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ABSTRACT

Intergenerational Transmission of Inequality: Maternal Endowments, Investments, and Birth Outcomes*

Newborn health is an important component in the chain of intergenerational transmission of disadvantage. This paper contributes to the literature on the determinants of health at birth in two ways. First, we analyze the role of maternal endowments and investments (education and smoking in pregnancy) on the probability of having a baby who is small for gestational age (SGA). We estimate both the total impact of maternal endowments on birth outcomes, and we also decompose it into a direct, "biological" effect and a"choice" effect, mediated by maternal behaviors. Second, we estimate the causal effects of maternal education and smoking in pregnancy, and investigate whether women endowed with different traits have different returns. We find that maternal cognition affects birth outcomes primarily through maternal education, that personality traits mainly operate by changing maternal smoking, and that the physical fitness of the mother has a direct, "biological" effect on SGA. We find significant heterogeneity in the effects of education and smoking along the distribution of maternal physical traits, suggesting that women with less healthy physical constitutions should be the primary target of prenatal interventions.

JEL Classification: I12, I14, J24

Keywords: health production, intergenerational transmission, human capital

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Contents

1 Introduction

Maternal cognitive, social and health endowments are important determinants of prenatal behaviors and an important link in the chain of the intergenerational transmission of inequality. Using UK data from the National Child Development Study (NCDS), the top panel of Figure 1 shows that mothers at the lowest quartile of the distribution of cognitive skills are almost 10 percentage points more likely to give birth to Small for Gestational Age (SGA)[1] babies than mothers in the upper quartile of the distribution.[2] Similarly, mothers with low social skills and mothers with less healthy physical constitution are several percentage points more likely to give birth to small for gestational age babies than their counterparts in the upper tail of the distribution. These maternal endowments can affect newborn health either directly, or through prenatal choices, such as education and smoking.[3] Indeed, the bottom panel of Figure 1 shows that both choices are highly correlated with the probability of giving birth to an SGA baby. These figures, however, only show correlations. In practice, we need to understand causal mechanisms to develop effective policies. That is our aim in this paper.

First, examining the effects of maternal endowments on newborn health is important because it helps us understanding how intergenerational transmission of disadvantage arises. On the one hand, more disadvantaged mothers (Currie (2009), Finch (2003), Kramer et al. (2001)) tend to give birth to babies in poorer health. On the other hand, in a circle of intergenerational transmission of disadvantage, infants with worse health also have worse labor market outcomes, lower test scores, poorer health later in life and even a higher probability of themselves giving birth to babies with worse health. This relationship persists even after controlling for maternal characteristics, environments, and for genetic endowments (Behrman and Rosenzweig (2004), Royer (2009), Oreopoulos et al. (2008), Currie and Hyson

[1] SGA is defined in this paper as being below the 10^{th} percentile of the distribution of birthweight by gestational age. Other definitions have been used, and we show that our results are robust to alternative definitions in Section C.3 in the Appendix.

[2] Endowments here are latent factor scores predicted from measurements of cognitive skill, social skill and physical constitution, respectively. All details of our estimation are reported in Section 2.

[3] Unfortunately, we are unable to examine other maternal prenatal behaviors due to data limitations.

(1999)). In this paper, we study how disadvantage is transmitted from mother to child, and consider low maternal cognitive, social and physical endowments as one particular form of disadvantage.

Critically, we go beyond simply assessing the relationship between maternal endowments and newborn health, to examine the mechanisms behind it. We estimate both the total impact of maternal endowments on birth outcomes, and we also decompose it into a direct, "biological" effect, and into a "choice" effect, which is mediated by maternal behaviors. Additionally, we estimate the causal effects of maternal education and smoking in pregnancy, and we investigate whether women endowed with different traits have different returns.

The paper is structured as follows. We discuss the relationship of our work with the previous literature in Section 2. In Section 3 we present our model and estimation strategy. We describe our data in Section 4, and discuss our results in Section 5. Section 6 concludes.

2 The Relationship of our Work with the Previous Literature

Our work is related to several different literatures. The first strand of literature we draw on is one studying the effects of early endowments. Maternal endowments have been found to be predictive of risky behaviors (Carneiro et al. (2007), Heckman et al. (2006), Conti et al. (2010)) and of child health (Rubalcava and Teruel (2004), Bhargava and Fox-Kean (2003), Currie and Moretti (2007)), both in the economic and in the medical/epidemiologic literature. The latter has mostly focused on the link between maternal birth weight and height and offspring birth weight (Gluckman and Hanson (2004), Subramanian et al. (2009), Kramer (1987)).

However, none of these papers analyzes the mechanisms through which maternal endowments operate. Nonetheless, the importance of accounting for them has long been recognized. For example, Rosenzweig and Schultz (1983) report that not accounting for unobserved parental health endowments leads to a substantial under-estimation of the benefits of early

prenatal care, while Fertig (2010) and Tominey (2007) find that selection explains between a third and a half of the association between prenatal smoking and birth weight.

The second strand of literature we contribute to is the one examining the effects of maternal prenatal behaviors on newborn health. Such literature reports estimates of varying magnitude of the negative effects on the baby, especially for what concerns prenatal smoking behavior. On the one hand, using panel data methods, prenatal smoking has been found to reduce birth weight on average by 150-250 grams (see e.g. Abrevaya (2006); Almond et al. (2005)). On the other hand, using instrumental variable (IV) techniques and maternal fixed effects, the magnitude of the effect is reduced to 50g (Lien and Evans (2005), Rosenzweig and Wolpin (1991), Walker et al. (2009), Abrevaya (2006)). Smoking also increases the probability of low birth weight by 2 to 7 percentage points (Sequí-Canet et al. (2022), Abrevaya (2006), Almond et al. (2005), Evans and Ringel (1999), Tominey (2007), Walker et al. (2009))[4] and reduces the length of gestation by around 0.3-0.7 weeks (Abrevaya (2006), Rosenzweig and Wolpin (1991)). Bharadwaj et al. (2014) analyze a 2004 law change in Norway that extended the smoking ban to restaurants and bars and find that mothers affected by the law had children with a 1.8 percentage point lower incidence of very low birth weight (i.e., less than 1,500 grams at birth) and a 2.5 percentage point lower likelihood of being born pre-term.[5] Also, smoking cessation during pregnancy prevents low birth weight (Yan and Groothuis (2015), Batech et al. (2013), Raatikainen et al. (2007), Harris et al. (2015)).[6]

A third strand of literature our work is related to is the one on the effects of education. Such literature has not reached a consensus yet on the effectiveness of educational policy on health. On the one hand, Currie and Moretti (2003) find that one extra year of maternal education, instrumented with college openings, reduces the probability of having a child with low birth weight by 1 percentage point; this is consistent with what was found in Chou et al. (2010). Also, Liu and Eriksson (2023) use Chinese compulsory schooling reforms

[4]See Di et al. (2022) for a review of the literature.

[5]The likelihood of very low birth weight and pre-term births in the sample are 2.5% and 6.4%, respectively.

[6]See Veisani et al. (2019) for a review of the Randomized Control Trials studies on the effect of prenatal smoking cessation interventions on birth weight.

and find that mothers' educational attainment has significant and sizable positive effects on their children's health, as measured by height, weight, and BMI Z-scores. On the other hand, Carneiro et al. (2013), McCrary and Royer (2011), and Arendt et al. (2021) find no significant impact of education on birth weight.

Currie and Moretti (2003) find that one extra year of education decreases the probability of smoking in pregnancy by 6 percentage points (equivalent to a 30% reduction). An effect of similar magnitude is found in Carneiro et al. (2013), while McCrary and Royer (2011), using age at school entry policies, find less evidence of an effect. It is well known, however, that instrumental variable estimators, at best, only identify a Local Average Treatment Effect (LATE), which measures the effect for those individuals induced into the treatment by the change in the instrument. If the effects of a treatment vary across individuals (given observed variables) and the agents act on the basis of their idiosyncratic returns, then marginal and average ex post returns will not be the same (see McCrary and Royer (2011) for a discussion of this in relation to maternal education and child health).

Methodologically, our approach is close to that in a series of papers by Li and Poirier (Li and Poirier, 2003a,b; Li et al., 2003), in which the authors estimate a structural equations input-output model using Bayesian methods. However, they neither explicitly model maternal endowments, nor the mechanisms through which they affect the health of the newborn.

Our paper also differs from literature on the causes and consequences of health at birth, because we use Small for Gestational Age (SGA), instead of low birth weight, as measure of newborn health.[7] The literature on the long term impact of SGA in the medical field is extensive: SGA has been found to be associated with adult educational attainment, income and also with height (Strauss, 2000), cognitive outcomes in adolescence (Ido et al., 1995), height in adolescence (Frisancho et al., 1994), as well as hypertension at 50 years (Spence et al., 2012). SGA is arguably a better indicator of fetal growth than low birth weight,

[7] Among the papers in economics which control for the time spent in the womb, see Behrman and Rosenzweig (2004) and Oreopoulos et al. (2008).

because it identifies babies who are small even after accounting for the time spent in utero.[8] Indeed, the term SGA is often used interchangeably with Intrauterine Growth Restriction (IUGR) (Meyer et al. (2009), Karlberg et al. (1995)). However, SGA does not necessarily imply IUGR, and vice versa (Lee et al. (2003)).[9]

In the following section we describe how we account for all these mechanisms in a unified modeling and estimation framework, which allows us to investigate the channels through which disadvantage is transmitted through generations.

3 A Model for Maternal Endowments, Investments, and Birth Outcomes

In this paper we estimate a sequential selection model with a factor structure,[10] building on, and expanding, the approach of Hansen et al. (2004), Heckman et al. (2006) and Conti et al. (2010). In our model, women first choose whether to continue education beyond the minimum compulsory level, and then, conditional on the educational choice, they choose their prenatal smoking behavior, which ultimately affects the probability of delivering a SGA baby.[11] We model both maternal choices and newborn outcomes as function of observable characteristics and latent traits. Maternal endowments $\Theta = (\theta_C, \theta_F, \theta_P)$ are composed of

[8] Note that this is effectively one of the advantages of using twin-based designs: since twins have the same gestational age, any difference in birth weight between them is informative about differences in fetal growth.

[9] We re-estimate all the models in our paper using low birthweight instead of SGA, present the full results in Appendix C, and discuss them in the paper when significant differences with the SGA results emerge.

[10] Factor models have become increasingly used in the economics literature (see e.g. Goldberger (1972); Joreskog and Goldberger (1975) and more recently Conti et al. (2010); Heckman et al. (2006, 2018); Piatek and Pinger (2016)).

[11] In a previous version of the paper, we also modeled the pregnancy choice. However, its inclusion does not affect the results, so it has been omitted. This suggests that there are no other (observed or unobserved) determinants which affect the pregnancy choice, outside from those already included in the model.

cognitive (C) and social skills or soft skills (F)[12,13] of the mother, as well as her physical constitution (P).[14,15]

While the inclusion of cognitive and personality traits in economic models of behavior is by now well-established, the use of a maternal factor to capture the biological propensity to have a baby of a certain size (given the constraint imposed by the physical constitution of the mother) is new.[16] Additionally, it is based on a solid economic and medical literature, which establishes that maternal birth weight and height affect the health of the newborn (Lumey and Stein, 1997). For example, using the same data as analyzed here, Hennessy and Alberman (1998) show that maternal physical constitution is a key determinant of SGA. It is by now well documented that conditions in utero are determined by factors going back two generations (Kuzawa and Quinn (2009)) and that maternal phenotype embodies her own cumulative environmental experiences which are in turn transmitted through biological vectors to the fetus, determining its health (Kuzawa, 2005). Consistently with this literature, we use maternal height in childhood and her birthweight, and grandmaternal height, as measurements for this factor, and we interpret it as a proxy for healthy physical constitution, arising from good early environments.[17] Controlling for maternal physical constitution also allows us to interpret the effects of maternal choices on the probability of delivering a small

[12]The letter "S" is used to denote smoking choice later in this section, so we denote social skills by "F" to avoid confusion.

[13]We use the term "social skills" for consistency with the previous literature in economics and for simplicity, but the scale we use more specifically measures behavioral problems and social adjustment, as we will detail in Section 4. Social skills could alternatively be called socio-emotional endowments or social abilities.

[14]Note that the number of latent factors in our model is specified a priori; see Conti et al. (2014) for a model in which the number of latent factors and the measurements they are proxied by is not pre-specified. See also, Zhou et al. (2022).

[15]It would be interesting to estimate the joint maternal and paternal effects on the probability of delivering a SGA baby. However, this is not possible due to the lack of required information on the partners of the mothers in our dataset. Hennessy and Alberman (1998) estimate the marginal effects of maternal and paternal characteristics (such as age, height, birthweight, gestational age, and smoking behavior) on fetal growth and find that the paternal effects were generally weaker than the maternal ones ("15% of the variability in the child's fetal growth can be explained by the mother's characteristics and 7% by the father's"). They acknowledge that these weak paternal effects are likely to be a proxy for those of their partners (for various reasons such as shared environment and assortative mating), rather than direct influences.

[16]See however Conti et al. (2010) for the use of a health factor.

[17]As described in the next section, we also condition on maternal weight, in order to avoid capturing the effect of a higher body mass, which is associated with bigger stature.

for gestational age baby as the result of environmentally-driven growth restrictions, rather than of a biological predisposition.

3.1 The measurement system

We assume that the observed measurements of maternal endowments are a function of observable characteristics X_C, X_F, X_P (proxies for early family environments), and of latent traits Θ=(θ_C, θ_F, θ_P), where θ_C indicates cognitive skills, θ_F social skills and θ_P physical constitution. The measurements are dedicated, but the factors are allowed to be correlated ($Cov(\theta_i, \theta_j) \neq 0$) $\forall\ i \neq j$.[18] These traits are assumed to be fully determined at the time of the measurement (age eleven of the mother) and imperfectly observed by the researcher, but known to the mother.

For individual i and measurement n, the cognitive skill and physical constitution measurement systems are:

$$M_{Cn,i} = X_{Cn,i}\beta_{Cn} + \lambda_{Cn}\theta_{C,i} + \epsilon_{Cn,i} \quad \text{for } n = 1, \ldots, N_C$$

and

$$M_{Pn,i} = X_{Pn,i}\beta_{Pn} + \lambda_{Pn}\theta_{P,i} + \epsilon_{Pn,i} \quad \text{for } n = 1, \ldots, N_P,$$

respectively.

