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ABSTRACT

The paper focuses on the long-term effects of early-life conditions with comparison to lifestyles and current socioeconomic factors on health status in a cohort of British people born in 1958. Using the longitudinal follow-up data at age 23, 33, 42 and 46, we build a dynamic model to investigate the influence of each determinant on health and the mediating role of education and lifestyles in the relationship between early-life conditions and later health. Direct and indirect effects of early-life conditions on adult health are explored using auxiliary linear regressions of education and lifestyles and panel Probit specifications of self-assessed health with random effects addressing individual unexplained heterogeneity. Our study shows that early-life conditions are important parameters for adult health, their contribution to health disparities increases from 17.8% to 23% when mediating effects are identified. They also shape other health determinants: the contribution of lifestyles reduces from 28% down to 22% when indirect effects of early-life conditions are distinguished. Noticeably, the absence of father at the time of birth and experience of financial hardships represent the lead factors for direct effects on health. The absence of obesity at 16 influences health both directly and indirectly working through lifestyles.

Keywords: cohort; decomposition; early-life conditions; education; lifestyles

Codes JEL: D63; I12.

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1. Introduction

Numerous literature references have agreed the important role played by current individual social characteristics, such as income, education level, wealth, and social status e.g. (van Doorslaer and Koolman 2004, Cutler et al. 2006, Lantz et al. 2010) in the explanation of health inequalities. More recently, several studies have also found early-life conditions as a relevant determinant of health inequalities with a large range of social background factors, such as low parental socioeconomic status (e.g. Currie and Stabile 2003, Case et al. 2005, Lindeboom et al. 2009, Rosa-Dias 2009, Jusot et al. 2010, Trannoy et al. 2010); family issues, such as living in a single parent family or experiencing marital discord (Case and Katz 1991, Francesconi et al. 2010); parents' health status (Trannoy et al. 2010) or health-risk lifestyles (Anda et al. 2002, Gohlmann et al. 2010, Jusot et al. 2010). However, the importance of lifestyles in the magnitude of health inequalities is less clear. Whereas epidemiological literature concluded until recently that lifestyles make a relatively minor contribution to the social gradient in health (Khang et al. 2009, Lantz et al. 2010, Skalicka et al. 2009, Van Oort et al. 2005), health economists have shown that differences in lifestyles can explain a relevant part of health and mortality inequalities (Contoyannis and Jones 2004, Häkkinen et al. 2006, Balia and Jones 2008) and a few recent epidemiological studies (Laaksonen et al. 2008, Menvielle et al. 2009, Strand and Tverdal 2004, Stringhini et al. 2010) have also confirmed that the impact of lifestyles on health and mortality disparities would be larger than it was previously estimated, particularly if lifestyles are observed longitudinally.

The issue at stake is that the three broad determinants of health: early-life conditions, current socioeconomic status (SES), and lifestyles cannot be considered as independent (see Figure 1). Several studies provide evidence on the transmission of SES over different generations and its relevance in the explanation of health inequalities e.g. (Marmot et al. 2001, Case et al. 2005, Trannoy et al. 2010). Moreover, parents' characteristics and early-life conditions would also be associated

with health-related behaviours in later life (Anda et al. 2002, Rosa-Dias 2009, Gohlmann et al. 2010, Jusot et al. 2010). Similarly, several studies uncovered mechanisms through which education affects lifestyles such as obesity and exercise (Kenkel 1991, Park and Kang 2008, Webbink et al 2010), smoking (Kenkel 1991, de Walque 2007, Etilé and Jones 2010). It is therefore essential to understand the interrelationships between those various determinants of health in order to evaluate their respective contribution to the magnitude of health inequalities.

Fig. 1: Early-life conditions, socioeconomic factors, lifestyles and later-life health status

The objective of this study is to explore the long term effects of social and health-related early-life conditions, education, and lifestyles on health and to understand the interdependence between those three health determinants. Relying on a dynamic model of health status over the life-cycle, our empirical analysis aims to investigate the effect of each determinant in overall health inequality and determines whether early-life conditions influence health directly or indirectly, that is via affecting education and lifestyles.

Our findings provide new elements on the determinants of health inequalities which are relevant for policy makers and that remained to be empirically assessed. Firstly, the role of early-life conditions is explored in direct and indirect terms with a larger set of indicators than previous analyses, including parental social and health conditions in addition to the individual's initial health status. Secondly, this research analyses the evolution of unhealthy lifestyles, their changes over an extended period of time and their association with health status. Finally, the longitudinal dimension of those data allows using dynamic panel analysis in order to control for unexplained individual heterogeneity and explain impact of past health status.

The structure of the paper is as follows. Section 2 describes the model that is empirically tested. Section 3 describes the National Child Development Study (NCDS) data and the variables of interest. Section 4 presents the empirical results and section 5 concludes.

2. The model

2.1 General health production function

In contrast with Jusot et al. (Jusot et al. 2010), who focused on a reduced-form model of childhood circumstances and lifestyles, we use a full model specification including individual qualification. Our approach also differs from Contoyannis and Jones (2004), Häkkinen et al. (2006), and Balia and Jones (2008), as our health production function includes early-life conditions as a potential determinant for health in addition to education and lifestyles. Furthermore, we built a dynamic model of health using longitudinal data.

The individual health status H can be written using the following health production function:

$$H = f(C, E, L, D, e) \qquad \text{(Eq. 1)}$$

The vector of early-life conditions C consists of a set of variables beyond individual control which may be related to health status. The literature on health determinants suggests an influence of childhood conditions and family background on health status in adulthood see for example Currie and Stabile 2003, Case et al. 2005, Rosa-Dias 2009, Lindeboom et al. 2009, Trannoy et al. 2010). Moreover, initial health such as birth weight and health problems during childhood and adolescence also significantly influence health in adulthood and the most adverse health risks in adulthood tend to be experienced by people having experienced poor health in childhood and adolescence (Moser et al. 2003, Case et al. 2005). The vector E represents individual's education level measured by the highest qualification achieved at age 46 and is not a time-variant variable. Researchers in many countries have found a relevant and persistent association between education and health as measured by various health measures (Grossman 2006). We assume that qualification is a reliable proxy of other social outcomes such as social class, employment status, housing or income. The vector of health-related behaviours L captures individual decisions to invest in health capital, such as lifestyles (Balia and Jones 2008, Contoyannis and Jones 2004, Häkkinen et al. 2006, Rosa-Dias 2009, Jusot et al.

2010). The vector D represents demographic characteristics which are biological determinants of health status, only captured by gender in cohort data. Finally, the residual term e represents unobserved heterogeneity related to other random factors, which cannot be captured by observed determinants.

More concretely, let us assume that health of individual i at wave t is measured by a continuous latent variable H_{it}^{*} which is measured using a binary variable H_{it} as follows:

$H_{it}^{*} \geq 0$ when $H_{it} = 1$

$H_{it}^{*} < 0$ when $H_{it} = 0$

The general health production model can be written as follows:

$$H_{it}^{*} = a_1 C_i + a_2 D_i + b_1 E_i + c L_{it-1} + u_i + v_{it}$$

with $i = 1, \dots, N$ and $t = 1, \dots, T_i$ (Eq. 2, model 1a)

We include lagged values of lifestyles into the model $L_{i,t-1}$ as past lifestyles are more likely to be important for health status than just acquired lifestyles. The time variant individual specific error term is captured by v_{it}, which is assumed to be normally distributed and uncorrelated across individuals and waves. The individual time invariant unobserved effect is captured by u_i ; it captures unobserved individual characteristics, such as genetics and personality traits. We firstly estimate a static model with random effects assuming that the errors are independent over time and uncorrelated with the explanatory variables. This model provides us with base estimates, with which we can compare results from models that incorporate unobserved heterogeneity and state dependence.

