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Short communication

Do childhood infections affect labour market outcomes in adulthood and, if so, how?

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ABSTRACT

A burgeoning body of literature suggests that poor childhood health leads to adverse health outcomes, lower educational attainment and weaker labour market outcomes in adulthood. We focus on an important but under-researched topic, which is the role played by infection-related hospitalization (IRH) in childhood and its links to labour market outcomes later in life. The participants aged 24–30 years in 2001 N = 1706 were drawn from the Young Finns Study, which includes comprehensive registry data on IRHs in childhood at ages 0–18 years. These data are linked to longitudinal registry information on labour market outcomes (2001–2012) and parental background (1980). The estimations were performed using ordinary least squares (OLS). The results showed that having an additional IRH is associated with lower log earnings (b = -0.110, 95 % confidence interval (CI): −0.193; −0.026), fewer years of being employed (b = −0.018, 95 % CI: −0.031; −0.005), a higher probability of receiving any social income transfers (b = 0.012, 95 % CI: −0.002; 0.026) and larger social income transfers, conditional on receiving any (b = 0.085, 95 % CI: 0.025; 0.145). IRHs are negatively linked to human capital accumulation, which explains a considerable part of the observed associations between IRHs and labour market outcomes. We did not find support for the hypothesis that adult health mediates the link.

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1. Introduction

Childhood health has lasting effects on socioeconomic outcomes. Research has established the importance of both initial health endowment and health events in childhood for subsequent socioeconomic outcomes. Both strands of the literature conclude that early health conditions are related to worse adult health, lower educational attainment, and lower earnings (Douglas et al., 2018; Prinz et al., 2018).

Health disparities have early origins. Both twin studies and genome-wide association studies (GWAS) show that variation in individual health is partly explained by genetic makeup. GWASs have identified numerous single-nucleotide polymorphisms (SNPs) that are associated with health-related outcomes such as body mass index (BMI) (Locke et al., 2015) and a higher incidence of type 2 diabetes (Andersson et al., 2013). A meta-analysis based on twin studies also concluded that there is a significant genetic component to health-related traits (Polderman et al., 2015). In addition to genetic inheritance, the in utero environment has consequences with regard to initial health endowment. It is challenging to distinguish between the effects of genetic inheritance and those of prenatal conditions, but the results based on monozygotic twins with similar genetic makeup suggest that differences in in utero conditions cause differences in birth weight, which is often used as a proxy for initial health endowment (Behrman and Rosenzweig, 2004). Further evidence for the importance of the effect of in utero conditions on subsequent health comes from studies exploiting exogenous shocks to foetal health, such as the 1918 influenza pandemic (Almond, 2006; Almond and Mazumder, 2005; Beach et al., 2018), the Dutch hunger winter (Roseboom et al., 2011), exposure to Ramadan (Kunto and Mandemakers, 2019; Majid, 2015; Schoeps et al., 2018) and a sudden reduction in pollution due to plant closures during pregnancy (Chay and Greenstone, 2003; Parker et al., 2008).

Studies also show that childhood health may have long-term effects on socioeconomic outcomes in adulthood. For example, lower self-reported retrospective measures of childhood health and specific childhood health conditions such as Type 1 diabetes have been associated with lower educational attainment and lower earnings (Case et al., 2005; Haas et al., 2011; Persson et al., 2016; Smith, 2009). To what extent these childhood conditions reflect initial health endowment, exogenous shocks to childhood health or childhood environment are unclear because identification strategies have typically been unable to distinguish among these competing explanations.

There are many potential mechanisms linking childhood health and socioeconomic status in adulthood. First, worse health may affect human capital accumulation, e.g., due to school absences, or because disease management or complications increase the time needed for human capital accumulation (i.e., the marginal cost of educational investments increases). Early health conditions may also affect the ability to learn. Previous research has documented, for example, that exposure to Ramadan fasting in utero has negative effects on cognitive development and school success later in life (Majid, 2015). Increased uncertainty about future health and labour market performance may also weaken economic incentives to invest in higher education (Lundborg et al., 2014). Second, childhood health may affect health in adulthood, thereby affecting occupational choices (Butland et al., 2011), productivity and hours worked (Pelkowski and Berger, 2004; Vijan et al., 2004). Early infection-related hospitalizations (IRHs), for example, have been linked to metabolic outcomes (Burgner et al., 2015a) and adverse cardiovascular phenotypes in adulthood (Burgner et al., 2015b), which may affect labour market outcomes (e.g., Cawley, 2015). Third, those who have poor health may also be discriminated against in the labour market (Rooth, 2011).