Social skill measures are binary, so we write the measurement system in terms of a latent index structure:

$$M^*_{Fn,i} = X_{Fn,i}\beta_{Fn} + \lambda_{Fn}\theta_{F,i} + \epsilon_{Fn,i} \qquad \text{for } n = 1, \ldots, N_F,$$

[18] Dedicated measurements with correlated factors are widely used in economics (Barón and Cobb-Clark (2010); Conti et al. (2010); Cunha et al. (2010); Heckman et al. (2006)). Our dedicated representation here assumes that cognitive ability does not *directly* affect the measures of social skills. Our measures of social skills are based on teachers' ratings of students' behavior. We acknowledge that, in general, a student's cognitive ability, which affects her performance in class, could influence teachers' perception of the student's behavior. However, as discussed later, our multiple measures of social skills are based on the Bristol Social Adjustment Guide (BSAG) where teachers evaluated children on a range of 146 items of social adjustment, behavior, and attitudes, which were then recoded into 12 so-called 'syndromes'. The detailed, precise evaluation of children's behavior makes it unlikely that our measures of social skills are riddled with measurement errors that are correlated with children's cognitive ability.

$$M_{Fn,i} = I[M^*_{Fn,i} > 0],$$

where $X_{Cn,i}$, $X_{Fn,i}$, $X_{Pn,i}$, are vectors of covariates and λ_{Cn}, λ_{Fn} and λ_{Pn} are factor loadings, for the n^{th} measurement of the cognitive, social skill and physical constitution factors, respectively.[19] M_{Cn}, M_{Fn} and M_{Pn} are sets of measurements specific to each trait $T = \{C, F, P\}$. Continuous measurements are normalized to have mean zero and variance 1. Since the scale of each factor is arbitrary, we set the factor loading in the first measurement equation specific to each factor to unity to set the scale ($\lambda_{C1} = 1$, $\lambda_{F1} = 1$ and $\lambda_{P1} = 1$). Furthermore, we assume $E[\epsilon_{Tn}] = 0 \; \forall \; T$ and $\epsilon_{Tn} \perp\!\!\!\perp \epsilon_{T'm} \; \forall \; m, n$ and $T \neq T'$. Last, to ensure identification we require $N_T > 2$.[20]

3.2 Educational choice

We model the binary choice of obtaining compulsory ($E = 0$) versus post-compulsory education ($E = 1$) using a standard latent index model to characterize the decision rule: $E_i = \mathbb{1}[E^*_i > 0]$, where E^*_i is the net utility derived from post-compulsory schooling, and E_i is the observed choice.[21] Latent utility from education is determined by observable covariates and by the latent factors. We model utility as a linear function of observable covariates and latent factors, with additive separability in the error term:

$$E^*_i = Z_{E,i}\beta_E + U_{E,i}$$

where $Z_{E,i}$ is a vector of observed characteristics and $U_{E,i}$ is a random variable that determine an individual's latent utility from education, and $Z_E \perp\!\!\!\perp U_E$. $U_{E,i}$ is itself a linear and additive function of the latent factors and of an idiosyncratic error term:

$$U_{E,i} = \lambda_{CE}\theta_{C,i} + \lambda_{FE}\theta_{F,i} + \lambda_{PE}\theta_{P,i} + \epsilon_{E,i}.$$

[19] Factor loadings are allowed to differ across equations, so that measurements are given different weights.
[20] Identification of a model with three correlated factors is laid out in Zhou et al. (2022).
[21] So agents select into the treatment if the net utility from doing so is positive.

The idiosyncratic error terms are assumed to be independent of the observables and of the latent factors, i.e. $\epsilon_E \perp\!\!\!\perp (\theta_C, \theta_F, \theta_P, Z_E)$. Furthermore, they are independent of the idiosyncratic error terms of all the other equations (i.e. $\epsilon_E \perp\!\!\!\perp \epsilon_j \; \forall \; j \neq E$).

3.3 Smoking choice

Conditional on education, we then model the binary choice to smoke during pregnancy (S).[22,23] For mothers, this decision is the result of a utility maximization process, where both the health of the newborn and her own consumption of cigarettes are arguments of her utility function.[24] The net utility from smoking for a mother with education level $E \in \{0, 1\}$ is:

$$S_i^{*E} = Z_{S,i}^E \beta_S^E + U_{S,i}^E \quad \text{for } E \in \{0, 1\},$$

where, as before, $Z_{S,i}^E$ is a vector of observed characteristics, and $U_{S,i}^E$ is a random variable which affects the mother's utility from smoking, with $Z_S^E \perp\!\!\!\perp U_S^E$. The latter is specified

[22]Note that the Surgeon General Report on Smoking and Health came out in January 1964, when our mothers were 5 years old. Hence, we assume that the adverse effects of smoking were known by the time they were pregnant with their first child.

[23]We model education and smoking as binary choices. We do not estimate the intensity of prenatal smoking on SGA. Previous papers report mixed results regarding the dose–response effects of smoking on birth outcomes (Kataoka et al. (2018); Osborne and Bailey (2022); Soneji and Beltrán-Sánchez (2019)). Also, the impact of education on health investments may depend on the quality of education, labor market returns to education, and quality of peers, and not the duration of the education per se (Galama et al. (2018)). We abstract from these issues in this paper and leave it as a topic for future research.

[24]The model we have in mind is one like in Rosenzweig and Schultz (1983). Here, the child birth weight (H), in addition to consumption of non-health (X) and health goods (Y), enter the utility function, denoted by $U = U(X, Y, H)$. Newborn health is produced according to $H = \Gamma(Y, I, \mu)$, using health consumption goods (Y), health investment goods (I) and parental endowments μ as inputs. In their framework, maternal endowments are all captured by μ, and maternal smoking is one of the components of Y which enter both the health production function and the utility function.

as: $U^E_{S,i} = \lambda^E_{CS}\theta_{C,i} + \lambda^E_{FS}\theta_{F,i} + \lambda^E_{PS}\theta_{P,i} + \epsilon^E_{S,i}$ for $E \in \{0,1\}$,[25] where, as before, $\epsilon^E_S \perp\!\!\!\perp (\theta_C, \theta_F, \theta_P, Z^E_S)$.[26]

3.4 Birth outcomes

Finally, we model the probability of delivering a baby that is small for gestational age, conditional on the education and smoking choice. We focus on outcomes of firstborn children only, since extending the analysis to higher order parities would require to model the fertility choice (since the decision to give birth to a second child might be a function of the health of the first child), which is beyond the scope of this paper.[27] Let $\mathrm{P}(SGA^{E,S}_i | X^{E,S}_{SGA,i}, \theta_C, \theta_F, \theta_P)$ denote the conditional probability of delivering a SGA baby for the two education and smoking statuses, respectively. Furthermore, assume $SGA^{E,S}_i = \mathbb{1}\left[SGA^{E,S*}_i > 0\right]$, where:

$$SGA^{E,S*}_i = X^{E,S}_{SGA,i}\beta^{E,S}_{SGA} + U^{E,S}_{SGA,i}$$

and

$$U^{E,S}_{SGA,i} = \lambda^{E,S}_{CSGA}\theta_{C,i} + \lambda^{E,S}_{FSGA}\theta_{F,i} + \lambda^{E,S}_{PSGA}\theta_{P,i} + \epsilon^{E,S}_{SGA,i}.$$

for $E \in \{0,1\}$ and $S \in \{0,1\}$, $X^{E,S}_{SGA} \perp\!\!\!\perp U^{E,S}_{SGA}$, $\epsilon^{E,S}_{SGA} \perp\!\!\!\perp (\theta_C, \theta_F, \theta_P, Z^{E,S}_{SGA})$, and $X^{E,S}_{SGA}$ is a vector of observables.[28] The baby SGA outcome for individual i can thus be written in

[25] Note that education and smoking are binary variables and our probit model accounts not only for the direct effects of the cognitive, social, and physical endowments on education and smoking but also for interactions between them. For example, a person's cognitive endowment may have a bigger positive effect on her education if she has a greater social endowment.

[26] In other words, we assume that idiosyncratic error terms in the education equation are independent of the idiosyncratic error terms in all other equations. A person who is a good test taker may find it easier to complete more education. This is captured in our specification through the impacts of cognitive skills, social skills, and physical constitution on test scores and education choices. However, our specification does not allow for unobserved factors other than our three latent factors to affect both skills measures and education choices.

[27] The timing of fertility is another aspect we abstract away from in this paper. See Eshaghnia (2023) for the role of mothers' ages at childbirth on children's health status and cognitive development.

[28] Note that in this paper we focus on education and smoking as the only endogenous investments that affect health at birth. There might be other relevant decisions and behaviors that we abstract away from in this paper. For example, prenatal smoking might be associated with alcohol use or maternal caffeine intake. Our estimates of the detrimental effects of smoking on SGA might be picking up the impacts of these other smoking-related factors too. Nonetheless, some studies that use British data find no significant relationship between a measure of birthweight adjusted for gestational age and maternal caffeine intake (or stress) after controlling for smoking in pregnancy (as well as the mother's height, parity, and sex of the child). They also find that, after smoking was controlled for, alcohol had an effect only among smokers (Brooke et al. (1989)).

switching regression form (Quandt, 1972) as follows:

$$SGA_i = E_i\Big[S_i^1 SGA_i^{1,1} + (1 - S_i^1)SGA_i^{1,0}\Big] + (1 - E_i)\Big[S_i^0 SGA_i^{0,1} + (1 - S_i^0)SGA_i^{0,0}\Big].$$

3.5 Identification and estimation

Identification in our model is based on the following conditional independence assumption:

$$(SGA_0, SGA_1) \perp\!\!\!\perp \Big((E, S) \mid X, Z, \theta_C, \theta_F, \theta_P\Big),$$

where (SGA_0, SGA_1) are potential outcomes for the untreated and treated state, respectively.[29] This is a standard matching assumption, with the difference that a subset of the matching variables (Θ) is imperfectly observed, and instead is proxied by several measurements with error.

We implement the conditional independence assumption estimating the sequential selection model with a factor structure, as described in the previous section, by means of Bayesian methods. However, we also provide evidence from a stepwise approach, where factor scores are estimated in a first step and then used as observed covariates in the choice and outcome models in a second one.[30] As mentioned, we use Markov Chain Monte Carlo (MCMC) methods to estimate the parameters of our sequential selection model.[31] In MCMC estimation, the latent factors are sampled from their joint posterior distribution in each iteration.[32] These draws are then treated as additional data in the estimation of the other model parameters. The estimation follows Carneiro et al. (2003); Hansen et al. (2004) and is described in those papers.[33]

Finally, we make the following assumptions on the latent factors and the error terms:

[29] In practice, we have two sequential treatments and four potential outcomes, however here we refer only to two for simplicity.

[30] Here we apply the method proposed by Iwata (1992) to correct for attenuation bias, and we use bootstrapped standard errors. All the details of this alternative method are provided in Section D in the Appendix.

[31] We prefer Bayesian MCMC methods to classical Maximum Likelihood Estimation (MLE) for its computational convenience.

[32] We run 100,000 iterations in total, of which we discard the first 20,000 as burn-in period, and we then retain one out of 40 of the remaining 80,000.

[33] We use a Bayesian MCMC sampler and code written by Karsten Hansen, Salvador Navarro and George Yates.

1. The distribution of $\Theta = (\theta_C, \theta_F, \theta_P)$ is flexibly approximated by a trivariate mixture with two components.[34] The probability density function is:

$$f_\Theta(\theta_C, \theta_F, \theta_P) \sim p_1 N(\mu_1, V_1) + p_2 N(\mu_2, V_2),$$

where μ_1 and μ_2 are vectors of dimensions (3×1), and V_1 and V_2 are matrices of dimensions (3×3). We do not restrict the variance-covariance matrices to be diagonal, so as previously mentioned we allow the underlying factors to be correlated.

2. The idiosyncratic errors[35] associated with binary choice and outcome models are assumed to be distributed as follows:

$$\epsilon_{E,i}, \epsilon_{S,i}^{E}, \epsilon_{SGA,i}^{E,S}, \epsilon_{Fn,i} \sim N(0,1) \quad \text{for} \quad E \in \{0,1\} \quad \text{and} \quad S \in \{0,1\}.$$

The idiosyncratic errors of the continuous cognitive and physical constitution measurements equations are assumed to be distributed as follows:

$$\epsilon_{Cn,i}, \epsilon_{Pn,i} \sim N(0, \sigma^2) \quad \text{for} \quad n = 1, \ldots, N_C \quad \text{and} \quad n = 1, \ldots, N_P.$$

3. Uninformative normal priors with mean and precision zero are used for all factor loadings.

We can then write the density of outcomes given observables as:

$$f\Big(SGA, E, S, M_C, M_F, M_P \mid X, Z\Big),$$

where $f(.)$ is the joint density of choices, outcomes and measurements. Written in terms of unobservables, the density is:

$$\int \int \int_{\theta_C, \theta_F, \theta_P} f\Big(SGA, E, S, M_C, M_F, M_P \mid X, Z, t_C, t_F, t_P\Big) dF_\theta(t_C, t_F, t_P),$$

[34] It has been shown that mixtures of normals are able to closely approximate any smooth density (Ghosal and Van Der Vaart, 2001).

[35] Defined uniquenesses in relation to the measurement equations of factor models, see Aigner et al. (1984).

where $F_\theta(.)$ denotes the joint cumulative distribution function associated with unobserved cognitive, social and physical endowments. Notice that, conditional on unobserved factors and observed characteristics, (E, S, M_C, M_F, M_P) are independent, and the sample likelihood simplifies accordingly.

4 Data

We use data from the British National Child Development Study (NCDS), which follows a cohort of individuals born in Great Britain during the week of March 3-9, 1958. The female members of this cohort are the mothers we study. The first wave of the NCDS, called the perinatal mortality survey, was administered at birth, and collected a rich array of information on birthweight, gestational age and other birth health conditions, as well as family background characteristics. Subsequent follow-ups were conducted in 1965, 1969, and 1974. Each administered a parental interview, a medical and a school questionnaire, as well as an achievement test battery including measures on cognitive and social skills at ages 7, 11, and 16. Additional follow-ups were conducted in 1981 (age 23), 1991 (age 33), and 2001 (age 43), and administered questions on fertility, partnerships, employment, and children outcomes.

By restricting our sample to all the female cohort members with nonmissing information on the covariates and at least three measurements for each factor, we are left with a sample of 3,217 observations.

4.1 Measurements

The measurements we use to proxy maternal endowments have been collected in the age 11 wave. One of the advantages of the NCDS data is the availability of measures of cognition, personality and health long before the post-compulsory educational choice we model takes place, at a time when the schooling system in the U.K. is still homogeneous.[36]

[36]Then, at age 11 all pupils had to undertake an exam (now abolished almost everywhere) – the so-called "11-plus" – to be admitted to a selective school.

The tests to measure cognition (which can be considered relatively stable at 11 years) were designed by the National Foundation for Educational Research in England and Wales (NFER). They include an IQ-type test with verbal and non-verbal subscales, a 35-items reading comprehension test, and a 40-items arithmetic/mathematics test.

As measures of social skills, we use items from the Bristol Social Adjustment Guide (BSAG).[37] This test evaluates the type and extent of behavioral disturbance in children as rated by the children's teachers, who were asked to indicate whether their pupils scored positively on a range of 146 items of social adjustment, behavior and attitudes. The behaviors were then recoded into 12 so-called 'syndromes': inconsequential behavior, nervous symptoms, anxiety towards adults, anxiety towards children, writing off adults, hostility towards adults, miscellaneous symptoms, restlessness, unforthcomingness,[38] depression and withdrawal.[39] All the items have been recoded, so that the variables we use take the value of 1 in case of absence of a particular syndrome.

Finally, to proxy for the maternal physical constitution factor, we use maternal height at age 11, maternal birth weight, and the adult height of the grandmother. Importantly, we control for maternal weight at age 11 in the measurement system (as described in the next section), to isolate the impact of a healthy physical constitution (reflecting a good early nutritional environment) from that of a bigger body mass which is associated with being taller. We provide evidence of the validity of this factor in capturing the effect of a healthy early nutritional environment when comparing its effects on the probability of delivering a SGA versus a low birth weight baby in Section 5.

