Gene and Economic Interaction: The Case of Obesity

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Abstract

We develop a simple model of health and human capital formation that takes into account the dynamic interaction between genetic inheritance and parental choices of investment in children. Differences in the genetic makeup of children induce variations in the shadow cost of inputs in the production function of human capital, and in equilibrium this is mirrored by changes in the returns to investment. We take this model to the data using the Avon Longitudinal Study of Parents and Children and we focus on a particular facet of health: obesity. Different forms of investment are considered as inputs, notably physical exercise and dietary intake, and we evaluate their interaction with various genes which has been associated to increases in Body Mass Index in Genome-Wide Association Studies. Once we control for the environmental pathways that connect the genetic endowment to obesity, we find that allelic variations are not significant anymore in explaining Body Mass Index (BMI), while Gene-Environment interaction (GxE) plays a fundamental role. We also find that children who tend to exercise are less affected by the presence of obesity-related alleles, while those who eat a less healthy diet are more susceptible to this genetic liability. These results are consistent with the findings in molecular biology showing that certain genes are associated with the hypothalamic regulation of food intake, and shed light on the interdependence between genes and economic choices regarding parental investment and human capital formation.

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"Nature makes the boy toward, nurture sees him forward" *Mulcaster (1582)*

1 Introduction

The goal of this paper is to understand how economic choices of investment in the child's capabilities build on and interact with her genetic endowment in order to enable the full flourishing of her innate ability. Only through a sequence of targeted choices and investments aimed at developing a particular talent can an individual fully excel later in life.

This idea is nested within the debate on "nature vs nurture", which has long been discussed both in social and biological sciences. It was initially framed as an antagonistic relationship by Francis Galton (1874), who believed genetic inheritance to play the stronger role. "When nature and nurture compete for supremacy on equal terms in the sense to be explained, the former proves to be the stronger." (Galton, 1874, p. 12) On the other side in The Wealth of Nations Adam Smith (1776) argues the opposite, suggesting that observed differences are due to specialization and division of labor, rather than arising from natural talents.

> "The difference of natural talents in different men is, in reality, much less than we are aware of; and the very different genius which appears to distinguish men of different professions, when grown up to maturity, is not upon many occasions so much the cause, as the effect of division of labour. The difference between the most dissimilar characters, between a philosopher and a common street porter, for example, seems to arise not so much from nature, as from habit, custom, and education. When they come into the world, and for the first six or eight years of their existence, they were, perhaps, very much alike, and neither their parents nor play-fellows could perceive any remarkable difference." (Smith, 1776, pp.28-29)

Since then, a wide literature in behavioral genetics has tried to parse out the relative importance of these two components in determining any particular human trait, usually comparing identical and non-identical twins. A very coarse additive model that assigns unique variances to both genes and environments is imposed in order to determine the precise percentage of a particular trait that is 'heritable'[1]. For example, it has been estimated that roughly 80% of the variation in human height can be attributed to genetic inheritance (Yang et al. (2010)), that 28 to 85% of IQ is heritable (van der Sluis et al. (2008)), or that 16-85% of Body Mass Index is due to genes (Yang et al. (2007)),

[1]See (**?**, ch. 5) for a textbook description of the main models. A critique of the main assumptions of the these analysis has been provided by Goldberger (1976, 1979) and Manski (2011). A bivariate extension than joins research from social and biological sciences has been proposed in Kohler et al. (2011), who discuss at length the necessary identifying conditions.

while the rest is due to environment . In a horse race between the two, they tried to pick a winner.

We argue that such an antagonistic relationship is ill-posed and obsolete, and should be relinquished in favor of a more systemic view that considers the dynamic interaction between the genetic endowment of an individual, and the social, biological, and economic environment in which she grows and develops. This perspective conforms with the original idea of Richard Mulcaster, who first spoke of nature and nurture in more harmonious terms, stressing their collaborative effects; this view has recursively been put forth by other scholars, like Anastasi (1958), and more recently by Rutter (2006) and Heckman (2007), and it is deeply rooted in the evidence accumulated in molecular biology. Recent technological advances in the mapping of the genome allowed researchers to connect various human traits to specific genetic markers; through appropriate statistical models and using Genome-Wide Association Studies (GWAS), they contributed to our understanding of the genetic underpinning of human behaviors and characteristics. At the same time, the epigenetic work of Meany, Syzf and colleagues (Meaney and Szyf (2005); Meaney (2010); Szyf and Bick (2013)) has shown how the genetic endowment of an individual actively interacts and is shaped by the surrounding environment through the process of gene expression and DNA methylation, validating the claim that the environment gets under your skin. These strands of research combined gave a strong biological foundation for a better understanding of behavioral genetics and the development of studies regarding Gene-Environment interaction (GxE) and Gene-Environment correlation (rGE)[2]. This could explain why, for example, we have witnesses a dramatic increase in obesity rates in the recent decades, even though obesity has been estimated to be highly heritable, and the genetic pool of the population has not changed.

We contribute to the debate by introducing a framework for understanding how families shape the environment and make decisions on how to invest in the capabilities of children, so as to develop their full genetic potential while facing limits on the time and the resources available.

In order to achieve this goal, we develop a simple model of health and human capital formation that takes into account the dynamic interaction between genetic inheritance, and family choices of investment. The foundational conjecture is to consider the genetic code of an individual as delimiting the *possibility set* of every agent. The DNA is therefore modeled as a biological shifter of the individual cost of investments.In other words, the genetic endowment of an individual delineates the set of achievable combination of inputs (investments) and outputs (human capital) that an agent can attain[3]; therefore a particular genetic makeup that has been related to a certain trait, such as

[2]These terms were initially introduced by Plomin et al. (1977)

[3](Mas-Colell et al., 1995, chap 5b) define the production set as "The set of all production vectors that constitute feasible plans for the firm. [...] The production set is taken as a primitive datum of the theory". In this case the firm is the family, and the production vector is composed of investments and human capital.

cognitive ability[4], would entail a larger production set available to the individual and better chances to attain a higher cognitive standing; however the actual achievement of such ability would depend on the sequence of economic choices and family investments undertook in order to develop that particular trait.

This puts some structure on how the genetic potential of an individual interacts with the budget constraint faced by the family when making decisions about how to invest in the child, and generates some predictions that can be tested by the data.

Considering various types of investments, we evaluate how differences in the genetic makeup of the child induce variations in the shadow cost of inputs, and in equilibrium this is mirrored by changes in the returns to investments and the optimal allocation of resources within the family.

Drawing a connection with the household production model developed by Becker, the genetic endowment would be considered as an 'environmental variable' which influences the efficiency of the household production function but not its preferences; as explained in (Becker, 2007, p.48), such variables "reduce the cost of producing commodities, and thus would expand opportunities, even if the full income were not affected".

We test the prediction of the model using the Avon Longitudinal Study of Parents And Children (ALSPAC), a very rich epidemiological dataset that followed prospectively a birth cohort recruited in Avon, UK, in 1991/1992.

We focus our attention on a particular facet of health capital: obesity. A trait that has been reliably and consistently measured over time, it has become of prime importance due to the recent rise in the obesity epidemics, especially in children. Ogden (2002) show how obesity rates of children aged 2 to 5 doubled from the early 1970s to 2000, going from a prevalence of 5% to 10.4%, and tripled for children aged 6 to 19, from 5% to 15%. Furthermore, Cawley (2010) shows how both direct and indirect costs of obesity are considerable: childhood obesity costs $14.3 billion a year due to prescription drugs, emergency room, inpatient and outpatient costs; the figure for adults is 10 times greater, with an estimated $147 billion spent in obesity related illnesses. The indirect costs of obesity range from delayed skills acquisition, to lower wages, job absenteeism, and lower productivity.

Finally, we chose it because it has a strong biological underpinning that has been connected to various genetic markers, but at the same time the level of fitness of our body can be affected by both social and economic choices, such as diet and exercise.

To guide the empirical analysis we focus on precise measures of genes and environment[5]. We leverage the findings in molecular biology showing how minor variants

[4]See Davies et al. (2011); Butcher et al. (2006) for a discussion of the genes that have been associated with IQ and cognitive functioning

[5]We follow the suggestions of Moffitt et al. (2005) and Purcell (2002) who point out how considering genes and environment as latent variables still potentially allows for detection of GxE, but suffers from low power, is sensitive to non-normality of the trait, and most importantly does not shed any light on the underlying processes. On the other side, using well defined measures of gene and environment is more sensible both from a statistical perspective - it provides the most power for detecting GxE - as

in the FTO gene (rs9939609 Single Nucleotide Polymorphism) are associated with hypothalamic regulation of energy intake but not energy expenditure. In other terms, this particular gene has been linked to biological mechanisms in the brain that determine the control of appetite and feeding impulses, but it has not been associated with differences in metabolic rates. Therefore the presence of minor-allele variants of this gene increases the (psychological) cost of following a strict diet, but leaves the incentives to engage in physical activity unaltered.

This intuition is corroborated by our results: we find strong evidence in support of the interaction between the FTO gene and the quantity of food that children eat; furthermore, we also find evidence of interplay between genetic endowment and physical activity, albeit to a minor extent. While the gene is connected, on average, to higher levels of Body-Mass-Index (BMI), controlling for the environmental pathways that connect FTO to obesity, we find that allelic variations are not significant anymore in explaining adiposity. Therefore, the presence of a different genetic makeup does not predestine the child to be overweight, but rather her level of health is conditional on the family choices in the realm of diet and exercise. Furthermore I find some preliminary evidence on the substitution between these two investment decisions: children who are endowed with the minor variant of the FTO gene tend to have a higher food intake on average, but the effect on obesity rates is mitigated by their exercise choices.

2 The Model

We consider a simple model of health and human capital formation where the family have to decide between consumption and investment. We analyze a simple static model to derive the basic predictions and build some intuition.

The family solves the following utility maximization problem, subject to the time and income constraint and the production function of health and investment:

$$\begin{aligned} \max_{x_e, x_d, \{\tau_k\}_{k=e}^{d}, c} \quad & U(c, \tau_l, H) \qquad (1) \\ s.t. \quad & \\ \Omega &= \tau_l + \tau_e + \tau_d \\ Y &= p_c c + p_d x_d + p_e x_e \\ H &= f\left(I_d, I_e; g\right) \\ I_e &= I_e\left(x_e, \tau_e; g\right) \\ I_d &= I_d\left(x_d, \tau_d; g\right) \end{aligned}$$

well as from the analytical perspective, since it sheds light on the biological and causal links connecting endowment, choices, and the final outcome

where H is the stock of health (or human capital), which arises according to the production function $f(.)$, a function of different types of investments - in this case we consider two potential investment, exercise I_e and diet I_d. Ω is the total amount of time and energy that the person can choose to allocate to leisure τ_l, exercise τ_e, and effort spent following a diet τ_d; Y is income, which can be allocated to either consumption c or investments x, according to their relative prices p_k. Investments are a function of the time τ and the goods x devoted to them, as well as the genetic endowment g of the individual.

