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Parental Health and Child Schooling

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Abstract

This paper provides new evidence on the impact of parental health shocks on investment in child education using detailed longitudinal data from Bosnia and Herzegovina. Our study controls for individual unobserved heterogeneity by using child fixed effects, and it accounts for potential health misreporting by employing several, more objective, health indicators. Our results show that children of ill mothers, but not of ill fathers, are significantly less likely to be enrolled in education at ages 15-24. Moreover, there is some evidence that mothers’ health shocks have more negative consequences on younger children and sons.[JEL. I21 O15]

Keywords. Human Capital, Intrahousehold allocation, Health shocks, Education, Bosnia and Herzegovina
1 Introduction

The degree to which human capital is transmitted across generations has assumed great importance among both social scientists and policy makers because of its key implications for economic growth and inequality. Among different dimensions of human capital, the enjoyment of good health is crucial for wellbeing in that directly contributing to both individual utility and economic performance (Grossman 1972, Strauss and Thomas 1998, Deaton 2007, Currie 2009, Case and Paxson 2011). Globally though, one billion of people still lack access to health care, and major illness remains one of the most sizable and unpredictable shocks households may face (World Health Organization 2011; 2005). This is especially true in less developed countries where many individuals are not covered by formal insurance mechanisms and out-of-pocket payments are the most important means of financing health care (World Bank 1993; 1995). Ill health may be even more costly in terms of wellbeing and growth if its economic and non-economic consequences are transferred to future generations’ human capital. This paper addresses this issue by examining the impact of maternal and paternal health on children’s education. While evidence on the parent-child transmission of health is accumulating (see, among others, Currie and Moretti 2007, Bhalotra and Rawlings 2011) the extent to which parental health shocks affect investments in child education has received very little empirical attention.

There is a rich literature showing that exposure to income shocks is detrimental to child human capital accumulation when households cannot rely on formal or informal mechanisms to smooth out negative events (Jensen 2000, Edmonds 2006, Dureya et al. 2007). In particular, households facing adverse shocks may divert child time away from education and towards labour in order to substitute for adult work or generate immediate income (Jacoby and Skoufias 1997, Beegle et al. 2006, Kruger 2007). Yet, while evidence has been concentrated on the income effect of shocks in market production (e.g. agricultural income shocks, job losses, etc.), less attention has been paid to the direct and indirect consequences of household adult members’ health on children’s schooling decisions. As emphasized by Morduch (1995), income changes may be the result of ex-ante smoothing strategies, which is not the case for the
type of large and unpredictable shocks that are represented by changes in health status. Adverse health events typically have an impact on labour supply while also squeezing resources for consumption, due to forgone income and higher health care expenses (Hamoudi and Sachs 1999, Dercon and Krishnan 2000, Wagstaff 2007). Moreover, parental health status may have direct non-pecuniary effects on the child’s schooling, over and above the pecuniary effects. These influences may act through the reduction of the quantity and/or quality of parental time inputs into child rearing, or the emotional distress caused to children. In a related literature relevant for this paper, it has been shown that parental death has a significant negative impact on child education (Gertler et al. 2004, Case and Ardington 2006, Chen et al. 2009). The same body of literature though, recognizes that if ill health predates parental death, the former is an important confounding factor.

We examine the role of parental health in child schooling by using the detailed Bosnia and Herzegovina (BiH) Living Standards Measurement Survey (LSMS), one of the few household panel datasets available for transition countries, conducted by the World Bank between 2001 and 2004. The longitudinal nature of the BiH LSMS and its richness of health-related information allow us to tackle two important problems which commonly arise when estimating the effect of parental health on children’s education: unobserved heterogeneity and measurement error.

A first challenge is to disentangle spurious correlation, due to unobserved heterogeneity, from causality. Parents with high intertemporal discount rates, for instance, are likely to engage in health-damaging behaviour, have worse health, and at the same time invest less time and money in their children’s human capital. In order to address this endogeneity concern, we employ longitudinal data and, for the first time in this literature, a child fixed effects estimator. This allows us to identify the effect of parental health by considering changes in parents’ health status overtime, that is health shocks. A key advantage of the latter approach is that health shocks are less likely to reflect long-term parental illnesses or health-related behavior than health status.

A second difficulty in the identification of the treatment effect of interest is related to the
subjective nature of self-reported health. Indeed, if true and self-declared health differ due to a misclassification error, an attenuation bias will affect both the OLS and the fixed effects estimator, leading to a lower-bound estimate of the parental illness effect. We then address this issue by employing a detailed set of alternative indicators of parental health status, which are available in the BiH LSMS and are generally considered as less subject to reporting bias.

Besides these important features of the BiH panel survey, which help the identification of the causal effects of interest, we focus on a particularly interesting setting where to study the impact of parents’ health shocks on children’s human capital. Indeed, before the 1992-1995 war BiH enjoyed the economy, health status, and health care of a middle-income country, but the conflict left the country’s physical and human resources devastated. Health services, especially those supporting women and children, were severely disrupted, with over 35% of facilities destroyed or heavily damaged (DFID 2003). Half of the country’s schools were destroyed during the conflict, decreasing access to education (World Bank 2005). Thus, due to the pervasive destruction of both the health and the education systems, the effect of parental health on child schooling is of particular concern.

Our preferred child fixed effects estimates show that children with only mothers with self-reported poor health are about 7 per cent points less likely to be enrolled in education at ages 15–24 compared to children with healthier parents, while we do find much lower and statistically insignificant effects of paternal illness. Similar results are obtained using less subjective measures, such as an index of limitations in activity of daily living: children of mothers with severe limitations are 9 per cent points less likely to be in school, while no significant effect is found for fathers. Thus, it appears that—contrary to the common wisdom that shocks to the primary household earner should bear more negative consequences for child education—it is especially maternal health that makes a difference as far as child schooling is concerned. We also find that younger children and sons suffer the most from their mother’s illness.

The structure of the paper is as follows. Section 2 discusses the role of parental health in determining children’s human capital acquisition, as explored in the existing literature. Section 3 describes the context of Bosnia and Herzegovina. Section 4 presents the econometric
strategy and challenges to identification. Section 5 describes the data and reports some descriptive statistics. Results using parental self-reported poor health status and more objective measures of health are presented in Section 6. Section 7 presents heterogeneous results by child’s attributes. Section 8 concludes.

2 Background literature

In the absence of an adequate system of social protection, illness can take a large and unexpected toll on household well-being, leaving little scope for ex-ante income smoothing strategies (Morduch 1995, Gertler and Gruber 2002). Adverse health events impose to household members current pecuniary costs, both direct, in terms of the price of accessing health care, and indirect, in terms of the loss of income associated with reduced labour supply and productivity. As a result, having a major health shock may make a family experience both a short-term income fall and a prolonged poverty trap (Wagstaff 2007, Sun and Yao 2010). Based on the theory of full insurance, Gertler and Gruber (2002) test and reject the hypothesis of consumption smoothing in the context of Indonesia, showing that households significantly reduce both labour supply and consumption patterns when hit by an adverse health event. Similarly, Asfaw and von Braun (2004) show that in Ethiopia illness has a significant negative impact on the stability and the level of household consumption. Focusing on the direct monetary costs of health, instead, Wagstaff (2007) finds evidence that the financial implications of ill health in Vietnam can be catastrophic, being associated with a significant reduction of consumption in households with no access to insurance (see also Dercon and Krishnan 2000, Baeza and Packard 2005, Bredenkamp et al. 2010).

In countries with poor systems of social protection, though, ill health may have significant economic consequences for both current and future generations (Hamoudi and Sachs 1999, Wagstaff 2007). Drawing from the economic theory of the household, if families with ill members are not able to access formal insurance markets—as it is likely to be the case in less developed or poor contexts—they may be compelled to rely on other coping mechanisms such
as trading the future welfare of all or some of their members against current access to health care or forgone income for one of them (Strauss and Thomas 1995). This is to say that when hit by an adverse health event, households may increase their use of child labour, by having children substitute for adult labour supply, thus decreasing school attendance. In the absence of adequate health insurance, older children may also be asked to take care of the sick parent, reducing the time they can devote to work or schooling.