Summary statistics for all the measurements are displayed in Table 1. They show, not

[37] We preferred the BSAG over the Rutter scale, which also measures behavioral adjustment problems and was administered at age 11, because this latter was administered to the mother, and Achenbach et al. (1987) have shown that teacher assessments are better predictors of childhood problems than parental ones. Achenbach et al. (1987) estimated correlations between child behavioral problems and assessments of teachers, parents, health visitors, and of the children themselves, and found them to be higher for teachers.

[38] Unforthcomingness describes a collection of behaviors characterizing a child's unassertiveness, interpersonal and academic passivity, and avoidance of competition (McDermott and Watkins, 1981).

[39] Withdrawal describes behaviors reflecting general social detachment or induced isolationism (McDermott and Watkins, 1981).

surprisingly, that women who have chosen to continue education beyond the compulsory level and not to smoke in pregnancy are endowed with better traits under all the different dimensions.

4.2 Outcome variables and covariates

Our main outcome of interest is the probability of having, by the age of 33,[40] a firstborn child who is small for gestational age. As previously argued, we believe SGA to be a more appropriate measure of newborn health than low birth weight, as reflecting in utero conditions: conditioning on gestational age allows to identify those children who are small not because born early, but because restricted in their growth.[41]

An obvious issue which arises when using SGA, however, is the presence of measurement error, since at the time the survey was carried out, the use of ultrasounds to check accuracy was still not diffused. In the NCDS data, gestational age is computed based on the mother's self-report of the date of her last menstrual period (LMP), which is then checked against general practitioner records (Jefferis et al., 2002). This recall problem has been pointed out both in the medical (Campbell et al. (1985); Chervenak et al. (1998); Harville et al. (2010); Kramer et al. (1988)) and in the economics literature (Royer, 2009). However, error in recall is mostly problematic for gestational periods longer than 42 weeks and for very early deliveries, while for deliveries within the 37-41 weeks range the accuracy has been shown to be high (Poulsen et al. (2011), Wingate et al. (2007), Mustafa and David (2001)). Therefore, in order to minimize measurement error, we restrict our sample to deliveries occurred between 26 and 42 weeks of gestation; in doing so, we retain 97% of the women.[42] The distribution of gestational age is calculated using a growth chart published by Fenton

[40] In our sample, 75% of the women have given birth at least once by this age, and, given the historical period of our sample, this reasonably approximates all women who will ever give birth.

[41] As already mentioned, we re-estimate all the models in our paper using low birthweight instead of SGA. While all the results are presented in Appendix C, in the text we only refer to them when they significantly differ from those using SGA.

[42] Results for the complete sample are very similar and are available upon request.

(2003)[43] in BMC Pediatrics, which is based on a meta-analysis of published reference studies. We adopt the most common definition used in the literature and define a baby to be SGA if she lies below the 10^{th} percentile of the birthweight by gestational age distribution. Table 2 shows that 14% of the children in our sample are small for gestational age; this relatively high prevalence is caused in part by the fact that firstborn[44] children are, ceteris paribus, more likely to be SGA than later born children (see e.g. Shah (2010) for a meta study on the association between parity and pregnancy outcomes). We provide extensive robustness tests in Appendix C (by restricting the sample to full-term babies and adopting different definitions of SGA) which confirm our main results.

The educational decision (derived from the 1981 questionnaire) is defined as staying-on after the minimum compulsory school-leaving age. We see from Table 2 that in our sample about 35% of the women have continued schooling beyond age 16, and that this percentage is halved among the smokers. Finally, the decision to smoke during pregnancy after the first trimester[45] is also derived from the 1981 questionnaire, so it is asked retrospectively. While we are not worried about selective recall bias, still there is the possibility of women "lying" to the interviewer; however, if present, this should bias our results downwards.

The covariates we include in the measurement equations to control for family background characteristics are specified in Table 3.[46] We also include additional variables in the choice equations: regional smoking prevalence at the time the mother was pregnant and whether the grandmother was herself a smoker at the time she was pregnant for the smoking choice, and local labor market conditions (the change in the unemployment rate) in the region of residence at the time of the educational decision. Summary statistics for the covariates and outcomes of our model are reported in Table 2.

[43] The chart is the most recent version of Babson and Benda (1976), which is the most common chart used in neonatology reference books. Different specifications using different charts or using the 5^{th} percentile to define SGA did not change the results in any significant way, as documented in Appendix C.3.

[44] As mentioned above, we focus on firstborn children only.

[45] Women might not know they are pregnant in the first weeks of gestation, so smoking might not be a choice then.

[46] Note that some covariates are excluded from the choice and outcome equations due to insufficient variation to identify their effects, given the smaller sample sizes in the conditional models.

5 Empirical Results and Simulations

We now present our results. In this section we first provide the estimated posterior means of the coefficients and we describe our simulation algorithm (Section 5.1). We then present and comment the results of the simulation exercise in three parts. First, we present how mothers sort into education and smoking decisions on the basis of their traits, and also the effects of these traits on the choices (Section 5.2). Second, we show in Section 5.3 the results from our decomposition exercise of the overall effects of the maternal traits (displayed in the top panel of Figure 1) into the channels through which they operate. Third, we present the treatment effects of education and smoking (Section 5.4), we decompose the observed SGA differences by choice (displayed in the bottom panel of Figure 1) into selection and causal components, and we examine heterogeneity in the treatment effects.

5.1 MCMC results and Simulation Algorithm

The posterior means of the factor loadings in the measurement system, which reflect the correlation between the measurements and the latent factors, are reported in Table 4. Notice that the copying designs test contributes to the cognitive factor less than the other tests; that the social skills factor is mostly reflecting lack of hostile behavior rather than of anxious one;[47] and that the maternal physical constitution factor has the loading with the biggest magnitude on maternal height at age 11.

We then use the estimated distributions of parameters and the data[48] to simulate counterfactual choices and outcomes, in order to shed more light on the effects of maternal traits, the mechanisms through which they operate, and the treatment effects of smoking and education. More specifically, the Bayesian MCMC algorithm we use for estimation generates a sample of size K model parameters from their conditional posterior distributions that we can

[47] The items have been recoded, so that they take the value of 1 in case of the absence of a particular syndrome.

[48] We randomly draw with replacement individuals from our sample. This randomly drawn data has the same sample size as the actual data. Then, for each individual, we make $K = 10$ draws from the posterior distributions of the parameters, so to take estimation uncertainty into account.

use to simulate (binary) outcomes for each individual according to the following expression:

$$\begin{aligned}\hat{y}_i &= \frac{1}{K}\sum_{k=1}^{K} \Pr\left(U^*(k)_{y,i} \geq 0 \mid X_{y,i}, \beta_y(k), \lambda_y(k), \Theta_i(k)\right) \\ &= \frac{1}{K}\sum_{k=1}^{K} \mathbb{1}\Big[(X_{y,i}\beta_y(k) + \lambda_y(k)\Theta_i(k) + \epsilon) > 0\Big],\end{aligned} \tag{1}$$

where $\mathbb{1}$ denotes the indicator function and ϵ is drawn from a normal distribution. Furthermore, given independence between the factors and the covariates, we can simulate outcomes for each individual at each percentile of the factor distribution, according to the following:

$$\hat{y}_{i_{\theta_p},} = \frac{1}{K}\sum_{k=1}^{K} \mathbb{1}\Big[(X_i\beta(k) + \lambda(k)\Theta_{p,i}(k) + \epsilon) > 0\Big] \quad \text{for } p = 1, \ldots, 99. \tag{2}$$

Furthermore, we can use the predicted probabilities from the choice equations to weight the predicted outcomes, so as to generate expected outcomes for each individual, conditional or unconditional on choices.

The first thing we do using these simulated outcomes is to assess whether our model provides a good fit to the data. To do so, we compare the simulated means of the education and smoking decisions, and of child SGA, to the ones in the data. As shown in Table 5, the means are not statistically significantly different from each other; additionally, also the proportion of outcomes correctly predicted by the model is remarkably high, especially considering the small number of observations we have in the conditional SGA equations.

5.2 The Intergenerational Effects of Maternal Traits

In order to examine the effects of maternal traits, we first compute and report the average marginal effects of a one standard deviation change in each of them on the outcomes. As shown in Table 6, both cognitive and social skills are significant determinants of the educational choice. However, social skills are the only traits which are significant determinants of the smoking choice. Lastly, neither cognitive nor social skills are significant determinants of the probability of delivering a SGA baby, after conditioning on the education and smoking choices: the physical constitution of the mother, nonetheless, remains a strong determinant,

in particular for women with a low level of education. Importantly, this is no longer the case when we use low birth weight rather than SGA as measure of newborn health: this suggests that our maternal physical constitution factor is indeed capturing the capacity of the mother to deliver a healthy baby, rather than the sheer size of her body (see Table C-1 in the Appendix for the LBW results).

In order to understand better the sorting decisions of the mothers, Figure 2 and Figure 3 display the marginal distributions of cognitive, social skills and physical constitution, conditional on the education and smoking choices.[49] We first notice that the spread of the distributions of the three endowments is different: the standard deviations amount to 0.84 for cognition, 1.62 for social skills, and to 0.35 for the maternal physical constitution factor. This pattern has implications for considering the impacts of interventions aimed at changing the relative position of individuals along these distribution. Second, we see that mothers with higher cognition, better social skills and a healthier physical constitution sort into post-compulsory education, as confirmed by a Kolmogorov-Smirnov test. Additionally, women with a higher level of cognition and better social skills decide not to smoke after the third trimester of pregnancy; maternal body fitness, instead, seems to play no role. As mentioned in Section 3, we also estimate the model using a stepwise approach, in which factor scores are computed in a first step and then used as observed covariates in the choice and outcome models in a second step.[50] As we can see in Table 8, the results we obtain are remarkably similar to those obtained using Bayesian estimation.

Finally, simulated decisions and outcomes by quantiles of the endowments distributions are displayed in Figure 4, Figure 5 and Figure 6. As compared to what seen before, they allow us to assess the effects of an exogenous shift in each maternal endowment in turn at different parts of their distribution (so to detect nonlinearities in those effects), while holding the

[49]The unconditional joint distributions are presented in Figure B.1, Figure B.3 and Figure B.5 in the Appendix. The correlations among traits are displayed in Table 7. We see that the correlation between cognitive and social skills is 0.369, the one between the cognitive and the physical traits is 0.172, while maternal social and physical traits are basically uncorrelated.

[50]This procedure is described in details in Section D in the Appendix.

other two at their means, rather than the average marginal effect of one standard deviation change (as seen in Table 6). Additionally, we compute the effects of maternal endowments on choices and outcomes, rather than the conditional ones.[51] Figure 4 shows that women with higher cognitive skills are more likely to obtain post-compulsory education. In fact, an early childhood intervention which moves a girl from the 20^{th} to the 80^{th} percentile of the cognitive skill distribution (holding the other two endowments at the mean), would increase her probability of obtaining post-compulsory education from 20% to around 50%. For social skills and physical constitution the effect is of a smaller magnitude, but still significant.[52]

Second, the decision to smoke during pregnancy is predominantly influenced by social skills, as can be seen from Figure 5. We find that an intervention which moves a girl from the 20^{th} to the 80^{th} percentile of the social skills distribution (holding the other two endowments at the mean), would halve her probability to smoke during pregnancy (from 0.30 to 0.15), while a shift of the same magnitude along the distribution of cognitive skills would reduce this probability only by 0.05.[53]

Third, Figure 6 displays the overall effect of maternal traits on the probability of giving birth to a small-for-gestational-age baby.[54] We find that an early nutritional intervention which moves a girl from the 20^{th} to the 80^{th} percentile of the distribution of her physical constitution more than halves the probability of delivering a SGA baby (from 0.20 to less than 0.10). Importantly, we find a much smaller effect when using low birthweight as measure of newborn health: a similar intervention would reduce the probability of delivering a LBW baby by only 3 (rather than 10) percentage points (see Figure C-1 in the Appendix). This reassures us about the validity of our measures of SGA as proxying for fetal health, and of

[51] See the next section for a decomposition of the total effect into a direct and an indirect one, i.e. one working through the two channels of education and smoking.

[52] This latter result is consistent with the literature on height and education (Case and Paxson (2010); Magnusson et al. (2006)).

[53] On the other hand, the effects of an early nutritional intervention arising from a movement of a similar magnitude on the woman's physical constitution are not precisely estimated.

[54] In order to compute the overall effects of skills on child SGA we integrate education and smoking variables as well as covariates out, according to $P(SGA|\Theta = \theta_p) = \int_X \sum_{E=0}^{1} \sum_{S=0}^{1} P(SGA|\Theta = \theta_p, X = x, E = e, S = s) P(S = s|X = x, \Theta = \theta_p, E = e) P(E = e|X = x, \Theta = \theta_p) dF_x$.

maternal physical traits as capturing the fitness of the mother in delivering healthy babies. Then, the corresponding effects of interventions to promote cognitive or social skills are much smaller, with a comparable shift (from the 20^{th} to the 80^{th} percentile) in the respective distributions only yielding a 1 percentage point reduction in the probability of delivering a SGA baby, which is also less precisely estimated.[55] Interestingly, we detect nonlinearities in the effects of maternal endowments, with the bigger gains occurring for women at the bottom of their respective distributions.

In the next subsection we then decompose the effects of maternal endowments on the probability of having a SGA baby into the components which operate through the education and smoking channels, and a residual direct effect.

5.3 Understanding the Mechanisms through which Maternal Endowments Affect Newborn Outcomes

In order to describe our decomposition exercise, we first establish some notation. Let the conditional probabilities of giving birth to a SGA baby, of smoking during pregnancy (S) and of staying on in education beyond the compulsory level (E) be written respectively as:

$$P\Big(SGA = 1 \mid X = x, \Theta = \theta, E = e, S = s\Big); \tag{3}$$

$$P\Big(S = 1 \mid X = x, \Theta = \theta, E = e\Big); \tag{4}$$

$$P\Big(E = 1 \mid X = x, \Theta = \theta\Big), \tag{5}$$

where X is a vector of predetermined characteristics (which include different subsets of variables, as detailed in Table 3, and Θ is the vector of the three maternal endowments.