This is the fundamental assumption of our model: genes enter not only the production function of human capital, but also the investment functions: a different genetic makeup will induce variations in the rate of conversion of effort and goods into investments, and not only the substitutability between and effectiveness of different inputs into the production function of human capital. Therefore having a particular genetic endowment will impact the incentives that people face when making investment decisions, shifting their cost functions.

A very good example of this come from the analysis of a particular gene that has been associated to obesity, FTO. Various studies have shown how this gene is associated to obesity through the regulation of hunger and feeding patterns, and not energy expenditure[6]. For example Fredriksson et al. (2008) find that "detailed in situ hybridization analysis in the mouse brain showed abundant expression in feeding-related nuclei of the brainstem and hypothalamus, such as the nucleus of the solitary tract, area postrema, and arcuate, paraventricular, and supraoptic nuclei as well as in the bed nucleus of the stria terminalis. [...] The FTO was significantly up-regulated (41%) in the hypothalamus of rats after 48-h food deprivation." They conclude that "These results are consistent with the hypothesis that FTO could participate in the central control of energy homeostasis." Similarly, Olszewski et al. (2009) anlyze the level of gene expression in the brain of mice experimentally deprived of food and find that "FTO mRNA is present mainly in sites related to hunger/satiation control; changes in hypothalamic FTO expression are associated with cues related to energy intake rather than feeding reward. In line with that, neurons involved in feeding termination express FTO". Cecil et al. (2008) analyze 2,726 Scottish children, 4 to 10 years of age, and find that the "A allele was associated with increased energy intake ($P = 0.006$) independently of body weight", but it had no visible effect on reting energy expenditure and metabolism. Using our same dataset, Timpson et al. (2008) found the same effect of the A-allele of the FTO gene on increased total energy and total fat intake, conditional on BMI.

Similar biological connections have been found for other genes, such as MC4R, BDNF, SH2B1[7].

[6]See also Speakman et al. (2008); Fawcett and Barroso (2010); Wardle et al. (2008); Yeo and O'Rahilly (2012); Timpson et al. (2008); Cecil et al. (2008).

[7]See Huszar et al. (1997); Govaerts et al. (2005); Qi et al. (2008); Valette et al. (2012) for mice-knock-out models as well as human evidence of the relation between the melanocortin-4 receptor (MC4R) and excessive feeding (hyperphagia), high levels of insulin and blood sugar (hyperinsulinemia

In terms of our model, this means that being a carrier of the FTO-A alleleincreases the cost of following a strict diet, $\frac{\partial I_d(x_d,\tau_d,g)}{\partial g_{FTO}} < 0$, but has no effect on the exercise function, so that $\frac{\partial I_e(x_e,\tau_e,g)}{\partial g_{FTO}} = 0$.

The first order condition of the model, as detailed in appendix A, are:

$$\frac{\frac{\partial f(I_d,I_e,g)}{\partial I_e(x_e,\tau_e,g)}\frac{\partial I_e(x_e,\tau_e,g)}{\partial x_e}}{p_e} = \frac{\frac{\partial f(I_d,I_e,g)}{\partial I_d(x_d,\tau_d,g)}\frac{\partial I_d(x_d,\tau_d,g)}{\partial x_d}}{p_d}$$

Or

$$\frac{\frac{\partial f(I_d,I_e,g)}{\partial I_e(x_e,\tau_e,g)}\frac{\partial I_e(x_e,\tau_e,g)}{\partial x_e}}{\frac{\partial f(I_d,I_e,g)}{\partial I_d(x_d,\tau_d,g)}\frac{\partial I_d(x_d,\tau_d,g)}{\partial x_d}} = \frac{p_e}{p_d}$$

Rearranging the terms we obtain:

$$\frac{\frac{\partial f(I_d,I_e,g)}{\partial I_e(x_e,\tau_e,g)}}{\frac{\partial f(I_d,I_e,g)}{\partial I_d(x_d,\tau_d,g)}} = \frac{p_e/\frac{\partial I_e(x_e,\tau_e,g)}{\partial x_e}}{p_d/\frac{\partial I_d(x_d,\tau_d,g)}{\partial x_d}}$$

$$\frac{f'_{I_e}}{f'_d} = \frac{p_e/I'_e}{p_d/I'_d} = \frac{p'_e}{p'_d} \tag{2}$$

Where $f'_{I_k} = \frac{\partial f(I_d,I_e,g)}{\partial I_k(x_k,\tau_k,g)}$ is the marginal productivity of investment k, while $p_k\prime = p_k/I'_k$ represents its shadow cost.

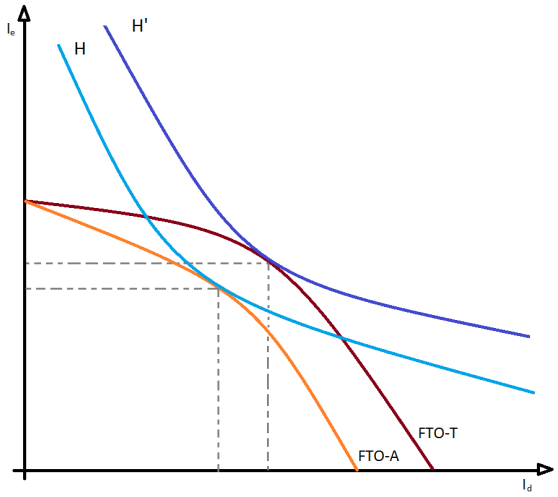
Since the genetic endowment of the agents enter the investment functions $I_e(.)$ and $I_d(.)$, by changing the genes we have a variation in the shadow price of inputs p'_k and, therefore, a change in the returns to investment. In the example of the FTO gene we can say that having at least one A allele, instead of a T allele, increases the cost of diet, $p'_d(A_{FTO}) > p'_d(T_{FTO})$, but has no effect on the price of exercise, $p'_e(A_{FTO}) = p'_e(T_{FTO})$. This will reflect in a shift in the budget set and, consequently, in a change of optimal allocation of **both** diet and exercise, as shown in Figure 1.

Therefore the predictions of the model are that a change in the genetic endowment of the child (say being born with an A allele instead of a T allele in the FTO rs9939609 gene-locus) will lead to a lower level of diet and, consequently, to a lower level of health (higher BMI). The effect on the optimal level of activity are not so straightforward: the restriction in the budget set will lead to a reduction in the optimal activity level; on the other side, the increased price of the other input might lead to a substitution effect toward exercise, which is now a relatively cheaper investment choice. Indeed, the higher is the use of the cheaper input I_e, the lower will be the effect of the FTO gene on

and hyperglycemia), and increase in food consumption; Gray et al. (2006); Unger et al. (2007) highlight the links between inhibition of food intake, energy homeostasis and the expression of brain-derived neurotrophic factor (BDNF) in the hypothalamus; Bochukova et al. (2010); Li et al. (2007); Ren et al. (2007) explain the relation between leptin, the SH2B1 gene, and eating and obesity. Finally Beckers et al. (2009) overviews the literature on the genetic basis of the leptin-melanocortin pathway to obesity

the observed level of health and BMI. Take an extreme example of a sportsman, who eats as much as he sees fit whenever he is hungry: the effort spent on diet is minimal, so that $I_d^* \approx 0$, but the amount of physical activity is very high; in this case, a difference in the genetic endowment would be barely noticeable on his obesity level.

Figure 1: Effect of Genes on the Production Function of Health



However the genes enter also the production function of health, $f(.)$. In order to obtain clear implications, we have to assume that the impact of genes on the production function $\frac{\partial f}{\partial g}$, which can call the *productivity effect*, does not counterbalance the *price effect* $\frac{\partial p_e(g)}{\partial g}$. A sufficient condition is for genes not to interact with the investments, so that the ratio of partial derivatives $\frac{\partial f(I_d,I_e,g)}{\partial I_e} / \frac{\partial f(I_d,I_e,g)}{\partial I_d}$ does not depend on g. For example, this is the case if the cross partials are equal to zero, $\frac{\partial f(I_d,I_e,g)}{\partial I_e \partial g} = \frac{\partial f(I_d,I_e,g)}{\partial I_d \partial g} = 0$.

This is consistent with the assumption that the family does *not* know the genetic endowment of the child, since it is not (easily) observable. However they do know the production functions and they observe the shadow prices of the investments: that is, they are aware of how hard or easy it is for the child to exercise and to follow a nutritious diet.

However we can be more general; since the FOC must hold $\forall g$, we can derive a more general condition by taking the derivative with respect to g, obtaining:

$$\frac{\frac{\partial f(I_d,I_e,g)}{\partial I_e \partial g}\frac{\partial f(I_d,I_e,g)}{\partial I_d} - \frac{\partial f(I_d,I_e,g)}{\partial I_d \partial g}\frac{\partial f(I_d,I_e,g)}{\partial I_e}}{\left[\frac{\partial f(I_d,I_e,g)}{\partial I_d}\right]^2} = \frac{p_e' p_d - p_e p_d'}{[p_d]^2}$$

therefore we have that the productivity effect does not overturn the price effect if the sign of the right hand side of the equation is the same as the sign of the difference between the two sides, or if $|p_e' p_d - p_e p_d'| > \left|\frac{\partial f(I_d,I_e,g)}{\partial I_e \partial g}\frac{\partial f(I_d,I_e,g)}{\partial I_d} - \frac{\partial f(I_d,I_e,g)}{\partial D \partial g}\frac{\partial f(I_d,I_e,g)}{\partial I_e}\right|$

3 Empirical Results

3.1 The Data

To bring the model to the data, I use the Avon Longitudinal Study of Parents and Children (ALSPAC), an ongoing investigation on the health and development of children (`http://www.alspac.bris.ac.uk`). An extremely rich dataset collected by epidemiologic researchers from the University of Bristol, the ALSPAC follows prospectively a cohort of pregnant women living in a district in the former County of Avon with an expected delivery date between April 1991 and December 1992 (Golding et al. (2001)). 14,541 pregnant women were enrolled at the beginning of the study; 13,988 children were alive at year 1.

Health data were collected through regular questionnaires, and medical and educational records. Anthropometric, Physical Activity, and dual-energy X-ray absorptiometry (DXA) measures were obtained during research clinic visits.

Anthropometric measures

Height was measured by using a Harpenden stadiometer (Holtain Ltd, Crymych, United Kingdom), and weight was assessed by using a weighing scale (Tanita TBF 305; TanitaUKLtd,Yewsley, United Kingdom). A Lunar Prodigy DXA scanner (GE Medical Systems Lunar, Madison, WI) provided measures of body composition, including fat, lean body mass, and bone mass. Body mass index (BMI = weight (kg)/height squared (m^2)), and BMI normal z-scores were calculated from the 1990 British Growth Reference[8]. Although multiple measure of obesity are provided, we mostly focus on BMI because it was most frequently measured, easily comparable to many other studies, and provides an easy yet reliable measure of obesity risk[9].