Model 1a does not allow us to address several important issues with relevant impact on the health determinants. Firstly, we do not know whether the model variables appropriately account for any unobserved individual characteristics that also influence time-variant variables. Especially, if the past lifestyles are correlated with u_i, we would expect to overestimate the effect of lifestyles in model 1a. Secondly, early-life conditions, education, and past lifestyles may affect current health directly but also indirectly, namely through affecting past health. If this is true, we would expect past health to influence current health, and the direct effects of early-life conditions, education, and past

lifestyles on current health to weaken or even disappear. Finally, the initial health state is likely to be not randomly assigned to the individual. To address the first issue, we use a random effect Probit specification allowing u_i and v_{it} to be correlated and introduce lifestyles averaged over time $\bar{L}_i$ as a set of controls for unobserved heterogeneity (Mundlak 1978). We now estimate the effects of changing lagged lifestyles on health but holding the average fixed[1] in the model 1b. While this model addresses part of the problem of unobserved heterogeneity, a dynamic model of health that incorporates both past health and unobserved effects is required to address the remaining issues. The inclusion of a rich set of early-life conditions in the model can be interpreted as a particular specification of the individual component. A well-explained vector strongly contributes to the reduction of the correlation between individual effects and initial conditions as it minimises unobserved time-invariant characteristics affecting individual outcomes at each point of time. Nevertheless, we need to account the potential endogeneity bias related to the respective correlations between early-life conditions, lifestyles and education with past health, which can be ruled out using a dynamic specification and introducing past health $H_{i,t-1}$ into the health production function. The introduction of past health status in our empirical model allows us to capture the state dependence in health reports and strongly reduces the impact of individual heterogeneity. In a dynamic context, initial health $H_{i,0}$ is likely to be correlated with unobserved heterogeneity u_i affecting H_{it} and if $H_{i,0}$ is considered exogenous this will lead to inconsistent estimators. We follow the alternative approach suggested by Wooldridge[2] (2002), which requires to specify the distribution of u_i given $H_{i,0}$ and other exogenous variables and so, include at least the first value of the independent variable, $H_{i,0}$.

[1] Lifestyles could therefore be regarded both as a measure of lifestyles shocks on health via the past lifestyle variables and as a measure of long-term or "permanent" lifestyles on health via the average lifestyle. Nevertheless, from our point of view the follow-up of lifestyles being limited to four points of time and to the use of binary lifestyle variables does not justify to interpret the effects of lifestyles on health in terms of permanent and transitory effects.

[2] Two other methods to address initial conditions problems could have been considered: Heckman (1981) and Orme (2001). The former suggests approximating the reduced form $f(H_{i,0}|x_i, u_i)$ and then specifying$f(u_i|x_i)$; $f(H_{i0}, \dots, H_{iT}|x_i)$ is then given by integrating out u_i (where x_i includes all the regressors). The two main difficulties with this method are specifying the distribution of initial health, and computing time. As for Orme (2001), he suggested a two-step bias corrected procedure that is locally valid when the correlation between H_{i0} and H_{it} is approximated to zero. A couple of recent works compared the relative performance of the three methods. Whereas Miranda (2007) concluded that the Heckman method delivers estimators that are hardly subject to bias and that are estimated with high precision; Arulampalam and Stewart (2009) concluded that none of the three estimators dominates the other two in

The ultimate latent health model that we estimate can be written as follows:

$H_{it}^{*} = a_1 C_i + a_2 D_i + b_1 E_i + c_1 L_{it-1} + c_2 \bar{L}_i + d_1 H_{it-1} + d_2 H_{i0} + u_i + v_{it}$

with $i = 1, \dots, N$ and $t = 1, \dots, T_i$ (Eq. 3, model 1c)

Using this dynamic model of health status over the life-cycle, we are now particularly interested in understanding the interdependent relationships linking the sets of health determinants: early-life conditions, education, and lifestyles. Therefore, we complement this primary specification with a mediating specification that aims to describe whether early-life conditions influence health directly or indirectly, that is via affecting education and lifestyles.

2.2 Mediating effects identification

The mediating specification aims to identify whether explanatory variables influence health directly or indirectly, that is by affecting or being affected by another explanatory variable. Let us firstly consider a more general case where individual health status H is defined according to a set of variables C, such as her early-life conditions, and a set of variables L, such as her education or lifestyles.

$H = F(C, L, \varepsilon)$ (Eq. 4)

We consider that L potentially mediates the relationship between C and H. For example, the early-life condition mother's qualification may affect adult health through an effect on individual's qualification attainment (see Figure 1) as exhibited in the *pathway model* that has been well-studied in both economic and epidemiological studies e.g. (Marmot et al. 2001, Case et al. 2005, Trannoy et al. 2010). We aim to evaluate the full effect of X on H such as follows

$H = \alpha_1 C + \varepsilon_1$ (Eq.5a)

However, as we are in a full model specification we cannot ignore the role played by L on H:

$H = \alpha_2 C + \beta L + \varepsilon_2$ (Eq.5b)

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all cases. Moreover, the authors found that it is advantageous to allow for correlated random effects using the approach of Mundlak (1978).

The total effect of C on H is measured by α_1 whereas the direct effect of C on H is measured by α_2. The difference between α_1 and α_2 represents the mediating effects of C on H that work through L. Moreover, the mediation effects can be written using the following auxiliary equation, where φ captures the effect of C on L:

$$L = \varphi C + \omega \quad \text{(Eq. 6)}$$

Using a linear model to estimate the relationship between L and C, we can rewrite (Eq. 5b) as follows:

$$H = \alpha_2 X + \beta(\varphi X + \omega) + \varepsilon_2$$

$$H = (\alpha_2 + \beta.\varphi)X + \beta.\omega + \varepsilon_2 = (\alpha_2 + \beta.\varphi)X + \varepsilon \quad \text{(Eq. 5c)}$$

Where $\beta.\varphi$ represents the mediating effect, namely the indirect effect of C on H working through L. Prior to the estimation of equation Eq. 5c, estimated residuals $\hat{\omega}$ must be estimated from the auxiliary equations Eq. 6.

Following Bernt-Karlson et al. (2010), we can express the respective direct, mediating and total effects of C on H as follows:

Direct effects	α_2
Mediating effects	$\beta.\varphi$
Total effects	$\alpha_1 = \alpha_2 + \beta.\varphi$

Let us now consider our present study, using Figure 1, the set of variables C could represent early-life conditions and the set of variables L could be both education and lifestyles. In addition, the dashed arrow in Figure 1 suggests that the set of variables C could represent both early-life conditions and education, and the set of variables L be lifestyles only, hence there are potentially two layers of mediating effects to distinguish: mediating effects between early-life conditions and health working through education and lifestyles (mediation 1), and mediating effects between education and health working through lifestyles (mediation 2). The two different mediating specifications will be tested and compared. In concrete terms, Eq.5b corresponds to the general health production function (Eq. 3, model 1c) whereas the mediating specification is a two-step estimation based on auxiliary

equations and then the estimation of the health production function described in (Eq. 5c). The sets of auxiliary equations being estimated can be written as follows:

$$E_i = \varphi_1 C_i + \varphi_2 D_i + e_i \qquad \text{(Eq. 6a)}$$

$$L_{it} = \varphi_1^a C_i + \varphi_2^a D_i + l_i \qquad \text{(Eq. 6b, } \textit{mediation 1}\text{)}$$

$$L_{it} = \varphi_1^a C_i + \varphi_2^a D_i + \varphi_3^a E_i + l_i \qquad \text{(Eq. 6b, } \textit{mediation 2}\text{)}$$

$$\bar{L}_i = \varphi_1^b C_i + \varphi_2^b D_i + \bar{l}_i \qquad \text{(Eq. 6c, } \textit{mediation 1}\text{)}$$

$$\bar{L}_i = \varphi_1^b C_i + \varphi_2^b D_i + \varphi_3^b E_i + \bar{l}_i \qquad \text{(Eq. 6c, } \textit{mediation 2}\text{)}$$