We first compute these probabilities using simulation (as described in the previous section), and then we apply the product rule to disentangle how a change in maternal endow-

[55] The attentive reader might have noticed that the effect of physical traits on the probability of smoking in pregnancy is much smaller than the one estimated in Table 6. The reason is the small and positive correlation between the cognitive and physical traits, which have opposite effects on smoking behavior – so that, when they are both allowed to vary (like in Table 6), they counterbalance each other out.

ments (θ_t) affects the newborn SGA through the different channels, as follows:

$$\overbrace{\frac{\partial P\left(SGA=1|X=x,\Theta=\theta\right)}{\partial\theta_t}}^{\text{Total effect}}$$

$$=\overbrace{\sum_{S=0}^{1}\sum_{E=0}^{1}P(S=s|X=x,\Theta=\theta,E=e)P(SGA=1|X=x,\Theta=\theta,E=e,S=s)}^{\text{Effect through education}}$$

$$\times\quad\frac{\partial P(E=e|X=x,\Theta=\theta)}{\partial\theta_t}$$

$$+\overbrace{\sum_{S=0}^{1}\sum_{E=0}^{1}P(E=e|X=x,\Theta=\theta)P(SGA=1|X=x,\Theta=\theta,E=e,S=s)}^{\text{Effect through smoking}}$$

$$\times\quad\frac{\partial P(S=s|X=x,\Theta=\theta,E=e)}{\partial\theta_t}$$

$$+\overbrace{\sum_{S=0}^{1}\sum_{E=0}^{1}P(E=e|X=x,\Theta=\theta)P(S=s|X=x,\Theta=\theta,E=e)}^{\text{Residual effect}}$$

$$\times\quad\frac{\partial P(SGA=1|X=x,\Theta=\theta,E=e,S=s)}{\partial\theta_t}.$$

where $t=C,S,P$, alternatively. This formula shows that maternal traits can affect the newborn SGA in three ways: indirectly through the education (summand 1) and smoking (summand 2) choices, and directly (summand 3).[56]

[56] In practice we compute average partial derivatives, and investigate the impact of a one standard deviation change in the respective factor. Note that we could also investigate a large movement along the factor distribution using finite differences (i.e. neglecting cross-differences), according to the following formula:

$$\begin{aligned}
\Delta_\theta P\Big(SGA=1\mid X=x,\Theta=\theta\Big)=\sum_{S=0}^{1}\sum_{E=0}^{1}\Big[&P(S=s\mid X,\Theta,E=e)\times P\Big(SGA=1\mid X=x,\Theta=\theta,E=e,S=s\Big)\\
&\times\quad\Delta_\theta P\Big(E=e|X=x,\Theta=\theta\Big)\Big]\\
+P\Big(E=e\mid X=x,\Theta=\theta\Big)&\times P\Big(SGA=1\mid X=x,\Theta=\theta,E=e,S=s\Big)\times\Delta_\theta P\Big(S=s\mid X=x,\Theta=\theta,E=e\Big)\\
+P\Big(E=e\mid X=x,\Theta=\theta\Big)&\times P\Big(S=s\mid X=x,\Theta=\theta,E=e\Big)\times\Delta_\theta P\Big(SGA=1\mid X=x,\Theta=\theta,E=e,S=s\Big)\\
+P\Big(S=s\mid X=x,\Theta=\theta,E=e\Big)&\times\Delta_\theta P\Big(E=e\mid X=x,\Theta\Big)\times\Delta_\theta P\Big(SGA=1\mid X=x,\Theta=\theta,E=e,S=s\Big)\\
+P\Big(E=e\mid X=x,\Theta=\theta\Big)&\times\Delta_\theta P\Big(S=s\mid X=x,\Theta=\theta,E=e\Big)\times\Delta_\theta P\Big(SGA=1\mid X=x,\Theta=\theta,E=e,S=s\Big)\\
+P\Big(SGA=1|X=x,\Theta=\theta,E=e,S=s\Big)&\times\Delta_\theta P\Big(E=e|X=x,\Theta=\theta\Big)\times\Delta_\theta P\Big(S=s|X=x,\Theta=\theta,E=e\Big)\\
+\Delta_\theta P\Big(E=e\mid X=x,\Theta=\theta\Big)&\times\Delta_\theta P\Big(S=s\mid X=x,\Theta=\theta,E=e\Big)\times\Delta_\theta P\Big(SGA=1\mid X=x,\Theta=\theta,E=e,S=s\Big)\Big].
\end{aligned}\tag{6}$$

The results of this decomposition exercise are presented in Table 9 and Figure 7. We find that 70% and 95% of the overall effect on newborn SGA of cognitive and social endowments, respectively, passes through choices. More specifically, 68% of the overall effect of cognitive skills passes through the channel of education, and only 2% of it works by affecting smoking behavior. Moreover, 30% of the overall effect of cognitive skills on newborn SGA is a residual, i.e. it is likely to work through other behaviors not included in our model, such as prenatal nutrition.[57] Then, 64% of the effect of social skills works through the smoking choice, while 30% through education. On the contrary, maternal physical constitution has a direct, "residual" effect on newborn health, i.e. it is not mediated by education or smoking decisions. Figure 7 displays the same information, but in terms of percentage points reduction in the probability of having a SGA baby, and the respective contributions of the three maternal endowments to it. Importantly, when we repeat the decomposition exercise using LBW as outcome (Figure C-2 in the Appendix), we find a significant residual effect of both cognition and social skills on low birth weight. The lack of biological plausibility of this result reassures us again about our choice of outcome as more genuinely capturing the rate of fetal growth.

We now move to the estimation of a variety of treatment effects of education and smoking in the next section.

5.4 The Treatment Effects of Education and Smoking

In this section, we investigate the causal impact of education and smoking on the probability of having a baby that is small for gestational age. First, we compute the average treatment effects (ATE) of education and smoking, which can alternatively be interpreted as the average effect of the treatment for a person randomly selected from the population, or as the expected change in the average outcome if the treatment was exogenously given to every individual.[58]

[57] In other words, smarter women might have healthier babies because of better nutrition. Unfortunately, we are unable to model other maternal behaviors due to data limitations.

[58] In the following we present the formal definitions of treatment effects and we describe the details of their estimation only for the effect of education on newborn SGA. The effects of education on smoking and of smoking on child SGA can be defined analogously.

Formally, the ATE of maternal education on newborn SGA is defined as:

$$E\Big[SGA^{E=1} - SGA^{E=0}\Big] = \int\int E\Big[SGA^{E=1} - SGA^{E=0} \mid X, \Theta\Big] dF_{X,\Theta}, \tag{7}$$

where $SGA^{E=1}$ and $SGA^{E=0}$ denote the potential outcomes in terms of the probability of delivering a SGA baby, for mothers with and without post-compulsory education.

The problem of causal inference states that both potential outcomes are never observed for a single individual, but only either $(SGA^{E=1}|X, \Theta, E = 1)$ or $(SGA^{E=0}|X, \Theta, E = 0)$. Here we make use of the following matching assumption $\mathrm{E}\big[SGA^{E=0}|X, \Theta, E = 0\big] = \mathrm{E}\big[SGA^{E=0}|X, \Theta, E = 1\big]$ to simulate counterfactual outcomes and estimate treatment effects. We allow for mismeasured matching variables. In practice, using draws from the posterior distributions of the parameters and of the factors, as well as from the covariates (X) information from our sample, we compute the ATE as follows:

$$\begin{aligned} &\mathrm{E}\Big[\widehat{SGA^{E=1}} - \widehat{SGA^{E=0}}\Big] \\ &= \frac{1}{NK}\sum_{k=1}^{K}\sum_{i=1}^{N}\Big[\hat{m}^{e=1}(X_i, \Theta_i(k); \Psi(k)) - \hat{m}^{e=0}(X_i, \Theta_i(k); \Psi(k))\Big] \end{aligned} \tag{8}$$

where Ψ denotes a vector of all parameters but the factors, and $\hat{m}^e$ denotes the model prediction of $m^e(X, \Psi) = \mathrm{E}[SGA|X, \Theta, E = e]$ and N denotes the number of individuals in the sample.

The second treatment effect we estimate is the Average Treatment effect on the Treated (ATT). This measures the impact of the treatment on an individual drawn at random from the population and actually been treated (in our case, who selected into education). The

ATT of maternal education on newborn SGA is thus defined as:[59]

$$E\Big[SGA^{E=1} - SGA^{E=0} \mid E=1\Big] = \int\int E\Big[SGA^{E=1} - SGA^{E=0} \mid X, \Theta, E=1\Big] dF_{X,\Theta|E=1},$$

We compute the ATT as follows:

$$\begin{aligned} &\mathrm{E}\Big[\widehat{SGA^{E=1}} - \widehat{SGA^{E=0}} | E=1\Big] \\ &= \frac{1}{N_1 K} \sum_{k=1}^{K} \sum_{i:e=1}^{N_1} \Big[\hat{m}^{e=1}(X_i, \Theta_i(k); \Psi(k)) - \hat{m}^{e=0}(X_i, \Theta_i(k); \Psi(k))\Big]. \end{aligned} \tag{9}$$

Lastly, we define and estimate the average marginal treatment effect (AMTE), which is the average effect of the treatment for the marginal person, i.e. the person who is indifferent between participation ($E=1$) and nonparticipation ($E=0$) into the treatment (Carneiro et al., 2010). The AMTE is defined as:

$$\begin{aligned} &E\Big[SGA^{E=1} - SGA^{E=0} \mid |Z_E\beta_E + U_E| < \epsilon\Big] \\ &= \int\int E\Big[SGA^{E=1} - SGA^{E=0} \Big| X, \Theta, \mid Z_E\beta_E + U_E\Big| < \epsilon\Big] dF_{X,\Theta}. \end{aligned} \tag{10}$$

with Z_E and U_E being the observed and unobserved determinants of the educational choice, as defined in Section 3, and ϵ close to zero.[60] The AMTE is computed as follows:

$$\begin{aligned} &\mathrm{E}\Big[\widehat{SGA^{E=1}} - \widehat{SGA^{E=0}} | \, |Z_E\beta_E + U_E| < \epsilon\Big] \\ &= \frac{1}{N_{mar}} \sum_{k=1}^{K} \sum_{i:mar}^{N_{mar}} \Big[\hat{m}^{e=1}(X_i, \Theta_i(k); \Psi(k)) - \hat{m}^{e=0}(X_i, \Theta_i(k); \Psi(k))\Big]. \end{aligned} \tag{11}$$

where *mar* denotes the marginal individual.

[59] The Average Treatment Effect on the Non-Treated (ATNT) can be analogously defined as:

$$E\Big[SGA^{E=1} - SGA^{E=0} \mid E=0\Big] = \int\int E\Big[SGA^{E=1} - SGA^{E=0} \mid X, \Theta, E=0\Big] dF_{X,\Theta|E=0},$$

and can be computed as follows:

$$\frac{1}{N_0 K} \sum_{k=1}^{K} \sum_{i:e=0}^{N_0} \Big[\hat{m}^{e=1}(X_i, \Theta_i(k); \Psi(k)) - \hat{m}^{e=0}(X_i, \Theta_i(k); \Psi(k))\Big]$$

[60] The value chosen for ϵ is 0.3, with the net utility taking values from -6 to $+6$. Using this metric, 15% of individuals in our sample are defined to be "at the margin".

The estimated treatment effects are reported in Table 10.[61] The important message that we learn from this table is that the various average treatment effects are not statistically significantly different from each other, suggesting the absence of evidence of sorting on health gains: in other words, when deciding whether to continue education beyond the compulsory level, or whether to smoke in pregnancy, women do not take the health of the baby into account. Additionally, when we use LBW rather than SGA as outcome (see Table C-2 in the Appendix), we find treatment effects of smoking which are half the magnitude, once more reassuring us of our choice of using SGA as a better indicator of health at birth.

In order to gain a better understanding of the magnitude of these average treatment effects, we now decompose the difference in means between the outcomes of treated and untreated women, shown in the bottom panel of Figure 1. The observed difference in the occurrence of SGA between high and low educated mothers can be decomposed into average treatment effect, sorting gain and selection bias according to:[62]

$$\overbrace{\mathrm{E}[SGA|E=1]-\mathrm{E}[SGA|E=0]}^{\text{observed difference}}=$$
$$ATE+\overbrace{(ATT-ATE)}^{\text{sorting gain}}+\overbrace{\mathrm{E}\left[SGA^{E=0}|E=1\right]-\mathrm{E}\left[SGA^{E=0}|E=0\right]}^{\text{selection bias}}.$$

The sorting gain is the difference between the ATE and the ATT, and it is informative as to whether individuals sort into a treatment based on their expectations about their idiosyncratic gains or losses. The results of this decomposition exercise are presented in Table 11 and graphically displayed in Figure 8. We find stark differences between the two treatments. On the one hand, the differences in prenatal smoking behaviors and newborn health that we observe between high- and low-educated women are only partially due to education: selection accounts for between 40% and 60% of this difference. On the other hand, the difference in the probability of delivering a SGA baby observed between women

[61] We can also calculate partial treatment effects, for example by fixing the education treatment assignment when calculating the average treatment effect of the smoking treatment: $\mathrm{E}\left[SGA^{S=1}_{E=1}\right]-\mathrm{E}\left[SGA^{S=0}_{E=1}\right]$. For the sake of brevity these results are not presented here.

[62] The observed difference in smoking between high- and low-educated women, and in the occurrence of SGA between smokers and non-smokers, can be defined analogously.

smoking and non-smoking in pregnancy (which amounts to 11 pp) can be entirely attributed to the harmful effects of smoking. Although we do not decompose it, it is worth noticing that the selection bias component reflects differences in the distributions of observables and latent endowments, as well as differences in their support.[63]

5.5 Treatment Effect Heterogeneity

We now go beyond mean impacts and investigate whether education and smoking policies have different effects along the distribution of maternal endowments. This is important, since, when allocating public resources under constraints, policy makers will need to know who benefits the most from a given policy, so to allow a more effective targeting. In practice, we compute average treatment effects for individuals at different quantiles of the distributions of each of their latent traits in turn, while fixing the other two at their mean value. For example, to calculate the effect of education on the probability of delivering a SGA baby for a woman with an average endowment of physical and social traits, but below the 20^{th} percentile of the cognitive skills distribution, we compute:

$$\mathrm{E}\Big[\widehat{SGA^{E=1}} - \widehat{SGA^{E=0}}\Big] = \frac{1}{NK}\sum_{k=1}^{K}\sum_{i=1}^{N}\Big[\hat{m}^{e=1}\Big(X_i, \theta_C(p_{0-20})(k), \bar{\theta}_F(k), \bar{\theta}_P(k); \Psi(k)\Big) - \hat{m}^{e=0}\Big(X_i, \theta_C(p_{0-20})(k), \bar{\theta}_F(k), \bar{\theta}_P(k); \Psi(k)\Big)\Big]$$

The results of this exercise are presented in Table 12 and displayed in Figure 9. We make several observations. First, although not by a large magnitude, the treatment effect of education on smoking is bigger at the top of the distribution of each of the maternal endowments. However, we find no significant evidence of heterogeneity in the effect of education and of smoking on the probability of delivering a SGA baby along the distribution of cognitive and social skills. This is a relevant difference with respect to the results we obtain

[63] Support differences in the trait distributions between treated and nontreated individuals become apparent when looking at Figure 2 and Figure 3. Heckman et al. (1997) show that the selection bias component can be further decomposed into components due to differing supports of X and Θ for the treated and nontreated groups, into differing distributions of X, Θ over the same support in the two groups, and into differences in outcomes that are present even after controlling for observables and unobservables.

when using low birth weight instead of SGA as measure of newborn health (see Figure C-3 in the Appendix), where we find that the treatment effect of education on the probability of delivering a low birth weight baby is significantly higher at the *bottom* of the maternal traits distribution. Nonetheless, we find significant evidence of treatment effect heterogeneity along the distribution of physical traits, which suggests the presence of complementarities. On the one hand, we find that the effect of education in reducing the probability of having a SGA baby is bigger for mothers with a healthier physical constitution. On the other hand, smoking during pregnancy has a much more detrimental effect for mothers with a poor physical constitution; this effect declines along the distribution, and it only has a small and insignificant impact for females who are physically very fit.[64] Interestingly, these women are also more likely to choose smoking during pregnancy (see Figure 5).[65] This finding is consistent with a model where there is heterogeneity in health endowments and women sort into utility-generating risky prenatal behaviors by acting upon knowledge of the consequences of their choices: in other words, those women with a better physical constitution are both more likely to smoke, and at the same time less likely to suffer from the adverse consequences of their behavior, in terms of a reduction in their newborn's health.[66] Our results have important policy implications: they suggest that anti-smoking policies should target women who are physically more prone to have smaller babies, since these are the women whose newborns suffer most from prenatal smoking.