Dietary assessment

Three-day dietary records including 2 weekday and 1 weekend day were obtained from adolescents a few days before the clinic visit; parents provided assistance as needed. Participants were instructed to record all foods and beverages consumed by using standard household measures. Records were reviewed during clinic visits to improve completeness. Questionnaires queried for information on vitamin supplements, type of milk or fat spreads consumed, and details of other foods commonly eaten. Diet records were coded and analyzed by using the Diet In Data Out software (MRC Human Nutrition Research, Elsie Widdowson Laboratory, Cambridge, United Kingdom), which generates food codes and weights of each item recorded (Price et al. (1995)). Average daily nutrient intakes were calculated by using BRIGADE (University of Bristol, Bristol, United Kingdom) - a nutrient analysis program based on a nutrient databank that included the fifth edition of McCance and Widdowson's food tables and supplements. Nutrients for foods not in the databank were obtained from the National Diet and Nutrition Sur-

[8]See Cole et al. (1998)

[9]See Taylor et al. (2010) for a discussion of the reliability of BMI in predicting coronary heart disease, diabetes, and all-cause mortality, as compared to other measures of adiposity.

vey nutrient databases or calculated from the manufacturers label. Food groups were formed on the basis of nutrient composition and culinary use of foods consumed. Dairy and milk groups were categorized into full-fat, low-fat, and nonfat on the basis of fat content. Total milk intake included full-fat, low-fat and nonfat plain and flavored milk. Total dairy intake included milk, cheese, cream, and yogurt; butter was not included.

Physical activity

The Actigraph uni-axial accelerometer (Actigraph, Fort Walton Beach, FL) was used to measure physical activity (Mattocks et al. (2008)). The accelerometer, which is worn around the waist, captures the frequency and intensity of movement in the vertical plane. Adolescents were asked to wear the accelerometer for 7 days during waking hours and to remove the instrument only during showering, bathing, and swimming. Physical activity measured directly from accelerometers (not including time spent swimming or cycling) was used. The accelerometers used in this study measured 1-min epochs. Adolescents with more than 3 days of accelerometer data were included in the analyses. Variables derived from the Actigraph were counts per minute as an estimate of total activity, minutes of sedentary activity, and minutes of moderate-to-vigorous activity (MVPA). On the basis of the results from a calibration study (Mattocks et al. (2008)), daily minutes of MVPA were defined by using cutoffs developed for moderate activity (accelerometer output between 3600 and 6200 counts/min) and vigorous activity (more than 6200 counts/min); time spent performing MVPA were summed to quantify minutes of MVPA.

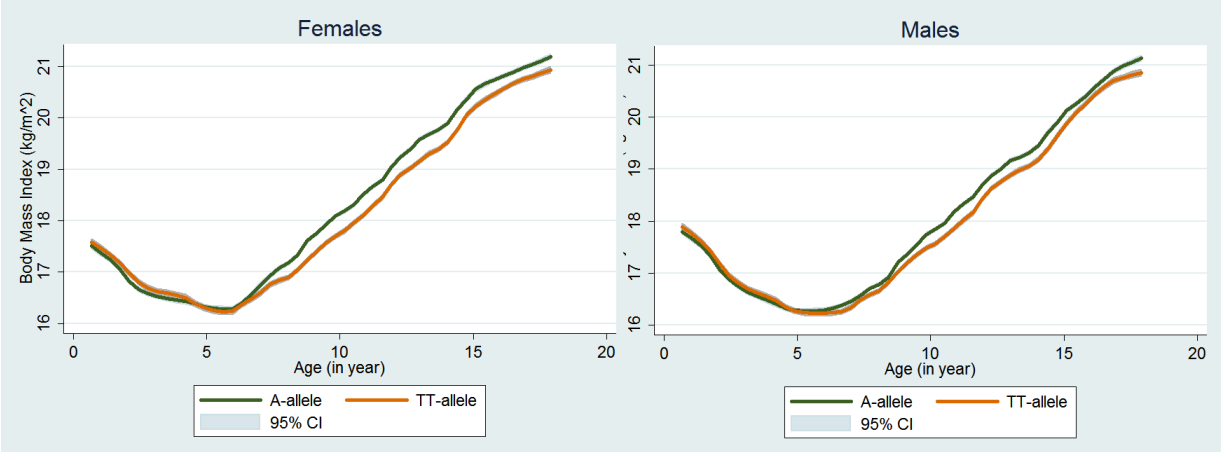
3.2 Evidence of interplay between genes and investment

First of all we look at the raw data and analyze the evolution of BMI over time, differentiated by gender; we compare the evolution of obesity between children who carry at least one risky A-allele in the FTO rs9939609 polymorphism (therefore those who are homozygous AA carries, or heterozygous AT) and those who don't (and therefore are homozygous TT carriers). As we can see in Figure 2, while in the first 5-6 years of their life there is no statistical difference between the two types, as children get older the distance between the two groups increases and then remains constant. This is a confirmation that in our dataset the FTO-gene has a significant effect on Body Mass Index, but that such effect is not present since birth, but actually arises as the child grows. This is consistent with the idea that the mere presence of the A-allele in not sufficient to induce a higher level BMI, but rather that the impact of the gene becomes pronounced as the effect of environment accumulates over time.

We now turn to the interaction between the genetic endowment of the child and the investment decisions of the family: the so-called Gene-Environment interaction (GxE)[10].

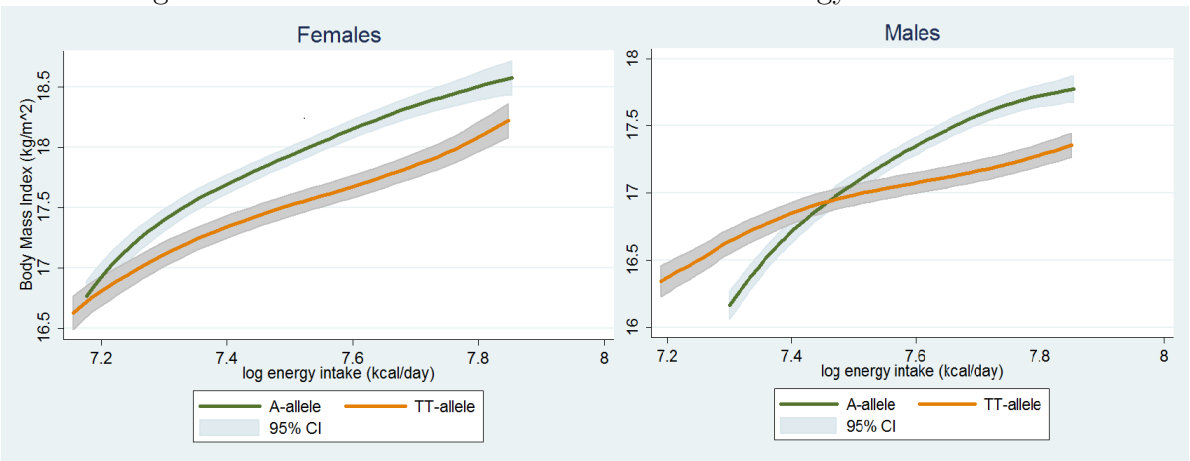
[10]The term was initially introduce by Plomin et al. (1977), and it is well explained in Moffitt et al. (2005, 2006) and in (Rutter, 2006, ch.9)

Figure 2: Evolution of Body-Mass-Index



Analyzing I_d by gender and genetic endowment, we use the data from the dietary questionnaires to investigate the relationship between the logarithm of the total amount of energy intake (kilo-calories per day) and the obesity level of the children aged 10 to 14 years old[11]. Not surprisingly, Figure 3 shows that higher energy intake is related to higher levels of BMI; furthermore, as predicted by the model, those children who carry at least one A-allele have a higher level of energy intake on average.The most interesting feature, however, is the significant difference in the slope describing the relationship between diet and BMI, and the fact that the two slopes intercept: genetic differences between children lead to differences in BMI *only when* they are abundant eaters. In other words, the impact of genes is *conditional* on a particular environment: the effect on the obesity-related-phenotype is evident only when *both* genes and environment are present and interact.

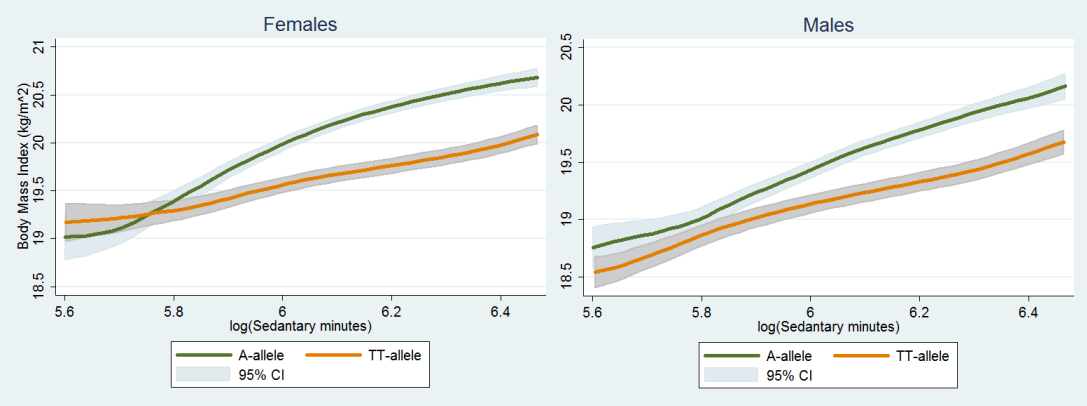
Figure 3: Gene-Diet Interaction: the Effect of energy intake on BMI



[11]Reliable data on food intake is limited to the clinical visits that started at those ages

A similar results can be found by analyzing the level of physical activity and exercise chosen by the adolescents, as depicted in Figure 4. The effect, as expected, is less pronounced; however we can still find very similar features: more time spent in a sedentary lifestyle leads to an increase in BMI, but significantly more so for those children who happen to carry the risky A-allele. Furthermore, the difference between the two types of children cannot be detected at low levels of sedentary activity (a high level of exercise), but only at higher ones, as predicted by the simple economic model.