This study examines the links between IRHs at ages 0–18 years and labour market outcomes in adulthood. A hospitalization that led to at least one overnight stay was defined as infection-related if either a primary or a secondary International Classification of Diseases code indicated an infection diagnosis (Liu et al., 2016). Using rich, longitudinal, population-based data, we examined to what extent links between IRHs and adult labour market outcomes are mediated through years of education and adult health and whether any associations remain intact after adjusting for initial health endowment (the formal mediation analysis is explained in Section 2.6). A large body of literature has emphasized the importance of prenatal health endowment for socioeconomic outcomes in adulthood. In this study, we focus on childhood and adolescent IRHs, which have garnered much less interest in the prior literature. We focus on IRHs for three reasons. First, infections are a major cause of hospitalizations among children, thus constituting a significant public health problem (Goto et al., 2016). Second, focusing on post-prenatal health is important because even if prenatal health endowment affects variability in health, it is unlikely to be completely deterministic. To that extent, it is worth investigating childhood health in its own right. Third, using information on IRHs that cover ages from 0 to 18, we can examine the relative importance of early (ages 0–5) and later (ages 6–18) IRHs. This analysis adds to the earlier literature, which has stressed the importance of early childhood health (Almond and Currie, 2011) but has also documented the crucial role of adolescent health in adult labour market outcomes (Lundborg et al., 2014).

2. Data and methods

The longitudinal data were obtained from three sources: 1) the Young Finns Study (YFS) / the Finnish Hospital Discharge Register (FHDR); 2) the Finnish Longitudinal Employer-Employee Data (FLEED) of Statistics Finland; and 3) the Longitudinal Population Census (LPC) of Statistics Finland.

The YFS is an on-going epidemiological study examining risk factors for cardiovascular diseases in adulthood. The study started in 1980 when subjects in six age cohorts born in 1962, 1965, 1968, 1971, 1974, and 1977 were randomly chosen from five university hospital regions in Finland to produce a nationally representative sample of Finnish children and adolescents. The original sample size was 3596 (Raitakari et al., 2008). The YFS contains information on IRHs at ages 0–18, which originate from the FHDR. The FHDR is a nationwide database with comprehensive records for patients discharged from hospitals from 1969 onwards. The data include information on admission and discharge days and diagnoses. The completeness and accuracy of the data are adequate, according to validation studies (Sund, 2012). In this study, we focused on the three youngest age cohorts, for whom we have access to information on infections for the entire age period from 0 to 18 years. The sample size in the baseline model is 1706. In 1980, these children were between 3 and 9 years old.

To obtain information on participants' educational attainment and labour market outcomes, the YFS was linked to the FLEED, and information on parental background (education and earnings in 1980) was drawn from the LPC data. The matching between datasets was based on unique personal identifiers, thus avoiding problems related to errors in record linkages (Ridder and Moffitt, 2007). The use of administrative data on income and educational attainment also eliminates the risk of systematic measurement error related to self-reported measures.

All participants of the YFS provided written informed consent, and the study was approved by the relevant institutional ethics committees. Parents or guardians provided written informed consent on behalf of the under-aged children enrolled in the study. The final combined YFS-FLEED-LPC data have been approved for research purposes by Statistics Finland under the ethics guidelines

of the institution, which comply with all national standards. A flow chart of the study subjects is documented in the Supplementary Appendix (Fig. A.1).

2.1. Outcome variables

We use four outcome variables, which were measured over the period 2001–2012: 1) the logarithm of average wage and salary earnings; 2) share of years employed; 3) indicator of having received social income transfers (1 = yes; 0 = no); and 4) the logarithm of social income transfers conditional on receiving any. The outcome variables were drawn from Statistics Finland's registry data FLEED.

2.2. Explanatory variables

The explanatory variable of interest was the number of IRHs at ages 0–18 years. A hospitalization that included at least one overnight stay was defined as infection-related if either a primary or a secondary International Classification of Diseases code indicated an infection diagnosis (Liu et al., 2016). All diagnoses were established by certified health care professionals. In robustness checks, we also used an indicator of having at least one infection between ages 0 and 18 as an explanatory variable.

2.3. Potential mediators

The first potential mediator was years of education in 2001. Information on the highest completed degree was drawn from the FLEED and was converted into years of education using the official estimates of Statistics Finland regarding the number of years needed to obtain a degree. As a second potential mediator, we used the adult health index. The index consists of seven objective indicators of the survey respondents' medical state (i.e., biomarkers; BMI, waist-hip ratio (WHR), triglycerides, HDL and LDL cholesterol levels, and systolic and diastolic blood pressure), which were based on measurements and blood tests conducted in 2001 by medical professionals at local health centres. An indicator variable for each biomarker was constructed to identify those individuals whose biomarker values exceeded the nationally recommended levels (1 = high risk; 0 = low risk; the cut-off values are documented in Supplementary Appendix Table A.1). These indicator variables were summed to create a comprehensive health measure index. Information on biomarkers was drawn from the YFS.

2.4. Control variables

All models were adjusted for indicators of the sex and birth cohort. Because differences in childhood health may reflect differences in family background (Milaniak and Jaffee, 2019), we also used the logarithm of family income in 1980 and parental education in 1980 as additional control variables. The indicator variable for high parental education level equalled one if at least one of the parents had obtained some university education. These control variables were based on Statistics Finland registry data FLEED and LPC.

