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Intergenerational Transmission of BMI and its Effects on Cognitive Skills

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Preface

In the last decades, obesity has become a real health plague in most of the developed and developing world, with serious economic and social consequences. Due to this reason, also the economists and social scientists recently focused their attention on this problem.

The first chapter of this dissertation presents data that give an idea of the size of the obesity epidemics and surveys the medical, economic and social literature that deals with the causes and consequences of weight excess, both at the individual and macro level.

In the second chapter, we shed new light on the intergenerational transmission of BMI and weight problems and study how the family environment, in particular parental education, influences this process.

Thanks to the longitudinal structure of our data, we can follow individuals over time, from their childhood to adulthood, exploiting information about their BMI when 10, 16 and 34 years old and therefore studying the persistence of parental influence.

We provide for the first time estimates of the intergenerational elasticity of BMI between *both* parents and their children and we find that it is extremely stable to the introduction of a large set of individual and family controls, suggesting a strong role for genetics in the transmission of weight. On the contrary, parental schooling seems to exert a minor influence on the persistence of BMI: only maternal education has a protective role, but exclusively for sons' BMI.

In the last chapter, we use again data from the BCS70 and we investigate whether obesity influences individuals' cognitive ability when 10 years old and their literacy and numeracy skills at 34 years. In order to understand whether this relationship is causal, we employ instrumental variables, using both parents' BMI as instruments for cohort members' Body Mass. We perform our analysis using also dummies describing individual weight status instead of the continuous BMI variable and we exploit information about individuals' BMI at different ages to study this issue from a dynamic point of view. Our results show that weight excess has a significant negative causal effect on both cognitive ability and basic skills.

Contents

Chapter 1

Causes and Consequences of Weight Excess: Data and Review of the Literature

1.1 Introduction

The surge in overweight and obesity rates has risen at the top of the public health policy agenda in recent decades, not only in the OECD area but also in developing countries, becoming one of the major public health concerns of the XXI century. Yet in 1997, the WHO formally recognized obesity as a global epidemic and since then the prevalence of people suffering from weight excess has increased dramatically.

Once considered a problem of high-income countries only, obesity now affects both the developed and developing world, especially in urban areas. The only remaining region where obesity is not common is sub-Saharan Africa.

A number of behavioural and environmental factors have contributed to the rise of weight problems in industrialised countries, including falling real prices of food and more time spent being physically inactive, because of an increased reliance on cars and mechanized manufacturing.

As it is well known, obesity is a *"key risk factor for numerous health problems, including hypertension, high cholesterol, diabetes, cardiovascular and coronary-heart diseases, respiratory problems (asthma), musculoskeletal diseases (arthritis) and some forms of cancer. Mortality risk also increases sharply once the overweight threshold is crossed*" (Health at a Glance 2011, OECD).

For this reason, obesity is the fifth risk factor for death in the world, causing every year the death of 2.8 million of adult individuals (WHO, 2008). Severely obese people die 8-10 years earlier than those of normal weight, a value similar to that for smokers (OECD Health Division, 2010). Moreover, psychological problems associated with weight excess are common and potentially serious.

As a consequence, obesity has substantial direct and indirect costs that put a strain on healthcare and social resources: according to the WHO estimates, obesity is currently responsible for about 2-8% of total health expenditure in Europe.

Definition

Overweight and obesity are defined as excessive weight presenting health risks because of the high proportion of body fat. In the social science literature, fatness has almost universally been measured using the Body Mass Index (BMI), since its computation requires only information about weight and height, which is fairly common in social sciences datasets.

The BMI is indeed a measure of a person's weight relative to her height, that correlates fairly well with body fat (see Figure 1) and it accepted as the most useful measure of obesity when these two data only are available. In particular, it is calculated as the result of dividing body weight (in kilograms) by body height (in meters) squared.

Based on the WHO classification, the following clinical weight classification is adopted:

- BMI less than 18.5: underweight;
- BMI between 18.5 and less than 25: normal weight
- BMI between 25 and less than 30: overweight;
- BMI equal or greater than 30: obese.

However, it is important to remember that "*this classification may not be suitable for all ethnic groups, who may have equivalent levels of risk at lower or higher BMI*" and "*the thresholds for adults are not suitable to measure overweight and obesity among children*" (Health at a Glance, 2011). The healthy BMI range varies with the age and sex of the child: overweight and obesity in children and adolescents are defined respectively as a BMI greater than the 85^{th} and 95^{th} percentiles of the weight distribution in the reference population.

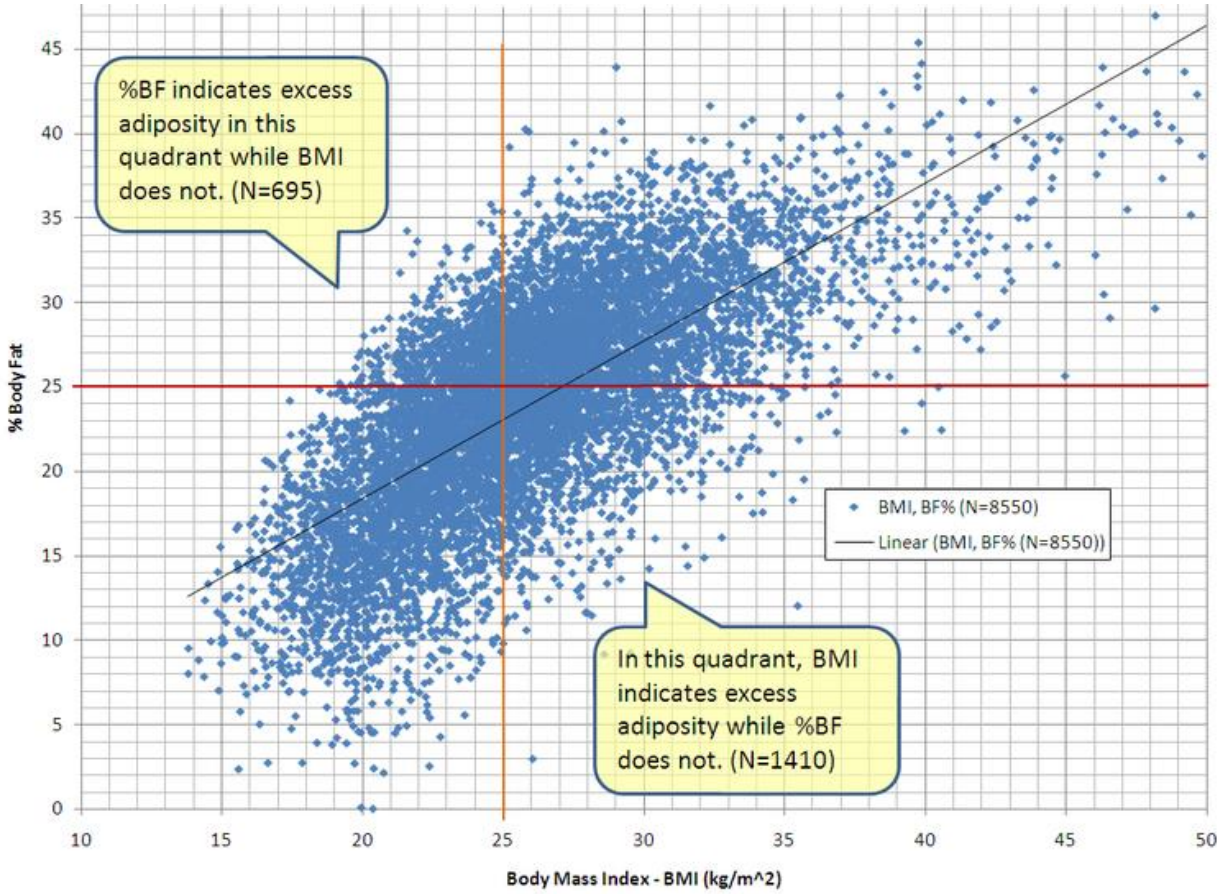
Despite its widespread use in social sciences, within the medical literature BMI is considered a limited measure of fatness and obesity because it is not able to distinguish the body composition. In particular, it cannot distinguish between fat and fat-free mass, such as muscle and bone, overestimating fatness among those who are muscular.

More accurate measures of fatness are for example the total body fat, the percentage of body fat and the waist circumference.

The use of different measures of obesity may lead to substantially different results. Burkhauser and Cawley (2008), show that when more accurate measures of fatness are used, "*the gap in obesity between white and African American men increases substantially, with white men significantly more likely to be obese*", while "*the gap (...) between African American and white women is cut in half*" with African American women still significantly more likely to be obese.

Moreover, they demonstrate that these different measures can be differently correlated with social science outcomes. For example, while BMI is positively correlated with the probability of employment disability, when body mass is divided into its components, fatness is positively associated with disability while the fat-free mass (such as muscle) is negatively correlated.

Figure 1 – Correlation between BMI and the percentage of body fat (in NHANES data)



1.2 Data

1.2.1 Adult Population

Looking at the world population in 2008, more than 1.6 billion of adults (> 20 years old) were overweight, among which 500 million were obese (200.000 males and 300.000 females) (Source: WHO). More recent data from the World Watch Institute (WWI) suggest that 1.9 billion of adult individuals suffered from weight excess in 2010.

Unfortunately, according to the WHO estimates, these numbers will raise even more: within 2015, 2.3 billion of the adult population will be overweight, with more than 700 million of them being obese.

Even if the situation is different from country to country, the general trend is similar and it is consistent with a sharp increase of weight problems all over the word.

OECD Area

Obesity rates have doubled or even tripled in many countries since 1980 and, according to the latest available OECD survey (OECD Health Data 2013), more than half of the adult population in the area reports that they are overweight or obese. Among those countries where height and weight were measured, the proportion is even greater, at 55.8%.

Even if weight problems are generally widespread in most of the countries (the prevalence of overweight and obesity among adults exceeds 50% in at least 19 of the 34 OECD countries), the situation is heterogeneous, with States like Japan and Korea and some European countries (France and Switzerland) where overweight and obesity rates are much lower, although increasing.

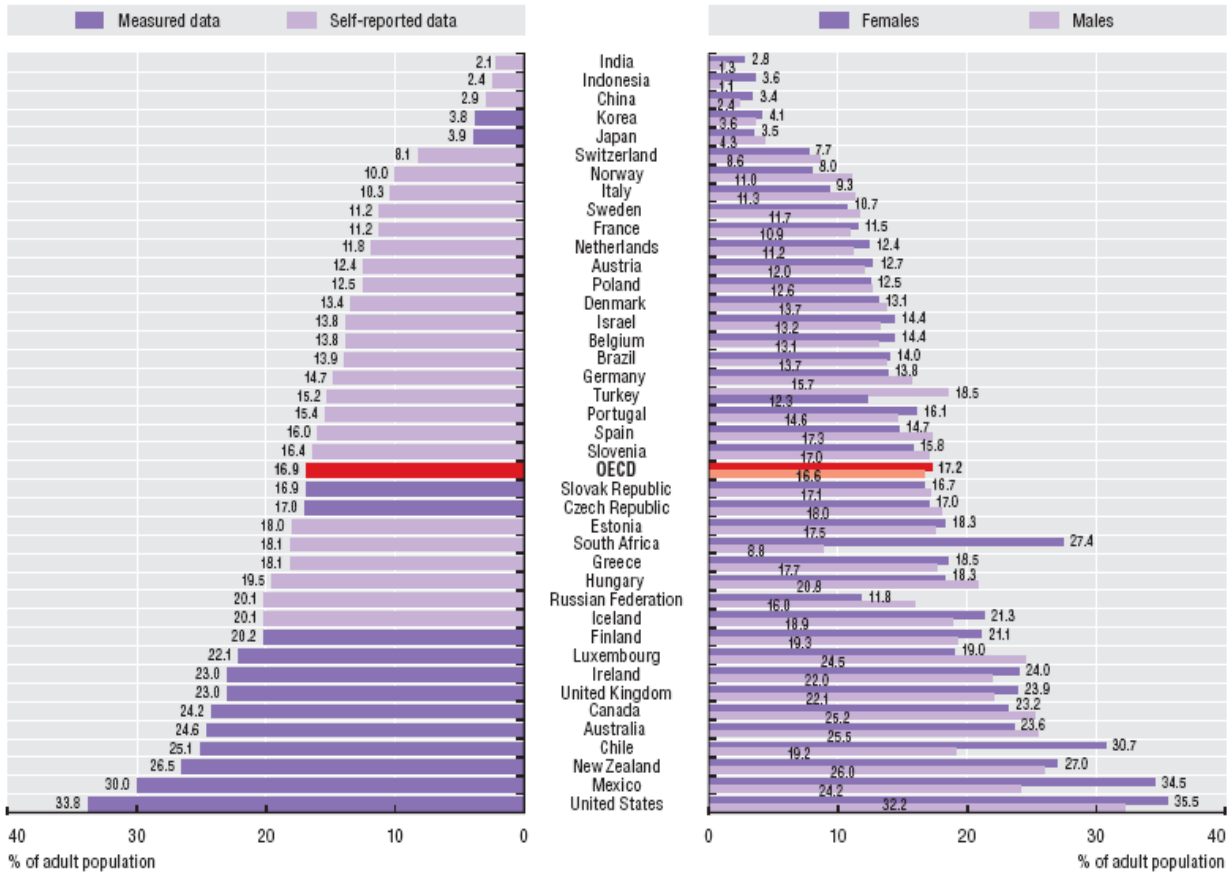
The same holds when we focus on the prevalence of obesity only: the average rate of obesity in the adult population across the entire OECD region is 17%. However, rates vary nearly tenfold among countries: from less than 4% in Japan and Korea, to 30% or more in the United States and Mexico.

Average obesity rates among men and women are similar, with the exception of South Africa, Chile, Turkey and Mexico where a greater proportion of women are obese and the Russian Federation, Luxembourg and Spain where males are much fatter than females.

The most alarming data are those concerning the rise of obesity prevalence: in the last 20 years the share of obese individuals has doubled in almost all the OECD countries for which data are available, with the exception of Czech Republic, U.K. and U.S. where it has increased by half. Obesity rates have also grown substantially over the past decade in some European States like Norway, Iceland and Luxembourg and in other countries as Mexico and New Zealand.

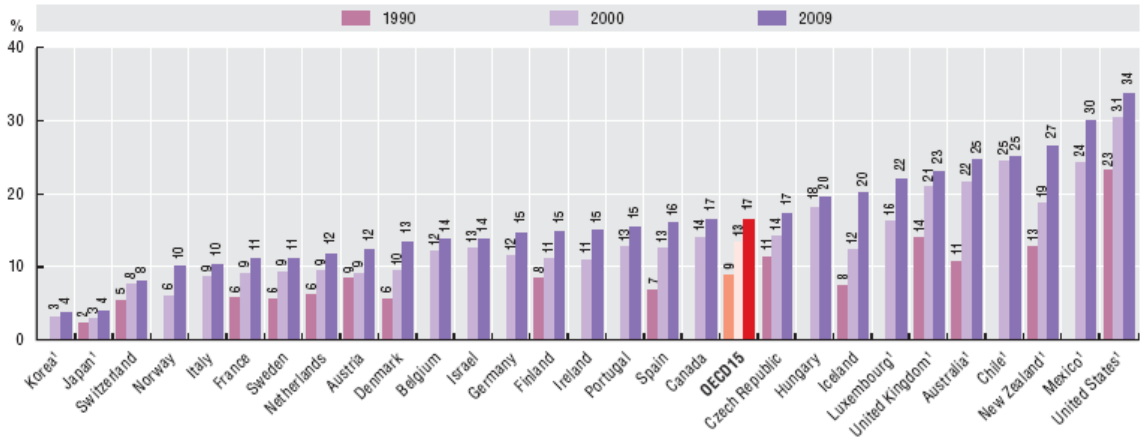
The rise in obesity has affected all population groups, regardless of sex, age, race, income or education level, but to different extents. In particular, recent studies have shown that obesity tends to be more common among individuals in disadvantaged socio-economic groups (Sassi et al., 2009) and among low-educated individuals (Sassi, 2010). In both cases the gradient in obesity is stronger in women than in men.

Figure 2 – Prevalence of Obesity among adults, 2009 (or nearest year)



Source: Health at a Glance 2011 (OECD Health Data 2011; national sources for non-OECD countries)

Figure 3 – Increasing obesity rates among the adult population in OECD countries, 1990, 2000 and 2009 (or nearest years)



1. Data are based on measurements rather than self-reported height and weight.

Source: Health at a Glance 2011 (OECD Health Data 2011)

Europe

As mentioned above, Unites States are the developed country mostly affected by weight problems, with the surprising share of 78.6% of the adult population being overweight (WWI, 2010) among which 35% are obese. However, the situation is now getting worse and worse also in Europe. According to the European Health Interview Surveys (EHIS), carried out by Eurostat in 2008: *"Weight problems and obesity are increasing at an alarming rate: over the last decade the proportion of the population that is overweight has increased considerably in most Member States, resulting in more than half the EU population being overweight or obese"*.

More precisely, the number of obese people in Europe (EU27) is increased by three times since 1980s and it is still raising. More than one half of the male population (with the exception of Estonia, Latvia and Romany) is overweight or obese (Source: International Association for the Study of Obesity, 2008). According to the Eurostat data released in 2008, among the 19 Member States for which information are available, the proportion of overweight and obese people in the adult population varied between 36.9 % and 56.7 % for women and between 51 % and 69.3 % for men.

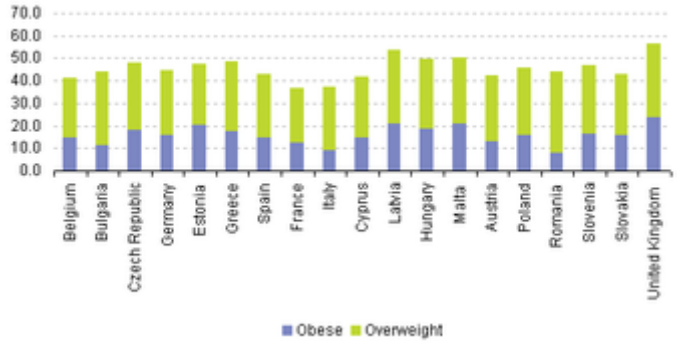


Figure 4: Overweight and obesity - women, 2008 (%) - Source: Eurostat

Figure 5: Overweight and obesity - men, 2008 (%) - Source: Eurostat

The worst data are those of U.K. where about 23% of adult men and women are obese.

There is no systematic difference in obesity between women and men across Member States: the proportion of obesity was higher for women in eight Member States, higher for men in ten and equal in one. However, for overweight there is a clear gender difference: in all the Member States the proportion of overweight individuals is much higher among men than among women.

It is interesting to notice that, like in the OECD data, the share of overweight and obese persons tends to fall with educational level[1]. This is especially true for females: the proportion of women who are obese or overweight falls as the educational level rises and the difference between lower and upper education level varies between 12.8 and 36.7 %.

For men, the evidence is less clear: differences are smaller and the distribution is different. In 8 of the available Member States, the highest share of overweight and obese men is observed for those with the lowest educational level, in 6 M.S. for those with a medium educational level while in 4 countries it is for those with a high educational level.

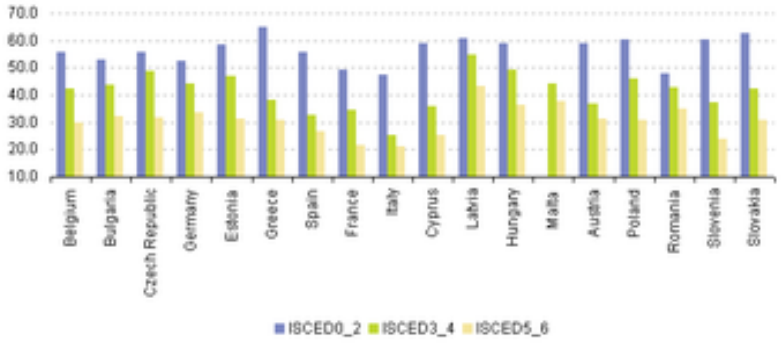


Figure 6: Overweight + obesity - women (%) - by educational level - Source: Eurostat

[1] The groups of educational level attained are defined according the International standard classification of education (ISCED), version of 1997:

- low level of education refers to pre-primary, primary and lower secondary education (ISCED level 0-2);
- medium level to upper secondary and post-secondary non-tertiary education (ISCED level 3-4);
- high level to tertiary education (ISCED level 5-6).

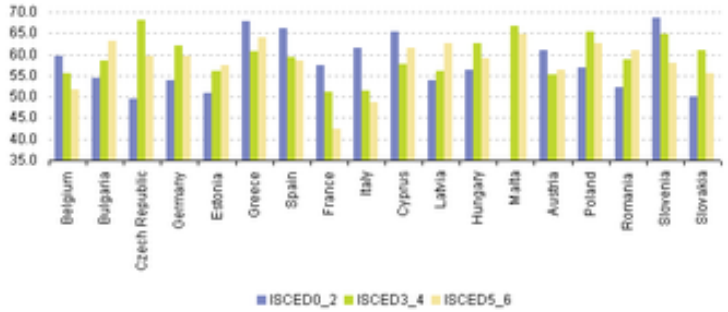


Figure 7: Overweight + obesity - men (%) - by educational level - Source: Eurostat

Finally, we can observe that the share of overweight and obese persons increases with age. For women there is a clear pattern in all the available Member States: the older the age group, the higher the share of overweight and obese persons. For men, the trend is slightly different: the increase in overweight and obesity is systematic till 65 years, while for the age group 65-74 the picture is less uniform.

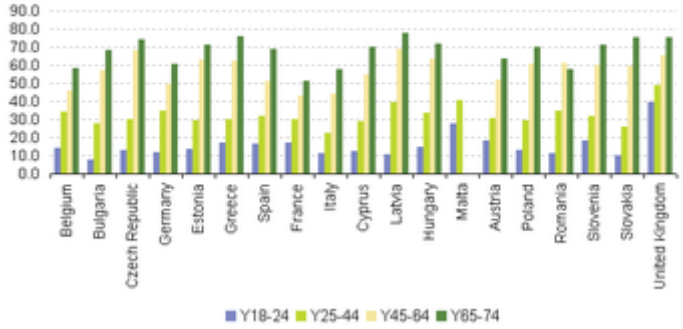


Figure 8: Overweight + obesity - women - by age (%) - Source: Eurostat

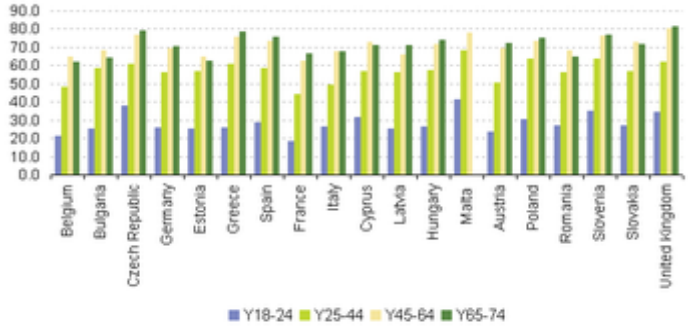


Figure 9: Overweight + obesity - men - by age (%) - Source: Eurostat

1.2.2 Childhood Obesity

Obesity and overweightness are especially dangerous for health when they afflict young people. Indeed, as pointed out in the OECD survey Health at a Glance (2011):

"*Children who are overweight or obese are at greater risk of poor health, both in adolescence and in adulthood. Being overweight in childhood increases the risk of developing cardiovascular disease or diabetes, as well as related social and mental health problems. Excess weight problems in childhood are associated with an increased risk of being an obese adult, at which point certain forms of cardiovascular diseases, cancer, osteoarthritis, a reduced quality of life and premature death can be added to the list of health concerns*"

Moreover, "*even if excess childhood weight is lost, adults who were obese children retain an increased risk of cardiovascular problems*".

Last but not least, overweight and obese children may suffer from psychological problems and disorders, because they are victims of discriminating behaviours.

According to the WHO, 43 millions of children below 5 years in the world are overweight, 35 million of which live in developing countries.

OECD Area

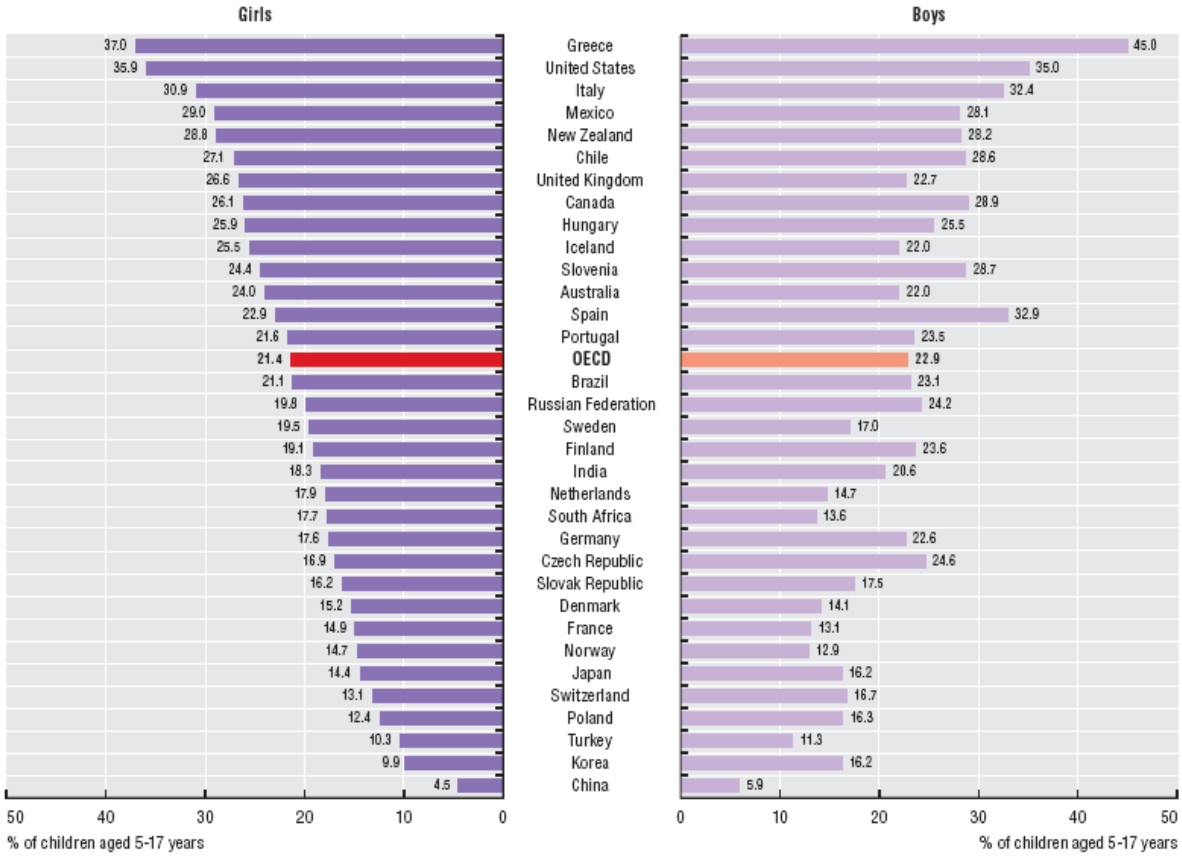
Childhood overweight and obesity has risen fast in the last decades, reaching double-figure rates in most OECD countries.