Furthermore, parents’ illness may have non-pecuniary, e.g., psychological, costs on children, which negatively impact on their school achievement (Pedersen and Revenson 2005, Sieh et al. 2010). Last but not least, as parents not only contribute monetary inputs but also time inputs into the “production” of child quality, their poor health status may reduce both the quantity and the quality of their time contributions, and negatively affect a child’s quality, in our specific case, education (Guryan et al. 2008, Gayle et al. 2011)

In the conclusion to their well known survey on the determinants of children’s attainments Haveman and Wolfe (1995) mentioned information on the health status of both parents and children as one of the most pressing data needs in this area of research. However, more than 15 years later papers written by economists on the effects of parental health on children’s educational achievement can still be counted on the fingers of one hand. In a related literature, some influential studies investigate the effect of that extreme form of health shock which is parental death. Gertler et al. (2004) use three repeated cross-sections of household data from Indonesia to test how the loss of a parent affects investment in children. They find that a parent’s recent death has a large effect on the child’s school enrollment, irrespective of the gender of the child and of the parent who dies. On the other hand, using longitudinal data Case and Ardington (2006) and Chen et al. (2009) present strong evidence that maternal death has a much larger impact on child education than paternal death in sub-Saharan Africa and Taiwan respectively. Adda et al. (2011) find for Sweden that mothers are somewhat more important for children’s cognitive skills and fathers for noncognitive ones. All the above mentioned papers, though, recognize that if important health problems predate parental death, the treatment effects might be seriously biased. To put it in other words, parental health is considered as a confounding
factor.

From a policy perspective, the international community is increasingly concerned about the growth-dampening effects of low levels of human capital on the one hand, and about the impact of better health care (and effective risk protection) on well-being and development on the other (e.g., the Millennium Development Goals; World Bank 2007). This is even more relevant if ill health has (intergenerational) implications in terms of intra-household resource allocation and investments in children’s human capital. Yet, as mentioned above, there is little empirical evidence pointing explicitly at the effect of parental health on child schooling. Sun and Yao (2010) investigate the consequences of adults’ health shocks on a child’s likelihood of entering and finishing middle school using Chinese panel data. They find that primary-school age children are the most vulnerable to severe health shocks, measured by out-of-pocket health expenditures larger than a given threshold, and that girls are more susceptible than boys to the damage of these shocks. Choi (2010) analyzes the long-run effects of parental self-reported poor health on children’s probability of having completed at least 15 years of schooling in Russia. Her results show that a father’s poor health status is a significant predictor of lower daughter’s educational attainment and probability of working during adulthood. Morefield (2010) investigates the effect of poor parental health, proxied by health conditions which limit an individual’s daily activities or ability to work, on children’s cognitive and non-cognitive skills in the US. Cognitive skills are measured by the Revised Woodcock–Johnson (WJ-R) applied problem achievement test.¹ His results indicate that parental health is determinant only for non-cognitive skills, that health shocks related to a vascular or cancerous condition bear more negative consequences, and that sons are more negatively affected than daughters.²

¹The WJ-R applied problem test evaluates a child’s ability to solve practical mathematical questions and is a measure of quantitative knowledge, while non-cognitive skills refer to behavioural problems and are measured using the Behavior Problems Index (BPI) developed by Peterson and Zill (1986).

²Although their paper is not primarily focused on the effect of parental health on child education, Goux and Maurin (2001) estimate an earnings equation and use parents’ health conditions as excluded instruments for education. They find a negative but statistically insignificant effect of mother’s death on child years of schooling, a positive and significant effect of father’s death, and no effect of mother’s severe health problems during the individual’s childhood. However, the latter result may be partly due to recall bias and small sample size (1019 observations).
Our study adds to the existing literature in several respects. As to the identification strategy, unlike previous studies, we take into account child unobserved heterogeneity using child fixed effects (see Section 4). To the best of our knowledge this is the first paper using such an identification strategy in the context of evaluating the effects of parental health on child schooling. Indeed, former studies have generally used longitudinal data but have focused on measures of schooling observed only at one single point in time, such as the highest level of schooling achieved at a given age (Sun and Yao 2010, Choi 2010). Moreover, we are able to account for potential measurement error or misreporting bias of health status by using multiple, more objective, measures of parental health shocks. This is particularly valuable for the difficult task of studying health consequences. Health status is multi-dimensional, many data sources isolate only a few dimensions of it, and health indicators are often reported with considerable error or are biased by the respondents' socioeconomic status or beliefs. Hence, in our analysis we employ different health measures such as parental self-reported health status, limitations in activities of daily living and indicators of mental health ('depression scales'). We further depart from the previous literature with respect to the specific outcome variable considered. Due to the limited length of our panel data, we focus on the short-run effects of parental health, namely the effects of recent shocks to parents’ health on the child’s current school enrollment. As mentioned above, Choi (2010) focuses on the long-term effects in terms of higher education achievement, while Morefield (2010) considers attainment in a standardized test. Both short-run and long-run effects are of interest. When considering the short-run effects of parents’ health shocks, it may be argued that a child’s school drop-out may be only temporary, since individuals could go back to education when the parents’ health improves. As we have panel data,

3In two related studies, Thirumurthy and Goldstein (2008; 2009) use longitudinal data combined with a treatment program in western Kenya to investigate the intra-household consequences of AIDS treatment. They show that health improvements through exogenously provided therapy have important implications for treated adults who begin or resume productive work. Furthermore, children living in households with HIV-infected adults who are on treatment are more likely to attend school than those in households with untreated adults. Yet, differently from our study, the above papers focus on a particularly debilitating and chronic disease among a very specific population of already-ill adults.

4The same is done by Gertler et al. (2004) in their study of parental deaths. Similarly to our paper, Sun and Yao (2010) investigate the likelihood of entering or finishing middle school, but they consider both long and short-run effects of parental health. Indeed, the retrospective section of their data allow them also to consider health shocks at children’s pre-school and primary school ages.
we do account for this potential issue unless the time elapsed between quitting and re-entering education is very long, in which case we claim drop-out can be considered as a particularly negative outcome. Moreover, the fact that an individual quitted school per se may make the option of re-entering the educational system less attractive, as the future benefits of schooling fall with age, while the costs, especially the psychological ones, are likely to increase with age and time spent out of education. Considering short-run effects also gives us the advantage that it is easier to keep under control potential confounding factors in the analysis, while in studies of long-term effects it is very difficult to account for all events which have potentially intervened between the time parental health worsened and the time children’s outcomes are observed. Moreover, as we are only using current and not retrospective information, self-reports of individuals on health are less likely to be affected by a recall bias.

3 The country context

Formerly one of the six federal units constituting the Socialist Federal Republic of Yugoslavia, BiH gained its independence during the Yugoslav wars of the 1990s and is now transforming its economy into a market-oriented system. With a population among the youngest in the European region, BiH is a country where health and education levels are substantially below those of neighbouring countries. Prior to the war, BiH was a country with a GDP of US$11 billion, a per capita income of US$2,400 and a sophisticated health system. Primary and secondary schools were free, with primary education (for those aged 7–15) compulsory so that the completion of the first nine years of schooling was virtually universal. The war, though, destroyed much of the country’s infrastructure and economy and the toll on the population was extremely severe (DFID 1999). By 1995, GDP had declined to US$2 billion, and the per capita

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5In our estimation sample, we observe 133 individuals who dropped out from school or did not go on in the next educational level among which only 2 re-entered education during our observation period. Thus, quitting (or not going on in) education seems to be quite a long-lasting or permanent state in our sample.

6For instance, in Sun and Yao (2010) individuals were asked in 2003 about major illnesses of any family members during 1987–2002.

7The provider network was publicly owned and financed through a para-state insurance system that provided health insurance, social security and disability insurance.
income to US$500. Unemployment was estimated to have risen to 80%. With the support for reconstruction provided by The World Bank, the European Commission (EC), and a broad coalition of donors, by the end of 2000 macroeconomic stability had been achieved despite extremely unfavourable conditions. Annual economic growth has averaged about 40% in real terms since 1995, and GDP reached US$7 billion in 2001, with per capita income approaching US$1,800 (DFID 2003).

The post-conflict transition posed major challenges also to labour force participation and employment in the new labour market. The 2007 Labor Force Study estimates the overall unemployment rate in BiH at 31.8%, with youth unemployment much higher than for adults (up to 60%). This is among the highest in the region, and according to a recent State commission’s study on youth issues, BiH’s unemployment rate is about four times the EU average (CCYI 2008).

On the side of social services, the Bosnian health system was devastated by the war. One third of all health infrastructures were totally destroyed. About 30% of the doctors and nurses left the country or were killed in the conflict. Government financing of the service is no longer in place. There are two health systems, one for the Federacija Bosna i Hercegovina (FBiH) and one for the Republika Srpska (RS). Both Ministries of Health lack the necessary financial resources and are highly dependent on external funding and humanitarian aid. Before the war, health care services were entirely covered by the social system, which collapsed during the war. In the FBiH it was replaced by an insurance fund that merged with the Federal Ministry of Health. In RS an insurance fund operating from Mostar came into operation. However, in reality the health system is funded through a diversity of sources (DFID 2003) and is still far from being able to provide financial protection against adverse health events, with only 60% of the population covered by health insurance. As with other countries in the region, the major reconstruction process is now focused on enhancing the efficiency of public spending. In the health sector, the main challenge is to make progress in the population’s health status while providing protection against the short- and long-term costs of illness in terms of human capital levels and growth. Indeed, health outcomes in BiH are below those found in other countries.
of the region. Some key outcome indicators raise concerns: the incidence of tuberculosis is four times higher than the EU average; disability, post traumatic stress, depression, and chronic diseases rank high on the burden of diseases. Accidents and injuries are at a high level and appear to be rising. The incidence of high-cost diseases of the heart and circulatory system, stroke, and cancer is above the European averages (World Bank 2005).