Previous studies have emphasized the role played by initial health endowment in subsequent health outcomes (e.g., Beach et al., 2018; Behrman and Rosenzweig, 2004; Majid, 2015). To explore how differences in initial health endowment affected our results, in the robustness checks, we adjusted the baseline models for birth weight (in kg), which is a commonly used proxy for initial health endowment (Behrman and Rosenzweig, 2004), and genetic risk scores for BMI (based on 32 SNPs (Speliotes et al., 2010)), WHR (14 SNPs (Heid et al., 2010)), triglycerides (41 SNPs (Hernesniemi et al., 2015)), HDL cholesterol (38 SNPs (Teslovich et al., 2010)), LDL cholesterol (58 SNPs (Hernesniemi et al., 2015)) and blood pressure (29 SNPs (Ehret et al., 2011)). As an additional control variable, we also used a genetic risk score for years of education (74 SNPs (Okbay et al., 2016)). These data were drawn from the YFS.

2.5. Additional variables

In the descriptive analyses, we compared childhood health and educational outcomes stratified by infection status in the following dimensions: the number of physician-diagnosed childhood chronic conditions, participation in remedial education (in 1980 and 1983) and school success, indicated by grade point average (GPA) in the ninth grade. This information was drawn from the YFS.

2.6. Analysis

Two-sample t-tests were utilized to compare the characteristics of those who experienced at least one IRH with those who did not experience any infections. The estimations of adult outcomes were performed using ordinary least squares (OLS). To test potential pathways that may mediate the association between IRH and adult labour market outcomes, we performed a Sobel (1982) mediation analysis. This test examines whether the indirect association between the independent variable (X, i.e., IRH) and the dependent variables (Y_k, where k refers to a labour market outcome) through potential mediators (M_j, where j refers to years of education or the adult health risk index) is significantly different from zero. Fig. 1 depicts the mediation model, which consists of three regression equations

$$Y_{ki} = \theta_1 + \gamma X_i + \varepsilon_{1i} \quad (1)$$

$$M_{ji} = \theta_2 + \alpha X_i + \varepsilon_{2i} \quad (2)$$

$$Y_{ki} = \theta_3 + \mu X_i + \beta M_{ji} + \varepsilon_{3i} \quad (3)$$

where i refers to an individual, γ refers to the gross association between IRHs and adult labour market outcomes (Fig. 1, Panel A, path c), α is the link between IRHs and the potential mediators (Fig. 1, Panel B, path a), β is the link between the mediator and the labour market outcomes (Fig. 1, Panel B, path b), and μ is the remaining link between IRHs and the labour market outcomes after controlling for the mediator (path c'). The mediated link is calculated as the product of the coefficients α and β (i.e., $\alpha\beta$), and the total proportion of the link that is mediated through mediator j is equal to $(\alpha\beta)/\gamma$.

3. Results

3.1. Descriptive statistics

Twenty-five percent of individuals had experienced at least one IRH (either primary or a secondary infection diagnosis) during childhood, of whom 61 % had one infection, 23 % had two infections and 16 % had at least three infections. Table 1 compares the observable characteristics of participants who had experienced at least one IRH to those who did not experience any infections. Among those who had experienced at least one IRH, the share of men was higher ($p < 0.01$), the average age was lower ($p < 0.01$), and the share of children with highly educated parents was smaller ($p < 0.05$). Otherwise, the results of the bivariate analyses without controls did not indicate the presence of differences in family background, initial health endowment (birth weight, GRSs) or the number of chronic conditions in childhood (in each case, $p > 0.10$).

Table 2 presents labour market outcomes, educational outcomes and adult health characteristics based on IRH status. The

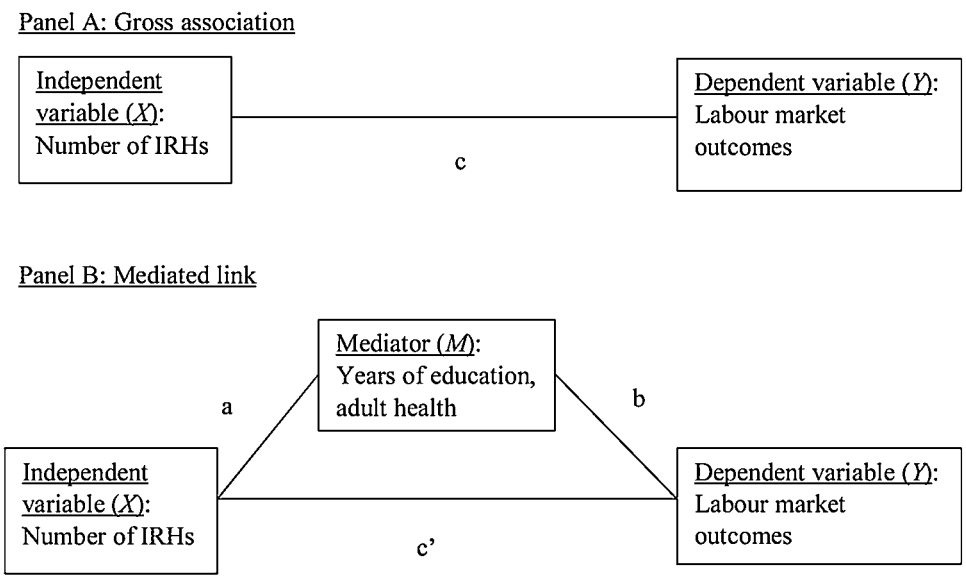

Fig. 1. Mediation model.