About 20% of children aged 5-17 years are affected by excess body weight across all countries, with Greece, United States and Italy being the States where the problem is most serious, reaching peaks of almost 30%. Only in China, Korea and Turkey the share of overweight children is less than 10%.

In most countries, boys have higher rates of overweight and obesity than girls with the exception of Nordic countries (Sweden, Norway, Denmark), as well as the United Kingdom, the Netherlands and Australia.

As for adults, the most impressive thing is the rapid increase in the rates of overweight among boys and girls: in many developed countries, child obesity levels doubled between the 1960s and 1980s, and have doubled again since then. Even in emerging countries, the prevalence of obesity is rising, especially in urban areas, characterized by more sedentary life-style and a easier access to energy-dense foods.

Figure 10 - Children aged 5-17 years who are overweight (including obese)



Source: Health at a Glance 2011

Europe

Focusing attention on Europe (EU27) only, Eurostat data asses that one out of five young person in Europe was overweight in 2008, for a total of 14 million (3 million of which were obese). Moreover, time-series suggest that every year this number is rising by 400.000 units. The worst data are again those of UK where 29% of male and female between 5 and 17 years are overweight. Also the situation of other Mediterranean countries (Italy, Spain, Cyprus and Malta) and of Portugal is worrying. Boys are in general more overweight than girls. On average, 16% of 11-years old boys are overweight or obese against 12% of girls. Among 13-years old guys 16% of males and 10% of females have weight excess, while among 15-years old these percentages are 17% and 10% respectively.

The data about childhood obesity are of particular interest because it is estimated that more than 60% of the children that are overweight before adolescence will be overweight when adult (Source: Health Behaviour in School-aged Children, HBSC 2005-06).

1.3 Potential determinants of obesity

Like many other medical conditions, obesity is the result of an interplay between genetic and environmental factors.

The percentage of obesity that can be attributed to genetics varies, depending on the population examined, from 6% to 85% (Yang, Kelly and He, 2007). Genetic predisposition eases the arising of weight problems in the presence of a sufficient calories intake. Medical researches (e.g. Poirier, Giles, Bray et al., 2006) have shown that the polymorphisms in various genes controlling appetite and metabolism are linked to the development of obesity when a favourable environment is present. Other studies, like Loos and Bouchard (2008), highlight the importance of FTO (fat mass and obesity associated gene) as an obesity-susceptibility gene: people with two copies of the FTO gene weight on average 3-4 kg more and have a 1.67-fold increased risk of obesity compared with those who did not inherit a risk allele.

While genetic influences are important to understanding obesity, they cannot explain the current dramatic increase seen within specific countries or globally. This is the result of multiple and interacting dynamics, which have led to a substantial change in people's lifestyle in relation to nutrition and physical activity.

Independently from genetic predisposition, weight excess arises when there is a lack of balance between calories intake and calories burned: a combination of excessive food energy intake and sedentary lifestyle, characterized by little physical activity, explain most cases of obesity.

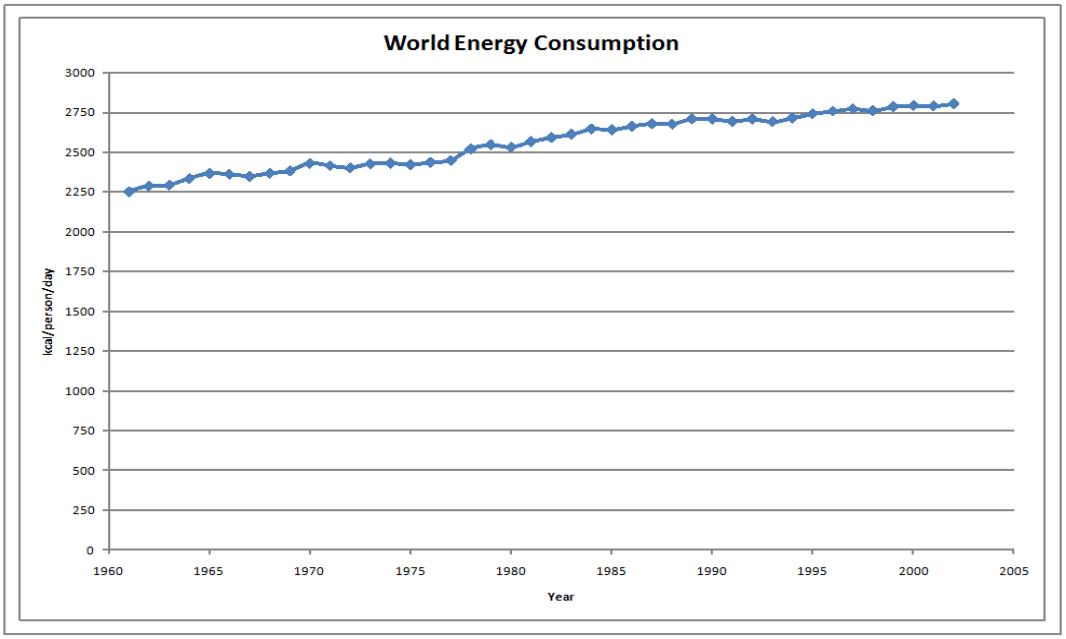
Even if the average calories available per person per day (the amount of food bought) varies markedly between different regions and countries, from the early 1970s to the late 1990s it has increased significantly in all parts of the world, except Eastern Europe (see Figure 5).

Most of this extra food energy comes from a rise in carbohydrate (corn, wheat and rice) and sugar consumption (in particular snacks and sweetened drinks) rather than fat consumption (Wright et al., 2003).

The introduction of new agricultural techniques and subsidization policies in the United States and Europe has lowered the prices of cereals, making them the main source of cheap food. However, the main responsible of overeating and poor dietary choices is likely to be the large consumption of energy-dense, big-portions and fast-food meals, the so called "junk-food" (Rosenheck, 2008).

The widespread availability of nutritional guidelines has done little to address this problem (Marantz, Bird and Alderman, 2008).

Figure 5 – The rise in pro-capita calories consumption per day



Source: World Resources Institute

Also a sedentary lifestyle plays a significant role in promoting obesity. Worldwide there has been a large shift towards less physically demanding work and, according to the WHO, globally, around 31% of adults were insufficiently active in 2008 (men 28% and women 34%). The prevalence of insufficient physical exercise was highest in the WHO Region of the Americas and the Eastern Mediterranean Region. In both these regions, almost 50% of women were insufficiently active, while the prevalence for men was 40% in the Americas and 36% in Eastern Mediterranean. The South East Asian Region showed the lowest percentages (15% for men and 19% for women).

Declining physical activity levels are primarily due to the increasing use of mechanized transportation and a greater prevalence of labor-saving technology both at the work-place and at home, causing sedentary behaviour during occupational and domestic activities. Also increased urbanization has resulted in several environmental factors that may discourage the participation in physical activity during leisure time, like high-density traffic, low air quality and pollution and the lack of parks, sidewalks and sports/recreation facilities.

Another determinant that have contributed to the reduction of the time devoted to physical exercise during leisure is the diffusion of television and computers: several studies, like Tucker and Bagwell (1991), Gortmaker et al. (1996) and Vioque, Torres and Quiles (2000) have found a strong positive correlation between television viewing time and the risk of obesity.

Minor potential contributors to the recent increase in obesity rates are (Keith, Redden, Katzmarzyk et al. 2006):

1. Insufficient sleep
2. Edocrine disruptors (in particular, environmental pollutants that interfere with lipid metabolism)
3. Malnutrition in early life. This is believed to play a key role in the rising rates of obesity in the developing world. Endocrine changes that occur during periods of malnutrition may promote the storage of fat once more food energy becomes available (Caballero, 2001)
4. Decreased rates of smoking. Smoking has a significant effect on an individual's weight, since it suppresses appetite. Those who quit smoking gain an average of 4.4 kg for men and 5.0 kg for women over ten years (Flegal et al., 1995). However, changing rates of smoking have little effect on the overall rates of obesity.
5. Increased use of medications that can cause weight gain (e.g., antipsychotics)
6. Proportional increases in ethnic and age groups that tend to be heavier
7. Pregnancy at a later age (which may cause susceptibility to obesity in children)
8. Epigenetic risk factors passed on generationally
9. Natural selection for higher BMI
10. Assortative mating leading to increased concentration of obesity risk factors

Education may have an important role in shaping individuals' health behaviours and, therefore, weight status and other health outcomes. This influence may be exerted via direct and indirect channels, that we are discussing the following paragraph.

1.3.1 Education and Health: Background

The discussion about the potential effects of education on weight can be generalized to almost all the individual's health outcomes.

Education is recognized to be a key factor in promoting health: policies designed to increase years of schooling for at-risk populations have led to substantial health improvements in many countries. For this reason, raising the general education level and increase information and prevention policies at the school-level is often one of the primary public interventions suggested to address also the issue of obesity.

It is indeed well known that education provides benefits that go far beyond the labor market productivity, ensuring higher wages and employment rates. Many contribution have shown and proved also the non-monetary returns of education: the traditional return to schooling estimates that only focus on monetary earnings greatly underestimate the benefits of schooling.

Increase in education leads for example to crime reduction (Lochner, 2004; Lochner and Moretti, 2004) and increase in voting and democratic participation (Barro, 1999; Glaeser et al., 2007; Acemoglu et al. 2005). Even a positive impact on happiness seems to exist (Oreopolous, 2007).

Economists and social scientists has focused their attention also on the potential link existing between education and health outcomes, showing a large and persistent relationship between schooling and health, that they called "Education Gradient".

The "Education Gradient" measures the effect of one more year of education on the particular measure of health under analysis (in the regression of a measure of an individual's health on the years of schooling and other covariates, it is the coefficient of the former).

This association has been observed in many countries and time periods and for a wide variety of health measures:

- Mortality rates (Kitagawa and Hauser, 1973; Cutler, Deaton and Lleras-Muney, 2006; Elo and Preston 1996; Lleras-Muney, 2005; Albouy and Lequien, 2009)
- Hospitalization (Arendt, 2008)
- Risk factors (Berger and Leigh, 1988; Sobel and Stunkard 1989, Adler et al., 1994)
- Long-term illness and chronic diseases (Kempter, Juerges and Reinhold, 2011; Fonseca and Zheng, 2011)
- Health behaviours (Kenkel, 1991; Cutler and Lleras-Muney, 2010; De Walque, 2007)
- Self-reported measures of health (Brunello, Fort, Schneeweis and Winter-Ebmer, 2011; Lundborg, 2008)
- Obesity and overweightness (Brunello, Fabbri and Fort, 2013 Grabner, 2009 MacInnis, 2006; Spasojevic, 2010)

1.3.2 Why education might affect health?

The obvious economic explanations -education is related to income or occupational choice- explain only a part of the education effect.

Increasing levels of education lead also to different thinking and decision-making patterns: schooling helps people to choose healthier life-styles by improving their knowledge about the relationships between health behaviors and health outcomes.

More in detail, there are three main potential channels through which education may influence health:

- *Income Effect*: education generally increases earnings, which makes costly health cares and health insurance purchase more affordable. Moreover, higher income increases consumption opportunities

and thus raises the demand for health. Last but not least, the availability of greater financial resources allow individuals to buy high-quality food, practice sport activities and therefore follow an healthier lifestyle.

- *Allocative Efficiency* (better allocation of health inputs): education may enhance the individual's ability to acquire and process health information or to follow more appropriate treatments (Rosenzweig and Schultz, 1982). Knowledge about more health options or inputs can improve health allowing the individuals to take advantage of more productive or less costly inputs;
- *Productive Efficiency* (better health production given a set of inputs): education may directly increase health production by raising the marginal productivity of health inputs or behaviors. Indeed, education may improve decision-making abilities, which should lead to better health decisions and more efficient use of health inputs (Grossman, 1972).

Among these three channels we can identify several mechanisms through which schooling might affect health:

1. **Income**: education may improve health simply because it results in greater resources, that allow the individual to spend more to remain healthy. As mentioned above, more educated individuals are more likely to spend more on costly treatments or to purchase better health insurance. Moreover, greater resources allow to lead a healthier lifestyle, spending more money in healthy food and exercising activities.
2. **Labor market**: highly educated individuals may have better jobs that, in addition to paying higher incomes and providing health insurance, offer safer work environments.
3. **Rank**: education might matter for health because it changes one's relative position or rank in society and rank by itself might affect health. Indeed, individuals at the lower end of the hierarchy have lower control over their lives and are constantly subjected to arbitrary demands by others, causing increases in stress, anxiety and depression and therefore in stress-related diseases.
5. **Social networks**: more educated individuals have larger social networks which provide financial, physical and emotional support and may in turn have a causal effect on health. Beyond support, friends and family provide peer recognition or disapproval. If more educated people have more educated friends, who are more likely to behave healthily and value health, the peer effects of networks exacerbates the effect of own education, acting as a multiplier effect.
5. **Information, cognitive skills and critical thinking**: education provides individuals with better access to information and improved critical thinking skills (although of course those with higher skills may also be more likely to get more education). The more educated appear to be better informed and to make use of new health related information first.

This allows more educated individuals to choose healthier lifestyle, including taking greater safety precautions, eating a healthier diet, drinking alcohol in moderation, not smoking and exercising more. However, today most individuals are well aware of the dangers associated with bad health behaviors which are more prevalent among uneducated. Therefore, education might matter for health not just because of the specific knowledge one obtains in school, but rather because it improves general skills, including critical thinking skills and decision-making abilities.

6. **Value of the future (discount rates) and preferences**: if education provides individuals with a better future along several dimensions, because it gives access to more income, it makes one happier and generally improve one's outlook of the future, people may be more likely to invest in protecting that future. In economic terms education increases the present discounted value of future lifetime utility. As income raises, willingness to pay for health improvements increases as well.

7. **Availability of knowledge and technology to prevent or treat diseases**: individuals with more education (or more income) are likely to use new knowledge and new techniques more rapidly and more effectively. This idea is consistent with the hypothesis that education gives an individual a wide range of serviceable resources, including money, knowledge, prestige, power and beneficial social conditions that can be used to one's health advantage.

All of the mechanisms listed above provide a potential explanation for explaining the link between schooling and health status but none of them is able to explain alone the entire size of the education gradient. Moreover, the relative magnitude of the different effects is unknown.

However, the most important issue to be solved is probably to understand whether the link between education and health is *causal*. Indeed, it is worth recognizing that education may be *correlated* with health even if there is no causal effect of schooling on the individual's health status. Reverse causality might indeed occur: healthier individuals are likely to be more efficient producers of human capital through schooling. More generally, unobservable individual characteristics, such as genetic endowments and family background, might simultaneously affect both education choices and health-related behaviors.

1.3.3 Is the effect of education on health outcomes causal?

To assess whether education has a causal effect on health outcomes the concerns about the *endogeneity of schooling* and the *unobserved heterogeneity in individuals' preferences* must be addressed.

The estimation techniques usually applied in order to face these problems are instrumental variable (IV) estimation and Regression Discontinuity (RD) techniques.

Most of the studies applying these methods use changes in compulsory schooling laws as instruments for educational attainment. School leaving age reforms provide an exogenous source of variation in schooling that is independent from individuals' unobservable characteristics and allow to see whether individuals who were forced to go to school through various policies have subsequently better health outcomes than those who were not. In other words, these reforms provide a quasi-natural experiment that allows to recover identification

Among the studies exploiting change in compulsory schooling leaving age as an IV we include Mazumder (2008), Llearas-Muney (2005), Clark and Royer (2010), Albouy and Lequien (2009), Oreopoulos (2006), Silles (2009), Kemptner, Jurges and Reinhold (2011), Brunello, Fabbri and Fort (2013).

However, also different types of instrumental variables have been employed in the health-economics literature. Among the others, we remember changes in school entry age laws (McCrary and Royer, 2009), the construction and openings of new schools or colleges (Currie and Moretti, 2003), changes in high school and GED requirements and policies (Kenkel, Lillard and Mathios, 2006) and, finally, even the risk of induction in the Vietnam War (De Walque, 2007; Grimard and Parent, 2007; MacInnis, 2006).

The studies mentioned in this paragraph investigate the impact of schooling on several different health outcomes:

- survival/mortality rates
- birth weight
- self-reported health status
- number of nights in hospital in last year
- long illness
- disability/limited mobility or activity
- hypertension
- obesity and overweight

Of course, we are mainly interested in the last of the health outcomes listed above and, from now on, we are going to focus our attention only on it. However, since the channels through which individuals' weight status is affected by their education level are the same we described in the previous paragraph for health outcomes in general, we are not going to discuss again the factors driving the influence of schooling on body mass.

Therefore, in the following, we first present a brief survey of the studies investigating the relationship between education and weight status (a deeper review will be presented in the second

chapter of the thesis) and then we will focus our attention on the explanation of the potential mechanisms linking parental educational level with their children body mass.

Table 1 – Studies investigating the *causal* effect of education on health outcomes

Paper	**Health Outcome**	**Data**	**Identification Strategy**	**Results**
Brunello, Fort, Schneeweis and Winter-Ebmer (2011)	Self-reported health and health behaviors	12 European countries	- IV: School Leaving age Reform (SLAR) - ADS Model (Card & Rothstein approach)	- Positive causal effect of education on self-reported health - Health behaviors contribute to explain the education gradient
Silles (2009)	Multiple measures of overall health	UK	IV: SLAR	Positive causal effect of education on health
Arendt (2008)	Hospitalization	Denmark	IV: SLAR	Protective causal effect of education on the probability of being hospitalized
Lundborg (2008)	- Self-reported health - Chronic conditions	USA	Sample of twins	- Positive causal effect of education on self-reported health - Negative causal effect of education on the number of chronic conditions
Lleras-Muney (2005)	Mortality rates	USA	IV: SLAR	Negative causal impact of education on mortality
Albouy and Lequien (2009)	Mortality rates	France	IV: SLAR	No significant effect of education on mortality rates
De Walque (2007)	Smoking behaviours	USA	IV: Vietnam War Draft Avoidance	Education affects smoking decisions: educated individuals are less likely to smoke, and among those who initiated smoking, they are more likely to have stopped.
Oreopoulos (2006)	Self-reported health	USA, UK, Canada	IV: SLAR	Students compelled to stay in school are less likely to report being in poor health
Fonseca and Zheng (2011)	- Self-reported health - Prevalence for diabetes and hypertension, cancer and other chronic conditions	13 OECD countries	IV: SLAR	Mixed evidence: - more education leads to lower probability of reporting poor health and lower prevalence for diabetes and hypertension; - however, positive relationship between education and cancer and insignificant impact on other chronic conditions

Juerges, Kruk and Reinhold (2010)	- Self-reported health - Biomarkers	UK	IV: SLAR	- Ambiguous causal effects of schooling on women's self-rated health - Insignificant causal effects on men's self-rated health and on biomarker levels in both sexes
Mazumder (2008)	- Mortality - Several long-term health outcomes	USA	IV: SLAR	No evidence for a causal link between education and health
Currie and Moretti (2003)	- Pre-natal health behavior Birth outcomes	USA	IV: openings of colleges by county	An extra year of college reduces the likelihood of low birth weight children, pre-term birth and smoking during pregnancy.

1.3.4 Education and obesity

The rise in obesity rates over the past few decades presents a major challenge for public health policy. Raising general education levels is one of the primary public interventions suggested to address this issue (Grabner 2008).

Several studies have investigated the relationship between individual's educational level and their weight status. However, the evidence they provide about the existence of a causal effect of the former on the latter is unclear.

Brunello, Fabbri and Fort (2013) focuses on the influence of education on the BMI and the probability of being overweight or obese of European females in an Instrumental Variables (IV) framework that uses the variation caused by compulsory school reforms implemented in European countries after the second World War as instrumental variables for education.

Their OLS estimates reveals the existence of a negative association between the BMI and years of schooling. The IV coefficients are always larger in size than OLS ones and the size of the estimated effect is not negligible: a 10% increase in years of schooling reduces females BMI by a minimum of 1.65 to a maximum of 2.27%.

Moving attention from the continuous BMI variable to a dummy indicating overweight, their evidence suggests that a 10% increase in years of schooling reduces the probability of being overweight by 6.71 to 6.82% when schooling is treated as exogenous and by 10.87 to 16.60% when it is treated as endogenous. Concerning obesity, IV estimates are imprecise and close to their standard probit estimates: a 10% increase in years of education reduces obesity by 11.57 to 16.70% when years of schooling are treated as endogenous.

A protective role of education on individuals' weight status is found also by Grabner (2009), using data from the first waves of the NANHES (US) and state-specific compulsory schooling laws between 1914 and 1978 as source of identification. He finds a strong and statistically significant negative effect of additional schooling on BMI. The effect is especially pronounced for females and it is larger than the one estimated by Brunello et al. (2013), suggesting that an extra year of education lowers a person's BMI by 1-4%. On the contrary, the estimated effect on the probability of being overweight or obese is smaller: a one-year increase in schooling reduces the probability of being obese by 2-4 percentage points. However, this result masks a considerable difference between genders, since the effect is again much stronger for women: an additional year of schooling is associated with a 6.5% reduction in overweight incidence and a 4.4% reduction in obesity incidence.

The evidence that the years of schooling have protective effect mainly on women BMI and obesity probability is a common finding in the literature. Also Braga and Bratti (2012) achieve this result exploiting the introduction of the Law n°1859 of 1962 in Italy as source of identification. Such a law, enforced in 1963, increased the length of compulsory schooling from 5 (primary education) to 8 years (5 years of primary education and 3 years of lower secondary education) and rose school-leaving age to 15, requiring individuals to attend school at least until graduation from lower secondary education. Using the eligibility for the 1963 reform as an instrumental variable in a "fuzzy" regression discontinuity design, they find that lower secondary schooling contributes to reducing women's BMI by 3.3 points, corresponding to -12.8%. As lower secondary schooling consists in Italy of three years of education, this corresponds to about a -4.3% reduction in BMI for every additional year of education, a magnitude which in line with that reported by Grabner (2009) for US women. Coherently, women's likelihood of being obese falls by 24%.

On the contrary, the estimated effect for males is much lower than for women, and never statistically significant.

A different identification strategy but similar results are found by MacInnis (2006) who exploits the close tie between the age of college attendance and the draft during the Vietnam war period as a quasi-natural experiment to be used in a regression discontinuity research design. In particular, in order to infer causation from education to obesity, he takes advantage of the common behavior among America guys to enrolling in college to avoid the draft. This draft avoidance behavior generates a strong instrument for post-secondary education, causing about a 5% increase in college completion, but has no effect for grades below high school.

The identification condition is that individuals have some, but not complete, control over their chance of completing college.

Using the National Health Interview Survey 1998–2003, they found that college education reduces the probability of obesity by about 40-60% and obesity co-morbidities such as hypertension, diabetes, and dental loss by about 30-60%.

On the other hand, Jurges, Reinhold and Salm (2009) do not find that education generally reduces weight problems. They use as an instrument for the years of education the number of academic track schools per square km in the state of residence when the respondent was age 10. During the post-war period German states started to build new academic track schools in order to increase the share of young people obtaining a university entrance diploma. The timing of the educational expansion differed between states, creating an exogenous variation in the availability of higher education, which allows estimating the causal effect of education on weight problems. Contrary to the previous studies, they find no causal effect of education on reduced overweight and obesity. Rather, their estimates indicate that education increases the probability of overweight and obesity. The authors justify this surprising result claiming the attendance of an academic track school might be related to changes in lifestyle that can increase the risk of weight excess such as a switch from manual blue collar work to more sedentary white collar employment.

Little evidence that education is associated with a lower probability of being overweight or obese for either men or women is provided also by Kenkel, Lillard and Mathios (2006). They use data from the National Longitudinal Survey of Youth 1979 to explore the relationships between high school completion and obesity controlling for several family background measures and individuals' cognitive ability. They find that high school completion seems not to be associated with the probability to suffer for weight problems and that health returns to GED receipt are much smaller than the returns to high school completion. When they repeat their analysis using as instruments for individuals' education variations in the State educational policy environment, in particular high school graduation requirements and GED policies, OLS results are confirmed, even if their IV estimates tend to be higly imprecise.

Clark and Royer (2010) try to overcome the difficulties associated with estimating the causal effect of education on health exploiting two changes to British compulsory schooling laws (in 1947 and 1972 respectively). These reforms generate a sharp differences in educational attainment among individuals born just months apart and allow to use regression discontinuity methods. They find that, even if the cohorts affected by these changes completed significantly more education than slightly older cohorts subject to the old laws, there is little evidence that this additional education improved health outcomes or changed health behaviors. In their analysis, they focus on a large set of health outcomes, using both subjective and objective measures of the health status. Among the objective measures they include individuals' BMI and probability of being obese and overweight.

While their OLS estimates suggest that education has beneficial effects on such outcomes, the instrumental variables estimates provide no evidence of a causal effect of education and weight excess. In particular, none of the IV estimates are statistically significant and moreover they often take the "wrong" sign.

The same approach is employed also by Arendt (2005), who reached identical conclusions. He uses data from the Danish National Work Environment Cohort Study (WECS) and exploits as instruments for education the Danish school reforms that took place in 1958 and 1975, that rose the minimum school leaving age to 14 and 16 years respectively. His OLS estimates confirm that education is negatively related to individuals' BMI. However, when instrumenting, he finds that, even if such relationship is amplified in magnitude, coefficients are no more statistically significant.

Table 2 – Studies investigating the *causal* effect of education on obesity

Paper	**Data**	**IV**	**Results**
Brunello, Fabbri and Fort (2013)	9 European countries	School Leaving Age Reform (SLAR)	- protective effect of education on female BMI but no effect on males BMI
Grabner (2009)	USA	SLAR	- negative causal effect of education on BMI, especially for females
Braga and Bratti (2012)	Italy	SLAR	- negative causal effect of education on females' BMI, but no significant effect for males
MacInnis (2006)	USA	Vietnam War Draft Avoidance	- negative causal effect of education on obesity and its co-morbidities
Jurges, Reinhold and Salm (2009)	Germany	Number of academic track schools in the region of residence	- negative effects of education on smoking - no causal effect of education on reduced overweight and obesity
Kenkel, Lillard and Mathios (2006)	USA	Variation in the State educational policy environment (high school graduation requirements and GED policies)	- causal link between education and the probability of smoking - little evidence of a causal link between education and the probability of being overweight or obese for either men or women
Clark and Royer (2010)	UK	SLAR	- no causal effect of education on health outcomes and health behaviors
Arendt (2005)	Denmark	SLAR	- no evidence for a causal link between education and health outcomes

1.3.5 Parental education and childhood obesity

As with adult obesity, wrong diet and decreased physical activity are believed to be the two most important determinants in causing the rising prevalence of weight problems among children and adolescents (see Dollman, Norton K. and Norton L., 2005).

