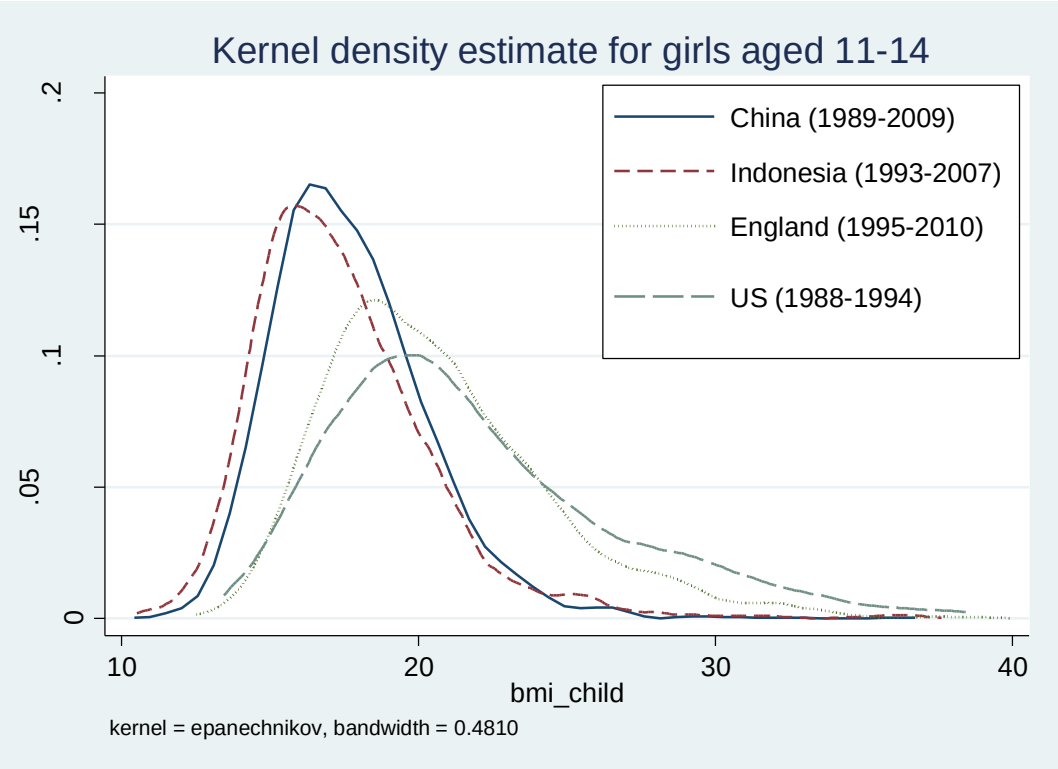

Table A2: Intergenerational BMI elasticity by country on children aged above five

	China	Indonesia	UK	UK	US
	CHNS (1989-2009)	IFLS (1993-2007)	BCS (1970-1996)	HSE (1995-2010)	NHANES 3 (1988-1994)
Dependent variable: Log (BMI of child)					
Log (BMI of father)	0.241***	0.161***	0.185***	0.186***	0.195***
	(0.0127)	(0.00939)	(0.00963)	(0.00768)	(0.0175)
Log(BMI of mother)	0.190***	0.147***	0.174***	0.196***	0.202***
	(0.0122)	(0.00780)	(0.00799)	(0.00646)	(0.0138)
Age of Child	-0.0086***	-0.0425***	0.0879***	0.0349***	0.0435***
	(0.00284)	(0.00414)	(0.00453)	(0.00270)	(0.00637)
(Age of Child) ^{2}	0.00158***	0.00369***	-0.0019***	-0.000145	-0.000351
	(0.000130)	(0.000220)	(0.000160)	(0.000125)	(0.000322)
Male Child	0.0271	-0.0377	0.531***	0.0713***	0.0608
	(0.0195)	(0.0258)	(0.0440)	(0.0177)	(0.0378)
Male*Age of Child	0.00104	0.0158***	-0.0880***	-0.0157***	-0.0136
	(0.00390)	(0.00583)	(0.00680)	(0.00366)	(0.00880)
Male*(Age of Child)^2	-0.000220	-0.0012***	0.0033***	0.00057***	0.000467
	(0.000178)	(0.000309)	(0.000243)	(0.000170)	(0.000447)
Constant	1.376***	1.842***	1.000***	1.334***	1.249***
	(0.0525)	(0.0378)	(0.0469)	(0.0335)	(0.0695)
Observations	11,082	12,884	19,594	22,103	4,207
R-squared	0.403	0.294	0.571	0.439	0.423

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Table A3: Intergenerational BMI elasticity by country on children aged between 5 and 16 years old

