Mediating role of education and lifestyles in the relationship between early-life conditions and health:

Evidence from the 1958 British cohort

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Introduction (1)

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Numerous studies agreed on various determinants of health inequalities:

- Current social status (income, education level, wealth, occupation …)

e.g. van Doorslaer & Koolman 2004; Cutler et al. 2006; Lantz et al. 2010

- Early-life conditions (social background, parental SES/health/lifestyles, childhood health,...)

e.g. Anda et al. 2002; Currie and Stabile 2003; Case et al. 2005; Lindeboom et al. 2009; Rosa-Dias 2009; Jusot et al. 2010; Gohlmann et al. 2010; Trannoy et al. 2010

But the role played by individual lifestyles is more controversial:

- Epidemiological literature:

"Lifestyles make a relatively minor contribution to the social gradient in health"

e.g. Khang et al. 2009; Lantz et al. 2010; Skalická et al. 2009; van Oort et al. 2005

"The impact of lifestyles on health disparities would be larger than it was previously estimated"

e.g. Laaksonen et al 2008; Menvielle et al 2009; Strand & Tverdal 2004; Stringhini et al 2010;

- Health economics:

"Differences in lifestyles can explain a relevant part of health and mortality inequalities"

e.g. Contoyannis and Jones 2004; Häkkinen et al. 2006; Balia and Jones 2008

Introduction (2)

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The design of public policies tackling health inequalities requires to know:

- The determinants of health inequality
- Their respective contribution to the magnitude of health inequality

Because public policies will differ with the determinants found to be important:

- Tackling inequalities related to social determinants
  - Interventions in housing or working environment
- Tackling risky lifestyles
  - Interventions aimed at the whole population: increasing prices
  - Measures targeting the most vulnerable and disadvantaged groups such as minimum age or health promotion interventions

Introduction (3)

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Moreover in philosophical literature on social justice :

- *"some types of inequality are more objectionable than others"*

e.g. Dworkin 1981; Cohen 1989; Arneson 1989; Roemer 1998; Fleurbaey 2008

- Inequality linked to factors for which the individual is not responsible are considered as *"illegitimate"* differences in outcomes :
  - Circumstances, so called inequalities of opportunity
- Inequality linked to factors for which the individual is responsible are considered as *"legitimate"* differences in outcomes
  - Effort

Among the determinants of health inequality,

- Early-life conditions would represent circumstances (illegitimate source of inequality)
- But what about social status and lifestyles ?

Introduction (4)

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Lifestyles and social status might reflect

- Social reproduction, copying behaviours, inherited preferences: Constraints over the life cycle

But also

- Preferences, free choice, will, tastes: Individual effort

Therefore underlying public policy becomes less obvious and more complicated:

- Early-life conditions, current social status and lifestyles cannot be considered independent
- What are the early-life conditions to compensate (Principle of compensation in Equality of Opportunity theory)?

The aim of the paper

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1. To explore the long-term effects of early-life conditions, education and lifestyles on health

2. To investigate the effect of each determinant in overall health inequality

3. To understand the interdependence between early-life conditions, education and lifestyles

4. To determine whether early-life conditions influence health directly or indirectly, that is via affecting lifestyles and education

Data - cohort

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Data

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National Child Development Study (NCDS) : a longitudinal study with all the people born in one week in March 1958 in England, Scotland and Wales

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		

Parent's data
Child health

Cohort member's data
Health, lifestyles
Education

- Attrition:
  - Attrition in the NCDS is not related to social status (Case et al. 2005)
  - Modest correlation between attrition and employment status (Lindeboom et al. 2006)

Variables (1)

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- Measurement of health / outcome of interest:
  - Self-assessed health : 4 or 5-point categorical scale ranging from P*oor* (age 23, 33, 45) or *Very poor* (age 46) to *Excellent health* (all waves)
  - Used as a binary variable : 1 if health rated as good or higher, and 0 otherwise.

	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%**

Variables (2)

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- Measurement of early-life conditions
  - Social background
    - Father's social class at the time of birth (3 categories + no male figure)
    - Father and mother's education (dropped out from school before or at minimum schooling age)
    - Report of financial hardships (age 16)
  - Parents' health and lifestyles
    - Parental report of chronic illness (age 16)
    - Parents' smoking (age 16)
  - Childhood health
    - Report of chronic condition (age 16)
    - Low birth weight (<2,5 kg)
    - Obesity status (age 16)

Variables (3)

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- Measurement of education (discrete outcome)
  - We assume that education level is a reliable proxy of other social outcomes (employment, housing, income, etc.)