There are several risk factors, which often act in combination, that may facilitate the inheritance of obesity. The most important one is the obesity of both parents. The studies by Kopelman (2005) and Kolata (2007) found that about 80% of the offspring of two obese parents were obese in contrast to less than 10% of the offspring of two parents who were of normal weight. This may be reflected by the family's environment and genetics.

As for adulthood obesity, childhood obesity is often the result of an interplay between many genetic and environmental factors. Mothers' over-weightiness predisposes their offspring to obesity by epigenetic, prenatal effects. Moreover, the transmission of polymorphisms in various genes controlling appetite and metabolism, that predispose individuals to obesity, may take place from parents to their children. As such, obesity may be the consequence of a number of rare genetic conditions that often present in childhood:

- Prader-Willi syndrome
- Bardet-Biedl syndrome
- MOMO syndrome
- Leptin receptor mutations
- Congenital leptin deficiency
- Melanocortin receptor mutations

As mentioned above, according to Yang, Kelly and He (2007), the percentage of obesity that can be attributed to genetics varies from 6% to 85% depending on the population analysed. Other studies, such as Comuzzie and Allison (1998), suggest that genes explain roughly one-half of the cross-sectional and temporal variation in individual weight. Individual environment and behaviours are responsible for the other half.

While genetics transmission is clearly independent from parents' educational level, there are several mechanisms through which parental schooling may affect children's weight. They can be classified into *direct* and *indirect* channels.

Parental education might have a *direct* impact on child BMI because it may result in better parenting, that is parents' ability to bring up their children, including the knowledge of what is good for their health. Indeed, especially during children's early life, parents make decisions that can affect crucially children weight status. These decisions are likely to be influenced by parental education and ability to acquire and process health information. The first fundamental choice

concerns breast-feeding: exclusive breast-feeding is recommended in all new-born infants for its nutritional and other beneficial effects, among which protection against obesity in later life with the duration of breast-feeding inversely associated with the risk of being overweight later on (Quak, Furnes, Lavine and Baur, 2008).

Other important decisions concern food choices and investments in sport and exercising activities. Well educated parents are usually more informed about nutrition facts and appropriate food portions and therefore are more likely to make their children have an healthy diet. Moreover, they are aware of the benefits of exercising and are therefore more willing to invest in children's sport activities and keep their sedentary behaviours at a minimum.

There are also several *indirect* pathways through which parental education may facilitate the intergenerational *transmission* of weight problems. In the following we examine in details these channels.

a. Family income

Higher education usually give access to better paid jobs and the higher income can be used to invest in health. In the presence of assortative mating, individuals with a higher level of education also marry partners with higher schooling, which positively affect family income. Furthermore, attending school for a longer time could lead to a change in preferences by either lowering the discount rate or increasing risk-aversion (Cutler and Lleras-Muney, 2010).

The availability of financial resources has an important role in determining children weight conditions. Households with greater resources can afford more costly and healthy food, can spend more on costly treatments or to purchase better health insurance and can invest more in exercising activities. This has a double effect on children's BMI: first, by allowing children to have an healthier lifestyle and, secondly, via the imitation of parental behaviors and the possibility to be included in a more favorable social network.

b. Imitation of parental health behaviors and lifestyle

It is likely that children tend to imitate parental behaviors. This, of course, holds also for food choices. Parents with disordered eating habits may set a bad example to their children. This problem is worsened by the fact that calorie-rich drinks and foods are easily accessible to children. Calorie-rich, prepared snacks are available in many locations frequented by children (for example snack vending machines in schools). Moreover, eating at fast food restaurants and consuming sugar-laden soft drinks is very common among young. Children living in families with unhealthy eating habits are more prone to consume junk food both because their parents are likely to do the same and because they received a nutritional education that tolerates these behaviours.

The same reasoning holds for physical exercise. Physical inactivity of children has shown to be a serious cause of overweightness. Children who fail to engage in regular physical activity are at greater risk of obesity since staying physically inactive leaves unused energy in the body, most of which is stored as fat.

Many children fail to exercise because they spend time doing stationary activities such as computer usage, playing video games or watching television. Two studies, one by Horton et al. (1995) and the other by Epstein et al. (2008) discovered that children were 21.5% more likely to be overweight when watching more than four hours of TV per day and 4.5% more likely when using a computer one or more hours per day.

As for eating habits, imitation of parental behaviour can play a key role in influencing children attitudes: sedentary parents might transmits a wrong message regarding the importance of having regular exercising in leading an healthy life.

c. Social network and Peer Effect

Children of more educated parents usually live in a better social environment and hang around with peers that are more likely to value health and behave healthily. This can influence their own behaviors, strengthening the habits and the teachings acquired from their parents. Also the school environment provides an opportunity to ensure that children understand the importance of good nutrition and physical activity, and can benefit from both.

There are also other factors that may have an impact on children weight status. For example, the time spent by parents with their children has been shown to be important. In 2011, researchers from American University, Cornell University and the University of Chicago released a study that found the more mothers work during their children's lifetimes, the more likely their kids are to be overweight or obese. Another study by Videon and Manning (2003) discovered that compared to adolescents who ate three or fewer family meals per week, those who ate four to five meals per week were 19% less likely to report poor consumption of vegetables, 22% less likely to report poor consumption of fruits and 19% less likely to report poor consumption of dairy foods. Adolescents who ate six to seven family meals per week, compared to those who ate three or fewer family meals per week, were 38% less likely to report poor consumption of vegetables, 31% less likely to report poor consumption of fruits, and 27% less likely to report poor consumption of dairy foods.

Finally, also psychological factors may contribute to generate weight problems. Strauss (2000) discovered a positive correlation between obesity and low self-esteem, while Goodman and Whitaker (2002) showed that feelings of depression can cause a child to overeat. Also stress can influence a child's eating habits, as documented by Ogg, Millar, Pusztai and Thom (1997).

In the second chapter of the thesis we will use data from the British Cohort Study (BCS70) to investigate the intergenerational transmission of BMI and weight problems from parents to children. We will compute intergenerational elasticity and, thanks to the large amount of information provided by our dataset, we will be able to distinguish between the direct and indirect channels driving the process by which weight is transmitted from one generation to the following.

1.4. Economic consequences of obesity

1.4.1 Health expenditure

Since it is associated with higher risks of chronic illnesses, obesity is linked to significant additional healthcare costs. Withrow and Alter (2011) show that obese individuals have medical costs that are approximately 30% greater than their normal weight peers.

According to the WHO estimates, obesity was responsible for 2-8% of total health expenditure in Europe in 2008 and, because of the time lag between the onset of obesity and related health problems, the rise in obesity over the past two decades will increase health care costs even more in the future. The Foresight Annual Report (2007), commissioned by the U.K. Government (Department for Bussiness, Innovation and Skills), foresees that in 2015 the total costs due to diseases linked to weight excess in England could rise by as much as 70% relative to 2007 and could be 2.4 times higher in 2025.

According to Cawley and Meyerhoefer (2012) the medical costs attributable to obesity in the US were about $190 billion in 2005, corresponding to more than 20% of the total medical expenditure.

In addition to the direct burden on the healthcare systems and therefore on States budgets, obesity has important additional economic costs due to its negative impact on labour market outcomes. Obese workers may indeed suffer disadvantages in employment and salaries, both because of their lower productivity and employers' discrimination.

1.4.2 Consequences on the labour market outcomes

Wages are the outcome mostly explored by the economists looking at the potentially negative effects of obesity on the labour market.

One of the first studies assessing the existence of a negative correlation between the weight and wages was Register and Williams (1990). They analysed a sample of 18 to 25 years olds from the 1982 wave of the National Longitudinal Study of Youth (NLSY), reporting mean hourly wage differences of minus 16% for obese women and (positive) 7% for obese men, compared to their not obese peers. However, when they control for age, race, region, education, health status, union status and employment characteristics, like work experience and industrial category, the pay differential falls to minus 12% for obese women and to minus 5% for obese men.

The fact that weight excess has a detrimental influence mainly on women's wage is a common finding in the literature. Averett and Korenman (1996) using a sample of 23 to 31 years olds drawn from the 1988 NLSY found a substantial wage penalty (around 20%) for white women who were obese both in 1988 and seven years before. However, white women who entered the obese category just between 1981 and 1988 had wages that were only about 6% below of normal-weight women. On the contrary, there is no evidence of a significant
correlation between the salaries of male workers and their probability of being obese.

Similar results are found in U.K. by Sargent and Blanchflower (1994). Using a sample of young adults, they provide evidence of a statistically significant inverse relationship between obesity and the earnings of 23 years old females, even when controlling for parental social class and the test score of ability tests sit in childhood (at ages 7, 11, and 16). Female adolescents who were in the top 10% of the BMI distribution at age 16 years earned 7.4% less than their non-obese peers; those in the top 1% earned 11.4% less. The inverse relationship between obesity at 16 years of age and earnings persisted whether the adolescent female remained obese (-6.4%) or moved into the non-obese category by age 23 years (-7.5%). As in Averett and Korenman (1996), no significant relationship between wages and BMI is instead found for men.

There are several reasons that can justify the negative relationship between BMI and wages. While most explanations are conditional on having a job, Han, Norton and Powel (2009) provide empirical evidence on the BMI wage penalty stemming from two indirect pathways (education and occupation choices) compared to the direct effect that conditions on education and occupation. The direct BMI wage penalty operates primarily through discrimination by employers who prefer to shun obese employees who may have lower productivity, higher health care costs and, as in sales industries, customers may dislike. On the other hand, the indirect channels operate through education and job preferences: the BMI wage gap may stem in part from choices made before being hired. Obesity may indeed harm the human capital accumulation process, resulting in lower educational achievements and therefore lower wages. Moreover, obese individuals might choose their job according to their weight condition, sorting into occupations in which their body weight does not lower their marginal productivity, avoiding jobs like sales or requiring social interactions with customers and colleagues.

Exploiting data from the 1979 NLSY, that provides information on both late teen and contemporaneous BMI of individuals, the authors can distinguish between the portion of the wage gap caused by employers' distaste for contemporaneously obese workers or by lower productivity and the fraction related to characteristics associated with BMI in late teenage years. Their results show that a significant portion of the effect of BMI on labour market outcomes occurs prior to

employment. Therefore, previous studies that estimate the BMI wage penalty *conditional* on education and occupation underestimate the gap: the authors suggest that the total BMI wage penalty for women is underestimated by 19% without including indirect effects. For men there is no statistically significant direct BMI penalty, but only a small indirect association driven by education.

However, these results should be taken with caution given that their model may not fully account for the potential endogeneity of BMI. Omitted variables related to both BMI and wages and unobserved individual-level heterogeneity (such as time preference) will still cause endogeneity bias. For example, if obese teenagers have a higher discount rate, estimated coefficients in the wage regression would be biased downwards.

The endogeneity issue is addressed by Cawley (2004) using instrumental variables. Specifically, he employed a sibling's BMI as an instrument for individual BMI. His OLS results show that heavier white, black and Hispanic females and Hispanic males tend to earn less than their lighter counterparts. However, IV estimates indicate that the hypothesis that weight does not lower wages can be rejected only for white females. In this subsample, IV coefficients reveal that an increase of two standard deviations from the mean weight in pounds leads to a decrease in wages of 18%.

The same approach is followed by Brunello and D'Hombres (2007), using as an instrument for a worker's BMI the BMI of a biological family member[2]. They use data from the European Community Household Panel and, contrary to the previous studies, they found that obesity hurts wages *independently* of gender. However, given the heterogeneity of the European labour markets, they decide to split their sample between the countries of the "olive belt" (Greece, Italy, Portugal and Spain) and those of the "beer belt" (Austria, Denmark, Belgium, Finland and Ireland), finding out that the effect of BMI on wages is negative only in the countries belonging to the "olive belt", while it is positive in the Northern and Central Europe. They suggest that this difference can be partly explained by the interaction between the weather, BMI and individual unobserved productivity.

A different choice concerning the instrumental variables employed to face the endogeneity of individuals' weight is done by Morris (2006) who uses two area-level indicators as instruments to control for environmental factors affecting individual obesity in the form of endogenous and exogenous peer group effects. These indicators are the mean BMI and the mean proportion of adults classified as obese in the respondent's health authority. Area-based measures have been previously used as instruments for individual level variables but not for BMI. Using pooled data from the 1997

[2] For individuals with several available relatives, they average out all available BMIs.

and 1998 rounds of the Health Survey for England (HSE), Morris analyses the impact of BMI on earnings in U.K. His OLS estimates show that BMI has a positive and significant effect on mean hourly earnings in males and a negative and significant effect in females. Specifically, the elasticity for men is 0.08, meaning that a 10% increase in BMI leads on average to a 0.8% increase in mean occupation wage. However, the direct effect of being obese on occupational attainment is not statistically significant. In females, the direct effect of BMI on occupational attainment is negative and significant: a 10% increase in BMI leads on average to a 0.4% decrease in mean occupation earning, while being obese has a weakly significant and negative direct effect. When controlling for endogeneity, IV coefficients are insignificant in all models. However, using a Hausmann test, Morris fails to reject the hypothesis that the OLS and IV coefficients are equal. This means that, if the instruments are valid, the endogeneity of BMI and occupational attainment does not significantly affect the OLS estimates, which should then be preferred because they result in lower standard errors.

Wages are not the only economic outcome that can be damaged by weight excess. Negative effects of obesity have been found also on the employment probability, even if, at the moment, the evidence is still unclear. Cawley and Danziger (2005) use U.S. data from the Women's Employment Study to investigate whether obesity is a barrier both to employment and earnings for welfare recipients. They provide evidence that high body weight is a great barrier to labour market success for white women: a 10% increase in weight from the mean is associated with a 12% decrease in the probability of current employment, an 8.9% decrease in the probability of fulltime employment, 5.4% fewer hours worked, 16% more months spent on welfare between survey waves and 10% lower earnings. On the contrary, among African American respondents, weight is only correlated with welfare receipt: a 10% increase in weight in pounds is associated with a 10.9% increase in the percentage of months spent on welfare. A limitation of this paper is that the authors, having no instruments to deal with the endogeneity issues, can only speculate on the causal relationship embedded in the correlations observed. However, the difference highlighted between the two ethnic groups is in line with the results of Cawley (2004), who found a negative causal effect of BMI only on the salaries of white females.

The causality of the relationship between obesity and employment probability is investigated in Morris (2007) exploiting three different estimation approaches: a univariate probit model, propensity score matching and IV regression using a recursive bivariate probit model. As in his previous work (Morris, 2006) he analyses data from the Health Survey for England (HSE) and uses as instrument for the individuals' weight condition the mean proportion of adults classified as obese in their health authority. Conditional on a large set of covariates, his results show that obesity has a

statistically significant and negative effect on employment for both males and females. While for males the results are similar across the three methods, failure to account for endogeneity leads to underestimation of the negative impact of obesity on female employment.

Opposite findings are obtained by Norton and Han (2008) using genetic information to identify the effect of obesity on wages and employment probability. They analysed data from the third wave of the Add Health dataset (when respondents are in their mid-twenties) and exploit the exogenous variation in the phenotype provided by specific genes linked to obesity as an instrument for individuals' BMI finding no evidence of a negative effect of weight on labour market outcomes.

Table 3 – Studies investigating the influence of obesity on labour market outcomes

Paper	Data	Outcome	IV	Main Findings
Register and Williams (1990)	U.S.	Wages	-	Negative correlation between obesity and wages, especially for females
Averett and Korenman (1996)	U.S.	Wages	-	Evidence of a wage penalty for white women but not for men.
Sargent and Blanchflower (1994)	U.K.	Wages	-	Negative correlation between obesity and wages for females but not for males
Han, Norton and Powel (2009)	U.S.	Wages	-	Negative correlation between late teen BMI and female future wages: a significant portion of the effect of BMI on labor market outcomes occurs prior to employment (role for indirect effects of BMI like education and occupation choice)
Cawley (2004)	U.S.	Wages	BMI of a sibling	Negative effect of obesity on wages only for white females
Brunello and D'Hombres (2007)	E.U.	Wages	BMI of a biological family member	Obesity affects wages independently from gender: the effect is negative in the Southern Europe and positive in Central and Northern Europe.
Morris (2006)	U.K.	Wages	Area-levels indicators	Positive correlation of bw obesity and wages for males, negative for females. IV estimates are not statistically significant. However, an Hausman test fails to reject the hypothesis that the OLS and IV coefficients are equal.
Cawley and Danziger (2005)	U.S.A.	Employment probability, wages and hours worked	-	Significant negative correlation between weight and labour market outcomes for white women
Morris (2007)	U.K.	Employment probability	Area-levels indicators	Obesity has a statistically significant and negative effect on employment for both males and females
Norton and Han (2008)	U.S.A.	Wages and employment probabilità	Genetic markers	No effect of obesity on labour market outcomes

1.4.3 Effects on individuals' cognitive skills

As we have seen in the previous paragraph, several studies have highlighted the negative effect of weight excess on labour market outcomes. However, not enough attention has been put on the potential channels driving this relationship. One of the possible interpretations provided to explain the wage and employment discrimination against overweight individuals is that obese workers have a lower productivity, because of health and psychological problems, higher absenteeism but also lower human capital. Cognitive skills and educational achievements are important determinants of individual's productivity: a decline in cognitive ability caused by obesity might explain part of the wage gap found in the literature between obese and non-obese workers.

We will investigate empirically the link between obesity and cognitive skills in the third chapter of the thesis, providing evidence of a negative causal effect stemming from weight excess.

For the moment we present and analyse the existing economic literature that, with few exceptions, highlights the presence of a negative influence of obesity on cognitive and educational achievements.

It is important to distinguish between the studies assessing the existence of a simple *association* and those looking for the *causal effect* of body weight. While the first category counts several contributions, just few studies have focused on the causality of the relation between obesity and cognitive skills.

Moreover, most of the works uses academic achievements (like maths and reading test scores, grade point averages in specific subjects, highest grade attended, etc.) as indicators of cognitive skills, while only few can rely on the result of standardized cognitive tests.

The relationship between obesity and cognitive skills has been investigated at different ages, from early childhood (Cawley and Spiess, 2008) to college-age students (Kaestner et al., 2009; Fuxa and Fulkerson, 2011; Karnehed et al., 2006), passing through elementary school children (Datar, Sturm and Magnabosco, 2004 and Datar and Sturm, 2006) and adolescents (Mo-Suwan et al., 1999; Sigfusdottir, Kristjansson and Allegrante, 2007).

The correlation assessed by the large majority of these studies is negative and significant.

Cawley and Spiess (2008) investigate the association between obesity and skill attainment (measured by verbal skills, activities of daily living, motor skills and social skills) in children aged 2-4 years, using data from the German Socio-Economic Panel Study. Even controlling for a rich set of child, parent, and family characteristics, they find that, among boys, obesity is associated with reduced verbal, social and motor skills, and activities of daily living, while

among girls, it is associated with reduced verbal skills only. Further analysis show that such correlations exist even for those preschool children who spend no time in day care, suggesting that it cannot be due solely to discrimination by teachers, classmates or day care providers.

Weight excess is found to be negatively correlated with individuals' human capital also at older ages: Sigfusdottir, Kristjansson and Allegrante (2007) explored the relationship between health behaviours and academic outcomes in Icelandic 14-15 years old students, showing that BMI, diet and physical activity explained up to 24% of the variance in academic achievement when controlling for gender, parental education, family structure and absenteeism.

Using a sample of Minnesota students, Fuxa and Fulkerson (2011) found that overweight and obese adolescents were significantly less likely to plan to go to college, more likely to report lower academic grades and skipping school than non-overweight peers. Similarly, Karnehed et al. (2006), analyzing a cohort of more than 700,000 Swedish men born from 1952 to 1973, provided evidence that 18 years old obese students were 50% less likely to get into higher education than their normal-weight counterparts. This result was confirmed even after adjustments for intelligence and parental socioeconomic status, suggesting that discrimination in the educational system may explain the strong association between obesity and educational achievements.

However, in some cases the evidence is not definitely clear and results are not always robust. In Datar, Sturm and Magnabosco (2004) the differences in school performance between obese and non-obese U.S. students, except for boys' math scores, become insignificant after controlling for socioeconomic and behavioral variables, suggesting that overweight is a marker but not a causal factor. Similarly, Li, Dai, Jackson and Zhang (2008) found that the association between BMI and academic achievements they detected in a sample of U.S. children aged 8–16 years was not significant after adjusting for parental and family characteristics. However, the negative correlation with cognitive functioning, measured by using standardized neuropsychological tests, remained significant after adjusting for family background, physical activity, hours spent watching TV and some health and psychosocial indicators.

Moreover, Mo-Suwan, Lebel, Puetpaiboon and Junjana (1999), using a sample of Thai students, showed that, while being or becoming overweight during adolescence (grades 7-9) was associated with poor school attainment, such an association did not exist using a two-years lagged BMI value, raising some concerns about the presence of contemporaneous effects of school performance on weight, caused for example by psychological reasons.

The only studies that don't find any significant evidence of a negative correlation between obesity and educational achievements are Kaestner and Grossman (2009) and Kaestner, Grossman and Yarnoff (2009). The former study analyses a sample of U.S. children's between the ages of 5 and 12 and uses the Peabody Individual Achievement Tests in math and reading as an outcome, while the latter focuses on U.S. adolescents (between the ages of 14 and 18) and uses highest grade attended, highest grade completed and drop out status as indicators of school performance. In both the cases the results suggest that on average overweight or obese students have levels of attainment that are about the same as normal-weight peers.

As already stressed at the beginning of the paragraph, just few researchers have focused on the *causality* of the relationship between obesity and cognitive skills. We have found only five studies in the literature facing the endogeneity problem that is likely to affect individuals' weight. All of them follow an instrumental variable approach, but two different kinds of instrument are employed: either a relative's BMI and weight status (Sabia, 2007; Averett and Stiefel, 2007) or genetic markers (Fletcher and Leherer, 2008; Ding et al., 2009 and Von Hinke Kessler Scholder et al., 2010).

Sabia (2007) studied the relationship between body weight of U.S. adolescents aged 14-17 and their academic achievement to understand whether early human capital accumulation is adversely influenced by obesity. Using as exclusion restrictions parental self-reports of obesity, he found consistent evidence of a significant negative effect of BMI on grade point average in math and English language for white females, while for non-white females and males the evidence of a causal link between body weight and academic performance is less convincing. Averett and Stiefel (2007) focused on two types of childhood malnutrition: not only over- but also under-weightiness. They analysed a sample of 5-years old children from the NLSY79 using maternal BMI as an IV for children's BMI and concluding that malnourished children tend to have lower cognitive abilities when compared to well-nourished children.

Concerning the studies using genetic markers to recover identification, Fletcher and Leherer (2008) were the first employing these instruments. They exploit differences in genetic inheritance among children within the same family to estimate the impact of several poor health conditions, one of which is obesity, on children's academic outcomes. Using the Add Health data they find no evidence that being overweight (or obese) significantly affects school performance when controlling for poor mental health. Different findings are obtained by Ding et al. (2009), who take a similar approach to Fletcher and Leherer (2008) but use a different dataset, the Georgetown Adolescent Tobacco Research (GATOR) study. Exploiting natural

variation within a set of genetic markers across individuals, they investigate the effects of a rich set of health outcomes on adolescents' academic achievements. Their IV results show large and significant negative effect of obesity on female's grade point average (GPA), but not for males. GPAs for obese girls are on average 0.8 points lower than those for non-obese peers.

However, Von Hinke Kessler Scholder et al. (2010) cast some doubts about the validity of the results obtained by these two studies. As the authors pointed out, there is a week and inconsistent evidence in the medical literature that the genetic variants employed by Fletcher and Leherer (2008) and Ding et al. (2009) are robustly associated with fatness in large population samples. This is a serious problem since weak association may result in biased estimates. Moreover, even if a suitable and robust genetic instrument is available, it may explain little of the variation in observed phenotype: if the alleles shift the adiposity distribution by a very small amount, the effect of fatness on test scores is identified only by this small difference in mean adiposity. The variants used by Von Hinke Kessler Scholder et al. (2010) are currently the best candidates to be used as genetic markers, since they have been shown to be associated with adiposity in large population samples. However, the authors admit that, while their instruments are not weak in a statistical sense, *"their effects may be too small to impact on the possible pathways to academic performance*", concluding that genetic instruments should be used with care.

Other important contributions of this study are the use of fat mass (determined by a dual-energy X-ray absorptiometry scan) as a measure of adiposity in place of the Body Mass Index, and the use of a U.K. dataset, the Avon Longitudinal Study of Parents and Children (ALSPAC). While their OLS results suggest that leaner children perform slightly better in school tests compared to their counterparts with more body fat, the IV estimates show no evidence that fat mass affects academic outcomes.

In the third chapter of the thesis we will contribute to this literature shedding new light on the causality of the link existing between obesity and human capital. Unlike the majority of existing researches, that focus on the consequences on educational achievements, we use as outcome the score reported in two standardized cognitive tests sit when individuals are 10 and 34 years old respectively.

We are the first ones to use data from the British Cohort Study (BCS70) in this kind of analysis. This dataset contains information on both parents' BMI, allowing us to use two instrumental variables: this guarantees an efficiency gain in the estimation and let us performs an over-identification test to check our instruments' exogeneity. Such a test was not feasible

in the previous studies using a relative's BMI as an IV for the individual's BMI, since they all use only one instrument and therefore have just-identified models.

Moreover, the BCS70 allows us to control for many important variables (such as birthweight, whether the individual was breast-fed when infant, parental education and school sentiment, etc.) that were often missed in the studies we presented above.

Finally, thanks to the longitudinal structure of our dataset and the availability of two tests sit in different periods of life (childhood and adulthood), we can follow individuals over time, evaluating also the lagged effect of obesity.