The war hampered access to education as well. Many school buildings were damaged, destroyed or forced to be converted into refugee centres and hospitals during the war (Mazowiecki 1994, Swee 2009). Reliable enrollment data during the conflict is very rare but it has been estimated that 50% of the schools in BiH required repair or reconstruction after the conflict (World Bank 2005). Furthermore, teachers also became a scarce resource due to out-migration, decreasing access to education even further. Even though several reports suggest that the remaining teachers strove to share energy and resources in order to informally organize classes in occasional locations, this was easier for primary education but more difficult for secondary education and above, due to the more specialized curricula.

Currently, the BiH education system is articulated in three levels: primary, secondary and university. Primary education is free and compulsory, and lasts for 9 years, between ages 7 and age 14. Secondary education is also free and lasts for 3 or 4 years, for the general and the technical school tracks, respectively. Students graduating from the general track can enroll in university, where undergraduate courses typically last for three to four years.

Overall, education access has suffered seriously as a result of conflict, leaving a lasting impact and developmental lag. Primary school enrollment recovered rapidly following the conflict. By contrast, secondary and tertiary education display less consistent patterns of resilience, although they suffered equal or greater damage during the conflict, and the gross enrollment rates started from a much lower level (World Bank 2005). Overall, 40% of the students do not acquire basic skills and knowledge by the end of fourth grade, while many students enrolled in costly vocational schools receive insufficient general education and are ill-equipped to meet the challenges of today’s labour markets. While primary education enrollment rates remain high at about 93%, BiH has the lowest rate of net secondary enrollment (73% overall, with only
57% of the poor attending) of all transition countries for which data are available (World Bank 2005). Overall, free access to the education system and high youth unemployment rates make child schooling a relatively low-cost investment in BiH.

4 Econometric model and identification strategy

We use a simple conceptual framework to motivate our empirical strategy. Let us assume that a child’s school enrollment is determined according to the following process:

\[ s_{it} = f(M_{it}, T_{it}, E_{it-1}, a_i, \rho_i) + \epsilon_{it} \]  

(1)

where \( i \) and \( t \) are individual and time subscripts, respectively. \( s_{it} \) is a dummy indicator for school enrollment, which takes value one in case the child is enrolled in education and zero otherwise. \( M_{it} \) and \( T_{it} \) are, respectively, the money and (effective) time that parents invest in child quality. \( M_{it} \) will include the money spent on school quality and books, for instance, while \( T_{it} \) captures parents’ involvement in a child’s education, e.g., the time spent helping the child with homework, talking to teachers, etc. \( a_i \) and \( \rho_i \) are usually unobservable, and stand for child inherited ability and parents’ intertemporal discount rates, respectively. \( E_{it-1} \) is the stock of education acquired by the child at time \( t - 1 \) (e.g., the highest educational title acquired).\(^8\) \( \epsilon_{it} \) is a random shock to child enrollment, which is assumed to be orthogonal to all the other variables entering \( f(\cdot) \). For the sake of simplicity, we assume that the relation \( f(\cdot) \) is linear.

Parents have to choose the optimal investments in a child’s education, subject to a time and a budget constraint.

Hence, money and time inputs invested in a child’s education are the outcome of optimal

\(^8\)In the language of Todd and Wolpin (2003) we specify a ‘value added’ model. In equation (1) we assume that the impact of past parental inputs is fully captured by \( E_{it-1} \) and that random shocks to education (\( \epsilon_{it} \)) are not serially correlated. This specification is often used in the literature. For a recent application see Cunha and Heckman (2007).
parents’ choices (Becker 1981),

\[ M_{it} = M(w_i, w_{it}, H_{it}, a_i, \rho_i) \]  

(2)

\[ T_{it} = T(w_i, w_{it}, H_{it}, a_i, \rho_i) \]  

(3)

where \( w_i \) and \( w_{it} \) are time invariant and time variant parents’ characteristics, respectively, such as education, income and wealth, and \( H_{it} \) is parental health.\(^9\) \( H_{it} \) is likely to affect both money inputs into a child’s education, as health expenditures reduce the household income available for alternative uses, and time inputs, by reducing parental involvement in child education. Again, \( M(.) \) and \( T(.) \) are assumed to be linear functions.

Furthermore, parents’ current health is itself the result of past parents’ investments (Grossman 1972, Dupas 2011), which depend on parents’ characteristics

\[ H_{it} = H(w_i, \rho_i, w_{it}) + \xi_{it} \]  

(4)

where \( \xi_{it} \) is a random ‘health shock’, and \( H(.) \) a linear function. Parents’ health depends on predetermined (and largely time-invariant) characteristics such as their levels of education (see, for instance, Currie and Moretti 2003, Lleras-Muney 2005, de Walque 2010), but also on their discount rates (Farrell and Fuchs 1982), both of which will determine their health investment, and on time variant factors (e.g., the local supply of health facilities).\(^{10}\)

As money and time inputs are rarely observed, it is often customary in this literature to substitute equations (2) and (3) into equation (1) to obtain the estimable form

\[ s_{it} = \tilde{f}(w_i, w_{it}, H_{it}, E_{it-1}) + \tilde{a}_i + \tilde{\rho}_i + \varepsilon_{it} \]  

(5)

where \( \tilde{f}(.) \) is a function linear in all arguments, and \( \tilde{a}_i \) and \( \tilde{\rho} \) are two linear functions in child

\(^9\)For simplicity, we refer to parental health, although in the empirical analysis we consider mother’s and father’s health separately.

\(^{10}\)For the sake of brevity, we have specified a ‘reduced form’ for parents’ health omitting further equations for parents’ investments in their own health.
ability and the intertemporal discount rate, respectively. The new error term becomes \( v_{it} = \tilde{a}_i + \tilde{p}_i + \varepsilon_{it} \). Thus, equation (5) shows that \( H_{it} \) is endogenous with respect to child education, i.e. both variables depend on the same unobservable factors. OLS estimation of (5) will then give inconsistent estimates of the treatment effect of interest \( \frac{\partial \tilde{s}_{it}}{\partial H_{it}} \). A possible remedy to this problem is to use a child fixed effects estimator. Child fixed effects are likely to capture the impact of all (observable and unobservable) time-invariant characteristics of both children and parents. Conditional on the time-varying controls \( w_{it} \), the only remaining variation in parents’ health status to be exploited is the one coming from health shocks \( \xi_{it} \), which are assumed to be random.

Our aim is to estimate a child’s school enrollment equation in which parents’ self-reported health status appears as a regressor. We use a linear probability model (LPM hereafter):

\[
s_{ict} = \alpha_0 + \alpha_1 PM_{it} + \alpha_1 f PF_{it} + \alpha_1 mf PMF_{it} + \alpha_2 x_{it} + \alpha_3 w_{it} + \delta_c + \delta_t + v_{it} \tag{6}
\]

where \( i, c, t \) are subscripts for individuals, cities of current residence, and calendar years, respectively, \( s_{ict} \) is a dichotomous indicator for a child’s school enrollment, and \( PM_{it} \), \( PF_{it} \) and \( PMF_{it} \) are indicators of poor health status (self-reported) by parents. We have included three different indicators: \( PM_{it} \) takes the value one if only the child \( i \)’s mother reported poor health at time \( t \), \( PF_{it} \) equals one if only the child’s father reported poor health, and \( PMF_{it} \) equals one if both parents reported poor health. This way of specifying the child schooling equation—instead of including the mother’s and father’s poor health as separate regressors—has two main

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11Indeed, \( f(\cdot) \) being linear is separable in the arguments.
12Hence, the main identifying assumption in the child fixed effects estimator is that \( \varepsilon_{it} \) is orthogonal to the included regressors.
13This simple conceptual framework also suggests why in general it is much more difficult to identify the effect of long-term parental illness. In the latter case, indeed, it is not possible to control for child fixed effects, as there is no time variation (or too little variation) in parents’ poor health status. However, without controlling for child fixed effects one cannot be sure that parents’ chronic illness will not be capturing the effect on child education of other parental unobservable attributes which do not vary overtime (e.g., their intertemporal discount rate).
14We follow the recent literature on treatment effects (see for instance, Angrist 2001) and use LPMs. In our case, the LPM is preferable compared to non-linear models such as probit, as we include in the estimation child fixed effects. Unlike the fixed effects estimator, indeed, random effects probit models assume that random effects are uncorrelated with the regressors. Using conditional logit models, instead, would force us to focus only on ‘movers’, that is on parents whose health status changes during the observation period, further reducing our sample size and the precision of our estimates.
advantages: (i) it reduces potential multicollinearity problems between the mother’s and father’s health status as the three health indicators are mutually exclusive, (ii) it is more general, in that it allows for non-linearities in the effect of parental health and relaxes the additivity assumption.15 \( x_{it} \) is a vector of individual time-varying and time-invariant characteristics such as age and sex,16 \( w_{it} \) a vector of child \( i \)'s parental time-varying and time-invariant characteristics such as education and age, \( \delta_c \) and \( \delta_t \) are city and calendar year fixed effects, respectively, which capture city-specific time-invariant unobservables and nationwide time trends or macroeconomic conditions, and \( v_{it} \) is a time-varying individual error term. The \( \alpha \)'s are parameters to be estimated.