	China	Indonesia	UK		US
	CHNS (1989-2009)	IFLS (1993-2007)	BCS (1970-1996)	HSE (1995-2010)	NHANES (1988-1994)
Dependent variable: Log (BMI of child)					
Log (BMI of father)	0.246***	0.161***	0.173***	0.184***	0.195***
	(0.0130)	(0.00939)	(0.00957)	(0.00793)	(0.0175)
Log(BMI of mother)	0.195***	0.147***	0.161***	0.193***	0.202***
	(0.0126)	(0.00780)	(0.00813)	(0.00666)	(0.0138)
Age of Child	-0.0191***	-0.0425***		0.0240***	0.0435***
	(0.00324)	(0.00414)		(0.00302)	(0.00637)
(Age of Child) ^{2}	0.00213***	0.00369***	0.00149***	0.000426***	-0.000351
	(0.000154)	(0.000220)	(1.78e-05)	(0.000145)	(0.000322)
Male Child	0.00925	-0.0377		0.0389**	0.0608
	(0.0215)	(0.0258)		(0.0192)	(0.0378)
Male*Age of Child	0.00518	0.0158***	-0.00168***	-0.00817**	-0.0136
	(0.00447)	(0.00583)	(0.000604)	(0.00411)	(0.00880)
Male*(Age of Child)^2	-0.000436**	-0.00124***	-1.43e-06	0.000179	0.000467
	(0.000213)	(0.000309)	(4.58e-05)	(0.000198)	(0.000447)
Constant	1.391***	1.842***	1.623***	1.398***	1.249***
	(0.0541)	(0.0378)	(0.0369)	(0.0350)	(0.0695)
Observations	10,474	12,884	15,658	20,431	4,207
R-squared	0.379	0.294	0.450	0.421	0.423

Note: the variable for "age of child" is omitted due to collinearity for BCS sample. Robust standard errors in parentheses, *** p<0.01, ** p<0.05, * p<0.1.

Table A4: Intergenerational BMI elasticity for parents and child on Pooled Country Data, Indonesian as the reference group

Dependent variable: log (BMI of child)				
Log (BMI of father)	0.161***	0.200***		0.161***
	(0.00402)	(0.00405)		(0.00419)
Log (BMI of mother)	0.163***		0.182***	0.162***
	(0.00342)		(0.00256)	(0.00359)
Obese father*				0.0129***
Obese mother				(0.00447)
Underweight father*				0.0123***
Underweight mother				(0.00271)
Age of Child	-0.0148***	-0.0141***	-0.0142***	-0.0147***
	(0.000494)	(0.000499)	(0.000436)	(0.000493)
(Age of Child) 2	0.00185***	0.00182***	0.00183***	0.00184***
	(2.46e-05)	(2.50e-05)	(2.22e-05)	(2.46e-05)
Male Child	0.0521***	0.0494***	0.0467***	0.0520***
	(0.00296)	(0.00298)	(0.00256)	(0.00296)
Male*Age of Child	-0.0131***	-0.0125***	-0.0117***	-0.0131***
	(0.000687)	(0.000693)	(0.000613)	(0.000687)
Male*(Age of Child)2	0.000597***	0.000575***	0.000530***	0.000597***
	(3.51e-05)	(3.55e-05)	(3.21e-05)	(3.51e-05)
China	0.0433***	0.0370***	0.0464***	0.0438***
	(0.00169)	(0.00172)	(0.00152)	(0.00169)
Britain	0.0445***	0.0409***	0.0619***	0.0457***
	(0.00158)	(0.00160)	(0.00144)	(0.00158)
England	0.0704***	0.0821***	0.102***	0.0710***
	(0.00165)	(0.00168)	(0.00135)	(0.00166)
US	0.0668***	0.0778***	0.0954***	0.0674***
	(0.00224)	(0.00229)	(0.00196)	(0.00224)
Spain		0.0866***	0.120***	
		(0.00430)	(0.00252)	
Mexico			0.0592***	
			(0.00197)	
Constant	1.722***	2.107***	2.154***	1.724***
	(0.0151)	(0.0126)	(0.00817)	(0.0168)
Observations	87,041	89,439	99,268	87,041
R-squared	0.500	0.471	0.467	0.501

Notes: Robust standard errors in parentheses.The dummy Obese_father*mother=1 if the BMI of father and mother are above 30, Under_father*mother=1 if the BMI of father and mother are below 20, *** p<0.01, ** p<0.05, * p<0.1

Table A5: Intergenerational BMI elasticity for parents and child, including the interactions of BMI with country dummies

VARIABLES	(1) Log (BMI of child)
Log (BMI of father)	0.164***
	(0.00633)
Log (BMI of mother)	0.143***
	(0.00581)
Age of Child	-0.0148***
	(0.000494)
(Age of Child)^2	0.00184***
	(2.46e-05)
Male Child	0.0520***
	(0.00296)
Male*Age of Child	-0.0131***
	(0.000687)
Male*(Age of Child)^2	0.000597***
	(3.51e-05)
Log (BMI of father)*China	0.0144
	(0.0103)
Log (BMI of father)*Britain	0.00257
	(0.00834)
Log (BMI of father)*England	-0.0117
	(0.00725)
Log (BMI of father)*USA	-0.0121
	(0.0106)
Log (BMI of mother)*China	-0.000530
	(0.0102)
Log (BMI of mother)*Britain	0.0117
	(0.00835)
Log (BMI of mother)*England	0.0341***
	(0.00723)
Log (BMI of mother)*USA	0.0334***
	(0.0107)
Constant	1.772***
	(0.0155)
Observations	87,041
R-squared	0.501