If we replace those auxiliary equations into equation Eq.3, the health production function in the mediating specification becomes:

$$H_{it}^* = (a_1 + \varphi_1)C_i + (a_2 + \varphi_2)D_i + b_1\hat{e}_i + c_1\hat{l}_{it} + c_2\bar{\hat{l}}_i + d_1 H_{it-1} + d_2 H_{i0} + u_i + v_{it}$$

with $i = 1, \dots, N$ and $t = 1, \dots, T_i$ (Eq. 5d, *mediation 1*)

$$H_{it}^* = (a_1 + \varphi_1)C_i + (a_2 + \varphi_2)D_i + \varphi_3 E_i + b_1\hat{e}_i + c_1\hat{l}_{it} + c_2\bar{\hat{l}}_i + d_1 H_{it-1} + d_2 H_{i0} + u_i + v_{it}$$

with $i = 1, \dots, N$ and $t = 1, \dots, T_i$ (Eq. 5d, *mediation 2*)

where, $\hat{e}_i$, $\hat{l}_{it}^{a1}$, $\hat{l}_i^{b1}$ (respectively $\hat{l}_{it}^{a1}$, $\hat{l}_i^{b1}$ in mediation 2) represent the estimated residuals in each auxiliary equation and can be written as follows:

$$\hat{e}_i = E(e_i | C_i, D_i)$$

$\hat{l}_{it}^{a1} = E(l_i | C_i, D_i)$ (*mediation 1*) and $\hat{l}_{it}^{a2} = E(l_i | C_i, D_i, E_i)$ (*mediation 2*)

$\hat{\bar{l}}_i^{b1} = E\left(\bar{l}_i \middle| C_i, D_i\right)$ (*mediation 1*) and $\hat{\bar{l}}_{it}^{b2} = E\left(\bar{l}_i \middle| C_i, D_i, E_i\right)$ (*mediation 2*)

These estimated residuals[3] are estimated as linear probability models for time-invariant outcomes and pooled linear probability models otherwise. The estimated residuals are then introduced in the health equation in replacement of the actual explanatory variables as in Eq. 5c. Using Eq. 5d, we can express the respective direct, mediating (indirect) and total effects of early-life conditions and education on health as follows:

[3] For binary outcomes one estimated residual will be predicted from the OLS estimation whereas for discrete outcomes in k categories, $k - 1$ estimated residuals will be predicted corresponding to each category except the category taken as reference.

Direct effects of early-life conditions on health	a_1
Total mediating effects of early-life conditions on health	$\varphi_1 = b_1\varphi_1 + c_1\varphi_1^a + c_2\varphi_1^b$
Mediating effects via education	$b_1\varphi_1$
Mediating effects via lifestyles	$c_1\varphi_1^a + c_2\varphi_1^b$
Mediating effects of education on health via lifestyles	$\varphi_3 = c_1\varphi_3^a + c_2\varphi_3^b$

2.3 Health determinants decomposition

The second part of our empirical analysis inquires to which extent the account of mediating effects influences the contribution of each determinant to health disparities. Shorrocks (1982) showed that if we are interested in an absolute measure of inequality, i.e. a measure invariant to one translation, the variance is a good index and its natural decomposition presents the desired properties. The alternative specifications of the health production function we considered in sections 2.1 and 2.2 are based on strictly identical regressors and so, they have the same variance. The variance of both models is estimated using bootstrap method to assess variability of estimated coefficients from the panel random effect Probit (using 300 replications).

In a linear case, the share of variance explained for example by early-life conditions C_i simply consists in the share of the R^2 of the model which is explained by C_i. In a non linear context it is not straightforward as H_{it}^* can only be measured as a prediction and, ω_i and ε_{it} are defined as independent of the set of K explicative variables. A variance estimated from the data is attributed to the time invariant individual error term ω_i whereas the time variant individual error term ε_{it} has a variance normalised to be equal to 1 in the case of a Probit model. We use the pseudo R^2 proposed by McKelvey and Zavoina (1975) in order to measure the share of variance explained by the variable X^k having an associated coefficient η^k, which is based on predictions of the latent endogenous variables:

$$\widehat{H}_{it}^* = \sum \eta^k X_{it}^k \qquad \text{(Eq. 7)}$$

Assuming that the variance of the error term u_i and v_{it} follows a normal distribution in this random effect Probit model, we can write:

$$R^2 = \frac{V(\widehat{H}^*)}{V(\widehat{H}^*)+\sigma_u+1} \qquad \text{(Eq. 8)}$$

Given the longitudinal data, the variance of the latent health variable can either be decomposed directly using the explained variance of the latent health variable, as measured by the pseudo-R^2 based on all the waves. The share of inequality associated to the variable X^k at time t can thus be written as:

$$I(X^k) = \frac{V(\eta^k X^k)}{V(\sum_{k=1}^{K} \eta^k X^k)} \quad \text{(Eq. 9)}$$

3. The National Child Development Study

The National Child Development Study (NCDS) is a continuing, multi-disciplinary longitudinal study which focuses on all the people born in one week in March 1958 in England, Scotland and Wales. Information was gathered from almost 17,500 babies. Following the initial birth survey in 1958, there have been seven attempts to trace all members of the birth cohort in order to monitor their physical, educational, social and economic development. These were carried out in 1965, 1969, 1974, 1981, 1991, 1999/2000 and 2004. For the birth survey, information was obtained from the mother and from medical records by the midwife. For the purpose of the first three NCDS surveys, information was obtained from parents, head teachers and class teachers, the schools health service and the subjects themselves (who completed tests of ability and, latterly, questionnaires). In the 1981 and later surveys, information was gathered by professional survey research interviewers. In 1981 information was obtained from cohort members and from the 1971 and 1981 Censuses. In the 1991 survey there was a professional interview with the cohort member along with self-completion questionnaires from NCDS subjects and husbands, wives, and cohabiters. For the 1999-2000 sweeps, information was obtained from cohort members by interviewer and self-completion using CAPI. The 2004 survey was administered by telephone.

3.1 The sample

For the purpose of our study, we focus on the four last sweeps of the cohort (t= 0, ..., 3) in order to have repeated measures of both lifestyles and health status as an adult. Data collected before age 23 are used to inform individual early-life conditions (see in Appendix Table A.I). We have excluded cohort members who missed at least one of the four first sweeps in order to ensure a description of childhood conditions with a limited non response. The balanced sample for which individuals have fully informed health status and lifestyles in all the sweeps 4 to 7 contains 4,480 individuals whereas the unbalanced sample contains between 5,900 and 7,900 individuals. A description of the distribution of relevant variables in the balanced sample at t=0 is available in appendix (see Table A.II)

3.1.1 Health variable

The NCDS includes only one repeated measure of the respondent's health in the cohort, namely self-assessed health (SAH). Respondents are asked to rate their own health on a four or five point categorical scale ranging from poor (sweeps 4, 5 and 6) or respectively very poor (wave 7) to excellent health status. Given the changes in scale in the variable over the different waves, we use SAH as a binary variable[4] which takes the value one if the individual rates her health as good health or higher, and zero if she rates her health less than "good". Self-assessed health has been shown to be a good predictor of mortality, morbidity and subsequent use of health care (Idler and Benyamini 1997). The distribution of health status in the balanced sample shows the age effect on health status over the life-cycle (see Table A.III). Whereas good health represents 92.7% of respondents at 23 years old, the proportion of respondents reporting a good health declines to 78.3% at 46 years old. Between the first three sweeps the mean is declining by a constant rate of 4 percentage points. There is a break with a decrease of 6 percentage points between the two last sweeps despite they are separated by four years only. This difference could be explained by an increasing effect of ageing on

[4] Dichotomisation was also required as the necessary condition on the proportional odds assumption for ordinal Probit models turns out not found to be valid.

health when the cohort member enters her forties. This shift could also come from the change in the categorical scale of self-assessed health between sweep 6 and sweep 7 and this latter issue is minimised by the dichotomisation of health.