Table 1
Descriptive statistics by IRH status.

	All Mean (SD)	At least one IRH Mean (SD)	No IRHs Mean (SD)	Difference	t-statistics	N
Number of IRHs (0–18 years)	0.438 (1.075)	1.779 (1.522)	0 (0)	1.779	41.936***	1706
Female (%)	0.505 (0.500)	0.440 (0.497)	0.526 (0.499)	−0.086	−3.066***	1706
Age in 2001	27.070 (2.469)	26.743 (2.474)	27.177 (2.459)	−0.434	−3.139***	1706
High level of parental education (%) (1980)	0.155 (0.362)	0.126 (0.332)	0.164 (0.370)	−0.038	−1.970**	1706
Parental income (1980)	12827.27 (7466.35)	12430.26 (7207.67)	12956.92 (7547.13)	−526.66	−1.255	1706
Initial endowment						
Birth weight	3.516 (0.538)	3.492 (0.565)	3.524 (0.529)	−0.032	−1.033	1578
Body mass GRS	29.055 (3.327)	29.132 (3.295)	29.031 (3.339)	0.100	0.422	1090
Waist-hip ratio GRS	15.170 (2.423)	15.095 (2.425)	15.193 (2.424)	−0.097	−0.562	1090
Triglyceride GRS	0.985 (0.094)	0.987 (0.099)	0.984 (0.092)	0.003	0.394	1090
HDL cholesterol GRS	44.698 (3.671)	44.900 (3.815)	44.636 (3.626)	0.264	1.007	1090
LDL cholesterol GRS	0.960 (0.078)	0.954 (0.082)	0.961 (0.076)	−0.007	−1.312	1090
Blood pressure GRS	30.376 (3.122)	30.378 (3.099)	30.376 (3.130)	0.003	0.011	1090
Childhood health (0–18 years)						
Number of chronic conditions	0.104 (0.382)	0.110 (0.412)	0.102 (0.372)	0.008	0.339	1706

Note: Table reports means and standard deviations (SD) in parentheses. The unit of analysis is the individual. Differences between groups were tested using a two-sample t-test. The three cohorts studied (aged 24, 27, and 30 in 2001) were drawn from the YFS. The indicator for high parental education equalled one if at least one of the parents had obtained some university education (based on the LPC data from 1980). IRH refers to infection-related hospitalization and GRS to the genetic risk score. Statistical significance: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

bivariate analysis revealed that those who experienced IRHs during childhood were more likely to participate in remedial education ($p < 0.01$), had lower GPA at the ninth grade, had fewer years of education in adulthood ($p < 0.01$) and were less likely to complete high education ($p < 0.01$). Participants with childhood IRHs also had lower earnings and a lower share of years employed ($p < 0.01$). If we compare adult health between the two groups, those who experienced at least one IRH had lower HDL cholesterol levels ($p < 0.1$), higher BMI and WHR ($p < 0.01$) and higher systolic blood pressure in 2001 ($p < 0.1$).

3.2. IRH and labour market outcomes

Table 3 shows the results from four mediation models with four different outcome variables (log of average earnings, share of years employed, indicator for obtaining any social income transfers and

Table 2
Descriptive statistics and comparison of labour market and educational outcomes and adult health by IRH status.

	All Mean (SD)	At least one IRH Mean (SD)	No IRHs Mean (SD)	Difference	t-statistics	N
Labour market outcomes						
Log of average earnings, 2001–2012	9.397 (1.904)	9.158 (2.269)	9.475 (1.762)	−0.317	−2.618***	1706
Share of years employed, 2001–2012	0.788 (0.293)	0.747 (0.324)	0.801 (0.281)	−0.054	−3.075***	1706
Indicator for social income transfers, 2001- 2012	0.889 (0.314)	0.898 (0.304)	0.886 (0.317)	0.011	0.632	1706
Log of average social income transfers, 2001–2012	7.056 (1.416)	7.109 (1.446)	7.039 (1.406)	0.071	0.839	1517
Educational outcomes						
Education, years	12.800 (2.433)	12.424 (2.296)	12.922 (2.464)	−0.498	−3.659***	1706
High education level	0.192 (0.394)	0.148 (0.355)	0.206 (0.405)	−0.058	−2.826***	1706
Indicator of remedial education	0.253 (0.435)	0.328 (0.471)	0.243 (0.429)	0.086	2.140**	1070
Grade point average in ninth grade	7.950 (0.936)	7.737 (0.987)	7.978 (0.926)	−0.241	−2.481**	906
Adult health						
Body mass index 2001	24.456 (4.335)	25.275 (4.928)	24.204 (4.106)	1.071	3.000***	982
Waist-hip ratio in 2011	0.823 (0.077)	0.838 (0.076)	0.818 (0.076)	0.020	3.509***	982
Triglycerides in 2001	1.255 (0.618)	1.307 (0.644)	1.240 (0.609)	0.067	1.409	982
HDL cholesterol in 2001	1.284 (0.317)	1.253 (0.327)	1.294 (0.313)	−0.040	−1.682*	982
LDL cholesterol in 2001	3.097 (0.775)	3.126 (0.782)	3.088 (0.774)	0.038	0.646	982
Diastolic blood pressure in 2001	71.249 (7.802)	71.626 (7.571)	71.133 (7.873)	0.492	0.839	982
Systolic blood pressure in 2001	120.930 (13.088)	122.342 (13.463)	120.495 (12.948)	1.847	1.878*	982
Health risk index in 2001	1.435 (1.256)	1.606 (1.330)	1.382 (1.228)	0.224	2.375**	982