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table A6: Intergenerational BMI elasticity on sample with approaching adult children (age>16), Indonesian as the reference group

	(1)	(2)	(3)	(4)	(5)	(6)
Dependent Variable: Log(BMI of child)						
Log(BMI of father)	0.189***	0.246***		0.195***	0.234***	
	(0.0102)	(0.0102)		(0.0108)	(0.0110)	
Log(BMI of mother)	0.163***		0.206***	0.185***		0.210***
	(0.0088)		(0.0088)	(0.0090)		(0.0090)
Male child	-0.39***	-0.37***	-0.38***	-0.28***	-0.28***	-0.29***
	(0.0649)	(0.0658)	(0.0650)	(0.0641)	(0.0654)	(0.0642)
Britain				1.866***	1.850***	209***
				(0.0760)	(0.0767)	(0.0749)
England				0.876***	1.388***	1.696***
				(0.104)	(0.105)	(0.0983)
US				1.368***	030***	116***
				(0.274)	(0.283)	(0.285)
Constant	13.58***	16.11***	17.29***	11.46***	14.82***	15.27***
	(0.288)	(0.251)	(0.207)	(0.302)	(0.256)	(0.217)
Observations	13,881	13,967	14,409	13,881	13,967	14,409
R-squared	0.099	0.063	0.064	0.128	0.085	0.094

Robust standard errors in parentheses, *** p<0.01, ** p<0.05, * p<0.1

Table A7 : OLS estimates of the intergenerational BMI elasticity, controlling for age dummies and the interactions between age and gender

	China	Indonesia	UK		CHNS
	CHNS	IFLS	BCS	HSE	
	(1989-2009)	1993-2007)	(1970-1996)	(1995-2010)	(1989-2009)
Dependent variable: log (BMI of child)					
Log (BMI of father)	0.212***	0.129***	0.177***	0.166***	0.150***
	(-0.0115)	(-0.0077)	(-0.0092)	(-0.0067)	(-0.0123)
Log (BMI of mother)	0.173***	0.126***	0.163***	0.178***	0.152***
	(-0.0108)	(-0.0064)	(-0.0076)	(-0.0057)	(-0.0096)
Constant	1.689***	1.960***	1.747***	1.699***	1.817***
	(-0.046)	(-0.0281)	(-0.0351)	(-0.0274)	(-0.045)
Observations	14,081	18,650	21,253	26,476	6,581
R-squared	0.37	0.231	0.566	0.468	0.469

Notes: The regression also includes the child age dummies and their interactions with gender. Robust standard errors in parentheses, *** p<0.01, ** p<0.05, * p<0.1

Table A8: The intergenerational transmission of height.

	China	Indonesia	UK		US
	CHNS	IFLS	BCS	HSE	NHANES 3
	(1989-2009)	(1993-2007)	1970-1996)	(1995-2010)	1988-1994)
VARIABLES: Log (height of child)					
Log (height of	0.428***	0.302***	0.288***	0.277***	0.199***
	(0.0180)	(0.0208)	(0.00840)	(0.00829)	(0.0125)
Log(height of	0.421***	0.419***	0.368***	0.341***	0.300***
	(0.0187)	(0.0191)	(0.00902)	(0.00828)	(0.0129)
Age of Child	0.0891***	0.0991***	0.0748***	0.0780***	0.0835***
	(0.000561)	(0.000677)	(0.000229)	(0.000326)	(0.000621)
(Age of Child)^2	-0.0024***	-0.0031***	-0.0019***	-0.0020***	-0.0023***
	(2.71e-05)	(4.01e-05)	(8.47e-06)	(1.59e-05)	(3.46e-05)
Male Child	0.00124	0.0145***	0.0340***	0.0210***	-0.00438**
	(0.00281)	(0.00287)	(0.00154)	(0.00138)	(0.00214)
Male*Age of Child	0.00145***	-0.0016***	0.00529***	0.00403***	0.00203***
	(0.000234)	(0.000313)	(0.000102)	(0.000125)	(0.000255)
Male*(Age of	0.00069***	0.00015*	0.00049***	0.00086***	0.00091***
Child)^2	(5.32e-05)	(8.00e-05)	(1.52e-05)	(3.07e-05)	(6.66e-05)
Constant	-0.0484	0.562***	1.005***	1.206***	1.800***
	(0.104)	(0.130)	(0.0556)	(0.0519)	(0.0823)
Observations	14,081	18,650	22,458	26,476	6,581
R-squared	0.944	0.909	0.956	0.952	0.954

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Appendix: Transition Matrices