  > Highest qualification achieved over the period

    - lower than O-level; O-level or A-level; higher than A-level

- Measurement of lifestyles (binary outcome)
  - Exercising: cohort member is regularly doing exercise or sports (at least once in the last 4 weeks)
  - Non smoking: cohort member is not a current smoker at wave t
  - Drinking prudently: the # of units of alcohol drinks taken the week before the interview (gender-specific)
  - Absence of obesity: BMI strictly lower than 30

Estimation strategy (1)

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Let us assume that individual health status *H* can be written using the following health production function:

$$H = f(C, D, E, L, u)$$

$$u = \omega_i + \varepsilon_{it}$$

ω_i unobserved individual characteristics (e.g. genetics, personality traits)

ε_{it} time variant individual specific error term

- Lifestyles introduced as lagged variables:
  - influence health at the next period / potential reverse causality if contemporaneous
- ω_i may be correlated with lifestyles at each wave:
  - A random effect Probit specification allowing ω_i and ε_{it} to be correlated introducing a vector of average individual past variables (Mundlak, 1978)
  - Therefore a measure of transitory effects and a measure of long-term or permanent effects on health

Estimation strategy (2)

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- Furthermore we need to distinguish between ω_i and past health:
  - a lagged dependent variable in the model $H_{i,t-1}$
    - Captures state dependence in health reports
    - Reduces the impact of individual heterogeneity
- The initial health is likely not to be randomly assigned and correlated with ω_i
  - The initial conditions problem (Wooldridge, 2005): H_{i0}

Concretely the latent health model that we estimate can be written as follows:

$$H_{it}^{*} = \alpha_1 C_i + \alpha_2 D_i + \beta_1 E_i + \delta_1 L_{it-1} + \delta_2 \bar{L}_i + \gamma_1 H_{it-1} + \gamma_2 H_{i0} + \omega_i + \varepsilon_{it}$$

Some base estimates in the paper:

- Model 1: a static model / Model 2: introduction of average past lifestyles / Model 3: a dynamic model

Measurement of inequality

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- An inequality index decomposable by sources : natural decomposition of the variance (Shorrocks, 1982)
- In a non linear context, H_{it}^{*} can only be measured as a prediction
- We use the pseudo R^2 (McKelvey and Zavoina 1975) in order to measure the share of variance explained by the K variables having an associated coefficient η_k

$$\hat{H}_{it}^{*} = \sum \eta_k X_{it}^{k}$$

$$R^2 = \frac{V(\hat{H}^{*})}{V(\hat{H}^{*}) + \sigma_{\varpi} + 1}$$

- ω_i and ε_{it} are defined as independent of the set of K explanatory variables:
  - a variance estimated from the data is attributed to ω_i
  - a variance normalised to be equal to 1 is attributed to ε_{it} (case of a Probit)

As many sources of inequalities in health as regressors (additive index)

Mediating effect identification (1)

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To help design public policies we need to understand interdependent relationships:

1. Baseline specification

$$H_{it}^{*} = \alpha_1 C_i + \alpha_2 D_i + \beta_1 E_i + \delta_1 L_{it-1} + \delta_2 \bar{L}_i + \gamma_1 H_{it-1} + \gamma_2 H_{i0} + \omega_i + \varepsilon_{it}$$

Potential mediated effects between early-conditions and health via adult lifestyles and education.

$$E_i = \theta_1^a C_i + \theta_2^a D_i + e_i$$

$$L_{it} = \theta_1^b C_i + \theta_2^b D_i + \theta_3^b E_i + l_{it}$$

$$\bar{L}_i = \theta_1^c C_i + \theta_2^c D_i + \theta_3^c E_i + \bar{l}_i$$

Mediating effect specification (2)

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To estimate mediating effect: (Bernt-Karlson et al. (2010))

1. Estimating the corresponding residual in each auxiliary equation (LPM)

2. Including the residuals in the health production function instead of the original variables

$$H_{it}^{*} = \alpha_1^2 C_i + \alpha_2^2 D_i + \beta_1^2 \hat{e}_i + \delta_1^2 \hat{l}_{it-1} + \delta_2^2 \bar{\hat{l}}_i + \gamma_1^2 H_{it-1} + \gamma_2^2 H_{i1} + \omega_i + \varepsilon_{it}$$

3. In the case of linear auxiliary equation estimates (not exact if probit, and generalised residuals), we can rewrite the baseline equation and obtain:

$$\alpha_1^{\,2} = \alpha_1 + \beta_1.\theta_1^{\,a} + \delta_1.\theta_1^{\,b} + \delta_2.\theta_1^{\,c} \qquad \delta_1^{\,2} = \delta_1$$

$$\alpha_2^{\,2} = \alpha_2 + \beta_1.\theta_2^{\,a} + \delta_1.\theta_2^{\,b} + \delta_2.\theta_2^{\,c} \qquad \delta_2^{\,2} = \delta_2$$

$$\beta_1^{\,2} = \beta_1 + ...\delta_1.\theta_3^{\,b} + \delta_2.\theta_3^{\,c}$$

Results – baseline model

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Variables	Baseline model	
Gender Male	0,031	
Fathers' social class (Ref.: I and II)		
III	-0,073	
IV and V	-0,208	**
No male head	-0,377	***
Financial hardship (Ref.: None)		
Yes	-0,252	***
Non response	0,118	
Father's education (Ref.: beyond the min age)		
Before or at min age	-0,045	
Mother's education (Ref.: beyond the min age)		
Before or at min age	-0,146	**
Parental illness (Ref.: None)		
Father's illness	-0,171	**
Mother's illness	-0,121	
Parental smoking (Ref.: None)		
Father's smoking	0,072	
Non response	-0,012	
Mother's smoking	-0,076	*
Non response	-0,068	
Chronic condition at 16 (Ref.: None)		
Yes	-0,012	
Non response	0,127	
Low birth weight	-0,079	
Obesity at 16 (Ref.: Yes)		
No	-0,307	*
Non response	-0,166	