[64]Our results are consistent with complementarities between education and physical constitution in producing better birth outcomes. Previous studies document the positive effect of education on the demand for health investment, even after controlling for wage income (Grossman (2017); Kitagawa and Hauser (1973)). Our results are also consistent with the findings of previous papers that individuals with higher endowed health have a higher compensating premium for participating in activities detrimental to their health (Ehrlich and Chuma (1990)).

[65]Note that both results have been obtained while fixing the other two endowments at their means, in turn.

[66]See, for example, Rosenzweig and Schultz (1983).

6 Conclusions and Policy Implications

This paper presents new evidence on the impact of maternal cognitive, social and physical endowments on newborn health. We use a sequential selection model with a factor structure to analyze the role of maternal endowments and investments (education and smoking in pregnancy) on the probability of having a baby who is small for gestational age (SGA). We estimate the total impact of maternal endowments on birth outcomes, and we also decompose it into a direct, "biological" effect and a "choice" effect, mediated by maternal behaviors. Using this model, we estimate the causal effects of maternal education and smoking in pregnancy. We investigate whether women endowed with different traits have different returns. We find that cognition affects birth outcomes primarily through education, that personality traits mainly operate by changing smoking behavior, and that the physical fitness of the mother has a direct, "biological" effect on SGA. We also find significant heterogeneity in the effects of education and smoking along the distribution of maternal physical traits, suggesting that women with a less healthy physical constitution should be the primary target of prenatal interventions.

What is then the most effective policy to close the SGA gap by education (4 percentage points) and smoking (11 percentage points)? On the one hand, our results show that a one standard deviation increase in cognitive, social and physical endowments reduce the probability of giving birth to a SGA baby by around 1, 0.5 and 6.5 percentage points, respectively. Additionally, we find that these endowments operate through different mechanisms: cognitive ability predominantly through education, social skills by affecting the smoking choice, while the physical constitution of the mother directly affects the health of the newborn. Indeed, our decomposition exercise has shown that 70% and 95% of the gap in SGA between women at the bottom and at the top quartiles of the cognitive and social endowments distributions, respectively, is explained by maternal choices. Figure 10 shows that, once we condition on them, no gap in the probability of delivering a SGA baby remains. On the contrary, the SGA differential between women endowed with a poor and with a healthy con-

stitution increases once we condition on the prenatal smoking choice. This occurs because the physical constitution of the mother has both a direct, positive effect on the health of the newborn, and it is also positively associated with her prenatal smoking decisions. On the other hand, we estimate that the average treatment effect of continuing education beyond the minimum compulsory leaving age and of smoking after the third trimester of pregnancy on the probability of delivering a SGA baby are -1.6 and 11 percentage points, respectively.

These results allow us to compare the effectiveness of three different policies in reducing the prevalence of SGA: an early childhood intervention providing both a stimulation and a nutritional component, an educational policy, and a smoking cessation intervention. In Table 13, we compare them by computing the change in terms of standard deviations of the endowments distributions that an early childhood intervention would have to achieve in order to cause the same change in the prevalence of SGA as an educational policy or a smoking cessation intervention.[67] Nutritional intervention that improves the physical constitution of the mother appears to be a very effective option. Furthermore, when analyzing heterogeneity in the treatment effects, we find evidence of substitutability. A prenatal anti-smoking intervention is more effective (in terms of reduction in the probability of delivering a SGA baby) for those women with a poor physical constitution. Of course, a comparison of the overall benefits of the two interventions, and of their respective costs, is necessary before drawing any definite conclusion. However, these results suggest that prenatal interventions, such as home-visiting programs, aimed at (among other things) reducing the prevalence of risky behaviors in pregnant women, and targeting low-income mothers, seem to be an effective way to compensate for maternal endowments differentials, and to guarantee a healthy start of life for the next generation.

[67] So, for example, it shows that an educational policy can achieve the same outcome as an intervention which raises IQ by 1.8 SD.

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7 Tables

Table 1: Summary Statistics, measurements, whole sample, Cohort 1958 [administered at age 11]

Variables	TOTAL		Education				Smoking			
	ALL		**Post-compulsory**		**Compulsory**		**Smoking in preg**		**Non-smoking in preg**	
Cognitive Skill Measures										
Verbal IQ	24.482	(8.67)	28.967	(6.99)	22.109	(8.53)	20.964	(8.66)	24.692	(8.45)
Non-verbal IQ	22.134	(7.22)	25.704	(6.27)	20.245	(6.97)	19.548	(6.88)	22.255	(7.15)
Reading comprehension	16.799	(5.76)	20.182	(5.15)	15.010	(5.23)	14.281	(5.29)	16.829	(5.52)
Mathematics	17.909	(9.89)	23.913	(8.93)	14.733	(8.84)	13.803	(8.58)	17.980	(9.75)
Copying designs test	8.394	(1.34)	8.704	(1.37)	8.229	(1.29)	8.118	(1.27)	8.407	(1.34)
Social Skill Measures										
Inconsequential Behavior	0.416	(0.49)	0.298	(0.46)	0.478	(0.50)	0.565	(0.50)	0.392	(0.49)
Nervous symptoms	0.067	(0.25)	0.040	(0.19)	0.081	(0.27)	0.099	(0.30)	0.062	(0.24)
Anxiety acceptance, adults	0.288	(0.45)	0.245	(0.43)	0.310	(0.46)	0.330	(0.47)	0.280	(0.45)
Anxiety acceptance, children	0.129	(0.33)	0.102	(0.30)	0.143	(0.35)	0.179	(0.38)	0.119	(0.32)
Hostility toward children	0.130	(0.34)	0.091	(0.29)	0.151	(0.36)	0.207	(0.41)	0.110	(0.31)
Writing off adults	0.324	(0.47)	0.233	(0.42)	0.372	(0.48)	0.471	(0.50)	0.293	(0.46)
Hostility towards adults	0.232	(0.42)	0.173	(0.38)	0.263	(0.44)	0.361	(0.48)	0.205	(0.40)
Miscellaneous symptoms	0.318	(0.47)	0.230	(0.42)	0.365	(0.48)	0.395	(0.49)	0.315	(0.46)
Restlessness	0.112	(0.32)	0.058	(0.23)	0.141	(0.35)	0.194	(0.40)	0.100	(0.30)
Unforthcomingness	0.495	(0.50)	0.430	(0.50)	0.529	(0.50)	0.515	(0.50)	0.487	(0.50)
Depression	0.364	(0.48)	0.243	(0.43)	0.429	(0.50)	0.498	(0.50)	0.336	(0.47)
Withdrawal	0.159	(0.37)	0.118	(0.32)	0.181	(0.38)	0.192	(0.39)	0.156	(0.36)
Measures of Physical Constitution										
Maternal birthweight (grams)	3271.581	(501.80)	3318.091	(461.23)	3246.977	(520.42)	3264.149	(536.95)	3255.021	(490.09)
Grandmaternal height (inches)	63.484	(2.49)	63.614	(2.42)	63.415	(2.53)	63.366	(2.58)	63.506	(2.49)
Height, age 11 (cm)	57.056	(2.92)	57.497	(2.84)	56.823	(2.93)	56.580	(3.08)	57.065	(2.83)
N	3217		1113		2104		588		1797	

Source: National Child Development Study (NCDS), Birth cohort 1958.

Notes: The analytical sample on which these estimates are based consists of all female cohort members that have no missings in any of the covariates.

We measure cognitive and social skills at age 11 of the mothers. Standard errors in brackets. Own calculations.

Table 2: Summary statistics, outcomes and covariates, whole sample, Cohort 1958

Variables	TOTAL		Education				Smoking			
	ALL		**Post-compulsory**		**Compulsory**		**Smoking in preg**		**Non-smoking in preg**	
Outcome Variable and Treatments										
Child Small for Gestational Age (SGA)	0.140	(0.35)	0.110	(0.31)	0.153	(0.36)	0.221	(0.42)	0.114	(0.32)
Post-compulsory education	0.346	(0.48)	1.000	(0.00)	0.000	(0.00)	0.136	(0.34)	0.354	(0.48)
Smoking past 3rd month of pregnancy	0.247	(0.43)	0.112	(0.32)	0.304	(0.46)	1.000	(0.00)	0.000	(0.00)
Mother lived in a broken home at age 7	0.091	(0.29)	0.069	(0.25)	0.103	(0.30)	0.128	(0.33)	0.087	(0.28)
Mother household size at age 7	5.073	(1.53)	4.783	(1.35)	5.226	(1.60)	5.602	(1.71)	4.976	(1.46)
Mother is firstborn	0.391	(0.49)	0.465	(0.50)	0.352	(0.48)	0.306	(0.46)	0.421	(0.49)
Grandmother had post-compulsory schooling	0.284	(0.45)	0.460	(0.50)	0.192	(0.39)	0.168	(0.37)	0.282	(0.45)
Grandmother age at mother's birth	27.601	(5.57)	28.175	(5.26)	27.298	(5.70)	26.910	(5.67)	27.316	(5.45)
Grandparents high SES	0.181	(0.39)	0.319	(0.47)	0.108	(0.31)	0.071	(0.26)	0.179	(0.38)
Grandparents medium SES	0.588	(0.49)	0.554	(0.50)	0.606	(0.49)	0.575	(0.49)	0.607	(0.49)
Mother living in London at age 11	0.089	(0.28)	0.081	(0.27)	0.093	(0.29)	0.070	(0.25)	0.093	(0.29)
Mother living in Wales at age 11	0.061	(0.24)	0.072	(0.26)	0.055	(0.23)	0.070	(0.25)	0.060	(0.24)
Mother living in Scotland at age 11	0.111	(0.31)	0.136	(0.34)	0.098	(0.30)	0.167	(0.37)	0.090	(0.29)
Mother living in London at age 16	0.090	(0.29)	0.083	(0.28)	0.094	(0.29)	0.071	(0.26)	0.092	(0.29)
Mother living in Wales at age 16	0.062	(0.24)	0.075	(0.26)	0.055	(0.23)	0.068	(0.25)	0.062	(0.24)
Mother living in Scotland at age 16	0.112	(0.32)	0.137	(0.34)	0.099	(0.30)	0.163	(0.37)	0.092	(0.29)
Change in regional unemployment rate	-1.211	(0.38)	-1.209	(0.40)	-1.212	(0.37)	-1.251	(0.41)	-1.195	(0.37)
Regional prevalence of smokers	0.332	(0.04)	0.318	(0.04)	0.338	(0.04)	0.350	(0.04)	0.326	(0.04)
Grandmother smoked in pregnancy	0.317	(0.47)	0.254	(0.44)	0.351	(0.48)	0.413	(0.49)	0.301	(0.46)
Child sex (female=1)	0.484	(0.50)	0.475	(0.50)	0.488	(0.50)	0.497	(0.50)	0.480	(0.50)
Grandmother weight at mother's birth (stones)	8.853	(1.47)	8.834	(1.35)	8.863	(1.53)	8.976	(1.67)	8.813	(1.41)
Mother relative weight at age 11	100.914	(14.79)	100.209	(14.42)	101.288	(14.98)	102.303	(15.56)	100.212	(14.38)
Mother gestational age (in weeks)	40.201	(1.73)	40.215	(1.54)	40.194	(1.83)	40.210	(1.95)	40.207	(1.70)
N	3217		1113		2104		588		1797	

Source: National Child Development Study (NCDS), Birth cohort 1958.

Notes: The analytical sample on which these estimates are based consists of all female cohort members that have no missings in any of the covariates.

Standard errors in brackets. Own calculations. The variable "regional prevalence of smokers" refers to the smoking prevalence in the mothers' region during the time when the mother was pregnant, and it has been obtained from `www.statistics.gov.uk`.

The variable "change in regional unemployment rate" refers to the change in unemployment between 1972 and 1973, at the time of her educational decision (source: Department of Employment Gazzette 1975).

"Grandparents high SES" refer to professional and managerial positions, "Grandparents medium SES" refer to non-manual and manual skilled positions, following the General Register Office classification, Census 1951.

"Mother relative weight at age 11" is the weight as the percentage of the average weight in the sample (100%).

Table 3: Covariates in the different model equations

		Meas (C,F)	Meas (P)	Education	Smoking	SGA
Covariates	Constant	✓	✓	✓	✓	✓
	Mother lived in a broken home at age 7	✓	✓			
	Mother household size at age 7	✓	✓			
	Mother is firstborn	✓	✓	✓	✓	
	Grandmother had post-compulsory schooling	✓	✓	✓	✓	✓
	Grandmother age at mother's birth	✓	✓	✓	✓	
	Grandparents high SES	✓	✓	✓	✓	
	Grandparents medium SES	✓	✓	✓	✓	
	Mother living in London at age 11	✓	✓	✓	✓	✓
	Mother living in Wales at age 11	✓	✓	✓	✓	✓
	Mother living in Scotland at age 11	✓	✓	✓	✓	✓
	Mother living in London at age 16			✓	✓	✓
	Mother living in Wales at age 16			✓	✓	✓
	Mother living in Scotland at age 16			✓	✓	✓
	Change in regional unemployment rate			✓		
	Regional smoking prevalence				✓	
	Grandmother smoked in pregnancy				✓	
	Child sex					✓
	Grandmother weight at mother's birth		✓			
	Mother relative weight at age 11		✓			
	Mother gestational age		✓			

Table 4: Factor loadings in the measurement system

	Cognitive Skills	Social Skills	Physical Constitution
Verbal IQ	1	-	-
Non-verbal IQ	0.912	-	-
	(0.01)		
Reading comprehension	0.795	-	-
	(0.02)		
Mathematics	0.904	-	-
	(0.02)		
Copying designs test	0.345	-	-
	(0.02)		
Inconsequential behavior	-	0.564	-
		(0.04)	
Nervous symptoms	-	0.369	-
		(0.04)	
Anxiety acceptance, adults	-	0.207	-
		(0.02)	
Anxiety acceptance, children	-	0.436	-
		(0.04)	
Hostility towards children	-	0.686	-
		(0.06)	
Writing off adults	-	0.575	-
		(0.04)	
Hostility towards adults	-	0.876	-
		(0.07)	
Miscellaneous symptoms	-	0.574	-
		(0.04)	
Restlessness	-	0.663	-
		(0.06)	
Unforthcomingness	-	0.335	-
		(0.03)	
Depression	-	1	-
Withdrawal	-	0.519	-
		(0.04)	
Maternal height	-	-	1.757
			(0.2)
Maternal birthweight	-	-	1
Grandmaternal height	-	-	1.09
			(0.11)

Note: National Child Development Study (NCDS), Birth cohort 1958. The analytical sample on which these estimates are based consists of all female cohort members that have no missings in any of the covariates. Standard errors in brackets.