3.1.2 Socioeconomic status

The NCDS provides several current social characteristics. Education is provided at each wave and we use the highest qualification achieved over the period, generating a three categories discrete variable: having a qualification lower than O-level, having O-level or A-level, and having a qualification higher than A-level. About one fifth of respondents have a qualification lower than secondary school.

3.1.3 Lifestyles variables

The NCDS includes a longitudinal follow-up of lifestyles and health records at age 23, age 33, age 42 and age 46. We consider four lifestyles binary variables (presented in Table A.IV). Exercising indicates whether the cohort member is regularly doing exercise or sports; it equals one if the cohort member did exercise at least once in the last four weeks and zero otherwise. Non smoking informs whether the cohort member is a current smoker at the time of the wave; it equals one if she does not currently smoke and zero otherwise. Drinking prudently is a gender-specific indicator based on the number of units of alcohol drinks taken the week before the interview. Males are considered to drink prudently if they drank between 0 and 21 units of alcohol whereas it is between 0 and 14 units a week for females (Working Party of the Royal College of Physicians UK 2001, Balia and Jones 2008). The binary variable takes the value one if the respondent drinks prudently and zero otherwise. The absence of obesity is the fourth lifestyle that we consider. Obesity may appear as an intermediated or genetic outcome of health and not a pure lifestyle. Given that we can control the genetic and the family transmitted effect on obesity using the respondent's obesity status when she was 16, the absence of obesity will thus captures aggregated effects of lifestyles. Absence of obesity

is constructed using the reported height and weight and calculating individuals' body mass index (BMI[5]). The absence of obesity is a binary variable taking the value one if the cohort member's BMI is strictly lower than 30 and zero otherwise.

3.1.4 Early-life conditions

The vector of early-life conditions that we consider has three main types of variables: social conditions in childhood, parents' health and health-related behaviours, and child and adolescence health. Social conditions in childhood include the father's social class at the time of birth, the father and the mother's education level, and parental reports of financial hardships when the cohort member was 16. Father's social class is described in three large categories: a top class (I/II) including professional and managerial or technical workers, a middle class (III) including skilled workers and armed services, and a low class (IV/V) including partly skilled and unskilled workers and a fourth category is added if the mother reported no male figure in the household at the time of birth. Parental education consists in a two categories variable: parents who dropped out from school before or at the minimum age (14-15 years depending on the year of birth) and parents who were still at school after this age. Parents' health is measured by parental report of chronic illness when the cohort member was 16 years old. Regarding parents' health related lifestyles, we used a smoking indicator for each parent taking the value 1 if reported to be a smoker. Respondent's health in childhood and adolescence are used as control variables but also as achievement variables since they may represent health-related difficulties from the living environment during childhood. We use the same approach as Case et al. (2005) who considered the report of at least one chronic condition at 16 as well as a birth weight below 2.5kg as health indicators before adulthood using the same dataset. Furthermore, we include obesity at 16 years old. We have computed BMI using medical assessment

[5] BMI in kg/m2= weight/height²

of height and weight and evaluated obesity level using gender-specific thresholds values found to be a good predictor of obesity at 18 (Lahti-Koski and Gill 2004).

4. Results

4.1 Random effect dynamic panel Probit results

The results of the random effect panel Probit of the general specification are presented in Table I. Three different models are reported: model 1a and model 1b are static models with random effect with model 1b including the average individual lifestyles over the studied period; whereas model 1c is a dynamic random effects Probit model.

Table I. Random effect Probit models results

The results show that several early-life conditions have a statistically significant effect on the probability to report good health regardless of the model. Individuals whose father belonged to the lowest social class, namely partly skilled and unskilled workers as well as individuals who had no father at the time of their birth are significantly less likely to report good health. Similarly, the experience of financial hardship during childhood has a significant and negative effect on reports of good health. The mother's education level is also found as a statistically significant determinant of poor health reports whereas the father's education is not significant in any of the models. This may be explained by father's social class being significant and so, absorbing the effect of father's education on descendant's health. Unlike mother's illness, father's illness significantly reduces the probability to report good adult health. Mother's smoking behaviour appears to be significant for descendant's report of good health but the significant level weakens in the dynamic model. Individual education level is also found statistically and significantly associated with health: low qualification is negatively associated with the report of a good health. Regarding lifestyles variables, the four lagged lifestyles are significantly associated with reports of good health in model 1a (at the

10% level for drinking prudently). However as soon as average lifestyles are added to the model, the lagged lifestyles are not significant for health anymore. The average behaviours in exercise, absence of obesity and to a lesser extent the absence of smoking are found strongly and significantly associated with reports of good health, whereas the average behaviour towards drinking is significant at the 10% level only.

This first table of results emphasises the relevance of a dynamic model; past health status and initial health in model 1c are significantly associated with reports of good health and the share of individual unexplained heterogeneity addressed by the model equals 36% of the unexplained heterogeneity. Furthermore, when past and initial health variables are introduced a reduction in the magnitude of the model's estimated coefficients is observed.

4.2 Auxiliary equations estimations

Prior to the mediating specification, the auxiliary equations are estimated. The results are presented in Appendix (Table A.V to Table. IX). Education is estimated as a linear regression model for the two binary variables within model 1c (having a qualification lower than O-level and having a qualification between O-level and A-level). Having a qualification lower than O-level is found positively and significantly associated with low father's social class and absence of a male head at the time of birth, experience of financial hardship, both parents' having left school before or at the minimum legal age and both parents' being smokers; presence of a chronic disease at 16 years old, obesity at 16, and low birth weight. Inversely, having a qualification between O-level and A-level is found positively and significantly associated with low father's social class, both parents' having left school before or at the minimum legal age. On the other hand it is found negatively associated with having experienced financial hardships; mother's smoking and having a chronic disease at 16 years old. The four lifestyles are estimated as OLS models on the pooled sample. Exercising is significantly and negatively associated with reports of chronic disease at 16, father's low social class,

both parents' low education and experience of financial hardship. When education is controlled (Table A.VI, column b), both low and middle qualifications are found significantly and negatively associated with regular exercise. Moreover, it weakens the significant effect of financial hardships, father's social class and parental education. With regard to smoking behaviour, the absence of smoking is strongly and negatively associated with both parents' smoking, financial hardship at 16, father's low social class and the absence of male head at the time of birth. When individual education is introduced (Table A.VII, column b), both qualification lower than O-level and between O-level and A-level are found negatively correlated with the absence of smoking. Moreover, the inclusion of individual education absorbs the effect of father's social class and weakens the impact of financial hardship. Drinking prudently is strongly higher among female and found negatively and significantly correlated with father's smoking. Noticeably, having a father who was in lower social class and low birth weight appear to be positively associated with prudent drinking. Middle qualification is found to be negatively associated with drinking prudently when education is introduced in the auxiliary equation (Table A.VIII, column b). Finally, the absence of obesity at 16 is statistically associated with non obesity in adulthood; in addition, mother's smoking, father's SES and mother's low education are found statistically significantly for the reduction of the absence of obesity. When education is included within the auxiliary equation (Table A.IX, column b), low individual education appears to be significantly and negatively associated with the absence of obesity and the introduction of individual education erases the significant effect of parents' education which was previously observed.

4.3 Random effect dynamic panel Probit results: mediating specification

The results of the mediating specifications of the health production function are presented in Table II. They have been obtained by replacing actual variables of education and lifestyles by the

estimated residual terms of the different auxiliary equations whose results were described in the previous section. The two mediating specifications are presented.