The most common way of analysing intergenerational transmission is to present transition matrices which tabulate the empirical discrete distribution associated with BMI for parent and child (Bhattacharya and Mazumder 2011). This analysis is not conditional on covariates but it does informatively summarize the bivariate distribution of child and adult BMI. Hence, we calculate the conditional transition probabilities to describe the rates of movement across specific categories of the BMI distribution across generations. We adopt different BMI measures when we classify the BMI category of mothers and children. We classify mothers' BMI status based on their raw BMI: under 18.5 is classified as underweight, 18.5-24.9 as normal weight, 25-29.9 as overweight, and above 30 as obese. Whereas the classification of children's BMI status is based on the WHO classification by BMI z-score: underweight if BMI z-score <-1.04; normal if -1.04<=BMI z-score<1.04; overweight if 1.04<=BMI z-score<1.64; obese if BMI z-score>=1.64. This BMI z-score is calculated with respect to the WHO reference population which varies by age and gender rather than with respect to the sample used here. We do not use raw BMI when we classify the BMI status of children because raw BMI levels are interpreted differently for adults and children. For adults, BMI classifications are independent of age or gender, whereas for children of all ages, BMI needs to be interpreted relative to a child's age and gender, since the amount of typical child body fat varies by age and gender.

Based on this classification, Table A9 to Table A15 present the transition probabilities of BMI status across generations in the CHNS (1989-2009) (China), IFLS (1993-2007) (Indonesia), BCS1970 cohort (Britain), HSE (1995-2010) (England), NHANES (1988-1994) (USA), ENS-2006 (Spain) and ENCELURB (2002-2009) (Mexico), respectively. These transition probabilities describe the distribution of child's BMI status conditional on mother's BMI status, they are similar to transition matrices across the discretized bivariate distribution. The interaction terms between mother and child of different BMI status. For instance, in Table A9, the numbers in the first row of matrix indicate of the total number of children whose mothers were "underweight", 20.56% were "underweight", 70.33% were "normal", 4% were "overweight, and 5.11% were "obese". For mothers in the "underweight" category, 20.56 % of their children appear in the same category "underweight", and 70.33% were in the "normal" category. Compared with other categories, there seems a stronger transmission of the same BMI status in the "underweight" category. In the case of Indonesia, Table A16 suggests there is a larger proportion of children in the "underweight" category, and a larger proportion of mothers in the "obese" category. This distribution is in line with recent studies, which suggest the possible coexistence of "under nutrition" and "obesity"

clustering within the same country or even within a single household ("dual burden households") in some developing countries, such as Indonesia (Doak et al. 2004). Moreover, we see there is a stronger intergenerational transmission of "underweight" (26.06%) in the IFLS sample compared to the CHNS sample.

In terms of the UK, as shown in Table A11 and A12, there is a significant greater fraction of mothers and children in the category of "overweight" and "obesity". Moreover, comparing Table A11 (based on BCS 1970 cohorts) and Table A12 (HSE sample), the fraction of "overweight" is larger for both mothers and children in the HSE (1995-2010) sample than in the BCS 1970 cohorts survey which follows the cohorts from the time when they were born (1970) up until they were 26 years old (1996). Considering the timing, Table A11 and A12 indicate an increasing proportion of "overweight" among adults and children over time from the period 1970-1996 to 1995-2010. In the case of the US, based on the NHANES3 sample (1988-1994), Table A13 suggests there is a large fraction of "overweight" and "obese" for both mothers and children. Similarly, Table A14 suggests a strong transmission of "obese" status (47.34%) from mothers to children. In the case of Mexico, Table A15 shows a strong transmission of "obese" status, given a large fraction of "obese" mothers (27.03%) in the mothers' BMI distribution. The relatively larger prevalence of "overweight" and "obese" compared to other developing countries is consistent with the fact there is a substantially rising trend of obesity in Mexico during the survey period (Neufeld, *et al*. 2008).

Table A9: The transition probabilities of mother and child's BMI z-score in CHNS 1989-2009 (China)

Full sample	BMI z-score		*Child's BMI status by BMI z-score (%)*				Mother's distribution	*Observations*
			BMI z-score Category					
		Category	<-1.64 *Underweight*	-1.64-[illegible] *Normal*	1.04-1.64 *Overweight*	>1.64 *Obese*		
Mother's BMI status by BMI(%)	< 18.5	*Underweight*	**20.56**	70.33	4	5.11	6.47	*900*
	18.5-24.9	*Normal*	10.86	**75.25**	6.64	7.26	76.53	*10,649*
	25-29.9	*Overweight*	6.25	74.71	**9.59**	9.45	15.29	*2,127*
	>30	*Obese*	7.98	65.55	13.45	**13.03**	1.71	*238*
	Child's distribution		10.73	74.68	7.04	7.55		
	Observations		*1,493*	*10,391*	*979*	*1,051*		*13,914*

Table A10: The transition probabilities of mother and child's BMI z-score in IFLS 1993-2007 (Indonesia)