The potential endogeneity of parental health status must be tackled when pursuing the task of estimating the causal effect of parental health on a child’s schooling. As we stressed in our conceptual framework, parents’ unobservables are likely to determine both parental health and child schooling and enter the error term \( v_{it} \) causing an endogeneity problem. One possible example of such unobservables are parents’ intertemporal discount rates: parents with low discount rates will invest more both in their health and in their children’s education. OLS estimates of equation (6) are likely to be affected by this source of bias and provide larger negative estimates of the impact of parental health on child schooling. If endogeneity is only generated by time-invariant individual unobservable characteristics, one possibility for getting rid of the endogeneity bias is by using a fixed effect (FE) estimator.

Let us assume that the individual error term in equation (6) is additive and consists of a time invariant part \( (u_i) \), which may be correlated with the regressors included, and a white-noise \( (\varepsilon_{it}) \), i.e., \( v_{it} = u_i + \varepsilon_{it} \). Then equation (6) can be rewritten:

\[
\begin{align*}
s_{ict} &= \alpha_0 + \alpha_{1m} P_{Mi} + \alpha_{1f} P_{Pi} + \alpha_{1mf} P_{MFi} + \alpha_{2x} x_{it} + \alpha_{3w} w_{it} + \delta_c + \delta_t + u_i + \varepsilon_{it} \\
&= \alpha_0 + \alpha_{1m} P_{Mi} + \alpha_{1f} P_{Pi} + \alpha_{1mf} P_{MFi} + \alpha_{2x} x_{it} + \alpha_{3w} w_{it} + \delta_c + \delta_t + u_i + \varepsilon_{it}
\end{align*}
\]

(7)

and the FE estimator with child fixed effects will deliver consistent estimates of the treatment.

\[15\] In more detail, the effect of having two parents with poor health is no longer equal to the sum of the effects of having each parent in bad health conditions.

\[16\] These variables have not been considered for brevity in the conceptual framework.
effects of interest ($\alpha_{1m}$, $\alpha_{1f}$ and $\alpha_{1mf}$). As the reader will notice, only one source of potential endogeneity remains unaddressed by the FE estimator above: the one coming from unobservable determinants (or correlates of) time-varying *yearly* shocks to parental health that are also correlated with factors directly affecting a child’s education ($\epsilon_{it}$). It is hard to think what these unobservables might be: they must be factors which change very suddenly (during one year), and simultaneously affect both parental health and children’s education. In any case we believe that they are unlikely to be frequent. For instance, they might be serious accidents which involve parents and children, causing both a deterioration of the health status of parents and children and a reduction in the school attendance of the latter. To avoid these odd cases, we will check the sensitivity of our estimates to including in the empirical specifications also the children’s (and their siblings’) health status. Were time-varying unobservable shocks common to all family members the main cause responsible for the correlation between parental health and child schooling, we would expect the coefficient on parental health to significantly decline after including the additional regressors.

It is important to stress what are our main source of identification and identifying assumption. In the child fixed effects estimator, identification comes from time variation within the same individual in parental poor health status, i.e., by ‘health shocks’ (either positive or negative) which trigger parental poor health. Accordingly, the main identifying assumption is that, conditional on child and parent observables, such shocks are exogenous. The idea is that children living in certain families may be systematically (i.e., in each period) more likely to live with ill parents, but that the timing of the deterioration or improvement of their parents’ ‘poor’ health status is substantially random with respect to their demand for education after controlling for a large set of covariates.

As it is well known, self-reported health may be affected by a considerable measurement error (reporting bias). Due to the dichotomous nature of our ‘poor health’ indicator (see the next section), the measurement error is non-classical and takes the form of misclassification error (see, among others, Aigner 1973, Klepper 1988). Under rather general conditions Lewbel
(2007) shows that misclassification error produces an attenuation bias, which is likely to be exacerbated using a FE model. For this reason, our child FE model may provide lower-bound estimates of the effect of parental health on child schooling. In order to assess the potential implications of misclassification on our estimates, however, we consider in Section 6.2 alternative indicators of parental health, which are generally thought to be less prone to reporting bias.

How does our strategy compare with the identification strategies employed in the past literature on the effects of parental health or death? There are mainly three types of studies. A first group makes an attempt to address endogeneity by simply including in the estimation children’s observable characteristics (Gertler et al. 2004, Sun and Yao 2010, Choi 2010) or using matching techniques to compare ‘similar’ individuals (Gertler et al. 2004), relying in both cases on a ‘selection on observables’ assumption. Similar in spirit is Morefield (2010), who uses a ‘value-added plus lagged inputs’ model (Todd and Wolpin 2003) by including in the current child attainment equation both lagged attainment and lagged parental inputs. As stressed by the author, this approach enables him to take into account lagged unobserved heterogeneity but not current unobserved heterogeneity, which may remain an important source of endogeneity of health status. Thus, the author makes an attempt to address this issue by including many parents’ and children’s observable characteristics.

A second group of studies seeks to improve identification by using household fixed effects, and exploiting differences in educational achievement between siblings (Chen et al. 2009, Adda et al. 2011). Chen et al. (2009), for instance, identify the causal effect of parental death by estimating differences in college enrollment between siblings who are orphaned by an unexpected accident before versus after the age of 18. The main identification assumption, there and in similar studies, is that the best control group for an individual are his/her siblings. However, this is not necessarily the case, as children of the same parents may differ by their ability levels or non-cognitive attributes, and parents may vary their monetary and time inputs across children.

17 The two conditions are that misclassification does not affect the true expected outcome and that on average observations of the treatment are more accurate than pure guesses.
using compensatory or reinforcing policies (Ermisch and Francesconi 2000). Our identification strategy instead explicitly accounts for children’s specificities by using child fixed effects, and allows us to consider also one-child families.

A third stream of studies makes an attempt to address endogeneity, or to bound the endogeneity bias, by using ‘most exogenous’ sources of parental death or some assumptions about the time-pattern of endogeneity respectively (Adda et al. 2011).

5 Data and descriptive statistics

Our empirical analysis is based on BiH LSMS, a panel survey conducted by the World Bank in four consecutive years (2001, 2002, 2003, and 2004). The 2001 survey is nationally representative and contains over 5,400 households and more than 9,000 individuals, half of which were re-interviewed for the panel in the following years. The attrition rate across waves is around 5%, which is relatively low compared to other national panels. Questions were asked to each individual household member of age 15 or older, while for younger members information was provided by parents or guardians. The survey contains detailed information on individual health status (both self-reported health and physical disabilities) and educational levels along with detailed demographic characteristics of household members, household asset endowments and wealth position, ethnicity, and area of residence. Consumption and income aggregates are available only for the 2001 and 2004 waves, while self-reported health status was asked in 2002, 2003 and 2004. Hence, we restrict our analysis to the last three waves. Our population

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18 Other potential weaknesses of the household fixed effects estimator are stressed by Adda et al. (2011) and concern the fact that it implicitly uses for identification only children in families with two or more children, and in the case of Chen et al. (2009) with a certain spacing between births. These subpopulations, and the treatment effect there estimated, may not necessarily be representative of that in the general population. Moreover, focusing on families with more than one child only, reduces the sample size and therefore the precision of the estimates. Finally, this approach may induce a sample selection bias as specific health conditions are likely to affect women’s or men’s fertility (diabetes, high blood pressure, obesity, etc.), which could influence children’s educational attainment (see, for instance, Cáceres-Delpiano 2006, Booth and Kee 2009).

19 Adda et al. (2011) assume that the amount of endogeneity is constant or decreasing during childhood. To put it in other words, they assume that parental deaths at early ages are more likely to be endogenous than at later ages (p. 10).
of interest comprises children aged 15–24 living in families with both parents currently alive. This is so as in BiH primary education starts at age 7, is free and compulsory till age 14, and compliance with the school obligation is universal. In our data, for instance, the average school enrollment rate between ages 7 and 14 is 99 per cent.