Table II. Random effect Probit models coefficients of the mediating specifications

Noticeably, the estimated coefficients associated to education and lifestyles in mediation 1 and to lifestyles only, in mediation 2 are strictly identical with the estimated coefficients in model 1c (see Table I) as expected when comparing Eq. 3 and Eq. 5d. The results of the mediating specification permit confirming the existence of indirect effect of early-life conditions variables on health over the life cycle in addition to their direct effect previously shown by the initial specification. There is a clear increase in the magnitude of all the estimated coefficients associated to early-life conditions in the mediating specification compared to the results of model 1c. The effect of early-life conditions is magnified when mediating effects with other health determinants such as education and lifestyles are disentangled. Moreover, this specification highlights the indirect effect of mother's smoking status on cohort member's health, which is found significantly associated with health in the two mediation specification but not in model (1c). The mediating specification 2 also emphasised that education level has a direct and an indirect effect on health as education as the estimated coefficients associated to the education variables are larger and the significance of having a qualification between O-level and A-level becomes statistically significant. The mediating specifications allow us to evaluate the magnitude of the direct and indirect effects of early-life conditions on adult health, working through education and lifestyles.

Table III presents the magnitude of direct and indirect effects of each of the variables within the vector of early-life conditions. The absence of male head at the time of birth has the highest direct effect on adult health. The experience of financial hardships during childhood is also an important early-life condition influencing adult health directly. The absence of obesity at 16 years has both relevant direct and indirect effects on adult. As an indirect effect, it essentially works

through lifestyles. The indirect effect of father's smoking also appears to work mainly through lifestyles whereas the indirect effect of mother's smoking works through both education and lifestyles. Noticeably, most of the indirect effects related to the father's SES and both parents' education level work through education. Finally, individual qualification influences health both directly and indirectly through lifestyles.

Table III. Early-life conditions direct and indirect effects on health

4.4 Decomposition of health inequality

Table IV presents the results of the decomposition of the variance of the predicted latent health within the longitudinal panel data analysis for the alternative specifications (model 1c and specification 2).

Table IV. Decomposition of health inequality (with bootstrapped 95% confidence intervals)

The decomposition in the baseline specification shows that the most important contribution to health inequalities comes from the state dependence of health and the initial health, which would explain 33.3% of the variance in the predicted health. Lifestyles are directly explaining 28.5% of health inequalities, which confirm that they are important determinants of health inequalities. Early-life conditions explain about 18% of health inequalities. If we add their indirect contributions to health inequalities, as done in the mediating specification, the relative contribution of early-life conditions increases and would represent 24% of health inequalities. This increase underlines the importance of the mediating effects of early-life conditions with education and lifestyles. On the contrary, the contribution of lifestyles on health inequalities reduces and would represent 22.2% of inequalities. Lifestyles are thus strongly influenced by early-life conditions and to a lesser extent by educational level whose contribution to health inequalities slightly increases in the mediating approach. Comparison between the decompositions of the general specification and the mediating

model suggest that the correlation between early-life conditions, and respectively lifestyles and education, is important. When we purge the contribution of lifestyles to health inequalities from their mediating effect with early-life conditions and education, we reduce their contribution to health inequalities and emphasise the importance of early-life conditions for health inequalities over the life cycle.

We thoroughly studied the share of early-life conditions in health inequalities and explore the relative contribution of social background, parent's health and lifestyles, and initial health to this vector. The decomposition in the general specification model suggests that social background variables are the leading contributing factor, representing about 66.3% of the share of early-life conditions in health inequalities. Parent's health and lifestyles represent about 19.3%.

5. Conclusion

In this paper, we developed a model to evaluate the contribution of several essential determinants of health to health inequalities using a representative cohort of individuals born in 1958 and a unique follow-up of health status, lifestyles as well as a good description of early-life conditions. Our results showed the indirect effects of early-life conditions variables on health over the life cycle in addition to their direct effects. Early-life conditions have a predominant contribution in health inequality when their indirect role on education achievement as an adult and lifestyles were taken into account representing 24% of overall health inequality. This latter result underlines the relevance of mediating effects between the determinants of health and outperforms previous works excluding early-life conditions as a relevant determinant of health inequalities along with socioeconomic factors and lifestyles. Among early-life conditions, social background seems to be the most important determinant of overall health inequalities. Lifestyles show an impressive contribution to health inequalities, namely 28.6%, in the general specification but appear to be determined by both early-life conditions and education, confirming previous results which have underlined social

determinism along with the existence of an accumulation of risk on causal pathways in the life course (Kuh et al. 2003).

The study exhibits among the early-life conditions those which influence adult health directly, indirectly and both directly and indirectly. The absence of father at the time of birth and experience of financial hardships represent the lead factors for direct effect on health. The absence of obesity at 16 influences health both directly and indirectly working through lifestyles. Indirect effects dramatically increase the relative contribution of early-life conditions to health inequalities, since their contribution equals 24% in the mediating model and thus becomes directly comparable to the contribution of lifestyles (22.2%).

Finally, the dynamic panel analysis permits controlling a large part of individual unexplained heterogeneity as well as the important effect of health state dependence over time. Our study has some limitations. The inequality measure is based on the explained part of the variance that is allowed by the model specifications. According to the pseudo-R^2 that is built using the variance of the latent variable, we would be able to explain about 18%. Therefore the unexplained health inequality remains very large. The panel data perspective also presents several limits. The first problem is the presence of attrition due to mortality in the cohort that we have ignored in the analysis. This leads us to an underestimation of the effect of early-life conditions, adult socioeconomic factors and lifestyles on health inequality as we worked on a selected sample of British people still alive at 46 years old. We did investigate mortality in our data and we found that mortality rate appears to be more important before age 23 than between age 23 and age 46. Finally, the NCDS cohort has a singular structure as the different waves are not equidistant in time. In particular there is a four year interval between the two last sweeps whereas there were about ten years between the past sweeps. We tried to catch this effect by introducing a year dummy into the

models. Therefore, the estimated coefficients in the models can be interpreted as a mean of the effects of lifestyles, education, and early-life conditions over time.

The study of the social determinants of health inequality together with lifestyles and the evaluation of their respective contribution to the magnitude of health inequality is particularly relevant for policy makers. The legitimacy of policies to tackle health inequalities is related to the relative contribution of each broad factor. Early-life conditions, which are factors that cannot be chosen by the individual, appear to be leading factors of health disparities. They are thus considered as the most illegitimate sources of health inequalities (Roemer 1998; Dworkin 1981) and would undoubtedly justify policy interventions that aim to compensate individual for inequalities of opportunity in health.

6. References

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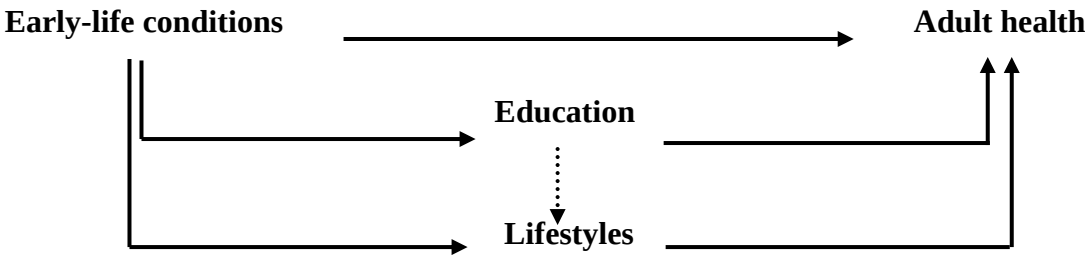
7. Figures

Fig. 1 Early-life conditions, socioeconomic factors, lifestyles and later-life health status



8. Tables

Table I. Random effect Probit models results with estimated coefficients (with bootstrapped 95% confidence intervals)