Note: Infections were measured between 0 and 18 years of age apart from the results concerning participation in remedial education and GPA in ninth grade in which case the number of infections was measured between ages 0 and 5.See Table 1 notes for additional details.

Table 3
Mediation by educational attainment.

Outcome variable (*Y*)	(1) Relation between number of IRHs and labour market outcomes (path c)	(2) Relation between number of IRHs and years of education (path a)	(3) Relation between years of education and labour market outcomes (path b)	(4) Mediated link between IRHs and the outcome variable through years of education (Sobel test)	(5) Proportion of total connection that is mediated
Model 1: *Y* = Log of average earnings, 2001-2012 (n = 1706)	−0.110** [−0.193; −0.026]	−0.169*** [−0.273; −0.066]	0.210*** [0.173; 0.247]	−0.036***	0.323
Model 2: *Y* = Share of years employed, 2001-2012 (n = 1706)	−0.018*** [−0.031; −0.005]	−0.169*** [−0.273; −0.066]	0.032*** [0.026; 0.037]	−0.005***	0.294
Model 3: *Y* = Indicator for social income transfers, 2001-2012 (n = 1706)	0.012* [−0.002; 0.026]	−0.169*** [−0.273; −0.066]	−0.008** [−0.015; −0.002]	0.001**	0.115
Model 4: *Y* = Log of average social income transfers, 2001-2012 (n = 1517)	0.085*** [0.025; 0.145]	−0.165*** [−0.271; −0.059]	−0.076*** [-0.104; −0.047]	0.012***	0.147

Note: Results are based on the linear regression (OLS) method. Each row refers to one of the four estimated mediation models. The unit of analysis is the individual. The paths are explained in Fig. 1. The three cohorts under study (aged 24, 27, and 30 in 2001) were drawn from the YFS. All models included controls for the birth year, sex, parental education level (1980), and parental earnings (1980). The 95 % confidence intervals for the parameter estimates are reported in parenthesis. Statistical significance: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 4
Mediation by adult health.

Outcome variable (*Y*)	(1) Relation between number of IRHs and labour market outcomes (path c)	(2) Relation between number of IRHs and health (path a)	(3) Relation between health and labour market outcomes (path b)	(4) Mediated link between IRHs and the outcome variable through health (Sobel test)	(5) Proportion of total connection that is mediated
Model 1: *Y* = Log of average earnings, 2001-2012 (n = 982)	−0.068* [−0.145; 0.010]	0.016 [−0.053; 0.086]	−0.139*** [−0.208; −0.069]	−0.002	0.033
Model 2: *Y* = Share of years employed, 2001-2012 (n = 982)	−0.019** [−0.033; −0.006]	0.016 [−0.053; 0.086]	−0.016** [−0.028; −0.003]	−0.000	0.013
Model 3: *Y* = Indicator for social income transfers, 2001-2012 (n = 982)	0.009 [−0.009; 0.027]	0.016 [−0.053; 0.086]	0.009 [−0.007; 0.026]	0.000	0.017
Model 4: *Y* = Log of average social income transfers, 2001-2012 (n = 869)	0.068* [−0.004; 0.141]	0.013 [−0.058; 0.085]	0.057* [−0.010; 0.125]	0.001	0.011

Note: Results are based on linear regression (OLS) method. Each row refers to one of the four estimated mediation models. The unit of analysis is the individual. The paths are explained in Fig. 1. The three cohorts under study (aged 24, 27, and 30 in year 2001) are drawn from the YFS. All models included controls for the birth year, sex, parental education (1980), and parental earnings (1980). The 95 % confidence intervals for the parameter estimates are reported in parenthesis. Statistical significance: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

log of average social income transfers). The results presented in column 1 show the overall (gross) association between IRHs and each of these labour market outcomes (path c, Fig. 1). A higher number of IRHs was associated with lower earnings, a lower share of years employed, a higher probability of receiving any social income transfers (i.e., the extensive margin of adjustment) and higher social income transfers if receiving any (i.e., the intensive margin of adjustment). Having an additional infection in childhood was associated with 11.0 % lower earnings (b = −0.110, 95 % confidence interval (CI): −0.193; −0.026; $p < 0.010$), a 1.8 percentage point lower share of years employed (b = −0.018, 95 % CI: −0.031; −0.005, $p < 0.006$), a 1.2 percentage point higher probability of receiving any social income transfers (b = 0.012, 95 % CI: −0.002; 0.026, $p < 0.087$) and an 8.5 % increase in the size of social income transfers conditional on receiving any (b = 0.085, 95 % CI: 0.025; 0.145, $p < 0.006$).