Moreover, since we need information on parental data, we necessarily have to focus on individuals who reside with their parents. Among individuals aged 15–24 in the BiH LSMS, the vast majority (83%) live with their parents. The corresponding percentages are 90% in the 15–19, and 74% in the 20–24 age group.

Co-habitation could introduce a sample selection bias, if any, but this is a real possibility only for older children. Indeed, we may expect the latter to be more likely to coreside with parents in bad health conditions to offer them daily assistance. This could also be negatively correlated with children’s school enrollment and generate an upward bias in our estimates. However, if cohabitation is more likely when parents suffer from long-term, i.e., chronic, health impairments, child fixed effects are likely to attenuate the severity of this selection bias.

The sample selection criteria are detailed in Table 1. The final sample is an unbalanced panel of 785 individuals and 2,060 observations.

Current school enrollment of children aged 15–24 is the outcome of interest, which we measure with a dummy variable equal to one if the individual is enrolled in education. This variable allows us to estimate both the probability of dropping out and of not enrolling in the next level of education. We do not distinguish between the two, since this would require modeling also past student status (dynamic panel), but we do not have enough observations to distinguish between the two outcomes. Yet, both dropping out and not continuing can be considered as negative outcomes, as in both cases children will end up with fewer years of

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20 Indeed, in order to focus on parental health only, and to avoid its effect being confounded with those of parental absence and parents’ deaths, we exclude single parent households and parental deaths.  
21 Unfortunately, since the LSMS does not provide information on parents’ health for children who left parental home, we cannot run an analysis of the probability of co-habitation, and of the impact of parental illness on the latter.  
22 However, as we will see later, we do not find an effect of parents’ illness on older children’s school enrollment.  
23 Due the sample selection we made before the analysis, we did not use survey weights as they no longer reflect population proportions.  
24 The survey’s question is: ‘Are you presently attending school?’.
schooling. We do control for the (time varying) highest diploma achieved by the individual, though. This is done so as to capture the fact that some individuals do not go on in education because they already achieved their desired level of schooling (e.g., many individuals quit at the end of secondary schooling irrespective of parental health).\footnote{By including past educational achievement, our model resembles a value-added model (Todd and Wolpin 2003). To check the robustness of our results, we have also estimated a model controlling for lagged school enrollment and the results (in Table A1) are very similar to those presented in the main text.} Hence, after controlling for the highest diploma held by individuals, we are able to estimate whether parental health has a contemporaneous effect over and above the level of education already achieved. In the whole sample the school enrollment rate is 52%, 73% in the age group 15–19, and 48% in the age group 20–24. Boys’ (girls’) school enrollment rate is 43% (63%). In the age group 15–19 (20–24) 70% (38%) of boys and 78% (60%) of girls are enrolled in education. Thus, school enrollment generally appears to be more frequent among girls, especially after age 19.

In order to measure a parent’s poor health status, i.e., a major illness’s having potentially severe consequences for the rest of the family, we use a dummy variable equal to one if the individual reported her/his health condition over the last fourteen months as ‘poor’ or ‘very poor’ (compared to the other categories provided by the survey question, that are ‘excellent’, ‘good’, and ‘fair’).\footnote{In 2004, the survey question is: ‘Please think back over the last fourteen months about how your health has been. Compared to other people of your own age would you say that your health has been on the whole’, with the possible answers reported in the main text. In 2002 and 2003 the question refers to the last twelve months. In principle, it would be interesting to consider the effect of the severity of illness, and all different categories of the Likert scale. However, we are forced to dichotomize the health indicator as there are too few individuals who declare to have ‘very poor health’ (2.8 per cent of fathers and 2.9 per cent of mothers in our estimation sample).} In what follows, we will refer to these parents as those with ‘poor health’ or ‘ill’. It should be noted that this indicator encompasses both temporary and chronic diseases. Our identification strategy based on changes in health status (child fixed effects), however, exploits the occurrence of a sudden illness and not long-standing chronic diseases, whose effect is captured by child fixed effects.

As we mentioned above, self-reported health status may contain a considerable amount of noise, and we will also consider in Section 6.2 information on more objective indicators of physical disabilities and mental health. In our sample, for about 9.8%, 10% and 9.6% of observations only the mother’s, only the father’s, and both parents’ health status is poor, re-
Since in our preferred child fixed specification the effect of parental health is identified by ‘switchers’, i.e., children for whom parental poor health status changes at least once during the observation period, it may be important to assess for how many individuals this happens. In our main estimation sample, the mother’s poor health changes for 147 observations, the father’s poor health for 175 observations, and both parents’ poor health for 152 observations. Health ‘shocks’ are almost evenly split between bad shocks (i.e., changes in poor health from 0 to 1) and good shocks (changes from 1 to 0). Indeed, in the sample positive shocks for mothers, fathers, and both parents are 70, 83 and 79, respectively. The corresponding figures for negative shocks are 77, 92 and 73. From these statistics, it appears that the incidence of both positive and negative shocks is quite similar for mothers and fathers.

Tabulations for the population of 15–24 years old children indicate that 57% of those living with ‘healthy’ parents are students, while the enrollment rate drops to 37% if both parents report poor health (see Table 2). Interestingly though, the enrollment rate is 36% if only the mother reports poor health and 52% if only the father does it. The same pattern holds if we split the sample according to child age (i.e., if we look at secondary and tertiary education ages, separately).

In all OLS specifications we include the following standard controls: a child’s age, sex, ethnic group and (time variant) highest educational qualification; the mother’s and father’s age and the (time variant) highest educational qualification; a dummy variable for the household owning a farm; variables related to the household’s demographic structure; a set of indicators of household wealth (house ownership, logarithm of the number of rooms, availability of water, telephone and house connected to sewer); city of current residence and calendar year fixed effects. Possibly, other explanatory variables may be added to our specification, such as war-

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27 In the survey, there is no way to distinguish between natural parents and other parental figures, and for the purpose of the current analysis we refer to fathers and mothers as to the paternal and maternal figures present in the household at the time of the interview.
28 Results including a quadratic in parents’ ages, which accounts for potential nonlinearities of changes in health status, is reported in Table A1.
29 Proxies for household wealth are generally considered to be less noisy indicators of households’ permanent economic conditions than income variables.
30 We also estimated models including city by time fixed effects with no significant change in the results (see
exposure indicators so as to capture the long-term effect of the conflict on school enrollment.\textsuperscript{31} Yet, such variables are related to the city of residence before the war, hence they are time-invariant and perfectly absorbed by the individual fixed effects. For the same reasons, other time-invariant controls (e.g. birth order) are excluded. Parents’ attributes, such as age and education, are included as they are likely to be correlated with both their health and investments in their progeny; the child’s age and highest educational qualification achieved are included as the likelihood of school enrollment tends to decrease with both these variables; proxies of household wealth and demographic structure are included as they affect both the health status and the schooling level of household members;\textsuperscript{32} time and city fixed effects capture macroeconomic and local conditions (such as the local provision of health services), respectively. In some specifications, we also include the child’s and his/her siblings’ poor health status, and are mainly intended as robustness checks. In particular, after conditioning on child FE, these additional controls may capture time-variant household common health shocks. We exclude for the moment work-related variables, such as parents’ labour force status, working hours or salaries, as they are likely to be affected by parents’ health status. Hence, we will be estimating the overall effect of parents’ illness, including both pecuniary and non-pecuniary effects. Yet, as a robustness test, we will also present later on estimates with controls for parents’ salaries.\textsuperscript{33}

Table \textmd{3} reports sample summary statistics for the main dependent and independent variables used in the empirical analysis.

\textsuperscript{31}Swee (2009) for example studies the effect of the conflict on individual school attainment by using a set of war-destruction indicators at the level of the city of residence prior to the war.

\textsuperscript{32}We included the number of children, the number of sons and daughers in the age groups 0-6 and 7-15, and household size which conditional on the other controls captures the effect of other adults living in the household.

\textsuperscript{33}For the same reasons, we also exclude an indicator of health insurance.
6 Results

6.1 Results using parents’ self-reported health

In this subsection, we report the estimates obtained with OLS, random effects (RE), and fixed effects (FE) models. Table 4 illustrates the estimates for the child school enrollment equation on the full sample of 15–24 years old children.

Column (1) of Table 4 shows the estimates obtained with OLS, from which we can see that children in families in which only mothers have poor health status are 14.1 percentage points (p.p., hereafter) less likely to be enrolled in education than children of healthy parents. The probability of school enrollment of children with both parents in bad health conditions is also significant and negative but lower in magnitude (-7.2 p.p.). The effect of the father’s illness turns out to be much smaller in magnitude and statistically insignificant.