Table 5: Model fit

	Simulated	Data	Difference	p-val χ^2	% correctly predicted
Education	0.343	0.346	0.003	0.425	0.612
Smoking(E=1)	0.130	0.112	-0.018	0.969	0.732
Smoking(E=0)	0.294	0.304	0.011	0.839	0.631
SGA(E=1, S=1)	0.194	0.212	0.018	0.274	0.669
SGA(E=0, S=1)	0.227	0.239	0.012	0.916	0.646
SGA(E=1, S=0)	0.098	0.106	0.008	0.920	0.758
SGA(E=0, S=0)	0.143	0.141	-0.002	0.882	0.713

Note: National Child Development Study (NCDS), Birth cohort 1958. The analytical sample on which these estimates are based consists of all female cohort members that have no missings in any of the covariates. Table displays model fit.

Table 6: Average marginal effects of a one standard deviation change in maternal traits

	Cognitive Skills	Social Skills	Physical Constitution
Education	0.152	0.039	0.016
	(0.046)	(0.014)	(0.013)
Smoking(E=1)	-0.011	-0.063	0.003
	(0.022)	(0.04)	(0.026)
Smoking(E=0)	-0.004	-0.056	0.014
	(0.013)	(0.021)	(0.016)
SGA(E=1, S=1)	-0.008	0.075	-0.149
	(0.1)	(0.127)	(0.206)
SGA(E=0, S=1)	0.027	-0.023	-0.11
	(0.027)	(0.025)	(0.054)
SGA(E=1, S=0)	0.003	-0.012	-0.053
	(0.02)	(0.018)	(0.036)
SGA(E=0, S=0)	-0.015	0.009	-0.046
	(0.012)	(0.015)	(0.021)

Note: National Child Development Study (NCDS), Birth cohort 1958. The analytical sample on which these estimates are based consists of all female cohort members that have no missings in any of the covariates. Standard errors in brackets. E=education; S=smoking; SGA=small for gestational age.

Table 7: Correlation of maternal traits

	Cognition	Social Skills	Physical Constitution
Cognition	1.000	0.369	0.172
Social Skills	0.369	1.000	-0.003
Physical Constitution	0.172	-0.003	1.000

Note: National Child Development Study (NCDS), Birth cohort 1958. The analytical sample on which these estimates are based consists of all female cohort members that have no missings in any of the covariates.

Table 8: Results using the three-step estimation procedure

Variables	**Education**	**Smoking in Pregnancy**		**SGA**			
		E=1	**E=0**	**E=1, S=1**	**E=0, S=1**	**E=1, S=0**	**E=0, S=0**
Cognitive skills factor, bias corrected	0.135	-0.004	0.0003	-0.052	0.018	0.002	-0.021
	(0.00)	(0.76)	(0.98)	(0.52)	(0.41)	(0.92)	(0.09)
Social skills factor, bias corrected	0.027	-0.048	-0.050	0.048	-0.015	-0.015	0.00763
	(0.00)	(0.00)	(0.00)	(0.50)	(0.46)	(0.37)	(0.52)
Physical constitution factor, bias corrected	0.008	-0.003	0.003	-0.081	-0.081	-0.032	-0.0262
	(0.30)	(0.80)	(0.81)	(0.24)	(0.00)	(0.01)	(0.02)
Observations	3209	715	1664	79	480	614	1109
Pseudo R^2	0.211	0.110	0.074	0.100	0.039	0.026	0.017
Covariates included	YES	YES	YES	YES	YES	YES	YES

Note: The coefficients display the average marginal effect of a one standard deviation change in the factor.
The analytical sample on which these estimates are based consists of all female cohort members with no missings in any of the covariates. Own calculations based on the NCDS data.
p-values in brackets. Standard errors are bootstrapped using 100 replications.
The bias correction procedure is the one due to Iwata (1992) and described in Appendix D.

Table 9: Decomposition of the effects of maternal endowments on newborn SGA

	Cognitive Skills	Social Skills	Physical Constitution
Education	68	30	1
Smoking	2	64	-2.0
Residual	30	6	100
TOTAL	100	100	100

Note: Numbers in cell show the percentage of the overall effect of each maternal trait which works through the education and smoking choices, and the residual effect. National Child Development Study (NCDS), Birth cohort 1958. The analytical sample on which these estimates are based consists of all female cohort members that have no missings in any of the covariates.

Table 10: Treatment effects of smoking and education

Treatment effect of	ATE	ATT	ATNT	AMTE	Control Mean
education on smoking in pregnancy	-0.098	-0.094	-0.100	-0.100	0.304
	(0.01)	(0.009)	(0.01)	(0.01)	
education on the probability of	-0.016	-0.02	-0.014	-0.016	0.153
delivering a SGA baby	(0.009)	(0.008)	(0.009)	(0.009)	
smoking on the probability of	0.111	0.099	0.115	0.110	0.114
delivering a SGA baby	(0.009)	(0.009)	(0.009)	(0.009)	

Note: National Child Development Study (NCDS), Birth cohort 1958. The analytical sample on which these estimates are based consists of all female cohort members that have no missings in any of the covariates. The numbers in columns 2-5 are the treatment effects, as specified: ATE=Average Treatment Effect; ATT=Average Treatment Effect on the Treated; ATNT=Average Treatment Effect on the Non-Treated; AMTE=Average Marginal Treatment Effect. The last column displays the average outcome for the untreated group. Standard errors in brackets. For definitions of these treatment effects, see Heckman and Vytlacil (2007a,b).

Table 11: Decomposition of the observed difference in the outcomes into causal and selection components

Decomposition of the difference in	Observed Difference	ATE	Sorting Gain	Selection Bias	% Sorting gain + Selection Bias
smoking in pregnancy by education	-0.166	-0.098	0.004	-0.073	41.059
SGA by education	-0.049	-0.016	-0.004	-0.029	66.850
SGA by smoking	0.102	0.111	-0.012	0.003	8.759

Note: National Child Development Study (NCDS), Birth cohort 1958. The analytical sample on which these estimates are based consists of all female cohort members that have no missings in any of the covariates. The second column shows the difference in the observed outcomes between the treated and the untreated. The third, fourth and fifth columns decompose this difference into the average treatment effect, the sorting gain and the evaluation bias, respectively. The sixth column displays the percentage of the observed difference attributable to the selection component.

Table 12: Heterogeneity in average treatment effects of smoking and education along the distribution of maternal traits

Treatment effect of	<20%	20-39%	40-59%	60-79%	>=80%
education on smoking in pregnancy (C)	-0.09	-0.10	-0.11	-0.11	-0.12
education on smoking in pregnancy (F)	-0.08	-0.10	-0.11	-0.11	-0.11
education on smoking in pregnancy (P)	-0.09	-0.10	-0.11	-0.11	-0.12
education on SGA (C)	-0.02	-0.03	-0.03	-0.03	-0.02
education on SGA (F)	-0.04	-0.03	-0.03	-0.03	-0.04
education on SGA (P)	0.02	-0.01	-0.03	-0.03	-0.02
smoking on SGA (C)	0.05	0.07	0.08	0.08	0.08
smoking on SGA (F)	0.10	0.08	0.08	0.09	0.11
smoking on SGA (P)	0.28	0.16	0.08	0.04	0.01

Note: National Child Development Study (NCDS), Birth cohort 1958. The analytical sample on which these estimates are based consists of all female cohort members that have no missings in any of the covariates. The table shows the average treatment effect of education on smoking (rows 1-3) and on newborn SGA (rows 4-6), and of smoking on SGA (rows 7-9) at different quantiles of the traits distribution (with the other two traits in turn fixed at their mean values).

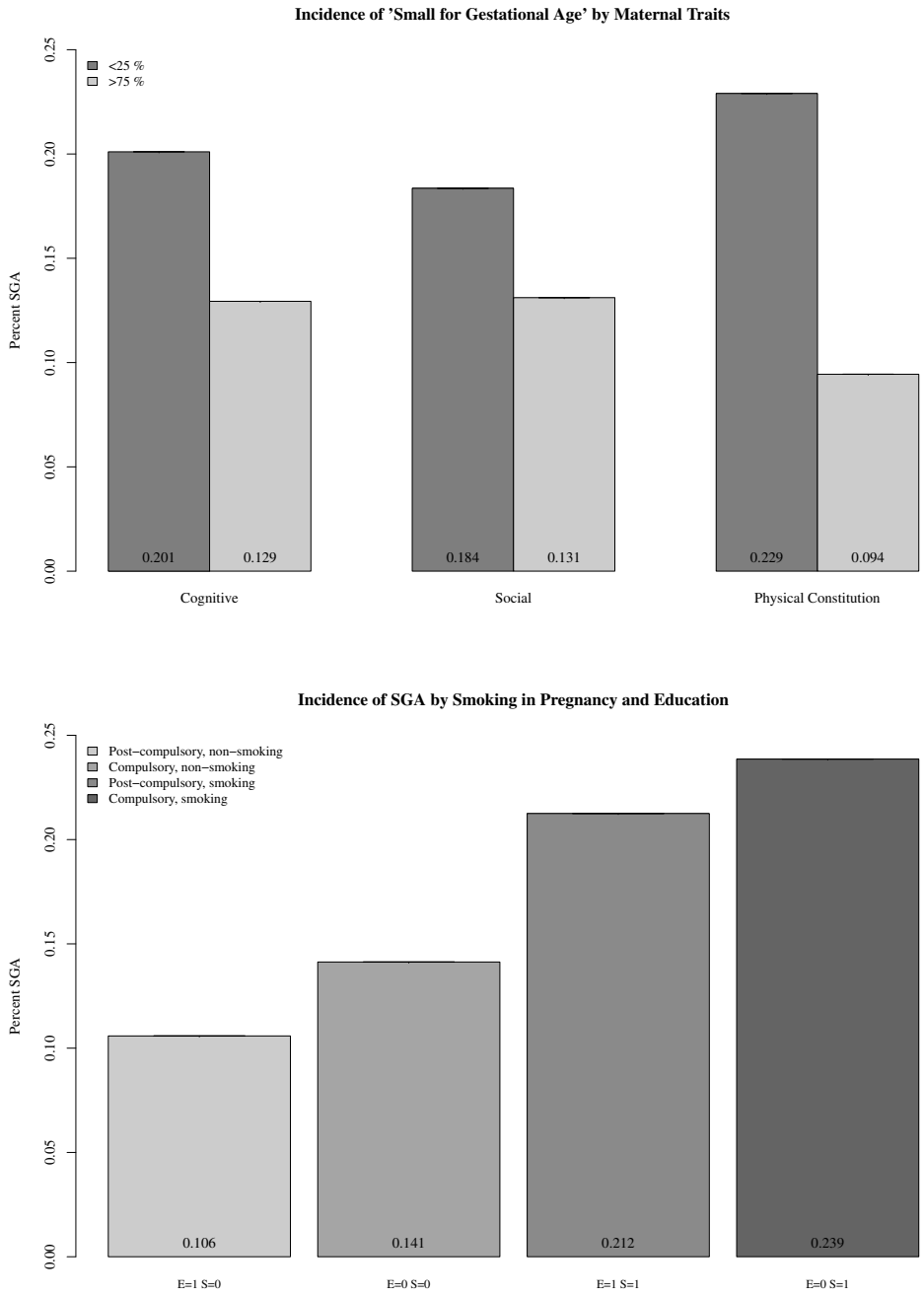
Table 13: Treatment effects equivalents in terms of standard deviations of the trait distribution

	ATE	Cognitive (sd)	Social (sd)	Physical Constitution (sd)
Education	-0.016	1.771	3.235	0.244
Smoking	0.111	-12.156	-22.2	-1.676

Note: National Child Development Study (NCDS), Birth cohort 1958. Columns 2-4 display the equivalent of the treatment effect in standard deviations of the endowment distribution. For example, in order to achieve the same effect as a policy that moves everybody from compulsory to post-compulsory education, every individual would need to be endowed with 1.8 sd higher cognitive abilities.

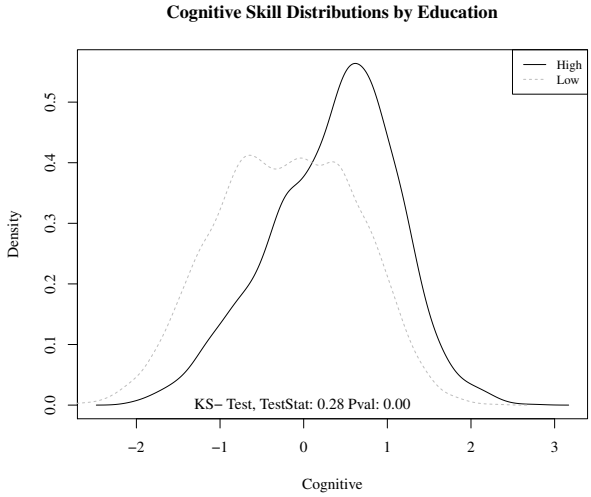
8 Figures

Figure 1: Probability of being born small for gestational age (SGA), by maternal traits and treatment status

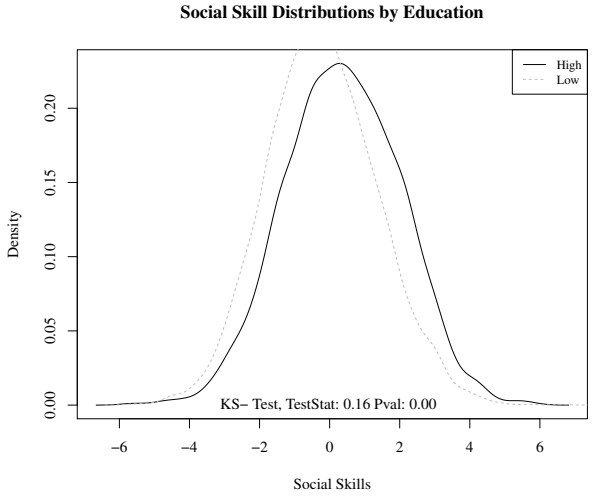


Note: National Child Development Study (NCDS), Birth cohort 1958. The analytical sample on which these figures are based consists of all female cohort members that have no missings in any of the covariates.