Figure 4: Gene-Activity Interaction: the Effect of physical activity on BMI



3.3 A Linear Production Function of Health

We now turn to the estimation of the production function of health. Following Ehrlich and Chuma (1990) as well as Galama et al. (2012); Galama (2011), we consider a model that allows for decreasing returns to scale in investment: $H_t = A(X)\left(I_{e,t}^{\alpha_e} I_{d,t}^{\alpha_d} g^{\alpha_g}\right)$; we log-linearize the function and allow for interplay between genes and investment by introducing an interaction term between g and both $I_{d,t}$ and $I_{e,t}$; finally we consider the persistence of health capital by introducing $(1-\delta)H_{t-1}$, where δ is the depreciation rate of the health stock, represented in our case by BMI. Therefore we estimate the following equation:

$$\begin{aligned} log(H_{i,t}) = \mu &+ \alpha_e log(I_{i,t}^e) + \alpha_d log(I_{i,t}^d) + \alpha_g g + \\ &+ \alpha_{GxE} log(I_{i,t}^e) \cdot g + \alpha_{GxD} log(I_{i,t}^d) \cdot g + (1-\delta) log(H_{i,t-1}) \\ &+ \gamma_g g_i^{mom} + \gamma_h log(H_i^{mom}) + \beta X_{i,t} + \kappa_t + \mu_i + \varepsilon_{i,t} \end{aligned} \tag{3}$$

where $g_{mom,i}$ and $log(BMI)_{mom,i}$ represent the genetic endowment of the mother and her weight before pregnancy; X_{it} are control variables introduced to proxy for family and individual specific characteristics that might influence obesity and investment[12]; κ_t

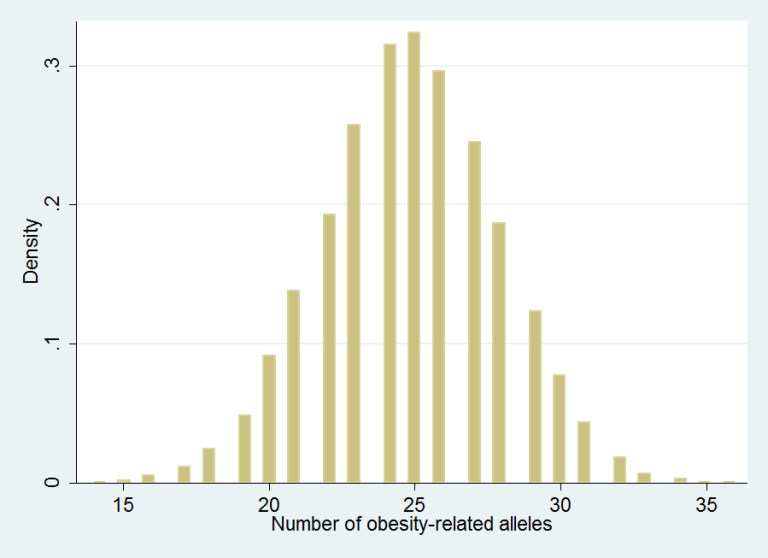
[12]We control for the age of the child at the clinic visit; the child's birth weight, as a proxy of prenatal investment; mother age at conception; dummies for different levels of mother and father Socio-Economic-Status and education levels; a dummy for teen-pregnancy; child parity

captures time effect and μ_i random effects idiosyncratic to the individual[13].

The α coefficients capture the relevant parameters of the production function of health; δ is the depreciation rate of the health stock; the γ parameters capture the level of intergenerational transmission of endowment and characteristics; finally the β capture the influence of demographic controls.

We ran the model separately for males and females, in order to capture potential gender differences in the production function, and we consider two different indexes of genetic endowment: first we let g be a dummy for whether the individual is carrying at least one minor allele of the FTO gene; then we consider a genetic-predisposition-score calculated as the number of obesity-related alleles of 26 different genes. We construct this score following Speliotes et al. (2010)[14] and Vimaleswaran and Loos (2010), and selected the appropriate obesity-related genes from the Genome-Wide-Association-Studies (GWAS) of Vimaleswaran and Loos (2010); Speliotes et al. (2010); Sandholt et al. (2012)[15]. As predicted by Mendel's law of independent assortment, the genetic score that we constructed displays a bell-shape similar to a normal, as shown in figure 3.3.

Figure 5: Distribution of the Genetic-Predisposition-Score



[13]Since the genetic endowment is fixed and not time-varying, we cannot run a fixed effect regression without losing information on the genetic influences. Running a random effect model or a simple regression does not change substantially the results.

[14]They call it "genetic-susceptibility" score

[15]The genes that we considered are: MC4R TMEM18 FTO TFAP2B BCDIN3D ETV5 BDNF GNPDA2 PPARG THADA IGF2BP2 TCF7L2 NPC1 MTCH2 PCSK1 KCTD15 SH2B1 NRXN3 HHEX CNR1 LYPLAL1 GCK NEGR1 PTER CDKN2 GCKR. All of them have been validated in various studies as obesity-related genetic loci, and for some there is evidence of potential environmental pathways through energy intake (diet) or energy expenditure (exercise). See the discussion in section 2.

Tables (1) and (2) report the coefficients of equation (3) for females and males respectively, when using the FTO gene as index of g.

As we can see from column (1) and (2), the genetic endowment of the child has a clear and strong effect on the obesity level, both for males and females, even after controlling for standard demographic characteristics. The effect is similar to the ones found by related studies[16], and comparable to a 10% increase in the BMI of the mother, which is quite substantial considering that it is the effect of a single genetic locus. Once we control for the investment choices of the family in column (3), the coefficient α_g does not change significantly; however, once we introduce the interaction between the gene and the two types of investment, we notice that the effect of FTO is not precisely estimated anymore, while the most important contribution to the evolution of BMI is due to diet and exercise, and their interaction with the genetic endowment. In this respect we find an interesting difference between males and females: for girls the most important investment is the dietary decision, which also display a significant interaction with the FTO gene, while their sedentary behavior is not predictive of BMI[17]; this is not true for boys, for whom both diet and exercise play and important role, and only the latter seem to interact with their genetic endowment. Notably, these effects are not distinguisable anymore once we omit the controls X in column (5), which prove to be a significant discriminating tool to parse out the difference between the general family enviroment and the particular effects of the investment decisions.

Finally it is worth noting the very high persistence of BMI: the depreciation coefficient δ is very low, more so for girls that for boys.

Tables (3) and (4) are similar to the previous tables, but this time we consider the genetic-predisposition-score as index of g. For comparability with the previous results we dichotomized the genetic score so that it is equal to one for the children who have more than the median number of obesity related alleles (number of 'fat-alleles' > 25).

We see that the main results carry through even when considering a polygenic approach: again we find gender-differences in the importance of various investments, and we find comparable magnitudes of persistence of BMI (δ). The main discrepancy with the previous set of results is that this time the effect of the genetic-score is significant in explaining the BMI of girls, even after controlling for the environment and its interplay with health behavioral decisions.

3.4 A CES Production Function of Health

One of the key predictions of the model was that, in equilibrium, the elasticity of substitution between the two investments depended on the genetic makeup of the individual (see equation (2)). In order to test this prediction we estimate a CES-production function of health, allowing all of the parameters to differ across genders and across genetic

[16] See Dina et al. (2007); Frayling et al. (2007); Timpson et al. (2008)

[17] It's worth noticing that once we control only for sedentary minutes and not for kilo-calories, then the investment in exercise becomes significant also for girls.

Table 1: Gene and Investment Interaction - Females

	(1)	(2)	(3)	(4)	(5)
Child FTO Gene	0.023	0.007	0.008	0.180	0.251
	[0.007]***	[0.003]**	[0.003]**	[0.132]	[0.133]*
Log(energy intake)			0.037	0.028	0.024
			[0.007]***	[0.008]***	[0.008]***
G×Diet				0.026	0.030
				[0.015]*	[0.015]**
Log(sedentary minutes)			0.010	0.012	-0.007
			[0.007]	[0.008]	[0.008]
G×Activity				-0.004	0.003
				[0.012]	[0.012]
Mom Gene		-0.006	-0.006	-0.006	
		[0.003]**	[0.003]**	[0.003]**	
$\log(BMI_{mom})$		0.075	0.074	0.074	
		[0.009]***	[0.009]***	[0.009]***	
$log(BMI)_{t-1}$		0.946	0.931	0.931	0.976
		[0.008]***	[0.009]***	[0.009]***	[0.007]***
Controls		X	X	X	
Observations	4418	4398	4398	4398	4398

Random effects model. * significant at 10%; ** significant at 5%; *** significant at 1%
Dependent variable: log BMI (kg/m^2); Controls: mom and dad education and SES;
mother age at pregnancy; parity; birth weight; age of child at clinic date;
dummy for single mother, time, low kilo-calories reporting, late respondent;

endowment. In other words, we don't consider DNA as a simple input into the production function, but rather we allow the genetic makeup of an individual to be a key determinant that sets the stage for the evolution of human capital.

We consider the following CES specification $H_t = A(X;g)\left[\alpha I_{e,t}^{\phi} + (1-\alpha) I_{d,t}^{\phi_d}\right]^{1/\phi}$. As before, we allow the multiplying constant $A(X;g)$ to depend on various demographic controls X as well as the depreciation rate of capital $(1-\delta)H_{t-1}$. Taking the logarithm we obtain the following equation to estimate:

$$\log H_{i,t} = \frac{1}{\phi}\log\left[\alpha {I_{i,t}^{e}}^{\phi} + (1-\alpha){I_{i,t}^{d}}^{\phi}\right] + (1-\delta)\log H_{i,t-1} + \\ + \gamma_h log(H_i^m) + \beta X_{i,t} + \kappa_t + \varepsilon_{i,t} \tag{4}$$

where the elasticity of substitution is captured by the parameter $\sigma = \frac{1}{1-\phi}$.

Assuming that the error term $\varepsilon_{i,t}$ follows a normal distribution, we can estimate equation (4) using maximum likelihood[18]. We split the sample according to gender and

[18]See section (B) for the details of the likelihood function

Table 2: Gene and Investment Interaction - Males

	(1)	(2)	(3)	(4)	(5)
Child FTO Gene	0.021	0.009	0.006	0.208	0.166
	[0.007]***	[0.003]***	[0.004]	[0.131]	[0.131]
Log(energy intake)			0.054	0.050	0.041
			[0.007]***	[0.009]***	[0.009]***
G×Diet				0.010	0.006
				[0.014]	[0.014]
Log(sedentary minutes)			0.027	0.020	0.003
			[0.006]***	[0.008]**	[0.007]
G×Activity				0.021	0.019
				[0.012]*	[0.012]
Mom Gene		-0.003	-0.003	-0.002	
		[0.003]	[0.003]	[0.003]	
$\log(BMI_{mom})$		0.069	0.069	0.068	
		[0.010]***	[0.010]***	[0.010]***	
$log(BMI)_{t-1}$		0.931	0.900	0.901	0.930
		[0.010]***	[0.010]***	[0.010]***	[0.009]***
Controls		X	X	X	
Observations	3952	3904	3904	3904	3904

Random effects model. * significant at 10%; ** significant at 5%; *** significant at 1%
Dependent variable: log BMI (kg/m^2); Controls: mom and dad education and SES;
mother age at pregnancy; parity; birth weight; age of child at clinic date;
dummy for single mother, time, low kilo-calories reporting, late respondent;

Table 3: Gene and Investment Interaction - Females

	(1)	(2)	(3)	(4)	(5)
Child Gene Score	0.039	0.006	0.005	0.312	0.320
	[0.006]***	[0.002]***	[0.003]*	[0.129]**	[0.129]**
Log(energy intake)			0.037	0.019	0.015
			[0.007]***	[0.011]*	[0.011]
G×Diet				0.030	0.031
				[0.014]**	[0.014]**
Log(sedentary minutes)			0.009	0.001	-0.015
			[0.007]	[0.010]	[0.010]
G×Activity				0.013	0.013
				[0.012]	[0.012]
Mom Gene		-0.006	-0.005	-0.005	
		[0.007]	[0.007]	[0.007]	
$\log(BMI_{mom})$		0.075	0.074	0.074	
		[0.009]***	[0.009]***	[0.009]***	
$log(BMI)_{t-1}$		0.944	0.930	0.931	0.976
		[0.009]***	[0.009]***	[0.009]***	[0.007]***
Controls		X	X	X	
Observations	4418	4398	4398	4398	4398