For the sake of completeness, although they are affected by the very same weaknesses as the OLS estimates, in column (2) we have reported the RE estimates, which show a reduction in the coefficient of the mother’s poor health. Column (3) reports our preferred specification. From the FE estimator we obtain that the mother’s poor health has a negative effect on child school enrollment of about −7 p.p., statistically significant at the 1% level. This is a sizeable effect given that the average school enrollment rate at ages 15–24 is 52 per cent. As it is expected from the combined effect of potential endogeneity and misclassification error, FE are lower than OLS estimates. It is hard to disentangle the separate effect of the two sources of bias, and FE estimates should be considered as lower-bound estimates. In the next section, though, we will explore in more depth the potential implications of misclassification on our estimates by using alternative—and more objective—indicators of parental health. On the other hand, when using panel estimators the effect of having both parents ill loses statistical significance with respect to OLS, suggesting that unobserved heterogeneity may be partly responsible for

\[ \text{PM} \]

\[ \text{PF} \]

\[ \text{PMF} \]

To check the sensitivity of the estimates to the model specification, we also estimated a random effects probit model. The marginal effect of \( \text{PM} \) (calculated at the sample mean) is -0.117 statistically significant at the 1% level. By contrast, the marginal effects of \( \text{PF} \) and \( \text{PMF} \) are much lower, at -0.028 and -0.025, respectively, and are not statistically significant at conventional levels. Thus, overall, the results are not driven by the assumption of linearity of the educational production function.
spurious correlation. A possible reading of this result is that while bad health shocks that hit only one parent are approximately random, the same is not true when these shocks hit simultaneously both parents, reflecting ‘positive assortative mating’ in terms of health status.\textsuperscript{35} Alternatively, when both parents report to be sick, this variable’s measurement error is likely to be larger and the FE estimates to be affected by a higher ‘attenuation bias’. We also estimated models including information on chronic diseases as additional control variables, but this did not change our FE results. Indeed, in those estimates parents’ permanent illnesses are likely to be captured by child fixed effects.

Columns (4)–(6), as we anticipated, report some robustness checks. Indeed, the FE estimator is not consistent when, for instance, time varying shocks to parental health are correlated with child’s health shocks (household common shocks), which are in turn correlated to child education. We already took the example of accidents involving the whole family, another one may be viral diseases. For this reason we have included the child’s and her/his siblings’ poor health status in the regression but the estimates of parental health effects remain unaffected.

Our finding of a stronger effect of mothers than of fathers is in line with most of the literature on parental absence. Chen et al. (2009) finds, for instance, that after conditioning on household fixed effects paternal deaths have a very small and statistically insignificant effect on children’s going to college. Case and Paxson (2001) show using US data that investments in child health are made, largely, by a child’s mother, and that step mothers are not substitutes for birth mothers in this domain. We find consistent evidence as far as investments in child education are concerned. Similar results are also found in the literature on migration, which is another form of ‘parental absence’. Cortes (2010) shows, for instance, that children of migrant mothers are more likely to lag behind in school compared to children of migrant fathers, supporting the fact that the mother’s absence has a stronger detrimental effect on child achievement than the father’s absence.

According to all of these studies, the asymmetric effects of mothers and fathers may be explained by the higher importance of time over pecuniary inputs into the production of child

\textsuperscript{35}This is to say that someone with bad health is likely to be married or partnered with someone with similarly bad health.
quality combined with the mother’s traditional role of child-rearer, which is complementary to
the child’s education. In our estimation sample, for instance, only 31% of mothers work for pay
(i.e., are employees, independent workers, or seasonal workers), compared to 73% of fathers
(see Table 3). Hence, a mother’s poor health condition in BiH could involve a reduction of
the quantity and/or quality of parenting time and have a more negative impact on children’s
outcomes than paternal illness, whose consequences may be mainly pecuniary in nature.\textsuperscript{36}

Several studies tend to stress the lower importance of current income as a determinant of human
capital, compared to other non-pecuniary, especially early, parental inputs (see Cameron and
Heckman 1998, Carneiro and Heckman 2002, among others).\textsuperscript{37}

Even though we do not have data on parents’ involvement in child education to directly test
the ‘intra-household time allocation hypothesis’ (our survey does not contain time diary data
or other information on household members’ time use), we can still provide some evidence
consistent with it.

In order to test whether pecuniary costs related to parental poor health (in particular each
parent’s earnings losses) play only a minor role for a child’s school enrollment, we run the same
regressions but including mother’s and father’s labour incomes as further control variables.
Even though they may be endogenous with respect to a child’s schooling (e.g., parents may
exhibit lower labour force participation if their children work), results in Table 5 suggest that
they are not the main mediating factor for the negative effect of parents’ illness on child school
enrollment. We consider two measures of salary available in all LSMS waves: the last paid
monthly salary or earning and the usual monthly net salary or earning (converted into hundreds
of ‘convertible marks’, KM).\textsuperscript{38} In both cases the coefficient of the mother’s poor health remains

\textsuperscript{36}In principle, poor health may also imply a larger amount of time spent with children for working parents, but
this is unlikely to be the case for Bosnian mothers, given their low level of labour force participation.

\textsuperscript{37}Blau (1999) reviews the literature studying the association between parental income and child development,
pointing out several difficulties involved in such an analysis (e.g. measurement of either permanent or current
parental income, confounding factors related to shared genes and environment vs. parental investment behav-
ior). By estimating the effect of parental income on children’s cognitive, social, and emotional development, this
study concludes that the income effect is small, especially in case of current income shocks. Family background
characteristics, instead, play a more important role than income in determining child outcomes.

\textsuperscript{38}Salaries were deflated using the GDP deflator and are expressed at 1996 value. As salaries are not available
for some working parents, we included a missing value dummy. Last salaries are missing for 4.3% of mother-time
observations and 10.6% of father-time observations, while usual salaries for 1.9% of mother-time observations
and 5.1% of father-time observations. We could not include health expenditures among the regressors as in the

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unaffected by the inclusion of parental salaries. Although the latter are not significant in the FE models, in OLS and RE models the child’s school enrollment seems to be more sensitive to the mother’s salary.\textsuperscript{39}

If household income loss is an important mediating factor for the effect of parents’ illness, we may expect children with older siblings to be less affected. Indeed, the burden of parental health shocks is likely to be borne mainly by older siblings, which may be the ones required to enter the labour market. To test this hypothesis we interacted all three dummies of parental health status with a dichotomous indicator for having either older brothers or older sisters (separate interactions for brothers and sisters produce small cell sizes) and in no case the interaction terms were statistically significant.\textsuperscript{40}

All of these pieces of evidence taken together suggest that the main causal pathway for the negative effect of the mother’s illness is likely to be non-pecuniary and related to a reduction of the quantity and/or the quality of parenting time.\textsuperscript{41}

It may argued, though, that results in this section reflect higher misclassification in self-assessment of health by fathers (men) than by mothers (women), hence producing a larger attenuation bias for the former. This may happen if, for instance, fathers are for any reason more likely to wrongly report poor health than mothers. In order to address this potential issue, we use the data from the World Health Survey (WHS) administered in BiH in 2002 by the World Health Organization (WHO). An important feature of these data is that it provides vignettes, which can be used to assess the existence and magnitude of a differential reporting bias by gender. The results of our analysis are reported in Appendix B, and show no evidence of a gender specific bias in self-reported health.

\textsuperscript{39}It is worth recalling that in FE models we are only exploiting transitory variations in parents’ current incomes. All specifications, though, do include wealth related variables among the controls.

\textsuperscript{40}Results available from the authors upon request.