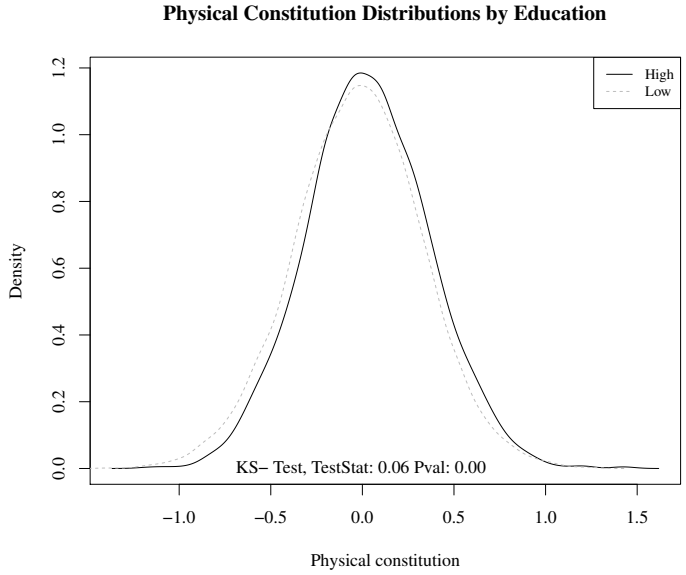
Figure 2: Marginal densities of maternal traits by education



(a) Cognitive



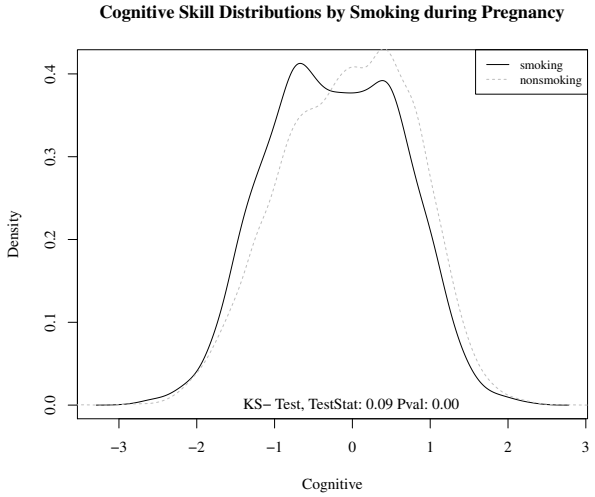
(b) Social



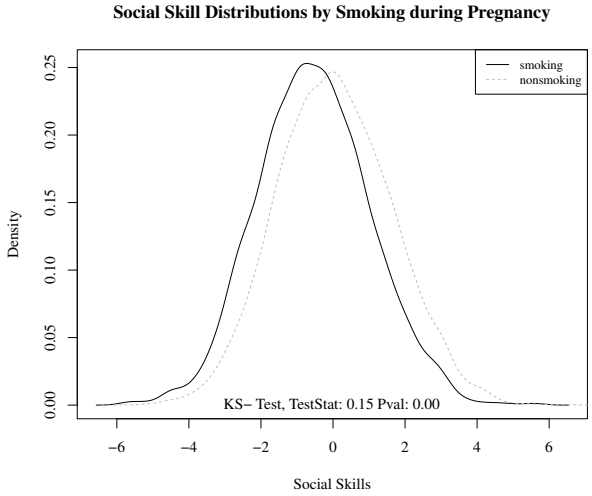
(c) Body Size

Note: National Child Development Study (NCDS), Birth cohort 1958. The analytical sample on which these estimates are based consists of all female cohort members that have no missings in any of the covariates.

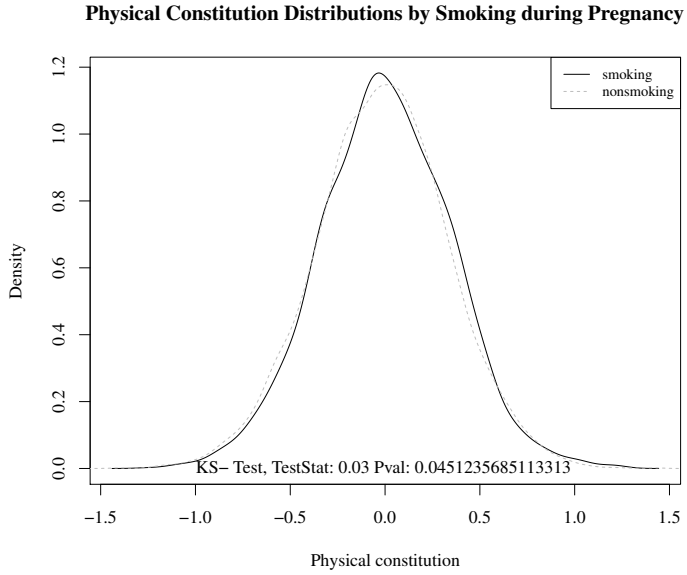
Figure 3: Marginal densities of maternal traits by prenatal smoking



(a) Cognitive



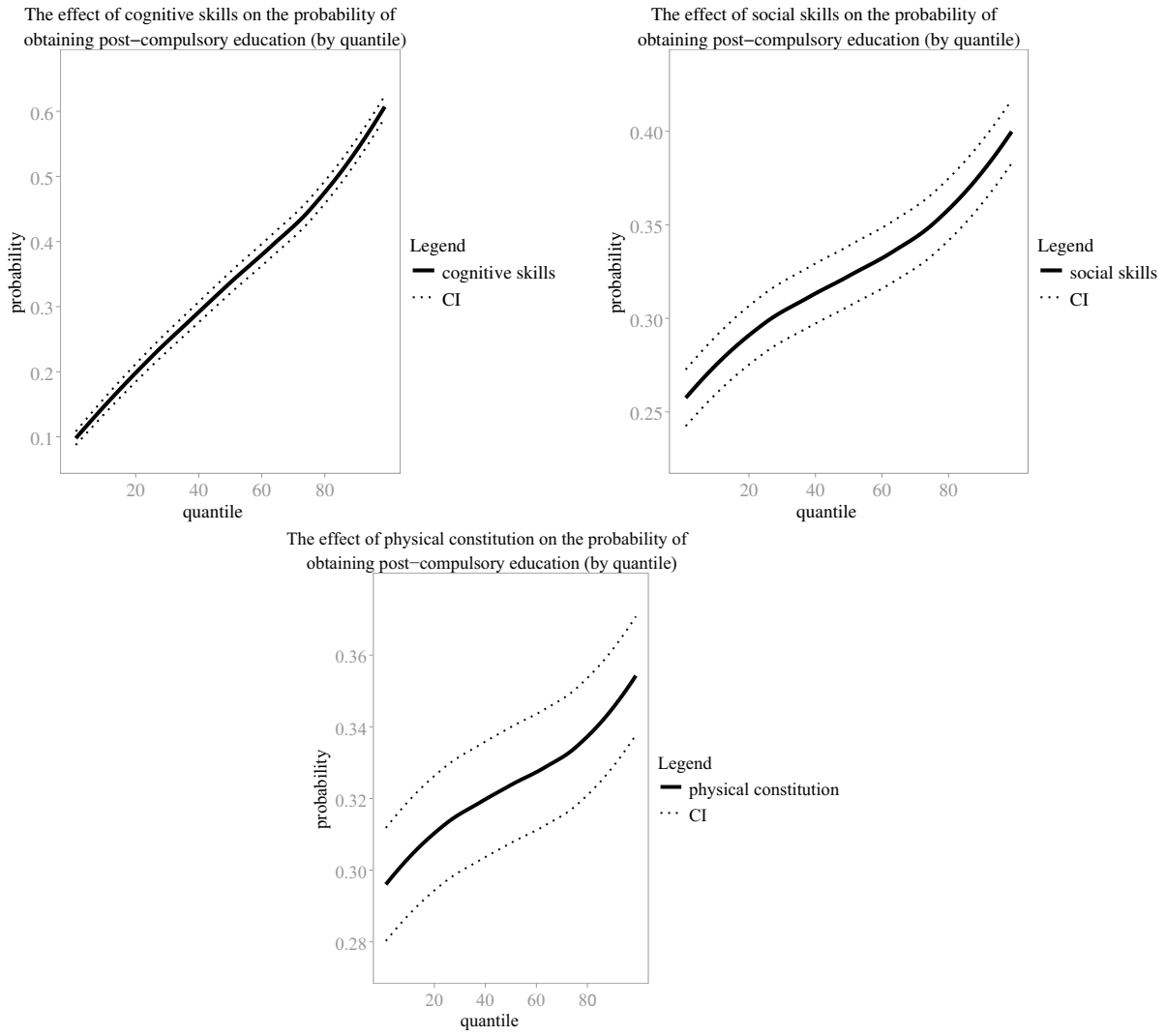
(b) Social



(c) Body Size

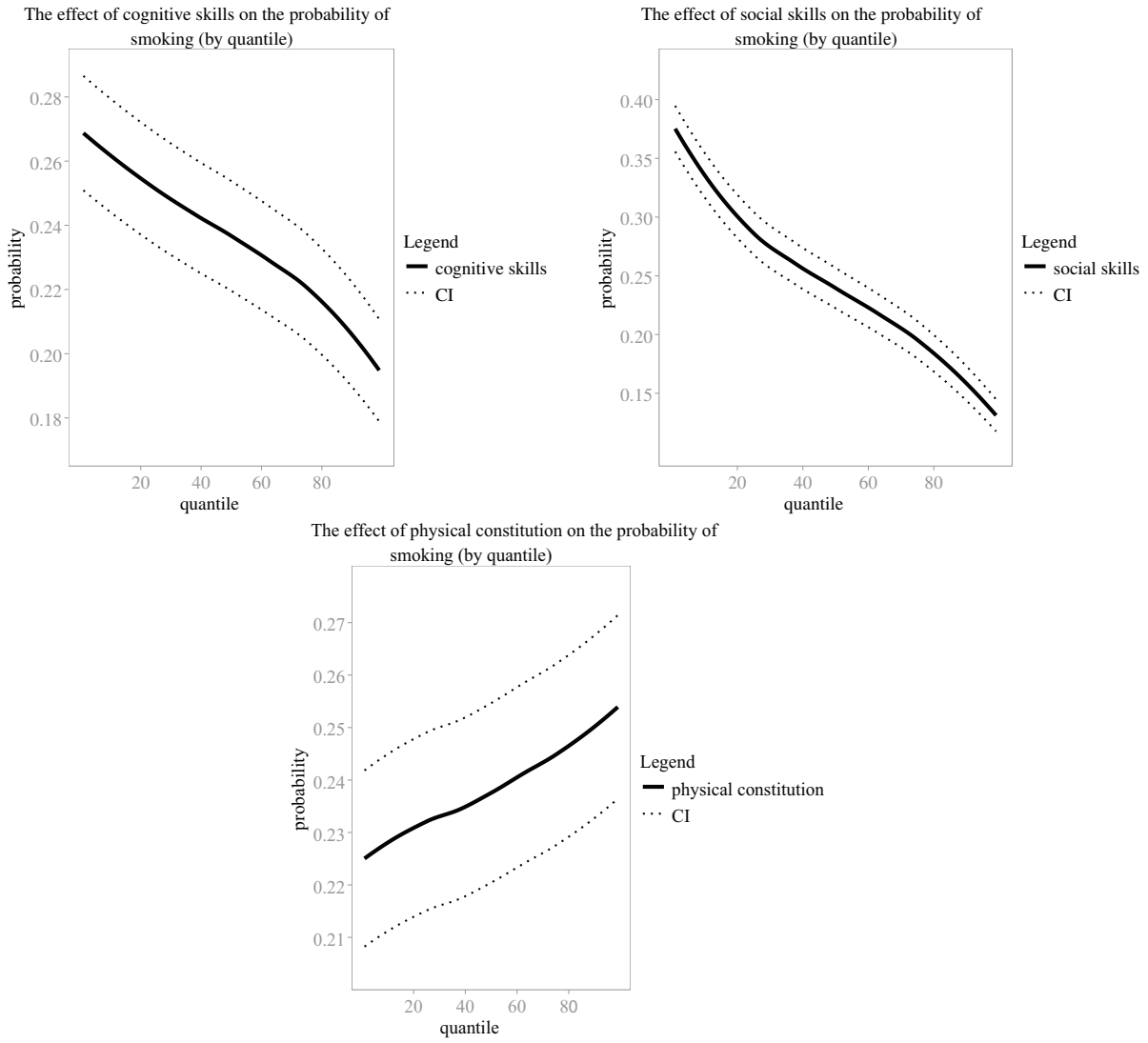
Note: National Child Development Study (NCDS), Birth cohort 1958. The analytical sample on which these estimates are based consists of all female cohort members that have no missings in any of the covariates.

Figure 4: Effects of maternal traits on education



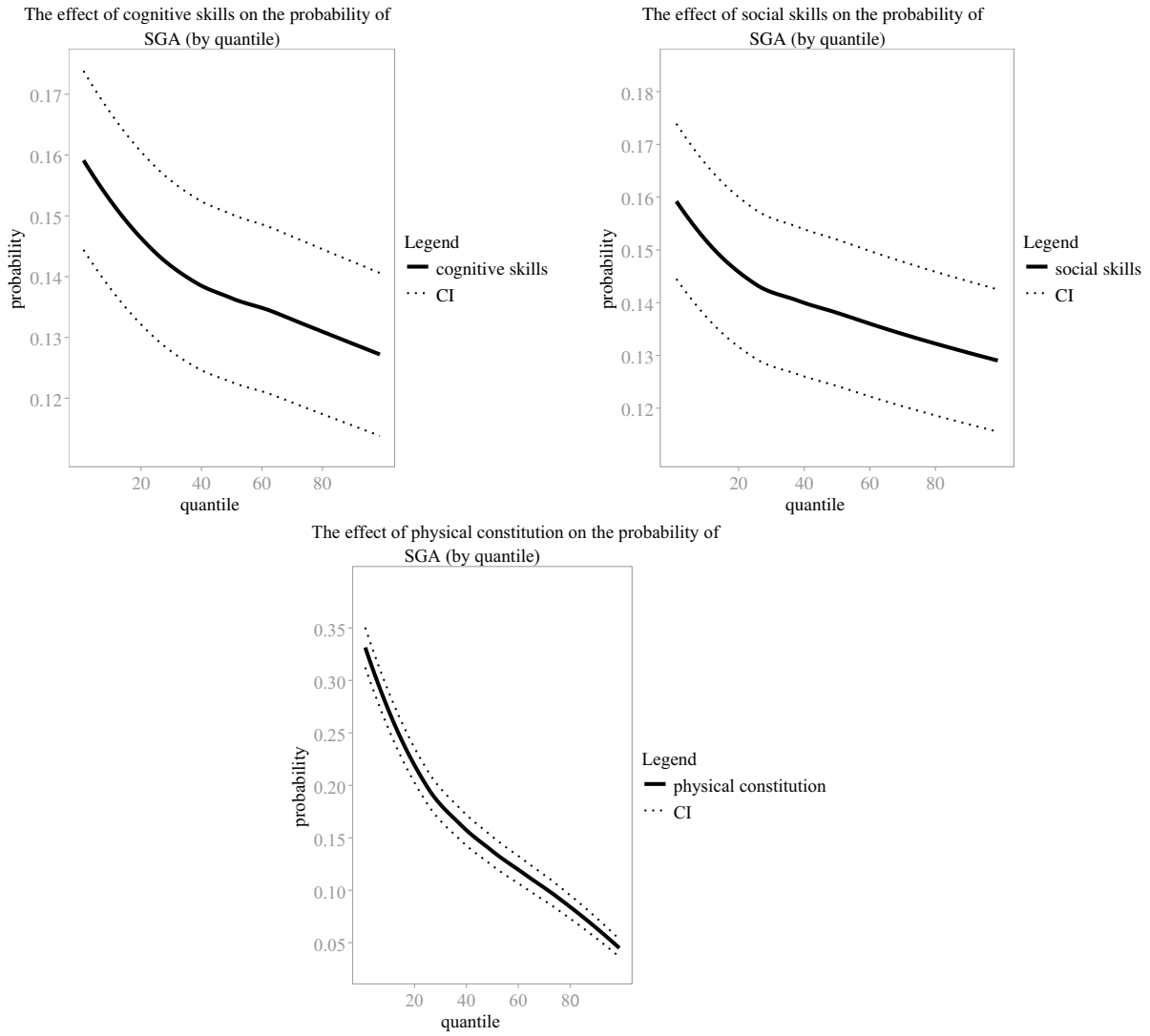
Note: National Child Development Study (NCDS), Birth cohort 1958. The analytical sample on which these estimates are based consists of all female cohort members that have no missings in any of the covariates. When computing the overall effect of each trait in turn on the educational choice, the other two traits are fixed at their respective means. 95% confidence intervals drawn.