3.3. Potential pathways

The results in Table 3 also show the mediation results using education as a potential pathway by which IRHs may affect adult labour market outcomes. Column 2 presents the relationship between IRHs and years of education (path a, Fig. 1), while column 3 presents the relationship between years of education and labour market outcomes (path b, Fig. 1). A higher number of IRHs was related to fewer years of education (Column 2, $p < 0.003$), whereas a higher number of years of education was associated with better labour market outcomes (Column 3, $p < 0.02$). Based on the Sobel mediation test (Column 4), the indirect association between IRHs and labour market outcomes mediated through education was statistically significant ($p < 0.048$), and the proportion of the total association mediated was 32 % in the earnings equation, 29 % in the share of years employed, 12 % in the probability of receiving social income transfers and 15 % in the amount of social income transfers conditional on receiving any (Column 5).

Table 4 shows the mediation results that were obtained using adult health as a potential mediator. Based on the results (Column 3), adult health was a significant predictor of labour market outcomes. A higher number of health risks in adulthood was related to lower earnings (b = -0.139, 95 % CI: -0.208; -0.069, $p < 0.000$) and a lower share of years employed (b = -0.016, 95 % CI: -0.028; -0.003, $p < 0.014$). The point estimates also suggest that a higher number of health risks was associated with a higher probability of receiving social income transfers (b = 0.009, 95 % CI: -0.007; 0.026, $p < 0.254$) and larger transfers received (b = 0.057, 95 % CI: -0.010; 0.125, $p < 0.095$). However, the relationship between childhood IRHs and the number of adult health risks (Column 2) was weak: one additional IRH was related to an ∼0.02 increase in the number of health risks ($p > 0.6$). Thus, IRHs do not seem to predict the adult health risk index. Consequently, the proportion of the total association that was mediated through the health risk index was low (1–3 %), and according to the Sobel test, this mediation pathway was non-significant ($p > 0.6$).

3.4. Robustness checks

Our results imply that education explains a considerable part of the link between IRHs and labour market outcomes, while the role of adult health as a mediator is small and statistically non-significant. However, missing information in the adult health risk index reduced our estimation sample in Table 4 (i.e., mediation through adult health), which limits the comparability between the results using the two different mediators. To explore whether varying sample size has implications for our findings, we estimated the models in Table 3 using the same sample as in Table 4. Based on the results (Table A.2), the sample size was not a major driver of our findings, and education remained a significant mediator in the reduced sample.

We also performed several robustness checks to reduce the possibility that our results were driven by confounders that affect both IRHs and labour market outcomes. First, we augmented the baseline models with the measures of initial health endowment i.e., birth weight and GRSs (Table 5). These control variables had only a modest impact on the point estimates that describe the connection between IRHs and labour market outcomes. Thus, the observed initial health endowment does not seem to be a major driver of our results. This pattern is in accordance with the findings based on bivariate analyses that showed that there were no statistically significant differences in initial endowment between those who had at least one IRH and those who had none (Table 1).

Table 5
IRHs in childhood and labour market outcomes. Baseline results and results with controls for initial health endowments based on the same sample size.

Outcome variable (*Y*)	(1) Relation between number of IRHs and labour market outcomes (path c)		(2) Relation between number of IRHs and years of education (path a)		(3) Relation between years of education and labour market outcomes (path b)		(4) Mediated link between IRHs and the outcome variable through years of education (Sobel test)		(5) Proportion of total connection that is mediated	
Model 1:	−0.035	−0.036	−0.204***	−0.208***	0.138***	0.137***	−0.028	−0.028***	0.802	0.790
Y = Log of average earnings, 2001-2012 (n = 1040)	[−0.112; 0.042]	[−0.113; 0.041]	[−0.342; −0.067]	[−0.345; −0.070]	[0.105; 0.171]	[0.104; 0.170]				
Controls for initial health endowments	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Model 2:	−0.011	−0.011	−0.204***	−0.208***	0.020***	0.020***	−0.004***	−0.004***	0.388	0.365
Y = Share of years employed, 2001-2012 (n = 1040)	[0.024; 0.003]	[0.025; 0.003]	[−0.342; -0.067]	[−0.345; −0.070]	[0.014; 0.026]	[0.014; 0.026]				
Controls for initial health endowments	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Model 3:	0.012	0.012	−0.204***	−0.208***	−0.004	−0.004	0.001	0.001	0.064	0.069
Y = Indicator for social income transfers, 2001-2012 (n = 1040)	[-0.006; 0.031]	[0.006; 0.031]	[−0.342; −0.067]	[−0.345; −0.070]	[−0.012; 0.004]	[−0.012; 0.004]				
Controls for initial health endowments	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Model 4:	0.077**	0.080**	−0.199***	−0.201***	−0.047***	−0.045***	0.009	0.009*	0.120	0.114
Y = Log of average social income transfers, 2001-2012 (n = 922)	[0.003; 0.152]	[0.006; 0.155]	[−0.341; −0.057	[−0.344; -0.059]	[−0.080; −0.013]	[−0.079; −0.012]				
Controls for initial health endowments	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes

Note: Initial health endowments include controls for birth weight, BMI GRS, WHR GRS, triglyceride GRS, HDL GRS, LDL GRS and blood pressure GRS. Results are based on linear regression (OLS) method. Each row refers to one of the four estimated models. The unit of analysis is the individual. The paths are explained in Fig. 1. The three cohorts under study (aged 24, 27, and 30 in year 2001) are drawn from the YFS. All models include controls for the birth year, sex, parental education (1980), and parental earnings (1980). The 95 % confidence intervals for the parameter estimates are reported in parenthesis. Statistical significance: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Second, the results remained intact when the mediation model was augmented with a genetic risk score for education (Supplementary Appendix Table A.3). Finally, we used Oster's (2019) method to evaluate the robustness of our results regarding omitted variable bias. The results in Table 6 suggest that unobservable factors may confound the links between IRHs and education years, and between education years and social income transfer. However, these results are highly conservative as setting $R_{max} = 1$ implies the outcome variable would be fully explained by the treatment and full set of controls. Otherwise, the results based on Oster's (2019) method suggest that the signs of our estimates indicating the links between years of education and labour market outcomes are robust to substantial selection on unobservables.

The results based on the model with education as a mediator remained robust if we replaced the number of IRHs with an indicator variable for those who had experienced at least one IRH in childhood (Supplementary Appendix Table A.4). In addition, we estimated separate mediation models using early (ages 0–5) and later (ages 6–18) IRHs as the explanatory variable of interest (Supplementary Appendix Tables A.5 and A.6). These results were in accordance with our main findings presented in Table 3. Thus, the links between IRHs and labour market outcomes are similar during early childhood and later childhood. We considered the possibility that this result reflects the correlation between early and later IRHs. However, this did not seem to be the case: the correlation between the number of early and later IRHs was weak (r = 0.14), and the link between later IRHs, education and labour market outcomes remained intact if the model was augmented with early IRHs (Supplementary Appendix Table A.7).

4. Discussion

Using linked longitudinal registry data, we found that children who experienced IRHs in childhood had weaker labour market outcomes in terms of earnings and employment than children who had not experienced IRHs in childhood. They also had a higher

Table 6
Robustness to omitted variable bias (Oster's method).

	Years of education		Log of average earnings		Share of employment years		Indicator for social income transfers		Log of average social income transfers	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Treatment variable	$\sim \delta$ for $\beta = 0$ given R_{max}	Identified set given $\delta = 1$ and Rmax	$\sim \delta$ for $\beta = 0$ given R_{max}	Identified set given $\delta = 1$ and R_{max}	$\sim \delta$ for $\beta = 0$ given R_{max}	Identified set given $\delta = 1$ and R_{max}	$\sim \delta$ for $\beta = 0$ given R_{max}	Identified set given $\delta = 1$ and R_{max}	$\sim \delta$ for $\beta = 0$ given R_{max}	Identified set given $\delta = 1$ and R_{max}
Number of IRHs	0.236	−0.169, 0.591	..	..	..	..	..	..	..	..
Years of education	..	..	0.575	0.210, 4.935	0.607	0.032, 0.742	0.083	−0.008, 2.335	−0.429	−0.369, -0.076
R_{max}	1.0		1.0		1.0		1.0		1.0	

Note: Results are computed using Oster's (2013) Stata module *psacalc*. Baseline models are based on OLS and they control for the birth year, sex, parental education (1980), and parental earnings (1980). Based on the results, the unobservable should be 0.236 (Column 1), 0.575 (Column 3), 0.607 (Column 5), 0.083 (Column 7), 0.429 (Column 9) times as important as the observables in order to produce a zero treatment effect (i.e. $\beta = 0$). Alternatively, Oster's method can be used to estimate bounds for the treatment effects assuming that unobservables are as important as observables ($\delta = 1$) as suggested by Altonji et al. (2005). Based on these results, we cannot reject the hypothesis that the links between IRH and years of education (Column 2) as well as between education years and social income transfers indicator (Column 8) are zero. Otherwise, the results based on Oster's (2019) method suggest that the signs of our estimates are robust to substantial selection on unobservables (Columns 4, 6, 10).

probability of receiving social income transfers. We also examined potential mechanisms that may explain the observed connection between IRHs and adult labour market outcomes.