Table 4 – Studies investigating the *correlation* between obesity and educational and cognitive achievements

Paper	Data	Outcome	Main Finding
Cawley and Spiess (2008)	Germany	Skill attainment in 2-4 years children	Obesity is associated with reduced verbal, social and motor skills for males and with reduced verbal skills for females
Sigfusdottir et al. (2007)	Iceland	Academic achievements	BMI, diet and physical activity explain a significant part of the variance in adolescents' academic achievements
Fuxa and Fulkerson (2011)	USA	College enrolment	Overweight adolescents are significantly less likely to plan to go to college
Karnehed et al. (2006)	Sweden	Enrolment in higher education	18 years old male student are 50% less likely to get into higher education than normal weight peers.
Datar and Sturm (2006)	USA	Maths and reading test scores, school absenteeism and grade repetition	Obesity is a significant risk factor for adverse school outcomes among elementary school girls
Datar, Sturm and Magnabosco (2004)	USA	Maths and reading test scores	Significant association between obesity and lower math and reading test scores in kindergarten and first grade children. However, controlling for socio-economic and behavioral variables, only the difference in boys' math score remain significant
Li, Dai, Jackson and Zhang (2008)	USA	Academic performance and cognitive functioning	Negative correlation between obesity and school-aged children and adolescents' cognitive functioning but no association with their academic performance
Mo-Suwan et al. (1999)	Thailand	School attainment	Negative correlation between contemporaneous BMI and school attainment; the correlation is not significant using 2-years lagged BMI values.
Kaestner and Grossman (2009)	USA	Maths and reading test scores	Obese children achievements are about the same of normal-weight peers
Kaestner, Grossman and Yarnoff (2009)	USA	Highest grade attended, highest grade completed and drop out rates	No significant effect of adolescent obesity on future academic outcomes

Table 5 – Studies investigating the *causal* effect of obesity on educational and cognitive achievements

Paper	Data	Outcome	IV	Main Finding
Sabia (2007)	USA	Self-reported GPA in maths and English language	Parents' perceived obesity	Negative effect of obesity for white female adolescents
Averett and Stiefel (2007)	USA	Cognitive ability test score	Mother's BMI	Negative effect of obesity and underweight
Fletcher and Leherer (2008)	USA	GPA + verbal test	Genetic markers	No significant effect of obesity
Ding, Weili, Lehrer and Rosenquist (2009)	USA	(Self-reported) GPA	Genetic markers	Large and significant negative effect of obesity on the GPAs of female adolescents but no effect on males
Von Hinke Kessler Scholder et al. (2010)	UK	KS3 score (English, math, science)	Genetic markers	No evidence that fat mass affects academic outcomes

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Chapter 2

Intergenerational Transmission of BMI and Weight Problems

2.1 Introduction

The share of obese and overweight people rose sharply in the last decades and this trend has even got faster in recent years. Obesity has become a real health and social plague with important economic consequences on State budgets through the increase in health expenditure. However, the flooding of obesity may have economic consequences that go well beyond the rise of medical costs. The persistence of weight problems across generations may also affect the economic mobility of the family. Given the importance of the "health capital" for education and earnings, it is possible that poor health during childhood and adolescence has an important influence on the intergenerational transmission of the economic status.

For this reasons, in recent years, also economists and social scientists have investigated the determiners of the obesity epidemic, focusing in particular on the role of risk factors like the individual's socio-economic status and educational level.

However, most of the studies have faced the issue only at the individual level, without analyzing the transmission of weight problems from parents to children. Just few works have taken an intergenerational perspective.

The aim of the present chapter is to shed new light on the intergenerational transmission of BMI and obesity and to investigate its origins. Specifically, we will focus on the role of parental education, which is a crucial determiner of the family environment and socio-economic status.

Medical research, like Comuzzie and Allison (1998), showed that genetics is crucial in determining individuals' weight condition: 40 to 70 percent of the variation in obesity-related phenotypes in humans is heritable. However, genetics alone cannot explain the dramatic increase in obesity rates observed almost all over the world. As recognized also by Comuzzie and Allison, "*the most common forms of human obesity arise from the interactions of multiple genes, environmental factors, and behavior*".

The intergenerational transmission of weight excess may stem from a variety of sources: it could reflect nature (genetics) or nurture (common environment), or the interaction between the two. Some studies, like Mansky (2011) and Heckman (2008) have indeed criticized the simple dichotomy between nature and nurture, embracing the epigenetics[3] approach. This theory asserts that the genes vs. environment distinction, that leads to the nature vs. nurture partition common to many economics papers, is obsolete: a gene-environment interplay takes place, in which environmental influences can alter the effects of genes.

[3] Epigenetics is the study of changes in gene expression or cellular phenotype, caused by mechanisms other than changes in the underlying DNA sequence

Empirical research has confirmed that the genes-environment interactions are central to explaining human development and that the genetic expression, or phenotype, is strongly influenced by environmental factors (Rutter, 2006; Rutter, Moffitt and Caspi, 2006).

Therefore, it is of interest to examine the interaction between parents' BMI and educational level: in the analysis that follows, we will emphasize this important feature of the intergenerational transmission process.

We contribute to the existing literature on intergenerational transmission in several directions. To our knowledge we are the first ones to: 1) compute IGE of BMI between *both parents* and their children, focusing not only on the transmission of the (continuous variable) BMI but also of weight problems (being overweight); 2) investigate the role of the interaction between parents' BMI (or weight status) and their educational qualification; 3) study the evolution of the IGE and of the transmission of weight excess over individuals' life; 4) use English data in this kind of analysis.

The choice of using a U.K. dataset (the British Cohort Study, BCS70) is of great relevance since United Kingdom is one of the countries with the highest prevalence of obesity among both children and adults.

Moreover, this dataset contains a huge set of information about individual and family background characteristics, providing us with a great advantage with respect to previous research. Thanks to its longitudinal structure, we can also follow cohort members over time. However, since we have information on parents' BMI only in 1980, we cannot set up a dynamic model.

Our results reveal the existence of a positive and highly statistically significant correlation between parents' and children's BMI, as well as between their probability to be overweight or obese. This positive association is extremely stable across different model specifications with the gradual inclusion of our controls, suggesting a strong influence of genetics in the intergenerational transmission of weight problems.

On the contrary, parents' schooling seems to play a minor role, having a small and often not significant impact on children BMI. Only the negative effect of maternal education on sons' BMI turns out to be significant from a statistical point of view.

However, when analyzing the influence of parental education we have to be careful mainly because of two reasons. First of all, parents schooling may exerts its influence through a better parenting, which is the parents' ability in raising children. Education may improve this "ability", providing parents with additional knowledge about what is good for children health and a better capability in acquiring and processing health information, thus preventing childhood obesity in a more effective way. However, parents' education also affects several variables that can in turn have an impact on children's BMI, like the family income, socio-economic status and social network, the parents'

health behaviors and the children own schooling. Therefore, finding no effect of parental education after controlling for parents' behaviors, parents' BMI, parents' social network and children's education, would only mean that the effect of better parenting is null, not that parents' education is not important.

Secondly, estimates of the parental schooling effect are likely to be biased by endogeneity issues, specifically by the presence of parents' unobservable characteristics, like ability, preferences and health endowments that can simultaneously affect both their educational level and their children weight status.

The outline of the chapter is the following. Section 2 contains a literature review. The data used are described in section 3, while section 4 presents the empirical models and the estimation strategies employed. The results of our estimates are shown in section 5. Conclusions follow in section 6, together with a discussion about the policy implications of our findings.

2.2 Literature Review

Our research deals with two different strands of literature: one studying the intergenerational transmission of health outcomes and the other analysing the link between parental education and children health.

2.2.1 Intergenerational Transmission

Economists and social scientists have long been interested in the study of the intergenerational transmission of earnings and education and in the computation of the corresponding intergenerational elasticities (IGE), which are essential measures to evaluate intergenerational mobility.

Only in recent years the attention has focused also on other family background characteristics, like IQ/ability, jobs and occupation, attitudes, social behaviours and health.

The literature on earnings transmission is potentially boundless and it is extensively reviewed in Solon (1999) and Black and Devereux (2011). For this reason we limit ourselves to mention only the most recent findings that agree on the evidence that Nordic countries have a higher level of mobility than U.S. and U.K.

Estimates of the IGEs and correlations for sons suggest that the lowest earnings persistence is in the Nordic countries (IGE lower than 0.3), the highest in U.S. (IGE of about 0.5-0.6) and the halfway level in U.K. (IGE of about 0.3) (see for example Jantti et al., 2006; Mazumder, 2005; Nicoletti and Ermish, 2007). Concerning other European countries, Italy has been estimates to have an IGE of about 0.5 (Piraino, 2007), France of 0.4 (LeFranc and Trannoy, 2005), while much lower values have been found for Germany (Vogel, 2008).

Father-daughter elasticities and correlations are smaller than the equivalent father-sons ones, but the pattern across countries is similar, with smaller IGEs in the Nordic countries and lager values in U.K and U.S. (Jantti et al., 2006).

Also the intergenerational transmission of education has been deeply investigated. Hertz et al. (2007) provide an extensive survey of correlation and regression coefficients for a sample of 42 countries in the period 1994-2004, finding that the correlation is about 0.6 in South America, 0.30 in U.K, between 0.3 and 0.4 in the Nordic countries, more than 0.5 in Italy and 0.46 in the U.S.

Their findings are confirmed by Chevalier et al. (2009), who focused only on the U.S. and European countries and by Behrman et al. (2001), who carried out a study on South America countries.

A growing literature has been moving beyond earnings and education to analyze the intergenerational relationship between other child and parents outcomes.

Substantial intergenerational transmission of IQ scores has been found in Norway (Black et al., 2009), Sweden (Bjorklund et al., 2010) and Germany (Anger and Heineck, 2009). De Coulon, Meschi and Vignoles (2008), using a U.K. cohort, showed that parents' basic skills in literacy and numeracy have a positive significant effect on their children's cognitive test scores.

Intergenerational mobility has been analyzed also for jobs and occupation, finding strong correlation between father and sons work activity (see Ermish and Francesconi, 2002; Carmichael, 2000; Di Pietro and Urwin, 2003).

Only few studies have investigated the existence of a positive intergenerational correlation in health outcomes. Currie and Moretti (2007) focused their attention on the intergenerational correlation in birth weight, finding that low birth weight mothers were significantly more likely to have low birth weight children. Moreover, the intergenerational transmission of low birth weight was stronger for mothers living in poor areas.

Using the German Socio-Economic Panel, Coneus and Spieß (2008) find intergenerational transmission of anthropometric (weight, height and BMI) and self-reported measures of health, but their analysis is carried out only on children younger than four years old. Similarly, Akbulut and Kugler (2007) examine the mother-child correlation in height, weight, BMI, depression and asthma in U.S., using data from the NLSY79 and revealing substantial inheritance of anthropometric and emotional health. Two other studies exploit the same dataset to carry out similar research. The first one is Classen and Hokayem (2005) that estimate the influence of child and maternal characteristics on the likelihood that a will become obese or overweight during youth, showing that among the explanatory variables that significantly influence the probability of youth obesity there are mothers' obesity and education.

The second one is Classen (2010), who provides estimates of the intergenerational persistence of BMI between mothers and their children when both are at similar stages of their lives (between the ages of 16 and 24). He finds a statistically significant intergenerational correlation of BMI, equal to 0.35 in the entire sample, 0.38 between mothers and daughters and 0.32 between mothers and sons. Strong dependence across generation is found also when considering the overweight and obesity outcomes.

Unfortunately, the lack of information about fathers' BMI in the NLSY79 prevents all the above mentioned studies from computing correlations and IGE between fathers and their children and to study whether and how the mother-children relationship varies when we include also the fathers in the analysis.

Finally, the transmission of smoking habits has been investigated by Loureiro et al. (2006), showing that 18 years old guys having both parents who smoke are about twice as likely to do the same than their peers whose parents don't smoke.

2.2.2 The impact of parental education on children health outcomes

Given the contrasting evidence provided by the different studies, it is difficult to draw any definitive conclusion about the effect of parental schooling on children health.

Currie and Moretti (2003) provided the first evidence that maternal education causally affects maternal pre-natal behavior and birth outcomes, exploiting the openings of colleges by county in the U.S. from 1940-96 as instruments for maternal education. Their IV estimates suggested that an extra year of college reduced the likelihood of low birth weight children, pre-term birth and smoking during pregnancy.

Similarly, Breierova and Duflo (2004) exploited a massive school construction program that took place in Indonesia between 1973 and 1978 to estimate the effect of education on fertility and child mortality. The different timing of school construction in the different regions of the country generated instrumental variables for the average education in the household and the difference in education between husband and wife. Using this identification strategy the authors showed that females' education played a stronger role than males' education in determining age at marriage and early fertility. However, female and male schooling seemed equally important factors in reducing child mortality.

On the contrary, McCrary and Royer (2006), exploiting school entry age laws to obtain an exogenous variation in women schooling attainment, found negligible effects of mothers' schooling on children's low birth weight, pre-maturity and infant mortality.

The intergenerational transmission of human capital was explored also by Lundborg, Nordin and Rooth (2011). Their study didn't rely on IV methods but used both an adoption and a twin design to investigate the effect of parental education on children's cognitive skills, non-cognitive skills and health (measured by an index of the overall health-status, including physical and mental conditions). Estimation results showed that parental education increased children's cognitive and non-cognitive skills, as well as their general health.

Grytten, Skau and Sorensen (2012) estimated the causal effect of parental education on infant health employing four different identifying strategies on the same dataset, the Norwegian Birth Registry: instrumental variables, a regression discontinuity design, a difference-in-difference approach and finally they focus only on twin parents. Following each of the estimation strategies, the result was always the same: parents' educational level had no impact on their offspring's birth weight, Apgar scores and mortality.

A different way to address the endogeneity of parental schooling was employed by Chen and Li (2006). They examined the effect of maternal education on young children health using a large sample of adopted children from China. As adoptees are genetically unrelated to the nurturing parents, the influence of parental education on their outcomes will reflect the nurturing effect only. Their analysis showed that mother's education was an important determinant of adopted children health (measured by height-for-age z-score) even after controlling for income, number of siblings, health environments and other socioeconomic variables. Interestingly, they also found that the effect of the maternal education on the adoptee sample was similar to that on the own birth sample. This suggests that the main influence of the mother's schooling on child health may be due to post-natal nurturing.

Doyle, Harmon and Walker (2005) analyzed the effect of parental background on children health, focusing on parents' education and family income as explanatory variables and treating both of them as endogenous. They identified the effect of parental education using the exogenous raise in schooling caused by the 1957 English School Reform and they accounted for the potential endogeneity of parental income using as an instrument the predicted parental education values from the first stage schooling equation. Then, they estimated the impact of parents' education and income on a four point ordinal variable defining child health status (Very Good, Good, Fair, Very Bad/Bad) with an ordered probit model. Their results provided little evidence of education effects and, when accounting for income endogeneity, they concluded that also income effects on child health were the result of spurious correlation rather than of some causal mechanism.

Exploring the child health income gradient in England, but looking just at correlation, Currie A. et al. (2004) found consistent and robust evidence of a significant family income effect on children

subjective measures of health. On the contrary, they found no evidence of an income gradient for objective indicators of health, derived from nurse measurements and blood test results. Finally, they showed that nutrition and family lifestyle choices had an important role in determining child health and child health outcomes were highly correlated within the family.

To our knowledge, only Lindeboom et al. (2009) studied the effect of parental education on children BMI. Using data from the National Child Development Study (NCDS 1958) and exploiting the U.K.'s 1947 raise in compulsory schooling age (from 14 to 15) to infer causality, they estimated small and statistically insignificant effects of parental education on birth outcomes (low birth weight and illness at birth) as well as on child chronic conditions and obesity at ages 7-16.

2.3 Data

The British Cohort Study of 1970 (BCS70), providing information about the BMIs of a sample of U.K. individuals and their parents, is one of the few available dataset that allows us to investigate the intergenerational transmission of weight. Since the United Kingdom is one of the E.U. Member States where weight problems are more severe, in adulthood as well as in childhood, the choice of this dataset seems particularly suitable.

The BCS70 is a longitudinal study collecting data on the births and families of about 17,000 babies born in England, Scotland, Wales and Northern Ireland in a particular week in April 1970 and following their lives until nowadays.

Over the course of cohort members lives, the BCS70 has gathered a huge amount of information about their health, physical, educational and socio-economic status.

Since the birth survey in 1970, there have been seven "sweeps" of cohort members at ages 5, 10, 16, 26, 30, 34 and 38. The age 42 survey began in May 2012 but is not jet available.

We focus our attention mainly on three sweeps, those carried out in 1980, 1986 and 2004, following the cohort members at ages 10, 16 and 34, that is when they are children, then adolescents and finally adults.

Unfortunately we cannot exploit all of the waves of the BCS70, because they not always contain all the information necessary for our analysis. For example, we have no information about neither cohort members' weight nor height when they are 5 (1975) so that we cannot compute BMI values, while in the 1996 sweep the dataset contains too many missing values in such variables.

The strength of the BCS70 is the large amount of information it provides about cohort members' environment, socio-economic status, family background, educational attainment and lifestyle. This allows us to include in our analysis a huge set of control variables that can be classified into four macro categories: geographical factors, socio-economic status of the household, family background

characteristics, health behaviors and health indicators. The full list of the covariates included in our estimations is provided in Appendix 1.

Since we have information also about the B.A.S. (British Ability Scale), a cognitive test taken when cohort members are 10 years old, we initially decided to include the test score in our set of controls. However, since this variable was missing for a lot of individuals (about 1,500) and its coefficient turned out to be not significant, we then decided to neglect it in our analysis.

The only drawback of the BCS70 is the reduction of observations over time. This problem is due mainly to two reasons. First of all, starting from the second wave, Northern Ireland was no more included in the survey, reducing the total number of observation in the sample by 626 units.

Moreover, after the birth survey in 1970, when the forms were filled by delivering mothers directly at the hospitals of the National Health Service[4], cohort members and their families were periodically interview by means of home and school questionnaire. This requires tracing cohort members during their lives that is obviously not an easy task.

In the first wave (1975), children were traced through the cooperation of the registration division of the Registrar General's Office (RGO), the National Health Service Central Register (NHSCR) and the Family Practitioner Committee (FPCs).

For the second waves the names and address of the cohort members were obtained from the School Register. Later on, this information was recorded with the cooperation of Local Health Authorities (LEAs) and District Health Authorities (DHAs). Despite the effort of these institutions and authorities, the lack of observations is not negligible.

Another problem is that questionnaires are sometimes only partially filled, so that the dataset contains missing values. Since some information, such as cohort members' and their parents' height, weight and years of schooling are essential for our analysis, we are forced to drop the observations where these characteristics are missing.

In addition, following the health economics literature, we drop from our sample also twins and multiple births, since they are not representative of the population.

Table A in Appendix 2 summarizes the total number of observations for each wave, the effective number of observations we can exploit and the reason that justify the reduction.

After merging the different waves and dropping out missing values, we are left respectively with 8145 observations in 1980, 3487 in 1986 and 5260 in 2004. In the light of the sensible drop in the number of observations over time, we might be concerned about attrition problems. For this reason

[4] Only for domiciliary births forms were sent to mother homes by the Medical Officers of Health of the local health authorities

we decide to test whether the sample reduction is due to selection issues. With this aim, we follow the approach developed by Heckman, using as selection equation the probability that a CM appears in a given sweep. Results and details of the technique applied are presented in Appendix 3. Since the coefficient (*lambda*) of the Inverse Mills ratio turns out to be not significant, we conclude that the drop of observations is due to exogenous factors and therefore our panel seems not to suffer from attrition problems.

In the following table, summary statistics for the variables of main interest are presented. Since we perform separate analysis for males and female, summary statistics are reported for each of the two genders groups. As it can be noticed, in both genders' subsamples fathers' BMI is on average about one unit greater than mothers' BMI. The same holds for the years of schooling: fathers received on average a little bit more education than mothers but data are extremely closed in the two subgroups. On the contrary, female cohort members are on average slightly more educated.

Table 1 – Average BMI values and obesity rates

	MALE CM		**FEMALE CM**	
Variable	**Mean**	**Std. Deviation**	**Mean**	**Std. Deviation**
Mother's BMI 1980	23.37	3.71	23.39	3.74
Father's BMI 1980	24.46	3.06	24.45	2.95
Cohort Member's BMI in 1980 (10 years old)	16.74	1.93	17.06	2.26
Cohort Member's BMI in 1986 (16 years old)	20.71	3.08	21.33	3.39
Cohort Member's BMI in 2004 (34 years old)	26.58	4.32	25.09	5.10
Mother's years of schooling	9.75	1.72	9.69	1.70
Father's years of schooling	9.97	2.29	9.96	2.28
Cohort Member's years of schooling	13.48	3.56	13.71	3.58

	MALE CM	FEMALE CM
Variable	**Percentage**	**Percentage**
Mothers' overweight rate	24.24%	23.58%
Fathers' overweight rate	36.49%	36.89%
Mothers' obesity rate	5.36%	5.78%
Fathers' obesity rate	4.78%	4.29%
CM obesity-risk rate in 1980 (10 years old)	18.39%	14.43%
CM obesity-risk rate in 1986 (16 years old)	19.62%	12.10%
CM overweight rate in 2004 (34 years old)	61.32%	39.69%
CM obesity rate in 2004 (34 years old)	16.79%	14.81%

Concerning cohort members' BMI, we can see that in both sexes it increases over time due to the physiological growth of individuals. It is greater for females at age 10 and 16 because of the earlier growth of girls but bigger for males in adulthood, as it was for parents' BMI.

Looking at parental overweight rates, we notice that they are much higher for fathers than for mothers (more than 36.5% vs. about 24%) while the situation is reverted when we consider obesity rates, even if the difference is not so definite.

Before analyzing the statistics relative to the cohort members, it is worth notice that overweight and obesity thresholds are computed in a different way for adults and children.

While for adult individuals overweight and obesity status are derived by simply comparing individuals' BMI with the BMI thresholds defined by the International Obesity Taskforce (25 and 30 respectively), the classification is a little bit more complex for children and boys below 18 years. In this case, it is necessary to compare children's BMI with those corresponding to the 85^{th} and 95^{th} percentiles in the BMI sample distribution, as illustrated in the figure in Appendix 5.

Among the cohort members, the situation is much worse for males. At all ages, the percentage of males suffering from weight problems is considerably higher. This is in line with the Health Interview Survey data, released by Eurostat in 2008.

Focusing our attention on the overweight and obesity rates, we can realize that they rose sharply in the last decades: they have increased from about 36.5% and 4.5% in 1980 to more than 61% and 16% in 2004 for males and from about 24% and 5.5% to almost 40% and 15% for females.

These percentages are extremely high but, again, they are perfectly in line with the Eurostat data. Moreover, it is important to remember that U.K. is the E.U. country where weight problems are most severe.

From Table 2 we can also notice that children born from obese parents are more prone to suffer from weight excess.

Table 2 – Children weight condition given parents' weight status

	Males (1980, 10 yrs old)			**Femles (1980, 10 yrs old)**		
	Normal weight	**Obesity risk** (>85° perc.)	**Obesity** (>95° perc.)	**Normal weight**	**Obesity risk** (>85° perc.)	**Obesity** (>95° perc.)
In the sample	85%	15%	5%	85%	15%	5%
Both parents are normal-weight	86.1%	13.9%	4.4%	86.7%	13.3%	4.2%
Obese mother and normal-weight father	75.3%	24.7%	10%	67.5%	32.5%	12.3%
Obese father and normal-weight mother	74.1%	25.9%	8.4%	71.5%	28.5%	15.3%
Both parents are obese	63.6%	36.4%	27.3%	59.3%	40.7%	17.4%

About 14% of the male children living in families where both parents are normal-weight are at risk of obesity and, among them, 4.4% are obese. These rates double when one of the parents is obese and reach respectively 36.4% and 27.3% if both parents are.

The trend is similar, if not even more pronounced, among 10 years old females.

2.4 Econometric Models

2.4.1 *OLS*

The first econometric model we are going to estimate is a simple OLS model, where we regress cohort member's BMI on their parents' BMI, starting with simple correlations and then adding progressively our controls. Therefore, the model is:

$$BMI_i^{cm} = \alpha + \beta BMI_i^J + \gamma S_i^J + \delta X_i^G + \phi X_i^{SES} + \eta X_i^{FB} + \varphi X_i^H + \varepsilon_i \qquad J = M, F \qquad (1)$$

where the control variable of main interest is parents' educational level (S^J), measured by years of schooling. $X^G, X^{SES}, X^{FB}, X^H$ are four sets of covariates that describe respectively the geographical factors, the socio-economic status of the household, the family background and the parents' health indicators and behavior, while ε_i is a random shock.

If the correlation between parents' and children's BMI is driven down by the inclusion of measures of the family background, socio-economic status and health behaviors, this suggests that it could be possible to intervene to break the intergenerational weight transmission. However, even if our set of control is remarkable, we cannot definitely rule out the presence of omitted variable bias.

We estimate model (1) for each of the three sweeps we are focusing on, that is when the cohort members are 10, 16 and 34 years old[5].

From this first set of regressions we can check the correlation between parents' and children's BMI (given by the coefficient β) and evaluate the persistence of such relation over time.

Intergenerational elasticities are obtained by simply taking the logarithms of individuals' and parents' BMIs in equation (1). In this case β will represent the IGE of BMI, while (1-β) is a measure of intergenerational mobility. Using the log of BMI has the advantage of allowing for non-linearities and to down weight very large BMI values.

As discussed in the introductive section, the individuals' weight status may be influenced also by the interaction between genetic, environmental and behavioral factors. Therefore, one potentially relevant limitation of (1) is that it does not allow for the interaction between parents' BMI and schooling. Hence, we also estimate:

$$BMI_i^{cm} = \alpha + \beta BMI^J + \gamma S_i^J + \delta X^G + \phi X_i^{SES} + \eta X_i^{FB} + \varphi X_i^H + \rho BMI^J * \deg ree^J + \varepsilon_i \quad J = M, F \qquad (2)$$

where $degree^J$ is a dummy variable that takes value 1 if parents hold a degree and 0 otherwise.

[5] In the 3rd regression, when cohort members are 34 years old, we control also for their own years of schooling. It is not necessary to include this variable in the first two regressions (at 10 and 16 years) because we are working with a cohort of individuals and the compulsory school leaving age in UK is just 16 years. Therefore, at least in the first two sweeps considered, all individuals have the same educational level.

2.4.2 *Probit*

Policy-wise, studying the intergenerational transmission of weight excess and establishing whether more educated parents can attenuate this process could be more relevant than analyzing BMI persistence.

With the linear regression we evaluated the relationship existing between the distributions of parents' and offspring's BMI and we estimated the influence that mothers' and fathers' schooling have on the entire BMI distribution of the offspring.

However, it could be more interesting to analyze whether the likelihood of being overweight is affected by the probability that parents suffer from weight excess and by their educational attainment. Indeed, we might expect that parental schooling displays a protective influence when we are close to the overweight or obesity threshold, rather than at other points of the BMI distribution.

Therefore, we now move our attention from the continuous variable BMI to a dummy variable describing individuals' weight condition. Our model becomes:

$$\Pr\left(Oby_i^{cm}=1 \mid Oby_i^J, S_i^J, X_i's\right)=\Phi\left(\alpha+\beta Oby_i^J+\gamma S_i^J+\delta X_i^G+\phi X_i^{SE}+\eta X_i^{FB}+\varphi X_i^H+\rho Oby_i^J * \deg ree_i^J\right) \qquad J=M,F \qquad (3)$$

where Φ is the cumulative density of the standard normal distribution and *Oby* is a dummy variable that takes value 1 if the individual is overweight or obese and 0 otherwise. The probabilities of being overweight or obese are calculated using the thresholds described at the end of Section 3.