Random effects model. * significant at 10%; ** significant at 5%; *** significant at 1%
Dependent variable: log BMI (kg/m^2); Controls: mom and dad education and SES;
mother age at pregnancy; parity; birth weight; age of child at clinic date;
dummy for single mother, time, low kilo-calories reporting, late respondent;

Table 4: Gene and Investment Interaction - Males

	(1)	(2)	(3)	(4)	(5)
Child Gene Score	0.032	0.009	0.011	0.042	0.024
	[0.006]***	[0.003]***	[0.003]***	[0.127]	[0.127]
Log(energy intake)			0.053	0.058	0.047
			[0.007]***	[0.011]***	[0.011]***
G×Diet				-0.007	-0.008
				[0.014]	[0.014]
Log(sedentary minutes)			0.028	0.019	0.003
			[0.006]***	[0.010]**	[0.009]
G×Activity				0.014	0.012
				[0.012]	[0.012]
Mom Gene		0.000	0.002	0.002	
		[0.008]	[0.007]	[0.007]	
$\log(BMI_{mom})$		0.068	0.069	0.068	
		[0.010]***	[0.010]***	[0.010]***	
$log(BMI)_{t-1}$		0.929	0.901	0.901	0.930
		[0.010]***	[0.010]***	[0.010]***	[0.009]***
Controls		X	X	X	
Observations	3952	3904	3904	3904	3904

Random effects model. * significant at 10%; ** significant at 5%; *** significant at 1%
Dependent variable: log BMI (kg/m^2); Controls: mom and dad education and SES;
mother age at pregnancy; parity; birth weight; age of child at clinic date;
dummy for single mother, time, low kilo-calories reporting, late respondent;

two different indicators of genetic endowment: whether the child carries at least one risky A-allele and whether the child has a genetic score higher than 25; we obtain the results displayed in tables (5) and (6) respectively.

Table 5: CES-Production function, by gender and FTO allele

		Females				Males			
	FTO:	T-Allele		Risky A-Allele		T-Allele		Risky A-Allele	
H_{t-1}	$(1-\delta)$	0.987	(0.043)	0.949	(0.066)	0.804	(0.157)	0.966	(0.055)
log(Kcal)	α	0.564	(0.317)	0.645	(0.261)	0.258	(0.399)	0.540	(0.269)
	ϕ	0.438	(0.707)	0.578	(0.465)	0.670	(0.616)	0.398	(0.566)
	$1/(1-\phi)$	1.778		2.367		3.026		1.662	
$\log(BMI_{mom})$	γ_h	0.041	(0.032)	0.050	(0.031)	0.047	(0.044)	0.049	(0.027)
Constant	β	-2.520	(0.462)	-2.676	(0.452)	-1.771	(0.594)	-2.717	(0.489)
Controls		X		X		X		X	
Obs		805		3614		693		3261	

Dependent variable: log BMI (kg/m^2); Controls: mom and dad education and SES; mother age at pregnancy; parity; birth weight; age of child at clinic date; dummy for single mother, low kilo-calories reporting, time;

Table 6: CES-Production function, by gender and Genetic Score

		Females				Males			
	FTO:	Low Score		Risky High Score		Low Score		Risky High Score	
H_{t-1}	$(1-\delta)$	0.932	(0.117)	0.743	(0.228)	0.819	(0.167)	0.977	(0.061)
log(Kcal)	α	0.505	(0.349)	0.758	(0.382)	0.632	(0.441)	0.512	(0.339)
	ϕ	0.584	(0.697)	0.280	(1.092)	0.089	(1.061)	0.685	(0.537)
	$1/(1-\phi)$	2.404		1.388		1.097		3.170	
$\log(BMI_{mom})$	γ_h	0.053	(0.040)	0.099	(0.066)	0.028	(0.052)	0.038	(0.036)
Constant	β	-2.805	(0.663)	-1.719	(0.803)	-1.842	(0.704)	-2.660	(0.424)
Controls		X		X		X		X	
Obs		2509		1910		2183		1771	

Dependent variable: log BMI (kg/m^2); Controls: mom and dad education and SES; mother age at pregnancy; parity; birth weight; age of child at clinic date; dummy for single mother, low kilo-calories reporting, time;

First of all we can notice that the elasticity of substitution is always bigger than one, however it is not precisely estimated. Furthermore, there are seizable difference between genders and across genetic pools: the depreciation rate seems to be smaller for males; the share α of investment in diet (kilo-calories consumed) is smaller for males and for those who carry a T-allele of the FTO gene; finally, the intergenerational transmission of BMI, depicted by γ_h, is very small and not substantially different between genders and genetic pools.

4 Conclusion

We introduce a simple economic framework that combines the recent discoveries of molecular genetics with a model of health and human capital formation in the early periods of life. This enables us to understand how family decisions of how much to invest in the human capital of the child are affected by the genetic endowment that the child is born with. We find that the genes change the shadow prices and the rate of return to different types of investment, inducing a shift in the optimal allocation of family resources.

We test our model using a novel epidemiological dataset that contains precise information on children Body Mass Index, their dietary pattern, their level of physical activity, and combines them with assays of the children DNA. We find that the predictions of the model are born out by the data: higher level of investment in exercise and a lower caloric intake can offset the negative consequences of being born with a particular genetic makeup, and the interplay between genes and the family investment decisions have long term effects on the children well-being.

Our analysis suggests that, although many genetic loci have been associated with higher levels of BMI, obesity rates are strongly determined by the interaction between genes and environment, and behavioral and economic choices can prevent and curtail the insurgence of obesity.

Although 40-60% of the variation in obesity-related phenotypes has been estimated to be heritable and due to genetic endowment, policies targeted at children that promote healthy behaviors, such as diet and regular physical activity, can be very effective in preventing adiposity and curtailing the recent trend in obesity rates.

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Appendices

A Appendix: The Model

In section $(A.1)$ we consider the simplest version of the static model, where the investment levels are decided directly by the family and their prices depend on the genetic endowment of the child. In the second part, section$(A.2)$, we develop a little further the static model by introducing investment functions that depend on the allocation of goods and time, in the spirit of Grossman1972. We then derive how the prices of the investments depend on the genetic endowment of the child and the (shadow) prices of goods and time.

A.1 Static Model Without Goods and Time Inputs

First let's look at the **static version** where food consumption and exercise are considered as direct inputs, whose price varies with genetic endowment.

$$\begin{aligned} \max_{I_d, I_e, c} \quad & U(c, H) \\ s.t. & \\ Y &= p_c c + p_d(g) I_d + p_e(g) I_e \\ H &= f(I_d, I_e, g) \end{aligned}$$

where H is the stock of health (or human capital), which arises according to the production function $f(.)$, a function of different types of investments - in this case we consider two potential investment, exercise I_e and diet I_d. Y is income, which can be allocated to either consumption c or investments I_e or I_d, according to their relative prices $p_x(g)$ for $k = e, d$, which depend on the genetic endowment of the individual.

This is the fundamental assumption of our model: genes enter not only the production function of human capital, but also the price of investment: a different genetic makeup will induce variations in the subjective cost of investments [19].

To find the solution of this model, we consider the Lagrangian associated to this maximization and we substitute all of the investment functions into the main production function of human capital

$$\begin{aligned} \mathcal{L} &= U[c, H] + \lambda_y (Y - p_c c - p_d(g) I_d - p_e(g) I_e) \\ &= U[c, f(I_d, I_e, g)] + \lambda_y (Y - p_c c - p_d(g) I_d - p_e(g) I_e) \end{aligned}$$

[19] See the discussion in the text for the molecular genetics bases of this assumption as well as a more general model that derives the shadow prices of these two inputs from a more basic investment function that depends on genes, goods, and time and effort.

Maximizing with respect to the goods c, I_d, I_e, and normalizing for the price of the consumption good ($p_c = 1$), we obtain the following first order conditions

$$\begin{aligned}
\frac{\partial \mathcal{L}}{\partial c} &= \frac{\partial U}{\partial c} - \lambda_y = 0 \\
\frac{\partial \mathcal{L}}{\partial I_e} &= \frac{\partial U}{\partial H}\frac{\partial f\left(I_d, I_e, g\right)}{\partial I_e} - \lambda_y p_e\left(g\right) = 0 \\
\frac{\partial \mathcal{L}}{\partial I_d} &= \frac{\partial U}{\partial H}\frac{\partial f\left(I_d, I_e, g\right)}{\partial I_d} - \lambda_y p_d\left(g\right) = 0
\end{aligned}$$

The first set of equations tell us that the optimal level of goods spent in investment (I_e^*, I_d^*) is such that the marginal productivity of every dollar spent on investment has to be equal to the marginal utility of every dollar spent on consumption

$$\lambda_y = \frac{\partial U}{\partial H}\frac{\partial f\left(I_d, I_e, g\right)}{\partial I_e}\frac{1}{p_e\left(g\right)} = \frac{\partial U}{\partial H}\frac{\partial f\left(I_d, I_e, g\right)}{\partial I_d}\frac{1}{p_d\left(g\right)} = \frac{\partial U}{\partial c}$$

Focusing on the investment part we have that

$$\frac{\frac{\partial f(I_d, I_e, g)}{\partial I_e}}{p_e\left(g\right)} = \frac{\frac{\partial f(I_d, I_e, g)}{\partial I_d}}{p_d\left(g\right)}$$

Or

$$\frac{\frac{\partial f(I_d, I_e, g)}{\partial I_e}}{\frac{\partial f(I_d, I_e, g)}{\partial I_d}} = \frac{p_e\left(g\right)}{p_d\left(g\right)}$$

Since the prices of investment depend on the genetic endowment of the individuals, by changing the genes we have a variation in the shadow price of inputs and, therefore, a change in the returns to investment. For example, if we know that a particular gene, like FTO, has an effect on energy intake (and therefore the amount of food required by the organism) and not on energy expenditure (and therefore the calories consumed by physical activity), then we can say that $\frac{\partial p_e(g)}{\partial g} = 0$ while $\frac{\partial p_d(g)}{\partial g} > 0$, meaning that being on a diet is more "expensive" for individuals that carry a short allele in the FTO gene, while it has no effect on their (shadow) price of exercising. However the genes also enter the production function of health, $f\left(.\right)$. In order to obtain clear implications, we have to assume that the impact of genes on the production function $\frac{\partial f}{\partial g}$, which can call the *productivity effect*, does not counterbalance the *price effect* $\frac{\partial p_e(g)}{\partial g}$. A sufficient condition is for genes not to interact with the investments, so that the ratio of partial derivatives $\frac{\partial f(I_d, I_e, g)}{\partial I_e} / \frac{\partial f(I_d, I_e, g)}{\partial I_d}$ does not depend on g[20].