	Model 1a *Static model*			**Model 1b** *Mundlak specification*			**Model 1c** *Dynamic model*		
Variables	**Coef.**		**S.E.**	**Coef.**		**S.E.**	**Coef.**		**S.E.**
Gender Male	0,084	*	0,047	0,042		0,050	0,031		0,043
Fathers' social class (Ref.: I and II - Professional and managerial/technical)									
III - Skilled	-0,105		0,081	-0,099		0,077	-0,073		0,066
IV and V - Partly skilled and unskilled	-0,269	***	0,086	-0,258	***	0,095	-0,208	**	0,085
No male head	-0,503	***	0,142	-0,487	***	0,134	-0,377	***	0,120
Financial hardship (Ref.: None)									
Yes	-0,347	***	0,084	-0,340	***	0,093	-0,252	***	0,077
Non response	0,064		0,175	0,099		0,172	0,118		0,161
Father's education (Ref.: beyond the min age)									
Before or at the min age	-0,081		0,076	-0,068		0,074	-0,045		0,065
Mother's education (Ref.: beyond the min age)									
Before or at the min age	-0,168	**	0,071	-0,155	**	0,075	-0,146	**	0,061
Parental illness (Ref.: None)									
Father's illness	-0,236	***	0,090	-0,227	**	0,094	-0,171	**	0,073
Mother's illness	-0,164		0,105	-0,144		0,111	-0,121		0,088
Parental smoking (Ref.: None)									
Father's smoking	0,086		0,055	0,109	**	0,055	0,072		0,044
Non response	0,024		0,103	0,020		0,110	-0,012		0,095
Mother's smoking	-0,142	***	0,052	-0,126	**	0,053	-0,076	*	0,046
Non response	-0,117		0,152	-0,114		0,155	-0,068		0,110
Chronic condition at 16 (Ref.: None)									
Yes	-0,112		0,084	-0,085		0,069	-0,012		0,064
Non response	0,194		0,164	0,173		0,161	0,127		0,139
Low birth weight	-0,127		0,113	-0,139		0,120	-0,079		0,103
Obesity at 16 (Ref.: Yes)									
No	0,068		0,202	-0,269		0,208	-0,307	*	0,184
Non response	-0,203		0,144	-0,187		0,147	-0,166		0,124
Educational level (Ref.: Higher than A-level)									
Before O-level	-0,394	***	0,073	-0,248	***	0,077	-0,207	***	0,067
O-level or A-level	-0,106	*	0,060	-0,051		0,061	-0,032		0,052
Lagged lifestyles									
Exercising	0,172	***	0,046	-0,029		0,042	-0,042		0,049
No smoking	0,319	***	0,046	0,083		0,070	0,072		0,070
Drinking prudently	0,107	*	0,063	0,032		0,072	0,033		0,064
No obesity	0,360	***	0,072	-0,026		0,084	-0,052		0,083
Mean lifestyles									
Exercising				0,746	***	0,095	0,566	***	0,092
No smoking				0,354	***	0,091	0,226	**	0,091
Drinking prudently				0,237	*	0,131	0,222	*	0,122
No obesity				0,912	***	0,136	0,760	***	0,116
Lagged health status							0,311	***	0,075
Initial conditions (Health status at 23)							1,007	***	0,100
Time dummies (Ref.: t=3)									
t=1	0,682	***	0,046	0,668	***	0,047	0,579	***	0,050
t=2	0,353	***	0,040	0,385	***	0,039	0,341	***	0,037
$V(\hat{H}^*)$	0,244			0,332			0,360		
σ_ω	1,129			1,126			0,639		
$\rho^{\#}$	0,530			0,530			0,390		
R^2 (McKelvey and Zavoina)	0,103			0,135			0,180		

[#] Share of individual unexplained heterogeneity measured by the share of σ_ω in the total unexplained variance ($\sigma_\omega + 1$)
Significance levels: *** 1%, **5%, *10%.

Table II. Random effect Probit models coefficients of the mediating specifications (with bootstrapped 95% confidence intervals)

	Mediating specification 1			Mediating specification 2		
Variables	**Coef.**		**S.E.**	**Coef.**		**S.E.**
Gender Male	0,081	*	0,044	0,081	*	0,042
Fathers' social class (Ref.: I and II - Professional and managerial/technical)						
III - Skilled	-0,104		0,065	-0,104		0,064
IV and V - Partly skilled and	-0,280	***	0,084	-0,280	***	0,077
No male head	-0,463	***	0,122	-0,463	***	0,121
Financial hardship (Ref.: None)						
Yes	-0,348	***	0,075	-0,348	***	0,070
Non response	0,063		0,140	0,063		0,153
Father's education (Ref.: beyond the min						
Before or at the min age	-0,093		0,064	-0,093		0,061
Mother's education (Ref.: beyond the min						
Before or at the min age	-0,199	***	0,059	-0,199	***	0,062
Parental illness (Ref.: None)						
Father's illness	-0,192	**	0,077	-0,192	**	0,080
Mother's illness	-0,141		0,095	-0,141		0,097
Parental smoking (Ref.: None)						
Father's smoking	0,021		0,045	0,021		0,046
Non response	-0,025		0,089	-0,025		0,095
Mother's smoking	-0,123	***	0,046	-0,123	***	0,044
Non response	-0,083		0,111	-0,083		0,118
Chronic condition at 16 (Ref.:						
Yes	-0,060		0,069	-0,060		0,062
Non response	0,151		0,139	0,151		0,134
Low birth weight	-0,096		0,101	-0,096		0,096
Obesity at 16 (Ref.: Yes)						
No	0,183		0,166	0,183		0,170
Non response	-0,219	*	0,127	-0,219	*	0,122
Educational level (Ref.: Higher than A-level)						
Before O-level	-0,207	***	0,065	-0,404	***	0,067
O-level or A-level	-0,032		0,051	-0,108	**	0,050
Lagged lifestyles						
Exercising	-0,042		0,046	-0,042		0,047
No smoking	0,072		0,069	0,072		0,070
Drinking prudently	0,033		0,070	0,033		0,068
No obesity	-0,052		0,087	-0,052		0,089
Mean lifestyles						
Exercising	0,566	***	0,084	0,566	***	0,094
No smoking	0,226	***	0,086	0,226	**	0,088
Drinking prudently	0,222	*	0,115	0,222	*	0,114
No obesity	0,760	***	0,127	0,760	***	0,127
Lagged health status	0,311	***	0,074	0,311	***	0,078
Initial conditions (Health status at	1,007	***	0,099	1,007	***	0,097
Time dummies (Ref.: t=3)						
t=1	0,577	***	0,044	0,577	***	0,046
t=2	0,337	***	0,036	0,337	***	0,039
$V(\widehat{H}^*)$	0,360			0,360		
σ_ω	0,639			0,639		
$\rho^{\#}$	0,390			0,390		
R^2 (McKelvey and Zavoina)	0,180			0,180		

[#] Share of individual unexplained heterogeneity measured by the share of σ_ω in the total unexplained variance ($\sigma_\omega + 1$)
Significance levels: *** 1%, **5%, *10%.