Since our dependent variable is now a binary one, we estimate our model using a probit regression. Specifically, we regress cohort members' probability of being overweight or obese on their parents' corresponding probabilities, including progressively parents' years of schooling and the same sets of covariates *X's* used for the OLS estimation. As for model (1), we next introduce also interaction terms between parents' obesity condition and their educational attainment.

Like in the previous OLS analysis, we perform separate estimations for each of the three sweeps considered and, when cohort members are adults, we control also for their own years of schooling.

2.5 Results

2.5.1 Unconditional IGEs, linear correlations and Spearman Rank Order Correlations

Before showing the results of our OLS regressions, we present a summary table with the unconditional (to our set of control variables) IGEs of BMI and the linear and rank order correlations between parents' and children's BMI.

Since (to our knowledge) we are the first ones to include both parents in this kind of analysis, we present novel estimates for the IGE, linear and rank order correlation between *fathers*' and sons and

we can check how IGEs change when considering mothers and fathers separately or jointly, comparing our results with those of previous research that included only mothers.

Table 3 – Unconditional IGEs, Linear Correlations and Rank Order Correlations

	IGE			Linear Correlation (ρ)			Rank Order Corr.		
	1980	1986	2004	1980	1986	2004	1980	1986	2004
Considering parents separately									
Mother-Child	0.171	0.229	0.324	0.214	0.212	0.271	0.217	0.210	0.251
Mother-Son	0.151	0.202	0.269	0.197	0.191	0.248	0.203	0.194	0.250
Mother-Daughter	0.192	0.254	0.380	0.230	0.231	0.302	0.230	0.232	0.269
Father-Child	0.196	0.244	0.346	0.194	0.186	0.231	0.190	0.198	0.226
Father-Son	0.148	0.253	0.278	0.174	0.202	0.209	0.171	0.213	0.220
Father-Daughter	0.248	0.233	0.413	0.215	0.172	0.259	0.210	0.184	0.251
Considering parents jointly									
Mother-Child	0.150	0.200	0.285						
Mother-Son	0.136	0.171	0.239						
Mother-Daughter	0.165	0.229	0.330						
Father-Child	0.167	0.202	0.284						
Father-Son	0.123	0.218	0.231						
Father-Daughter	0.215	0.185	0.334						

Note: all coefficients are significant at the 1% level

From Table 3 we can immediately see that introducing also the fathers into the analysis, the mother-children IGE of BMI goes down, suggesting that previous studies which, because of the lack of information, did not consider fathers' BMI, overestimate the role of mothers in the intergenerational transmission of BMI, likely because of the presence of assortative-mating.

Moreover, it can be noticed that, when considering parents separately, the father-children IGEs are usually greater than mothers' ones. This result is overturned when we move attention to the linear correlation ρ, which is a more suitable measure of intergenerational mobility, since it partially controls for differences in marginal distributions.

However, when both parents are simultaneously taken into account, the gap in IGEs is almost close: mothers-children and fathers-children elasticities turn out to be very similar (especially in 2004, when cohort members and their parents are at similar stages of their lives).

Comparing our 2004 results for the mother-child, mother-son and mother-daughter IGEs and linear correlations with those of Classen (2010) we notice that our values are substantially smaller, suggesting that BMI persistence is lower is U.K. than in U.S. and therefore intergenerational mobility is higher. This is consistent with the findings concerning the intergenerational elasticity in earnings and education, confirming that U.S. tends to be a less mobile society than U.K. and suggesting that the persistence of weight problems across generations might, to some extent, affect the economic mobility of a family.

In line with Classen (2010), we find that the IGEs and correlations between mothers and daughters are greater than those between mothers and sons. On the contrary, this evidence is unclear when looking at the relationship between fathers' and children's BMI.

Finally, we can notice that IGEs increase considerably between age 10 and age 16: this might be due to the convergence of the marginal distribution of offspring's BMI with those of their parents. The rise is indeed smaller, although not negligible, when we look at intergenerational correlations.

This result is confirmed when we compute the Spearman Rank Order Correlation, which fully controls for differences in marginal distributions[6], suggesting that the sharp increase in IGEs is only partially driven by the convergence of marginal distribution. We could interpret this result as a signal of the importance of genetics: even if, growing up, individuals are likely to become more "indipendent" from family habits and environment, their BMI comes to be more correlated with those of their parents.

It is worth noticing that, in the inter-generational context, the Spearman Index has the strong advantage of focusing on positional change, measuring the relationship between rankings of different ordinal variables.

Consistently with the linear correlation index, it displays higher correlation in BMI between children and their mothers, rather than their fathers, suggesting that mothers have a more important role in the transmission of BMI.

2.5.2 OLS estimates

The existence of a positive and statistically significant relationship between the BMIs of parents and 10 years old children is confirmed by OLS estimates that are presented in Tables 4 and 5, where we introduced progressively our set of controls. Parental BMI coefficients are always highly significant and extremely stable across the different model specifications: their size is almost unaffected by the progressive introduction of our sets of covariates, including parental education (columns 2). This

[6] This is the case because both marginal distributions would be standard uniform distributions.

result suggests that genetics plays a strong role in the intergenerational transmission of BMI since observable environmental factors seem not to influence the relationship between parents' and children's BMI. Moreover, as often informally argued by many researchers (see for example Altonji et al., 2005 and Currie and Moretti, 2007), the stability of an explanatory variable's coefficient to the inclusion of additional controls may be considered as a clue of its exogeneity.

Looking at IGEs, we can notice that they are slightly different between males and females: an increase of 10% in either mother's or father's BMI leads to a rise of about 1.3% in sons' BMI, while for daughters an increase of 10% in mother's BMI induces a rise of 1.7%; the same increase in fathers' BMI leads to a growth of more than 2%.

OLS estimates show also the existence of a small negative but significant correlation between maternal education and sons' BMI when they are 10 years old.

Table 4 – OLS estimates (10 years old Boys)

	(1) **BMI 1980**	(2) **BMI 1980**	(3) **BMI 1980**	(4) **BMI 1980**	(5) **BMI 1980**	(6) **BMI 1980**	(7) **BMI 1980**
BMIm	0.0892***	0.0893***	0.0895***	0.0931***	0.0930***	0.0895***	0.0895***
	(8.36)	(9.55)	(9.55)	(9.73)	(9.66)	(9.28)	(9.28)
BMIf	0.0830***	0.0834***	0.0852***	0.0853***	0.0856***	0.0864***	0.0864***
	(9.58)	(8.35)	(8.41)	(8.27)	(8.25)	(8.38)	(8.33)
YSM		-0.0193	-0.0305	-0.0436*	-0.0450*	-0.0392*	-0.0436*
		(0.95)	(1.45)	(1.87)	(1.92)	(1.68)	(1.77)
YSF		0.0186	0.0203	0.0310*	0.0333*	0.0330*	0.0277
		(1.23)	(1.31)	(1.76)	(1.89)	(1.86)	(1.51)
BMIm*degree_m							0.0045
							(0.50)
BMIf*degree_f							0.0055
							(1.09)
Geogr. Factors			Yes	Yes	Yes	Yes	Yes
S.E.S.				Yes	Yes	Yes	Yes
Family Backgr.					Yes	Yes	Yes
Health Indicators						Yes	Yes
IGE wrt BMIm	0.136***	0.136***	0.136***	0.141***	0.141***	0.135***	0.135***
	(11.77)	(10.61)	(10.58)	(10.76)	(10.67)	(10.16)	(10.18)
IGE wrt BMIf	0.123***	0.124***	0.126***	0.126***	0.127***	0.129***	0.129***
	(8.76)	(8.83)	(8.90)	(8.77)	(8.77)	(9.00)	(8.99)
Cons	12.63***	12.62***	15.17***	15.55***	16.45***	17.38***	17.51***
	(42.66)	(35.47)	(15.76)	(14.55)	(10.96)	(9.13)	(9.16)
N	4163	4163	4163	4163	4163	4163	4163

Robust std. errors; *t* statistics in parenthesis; * p < 0.10, ** p < 0.05, *** p < 0.01

In our most complete model specification (column 7), one additional year of mother's schooling is associated to a reduction of 0.0436 points in sons' BMI, while no significant association is found with father's schooling[7].

Concerning daughters, neither mother's nor father's education seems to influence BMI levels.

However, it is worth stressing that the small or insignificant coefficients of parents' years of schooling in column (7) don't imply that parental education is not important in affecting children weight. They simply mean that, once we control for the family background, parents' BMI, socio-economic status and health behaviors, the *direct* influence of parents' schooling, stemming from better parenting, is limited[8].

Table 5 – OLS estimates (10 years old Girls)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	BMI 1980	**BMI 1980**	**BMI 1980**	**BMI 1980**	**BMI 1980**	**BMI 1980**	**BMI 1980**
BMIm	0.110***	0.112***	0.111***	0.109***	0.115***	0.116***	0.113***
	(10.25)	(10.33)	(10.10)	(9.89)	(10.37)	(10.46)	(10.14)
BMIf	0.151***	0.152***	0.149***	0.148***	0.148***	0.146***	0.145***
	(12.02)	(12.06)	(11.61)	(11.56)	(11.72)	(11.61)	(11.38)
YSM		0.029	0.012	0.011	0.004	0.010	0.011
		(1.23)	(0.48)	(0.40)	(0.15)	(0.39)	(0.41)
YSF		0.00280	0.00376	-0.00129	-0.0062	-0.0037	-0.001
		(0.16)	(0.21)	(0.06)	(0.29)	(0.19)	(0.04)
BMIm*degree_m							-0.001
							(0.08)
BMIf*degree_f							-0.003
							(0.46)
Geogr. Factors			Yes	Yes	Yes	Yes	Yes
S.E.S.				Yes	Yes	Yes	Yes
Family Backgr.					Yes	Yes	Yes
Health Indicators						Yes	Yes
IGE wrt BMIm	0.165***	0.168***	0.167***	0.164***	0.166***	0.170***	0.169***
	(12.34)	(11.45)	(11.17)	(10.89)	(11.02)	(11.16)	(11.13)
IGE wrt BMIf	0.215***	0.219***	0.216***	0.214***	0.212***	0.209***	0.208***
	(12.85)	(12.71)	(12.30)	(12.15)	(12.05)	(11.90)	(11.90)
Cons	10.80***	10.42***	11.32***	11.37***	9.54***	8.16***	8.13***
	(30.17)	(23.97)	(11.74)	(10.32)	(6.17)	(4.45)	(4.44)
N	3982	3982	3982	3982	3982	3982	3982

Robust std. errors; *t* statistics in parenthesis; * p < 0.10, ** p < 0.05, *** p < 0.01

[7] However, in some other specifications (columns 4-6), the correlation between fathers' education and sons' BMI is positive and significant, even if small in size.

[8] As explained in the introductive section, we can distinguish between the "*direct*" effect of parental education, that is an improvement in parents' ability to raise children (quality of parenting) and the "i*ndirect*" influences, including all those effects that are mediated by other variables, like income, children education, parents' BMI, etc.

The interaction terms between parents' BMI and the dummy variable for holding a degree are not statistically significant, neither for sons or daughter, meaning that, once we control for our set of covariates, the intergenerational transmission of BMI is not influenced by parents' educational level: the IG transmission of BMI is not weaker for highly educated individuals.

Almost identical results hold when considering different stages of cohort members' lives. Table 6 shows the temporal evolution of IGEs, intergenerational correlations in BMI and parental education influence, comparing the results obtained when the cohort members are 10, 16 and 34 years old. Only specification (7) is reported in Table 6; the full set of regressions (columns 1-7) for ages 16 and 34, which highlight the stability of IGEs and BMI correlations to the inclusion of controls, are shown in Appendix 5. We can easily notice that parents' BMI is significantly and positively correlated with children's BMI in each of the three ages considered. Moreover, as the cohort members grow up, their BMI become more and more correlated with those of their parents.

Table 6 – OLS estimates, Persistence over time

	MALES			**FEMALES**		
	(1)	(2)	(3)	(1)	(2)	(3)
	BMI 1980	**BMI 1986**	**BMI 2004**	**BMI 1980**	**BMI 1986**	**BMI 2004**
BMIm	0.0895***	0.142***	0.257***	0.113***	0.200***	0.387***
	(9.28)	(5.07)	(9.17)	(10.14)	(8.00)	(11.97)
BMIf	0.0864***	0.183***	0.238***	0.145***	0.177***	0.358***
	(8.33)	(6.09)	(8.17)	(11.38)	(6.07)	(10.14)
YSM	-0.0436*	-0.075***	-0.129*	0.011	0.033	0.030
	(1.77)	(2.74)	(1.66)	(0.41)	(1.31)	(0.90)
YSF	0.0277	-0.007	0.153*	-0.001	-0.002	-0.029
	(1.51)	(0.30)	(1.81)	(0.04)	(0.09)	(1.00)
YSCM			-0.068**			-0.033
			(2.45)			(1.17)
BMIm*degree_m	0.0045	0.023	0.010	-0.001	-0.010	-0.049**
	(0.50)	(0.99)	(0.44)	(0.08)	(0.55)	(2.15)
BMIf*degree_f	0.0055	-0.004	-0.010	-0.003	-0.009	0.009
	(1.09)	(0.37)	(0.83)	(0.46)	(0.80)	(0.59)
Geogr. Factors	Yes	Yes	Yes	Yes	Yes	Yes
S.E.S.	Yes	Yes	Yes	Yes	Yes	Yes
Family Background	Yes	Yes	Yes	Yes	Yes	Yes
Health Indicators	Yes	Yes	Yes	Yes	Yes	Yes
IGE wrt BMIm	0.135***	0.166***	0.229***	0.169***	0.215***	0.358***
	(10.18)	(5.64)	(10.18)	(11.13)	(8.01)	(13.95)
IGE wrt BMIf	0.129***	0.209***	0.221***	0.208***	0.203***	0.328***
	(8.99)	(6.54)	(8.87)	(11.90)	(6.31)	(10.90)
Cons	17.51***	20.75***	17.85***	8.13***	7.41*	10.95**
	(9.16)	(5.35)	(3.93)	(4.44)	(1.76)	(2.21)
N	4163	1844	2808	3982	1903	3010

Robust std. errors; *t* statistics in parenthesis; * p < 0.10, ** p < 0.05, *** p < 0.001

In the same way, also IGEs increase. Interestingly, both correlations and IGEs are considerably higher for females, suggesting that the IG transmission of weight is stronger for daughters.

For example, in 2004 a rise of 10% in mother's BMI is associated to a corresponding growth of about 2.3% in the BMI of sons and of 3.6% in that of daughters. The same increase in father's BMI induces a rise of about 2.2% in sons and 3.3% in daughters' BMI.

The estimates in Table 6 provide evidence that mother's education is negatively correlated with sons' BMI also during their adolescence and adulthood. On the contrary, the coefficient of father's schooling is statistically significant (and positive) only in 2004.

When sons become adult, also their own education matters, being negatively correlated with their BMI (column 3).

Concerning daughters, neither mother's nor father's education is *directly* associated with girls' BMI, in none of their stages of life. Only the interaction term between mothers' BMI and the indicator for having a degree is negative and statistically significant, meaning that the IG transmission of BMI is lower for mothers with higher education.

Surprisingly, the daughters' own schooling coefficient turns out to be not significant. However, if we performed our estimation taking into account the whole sample, without splitting between males and females, the association between BMI and own education is negative and statistically significant.

2.5.3 Probit Estimates

Probit estimates for the probability of suffering from weight excess are presented in Table 7 and are consistent with the OLS results we found for the continuous variable describing individuals' BMI. The probability that parents' are overweight or obese is positively correlated with the probability that also their offspring suffer from weight problems. This result holds for both sons and daughters in each of the three ages considered. As for the intergenerational correlation in BMI, also the association between parents' and offspring's probability of suffering from weight excess tends to increase from childhood to adulthood.

The probability that a male cohort member is at risk of obesity (BMI over the 85^{th} percentile) at age 10 is 11.1 percentage points higher if his mother is overweight or obese. This is a large effect given that the average probability of being at risk of obesity for 10 years sons is about 18%. In percentage term this effect is about 60% of the average probability of obesity risk.

When sons are 16 years old, having a mother suffering from weight excess raises the obesity risk by 10.4 percentage points, while the rise is 18% at age 34.

Table 7 – Probit Estimates

	MALES			FEMALES		
	(1) **OBESITY RISK 1980**	(2) **OBESITY RISK 1986**	(3) **O.W. OBESITY 2004**	(1) **OBESITY RISK 1980**	(2) **OBESITY RISK 1986**	(3) **O.W. OBESITY 2004**
Marginal effect to OW/OB Mother	0.111***	0.104***	0.180***	0.113***	0.119***	0.234***
	(8.12)	(5.02)	(7.66)	(8.15)	(5.92)	(9.86)
Marginal effect to OW/OB Father	0.053***	0.060***	0.131***	0.078***	0.067***	0.153***
	(4.63)	(3.48)	(6.38)	(6.84)	(4.23)	(7.66)
Marginal effect to YSM	-0.008	-0.006	-0.007	0.002	0.000	0.001
	(1.60)	(0.82)	(1.23)	(0.46)	(0.09)	(0.27)
Marginal effect to YSF	0.004	-0.007	0.005	-0.006	0.000	-0.004
	(1.21)	(1.15)	(1.31)	(1.53)	(0.18)	(1.32)
Marginal effect to YSCM			-0.010***			-0.001
			(3.27)			(0.29)
Marginal effect to OW/Oby_m*degree_m	0.001	-0.001	0.002	-0.001	-0.002	-0.005
	(0.37)	(0.26)	(0.82)	(0.44)	(0.85)	(1.52)
Marginal Effect to OW/Oby_f*degree_f	0.001	0.000	-0.002	0.000	-0.001	-0.001
	(1.01)	(0.26)	(1.34)	(0.37)	(0.63)	(0.29)
Geographical Factors	Yes	Yes	Yes	Yes	Yes	Yes
S.E.S.	Yes	Yes	Yes	Yes	Yes	Yes
Family Background	Yes	Yes	Yes	Yes	Yes	Yes
Health Indicators	Yes	Yes	Yes	Yes	Yes	Yes
Cons	-1.01***	-0.98***	0.34	-11.44***	-13.03***	-4.86*
	(5.55)	(4.23)	(0.24)	(7.61)	(9.95)	(1.80)
N	4163	1844	2808	3982	1903	3010

t statistics in parenthesis; * $p < 0.10$, ** $p < 0.05$, *** $p < 0.001$

The effect of fathers' overweight is smaller: sons' obesity risk increases respectively by 5.3, 6.0 and 13.1 percentage points at ages 10, 16 and 34.

Concerning daughters, the growth in the obesity probability at the three different stages of life considered is 11.3, 11.9 and 23.4 percentage points if the mother suffers from weight excess and of 7.8, 6.7 and 15.3 if fathers are overweight or obese.

As pointed out by our OLS regressions, the direct influence of parental schooling in the IG transmission of weight seems limited. Only in 1980 the coefficient of mothers' schooling is weakly significant for boys (t-stat=1.60) while that of fathers' is weakly significant for daughters (t-stat=1.53). In both the cases the association is negative.

The interaction terms between the parents' indicators for being overweight and having a degree are never statistically significant, suggesting that the IG transmission of weight problems is not lower in families with more educated parents.

2.6 Intergenerational transmission of BMI and parental schooling

2.6.1 Potential sources of endogeneity

Estimating (1) and (3) as a single equation model will only produce consistent parameter estimates if our regressors are uncorrelated with the error term ε_i.

However, since parents' years of schooling are likely to be correlated with unobservable confounders, we may have a lack of orthogonality between the variable S_i^J and the random component ε_i. This would make our OLS and probit estimates of the coefficients' vector biased.

In principle, the same reasoning might hold also for parents' weight. However, after controlling for parental education and health behaviours, it is likely that the endogeneity issue related to this variable is solved and parents' BMI coefficients reflect the effect of genetics only. This seems to be confirmed by the strong stability of the parental BMI estimates to the gradual inclusion of control variables.

Several unobservable factors may cause a positive association between parental education and child health, including obesity. Among these unobservable confounders we include:

- health endowments: parents' poor health might affect their own educational level (that in turn may affect their weight) and be transmitted to their offspring;
- preferences for investments in education and health: less motivated parents might be unwilling to invest in their own and their children schooling and health prevention, obtaining low educational attainment and rising the probability of weight problems;
- peer group effects: more educated people usually have more educated friends, who are more likely to behave healthily. The social network might induce the individuals to conform their behaviors, exacerbating the effect of own education and acting as a "social multiplier";
- access to prenatal care: highly educated parents have better access to prenatal care. Good access to prenatal care improves both infant health and the health of the mother during and after pregnancy;
- parents' unobservable ability, which might simultaneously influence parents' schooling and children's BMI: parents with higher cognitive skills are likely to get more schooling and may be better in acquiring and processing information to provide an healthy diet and life-style to themselves and their children.

All of these potential sources of endogeneity suggest that the OLS estimates of the relationship between parental education and children BMI are likely to be downward biased.

Even if, in or analysis, we control for a large set of covariates, including:

- parents' health behaviors and some health indicators (as a proxy for parents' health endowments);

- parents' income and place where they live (as a proxy for access to prenatal care);
- type and social rating of the neighborhood (as a proxy for the social network)

not all the previous issues can be taken into account in our regression and therefore the effect of parental education is likely to be overestimated.

When we allow for the endogenity of parental education, the model we are going to estimate is:

$$BMI_i^{cm} = \alpha + \beta BMI^J + \gamma S_i^J + \delta X_i^G + \phi X_i^{SES} + \eta X_i^{FB} + \varphi X_i^H + \varepsilon_{1i} \qquad J = M, F$$

where $S_i^J = \lambda + \delta X_i^G + \phi X_i^{SES} + \eta X_i^{FB} + \varphi X_i^H + \varepsilon_{2i}$

and $Cov(\varepsilon_{1i}, \varepsilon_{2i}) \neq 0$

The most popular approach followed by the economists to solve endogeneity issues and hence identify the causal effect of education is the use of instrumental variables.

The instrument commonly employed to account for the endogeneity of schooling choice are education reforms, specifically rise in the compulsory school leaving age, which yield an exogenous variation in the years of schooling that an individual receive.

Unfortunately, we cannot apply the IV method in our setting, since there is no quasi-experimental variation across our sample that provides an instrument for parents' years of schooling. Three rising of compulsory education have been implemented in U.K. in the 1900s, the first one in 1918, the following in 1947 and the last one in 1973. While the first and third reforms didn't affect at all the educational attainment of cohort members' parents, on the contrary the 1947 reform (that raised compulsory schooling from age 14 to age 15), applying to all individuals born from 1933 onwards, affects almost all the parents in our sample, leaving us with a very small (and not representative, since too old) control group.

2.6.2 An attempt to address parents' schooling endogeneity: the Lewbel's approach

As already emphasized, the values of parental BMI coefficients and IGEs are extremely stable to the introduction of control variables, including parents' schooling. This is an argument in favor of the orthogonality of individuals' BMI to unobservable factors like parents' ability. However, the hypothesis of orthogonality cannot be tested with certainty.

Although the lack of a credible instrument in our setting prevents us from using a standard IV approach in order to get consistent estimates, in the last years several new econometric techniques have been suggested to obtaining identification without using exclusion restrictions but exploiting heteroskedasticity. Among the others, there are Hogan and Rigobon (2002), Klein and Vella (2009, 2010) and Lewbel (2012, 2014).

The main drawback shared by these methods, where identification comes from a heteroskedastic covariance restriction, is the need for strong additional assumptions. Moreover, imposing higher moment conditions, they are likely to provide less reliable estimates than those obtained with standard exclusion restrictions. Finally, they can usually be applied only when the dependent variable is continuous.

That being said, we make an attempt to attain identification using the technique developed by Lewbel (2012)[9], even if we are aware that the results we will found may have a limited relevance.

According to Lewbel (2012), two conditions are sufficient to identify a standard single-equation IV type model without exclusion restrictions: 1) the presence of heteroskedasticity of the errors in the first-stage regression; 2) the existence of a vector of observed exogenous variables *Z* (which might be a subset of the vector of regressors *X* or could equal *X*) uncorrelated with the product of the error terms of the main and secondary equations ($\varepsilon_1, \varepsilon_2$).

Formally, the parameters of interest are identified if there exist exogenous variables *Z* such that:

$$E\left(X^{'} * \varepsilon_j\right) = 0 \qquad j=1, 2 \tag{4}$$

$$Cov\left(Z, \varepsilon_2^2\right) \neq 0 \tag{5}$$

$$Cov\left(Z, \varepsilon_1 * \varepsilon_2\right) = 0 \tag{6}$$

While equation (4) simply requires that the *X*'s are exogenous and equation (5) implies heteroskedasticity, which can be tested using a Breusch-Pagan test[10], equation (6) is not testable (like the instrument excludability from the second-stage equation in standard IV models) and represents the key identification assumption in the Lewbel's approach. Intuitively, the *Z* vector must be orthogonal to the factors that generate the covariance between the error terms, like the family specific effects (for example parents' unobservable ability).

In our context, good candidates for the *Z* vector seem to be the variables contained in the vector of *geographical factors*, X^G, (cohort members' and their fathers', mothers' and maternal grandmothers' region of birth), the dummies accounting for individuals' ethnicity and those indicating whether mother and father are natural parents. All of these variables can arguably be considered exogenous in our setting and thus seem appropriate to be included in *Z*.

[9] For recent applications see Sabia (2007), Denny and Oppedisano (2013), Averett and Stiefel (2007), Giambona and Schwienbacher (2007), Emran and Hou (2013), Rashad and Markowitz (2007).

[10] Across all specifications of our model, the Breush-Pagan heteroskedasticity test reveals the presence of heteroskedasticity both in the first-stage schooling equation and in the second stage.

The estimator proposed in Lewbel (2012) takes the form of two-stage least squares and can be implemented as follows.

In the first stage the endogenous variables are regressed on all of the control variables (X) of the main equation, which include the Z vector:

$$S_i^J = \lambda + \delta X_i + \varepsilon_{2i}$$

The estimated residuals $\hat{\varepsilon}_{2i}$ from these first-stage regressions are then used to create instruments as follows:

$$(Z - \bar{Z})\hat{\varepsilon}_2$$

where $\bar{Z}$ is the sample mean of Z[11].

Finally, the primary equation is estimated by instrumental variables using X and $(Z - \bar{Z})\hat{\varepsilon}_2$ as instruments. The assumption that Z is uncorrelated with $\varepsilon_1\varepsilon_2$ means that $(Z - \bar{Z})\hat{\varepsilon}_2$ is a valid instrument for S^j, since it is uncorrelated with ε_1.

It is worth noting that the Lewbel's (2012) estimator can be applied only if the dependent variable is continuous. Therefore, we can use this procedure just in the analysis of the IG transmission of BMI, but not in studying the persistence of weight problems.

The estimates obtained applying the Lewbel's (2012) technique are presented in Table 8. As it can be immediately noticed, parents' BMI coefficients and the corresponding IGEs are extremely closed to the OLS ones. The same holds for the estimates of the parental schooling influence.