Figure 5: Effect of maternal traits on smoking during pregnancy



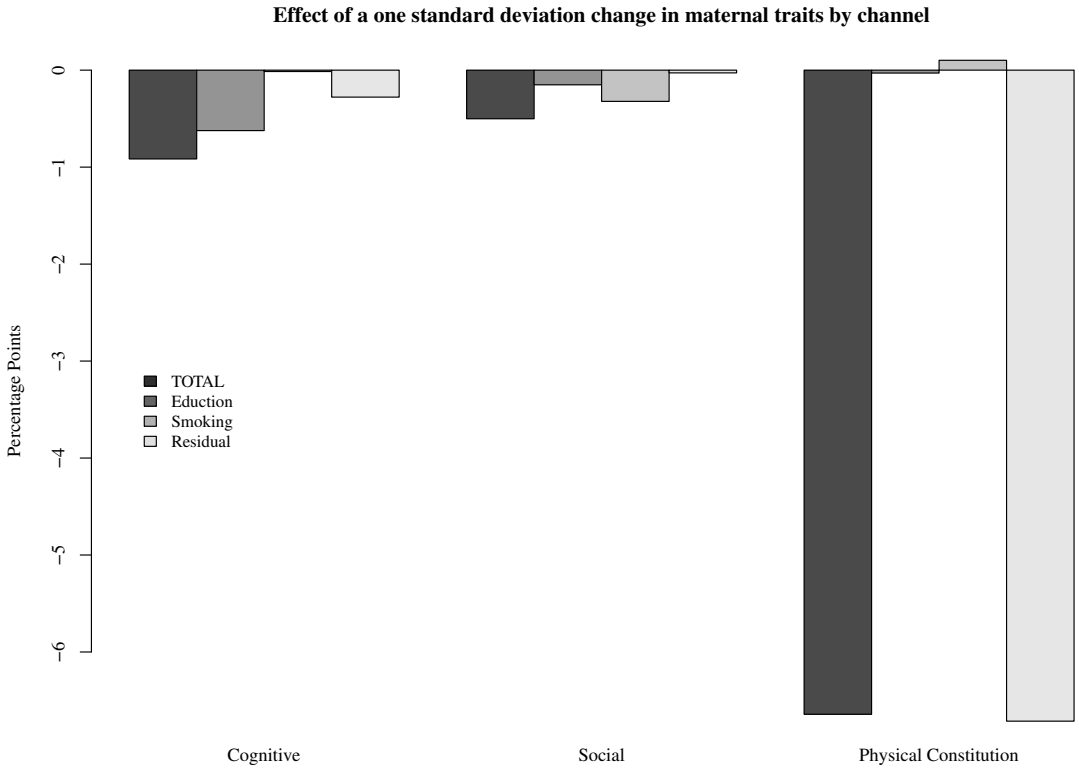
Note: National Child Development Study (NCDS), Birth cohort 1958. The analytical sample on which these estimates are based consists of all female cohort members that have no missings in any of the covariates. When computing the overall effect of each trait in turn on the smoking choice, the other two traits are fixed at their respective means. 95% confidence intervals drawn.

Figure 6: Effect of maternal traits on newborn SGA



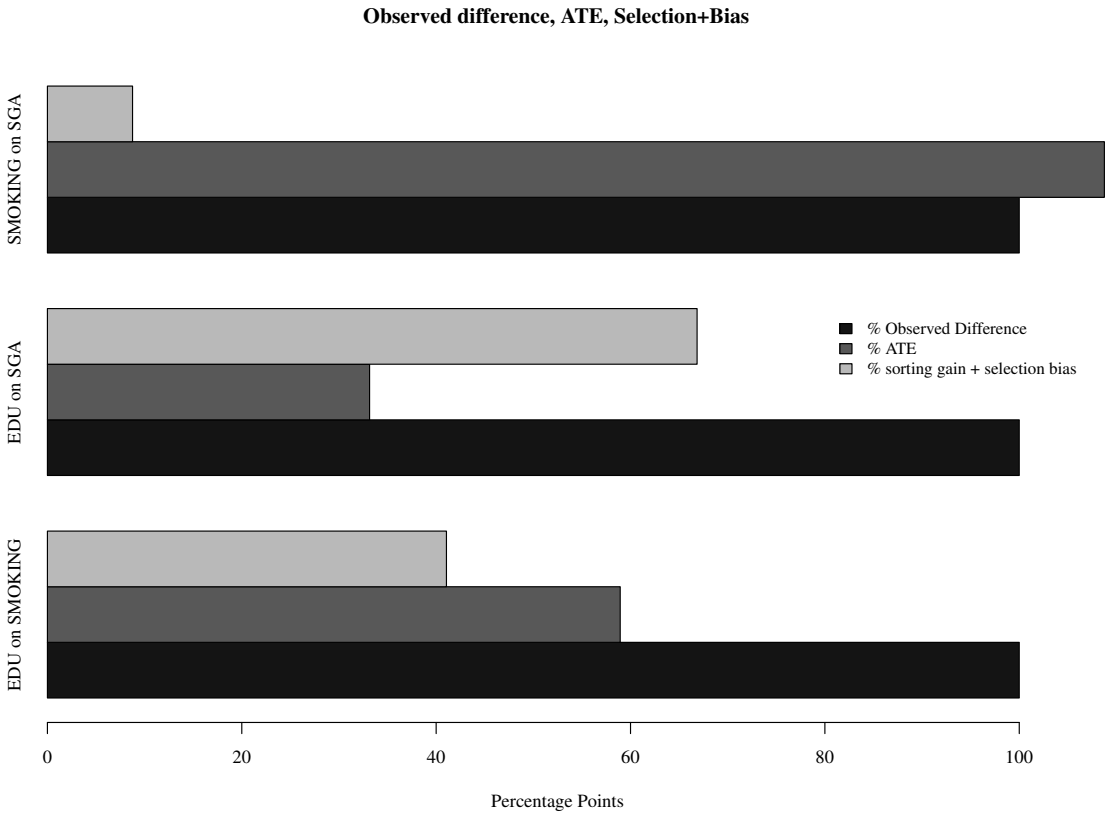
Note: National Child Development Study (NCDS), Birth cohort 1958. The analytical sample on which these estimates are based consists of all female cohort members that have no missings in any of the covariates. When computing the overall effect of each trait in turn on the probability of delivering a SGA newborn, the other two traits are fixed at their respective means. 95% confidence intervals drawn.

Figure 7: Decomposing the effects of maternal endowments on newborn SGA



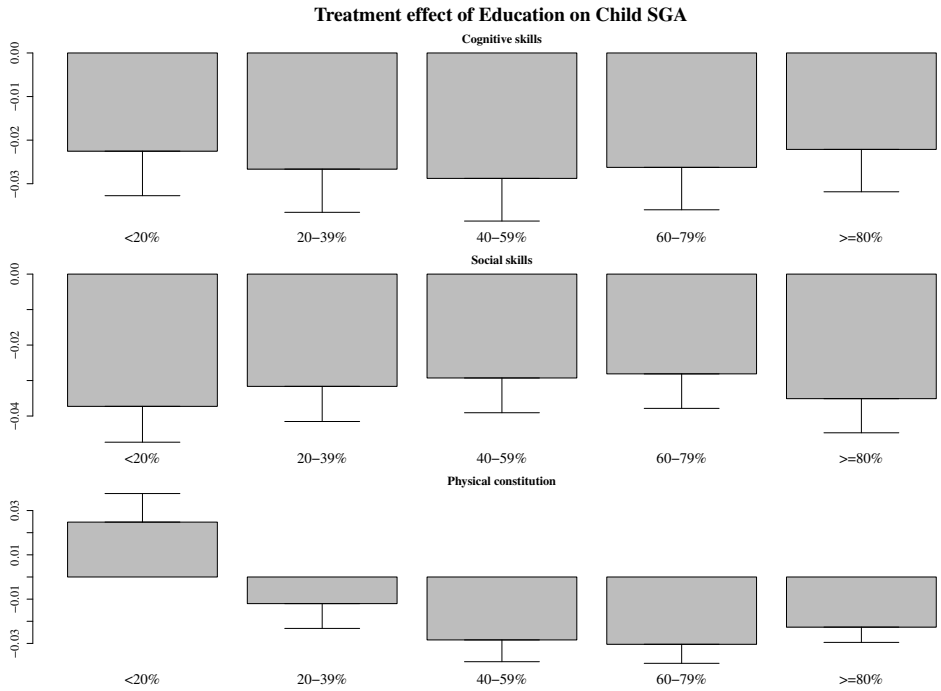
Note: National Child Development Study (NCDS), Birth cohort 1958. The analytical sample on which these estimates are based consists of all female cohort members that have no missings in any of the covariates. The length of the bar "Total" shows the overall reduction in the probability of delivering a SGA baby which is associated with a one standard deviation increase in each of the three maternal endowments. The respective contributions of the various channels are shown in the bars "education", "smoking" and "residual", respectively.

Figure 8: Decomposition of the observed differences in the outcomes into causal and selection components

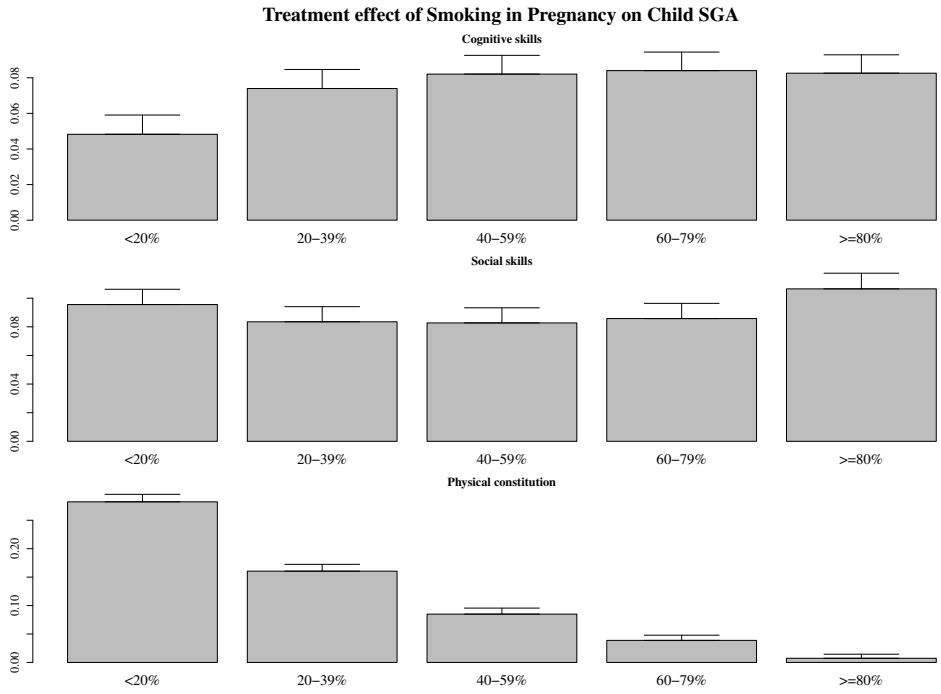


Note: National Child Development Study (NCDS), Birth cohort 1958. The analytical sample on which these estimates are based consists of all female cohort members that have no missings in any of the covariates.

Figure 9: Heterogenous treatment effects of education and smoking on SGA along the distribution of maternal traits



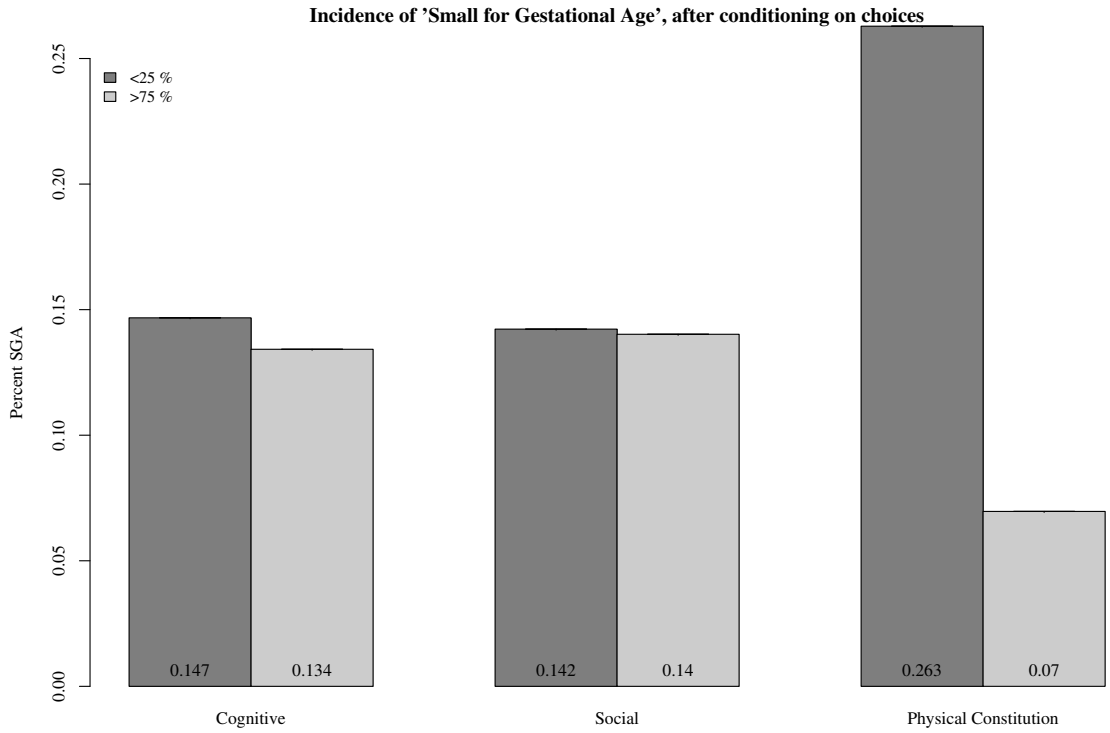
(a) Social



(b) Body Size

Note: National Child Development Study (NCDS), Birth cohort 1958. The analytical sample on which these estimates are based consists of all female cohort members that have no missings in any of the covariates. Whiskers display standard errors.

Figure 10: Difference in SGA after conditioning on maternal traits and choices



Note: National Child Development Study (NCDS), Birth cohort 1958. Graph displays outcomes for individuals with endowments in the highest/lowest quartile of the distribution after conditioning on education and smoking. Conditional SGA probabilities are weighted by unconditional smoking and education probabilities according to: $\sum_E \sum_S \left[P(SGA = 1|X = x, \Theta = \theta_{>75}, E = e, S = s) - P(SGA = 1|X = x, \Theta = \theta_{<25}, E = e, S = s)\right] P(S = s|E = e)P(E = e)$