Table III. Early-life conditions direct and indirect effects on health

Variables	Direct Effect a_1	Indirect effect via education $b_1\varphi_1$	Indirect effect via lifestyles $c_1\varphi_1^a + c_2\varphi_1^b$	Total Effect $a_1 + \varphi_1$
Fathers' social class				
III – Skilled	-0,073	-0,021	-0,010	-0,104
IV and V - Partly skilled and unskilled	-0,208	-0,059	-0,013	-0,280
No male head	-0,377	-0,050	-0,036	-0,463
Financial hardship				
Yes	-0,252	-0,073	-0,023	-0,348
Non response	0,118	-0,020	-0,035	0,063
Father's education				
Before or at the min age	-0,045	-0,036	-0,011	-0,093
Mother's education				
Before or at the min age	-0,146	-0,040	-0,013	-0,199
Parental illness				
Father's illness	-0,171	-0,002	-0,019	-0,192
Mother's illness	-0,121	0,002	-0,022	-0,141
Parental smoking				
Father's smoking	0,072	-0,014	-0,037	0,021
Non response	-0,012	0,000	-0,012	-0,025
Mother's smoking	-0,076	-0,026	-0,021	-0,123
Non response	-0,068	-0,020	0,004	-0,083
Chronic condition at 16				
Yes	-0,012	-0,022	-0,026	-0,060
Non response	0,127	-0,013	0,037	0,151
Low birth weight	-0,079	-0,033	0,017	-0,096
Obesity at 16				
No	-0,307	0,035	0,456	0,183
Non response	-0,166	-0,011	-0,041	-0,219
Educational level				
Before O-level	-0,207		-0,197	-0,404
O-level or A-level	-0,032		-0,076	-0,108

Table IV. Decomposition of health inequality (with bootstrapped 95% confidence intervals)

	Over the full period	
Variables	**Mean (%)**	**[95% Conf. Int]**
Baseline specification		
Sex	0,27	[0,24 ; 0,31]
Age	15,12	[14,95 ; 15,28]
Early-life conditions	17,81	[16,23 ; 19,39]
Social background	*11,81*	*[10,97 ; 12,77]*
Parent's health and lifestyles	*3,44*	*[3,10 ; 3,79]*
Initial health	*2,5*	*[2,11 ; 2,88]*
Lifestyles	28,55	[27,36 ; 29,74]
Education	4,92	[4,68 ; 5,17]
Health state-dependence	33,33	[32,78 ; 33,88]
Mediating specification		
Sex	0,65	[0,60 ; 0,69]
Age	15,09	[14,90 ; 15,28]
Early-life conditions	23,75	[22,07 ; 25,43]
Social background	*15,85*	*[14,85 ; 16,85]*
Parent's health and lifestyles	*4,67*	*[4,26 ; 5,08]*
Initial health	*3,23*	*[2,89 ; 3,58]*
Lifestyles	22,16	[20,99 ; 23,34]
Education	5,29	[5,10 ; 5,47]
Health state-dependence	33,06	[32,49 ; 33,64]

Appendix 1: Supplementary tables

Table A.I The original NCDS sample and the study sample

Year	**1958**	**1965**	**1969**	**1974**	**1981**	**1991**	**1999/00**	**2004**
Cohort member age	Birth	7	11	16	23	33	42	46
Cross-sectional original sample	17,416	15,051	14,757	13,917	12,044	10,986	10,979	9,175
		Early-life conditions			***t=0***	***t=1***	***t=2***	***t=3***
Unbalanced selected sample					7,874	6,956	6,999	5,990
Balanced selected sample						4,480		

Table A.II Descriptive statistics in the balanced sample at t=0

Variables	**N=4480**	**Proportion**
Gender		
Male	2065	46.09 %
Female	2415	53.91 %
Fathers' social class		
I/II - Professional and managerial/technical	854	19.06 %
III – Skilled	2651	59.17 %
IV/V - Partly skilled and unskilled	827	18.46 %
No male head	148	3.30 %
Financial hardship		
Yes	322	7.19 %
No	4055	90.51 %
Non response	103	2.30 %
Father's education		
Minimum schooling age and below	3460	77.23 %
Beyond the min age	1020	22.77 %
Mother's education		
Minimum schooling age and below	3464	77.32 %
Beyond the min age	1016	22.68 %
Parental illness		
Father's illness	314	7.01 %
Mother's illness	218	4.87 %
Parental smoking		
Father's smoking	2432	54.29 %
Non response	292	6.52 %
Mother's smoking	1962	43.79 %
Non response	148	3.30 %
Chronic condition at 16		
Yes	523	11.67 %
No	3475	77.57 %
Non response	482	10.76 %
Low birth weight	214	4.78 %
Obesity at 16		
Yes	59	1.32 %
No	3818	85.06 %
Non response	610	13.62 %
Cohort member's education:		
Higher than A-level	1314	29.33 %
O-level or A-level	2293	51.18 %
Before O-level	873	19.49 %

Table A.III Distribution of health status in the balanced sample

	Age 23 *t=0*	**Age 33** *t=1*	**Age 42** *t=2*	**Age 46** *t=3*
Excellent	45.85%	35.51%	31.54%	32.08%
Good	46.88%	53.21%	53.19%	46.21%
Good health	**92.72%**	**88.73%**	**84.73%**	**78.28%**
Fair	6.70%	10.09%	12.77%	14.98%
Poor	0.58%	1.18%	2.50%	5.07%
Very poor				1.67%
Poor health	**7.28%**	**11.27%**	**15.27%**	**21.72%**

Table A.IV Descriptive statistics of lifestyles variables in the balanced sample

	Age 23 *t=0*	Age 33 *t=1*	Age 42 *t=2*
Exercising	49.51%	79.93%	75.56%
No smoking	64.08%	71.52%	74.24%
Drinking prudently	87.61%	92.57%	84.98%
No obesity	97.37%	89.87%	85.65%

Table A.V Estimated coefficients of auxiliary equation of education (OLS model)

	Before O-level			**O-level or A-level**		
Variables	**Coef.**		**S.E.**	**Coef.**		**S.E.**
Gender Male	-0,057	***	0,011	0,017		0,015
Fathers' social class (Ref.: I and II - Professional and managerial/technical)						
III – Skilled	0,032	*	0,017	0,072	***	0,022
IV and V - Partly skilled and unskilled	0,128	***	0,021	0,066	**	0,027
No male head	0,110	***	0,034	0,048		0,045
Financial hardship (Ref.: None)						
Yes	0,212	***	0,023	-0,120	***	0,030
Non response	0,077	**	0,038	-0,102	**	0,050
Father's education (Ref.: beyond the min age)						
Before or at the min age	0,069	***	0,016	0,080	***	0,021
Mother's education (Ref.: beyond the min age)						
Before or at the min age	0,089	***	0,015	0,039	*	0,020
Parental illness (Ref.: None)						
Father's illness	0,006		0,022	-0,006		0,030
Mother's illness	0,003		0,026	-0,032		0,035
Parental smoking (Ref.: None)						
Father's smoking	0,031	**	0,012	0,015		0,016
Non response	-0,007		0,025	0,030		0,033
Mother's smoking	0,079	***	0,012	-0,049	***	0,016
Non response	0,061	*	0,032	-0,044		0,043
Chronic condition at 16 (Ref.: None)						
Yes	0,067	***	0,018	-0,048	**	0,023
Non response	0,059		0,037	-0,102	**	0,049
Low birth weight	0,094	***	0,026	-0,043		0,035
Obesity at 16 (Ref.: Yes)						
No	-0,104	**	0,049	0,070		0,065
Non response	0,014		0,034	0,055		0,045

Significance levels: *** 1%, **5%, *10%.