Full sample	BMI z-score		*Child's BMI status by BMI z-score (%)*				Mother's distribution	*Observations*
			BMI z-score Category					
		Category	<-1.64 *Underweight*	-1.64- *Normal*	1.04-1.64 *Overweight*	>1.64 *Obese*		
Mother's BMI status by BMI(%)	< 18.5	*Underweight*	**26.06**	64.51	2.91	6.52	9.32	*1,719*
	18.5-24.9	*Normal*	17.33	**72.62**	4.1	5.96	63.06	*11,635*
	25-29.9	*Overweight*	12.1	74.62	**5.8**	7.49	21.86	*4,034*
	>30	*Obese*	7.71	72.15	7.9	**12.23**	5.76	*1,063*
	Child's distribution		16.44	72.27	4.58	6.7		
	Observations		*3,034*	*13,335*	*845*	*1,237*		*18,451*

Table A11: The transition probabilities of mother and child's BMI z-score in British Cohort Studies 1970 (UK)

Full sample	BMI z-score		*Child's BMI status by BMI z-score (%)*				Mother's distribution	*Observations*
			BMI z-score Category					
			<-1.64	-1.64-1.04	1.04-1.64	>1.64		
		Category	*Underweight*	*Normal*	*Overweight*	*Obese*		
Mother's BMI status	< 18.5	*Underweight*	**7.72**	81.41	7.46	3.4	3.42	*764*
	18.5-24.9	*Normal*	3.85	**80.31**	9.54	6.31	72.36	*16,142*
by BMI(%)	25-29.9	*Overweight*	2.34	70.95	**14.72**	11.99	18.55	*4,137*
	>30	*Obese*	2.37	64.24	16.46	**16.93**	5.67	*1,264*
	Child's distribution		3.62	77.7	10.82	7.86		
	Observations		*807*	*17,332*	*2,414*	*1,754*		*22307*

Table A12: The transition probabilities of mother and child's BMI z-score in HSE 1995-2010 (UK)

Full sample	BMI z-score		*Child's BMI status by BMI z-score (%)*				Mother's distribution	*Observations*
			BMI z-score Category					
			<-1.64	-1.64-1.04	1.04-1.64	>1.64		
		Category	*Underweight*	*Normal*	*Overweight*	*Obese*		
Mother's BMI status	< 18.5	*Underweight*	**9.72**	63.33	9.17	17.78	1.38	*360*
	18.5-24.9	*Normal*	2.02	**59.29**	14.21	24.49	46.21	*12,092*
by BMI(%)	25-29.9	*Overweight*	1.37	54.62	**15.31**	28.7	31.54	*8,254*
	>30	*Obese*	0.92	44.04	16.24	**38.8**	20.87	*5,461*
	Child's distribution		1.69	54.69	14.91	28.71		
	Observations		*442*	*14,310*	*3,902*	*7,513*		*26,167*

Table A13: The transition probabilities of mother and child's BMI z-score in NHANES 3 1988-1994 (US)

Full sample	BMI z-score		***Child's BMI status by BMI z-score (%)***				Mother's distribution	*Observations*
			BMI z-score Category					
		Category	<-1.64 *Underweight*	-1.64- *Normal*	1.04-1.64 *Overweight*	>1.64 *Obese*		
Mother's BMI status	< 18.5	*Underweight*	**1.44**	47.12	24.04	27.4	3.19	*208*
	18.5-24.9	*Normal*	1.55	**43.79**	18.28	36.38	48.46	*3,156*
by BMI(%)	25-29.9	*Overweight*	0.89	40.65	**17.99**	40.47	25.95	*1,690*
	>30	*Obese*	0.55	33.1	16.24	**50.1**	22.4	*1,459*
	Child's distribution		1.15	40.69	17.93	40.23		
Observations			*75*	*2,650*	*1,168*	*2,620*		*6,513*

Table A14: The transition probabilities of mother and child's BMI z-score in ENS-2006 (Spain)

Full sample	BMI z-score		***Child's BMI status by BMI z-score (%)***				Mother's distribution	*Observations*
		Category	<-1.64 *Underweight*	-1.64- *Normal*	1.04-1.64 *Overweight*	>1.64 *Obese*		
Mother's BMI status	< 18.5	*Underweight*	**10.53**	43.42	14.47	31.58	2.26	*76*
	18.5-24.9	*Normal*	4.44	**46.61**	13.76	35.19	61.02	*2,049*
by BMI(%)	25-29.9	*Overweight*	2.85	42.92	**16.21**	38.01	26.09	*876*
	>30	*Obese*	2.24	36.41	14.01	**47.34**	10.63	*357*
	Child's distribution		3.93	44.49	14.44	37.14		
Observations			*132*	*1,494*	*485*	*1,247*		*3,358*

Table A15: The transition probabilities of mother and child's BMI z-score in ENCELURB (2002-2009) (Mexico)