However, since the FOC must hold $\forall g$, we can derive a more general condition by taking the derivative with respect to g, obtaining:

$$\frac{\frac{\partial f(I_d, I_e, g)}{\partial I_e \partial g}\frac{\partial f(I_d, I_e, g)}{\partial I_d} - \frac{\partial f(I_d, I_e, g)}{\partial I_d \partial g}\frac{\partial f(I_d, I_e, g)}{\partial I_e}}{\left[\frac{\partial f(I_d, I_e, g)}{\partial I_d}\right]^2} = \frac{p_e' p_d - p_e p_d'}{\left[p_d\right]^2}$$

[20]For example, this is the case if the cross partials are equal to zero, $\frac{\partial f(I_d, I_e, g)}{\partial I_e \partial g} = \frac{\partial f(I_d, I_e, g)}{\partial I_d \partial g} = 0$

therefore we have that the productivity effect does not overturn the price effect if the sign of the right hand side of the equation is the same as the sign of the difference between the two sides, or if $|p'_e p_d - p_e p'_d| > \left| \frac{\partial f(I_d,I_e,g)}{\partial I_e \partial g} \frac{\partial f(I_d,I_e,g)}{\partial I_d} - \frac{\partial f(I_d,I_e,g)}{\partial D \partial g} \frac{\partial f(I_d,I_e,g)}{\partial I_e} \right|$

A.1.1 Functional form Specification

For simplicity, let's assume a Cobb-Douglas functional form specification for the utility function, so that

$$U[c, H] = \phi \log c + (1 - \phi) \log H$$

Furthermore, we can assume different specification for the the production function of health, for example a Cobb-Douglas specification or a more general Constant Elasticity of Substitution (CES).

Cobb-Douglas First, let's assume a Cobb-Douglas functional form specification also for the production function of health: $f(I_d, I_e, g) = A(I_e^{\alpha_e} I_d^{\alpha_d} g^{\alpha_g})$. The problem becomes

$$\begin{aligned} \max_{I_d, I_e, c} \quad & \phi \log c + (1 - \phi) [\log A + \alpha_e \log I_e + \alpha_d \log I_d + \alpha_g \log g] \\ s.t. & \\ Y &= c + p_d(g) I_d + p_e(g) I_e \end{aligned}$$

The first order condition become:

$$\begin{aligned} \frac{\partial \mathcal{L}}{\partial c} &= \frac{\phi}{c^*} - \lambda_y = 0 \\ \frac{\partial \mathcal{L}}{\partial I_e} &= \frac{(1 - \phi)\alpha_e}{I_e^*} - \lambda_y p_e(g) = 0 \\ \frac{\partial \mathcal{L}}{\partial I_d} &= \frac{(1 - \phi)\alpha_d}{I_d^*} - \lambda_y p_d(g) = 0 \end{aligned}$$

We have that

$$\lambda_y = \frac{\phi}{c^*} = \frac{(1 - \phi)\alpha_e}{I_e^* p_e(g)} = \frac{(1 - \phi)\alpha_d}{I_d^* p_d(g)}$$

Expressing everything in term of consumption c^* we have

$$\begin{aligned} I_e^* &= \frac{(1 - \phi)}{\phi} \frac{\alpha_e}{p_e(g)} c^* \\ I_d^* &= \frac{(1 - \phi)}{\phi} \frac{\alpha_d}{p_d(g)} c^* \end{aligned}$$

Substituting everything into the budget constraint, in order to obtain the demand as function of prices and income, we have:

$$\begin{aligned} Y &= c^* + p_e(g) I_e^* + p_d(g) I_d^* \\ &= c^* + p_e(g) \frac{(1-\phi)}{\phi} \frac{\alpha_e}{p_e(g)} c^* + p_d(g) \frac{(1-\phi)}{\phi} \frac{\alpha_d}{p_d(g)} c^* \\ &= c^* \left[1 + \frac{(1-\phi)}{\phi} (\alpha_e + \alpha_d) \right] \end{aligned}$$

so that we have

$$\begin{aligned} c^* &= Y\phi \left[\frac{1}{\phi + (1-\phi)(\alpha_e + \alpha_d)} \right] \\ I_e^* &= Y \frac{\alpha_e (1-\phi)}{p_e(g)} \left[\frac{1}{\phi + (1-\phi)(\alpha_e + \alpha_d)} \right] \\ I_d^* &= Y \frac{\alpha_d (1-\phi)}{p_d(g)} \left[\frac{1}{\phi + (1-\phi)(\alpha_e + \alpha_d)} \right] \end{aligned}$$

Note that if we assume that $\alpha_e + \alpha_d = 1$, so that there are constant returns to scale to I_e and I_d in the production function of health, then the results simplifies to the usual result that the optimal consumption of c and H will be proportional to income, with weights proportional to their importance in the utility function (ϕ and $1-\phi$ respectively) and inversely proportional to their prices; for example $c^* = \phi Y$.

However it is interesting to focus on the investment, in order to better understand the choices of substitution between the two inputs:

$$\begin{aligned} \frac{\frac{\partial f(I_d, I_e, g)}{\partial I_e}}{\frac{\partial f(I_d, I_e, g)}{\partial I_d}} &= \frac{p_e(g)}{p_d(g)} \\ \frac{\alpha_e A I_e^{*\alpha_e - 1} I_d^{*\alpha_d} g^{\alpha_g}}{\alpha_d A I_e^{*\alpha_e} I_d^{*\alpha_d - 1} g^{\alpha_g}} &= \frac{p_e(g)}{p_d(g)} \\ \frac{\alpha_e I_d^*}{\alpha_d I_e^*} &= \frac{p_e(g)}{p_d(g)} \\ I_d^* &= \frac{\alpha_d}{\alpha_e} \frac{p_e(g)}{p_d(g)} I_e^* \end{aligned}$$

It is easy to see that the dependency on genes drops out of the production function, and g influences only the relative prices

CES A similar result holds if we consider the case of Constant Elasticity of Substitution, so that the production function of health becomes:
$f(I_d, I_e, g) = A \left[\alpha_e I_e^\eta + \alpha_d I_d^\eta + (1 - \alpha_e - \alpha_d) g^\eta \right]^{\frac{1}{\eta}}$
and the family problem:

$$\begin{aligned}
\max_{I_d,I_e,c} \quad & \phi \log c + (1-\phi)\left[\log A + \frac{1}{\eta}\log\left[\alpha_e I_e^{\eta} + \alpha_d I_d^{\eta} + (1-\alpha_e-\alpha_d)\, g^{\eta}\right]\right] \\
s.t. & \\
Y &= c + p_d(g)\, I_d + p_e(g)\, I_e
\end{aligned}$$

The first order condition become:

$$\begin{aligned}
\frac{\partial \mathcal{L}}{\partial c} &= \frac{\phi}{c^*} - \lambda_y = 0 \\
\frac{\partial \mathcal{L}}{\partial I_e} &= \frac{(1-\phi)\,\alpha_e I_e^{*\eta-1}}{\left[\alpha_e I_e^{*\eta} + \alpha_d I_d^{*\eta} + (1-\alpha_e-\alpha_d)\, g^{\eta}\right]}\frac{\eta}{\eta} - \lambda_y p_e(g) = 0 \\
\frac{\partial \mathcal{L}}{\partial I_d} &= \frac{(1-\phi)\,\alpha_d I_d^{*\eta-1}}{\left[\alpha_e I_e^{*\eta} + \alpha_d I_d^{*\eta} + (1-\alpha_e-\alpha_d)\, g^{\eta}\right]}\frac{\eta}{\eta} - \lambda_y p_d(g) = 0
\end{aligned}$$

We have that

$$\begin{aligned}
\lambda_y &= \frac{\phi}{c^*} \\
&= \frac{1}{p_e(g)}\frac{(1-\phi)\,\alpha_e I_e^{*\eta-1}}{f\left(I_e^*, I_d^*, g\right)^{\eta}/A} \\
&= \frac{1}{p_d(g)}\frac{(1-\phi)\,\alpha_d I_d^{*\eta-1}}{f\left(I_e^*, I_d^*, g\right)^{\eta}/A}
\end{aligned}$$

Regretfully there is no closed form solution to this problem[21]. However it can be useful to focus on the FOC for investment, in order to better understand the choices of substitution between the two inputs:

$$\frac{\frac{\partial f(I_d,I_e,g)}{\partial I_e}}{\frac{\partial f(I_d,I_e,g)}{\partial I_d}} = \frac{p_e(g)}{p_d(g)}$$

[21]Even if we use the relationship between the investments to substitute it into the FOC to obtain c^* as a function of I_e^*, we get:

$$\begin{aligned}
c^* &= \frac{\phi(1-\phi)\,\alpha_e}{p_e(g)}\frac{I_e^{*\eta-1}}{\left[\alpha_e I_e^{*\eta} + \alpha_d I_d^{*\eta} + (1-\alpha_e-\alpha_d)\, g^{\eta}\right]} \\
&= \frac{\phi(1-\phi)\,\alpha_e}{p_e(g)}\frac{I_e^{*\eta-1}}{\left[\alpha_e I_e^{*\eta} + \left(\frac{\alpha_d^2}{\alpha_e}\right)^{\eta}\left(\frac{p_e(g)}{p_d(g)}\right)^{\eta/\eta-1} I_e^{*\eta} + (1-\alpha_e-\alpha_d)\, g^{\eta}\right]} \\
&\quad \frac{\phi(1-\phi)}{p_e(g)}\frac{\alpha_e}{I_e^*\left[\alpha_e + \left(\frac{\alpha_d^2}{\alpha_e}\right)^{\eta}\left(\frac{p_e(g)}{p_d(g)}\right)^{\eta/\eta-1} + (1-\alpha_e-\alpha_d)\left(\frac{g}{I_e^*}\right)^{\eta}\right]}
\end{aligned}$$

which does not lead to a closed form solution. However it is interesting to note that the optimal consumption c^* will depend on the genetic makeup g even if the allocation of resources between the investments does not.

$$\begin{aligned}
\frac{A\left[\alpha_e I_e^{*\eta}+\alpha_d I_d^{*\eta}+\left(1-\alpha_e-\alpha_d\right)g^{\eta}\right]^{\frac{1-\eta}{\eta}}\alpha_e I_e^{*\eta-1}}{A\left[\alpha_e I_e^{*\eta}+\alpha_d I_d^{*\eta}+\left(1-\alpha_e-\alpha_d\right)g^{\eta}\right]^{\frac{1-\eta}{\eta}}\alpha_d I_d^{*\eta-1}} &= \frac{p_e\left(g\right)}{p_d\left(g\right)} \\
\frac{\alpha_e}{\alpha_d}\left(\frac{I_d^*}{I_e^*}\right)^{\eta-1} &= \frac{p_e\left(g\right)}{p_d\left(g\right)} \\
I_d^* &= \left[\frac{\alpha_d}{\alpha_e}\frac{p_e\left(g\right)}{p_d\left(g\right)}\right]^{1/\eta-1} I_e^*
\end{aligned}$$

Also in this case we have that the ratio of derivatives does not depend on g, and therefore the effect of genes is apparent only through the prices. These are example of quite general functions, allowing for different elasticities of substitutions between inputs, that still satisfy our assumption. Furthermore, in these cases we have the the optimal inputs are proportional to each other and to the ratio of their prices.