Even if we are aware that these results should be taken with caution, they suggest that the endogeneity of parents' education is likely a limited issue in our model and confirm that individual BMI seems to be orthogonal both to our set of observable controls and to unobservable confounders.

Table 8 – Lewbel's Estimates

	MALES			FEMALES		
	(1)	(2)	(3)	(4)	(5)	(6)
	BMI 1980	BMI 1986	BMI 2004	BMI 1980	BMI 1986	BMI 2004
BMIm	0.090***	0.143***	0.258***	0.113***	0.200***	0.387***
	(11.04)	(6.18)	(11.58)	(11.71)	(8.74)	(15.61)
BMIf	0.087***	0.182***	0.238***	0.139***	0.177***	0.359***
	(8.99)	(6.76)	(8.80)	(11.58)	(6.68)	(11.61)
YSM	-0.040*	-0.066*	-0.124***	0.002	0.029	0.025
	(1.68)	(1.81)	(2.69)	(0.13)	(0.89)	(0.58)

[11] Note that these are not conventional instrumental variables, since Z may appear in the individuals' BMI equation. Only the exogeneity of Z is needed.

YSF	0.011	-0.004	0.151***	-0.001	-0.005	-0.032
	(1.15)	(0.11)	(5.09)	(0.13)	(0.22)	(1.00)
YSCM			-0.069**			-0.032
			(2.85)			(1.22)
Geogr. Factors	Yes	Yes	Yes	Yes	Yes	Yes
S.E.S.	Yes	Yes	Yes	Yes	Yes	Yes
Family Backr.	Yes	Yes	Yes	Yes	Yes	Yes
Health Indicators	Yes	Yes	Yes	Yes	Yes	Yes
IGE wrt BMIm	0.136***	0.170***	0.230***	0.169***	0.214***	0.358***
	(11.42)	(6.71)	(11.67)	(12.13)	(8.21)	(15.70)
IGE wrt BMIf	0.129***	0.204***	0.221***	0.201***	0.203***	0.329***
	(9.17)	(6.85)	(9.33)	(11.80)	(6.64)	(11.86)
_cons	16.93***	21.61***	16.54***	8.76***	7.53	10.92*
	(7.08)	(3.68)	(4.07)	(4.13)	(1.58)	(1.99)
N	4163	1844	2808	3982	1903	3010

*t statistics in parenthesis; * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$*

2.7 Conclusions

In this chapter we used data from the British Cohort Study of 1970 to analyze the transmission of BMI and weight excess from one generation to the following and to investigate the factors that affect this process, focusing on the influence of parental education.

While economists broadly examined the intergenerational transmission of earnings and education, much less attention was devoted to the IG transmission of weight problems. The few existing studies focus on U.S. data and, due to the lack of information, only the role of mothers.

We contribute to this literature in several directions. To our knowledge, we are the first ones to use U.K. data and to include also fathers in this kind of analysis, computing intergenerational elasticities and correlations between the BMI of both parents and their children. Moreover, we introduce in our regressions interaction variables between parents' BMI and dummy variables indicating whether they hold a degree, in order to explore whether the IG transmission of weight differs according to the parents' educational qualification.

Finally, the longitudinal structure of our dataset allows us to follow individuals over time (at ages 10, 16 and 34), studying the temporal evolution of IGEs and BMI correlations.

The unconditional values of the IGE and of the linear and rank order correlations of BMI, computed in the first part of our empirical analysis, display a positive and statistically significant association between parents' and children BMI. This correlation increases over time and is stronger between mothers and children.

Interestingly, the mother-children IGE decreases when we consider both parents jointly, suggesting that previous studies (Classen, 2010) that did not take into account fathers' BMI, overestimated the role of mothers in the intergenerational transmission of BMI, likely because of the presence of assortative-mating

The following OLS analysis confirms the existence of a positive and highly significant correlation between parents' and children's BMI. This relationship is not influenced by the progressive introduction in our regressions of a large set of covariates accounting for individuals' environment, socio-economic status, family background and health behaviours: parents' BMI coefficients remain extremely stable across the different specifications of the model. This finding suggests a strong role for genetics in the intergenerational transmission of weight problems. On the contrary, parental years of schooling have a little and often not significant effect on their offspring's BMI. Only mothers' education turns out to have a protective effect, but exclusively on sons' BMI.

It is worth stressing that this modest effect doesn't mean that parental schooling is not important in influencing children's BMI. It simply means that, once we control for the parents' BMI and health behaviors, family background, socio-economic status and individuals' own education, the improvement in the quality of parenting that may stem from additional years of schooling has a little effect on children's weight.

Not even the interaction terms between parental BMI and indicators for having a degree turn out to be relevant, suggesting that the IG transmission of BMI is not weaker in more educated families.

All of these results are confirmed when we move our attention from the persistence of BMI to the transmission of weight excess, using dummy variables accounting for the existence of weight problems. Our probit estimates are consistent with OLS ones: overweight and obese parents' are more likely to have offspring suffering from weight excess and the role played by parental education is limited.

Despite the remarkable stability showed by parental BMI coefficients and IGEs to the introduction of control variables, including parents' schooling, is an argument in favor of the orthogonality of individuals' BMI both to observable and unobservable factors, the existence of several potential sources of endogeneity stemming from parental education suggests to explicitly face this issue.

Given the impossibility to find a credible instrument in our setting, we make an attempt to get consistent estimates using a recent method developed by Lewbel (2012), that exploits heteroskedasticity for identification and does not rely on standard exclusion restrictions, being aware that the results we will found may have a limited relevance.

The estimates obtained using the Lewbel's approach are very closed to the OLS ones, suggesting that the endogeneity of parental education is likely a limited issue in our model.

In conclusion, our results showed that a significant intergenerational transmission of BMI and weight excess takes place. The correlation between parents' and children's BMI is very stable across the different model specification in which we have gradually included a large set of control variables, suggesting a strong role for genetics in the intergenerational transmission of weight problems, as previously suggested by medical studies using samples of adoptees, like Grilo et al. (1991) and Vogler et al. (1995). On the contrary, after controlling for our set of covariates, OLS and Probit estimates reveal that parents' education has a little and often not significant effect on offspring's BMI, suggesting that the better parenting that may stem from additional schooling might be not so important in influencing the weight transmission.

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2.9 Appendices

Annex 1 – Classification of the control variables

Geographical Factors:

- mother's region of birth
- father's region of birth
- mother's mother region of birth
- child birth country
- type of neighbourhood where the household lives (e.g. inner urban area, rural, council estate, outskirts of town or city ….)

Socio-Economic Status of the Household:

- father socioeconomic group
- mother socioeconomic group
- father social class
- mother social class
- social rating of neighbourhood
- dummy for father being employed or unemployed
- dummy for mother being employed or unemployed
- father's type of employment
- mother's type of employment
- father's father occupation
- mother's father occupation
- dummy for mother being part-time or full-time employed
- total gross family income

Family Background Characteristics

- mother's age at delivery
- age of father
- ethnic group
- parents' marital status
- relationship to child of mother figure
- relationship to child of father figure

- number of persons in the household
- number of children in the household
- number of mother’ previous pregnancies

Health Indicators and Health Behaviours

- standing height of mother in cm
- birthweight of baby in grams
- dummy for breast feeding
- dummy for mother smoking during pregnancy
- mother's present smoking habits
- father's present smoking habits
- dummy for whether any other person in the house smokes
- mother’s smoking habits since 1970
- father’s smoking habits since 1970
- dummy for mother drinking during early pregnancy
- dummy for mother drinking during late pregnancy
- dummy for sport being an habitual spare time activity for the child
- dummy for watching TV being an habitual spare time activity for the child

Annex 2 - Causes of data reduction

Survey Year	Total Number of Observations	Lack of observations due to difficult tracing and deaths (with respect to the birth survey)	Number of observations dropped because of missing data (and multiple births)	Number of observations that can be exploited in our analysis
1970	17,198	-	588	16,610
1975	13,135	4,063	1,158	11,510
1980	14,875	2,323	6,730	**8,145**
1986	11,615	5,583	8,128	**3,487**
2004	9,655	7,543	4,395	**5,260**

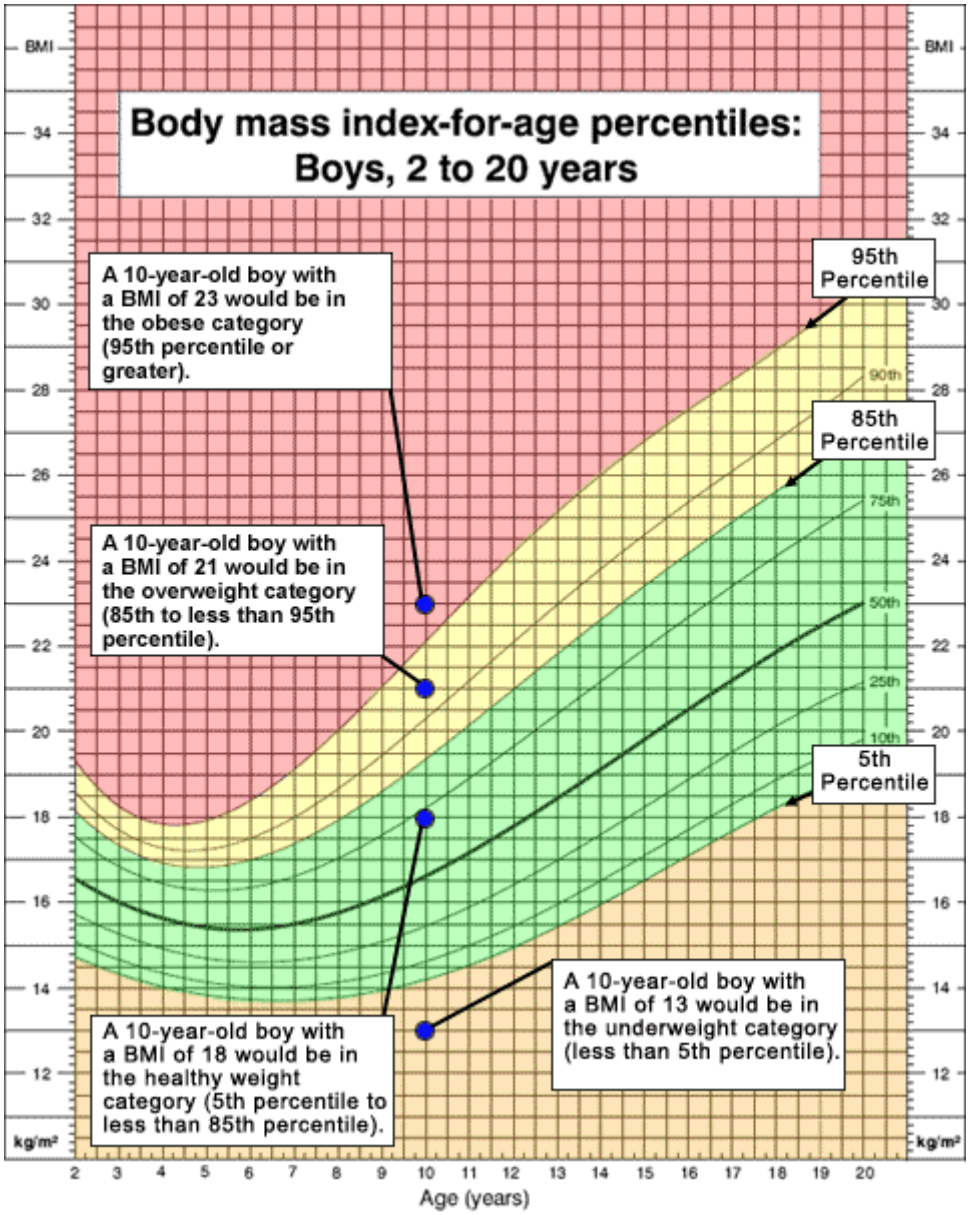
Annex 3 – Attrition Issues

This technique is based on a simple two-steps procedure. In the first step we study whether the probability that a cohort member appears in a given sweep depends on his/her observable characteristics and we compute predictions. From these predictions we create the Inverse Mills Ratio and finally we check whether its coefficient lambda is significant in the regressions having BMI (at different time) as the dependent variable.

The sweep of 1980 is the first one we are using in our analysis and therefore we are not going to check whether it might be affected by attrition issues.

Concerning the sweeps of 1986 and 2004, in none of the two cases the lambda coefficients turn out to be significant: their p-values are respectively 0.695 and 0.423, suggesting that, despite the sensible drop in the number of observations, our dataset seems not to suffer from attrition problems.

Annex 4 - BMI thresholds for children and boys below age 20



Source: Developed by the National Centre for Health Statistics in collaboration with the National Centre for Chronic Disease Prevention and Health Promotion

Annex 5 – OLS estimates for ages 16 and 34 (full set of model specifications)

Table A – Males, age 16

	(1) BMI 1986	(2) BMI 1986	(3) BMI 1986	(4) BMI 1986	(5) BMI 1986	(6) BMI 1986	(7) BMI 1986
BMIm	0.146***	0.141***	0.145***	0.148***	0.146***	0.143***	0.142***
	(6.17)	(5.82)	(6.02)	(6.03)	(5.40)	(5.13)	(5.07)
BMIf	0.188***	0.191***	0.185***	0.187***	0.185***	0.183***	0.183***
	(7.32)	(7.39)	(7.19)	(7.05)	(6.12)	(6.10)	(6.09)
YSM		-0.040***	-0.038**	-0.045**	-0.066**	-0.066**	-0.075***
		(2.89)	(2.24)	(2.16)	(2.50)	(2.45)	(2.74)
YSF		0.017	0.007	0.001	-0.008	-0.004	-0.007
		(1.31)	(0.46)	(0.07)	(0.35)	(0.18)	(0.30)
BMIm*degree_m							0.023
							(0.99)
BMIf*degree_f							-0.004
							(0.37)
Geogr. Factors			Yes	Yes	Yes	Yes	Yes
S.E.S.				Yes	Yes	Yes	Yes
Family Backr.					Yes	Yes	Yes
Health Indicators						Yes	Yes
IGE wrt BMIm	0.171***	0.165***	0.170***	0.172***	0.170***	0.167***	0.166***
	(7.01)	(6.63)	(6.74)	(6.62)	(5.97)	(5.71)	(5.64)
IGE wrt BMIf	0.218***	0.224***	0.216***	0.215***	0.211***	0.209***	0.209***
	(7.97)	(8.09)	(7.78)	(7.48)	(6.54)	(6.55)	(6.54)
_cons	12.85***	13.11***	22.43***	22.43***	23.98***	20.73***	20.75***
	(17.86)	(17.14)	(11.10)	(10.33)	(7.74)	(5.34)	(5.35)
N	2149	2080	2080	2080	1844	1844	1844

t statistics in parentheses
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table B – Females, age 16

	(1) BMI 1986	(2) BMI 1986	(3) BMI 1986	(4) BMI 1986	(5) BMI 1986	(6) BMI 1986	(7) BMI 1986
BMIm	0.208***	0.208***	0.204***	0.198***	0.199***	0.200***	0.200***
	(9.99)	(9.85)	(9.24)	(8.65)	(8.04)	(7.97)	(8.00)
BMIf	0.166***	0.169***	0.164***	0.161***	0.178***	0.177***	0.177***
	(6.36)	(6.47)	(6.24)	(5.85)	(6.08)	(6.07)	(6.07)
YSM		0.023	0.021	0.032	0.029	0.029	0.033
		(0.98)	(0.83)	(1.26)	(1.03)	(1.16)	(1.31)
YSF		0.0256	0.024	0.021	-0.000	-0.005	-0.002
		(0.99)	(0.98)	(0.95)	(0.01)	(0.28)	(0.09)
BMIm*degree_m							-0.011
							(0.55)
BMIf*degree_f							-0.009
							(0.80)
Geogr. Factors			Yes	Yes	Yes	Yes	Yes
S.E.S.				Yes	Yes	Yes	Yes
Family Backr.					Yes	Yes	Yes
Health Indicators						Yes	Yes
IGE wrt BMIm	0.229***	0.228***	0.225***	0.218***	0.216***	0.214***	0.215***
	(10.00)	(9.87)	(9.36)	(8.79)	(8.19)	(7.99)	(8.01)
IGE wrt BMIf	0.185***	0.192***	0.187***	0.183***	0.204***	0.203***	0.203***
	(6.51)	(6.69)	(6.36)	(5.97)	(6.32)	(6.31)	(6.31)
_cons	12.44***	11.89***	13.46***	13.98***	11.93***	7.531	7.410
	(16.09)	(13.56)	(5.99)	(5.79)	(3.36)	(1.79)	(1.76)
N	2201	2148	2148	2148	1903	1903	1903

t statistics in parentheses
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table C – Males, age 34

	(1) BMI 2004	(2) BMI 2004	(3) BMI 2004	(4) BMI 2004	(5) BMI 2004	(6) BMI 2004	(7) BMI 2004
BMIm	0.266***	0.266***	0.260***	0.263***	0.256***	0.258***	0.257***
	(11.07)	(10.81)	(10.61)	(10.24)	(9.41)	(9.21)	(9.17)
BMIf	0.244***	0.249***	0.237***	0.226***	0.239***	0.238***	0.238***
	(9.07)	(9.06)	(8.66)	(8.13)	(8.19)	(8.17)	(8.17)
YSM		-0.166**	-0.152**	-0.0992*	-0.143*	-0.125	-0.129*
		(2.44)	(2.28)	(1.70)	(1.81)	(1.59)	(1.66)
YSF		0.0955*	0.101*	0.113**	0.146*	0.150*	0.153*
		(1.69)	(1.72)	(2.11)	(1.75)	(1.81)	(1.81)
YSCM						-0.069**	-0.068**
						(2.51)	(2.45)
BMIm*degree_m							0.010
							(0.44)
BMIf*degree_f							-0.011
							(0.83)
Geogr. Factors			Yes	Yes	Yes	Yes	Yes
S.E.S.				Yes	Yes	Yes	Yes
Family Backr.					Yes	Yes	Yes
Health Indicators						Yes	Yes
IGE wrt BMIm	0.239***	0.237***	0.232***	0.232***	0.228***	0.230***	0.229***
	(12.25)	(11.89)	(11.60)	(11.15)	(10.32)	(10.22)	(10.18)
IGE wrt BMIf	0.231***	0.236***	0.227***	0.216***	0.222***	0.221***	0.221***
	(10.20)	(10.24)	(9.85)	(9.21)	(8.85)	(8.87)	(8.87)
_cons	14.53***	15.09***	17.04***	17.58***	19.00***	17.81***	17.85***
	(18.69)	(16.35)	(7.60)	(6.81)	(4.85)	(3.94)	(3.93)
N	3332	3230	3230	3230	2809	2808	2808

t statistics in parentheses
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table D – Females, age 34

	(1) BMI 2004	(2) BMI 2004	(3) BMI 2004	(4) BMI 2004	(5) BMI 2004	(6) BMI 2004	(7) BMI 2004
BMIm	0.357***	0.352***	0.347***	0.344***	0.374***	0.387***	0.387***
	(12.97)	(12.29)	(12.03)	(11.83)	(11.83)	(11.98)	(11.97)
BMIf	0.363***	0.353***	0.344***	0.337***	0.363***	0.359***	0.358***
	(12.14)	(11.54)	(10.94)	(10.52)	(10.28)	(10.16)	(10.14)
YSM		-0.0181	-0.0117	0.0122	0.0102	0.0246	0.0302
		(0.81)	(0.54)	(0.54)	(0.34)	(0.76)	(0.90)
YSF		-0.0302	-0.0279	-0.0199	-0.0323	-0.0321	-0.0294
		(1.21)	(1.21)	(0.78)	(1.05)	(1.08)	(1.00)
YSCM						-0.0322	-0.0327
						(1.15)	(1.17)
BMIm*degree_m							-0.0492**
							(2.15)
BMIf*degree_f							0.00883
							(0.59)
Geogr. Factors			Yes	Yes	Yes	Yes	Yes
S.E.S.				Yes	Yes	Yes	Yes
Family Backr.					Yes	Yes	Yes
Health Indicators						Yes	Yes
IGE wrt BMIm	0.330***	0.323***	0.318***	0.316***	0.346***	0.358***	0.358***
	(14.94)	(14.12)	(13.75)	(13.47)	(13.72)	(13.95)	(13.95)
IGE wrt BMIf	0.334***	0.326***	0.319***	0.312***	0.332***	0.329***	0.328***
	(12.90)	(12.33)	(11.82)	(11.34)	(11.03)	(10.92)	(10.90)
_cons	7.958***	8.817***	8.885**	8.151**	10.80**	10.92*	10.95*
	(8.88)	(8.73)	(2.94)	(2.63)	(2.65)	(2.20)	(2.21)
N	3560	3459	3459	3459	3011	3010	3010

t statistics in parentheses
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Chapter 3

Short and Long-run Effects of Obesity on Cognitive Skills

3.1 Introduction

Recent medical research[12] has proved the existence of a negative relationship between obesity and cognitive skills, finding that the Intelligent Quotient (IQ) and fatness are negatively correlated.

Also in the field of economics literature some studies analyzed the consequences of obesity on cognitive ability, measured by standardized tests, and educational outcomes, such as school performance (e.g. Datar, Sturm and Magnabosco, 2004; Kaestner and Grossman, 2009; Cawley and Spiess, 2008).

We investigate this issue using data from the British Cohort Study, which follows a cohort of U.K. individuals from their birth, in 1970, until nowadays. This longitudinal dataset contains the results of a cognive test, the British Ability Scale (BAS), sit in 1980 when individuals are 10 years old, and of a basic skills test, taken in 2004 when they are 34. In the same years information about individuals' BMI are recorded. Therefore, we can study the impact of current BMI levels on the individuals' test scores. Moreover, we can evaluate the lagged effect of 1980 Body Mass levels on the basic skill test performed in 2004 and study whether a change in the weight status from childhood to adulthood may affect the test result.

The topic is of great interest since cognitive ability, educational achievements and basic skills are important determinants of individual's productivity and economic outcomes. For this reason, policies targeted to reduce obesity rates may have economic implications that go beyond the well-known savings in health expenditure.

Several studies have indeed highlighted the negative effect of weight excess on wages and employment probability. However, not enough attention has been put on the channels driving these relationships: a decline in cognitive ability caused by obesity might explain part of the wage and employment gap found between obese and non-obese workers.

Therefore, our contribute aims to complement this strand of literature, shedding new light on the potential origins of such disparities.

In turn, the relationship between weight excess and individuals' test scores can be driven by several mechanisms. First of all, obesity could cause a decline in cognitive ability. One possible medical explanation is that the hormones secreted by fat could damage brain's cells (Cournot M. et al., 2006). Cognitive problems can also follow from deficiencies of certain micronutrients such as zinc, iron and iodine (Taras, 2005) for which overweight people may be at risk because of the consumption of cheap, energy-rich but nutrient-poor food (Nead et al. 2004). Moreover, it is well known that weight problems are responsible for many chronic diseases (such as cardiovascular

[12] Archana Singh-Manoux et al. (2012), Cournot M. et al. (2006), Thompson P.M. et al. (2010).

disease, hypertension, coronary-heart diseases, sleep disorders, etc.) that could alter cognitive functioning.

Obesity may also have adverse psychological effects. Strauss (2000) found a positive correlation between weight problems and low self-esteem, while Faith, Matz and Jorge (2002) documented a positive association linking depression with obesity. Psychological problems, as well as health disorders, could be responsible for a decline in cognitive functioning.

Finally, weight excess could harm human capital accumulation. For example, obese children are more likely to be absent from school than non-obese (Geiner et al. 2007), they can be discriminated by teachers (Redline et al. 1999) and bullied by their peers. This can negatively influence their learning environment, resulting in a lower educational achievement, which is important in determining cognitive ability. Moreover, educational attainment affects the probability of finding a good job, which in turn can influence the level of skills acquired at the workplace.

However, the association between obesity and cognitive skills might be driven also by unobservable confounders like individual and family background characteristics that simultaneously affect both weight and cognitive ability. In particular, parental cognitive skills[13], socio-economic position and attitudes toward education might be important omitted variables.

Part of the relationship linking obesity and cognitive ability could also be explained by reverse causality: differences in cognitive ability might cause differences in adiposity. Individuals with lower IQ and education might be less able to acquire and process health information and can therefore choose unhealthy behaviours and lifestyle that may lead to obesity. In addition, it is possible that they are more likely to suffer from psychological discomfort: discrimination, lower popularity and engagement in social activities could induce depression and over-eating.

We address the reverse causality and the unobserved heterogeneity issues by means of instrumental variables. Following the approach by Sabia (2007) and Averett and Stiefel (2007) we instrument individual's BMI with a relative's BMI.

Parental BMI was previously used as an instrument for offspring's BMI by Cawley (2000, 2004) and Brunello and d'Hombres (2005) that, however, focused on wages and employment disability as outcomes of interest.

[13] De Coulon, Meschi and Vignoles (2008) showed that parents' basic skills in literacy and numeracy have a positive significant effect on their children's cognitive test scores, over and above the positive effects of parental education and ability

The first requirement for the model identification is that parent's BMI is strongly correlated with that of their offspring (conditional on other covariates). This is likely to be the case as showed by recent research[14].

The second identification assumption requires that parental BMI is not correlated with unobservable determinants of children cognitive ability. This hypothesis could be problematic if parental obesity is correlated with some family-level environmental characteristics that can influence children cognitive skills. However, as highlighted by Cawley (2004) there is little empirical evidence of the effect of a common household environment on BMI: genetics rather than the family context is the key factor in explaining body weight. Despite this evidence, in order to enhance the credibility of our instrument, we control for a large set of variables describing parents' attitudes toward education and schooling that are available in our dataset.

We shed new light on the causality of the link existing between weight problems and individual's cognitive skills using for the first time in this kind of literature data from the British Cohort Study of 1970 (BCS70). This dataset contains information on *both* parents' BMI: the availability of two instrumental variables guarantees an efficiency gain in the estimation and provides us with an additional element to check their validity, that is, the possibility to perform an over-identification test. This was not possible in all of the previous studies, using just one relative's BMI as an IV for the individual's BMI.

Moreover, the BCS70 allows us to control for many important variables (such as birth-weight, the psychological condition, whether the individual was breast-fed when infant, parents' education and socio-economic status, parental attitudes and school sentiment, etc.) that were often missed in the above mentioned studies.

Finally, we focus in the same research on two different outcomes: a cognitive test performed in childhood and a literacy and numeracy skills test carried out in adulthood. This latter outcome is of particular interest, since most of the previous economic literature on this topic has focused only on children and adolescents.

The panel structure of our data provides us two different observations of individuals' BMI, one in 1980, when cohort members are 10 years old and the other in 2004, when they are 34. Therefore, in addition to the effect of current BMI on each of the tests, we can study both the impact of lagged Body Mass and the implications of a change in the weight status (from childhood to adulthood) on the test performed in 2004.