Full sample	BMI z-score		***Child's BMI status by BMI z-score (%)***				Mother's distribution	*Observations*
		Category	<-1.64 *Underweight*	-1.64- *Normal*	1.04-1.64 *Overweight*	>1.64 *Obese*		
Mother's BMI status	< 18.5	*Underweight*	**8.13**	75.61	4.88	11.38	1.69	*123*
	18.5-24.9	*Normal*	2.82	**75.63**	10.86	10.69	33.17	*2,413*
by BMI(%)	25-29.9	*Overweight*	1.95	68.65	**14.57**	14.83	38.11	*2,772*
	>30	*Obese*	1.73	62.26	15.67	**20.35**	27.03	*1,966*
	Child's distribution		2.28	69.36	13.47	14.89		
Observations			*166*	*5,045*	*980*	*1,083*		*7,274*

In summary, these transition probabilities reveal a wide disparity in the joint distribution of mother and child's BMI status across countries. The CHNS sample suggests a stronger persistence of "underweight" between mothers and children in China; the IFLS sample shows a coexistence of "underweight" children and "obese" mothers ("nutrition transition paradox")[24] in Indonesia; there is a significantly larger fraction of mothers and children in the category of "overweight" and "obesity" in the UK, similar in the US and Spain; there is a relatively larger prevalence of "obesity" in Mexico compared to other developing countries. These transition probabilities show a global mobility of BMI status across the entire distribution, in particular reveal the prevalence of large movements in the BMI distribution from one generation to the next.

The matrices from Table A16 to A22 are the counterparts to Tables A9 to A15 but using the quartiles of BMI. These tables - quite clearly show that there is a lot lower level of transitions between children of one quartile in their own BMI distribution compared to their parents when we calculate the transition matrices by quartile. This is not surprising. These transition matrices do also show that there is most movement in these transition matrices in the extremes of the distribution - i.e. in the quartile where the parent and child BMI are both in the top or the bottom quartile of their respective BMI distributions.

[24] Relatively, compared with developing countries.

Table A16: The transition probabilities of mother and child's BMI quartiles in CHNS 1989-2009 (China)

Full sample		***Child's BMI status by quartile***				Mother's distribution	*Observations*
	BMI	***BMI quartile***					
		quartile1	*quartile 2*	*quartile 3*	*quartile 4*		
Mother's BMI status by quartile	*quartile1*	34.63	28.11	21.68	15.59	25.06	3,478
	quartile 2	27.33	27.16	25.19	20.32	24.95	3,479
	quartile 3	21.8	24.13	26.14	27.93	24.99	3,478
	quartile 4	16.25	21.19	26.53	36.02	25	3,479
		25.01	25.15	24.88	24.96		
Observations		3,478	3,479	3,478	3,479		*13,914*

Table A17: The transition probabilities of mother and child's BMI quartiles in IFLS 1993-2007

(Indonesia)

Full sample		***Child's BMI status by quartile***				Mother's distribution	*Observations*
	BMI	***BMI quartile***					
		quartile1	*quartile 2*	*quartile 3*	*quartile 4*		
Mother's BMI status by quartile	*quartile1*	32.58	26.7	22.4	18.32	25.06	4,613
	quartile 2	26.01	26.34	26.21	21.44	24.95	4,612
	quartile 3	23.95	25.11	25.54	25.41	24.99	4,613
	quartile 4	17.46	21.86	25.85	34.82	25	4,613
		25.02	24.98	25.01	24.99		
Observations		4,613	4,612	4,613	4,613		18,451

Table A18: The transition probabilities of mother and child's BMI quartiles in British Cohort Studies 1970 (UK)

Full sample		***Child's BMI status by quartile***				Mother's distribution	*Observations*
	BMI	***BMI quartile***					
		quartile1	*quartile*	*quartile*	*quartile*		
Mother's BMI status by quartile	*quartile1*	31.67	25.03	24.87	18.44	25.45	5,577
	quartile 2	27.97	26.3	23.14	22.59	25.63	5,577
	quartile 3	23.57	25.5	25.57	25.36	24.49	5,577
	quartile 4	20.33	25.26	24.63	29.79	24.43	5,576
		25.97	25.52	24.54	23.97		
Observations		5,577	5,577	5,577	5,576		22,307

Table A19: The transition probabilities of mother and child's BMI quartiles in HSE 1995-2010 (UK)

Full sample		***Child's BMI status by quartile***				Mother's distribution	*Observations*
	BMI	***BMI quartile***					
		quartile1	*quartile*	*quartile*	*quartile*		
Mother's BMI status by quartile	*quartile1*	34.17	27.25	23.32	15.26	25	6,541
	quartile 2	26.31	26.65	25.41	21.63	25	6,542
	quartile 3	22.26	24.53	25.83	27.38	24.99	6,542
	quartile 4	17.25	21.57	25.44	35.73	25	6,542
		25	25	25	25		
Observations		6,541	6,542	6,542	6,542		26,167

Table A20: The transition probabilities of mother and child's BMI quartiles in NHANES 3 1988-1994 (US)