\textsuperscript{41}We also estimated specifications including an indicator for having health insurance, but this did not change our results. We present in the text specifications excluding this control as it may capture reverse causality (e.g., healthier individuals are more likely to be employed and covered by a health insurance).
6.2 Results using alternative health indicators

The index of self-reported parental poor health status we used so far may be affected by a reporting bias. Indeed, reported health status may contain a measurement error due to differences in individual reference points, e.g., more optimistic individuals may systematically overstate their health status. Unfortunately, the LSMS does not provide objective measures of health (e.g., medical records). Yet, we can employ alternative subjective measures of poor parental health to check the robustness of our results. First, we use parents’ self-reported ability to physically perform the activities of daily living (ADLs, hereafter) as an alternative proxy of parental poor health. Similar health indicators have been used in the economic literature by Strauss et al. (1993), Gertler and Gruber (2002), and Morefield (2010), among others. ADLs indicators are often considered to be ‘more objective’ than self-reported health status and less likely to be affected by differences in individual response scales: as they represent answers to very specific questions in which the interviewer asks for the ability to perform certain daily activities, they may limit the likelihood that respondents rationalize their own behavior through their answers. In particular, ADLs indicators have the advantage of recording specific facts related to an individual’s daily living rather than her opinions on her physical wellbeing. These measures have been validated both in the US and in East Asian countries (Andrews et al. 1986, Guralnik et al. 1989, Ju and Jones 1989) among others. In 2003 and 2004 only, the BiH LSMS asked individuals the following questions:

42 (i) Has your health limited your ability to perform vigorous activities such as lifting heavy objects, running, or participating in strenuous sports? (ii) Has your health limited your walking uphill? (iii) Has your health prevented you from bending, lifting, or stooping? The possible answers are: ‘No’, ‘Yes, less than three months’ and ‘Yes, more than three months’. Codes 1, 2 and 3 are given to the first, second and third answers, respectively. The scores to the single questions can be added to obtain a single health indicator, which we label the ADLs score and which increases with the severity of the disability (see Gertler and Gruber 2002). The latter variable can then be included as a continuous indicator of

42 The English translation is ours as the original survey questions were asked in the local language.
parental health in the child schooling equation. The ADLs score can be also dichotomized to build an indicator of poor health status. As questions on ADLs were not asked in the 2002 wave, and in order not to restrict too much the number of parents with poor health, we fixed the threshold of the dichotomous variable at an arbitrary level of 6 (corresponding, for instance, to individuals not being able to perform all three activities for less than three months, or only two activities for more than three months).

Table 6 reports the estimates on the 2002–2003 sample. The FE estimator shows that a one standard deviation increase (about two points, see Table 3) in the mother’s ADLs score (meaning worse health) is associated with a 6.2 p.p. penalty in the likelihood of child school enrollment for children of healthy fathers. The effect turns out to be statistically significant only at the 10% level. Effects are more precisely estimated when we use the dichotomous version of the indicator. Children with mothers with poor health (ADLs score $\geq 6$) are about 9 p.p. less likely to be enrolled in school. The overall picture is very consistent with the results of the previous section, showing a stronger effect of mothers’ poor health. Columns (7)–(9) report the estimates using self-reported poor health status, which show that the different estimation sample with respect to the analysis in Table 4 is not determining remarkable differences in the estimated effects. Under the assumption that the direction of the endogeneity bias is the same irrespective of the proxies of poor health that we consider, the table also suggests that the ADLs score is much less prone to measurement error than self-reported health: indeed, switching from OLS to FE in columns (1) and (3) is not causing a drop in the estimated coefficients, contrary to when self-rated health is used, in columns (7) and (9).

While the proxy of poor health considered in the previous section encompasses both physical and mental health, the one based on the ADLs score refers to physical disabilities only. Hence, we turn to indicators of mental health provided by LSMS. In particular, waves 2003

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43In this case, as the ADLs score is continuous, we cannot include mutually exclusive indicators of the mother’s and father’s health, but include an interaction term in the regression. Gertler and Gruber (2002) use the following formula to compute a disability index, $\text{ADLs} = (\text{score} - \text{Min score})/(\text{Max score} - \text{Min score})$. The correlation between this index and the variable we use is 1, and, therefore, the estimated coefficients in the regressions are invariant to this transformation.

44We also replicated the analysis with cut points of 5 and 7 without relevant differences in the results (available upon request). As older parents are more likely to be affected by ADLs limitations, it is crucial to control for parents’ ages in the child schooling equation, which may also have direct effects on children’s attainments.
and 2004 include a battery of questions that can be used to compute depression scales. Despite being subjective, as they ask respondents about their internal states and associated behaviour, these scales have been validated in the psychological literature. In particular, the Center of Epidemiological Studies Depression (CES-D) Scale (Radloff 1977) was administered to LSMS respondents.\footnote{For more information see Do and Iyer (2009).} This scale has been subjected to a specific validation for Bosnia and Herzegovina (Kapetanovic 2009). In the current study, we use the following seven items that are present in both the 2003 and the 2004 waves: ‘(i) For the next few questions please look at Showcard C and tell me if during the last week you felt low in energy, slowed down? (ii) During the last week did you blame yourself for different things? (iii) During the last week did you have problems falling asleep or sleeping? (iv) During the last week did you feel hopeless in terms of the future? (v) During the last week did you feel melancholic? (vi) During the last week did you feel that you worried too much about different things? (vii) During the last week did you feel that everything was an effort?’ The possible answers in Showcard C are ‘Not at all’, ‘A little’, ‘Quite a bit’, and ‘Extremely often’, which are assigned scores of 0, 1, 2, and 3, respectively. Scores in single questions can be summed to obtain an aggregate score ranging between a minimum of 0 (no depression symptoms) and a maximum of 21 (very severe depression symptoms). Higher CES-D scores means worse mental health.

Table 7 reports the estimates of the child schooling equation including the mother’s and the father’s CES-D continuous scales along with their interaction terms. Results show that the mother’s mental health is more important than the father’s mental health in explaining children’s school enrollment, although Table 3 also suggests that depression is relatively more widespread among mothers than fathers. Raising by one standard deviation (3.265) the CES-D score of the mother is associated to an about 10 p.p. lower probability of child school enrollment when fathers show no symptoms of depression (i.e., their score is zero). Consistent effects are found using a dichotomous indicator of mental health. As an indicator of poor mental health status we adopted the threshold of 5.6, which was set by reporting on our 21-point (and 7-item) scale the 16 score threshold suggested by Radloff (1977) on a 60-points scale including 20
items. The results are shown in columns (4)–(6): the mother’s depression is associated with a 9.1 p.p. negative premium in the child’s school enrollment. Also in this case, we do not find any evidence of an attenuation bias in the FE estimates, which suggests that the CES-D scale score is probably measured more precisely than self-reported health. Estimates using self-reported poor health status in the same sample are reported in columns (7)–(9).

Overall, the findings in this section are robust to considering alternative, less subjective, indicators of parental health status and confirm a primary role of maternal health shocks in negatively affecting child school achievement.

7 Heterogeneous effects by child age and gender

Up to now, we have assumed homogeneous effects of parental health on children of different age and gender. Yet according to the literature, changes in the household’s conditions may differentially affect the ‘treatment effects’ of children according to their attributes (Becker 1981). This is so as the marginal productivities of monetary resources and parental time, which are stretched by health shocks, are likely to change according to child maturity and gender.

Child age may lead to heterogenous treatment effects as, for instance, older children are likely to be more (financially) independent from their parents and less sensitive to shocks hitting them. Furthermore, if they have been already screened in the educational system, parents who are aware of their level of ability could be less likely to withdraw them from school in response to adverse health events (see Sun and Yao 2010). Heterogenous effects may be also expected with respect to child gender. The latter may depend on the degree to which returns to schooling are different between boys and girls, and whether sons and daughters play different (economic) roles in the household—by taking care of ill parents, for instance. In addition, parents may have different preferences for the ‘quality’ (i.e. education) of female and male children based

46Human capital theory argues that the underlying costs and benefits of schooling may vary with gender for several reasons (Schultz 1988). Gendered labour markets may provide lower wages for educated females than males, whilst girls’ unpaid domestic labour is often a significant component of family production (Smock 1981, McMahon 1999). Moreover, marriage markets may take a girls’ future income out of her family and transfer the benefits of education to the husband’s family (Boserup 1995).
either on social and cultural norms or on the child rearing technology (Thomas 1994, Alderman and Gertler 1997). 47

Thus, exploring age and gender patterns in the effects of poor parental health may shed more light into the causal pathways hidden behind the stronger negative effect of mother’s health we found above. For this reason, we present in what follows the effect of parental health estimated from samples splitted by child’s age and gender. It must be noted though that due to the smaller samples’ sizes estimates are less precise, and generally not statistically different across samples.

Tables 8 and 9 investigate heterogenous effects using self-reported poor health status as it allows for larger sub-samples and hence more precise estimates. 48 Columns (1)–(3) of Table 8 show the estimates on the sample of 15–19 years old children. The OLS estimator gives significant and negative effects for households in which only the mother is in poor health and for those in which both parents are in poor health, of -14.5 and -8.5 p.p., respectively. However, when we switch to panel estimators (RE and FE models) the latter effect falls and loses statistical significance, and only children in households where the mother (only) is in poor health turn out to have a significant penalty (-9.3 p.p. in the FE model) in the probability of school enrollment. Columns (4)–(5), reporting OLS and RE estimates, show that apparently also older children are affected by the mother’s poor health status. However, the FE estimator indicates that this negative association is likely to reflect a spurious correlation, and disappears when we control for unobserved individual time-invariant characteristics, which in the FE estimator are allowed to be correlated with both child’s schooling and parental health. Under some specific assumptions, the estimates in columns (4)–(6) can also be considered as a sort of falsification check. Indeed, if older children’s schooling decisions are less affected by their parents’ deci-

47While there are differences in the allocation of household resources depending on the gender of the child, these differences even vary with the gender of the parent. This may reflect either the role of preferences or differences in the technology of child rearing, e.g., it may be efficient for mothers to spend more time with daughters and fathers with sons. Thus, if the mother (father) has a preference for daughters (sons), she (he) will invest marginal resources in sons (daughters); correspondingly, sons (daughters) will lose more from a mother’s (father’s) shock (see also Duflo 2003, Pitt et al. 2003).