[14] See Comuzzie and Allison (1998) and Castelnovo (2013)

The outline of this chapter is as follows. Section 2 contains a literature review; the data used are described in section 3, while section 4 presents the empirical models and the estimation strategies employed. In section 5 we show our results. Conclusions follow.

3.2 Literature Review

It is well known from the medical literature that obesity has important negative consequences on individuals' health. This has in turn relevant economic implications that have drawn the attention of economists, who initially focused on the effect of obesity on outcomes such as wages and employment. Within this literature we can distinguish between studies investigating the existence of an association between weight excess and economic outcomes and those trying to understand whether such relationship is actually causal.

One of the first papers looking at the consequences of obesity on wages is Sargent and Blanchflower (1994), that showed a negative association between obesity at 16 years and earnings at 23 for British women but not for men. Female adolescents who were in the top 10% of the body mass index distribution at age 16 earned 7.4% less than their non-obese peers and those in the top 1% earned 11.4% less, while no statistically significant effect is found for males.

Han, Norton and Powell (2009) highlight the existence of a negative correlation between late teen BMI and future wages also in the U.S. and distinguish between the direct BMI wage penalty, operating through employers' discrimination, and the indirect effects driven by poor educational and occupational choices. Their results show a total 0.96% decrease in wages for each additional unit of late teen BMI among women. A significant portion (19%) of the total wage penalty is due to the indirect effects of BMI, that occurs prior to employment. As in Sargent and Blanchflower (1994), they didn't find a significant direct effect of BMI on wages for men. However, they showed that higher levels of late teen BMI for men slightly decrease hourly wages via the indirect pathways of education and occupational choice.

Averett and Korenman (1996) showed that in U.S.A. obese women live in families with low income while results for men are weaker and mixed. There is also some evidence of labor market discrimination against obese women. However, differences in marriage probabilities and in spouse's earnings account for 50 to 95% of their lower economic status.

Focusing on several economic outcomes, Cawley and Danziger (2005) found that high body weight is a great barrier to labor market success for white women but not for African-American women. Among white women, they found a significant negative correlation between weight and labor market outcomes such as employment, hours worked, and earnings.

All of the studies cited above established the existence of a negative association but not of a causal effect of obesity on wages, since they do not account for the potential endogeneity of individuals' BMI.

Probably the first study facing this endogeneity problem is Cawley (2000), who exploited information on a sibling's BMI as an IV for individual's own BMI. The outcome of interest is now employment disability and the results reveal no causal effect of body weight.

The same IV approach is used in Cawley (2004), but here the relation studied is the one between BMI and wages. Using data from the National Longitudinal Survey of Youth (NLSY) Cawley found that weight has a negative causal impact only on white females' wages. No evidence is found for males or for black females.

On the contrary, using data from 9 E.U. countries and the average of relatives' BMI as an instrument, Brunello and d'Hombres (2005) found that the causal impact of obesity on wages is independent of gender dimension. It is negative a statistically significant for countries belonging to the "olive belt" and positive for Northern and Central Europe States.

A different instrument, that is genetic markers (whose validity will be discussed later in the chapter), is employed in Norton and Han (2008), that found no causal effect of obesity on neither employment probability nor wages.

Finally, Pinkston (2012) is the first paper to consider effects of body mass on wages in a dynamic panel data model and uses fixed effects estimation to account for the endogeneity of body mass. This framework allows the author to consider the effects of both current and past BMI, while also controlling for past wages. Estimation results suggest that past levels of body mass negatively affect the wages of young workers more than current BMI and this is especially true for white men. The only evidence of an effect of current weight is a penalty faced by morbidly obese women. This finding supports the focus of Han et al. (2008) on the indirect effects of body mass on wages.

More recently, economists have focused also on the relationship between weight excess and academic and cognitive achievements. The issue has been investigated at different ages, from early childhood to university-age students. Also in this case it is possible to distinguish between studies establishing a simple correlation and those looking for a causal effect, this latter category being a minority. As explained in the following, the evidence provided is unclear.

Cawley and Spiess (2008) evaluated skill attainment in children from 2 to 4 years old, finding that, among boys, obesity is associated with reduced verbal, social and motor skills, while for girls is associated only with reduced verbal skills.

The link between weight excess and the academic performance of U.S. elementary school children was examined by several authors. Datar, Sturm and Magnabosco (2004) showed that overweight

children had significantly lower math and reading test scores compared with non-overweight peers in kindergarten and at the end of grade 1. However, these differences, except for boys' math scores became insignificant after controlling for socioeconomic and behavioral variables, suggesting that overweight is a marker but not a causal factor.

Datar and Sturm (2006) focused on several outcomes (math and reading standardized test scores, school absences, grade repetition) showing that change in overweight status during the first 4 years in school is a significant risk factor for adverse school outcomes among girls but not boys.

Different findings are obtained by Kaestner and Grossman (2009) using a sample of U.S. children's between the ages of 5 and 12 and the Peabody Individual Achievement Tests in math and reading as an outcome. Their results suggest that, in general, overweight or obese children get achievement test scores that are about the same as children with average weight

Li, Dai, Jackson and Zhang (2008), focusing on a sample of U.S. school-age children and adolescents provide evidence that while the association between BMI and academic performance was not significant after adjusting for parental and family characteristics, the one between cognitive functioning remained significant after adjusting for family background, physical activity, hours spent watching TV and some health and psychosocial indicators.

Sigfusdottir, Kristjansson and Allegrante (2007) explored the relationship between health behaviours and academic achievement in Icelandic 14- and 15-year old students. Body Mass

Index, diet and physical activity explained up to 24% of the variance in academic achievement when controlling for gender, parental education, family structure and absenteeism.

On the contrary, Kaestner, Grossman and Yarnoff (2009) using as outcomes highest grade attended, highest grade completed and drop out status, found that, in general, overweight or obese U.S. adolescents (between the ages of 14 and 18) have levels of attainment that are about the same as teens with average weight.

Contrasting results are obtained also by studies focusing on high school students.

Fuxa and Fulkerson (2011), using a sample of Minnesota students, found that overweight and obese adolescents were significantly less likely to plan to go to college and more likely to report skipping school and to have lower academic grades than non-overweight peers. Okunade, Hussey, Karakus (2009) suggest no adverse impact of overweight or obesity on timely high school completion for males, but a significant average negative effect on females, in particular white and Asian females. No significant effects were found for African-Americans. Finally, according to Karnehed et al. (2006), 18 years old Swedish obese students were ½ as likely to get into higher education.

Even if several researchers investigated the potential link between weight problems and educational or cognitive achievements, only few studies have focused on the causality of this relationship.

These works typically employ an instrumental variable approach to face the endogeneity problem that is likely to affect individuals' BMI. Among them, we can distinguish between studies using a relative's BMI as IV and those using genetic markers.

In the former category we include Sabia (2007), who studied the relationship between the body weight of U.S. adolescent and their academic achievement, founding consistent evidence of a significant negative relationship between BMI and grade point average in math and English language for white females aged 14-17. Instead, for non-white females and males, there was less convincing evidence of a causal link between body weight and academic performance after controlling for unobserved heterogeneity.

The main problem of this paper is the use of subjective and self-reported measures of parental obesity. Indeed, the variables used as instruments are neither parents' BMI levels nor dummies indicating whether they actually are overweight or obese, but rather variables stating whether they *feel* obese or not. Therefore, they inform about parents' *perceived* obesity, being not objective measures of their real weight status. Moreover, grade point averages are self –reported by students that may have an incentive to over-report their grades.

A second study using relatives' weight status as an IV is Averett and Stiefel (2007) that focused on two types of childhood malnutrition: not only over- but also under-weight. They use a sample of 5-years old children from the NLSY79 to investigate the cognitive consequences of child malnutrition, concluding that malnourished children tend to have lower cognitive abilities when compared to well-nourished children.

Because of the lack of information in their dataset, they can rely only on maternal BMI as an IV, so that they cannot perform an over-identification test to check the validity of the instrument.

The literature using genetic markers in order to recover identification includes Fletcher and Leherer (2008), Norton and Han (2008), Ding et al. (2009) and Von Hinke Kessler Scholder et al. (2010).

The latter study is the only one using a U.K. dataset, the Avon Longitudinal Study of Parents and Children[15] (ALSPAC), and moves a critique to the preceding works. As the authors pointed out, there is a week and inconsistent evidence in the medical literature that the genetic variants employed in the prior studies are robustly associated with fatness in large population samples. This is a serious problem since weak association may result in biased estimates.

Moreover, even if a suitable and robust genetic instrument is available, it may explain little of the variation in observed phenotype: if the alleles shift the adiposity distribution by a very small

[15] The ALSPAC dataset collects information about a cohort roughly 14.000 children born in one geographic area of England, the Avon, between April 1991 and December 1992.

amount, the effect of fatness on test scores is identified only by this small difference in mean adiposity.

The variants used by Von Hinke Kessler Scholder et al. (2010) are currently the best candidates to be used as genetic markers, since they have been shown to be associated with adiposity in large population samples. However, the authors admit that, while their instruments are not weak in a statistical sense, their effects may be "too small to impact on the possible pathways to academic performance", concluding that genetic instruments should be used with care. Taking into account these observations, it is not surprising that none of these studies find a significant effect of fatness on academic performance.

3.3 Data and summary statistics

We use data from the British Cohort Study (BCS70), a longitudinal dataset collecting information on the births and families of babies born in England, Scotland, Wales and Northern Ireland in a particular week in April 1970 and following their lives until nowadays. Since the birth survey there have been seven "sweeps" of cohort members at ages 5, 10, 16, 26, 30, 34 and 38. The strength of the BCS70 is the vast amount of information it provides about cohort members' environment, family background, educational attainment, socio-economic and health status. This characteristic allows us to control for a large set of covariates in our regressions.

In our analysis we focus mainly on the 2^{nd} (age 10) and 6^{th} (age 34) sweeps. In the 2^{nd}, carried out in 1980, cohort members are required to sit the British Ability Scale (B.A.S.) Test, while in the 6^{th} sweep (2004) they take a basic skills examination.

The B.A.S. has long been established as a leading standardised test in the UK for assessing a child's cognitive ability and educational achievement across a wide age range. The version of the test taken by individuals in 1980 was organized into four sections, for a total of 120 questions: 1) word definition; 2) word similarities; 3) recall of digits; 4) matrices (where children have to complete some patterns drawing the appropriate picture in an empty square).

On the contrary, the basic skills test of 2004 aimed at assessing individuals' literacy and numeracy skills and was part of a bunch of initiatives carried out to understand and tackle the problem of poor basic skills in a substantial minority of the U.K. adult population. It was divided into two sections (a literacy and a numeracy one), for a total of 60 questions.

From what has been said, it is clear that the two tests have different aims and measure different abilities, therefore they are hardly comparable.

Our sample consists of 4368 individuals, among which 2055 are males and 2313 females.

As it can be seen from Table 3, in adulthood, weight problems are more severe among men: average BMI level, overweight and obesity rates are considerably greater for males. The data presented in the table could appear surprisingly high (more than 61% of the male population is overweight) but they are perfectly in line with those from "The Health Interview Surveys", carried out by Eurostat in 2008.

The situation is different when we look at the weight statistics in 1980, during cohort members' childhood: the average BMI level is higher for females (this is due to their physiological earlier growth) and the overweight and obesity rates are substantially equal between genders.

Table 3 – Weight conditions

	Mean	**Std. Dev.**	**Observations**
BMI 2004	25.77	4.75	4368
BMI 2004 males	26.64	4.34	2055
Overweight rate	61.65%		1267
Obesity rate	17.86%		367
BMI 2004 females	25.01	4.96	2313
Overweight rate	38.82%		898
Obesity rate	14.61%		338
BMI 1980	16.90	2.07	4368
BMI 1980 males	16.75	1.91	2055
Overweight rate	15.13%		311
Obesity rate	5.01%		103
BMI 1980 females	17.02	2.20	2313
Overweight rate	15.05%		348
Obesity rate	5.02%		116
Weight Trends			
Males			
Switch from non-overweight in 1980 to overweight in 2004	56.94%		993
Overweight in 1980 and in 2004	88.1%		274
Non-Overweight in 1980 and in 2004	43.06%		751
Overweight in 1980 but not in 2004	11.9%		37
Females			
Switch from non-overweight in 1980 to overweight in 2004	33.03%		649
Overweight in 1980 and in 2004	71.55%		249
Non-Overweight in 1980 and in 2004	66.97%		1316
Overweight in 1980 but not in 2004	28.45%		99

Looking at the weight evolution over time it can be noticed that almost 57% of males in normal weight condition when 10 years old switch to the overweight status at the age of 34, while only 33% of females change their weight category over time in this direction.

In addition, overweight male children are also more likely to become overweight adults: 88% of overweight children suffer from weight problems also in adulthood, against 71.5% of females. These different trends in the weight evolution across genders explain the gap in the adult overweight and obesity rates, starting from a situation of almost equality.

The British Ability Scales has long been established as a leading standardized test in the UK for assessing a child's cognitive ability and educational achievement across a wide age range. The test version sit by cohort members in 1980 consists of four sections: word definition (explain the meaning of some given words), verbal similarities (tell a word that is related to three words told by the examiner), recall of digit (remember a progressively increasing number of digits) and matrices (complete some patterns drawing the appropriate shape in an empty square), for a total of 120 questions.

The Basic Skill Test sit by cohort members when they are 34 years old is, instead, divided into two sections: a literacy part, made up of 37 questions and a numeracy one, composed by 23 questions.

The total score is given by the number of correct answers (there is no penalty for wrong answers). Hence, the test score is an integer number between zero and 60.

As it can be seen from Table 4, males performed, on average, slightly better than females in both the tests. What is of interest in our context is to compare test results across weight categories. Concerning the B.A.S score we can observe opposite trends in the two genders: the score increases with BMI among males, while it decreases among females.

In the basic skill test the trend is instead the same in both sexes: the score decreases as the weight increases. This inverse relationship holds not only for both genders but also for both the ages considered (10 and 34), with the only exception of overweight females children, that, when adult, performed slightly better than non-overweight peers. However, when weight problems are more severe, resulting in obesity, the trend is restored, with obese female children performing significantly worse than normal-weights.

Table 4 – Test Scores

	Mean	Std. Dev.	Observations
B.A.S. TEST, 1980			
Test Score	63.69	11.71	3699
Males Test Score	64.20	12.09	1718
Females Test Score	63.25	11.36	1981
Score per Weight Categories			
Males, 1980			
Test Score if Normal Weight	64.03	12.07	1459
Test Score if Overweight	65.18	12.17	259
Test Score if Obese	65.98	11.95	83
Difference in mean: Normal vs Overweight	-1.15		
Difference in mean: Normal vs Obese	-1.95		
Females, 1980			
Test Score if Normal Weight	63.40	11.49	1698
Test Score if Overweight	62.34	10.48	283
Test Score if Obese	62.23	11.40	92
Difference in mean: Normal vs Overweight	1.06		
Difference in mean: Normal vs Obese	1.17		
BASIC SKILL TEST, 2004			
Test Score	50.83	7.17	4368
Males Test Score	51.57	7.09	2055
Females Test Score	50.17	7.18	2313
Score per Weight Categories			
Males, 2004			
Test Score if Normal Weight	51.97	7.11	788
Test Score if Overweight	51.32	7.07	1267
Test Score if Obese	51.19	7.11	367
Difference in mean: Normal vs Overweight	0.65**		
Difference in mean: Normal vs Obese	0.78*		
Females, 2004			
Test Score if Normal Weight	50.81	6.85	1415
Test Score if Overweight	49.15	7.56	898
Test Score if Obese	48.86	7.70	388
Difference in mean: Normal vs Overweight	1.66***		
Difference in mean: Normal vs Obese	1.95***		
Males, 1980			
Test Score if Normal Weight	51.61	7.04	1744
Test Score if Overweight	51.33	7.41	311
Test Score if Obese	50.86	7.83	103
Difference in mean: Normal vs Overweight	0.28		
Difference in mean: Normal vs Obese	0.75		
Females, 1980			
Test Score if Normal Weight	50.15	7.21	1966
Test Score if Overweight	50.28	7.02	347
Test Score if Obese	49.49	8.10	116
Difference in mean: Normal vs Overweight	-0.13		
Difference in mean: Normal vs Obese	0.66		

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.001$

3.4 Empirical Models

3.4.1 OLS Estimator

Following the literature on the effects of Body Mass on individuals' outcomes, we assume that our regression of interest takes the form:

$$y_i = \alpha + \beta BMI_i + \gamma X_{1i} + \delta X_{2i} + \varepsilon_i \quad (1)$$

where y_i is the test score reported by individual i in either the B.A.S or the Basic Skills tests, BMI_i is the cohort member Body Mass Index and X_{1i} and X_{2i} are two vectors of control variables. The former includes individual-level and family-level observables, such as years of schooling, birth and living country, ethnic group, family income and parents' education, while the latter collects information about the family cultural environment and parental attitudes towards children schooling. The complete list of control variables is provided in Appendix 1.

The estimate of β will be an unbiased estimate of the effect of Body Mass on individuals' basic skills only if there are no unobservable characteristics correlated with both BMI and test score, that is $E(\varepsilon|BMI)=0$. If this identification assumption is violated, as it is the case in presence of endogeneity or unobserved heterogeneity, our OLS estimate of β will be biased.

We estimate equation (1) using each test scores as dependent variables and individuals' BMIs as regressors, in order to study the simultaneous and lagged effects of Body Mass.

Then, we move our attention from the continuous variable BMI to a dummy variable (OBY_i) indicating whether the CM suffers from weight problems, estimating the following equation:

$$y_i = \alpha + \beta OBY_i + \gamma X_{1i} + \delta X_{2i} + \varepsilon_i \quad (2)$$

Also in this case, for the Basic Skills test sit in 2004, we focus on the individuals' weight status at 10 and 34 years old.

Finally, in order to investigate the potential effects of weight gain from childhood to adulthood, we create some dummy variables indicating whether CMs have changed their weight classification, from 1980 to 2004, moving from a normal-weight condition to overweight.

In this case, the model to be estimated is:

$$y_i = \alpha + \beta D_i + \gamma X_{1i} + \delta X_{2i} + \varepsilon_i \quad (3)$$

where y_i is the outcome of the Basic Skills Test and D_i is a vector of dummy variables indicating whether the CM became overweight, slimmed down or stayed overweight. We use individuals who are normal weight both in 1980 and 2004 as the reference category.

Clearly, both the equations (2) and (3) are subject to the same endogenity issue that affects model (1).

3.4.2 IV Estimator

As pointed out before, the OLS estimates are unbiased only in the absence of endogeneity and unobserved heterogeneity issues. This is hardly the case in our context: reverse causality may take place since individuals' skills could affect their Body Mass, influencing their diets and lifestyle choices or creating psychological discomfort. As already mentioned, stress can alter eating habits inducing individuals to consume less (or more) food than needed.

The presence of unobservable characteristics, both at the individual and family level, could also bias our results.

A common method for addressing these problems is the use of instrumental variables. This requires finding at least one observable variable that provide exogenous variation in individuals' BMI but is uncorrelated with the outcome of interest except through BMI itself.

Following the literature (e.g. Cawley 2000 and 2004; Brunello and d'Hombres 2005) we choose as instruments for model (1) the BMI of a relative. In particular, we are the first ones in this kind of literature that can exploit information on both parents' body mass. This allows us to perform an over-identification test, which supports the validity of our choice.

Parental BMI is likely to satisfy the first requirement for IVs, since it is strongly correlated with that of their offspring: Comuzzie and Allison (1998) estimated that 40 to 70 percent of the variation in obesity-related phenotypes in humans is heritable, while, in the second chapter of this thesis, we have highlighted the strong positive association existing between parental and offspring's BMI and we have computed intergenerational elasticity using the same data (BCS70) of the current chapter.

Moreover, parents' body mass must be uncorrelated with unobservable determinants of cognitive skills, that is with the error term ε. As suggested by Sabia (2007), this assumption may be problematic if parental obesity is correlated with unobserved family-level environmental characteristics that can influence children cognitive ability. However, as highlighted by Cawley (2004), there is medical evidence[16] from studies using samples of adoptees suggesting that genetics rather than household environment is the most important determinant of body weight. This result supports the use of biologically related individuals' BMI as a credible instrument.

As we will see in the next section, all of the tests we have performed in order to assess the validity of our instruments give satisfactory results.

[16] See Stunkar et al. (1986), Grilo and Pogue-Geile (1991) and Vogler et al. (1995).

However, given the concerns of their potential correlation with household attitudes toward education, following Sabia (2007), we control for several measures of "family-level school sentiment" and parents' general propensity to "intellectual" activities, such as reading newspapers or books. These are the control variables we included in the vector X_{2i}.

Since in model (2) the endogenous regressor is a dummy variable, we can estimate the model parameters applying different estimation strategies.

We start with a standard IV approach, where, following the previous reasoning, the instruments will be two dummy variables telling whether parents are overweight/obese or not.

Then, in order to check the robustness of our results, we move in the setting of endogenous treatment models, considering the overweight/obesity condition as the treatment.

In our first specification we assume homogeneous treatment effects (Dummy Endogenous Model) and we estimate the effect of obesity applying both the Heckmann correction (or Heckmann two-steps) procedure and maximum likelihood.

Later, we allow for heterogeneous effects in an Endogenous Switching Model (or Roy Model) applying again the Heckmann two-step technique.

It is worth to notice that the two Heckit models we are considering rely on different assumptions. In the Dummy Endogenous Model the treatment effect is assumed to be homogeneous in the population, that is, the idiosyncratic gain is zero for every individual. In other words, the impact of the treatment does not vary with individuals' observable characteristics and the unobservable determinants of the outcome are the same with or without treatment.

In the Endogenous Switching Model we relax the strong homogeneous effect assumption allowing for heterogeneous treatment, that is, for individual-specific effects: the average treatment effect (ATE) is allowed to vary across individuals with different observable characteristics and to affect the probability of individuals to "choose" the treatment.

3.5 Results

3.5.1 OLS Estimates using the BMI as measure of weight

We start by analyzing the effect of BMI in 1980 on the B.A.S. score, first using the entire sample and then for males and females separately.

The results of the OLS estimates are reported in Table 5. The coefficients in column (1) are those from the regression of the test score on individuals' BMI only (univariate regression). The specification in column (2) includes the set of control variables contained in the X_{1i} vector, while in column (3) we control also for parental attitudes towards education.

In each of the model specifications, once our sets of controls are included, we find no significant association between children's BMI and their cognitive ability.

Different results are obtained when we look at the correlation between the score of Basic Skill Test taken in 2004 and the individuals' current values of BMI (Table 6).

Considering the whole sample, the OLS estimates are negative and significant in all of the model specifications: one unit increase in individuals' BMI is associated to a test score reduction of 0.088 points (out of 60) in the univariate regression and of about 0.05 points in the two other specifications.

However, when we distinguish between males and females, this association turns out to be statistically significant only for women, which suffer a test score reduction of about 0.07 points for each additional unit of BMI. One possible explanation for this difference between genders is that OLS estimates reflect both a (negative) causal and a (positive) spurious effect, the latter given by the effort and behaviors that obese individuals put to use during school and work activities in order to offset the negative consequences of their weight condition. Since women are likely to spend less time in the labor market, because of pregnancies and their greater involvement in children education, this may harm their skills learning. Conversely, men may have more opportunities to build their human capital during the work activity, thus counterbalancing the potential negative effect of obesity with superior experience and "learning by doing".

On the contrary, using lagged BMI levels, we don't find any significant relationship, neither in the whole sample nor separating by sex (Table 7).

Summing up, our OLS estimates show no evidence of correlation between BMI and cognitive ability during childhood, while deficiencies in literacy and numeracy skills are associated with increased BMI in adulthood but aren't influenced by past BMI levels.

This last result could appear surprising: we might expect that a high BMI in childhood is more detrimental since it affects individuals during the educational process, when human capital accumulation is taking place.

However, this finding can be justified noticing that the variance in the BMI distribution is much higher in adulthood than in childhood. Therefore, the variance of the OLS estimator will be lower when using 2004 BMI values, implying more precise estimates. Another possible explanation for the different relationship between skills and Body Mass at different ages is that the effect of weight is non-linear. Given the non-linearity concerns, we decide to move our attention from the continuous BMI variable to a dummy indicating whether individuals suffer from weight problems.

Table 5 – Correlation between BMI and the B.A.S. score

	B.A.S. Test Score 1980								
	FULL SAMPLE			MALES			FEMALES		
	(1)	(2)	(3)	(1)	(2)	(3)	(1)	(2)	(3)
BMI 1980	0.037	0.030	0.049	0.322**	0.238	0.230	-0.123	-0.112	-0.063
	(0.39)	(0.33)	(0.55)	(2.02)	(1.56)	(1.51)	(1.07)	(1.01)	(0.59)
Controls in X_{1i}		X	X		X	X		X	X
Controls in X_{2i}			X			X			X
Cons	63.07***	44.18***	43.85***	58.82***	39.97***	41.12***	65.35***	46.00***	41.95***
	(30.10)	(16.25)	(15.55)	(21.87)	(10.24)	(9.68)	(32.86)	(12.23)	(11.15)
N	3699	3675	3675	1718	1704	1704	1981	1971	1971

t statistics in parenthesis; * p < 0.10, ** p < 0.05, *** p < 0.01

Table 6 - Correlation between current BMI and the Basic Skill Test score

	Basic Skills Test Score 2004								
	FULL SAMPLE			MALES			FEMALES		
	(1)	(2)	(3)	(1)	(2)	(3)	(1)	(2)	(3)
BMI 2004	-0.088***	-0.051**	-0.046**	-0.066*	-0.011	-0.010	-0.151***	-0.079**	-0.074**
	(3.15)	(2.16)	(1.99)	(1.83)	(0.33)	(0.30)	(5.05)	(2.83)	(2.66)
Controls in X_{1i}		X	X		X	X		X	X
Controls in X_{2i}			X			X			X
Cons	53.09 ***	47.17***	44.79***	53.32***	44.05***	43.12***	53.94***	38.84***	34.18***
	(83.46)	(30.51)	(31.96)	(54.77)	(8.62)	(8.53)	(70.69)	(19.59)	(18.04)
N	4368	4340	4340	2055	2046	2046	2313	2301	2301

t statistics in parenthesis; * p < 0.10, ** p < 0.05, *** p < 0.01

Table 7 - Correlation between lagged BMI and the Basic Skill Test score

	Basic Skills Test Score 2004								
	FULL SAMPLE			MALES			FEMALES		
	(1)	(2)	(3)	(1)	(2)	(3)	(1)	(2)	(3)
BMI 1980	0.015	0.028	0.047	0.079	0.056	0.036	0.009	0.028	0.042
	(0.20)	(0.49)	(0.88)	(0.97)	(0.72)	(0.46)	(0.13)	(0.44)	(0.68)
Controls in X_{1i}		X	X		X	X		X	X
Controls in X_{2i}			X			X			X
Cons	50.58***	47.71***	42.62***	50.24***	42.88***	44.13***	50.01***	36.15***	34.38***
	(51.58)	(28.58)	(30.29)	(36.32)	(8.36)	(8.61)	(42.91)	(17.16)	(16.41)
N	4368	4350	4350	2055	2046	2046	2313	2304	2304

t statistics in parenthesis; * p < 0.10, ** p < 0.05, *** p < 0.01

3.5.2 OLS Estimates using the overweight dummy

According to the medical literature, adult individuals are classified as overweight if their BMI is greater or equal to 25. The classification into this category is slightly more complicated for individuals' below 18 years. In this case, it is necessary to distinguish between males and females and the assignment to a weight category is done by looking at the relative position in the sample weight distribution. In particular, a child is classified as overweight if his/her BMI belongs to the 85^{th} percentiles or higher.