Full sample		***Child's BMI status by quartile***				Mother's distribution	*Observations*
	BMI	***BMI quartile***					
		quartile1	*quartile*	*quartile*	*quartile*		
Mother's BMI status	*quartile1*	33.95	29.47	22.97	13.6	25.47	1,628
	quartile 2	28.69	26.29	23.71	21.31	24.74	1,629
by quartile	*quartile 3*	21.7	24.33	25.86	28.12	24.86	1,628
	quartile 4	15.54	19.8	27.48	37.17	24.94	1,628
		25.01	25	25	25		
Observations		1,628	1,629	1,628	1,628		6,513

Table A21: The transition probabilities of mother and child's BMI quartiles in ENS-2006 (Spain)

Full sample		***Child's BMI status by quartile***				Mother's distribution	*Observations*
	BMI	***BMI quartile***					
		quartile1	*quartile*	*quartile*	*quartile*		
Mother's BMI status by quartile	*quartile1*	32.6	26.31	25.73	15.37	25.13	839
	quartile 2	27.25	27.02	25.15	20.58	25.01	840
	quartile 3	24	24	23.88	28.12	24.87	840
	quartile 4	18.62	21.43	25.41	34.54	24.99	839
		25.63	24.69	25.04	24.63		
Observations							3,358
		839	840	840	839		

Table A22: The transition probabilities of mother and child's BMI z-score in ENCELURB (2002-2009) (Mexico)

Full sample		***Child's BMI status by quartile***				Mother's distribution	*Observations*
	BMI	***BMI quartile***					
		quartile1	*quartile*	*quartile*	*quartile*		
Mother's BMI status by quartile	*quartile1*	33.14	25.8	23.96	17.11	25.02	1,818
	quartile 2	25.19	26.38	25.19	23.24	24.98	1,819
	quartile 3	22.57	25.92	25.43	26.08	25.01	1,819
	quartile 4	19.14	21.95	25.35	33.57	24.98	1,818
Observations	Child's	25.01	25.01	24.98	25		
		1,818	1,819	1,819	1,818		7,274

Appendix B: The Metrics and Magnitudes of BMI, z-score and logBMI Intergenerational Correlations.

There is some disagreement in what is the correct measure of adiposity to use in statistical work on intergenerational transmission. In this appendix we clarify the alternative measures that have been used, their inter-relationship and their relative merits and disadvantages.

The Different Measures of Adiposity used in Statistical and Econometric Research.

The most common measures of adiposity[25] used is statistical relations between one generation and another are:

i) The raw BMI,

ii) The BMI z-score relative to some population.

iii) The log of the BMI.

In thinking about how to compare the relative merits of these measures it should be remembered that the BMI measure is itself a specific non-linear measure of height and weight, defined as: [weight(kg)/height^2(cm)]*10,000. This means that transformations of the BMI need to be treated with care. In consequence, coefficients of regressions involving transformations of this ratio – especially when normed against a reference population - can be difficult to interpret and compare. Inevitably their interpretation also depends on what controlling regressors have been used in the estimation. All these factors contribute to difficulties in comparing coefficients across different published research findings.

Predominantly the medical, biological and epidemiological literature has mainly estimated an intergenerational BMI correlation (IBC) which is the size of the either the raw correlation of fathers BMI, y_f , with child's BMI[26], y_c , or the coefficient, β_1 , in equation (B1) of the regression of child's BMI on fathers BMI.

$$y_c = \alpha_1 + \beta_1 y_f + u_1 \qquad \text{(B1)}$$

[25] Some medical studies have access to other measure like percentage body obese or measures of skin pinches. This data was not available to this study.

[26] For the sake of simplicity we use the example of child and father in this Appendix – the same logic applies to mother and child.

Fortunately, if the simple regression in (B1) is transformed into z-score values the interpretation of the IBC coefficient, β_1 , does not change much to, β_2 in equation (B2).

$$Z_c = \alpha_2 + \beta_2 Z_f + u_2 \qquad \text{(B2)}$$

where $z = \frac{y-\bar{y}}{\sigma}$

Indeed it can be shown by simple algebra that $\beta_1 = \sigma\beta_2$ (under basic regularity conditions).

This means that the marginal effects interpretation of the IBC coefficient is very similar in equation (B1) and (B2). Specifically in equation B2, the IBC estimate will be the effect of a one unit change of the standard deviation of the fathers BMI z-score on the child's BMI z score. This means, that broadly speaking the IBC estimates from basic raw BMI regressions and those run in z-scores are comparable – relative to the reference group which norms these BMI values. (We discuss this below.)

Where the position becomes more complicated is when we use the logBMI as the variable in our regression. Specifically if we run the regression:

$$logy_c = \alpha_3 + \beta_3 logy_f + u_3 \qquad \text{(B3)}$$

then the comparison of β_3 with β_1 or β_2 is slightly more problematic. The reason we might want to do that is to compare the marginal effect in the coefficient β_3 – which is now an elasticity – indeed we can call the this the intergenerational BMI elasticity, IBE – with the literature on estimating the elasticity of transmission across generations. This elasticity has been estimated by many papers and it would be insightful to be able to compare this IBE with the corresponding estimates of elasticity of intergenerational transmission of income or education. Our difficulty is that this is not what has been estimated by the epidemiological literature on the intergenerational transmission of adiposity – here an IBC has been estimated. In this paper we use the IBE - but in this appendix we explain and calibrate the relationship between the IBE and the IBC. We also report in Table C1 the counterpart of Table 10c which reports the IBE for the same data and specification.