Previous studies have found that early IRHs are related to metabolic outcomes (Burgner et al., 2015a) and adverse cardiovascular phenotypes in adulthood (Burgner et al., 2015b). To test the possibility that health acts as a mediator in the relationship between IRHs and labour market outcomes, we constructed a health risk index based on biomarkers that are related to cardiovascular diseases. However, we did not find support for the hypothesis that adult health explains the worse labour market performance of individuals who had experienced IRHs in childhood. Instead, our results are consistent with the hypothesis that health problems in childhood are related to weaker human capital accumulation. Children who experienced at least one IRH were more likely to participate in remedial education, had lower GPAs in ninth grade, had fewer education years as adults and were less likely to complete university level education. Based on a mediation model, ~30 % of the total connection between IRHs and labour market outcomes (earnings and employment) was mediated through years of education. Mediation through education years also explained ~12 % and ~15 % of the associations between IRHs and the likelihood of receiving social income transfers and the amount of transfers received, respectively. These findings are consistent with earlier studies showing that health problems in childhood reduce possibilities or incentives to invest in human capital in terms of education and that poor childhood health has lasting effects on socioeconomic outcomes (Currie, 2020; Douglas et al., 2018; Karbownik and Wray, 2019; Majid, 2015; Prinz et al., 2018; Schurer et al., 2019). The links between IRHs, education and labour market outcomes were similar during early (ages 0–5) and later (ages 6–18) childhood. This challenges, to some extent, the earlier literature, which has stressed the overwhelming importance of early childhood conditions on human capital development and subsequent labour market outcomes (see, e.g., Almond and Currie, 2011). However, it is possible that mechanisms that link childhood IRHs to education are different in early and later childhood. IRHs in early childhood, for example, may be linked to brain development and the ability to learn, whereas later, school absences play a more significant role.

A limitation of this study is that there may be unobservable factors that explain the negative relationship between IRHs and adult labour market outcomes. To reduce their scope, we augmented all our models with sex, age cohort and parental background (income and education). We also evaluated the possibility that differences in observable initial health endowment or genetic predisposition towards higher education drive the findings, but this did not seem to be the case. Based on Oster's (2019) method, there may be unobservable factors that confound the links between IRHs and education years and between education years and social income transfers. However, these results were based on a conservative assumption that the outcome variable would be fully explained by the treatment and full set of controls. Previous literature has shown that prenatal nutrition (e.g., Prentice, 2017) and exposure to environmental toxicants (e.g., Winans et al., 2011) affect the development of the immune system and may thus produce variability in IRHs. We did not have a direct measure for initial differences in immune systems and thus, to the extent that initial differences in immune systems have an influence on both IRHs and labour market outcomes, this potential confounding factor may affect our interpretation of the connections between IRHs and labour market outcomes.

Based on our findings, IRHs do not seem to predict the adult health risk index among prime working-age individuals. Because health is a multidimensional concept, it may be that the health risk index does not cover all core health aspects that could explain the connection between IRHs and labour market outcomes. This second limitation may lead us to underestimate the role of health as a mediator. However, we did find a significant link between the adult health risk index and labour market outcomes, which suggests that our health risk index is highly relevant in a labour market context. Another potential limitation is that in our data, hospitalization was defined as infection-related if either a primary or a secondary International Classification of Diseases code indicated an infection diagnosis. Thus, we do not have exact information on the severity of the infection, and it is also possible that the primary reason for hospitalization was other than infection. Because our data originate from the FHDR, we were also unable to observe less severe infections that did not require hospitalization. However, hospitalization infection data are much less prone to variation in health-seeking behaviour by families and differences in medical treatment (Burgner et al., 2015b). Finally, our results may not only reflect the effects of IRHs but also the consequences of poor overall childhood health to an extent that was not captured in the health endowments incorporated into the models.

Our data are from Finland where the national health care system is based on universal public health care services to which everyone permanently residing in the country is entitled. Thus, access to health care is universal. The Finnish health care system pays particular attention to young children. In the 1920s, a child health clinic system was established to secure the health and wellbeing of the mother and baby during pregnancy and promote children's physical and mental development. This system, which is free of charge at the point of service and involves nearly all mothers and their children, includes regular nurse visits before and after birth until school age. After that, regular nurse and doctor visits continue at schools. Despite universal access to health care services, we find surprisingly large and robust associations between IRHs and adult labour market outcomes. In countries where such a health care system does not exist or where the coverage of health care services is not universal across the socioeconomic spectrum, these links may be much larger.

A higher incidence of IRHs is linked to lower long-term earnings, a lower share of years employed and a greater need for social income transfers. The associations are partly explained by the links between IRHs and lower educational attainment. These long-lasting associations highlight the need for policies and treatment practices that prevent childhood IRHs and/or provide adequate educational support to children who suffer from IRHs. Future research may further consider the most efficient way to tackle the problem and what type of educational support measures should be provided to children and adolescents who suffer from IRHs.

Declaration of Competing Interest

none

CRediT authorship contribution statement

Jutta Viinikainen: Conceptualization, Methodology, Formal analysis, Writing - original draft. **Alex Bryson:** Conceptualization, Writing - original draft. **Petri Böckerman:** Conceptualization, Writing - original draft. **Marko Elovainio:** Writing - original draft. **Nina Hutri-Kähönen:** Investigation, Writing - original draft. **Markus Juonala:** Conceptualization, Investigation, Writing - original draft. **Terho Lehtimäki:** Investigation, Writing - original draft. **Katja Pahkala:** Investigation, Writing - original draft. **Suvi Rovio:** Investigation, Writing - original draft. **Laura Pulkki-Råback:** Writing - original draft. **Olli Raitakari:** Investigation,

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Appendix A. Supplementary data

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