A.2 Static Model With Goods and Time Inputs

In order to give some microfoundations to the claim that the prices of investment depend on the genetic makeup of an individual, we can consider the model presented in equation (1), where the two investments are themselves function of market goods - which prices are equal for everybody, as well as time and effort, and the genetic endowment of the individual. To find a solution to this model, we consider the Lagrangian associated to this maximization and we substitute all of the investment functions into the main production function of human capital

$$\begin{aligned}
\mathcal{L} &= U\left[c,\tau_l,H\right]+\lambda_\tau\left(\Omega-\tau_l-\tau_e-\tau_d\right)+\lambda_y\left(Y-p_c c-p_d x_d-p_e x_e\right) \\
&= U\left[c,\tau_l,f\left(I_e\left(x_e,\tau_e,g\right),I_d\left(x_d,\tau_d,g\right),g\right)\right]+ \\
&\quad +\lambda_\tau\left(\Omega-\tau_l-\tau_e-\tau_d\right)+\lambda_y\left(Y-p_c c-p_d x_d-p_e x_e\right)
\end{aligned}$$

Assuming that all of the time not used investing is devoted to leisure we have that the time constraint is always binding and $\tau_l=\Omega-\tau_e-\tau_d$ so that

$$\begin{aligned}
\mathcal{L} &= U\left[c,\Omega-\tau_e-\tau_d,f\left(I_e\left(x_e,\tau_e,g\right),I_d\left(x_d,\tau_d,g\right),g\right)\right]+ \\
&\quad +\lambda_y\left(Y-p_c c-p_d x_d-p_e x_e\right)
\end{aligned}$$

Maximizing with respect to the goods c,x_e,x_d and time τ_e,τ_d, and normalizing for the price of the consumption good ($p_c=1$), we obtain the following first order conditions

$$\begin{aligned}
\frac{\partial\mathcal{L}}{\partial c} &= \frac{\partial U}{\partial c}-\lambda_y=0 \\
\frac{\partial\mathcal{L}}{\partial x_e} &= \frac{\partial U}{\partial H}\frac{\partial f\left(I_d,I_e,g\right)}{\partial I_e\left(x_e,\tau_e,g\right)}\frac{\partial I_e\left(x_e,\tau_e,g\right)}{\partial x_e}-\lambda_y p_e=0
\end{aligned}$$

$$\frac{\partial \mathcal{L}}{\partial x_d} = \frac{\partial U}{\partial H}\frac{\partial f\left(I_d, I_e, g\right)}{\partial I_d\left(x_d, \tau_d, g\right)}\frac{\partial I_d\left(x_d, \tau_d, g\right)}{\partial x_d} - \lambda_y p_d = 0$$

And

$$\begin{aligned}\frac{\partial \mathcal{L}}{\partial \tau_e} &= \frac{\partial U}{\partial H}\frac{\partial f\left(I_d, I_e, g\right)}{\partial I_e\left(x_e, \tau_e, g\right)}\frac{\partial I_e\left(x_e, \tau_e, g\right)}{\partial \tau_e} - \frac{\partial U}{\partial \tau_l} = 0\\ \frac{\partial \mathcal{L}}{\partial \tau_d} &= \frac{\partial U}{\partial H}\frac{\partial f\left(I_d, I_e, g\right)}{\partial I_d\left(x_d, \tau_d, g\right)}\frac{\partial I_d\left(x_d, \tau_d, g\right)}{\partial \tau_d} - \frac{\partial U}{\partial \tau_l} = 0\end{aligned}$$

The first set of equations tell us that the optimal level of goods spent in investment (x_e^*, x_d^*) is such that the marginal productivity of every dollar spent on investment has to be equal to the marginal utility of every dollar spent on consumption

$$\frac{\partial U}{\partial H}\frac{\partial f\left(I_d, I_e, g\right)}{\partial I_e\left(x_e, \tau_e, g\right)}\frac{\partial I_e\left(x_e, \tau_e, g\right)}{\partial x_e}\frac{1}{p_e} = \frac{\partial U}{\partial H}\frac{\partial f\left(I_d, I_e, g\right)}{\partial I_d\left(x_d, \tau_d, g\right)}\frac{\partial I_d\left(x_d, \tau_d, g\right)}{\partial x_d}\frac{1}{p_d} = \frac{\partial U}{\partial c}$$

Focusing on the investment part we have that

$$\frac{\frac{\partial f(I_d,I_e,g)}{\partial I_e(x_e,\tau_e,g)}\frac{\partial I_e(x_e,\tau_e,g)}{\partial x_e}}{p_e} = \frac{\frac{\partial f(I_d,I_e,g)}{\partial I_d(x_d,\tau_d,g)}\frac{\partial I_d(x_d,\tau_d,g)}{\partial x_d}}{p_d}$$

Or

$$\frac{\frac{\partial f(I_d,I_e,g)}{\partial I_e(x_e,\tau_e,g)}\frac{\partial I_e(x_e,\tau_e,g)}{\partial x_e}}{\frac{\partial f(I_d,I_e,g)}{\partial I_d(x_d,\tau_d,g)}\frac{\partial I_d(x_d,\tau_d,g)}{\partial x_d}} = \frac{p_e}{p_d}$$

Rearranging the terms we obtain:

$$\begin{aligned}\frac{\frac{\partial f(I_d,I_e,g)}{\partial I_e(x_e,\tau_e,g)}}{\frac{\partial f(I_d,I_e,g)}{\partial I_d(x_d,\tau_d,g)}} &= \frac{p_e/\frac{\partial I_e(x_e,\tau_e,g)}{\partial x_e}}{p_d/\frac{\partial I_d(x_d,\tau_d,g)}{\partial x_d}}\\ \frac{f'_{I_e}}{f'_d} &= \frac{p_e/I'_e}{p_d/I'_d} = \frac{p'_e}{p'_d}\end{aligned}$$

A.2.1 Functional form Specification

Let's make some functional form specifications that will ease the solution of the model. Consider a Cobb-Douglas specification for all of the functions

$$\begin{aligned}\max_{x_e, x_d, \{\tau_k\}_{k=e}^{d}, c} \quad & \phi_1 \log c + \phi_2 \log \tau_l + (1 - \phi_1 - \phi_2)\log H\\ s.t.\\ \Omega &= \tau_l + \tau_e + \tau_d\\ Y &= p_c c + p_d x_d + p_e x_e\end{aligned}$$

$$
\begin{aligned}
H &= A_H \left(I_e^{\alpha_e} I_d^{\alpha_d} g^{\alpha_g} \right) \\
I_e &= A_e \left(x_e^{\gamma_1} \tau_e^{\gamma_2} g^{\gamma_3} \right) \\
I_d &= A_d \left(x_d^{\delta_1} \tau_d^{\delta_2} g^{\delta_3} \right)
\end{aligned}
$$

Assuming that all of the time not used investing is devoted to leisure we have that the time constraint is always binding and $\tau_l = \Omega - \tau_e - \tau_d$ so that

$$
\begin{aligned}
\mathcal{L} &= \phi_1 \log c + \phi_2 \log \left(\Omega - \tau_e - \tau_d \right) \\
&\quad + \left(1 - \phi_1 - \phi_2 \right) \log A_H \left[\left(A_e x_e^{\gamma_1} \tau_e^{\gamma_2} g^{\gamma_3} \right)^{\alpha_e} \left(A_d x_d^{\delta_1} \tau_d^{\delta_2} g^{\delta_3} \right)^{\alpha_d} g^{\alpha_g} \right] + \\
&\quad + \lambda_y \left(Y - p_c c - p_d x_d - p_e x_e \right) \\
&= \phi_1 \log c + \phi_2 \log \left(\Omega - \tau_e - \tau_d \right) \\
&\quad + \left(1 - \phi_1 - \phi_2 \right) \log A_H A_e^{\alpha_e} A_d^{\alpha_d} g^{\gamma_3 \alpha_e + \delta_3 \alpha_d + \alpha_g} \\
&\quad + \left(1 - \phi_1 - \phi_2 \right) \gamma_1 \alpha_e \log x_e + \left(1 - \phi_1 - \phi_2 \right) \delta_1 \alpha_d \log x_d + \\
&\quad + \left(1 - \phi_1 - \phi_2 \right) \gamma_2 \alpha_e \log \tau_e + \left(1 - \phi_1 - \phi_2 \right) \delta_2 \alpha_d \log \tau_d + \\
&\quad + \lambda_y \left(Y - p_c c - p_d x_d - p_e x_e \right)
\end{aligned}
$$

It is easy to see that in this case genes g do not affect anything, but simply enter as a constant, similar to the effect of A. Maximizing with respect to the goods c, x_e, x_d and time τ_e, τ_d, and normalizing for the price of the consumption good ($p_c = 1$), we obtain the following first order conditions

$$
\begin{aligned}
\frac{\partial \mathcal{L}}{\partial c} &= \frac{\phi_1}{c} - \lambda_y = 0 \\
\frac{\partial \mathcal{L}}{\partial x_e} &= \frac{\left(1 - \phi_1 - \phi_2 \right) \gamma_1 \alpha_e}{x_e} - \lambda_y p_e = 0 \\
\frac{\partial \mathcal{L}}{\partial x_d} &= \frac{\left(1 - \phi_1 - \phi_2 \right) \delta_1 \alpha_d}{x_d} - \lambda_y p_d = 0
\end{aligned}
$$

And

$$
\begin{aligned}
\frac{\partial \mathcal{L}}{\partial \tau_e} &= \frac{\left(1 - \phi_1 - \phi_2 \right) \gamma_2 \alpha_e}{\tau_e} - \frac{\phi_2}{\Omega - \tau_e - \tau_d} = 0 \\
\frac{\partial \mathcal{L}}{\partial \tau_d} &= \frac{\left(1 - \phi_1 - \phi_2 \right) \delta_2 \alpha_d}{\tau_d} - \frac{\phi_2}{\Omega - \tau_e - \tau_d} = 0
\end{aligned}
$$

Looking at the optimal allocation of goods, we have that

$$
\begin{aligned}
\frac{\phi_1}{c} &= \frac{\left(1 - \phi_1 - \phi_2 \right) \gamma_1 \alpha_e}{p_e x_e} = \frac{\left(1 - \phi_1 - \phi_2 \right) \delta_1 \alpha_d}{x_d p_d} \\
x_e &= \frac{\left(1 - \phi_1 - \phi_2 \right)}{\phi_1} \frac{\gamma_1 \alpha_e}{p_e} c
\end{aligned}
$$

$$x_d = \frac{(1-\phi_1-\phi_2)}{\phi_1}\frac{\delta_1\alpha_d}{p_d}c$$

Substituting into the budget constraint we obtain that

$$\begin{aligned} Y &= c + p_e x_e + p_d x_d \\ &= c\left[1 + p_e\frac{(1-\phi_1-\phi_2)}{\phi_1}\frac{\gamma_1\alpha_e}{p_e} + p_d\frac{(1-\phi_1-\phi_2)}{\phi_1}\frac{\delta_1\alpha_d}{p_d}\right] \\ &= c\left[\frac{\phi_1 + (1-\phi_1-\phi_2)(\gamma_1\alpha_e + \delta_1\alpha_d)}{\phi_1}\right] \end{aligned}$$

so that the optimal allocations become:

$$\begin{aligned} c^* &= \frac{\phi_1}{\phi_1 + (1-\phi_1-\phi_2)(\gamma_1\alpha_e + \delta_1\alpha_d)}Y \\ x_e^* &= \frac{(1-\phi_1-\phi_2)\gamma_1\alpha_e}{\phi_1 + (1-\phi_1-\phi_2)(\gamma_1\alpha_e + \delta_1\alpha_d)}\frac{Y}{p_e} \\ x_d^* &= \frac{(1-\phi_1-\phi_2)\delta_1\alpha_d}{\phi_1 + (1-\phi_1-\phi_2)(\gamma_1\alpha_e + \delta_1\alpha_d)}\frac{Y}{p_d} \end{aligned}$$