The logical question is – how might the IBC and IBE compare more generally for the same data. This is particularly important if we wish to compare our estimates with those of Classen (2010), as he estimates an IBE and other others in other literatures who estimate the IBC. The answer is that the magnitude of the IBE will strictly be not directly comparable with the magnitude of IBC. What is logically the case is that the IBC will typically be less than the IBE – but this is not surprising as

the IBC is based on a regression which has been transformed in natural logs so that the metric of adiposity are much smaller numbers. There is no uniform look up comparison possible though as the estimated intercept needs to be taken into account. Explicitly it should be remembered that the log transformation is a specific non-linear transformation (which makes appropriate the proportionate change interpretation consistent with an elasticity). To see this we can verify with a numerical example from our data. Using our data from our Table B1 below we can compare the IBC and IBE for the whole of our pooled data (using only the mothers BMI as the only regressor).

Table B1 - Estimates of IBC, IBE parameters for Pooled Data.

Equation	**Intercept**	**Slope**	**Obs**	**Means**
B1 - (IBC)	13.78	.166	99,268	$y_f = 24.479,\ y_f = 17.848$
B2	1.17e-06	.2204	99,268	Z_f = -0.030 $Z_c = 1.35e06$
B3 - (IBE)	2.129	.231	99,268	$logy_f = 3.180,$ $logy_c =$ 3639

We can also verify the property that the z-score regression goes through the mean of the data:

$$\bar{Z}_c = \hat{a}_2 + \hat{b}_2 \bar{Z}_f$$

-0.21 = 1.35e-06 + .2204(-0.030)

Likewise, using our data the values in Table B1 we can verify the property that the double log regression goes through the mean of the logs:

$$\log \bar{y}_c = \hat{a}_3 + \hat{b}_3 \log \bar{y}_f$$

Log(2.8639) = 2.129 + .231 log(3.1806)

This is confirmation that our relative estimates of an IBC of .166 and an IBE of .220 are comparable. This means that Classen's (2010) estimate of an IBE of .35 needs to be potentially considerably tempered in comparing it with any IBC estimates or our own IBE estimates in this paper. Indeed our own estimates of the IBE for mothers and child for the USA is .18 and for fathers and child is .19 for the USA from Table 10a. In essence what to report is a matter of personal preference – but one that should be informed by a comparison of the two different numbers for the same data and a clarity on the comparison of what exactly is being estimated in each case.

The reader should keep in mind the distinction when comparisons with other papers are made. The disadvantage of the double log transformation is that it is a particular non-linear transformation (on

an already transformed BMI variable) which makes interpretation of the effects more difficult. The disadvantage of using the z-score method is that it will depend on the validity of the reference group[27].

[27] We also need to be careful in handling data in terms of z-scores as obviously approximately half of these values are negative.

Table B2. Estimates for IBC Comparable to Table 10c on IBE.

	China	Indonesia	Britain	England	US
Dependent Variable: BMI z-score of child					
BMI z-score_father	0.244***	0.157***	0.193***	0.188***	0.174***
	(0.0133)	(0.00984)	(0.00978)	(0.00779)	(0.0141)
BMI z-score_mother	0.239***	0.189***	0.194***	0.223***	0.177***
	(0.0144)	(0.00961)	(0.00915)	(0.00746)	(0.0106)
Age of Child	-0.278***	-0.265***	-0.146***	-0.484***	-0.395***
	(0.0134)	(0.0142)	(0.00792)	(0.0104)	(0.0208)
(Age of Child) ^{2}	0.0108***	0.0142***	0.00627***	0.0184***	0.0158***
	(0.000685)	(0.000881)	(0.000305)	(0.000535)	(0.00123)
Male Child	-0.0823	0.0330	0.0882	-0.111*	-0.171*
	(0.0806)	(0.0722)	(0.0700)	(0.0606)	(0.0924)
Male*Age of Child	0.0783***	0.0280	-0.0331***	0.0240	0.0364
	(0.0186)	(0.0202)	(0.0114)	(0.0146)	(0.0297)
Male*(Age of Child)^2	-0.00497***	-0.00372***	0.00172***	-0.00154**	-0.00196
	(0.000953)	(0.00126)	(0.000447)	(0.000760)	(0.00178)
Constant	1.104***	0.417***	0.665***	3.021***	2.674***
	(0.0588)	(0.0510)	(0.0496)	(0.0450)	(0.0672)
Observations	14,010	18,650	21,253	26,476	6,581
R-squared	0.185	0.114	0.111	0.321	0.247

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1