Variables	Baseline model	
Educational level (Ref.: Higher than A-level)		
Before O-level	-0,207	***
O-level or A-level	-0,032	
Lifestyles (lagged)		
Exercising	-0,042	
No smoking	0,072	
Drinking prudently	0,033	
No obesity	-0,052	
Mean lifestyles		
Exercising	0,566	***
No smoking	0,226	**
Drinking prudently	0,222	*
No obesity	0,760	***
Lagged health status	0,311	***
Health status at 23	1,007	***
Time dummies (Ref.: t=3)		
t=1	0,579	***
t=2	0,341	***
V(H^*)	0,360	
σ_ω	0,639	
ρ#	0,390	
R^2 (McKelvey and Zavoina)	0,180	

Results- comparisons with mediated model

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Variables	Baseline model	Mediated model
Gender Male	0,031	0,081 *
Fathers' social class (Ref.: I and II)		
III	-0,073	-0,104
IV and V	-0,208 **	-0,280 ***
No male head	-0,377 ***	-0,463 ***
Financial hardship (Ref.: None)		
Yes	-0,252 ***	-0,348 ***
Non response	0,118	0,063
Father's education (Ref.: beyond the min age)		
Before or at min age	-0,045	-0,093
Mother's education (Ref.: beyond the min age)		
Before or at min age	-0,146 **	-0,199 ***
Parental illness (Ref.: None)		
Father's illness	-0,171 **	-0,192 **
Mother's illness	-0,121	-0,141
Parental smoking (Ref.: None)		
Father's smoking	0,072	0,021
Non response	-0,012	-0,025
Mother's smoking	-0,076 *	-0,123 ***
Non response	-0,068	-0,083
Chronic condition at 16 (Ref.: None)		
Yes	-0,012	-0,060
Non response	0,127	0,151
Low birth weight	-0,079	-0,096
Obesity at 16 (Ref.: Yes)		
No	-0,307 *	-0,183
Non response	-0,166	-0,219 *

Variables	Baseline model	Mediated model
Educational level (Ref.: Higher than A-level)		
Before O-level	-0,207 ***	-0,404 ***
O-level or A-level	-0,032	-0,108 **
Lifestyles (lagged)		
Exercising	-0,042	-0,042
No smoking	0,072	0,072
Drinking prudently	0,033	0,033
No obesity	-0,052	-0,052
Mean lifestyles		
Exercising	0,566 ***	0,566 ***
No smoking	0,226 **	0,226 **
Drinking prudently	0,222 *	0,222 *
No obesity	0,760 ***	0,760 ***
Lagged health status	0,311 ***	0,311 ***
Health status at 23	1,007 ***	1,007 ***
Time dummies (Ref.: t=3)		
t=1	0,579 ***	0,577 ***
t=2	0,341 ***	0,337 ***
V(H^*)	0,360	0,360
σ_ω	0,639	0,639
ρ#	0,390	0,390
R^2 (McKelvey and Zavoina)	0,180	0,180

Decomposition of health inequality

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	Over the full period			
	Baseline specification		Mediating specification	
Variables	Mean (%)	[95% Boot. Conf. Int]	Mean (%)	[95% Boot. Conf. Int]
Sex	0,27	[0,24 ; 0,31]	0,65	[0,60 ; 0,69]
Age	15,12	[14,95 ; 15,28]	15,09	[14,90; 15,28]
Early life conditions	17,81	[16,23 ; 19,39]	23,75	[22,07 ; 25,43]
Social background	*11,81*	*[10,97 ; 12,77]*	*15,85*	*[14,85 ; 16,85]*
Parent's health and lifestyles	*3,44*	*[3,10 ; 3,79]*	*4,67*	*[4,26; 5,08]*
Initial health	*2,50*	*[2,11 ; 2,88]*	*3,23*	*[2,89; 3,58]*
Lifestyles	28,55	[27,36 ; 29,74]	22,16	[20,99 ; 23,34]
Education	4,92	[4,68 ; 5,17]	5,29	[5,10 ; 5,47]
Health state-dependence	33,33	[32,78 ; 33,88]	33,06	[32,49 ; 33,64]

Conclusion …

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- Impressive contribution of lifestyles to health inequalities (28% baseline / 22% mediated)
- Health significantly influenced by average past lifestyles : average past lifestyles matter more
- Advantages of dynamic panel analysis :
  - to control a large part of individual unexplained heterogeneity
  - to evaluate the effect of health state dependence over time

- Early life conditions and education would shape other factors: mediated effects
  - When lifestyles and social factors are purged from the association with early life conditions and education :
    - reduction of their contribution to health inequalities
    - higher contribution of early life conditions to health inequalities
    - higher contribution of education to health inequalities