Looking at the FOC for time allocation, we have that the ratio of the time spent investing in the two inputs is constant and equl to the relation of productivity coefficients: $\frac{\tau_d}{\tau_e} = \frac{\delta_2\alpha_d}{\gamma_2\alpha_e}$. Remembering that $\Omega - \tau_e - \tau_d = \tau_l$, we have that $\frac{\tau_l}{\tau_e} = \frac{(1-\phi_1-\phi_2)\gamma_2\alpha_e}{\phi_2}$. Substituting into the time budget constrain:

$$\begin{aligned} \Omega &= \tau_l^* + \tau_e^* + \tau_d^* \\ &= \left[\frac{(1-\phi_1-\phi_2)\gamma_2\alpha_e}{\phi_2} + 1 + \frac{\delta_2\alpha_d}{\gamma_2\alpha_e}\right]\tau_e^* \end{aligned}$$

so that the optimal allocation becomes

$$\begin{aligned} \tau_e^* &= \frac{\phi_2\gamma_2\alpha_e}{(1-\phi_1-\phi_2)\gamma_2^2\alpha_e^2 + \phi_2\gamma_2\alpha_e + \phi_2\delta_2\alpha_d}\Omega \\ \tau_d^* &= \frac{\phi_2\delta_2\alpha_d}{(1-\phi_1-\phi_2)\gamma_2^2\alpha_e^2 + \phi_2\gamma_2\alpha_e + \phi_2\delta_2\alpha_d}\Omega \\ \tau_l^* &= \frac{(1-\phi_1-\phi_2)\gamma_2^2\alpha_e^2}{(1-\phi_1-\phi_2)\gamma_2^2\alpha_e^2 + \phi_2\gamma_2\alpha_e + \phi_2\delta_2\alpha_d}\Omega \end{aligned}$$

CES for the Investment Functions Let's assume instead that the investment functions follow a Constant Elasticity of Substitution functional form, while the production function a normal Cobb-Douglas. Therefore we have the following maximazation

$$\max_{x_e, x_d, \{\tau_k\}_{k=e}^d, c} \phi_1 \log c + \phi_2 \log \tau_l + (1-\phi_1-\phi_2)\log H$$

$$
\begin{aligned}
&s.t.\\
&\Omega = \tau_l + \tau_e + \tau_d\\
&Y = p_c c + p_e x_e + p_d x_d\\
&H = A_H \left(I_e^{\alpha_e} I_d^{\alpha_d} g^{\alpha_g}\right)\\
I_e\left(x_e, \tau_e, g\right) &= A_e \left[\gamma_1 x_e^{\eta_e} + \gamma_2 \tau_e^{\eta_e} + \left(1-\gamma_1-\gamma_2\right) g^{\eta_e}\right]^{\frac{1}{\eta_e}}\\
I_d\left(x_d, \tau_d, g\right) &= A_d \left[\delta_1 x_d^{\eta_d} + \delta_2 \tau_d^{\eta_d} + \left(1-\delta_1-\delta_2\right) g^{\eta_d}\right]^{\frac{1}{\eta_d}}
\end{aligned}
$$

Therefore we have that

$$
\begin{aligned}
\frac{\partial I_e\left(x_e, \tau_e, g\right)}{\partial x_e} &= \gamma_1 x_e^{\eta_e - 1} \frac{\eta_e}{\eta_e} A_e \left[\gamma_1 x_e^{\eta_e} + \gamma_2 \tau_e^{\eta_e} + \left(1-\gamma_1-\gamma_2\right) g^{\eta_e}\right]^{\frac{1}{\eta_e}-1}\\
&= \gamma_1 x_e^{\eta_e - 1} I_e^{1-\eta_e} A_e^{\eta_e}\\
\frac{\partial I_d\left(x_d, \tau_d, g\right)}{\partial x_d} &= \delta_1 x_d^{\eta_d - 1} \frac{\eta_d}{\eta_d} A_d \left[\delta_1 x_d^{\eta_d} + \delta_2 \tau_d^{\eta_d} + \left(1-\delta_1-\delta_2\right) g^{\eta_d}\right]^{\frac{1}{\eta_d}-1}\\
&= \delta_1 x_d^{\eta_d - 1} I_d^{1-\eta_d} A_d^{\eta_d}
\end{aligned}
$$

Focusing only on the investment decisions, we know that it must hold

$$
\begin{aligned}
\frac{\frac{\partial f(I_e, I_d, g)}{\partial I_e(x_e, \tau_e, g)}}{\frac{\partial f(I_e, I_d, g)}{\partial I_d(x_d, \tau_d, g)}} &= \frac{p_e / \frac{\partial I_e(x_e, \tau_e, g)}{\partial x_e}}{p_d / \frac{\partial I_d(x_d, \tau_d, g)}{\partial x_d}}\\
\frac{\alpha_e H I_e^{-1}}{\alpha_d H I_d^{-1}} &= \frac{p_e / \gamma_1 x_e^{\eta_e - 1} I_e^{1-\eta_e} A_e^{\eta_e}}{p_d / \delta_1 x_d^{\eta_d - 1} I_d^{1-\eta_d} A_d^{\eta_d}}\\
\frac{\alpha_e I_e^{-1} x_e^{\eta_e - 1} I_e^{1-\eta_e}}{\alpha_d I_d^{-1} x_d^{\eta_d - 1} I_d^{1-\eta_d}} &= \frac{p_e / \gamma_1 A_e^{\eta_e}}{p_d / \delta_1 A_d^{\eta_d}}\\
\frac{x_e^{\eta_e - 1} / \left(A_e I_e\right)^{\eta_e}}{x_d^{\eta_d - 1} / \left(A_d I_d\right)^{\eta_d}} &= \frac{p_e / \gamma_1 \alpha_e}{p_d / \delta_1 \alpha_d}\\
\frac{x_e^{\eta_e - 1} / \left[\gamma_1 x_e^{\eta_e} + \gamma_2 \tau_e^{\eta_e} + \left(1-\gamma_1-\gamma_2\right) g^{\eta_e}\right]}{x_d^{\eta_d - 1} / \left[\delta_1 x_d^{\eta_d} + \delta_2 \tau_d^{\eta_d} + \left(1-\delta_1-\delta_2\right) g^{\eta_d}\right]} &= \frac{p_e / \gamma_1 \alpha_e}{p_d / \delta_1 \alpha_d}
\end{aligned}
$$

And, looking at the time allocation, we get a similar result:

$$
\begin{aligned}
\frac{\partial f\left(I_e, I_d, g\right)}{\partial I_e\left(x_e, \tau_e, g\right)} \frac{\partial I_e\left(x_e, \tau_e, g\right)}{\partial \tau_e} &= \frac{\partial f\left(I_e, I_d, g\right)}{\partial I_d\left(x_d, \tau_d, g\right)} \frac{\partial I_d\left(x_d, \tau_d, g\right)}{\partial \tau_d}\\
\alpha_e H I_e^{-1} \gamma_2 A_e^{\eta_e} I_e^{1-\eta_e} \tau_e^{\eta_e - 1} &= \alpha_d H I_d^{-1} \delta_2 A_d^{\eta_d} I_d^{1-\eta_d} \tau_d^{\eta_d - 1}\\
\frac{\tau_e^{\eta_e - 1} / \left(A_e I_e\right)^{\eta_e}}{\tau_d^{\eta_d - 1} / \left(A_d I_d\right)^{\eta_d}} &= \frac{\alpha_d \delta_2}{\alpha_e \gamma_2}
\end{aligned}
$$

So we have that the optimal level of goods $\left(x_e, x_d\right)^*$ and time $\left(\tau_e, \tau_d\right)^*$ will depend on the genetic makeup, but their ratio will be constant

$$
\frac{\tau_e^{1-\eta_e}}{\tau_d^{1-\eta_d}} \frac{\alpha_d \delta_2}{\alpha_e \gamma_2} = \frac{p_e / \gamma_1 \alpha_e}{p_d / \delta_1 \alpha_d} \frac{x_e^{1-\eta_e}}{x_d^{1-\eta_d}}
$$

$$\frac{(x_e/\tau_e)^{1-\eta_e}}{(x_d/\tau_d)^{1-\eta_d}} = \frac{p_d}{p_e}\frac{\gamma_1\delta_2}{\gamma_2\delta_1}$$

$$\frac{x_e}{\tau_e} = \left(\frac{p_d}{p_e}\frac{\gamma_1\delta_2}{\gamma_2\delta_1}\right)^{1/1-\eta_e}\left(\frac{x_d}{\tau_d}\right)^{\frac{1-\eta_d}{1-\eta_e}}$$

Therefore, the shadow price of investment k is $p'_k = p_k x^*_k + w\tau^*_k$, where w is the shadow price of time, and it will depend on the genetic endowment of the child.

B Likelihood

In section (3.4) we estimate a Constant Elasticty of Substitution production function. We have the following specification for $\log(H_{i,t})$, the logarithm of Body-Mass-Index of individual i at time t:

$$\log H_{i,t} = \frac{1}{\phi}\log\left[\alpha {I^e_{i,t}}^{\phi} + (1-\alpha){I^d_{i,t}}^{\phi}\right] + (1-\delta)\log H_{i,t-1} + \beta X_{i,t-1} + \varepsilon_{i,t}$$

where $X_{i,t-1}$ contains demographic covariates, as well as mother characteristics. We assume that $\varepsilon_{i,t}$ follows a normal distribution with mean zero and variance σ^2_ε. The associated log-likelihood function is the following:

$$\log \mathscr{L} = \sum_{i=1}^{n}\log\left[\frac{1}{\sqrt{2\pi\sigma^2_\varepsilon}}\exp\left(-\frac{\left(\log H_{i,t} - \frac{1}{\phi}\log\left[\alpha {I^e_{i,t}}^{\phi} + (1-\alpha){I^d_{i,t}}^{\phi}\right] - (1-\delta)\log H_{i,t-1} - \beta X_{i,t-1}\right)^2}{2\sigma^2_\varepsilon}\right.\right.$$

$$= -\frac{n}{2}\log\left(\sigma^2_\varepsilon\right) - \frac{1}{2\sigma^2_\varepsilon}\sum_{i=1}^{n}\left(\log H_{i,t} - \frac{1}{\phi}\log\left[\alpha {I^e_{i,t}}^{\phi} + (1-\alpha){I^d_{i,t}}^{\phi}\right] - (1-\delta)\log H_{i,t-1} - \beta X_{i,t-1}\right)^2$$