Table A.VI Estimated coefficients of auxiliary equation of exercising (OLS model)

Exercising	**Early conditions (a)**			**Early-life condition + Education (b)**		
Variables	**Coef.**		**S.E.**	**Coef.**		**S.E.**
Gender Male	0,095	***	0,009	0,086	***	0,009
Fathers' social class (Ref.: I and II - Professional and managerial/technical)						
III – Skilled	-0,012		0,013	-0,002		0,012
IV and V - Partly skilled and unskilled	-0,052	***	0,016	-0,025		0,016
No male head	-0,040		0,026	-0,018		0,026
Financial hardship (Ref.: None)						
Yes	-0,044	**	0,019	-0,016		0,018
Non response	-0,027		0,028	-0,021		0,029
Father's education (Ref.: beyond the min age)						
Before or at the min age	-0,039	***	0,012	-0,022	*	0,012
Mother's education (Ref.: beyond the min age)						
Before or at the min age	-0,039	***	0,011	-0,021	*	0,011
Parental illness (Ref.: None)						
Father's illness	-0,001		0,018	0,000		0,018
Mother's illness	-0,018		0,021	-0,020		0,020
Parental smoking (Ref.: None)						
Father's smoking	-0,017	*	0,009	-0,011		0,009
Non response	0,015		0,019	0,016		0,018
Mother's smoking	-0,006		0,009	0,004		0,009
Non response	-0,025		0,025	-0,018		0,024
Chronic condition at 16 (Ref.: None)						
Yes	-0,061	***	0,014	-0,053	***	0,014
Non response	0,047		0,030	0,050	*	0,029
Low birth weight	-0,021		0,021	-0,008		0,021
Obesity at 16 (Ref.: Yes)						
No	0,001		0,035	-0,013		0,037
Non response	-0,051	*	0,027	-0,045	*	0,027
Educational level (Ref.: Higher than A-level)						
Before O-level				-0,172	***	0,014
O-level or A-level				-0,069	***	0,010
Time dummies (Ref.: t=3)						
t=1	0,304	***	0,009	0,304	***	0,009
t=2	0,260	***	0,009	0,260	***	0,009

Significance levels: *** 1%, **5%, *10%.

Table A.VII Estimated coefficients of auxiliary equation of non smoking (OLS model)

Non smoking	**Early conditions (a)**			**Early-life condition + Education (b)**		
Variables	**Coef.**		**S.E.**	**Coef.**		**S.E.**
Gender Male	0,014		0,012	0,001		0,012
Fathers' social class (Ref.: I and II - Professional and managerial/technical)						
III - Skilled	-0,017		0,016	-0,002		0,016
IV and V - Partly skilled and unskilled	-0,048	**	0,021	-0,011		0,021
No male head	-0,147	***	0,038	-0,116	***	0,038
Financial hardship (Ref.: None)						
Yes	-0,087	***	0,026	-0,047	*	0,026
Non response	-0,021		0,040	-0,012		0,038
Father's education (Ref.: beyond the min age)						
Before or at the min age	-0,009		0,016	0,016		0,016
Mother's education (Ref.: beyond the min age)						
Before or at the min age	-0,013		0,015	0,013		0,015
Parental illness (Ref.: None)						
Father's illness	-0,029		0,025	-0,028		0,024
Mother's illness	-0,041		0,029	-0,043		0,028
Parental smoking (Ref.: None)						
Father's smoking	-0,081	***	0,013	-0,072	***	0,013
Non response	-0,078	***	0,027	-0,077	***	0,026
Mother's smoking	-0,044	***	0,013	-0,029	**	0,013
Non response	-0,007		0,036	0,003		0,034
Chronic condition at 16 (Ref.: None)						
Yes	-0,004		0,019	0,008		0,019
Non response	0,030		0,043	0,035		0,042
Low birth weight	-0,006		0,029	0,013		0,028
Obesity at 16 (Ref.: Yes)						
No	0,083		0,053	0,064		0,054
Non response	-0,073	*	0,039	-0,064	*	0,038
Educational level (Ref.: Higher than A-level)						
Before O-level				-0,242	***	0,019
O-level or A-level				-0,097	***	0,013
Time dummies (Ref.: t=3)						
t=1	0,074	***	0,006	0,074	***	0,006
t=2	0,102	***	0,006	0,102	***	0,006

Significance levels: *** 1%, **5%, *10%.

Table A.VIII Estimated coefficients of auxiliary equation of drinking prudently (OLS model)

Drinking prudently	**Early conditions (a)**			**Early-life condition + Education (b)**		
Variables	**Coef.**		**S.E.**	**Coef.**		**S.E.**
Gender Male	-0,088	***	0,007	-0,089	***	0,007
Fathers' social class (Ref.: I and II - Professional and managerial/technical)						
III - Skilled	0,011		0,010	0,013		0,010
IV and V - Partly skilled and unskilled	0,025	**	0,012	0,028	**	0,012
No male head	0,011		0,020	0,013		0,020
Financial hardship (Ref.: None)						
Yes	0,008		0,012	0,009		0,012
Non response	-0,022		0,027	-0,023		0,027
Father's education (Ref.: beyond the min age)						
Before or at the min age	0,009		0,010	0,012		0,010
Mother's education (Ref.: beyond the min age)						
Before or at the min age	0,010		0,009	0,012		0,009
Parental illness (Ref.: None)						
Father's illness	0,012		0,012	0,012		0,012
Mother's illness	-0,005		0,016	-0,006		0,016
Parental smoking (Ref.: None)						
Father's smoking	-0,021	***	0,007	-0,021	***	0,007
Non response	-0,030	*	0,016	-0,029	*	0,016
Mother's smoking	-0,008		0,007	-0,008		0,007
Non response	0,021		0,017	0,021		0,017
Chronic condition at 16 (Ref.: None)						
Yes	0,018	*	0,010	0,018	*	0,010
Non response	0,022		0,022	0,021		0,022
Low birth weight	0,030	**	0,013	0,030	**	0,013
Obesity at 16 (Ref.: Yes)						
No	-0,027		0,027	-0,027		0,027
Non response	-0,016		0,021	-0,015		0,021
Educational level (Ref.: Higher than A-level)						
Before O-level				-0,012		0,010
O-level or A-level				-0,017	**	0,008
Time dummies (Ref.: t=3)						
t=1	0,050	***	0,006	0,050	***	0,006
t=2	-0,026	***	0,006	-0,026	***	0,006

Significance levels: *** 1%, **5%, *10%.

Table A.IX Estimated coefficients of auxiliary equation of non obese (OLS model)

Non obese	**Early conditions (a)**			**Early-life condition + Education (b)**		
Variables	**Coef.**		**S.E.**	**Coef.**		**S.E.**
Gender Male	0,011	*	0,006	0,009		0,006
Fathers' social class (Ref.: I and II - Professional and managerial/technical)						
III - Skilled	-0,019	**	0,008	-0,017	**	0,008
IV and V - Partly skilled and unskilled	-0,012		0,011	-0,006		0,011
No male head	0,001		0,018	0,006		0,018
Financial hardship (Ref.: None)						
Yes	-0,013		0,014	-0,004		0,014
Non response	-0,022		0,022	-0,020		0,022
Father's education (Ref.: beyond the min age)						
Before or at the min age	-0,014	*	0,008	-0,010		0,008
Mother's education (Ref.: beyond the min age)						
Before or at the min age	-0,017	**	0,008	-0,012		0,008
Parental illness (Ref.: None)						
Father's illness	-0,019		0,014	-0,019		0,014
Mother's illness	0,004		0,014	0,004		0,014
Parental smoking (Ref.: None)						
Father's smoking	-0,008		0,007	-0,007		0,007
Non response	0,014		0,013	0,014		0,013
Mother's smoking	-0,020	***	0,007	-0,017	**	0,007
Non response	0,008		0,017	0,011		0,017
Chronic condition at 16 (Ref.: None)						
Yes	-0,010		0,011	-0,007		0,011
Non response	-0,009		0,019	-0,007		0,019
Low birth weight	0,009		0,015	0,013		0,015
Obesity at 16 (Ref.: Yes)						
No	0,640	***	0,043	0,636	***	0,043
Non response	0,007		0,017	0,008		0,017
Educational level (Ref.: Higher than A-level)						
Before O-level				-0,045	***	0,011
O-level or A-level				-0,009		0,007
Time dummies (Ref.: t=3)						
t=1	-0,075	***	0,004	-0,075	***	0,004
t=2	-0,117	***	0,005	-0,117	***	0,005

Significance levels: *** 1%, **5%, *10%.