When using the overweight dummies instead of BMI values our results don't change: no statistically significant association is found between weight excess in 1980 and neither the B.A.S. score taken in the same year (Table 8), nor the basic skills test score reported in 2004 (Table 9). On the other hand, as it can be seen from Table 10, being overweight in 2004 is strongly associated with a decrease in basic skills for females but not for males.

Since the findings obtained using indicators for being overweight are perfectly in line with those previously found employing the continuous variable BMI, the non-linearity concerns seem not relevant in our analysis.

Our estimates revealed a negative association between obesity and basic skills at age 34 but not at age 10. Therefore, it is interesting to investigate the issue also from a dynamic point of view, studying how individuals' weight evolution over time is associated with their test scores.

Looking at the summary statistics presented in Table 3 we can notice that overweight rates are much higher in adulthood than in childhood (about 50% vs. 15%). Data about weight transition over time confirm that many cohort members have moved from a normal weight status when 10 years old to the overweight condition at age 34. Therefore, we create dummy variables to identify the individuals who become overweight, those who remain overweight and those who slim down and we study whether weight transition affect their test scores[17](individuals that are normal-weight both in 1980 and 2004 are our reference category).

Since the starting age from which we measure changes in BMI category is childhood, we have to perform separate analysis for males and females. As it can be seen from Table 11, weight gain is strongly associated with a lower basic skill level at age 34 for females, but not for males, at least when we include our controls in the regression.

In the univariate regression, becoming overweight is associated with a test score reduction of about 0.6 points (out of 60) for males and 2 for females, while when including all our controls the reduction is of about 1.2 points for females and there is no statistically significant effect for males.

[17] Our reference category is given by individuals that are normal-weight in both 1980 and 2004.

Table 8 – Correlation between the probability of being overweight in 1980 and the B.A.S. score

	B.A.S. Test Score 1980					
	MALES			FEMALES		
	(1)	(2)	(3)	(1)	(2)	(3)
Overweight in 1980	1.15	1.16	1.06	-1.06	-0.84	-0.75
	(1.40)	(1.52)	(1.38)	(1.56)	(1.27)	(1.16)
Controls in X_{1i}		X	X		X	X
Controls in X_{2i}			X			X
Cons	64.03***	43.39***	44.45***	63.40***	44.34***	41.04***
	(202.65)	(13.18)	(12.14)	(227.29)	(13.70)	(12.50)
N	1718	1704	1704	1981	1971	1971

t statistics in parenthesis; * p < 0.10, ** p < 0.05, *** p < 0.01

Table 9 - Correlation between the probability of being overweight in 1980 and Basic Skills

	Basic Skills Test Score 2004					
	MALES			FEMALES		
	(1)	(2)	(3)	(1)	(2)	(3)
Overweight in 1980	-0.284	-0.154	-0.240	0.125	0.301	0.293
	(0.65)	(0.37)	(0.58)	(0.30)	(0.77)	(0.76)
Controls in X_{1i}		X	X		X	X
Controls in X_{2i}			X			X
Cons	51.61***	43.71***	44.66***	50.15***	36.55***	35.03***
	(303.78)	(8.74)	(8.94)	(309.52)	(20.05)	(19.26)
N	2055	2039	2039	2313	2301	2301

t statistics in parenthesis; * p < 0.10, ** p < 0.05, *** p < 0.01

Table 10 - Correlation between the probability of being overweight in 2004 and Basic Skills

	Basic Skills Test Score 2004								
	FULL SAMPLE			MALES			FEMALES		
	(1)	(2)	(3)	(1)	(2)	(3)	(1)	(2)	(3)
Overweight 2004	-0.81***	-0.64**	-0.59**	-0.66**	-0.26	-0.25	-1.66***	-1.01***	-0.93***
	(3.73)	(3.07)	(2.85)	(2.04)	(0.86)	(0.83)	(5.45)	(3.54)	(3.29)
Controls in X_{1i}		X	X		X	X		X	X
Controls in X_{2i}			X			X			X
Cons	51.23***	46.41***	46.69***	51.97***	44.02***	44.96***	50.81***	37.16***	35.61***
	(335.63)	(9.57)	(9.72)	(205.82)	(8.78)	(8.98)	(267.77)	(20.38)	(19.56)
N	4368	4350	4350	2055	2039	2039	2313	2301	2301

t statistics in parenthesis; * p < 0.10, ** p < 0.05, *** p < 0.01

On the opposite, even if their signs are in the expected direction, the coefficients of the dummies for staying overweight and slimming down from the overweight to the normal-weight status are never statistically significant.

Table 11 – The impact of weight gain from childhood to adulthood

	Basic Skills Test Score 2004					
	MALES			FEMALES		
	(1)	(2)	(3)	(1)	(2)	(3)
Get overweight	-0.61*	-0.21	-0.18	-1.99***	-1.29***	-1.19***
	(1.77)	(0.66)	(0.56)	(5.83)	(4.03)	(3.75)
Slim down	0.34	0.35	0.32	0.07	0.15	0.16
	(0.28)	(0.31)	(0.29)	(0.10)	(0.21)	(0.23)
Stay overweight	-0.76	-0.36	-0.44	-0.77	-0.24	-0.21
	(1.52)	(0.76)	(0.92)	(1.56)	(0.52)	(0.45)
Controls in X_{1i}		X	X		X	X
Controls in X_{2i}			X			X
Cons	51.95***	43.99***	44.91***	50.81***	37.15***	35.60***
	(200.78)	(8.77)	(8.96)	(258.39)	(20.37)	(19.56)
N	2055	2039	2039	2313	2301	2301

t statistics in parenthesis; * p < 0.10, ** p < 0.05, *** p < 0.01

3.5.3 IV Estimates using the BMI as measure of weight

Following the previous literature, we instrument individual BMI with that of biological family members. In particular, we can exploit information on both cohort members' parents, so that we can rely on two instruments.

Again, we start by focusing on the B.A.S. test. IV coefficients are always negative, significant and larger in size than OLS ones. In particular, in our preferred model specification (columns 3), they are 10% significant and about 5 times greater (in absolute value) than OLS ones for males and 1% significant and about 20 times greater for females (Table 12).

This is in line with the field literature: increases of comparable size are found, when instrumenting, by Sabia (2007), Brunello and d'Hombres (2005), Averett and Stiefel (2007) and Cawley (2004), at least for some, if not all, the population subgroups they considered.

According to our IV results, one unit increase in individuals' BMI causes an average test score reduction of about 1.10 points out of 120 for males and 1.38 for females.

One possible explanation for this rise in coefficients' size is that OLS estimates are biased upwards by the positive correlation between unobservables, such as motivation or perseverance, and the BMI: overweight and obese children compensate the potential negative effect of weight with

unobservable behaviours (such as the effort put in the learning process and the time devoted to studying) that improve their skills.

Table 12 - Effect of BMI on the B.A.S. score

	BAS Test Score 1980								
	FULL SAMPLE			MALES			FEMALES		
	(1)	(2)	(3)	(1)	(2)	(3)	(1)	(2)	(3)
BMI 1980	-2.70***	-1.33***	-1.25***	-2.55***	-1.13*	-1.10*	-2.75***	-1.48***	-1.38***
	(7.13)	(4.01)	(3.83)	(3.51)	(1.73)	(1.70)	(6.37)	(3.99)	(3.77)
Controls in X_{1i}		X	X		X	X		X	X
Controls in X_{2i}			X			X			X
Sargan Test p-value	0.380	0.400	0.220	0.510	0.284	0.452	0.064	0.023	0.011
Endogeneity Test p-value	0.000	0.000	0.000	0.000	0.028	0.030	0.000	0.000	0.000
Underid. Test p-value	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Cragg-Donald Wald F-stat.	151.51	147.78	146.81	49.40	45.53	45.21	100.82	100.96	98.84
R^2 first stage	0.076	0.094	0.095	0.055	0.076	0.079	0.093	0.108	0.114
Cons	109.26***	63.10***	62.06***	106.88***	59.42***	60.14***	109.93***	68.23***	63.44***
	(17.09)	(6.72)	(6.71)	(8.78)	(4.69)	(4.78)	(14.98)	(9.93)	(9.31)
N	3699	3675	3675	1718	1704	1704	1981	1971	1971

t statistics in parenthesis;
* p < 0.10, ** p < 0.05, *** p < 0.01

Similar results hold when we consider the Basic Skill Test. Using 2004 Body Mass as a regressor (Table 13), IV coefficients are negative, like OLS ones, but highly significant for both sexes and not just for females. Again, they also increase in magnitude. The rise is now larger for males than for females: it is of about 5.5 times for the former and 3.5 times for the latter. One unit increase in 2004 average BMI causes a reduction of 0.55 points out of 60 in the average males' score and of 0.27 points in the females' one.

When looking at the effect of lagged BMI (Table 14), we observe a change in coefficients' sign and significance with respect to OLS estimates[18]: the effect of 1980 Body Mass becomes negative and highly significant. The increase in size is in absolute value much greater than the one of current BMI. According to our IV estimates, a unitary increase in average Body Mass in 1980 leads to an average test score reduction of about 1.2 points for males and 0.65 for females.

Therefore, contrary to OLS results, it seems that high BMI levels are more penalizing when recorded during childhood, that is, in the meanwhile of the educational process. This is a reasonable

[18] The same change in coefficients' size can be found in Brunello and d'Hombres (2005) for the male population.

finding, since it is during the human capital accumulation process that obesity may influence more seriously skills acquisition.

Table 13 - Effect of current BMI on the Basic Skill Test score

	Basic Skills Test Score 2004								
	FULL SAMPLE			MALES			FEMALES		
	(1)	(2)	(3)	(1)	(2)	(3)	(1)	(2)	(3)
BMI 2004	-0.69***	-0.39***	-0.37***	-0.78***	-0.52***	-0.55***	-0.60***	-0.31***	-0.27***
	(8.38)	(5.12)	(4.93)	(5.06)	(3.47)	(3.63)	(6.65)	(3.56)	(3.22)
Controls in X_{1i}		X	X		X	X		X	X
Controls in X_{2i}			X			X			X
Sargan Test p-value	0.742	0.717	0.565	0.625	0.515	0.593	0.384	0.223	0.153
Endog. Test p-value	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Underid. Test p-value	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.005	0.011
F-Test.	211.12	200.18	198.92	71.59	61.27	60.784	156.83	139.95	138.27
R^2 first stage	0.088	0.131	0.133	0.065	0.082	0.084	0.120	0.132	0.136
Cons	68.70***	58.25***	58.05***	72.33***	59.97***	61.66***	65.25***	46.08***	43.31***
	(32.15)	(10.49)	(10.57)	(17.61)	(8.52)	(8.76)	(28.70)	(14.99)	(14.12)
N	4368	4340	4340	2055	2039	2039	2313	2301	2301

t statistics in parenthesis; * p < 0.10, ** p < 0.05, *** p < 0.01

Table 14 - Effect of lagged BMI on the Basic Skill Test score

	Basic Skills Test Score 2004								
	FULL SAMPLE			MALES			FEMALES		
	(1)	(2)	(3)	(1)	(2)	(3)	(1)	(2)	(3)
BMI 1980	-1.71***	-0.93***	-0.88***	-1.84***	-1.18***	-1.24***	-1.57***	-0.73***	-0.65***
	(8.04)	(5.00)	(4.81)	(4.89)	(3.44)	(3.61)	(6.13)	(3.37)	(3.02)
Controls in X_{1i}		X	X		X	X		X	X
Controls in X_{2i}			X			X			X
Sargan Test (p-value)	0.475	0.500	0.370	0.571	0.500	0.594	0.151	0.129	0.090
Endog. Test (p-value)	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Underid. Test (p-value)	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
F-Test	177.64	175.31	174.97	65.62	61.74	61.17	109.86	111.27	109.32
R^2 first stage	0.075	0.095	0.097	0.060	0.082	0.086	0.087	0.107	0.111
Cons	79.82***	58.94***	58.63***	82.41***	61.01***	62.89***	76.85***	49.86***	46.54***
	(22.12)	(10.31)	(10.39)	(13.06)	(8.37)	(8.61)	(17.64)	(11.98)	(11.30)
N	4368	4340	4340	2055	2039	2039	2313	2301	2301

t statistics in parenthesis; * p < 0.10, ** p < 0.05, *** p < 0.01

The tests performed to check the goodness of our instruments are almost always satisfied[19]: the high p-value of the Sargan over-identification test supports their validity (there is no correlation with the error term), while both the low p-value of the Anderson under-identification test and the high Cragg-Donald Wald F-statistic suggest that excluded restrictions are relevant, that is correlated with individual BMI, whose endogeneity is confirmed by the zero p-value of the endogeneity test performed.

Even if the instruments are strong predictors of body weight and satisfy the Sargan over-identification test, the quite low first-stage R^2 values raise some doubts about the presence of a weak instrument problem, resulting in size distortion.

3.5.4 IV Estimates using the overweight dummy

When we move attention to the effect of being overweight, our results are in line with those we previously got using BMI. The IV coefficients for the overweight dummy are summarized in Table 15, while the full set of results, which include the estimates obtained applying the Heckman control function approach and the tests for the validity of our instruments are left in Appendix 3.

Looking at the Basic Skills Test and using information about the weight status, estimates accounting for endogeneity are, in the full sample, about 8-9 times greater in absolute value with respect to OLS ones: being overweight or obese reduces (on average) the test score by about 5 points (out of 60). Again, a possible explanation for the change in coefficients' size when we allow for endogeneity is that individuals suffering from weight problems compensate the negative effect of weight with unobservable characteristics and behaviours, like the effort choice, that bias OLS estimate downwards.

It is important to notice that all of the estimation strategies we used gave very similar results, suggesting evidence of a significant negative relation between current BMI and basic skills (Table A, Appendix 2).

However, when distinguishing between males and females the outcomes of the different procedures are less homogeneous: the test score reduction ranges from about 3.9 to 5.8 points for males and from 4 to 5.6 points for females (Table B, Appendix 2).

In Models (4) the coefficient associated to the Heckman correction term (*lambda*) is significant at 1% and positive, meaning that endogeneity concerns were justified and positive self-selection into treatment takes place. In other words, there is a positive correlation between unobservable

[19] The only exception is given by the Sargan over-identification test in the female sub-sample that, in some cases, failed.

characteristics, included in the error term, and individuals' BMI: overweight and obese people seem to have on average better unobserved features than non-obese peers.

Similar results hold when we study the effect of weight excess in 1980 on the Basic Skills Test, but in this case the negative impact of over-weightiness is even greater and heterogeneous across the different estimation techniques: it ranges from about 6.1 to 9.6 for males and from 2.7 to 9.5 for females (Table C, Appendix 2).

Finally, looking at the B.A.S. test, we can notice that being overweight has a strong negative effect only on females' score, while it seems not to affect males' results (only using MLE the coefficient of the dummy indicating weight excess is significant at 10% and negative, as it can be seen from Table D, Appendix 2).

Table 15 – Summary of IV estimates

	Basic Skills Test Score 2004					
	FULL SAMPLE		MALES		FEMALES	
	OLS	IV	OLS	IV	OLS	IV
Overweight/Oby 2004	-0.59**	-5.09***	-0.25	-6.30***	-0.93***	-4.37***
	(2.85)	(4.64)	(0.83)	(3.24)	(3.29)	(3.34)
Controls in X_{1i}	X	X	X	X	X	X
Controls in X_{2i}	X	X	X	X	X	X
Cons	46.69***	45.21***	44.96***	44.41***	35.61***	38.74***
	(9.72)	(15.76)	(8.98)	(11.70)	(19.56)	(8.77)
Overweight/Oby 1980			-0.240	-8.71***	0.293	-6.68***
			(0.58)	(2.93)	(0.76)	(3.13)
Controls in X_{1i}			X	X	X	X
Controls in X_{2i}			X	X	X	X
Cons			44.66***	39.02***	35.03***	36.93***
			(8.94)	(11.60)	(19.26)	(8.27)
N	4350	4350	2046	2046	2304	2304

Table 15 -continued

	B.A.S. Test			
	MALES		FEMALES	
	OLS	IV	OLS	IV
Overweight/Obesity in 1980	1.06	-4.18	-0.75	-15.27***
	(1.38)	(0.75)	(1.16)	(4.19)
Controls in X_{1i}	X	X	X	X
Controls in X_{2i}	X	X	X	X
Cons	44.45***	38.77***	41.04***	48.51***
	(12.14)	(6.99)	(12.50)	(6.50)
N	1710	1710	1974	1974

t statistics in parenthesis;
* p < 0.10, ** p <0.05, *** p < 0.01

3.6 Conclusions

In this chapter we studied the relationship between Body Mass and two measures of cognitive and educational achievements: the British Ability Scale test, taken when individuals are 10 years old, and a literacy and numeracy test, sit at the age of 34.

The issue is of great relevance since cognitive ability and educational outcomes are important determinants of individuals' productivity and wages. Economists have indeed studied the link between obesity and economic outcomes like employment probability, work absenteeism and wages finding that weight excess has a negative effect on them. However, they have not clarified the potential channels that can explain this relationship.

In our analysis we used as a sample a cohort of British individuals for which we have information about BMI in the years when the two tests are undertaken. In this way, for the second test, we are able to study both the current and lagged effects of Body Mass.

We started by analyzing the association between weight and tests' outcome and then we focused on the causal link between the two, following an instrumental variable approach.

According to our OLS estimates, a negative association exists between current BMI and the basic skills test score sit in 2004. However, when we distinguished between males and females, such relationship turned out to be significant only for the latters. In our preferred specification, one unit increase in females average BMI was associated to a test score reduction of about 0.07 points out of 60 (in the whole sample the reduction was of about 0.05 points).

On the contrary, we didn't find any significant association with lagged Body Mass and between the B.A.S. score obtained in 1980 and then BMI levels.

The subsequent OLS analysis carried out using weight categories instead of the continuous variable BMI confirmed all these results. Being overweight when sitting the basic skills test is associated to a decrease of about 0.9 points in females average test score (0.6 points in the overall sample).

However, our OLS results are likely to suffer from endogeneity and unobserved heterogeneity problems. In order to understand whether the effect of obesity is causal, we decided to use the instrumental variable estimation method. Following Cawley (2000, 2004), Brunello and d'Hombres (2005), Sabia (2007) and Averett and Stifel (2007) we chose relatives' BMI as an IV. Contrary to previous studies, our dataset provides information on both parents' BMI and therefore we have an additional element to check our instruments' validity by means of an over-identification test.

Our IV estimates revealed the existence of a negative and statistically significant causal effect of current BMI on the outcome of both the B.A.S. and the Basic Skills tests. The effect was significant also for males (even if only at 10% in the B.A.S. test) and larger in absolute value with respect to OLS. A raise in coefficients' size was found, when instrumenting, by most of the above mentioned studies, at least for some the population subgroups considered. This increase can be explained by the presence of a positive correlation between unobservables, like motivation or perseverance, and the BMI, that makes OLS coefficients downward biased. Intuitively, overweight and obese individuals may compensate the negative effect of weight with characteristics, attitudes and behaviours that are unobservable to us and that can improve their skills. For example they might put more effort in their activities or devote more time to studying, maybe because they are less involved in social and sport activities.

As for OLS, the negative effect of weight is confirmed by the analysis carried out using a dummy that identifies overweight and obese individuals instead of using the continuous variable BMI.

Finally, exploiting the availability of BMI data at different ages, we studied the effect of weight gain over time, finding that moving from a normal-weight condition in childhood to overweightness in adulthood is associated with lower basic skills levels. Of course, in this case, reverse causality may still be an issue.

3.7 References

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3.8 Appendices

Annex 1 – List of Control Variables

The observables included in vector X_{1i} are:

- Years of schooling of the cohort member (CM)
- Sex of the CM
- Birth country of the CM
- Living country of the CM
- Ethnic group of the CM
- Years of schooling of CM's mother
- Years of schooling of CM's father
- Family income when CM was 10 years old
- Number of household members when CM was 10 years old
- Birth-weight of CM
- Whether the CM was breast-fed

The variables included in vector X_{2i} are dummies indicating whether, in 1986:

- newspapers are usually available at home
- magazines or Sunday papers are usually available at home
- mother reads books or magazines
- father reads books or magazines
- parents help in homework
- parents have no expectation about school
- parents visit school
- mother is unable to help in homework
- father is unable to help in homework
- both parents unable to help in homework
- parents impose curfew in schooldays

Annex 2 – Estimates accounting for endogeneity: full set of results

Table A - Effect on the Basic Skill Test score of being overweight in 2004 (Full Sample)

	Basic Skills Test Score 2004				
			Heckit Models		
	OLS	IV	DEM 2-steps	DEM MLE	Roy Model 2-steps
Overweight/Obesity in 2004	-0.586**	-5.09***	-4.98***	-4.66***	-4.89***
	(2.85)	(4.64)	(4.61)	(4.92)	
Controls in X_{1i}	X	X	X	X	X
Controls in X_{2i}	X	X	X	X	X
Sargan Overid. Test p-value		0.993			
Endogeneity Test p-value		0.000			
Underid. Test p-value		0.000			
F-Test.		87.007			
Λ			2.82***	2.62***	
			(4.18)	(4.37)	
R^2 first stage		0.105			
Cons	46.69***	45.21***	45.08***	44.69***	
	(9.72)	(15.76)	(15.79)	(16.14)	
N	4350	4350	4350	4350	4350

t statistics in parenthesis;
* p < 0.10, ** p < 0.05, *** p < 0.01

Table B - Effect on the Basic Skill Test score of being overweight in 2004 (Males vs Females)

	Basic Skills Test Score 2004									
	MALES					FEMALES				
			Heckit Models					Heckit Models		
	OLS	IV	DEM 2-steps	DEM MLE	Roy Model 2-steps	OLS	IV	DEM 2-steps	DEM MLE	Roy Model 2-steps
Overweight/Obesity in 2004	-0.25	-6.30***	-3.89***	-3.89***	-5.46***	-0.93***	-4.37***	-4.22***	-5.63***	-4.56***
	(0.83)	(3.24)	(3.09)	(3.18)		(3.29)	(3.34)	(3.26)	(4.05)	
Controls in X_{1i}	X	X	X	X	X	X	X	X	X	X
Controls in X_{2i}	X	X	X	X	X	X	X	X	X	X
Sargan Overid. Test p-value		0.391					0.428			
Endogeneity Test p-value		0.001					0.005			
Underid. Test p-value		0.000					0.000			
F-Test.		30.02					57.93			
Λ			3.57***	2.35***				2.11***	3.01***	
			(3.05)	(3.61)				(2.61)	(2.70)	
R^2 first stage		0.051					0.074			
Cons	44.96***	44.41***	43.96***	42.20***		35.61***	38.74***	38.58***	39.97***	
	(8.98)	(11.70)	(11.78)	(12.56)		(19.56)	(8.77)	(8.76)	(8.79)	
N	2046	2046	2046	2046	2046	2304	2304	2304	2304	2304

t statistics in parenthesis;
* p < 0.10, ** p < 0.05, *** p < 0.01

Table C - Effect on the Basic Skill Test score of being overweight in 1980 (Males vs Females)

	Basic Skills Test Score 2004									
	MALES					FEMALES				
			Heckit Models					Heckit Models		
	OLS	IV	DEM 2-steps	DEM MLE	Roy Model 2-steps	OLS	IV	DEM 2-steps	DEM MLE	Roy Model 2-steps
Overweight/Obesity in 1980	-0.240	-8.71***	-6.18**	-9.62***	-8.00***	0.293	-6.68***	-4.27**	-9.53***	-2.71***
	(0.58)	(2.93)	(2.48)	(16.77)		(0.76)	(3.13)	(2.33)	(17.70)	
Controls in X_{1i}	X	X	X	X	X	X	X	X	X	X
Controls in X_{2i}	X	X	X	X	X	X	X	X	X	X
Sargan Overid. Test p-value		0.112					0.290			
Endogeneity Test p-value		0.002					0.000			
Underid. Test p-value		0.000					0.000			
F-Test.		23.71					42.90			
Λ			3.35**	5.41**				2.62**	5.69**	
			(2.42)	(2.44)				(2.56)	(2.36)	
R^2 first stage		0.041					0.073			
Cons	44.66***	39.02***	38.91***	39.06***		35.03***	36.93***	36.03***	37.99***	
	(8.94)	(11.60)	(12.11)	(11.39)		(19.26)	(8.27)	(8.41)	(8.15)	
N	2046	2046	2046	2046	2046	2304	2304	2304	2304	2304

t-statistics in parenthesis;
* p < 0.10, ** p < 0.05, *** p < 0.01

Table D - Effect on the B.A.S. score of being overweight in 1980

	B.A.S. Test Score 1980									
	MALES					FEMALES				
			Heckit Models					Heckit Models		
	OLS	IV	DEM 2-steps	DEM MLE	Roy Model 2-steps	OLS	IV	DEM 2-steps	DEM MLE	Roy Model 2-steps
Overweight/Obesity in 1980	1.06	-4.18	-6.46	-6.31*	-8.81	-0.75	-15.27***	-11.04***	-11.18***	-9.61***
	(1.38)	(0.75)	(1.31)	(1.85)		(1.16)	(4.19)	(3.60)	(6.23)	
Controls in X_{1i}	X	X	X	X	X	X	X	X	X	X
Controls in X_{2i}	X	X	X	X	X	X	X	X	X	X
Sargan Overid. Test p-value		0.101					0.029			
Endogeneity Test p-value		0.332					0.000			
Underid. Test p-value		0.000					0.000			
F-Test.		15.87					41.87			
Λ			4.22	4.14				5.91***	5.60***	
			(1.55)	(1.49)				(3.48)	(3.77)	
R^2 first stage		0.036					0.059			
Cons	44.45***	38.77***	38.76***	38.76***		41.04***	48.51***	47.04***	47.09***	
	(12.14)	(6.99)	(6.88)	(6.89)		(12.50)	(6.50)	(6.65)	(6.70)	
N	1710	1710	1710	1710	1710	1974	1974	1974	1974	1974

t -statistics in parenthesis;
* p < 0.10, ** p < 0.05, *** p < 0.01

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