48We also ran regressions using alternative indicators of parental health status such as the ADLs and CES-D scores. Due to the smaller sub-samples (as ADLs and CES-D scores are not available for the same waves), the estimates are less precise but are overall consistent with the results reported in this section.
sions or conditions (‘life-course hypothesis’, Shavit and Blossfeld 1993), finding an effect of parental health in the age group 20–24 as large as the one in the 15–19 age group could be considered as a symptom that we are catching a spurious correlation due to unobserved heterogeneity. This is not the only possible interpretation of course. For instance, older children in general have a higher earnings potential and therefore their human capital investment decisions may be more sensitive to parental health shocks if they mainly involve pecuniary costs to the household. However, as we already said, the fact that for both age groups the higher coefficient is attached to the mother’s poor health, which is also the one more often statistically significant, makes us propend for a greater relative importance of non-pecuniary parental inputs rather than pecuniary ones, since mothers are less likely to be working in the labour market and earn an income.

Table 9 reports the results of the estimates split by child gender. Columns (1)–(3) list the results for daughters and columns (4)–(6) for sons. For daughters, the coefficient estimates from RE and FE estimators are much lower than those obtained using OLS, and insignificant in the FE model, suggesting that unobserved heterogeneity is partly responsible for the negative association between parental health and their schooling. By contrast, for sons the estimates of OLS, RE and FE are much more stable and when using the FE estimator sons show an 8.4 p.p. lower probability of school enrollment, statistically significant at the 1% level.

Although, due to the small sample size, the two effects are not very precisely estimated and are not statistically different, this seems to suggest that according to the several intra-household allocation hypotheses, either the mother spends more time with daughters and a reduction in the quantity of her parenting time due to illness is especially harmful to sons (for whom the marginal productivity of time is higher) or, assuming that boys and girls are equally dependent on maternal time, boys have a higher opportunity cost of education when mothers are ill.49

These results are robust to including parental salaries (results are not reported here).

49We made an attempt to distinguish between the two hypotheses by estimating a regression using a child’s labour force participation (LFP) as the dependent variable. In the pooled sample by gender, the mother’s poor health is significantly (at the 10%) positively correlated with children’s LFP in the FE model. When we split the sample by child gender, the mother’s coefficient is very imprecisely estimated. However, for both sons and daughters the coefficients have a positive sign for the mother’s illness and a negative sign for the father’s illness.
Overall, findings in this section suggest that younger children and sons are likely to be more sensitive to maternal illness and pay the highest toll in terms of low education achievement. However, a thorough analysis of this heterogeneity would require larger samples.

8 Concluding remarks

A major parental illness is one of the most sizeable and least predictable shocks to household welfare, with potentially long-lasting consequences if investment in children is affected. Unlike the effect of parental death on children’s outcomes, the role of parental illness on investments in children’s human capital has been rarely investigated by the economic literature. Yet, lack of access to both health care and insurance mechanisms is increasingly perceived by policymakers as a crucial hurdle for household well-being and economic development.

We explore this issue by estimating the effects of parental illness on child current school enrollment, using a detailed longitudinal panel dataset from Bosnia and Herzegovina. The latter is a transition country where the 1992–1995 conflict left both health and schooling infrastructures in a very poor state and where the levels of educational and health achievements in the population are low compared to neighbouring countries.

Methodologically—to the best of our knowledge—this is the first paper which seeks to address the potential endogeneity of parental health status with respect to a child’s education using longitudinal data and child fixed effects. This identification strategy allows us to exploit sudden changes in parents’ health status, i.e. health shocks, which are less likely to be correlated with mother’s and father’s unobserved persistent traits (e.g., higher discount rates) also affecting their children’s education than parental health status. We further control for remaining household-level unobservable factors that vary from year to year and potentially affect both children’s education and parental health by including child’s own and his/her siblings’ health status as additional covariates.

Our findings show that, contrary to the common wisdom that shocks to the primary household earner bear more negative consequences for child education, it is especially maternal
health that makes a difference as far as child school enrollment is concerned. If the mother self-reports to be in poor health, our FE model suggests that her child is 7 p.p. less likely to be enrolled in education at ages of 15–24 years. The results are robust to considering other—more precise—measures of parental physical and mental health, such as limitations in activities of daily living and depression scales, which have been validated in the medical and psychological literature. We also find heterogeneous patterns of parental effects by child age and gender: younger children (aged 15–19) and sons seem to be more negatively affected by maternal illness.

By finding that the negative effects of parental health shocks are stronger when the mother is ill, our analysis supports the hypothesis that the maternal non-financial support to children is a key input for their school achievement. Furthermore, our findings point to important policy implications. Women’s access to health care services is likely to be particularly difficult in developing and transition countries (see, for instance, Oster 2009). For this reason, especially in those countries, the implementation of an adequate system of social protection, better prevention and improved women’s access to health care may contribute to greatly reducing the intergenerational cost of low levels of human capital.

Appendix. Differential self-report bias by gender?

We have already noted that the larger effect of self-reported health on child’s schooling for mothers may be determined by a larger measurement error in father’s self-reported health, which causes a more severe attenuation bias. Although in Section 6.2 we indirectly address this issue by considering less subjective measures of individual health, in the present Appendix we provide a more direct assessment of differences in report bias by gender.

As we have noted, the LSMS does not provide vignettes, however, the latter are available in the WHS administered in BiH in 2002 by the WHO. Vignettes are hypothetical situations in which individuals are asked to rate the health of a third person, whose health conditions are carefully described. An example is the following:
Anton does not exercise. He cannot climb stairs or do other physical activities because he is obese. He is able to carry the groceries and do some light household work.

Q2105 Overall in the last 30 days, how much of a problem did [name of person] have with moving around? 1. None 2. Mild 3. Moderate 4. Severe 5. Extreme/Cannot do

Q2106 In the last 30 days, how much difficulty did [name of person] have in vigorous activities, such as running 3 km (or equivalent) or cycling? 1. None 2. Mild 3. Moderate 4. Severe 5. Extreme/Cannot do.

In the questions the first name of the vignette’s person matches the respondent’s gender. The idea is that differences in individual responses completely stem from differences in reporting styles, as the true health of the persons described in the vignettes cannot depend on the respondents’ characteristics. In what follows, we present estimates referring to the twenty vignettes of Set-A (‘mobility and affect’) for which data are available for 262 individuals. The full set of vignettes is described in the WHS website.

We test gender differential in self-report bias in two ways. First, we estimate an ordered probit model where the cut points depend on individual observable characteristics, among which gender. The latent health index for the vignette’s person is:

\[ Y_{vi}^* = \theta_v + \epsilon_{vi} \]  \hspace{1cm} (8)

where the subscripts \(v\) and \(i\) stand for the vignette and the individual, respectively. \(\theta_v\)’s are vignettes’ specific fixed effects. The error term \(\epsilon_{vi}\) is assumed to be standard normally distributed. The observational rule for self-rated health is instead:

\[ Y_{vi} = j \text{ if } \tau_{i}^{j-1} < Y_{vi}^* \leq \tau_{i}^{j} \text{ for } j = 1 \ldots 5 \]  \hspace{1cm} (9)
where \( j = 1 \ldots 5 \) are the points of the Likert scale (none, mild, moderate, severe, extreme). The cut points are modelled as:

\[
\tau^j_i = a_j + \mathbf{x}'_i \boldsymbol{\gamma}.
\]

In the vector of controls affecting report bias (\( \mathbf{x}_i \)) we include an individual’s gender, age and years of completed education. In this model, individual characteristics of the respondents produce the same shift in all cut points. Given the linearity of the cut points, this model can be estimated using Maximum Likelihood by simply including in an ordered probit specification the additional variables affecting the thresholds. The results are reported in column (1) of Table B1 and show that gender does not affect report bias. The only statistically significant variables turn out to be the vignettes’ fixed effects (we do not report the coefficients but the Wald test for their joint exclusion).

Second, we estimated a model allowing for self-report bias to affect the last two cut points only, which are the most important for reporting ‘poor health’ in our analysis based on the LSMS. In this case, we modelled the cut points following Pradhan and van Soest (1995):

\[
\begin{align*}
\tau^j_i & = a_j & \text{for } j = 1,2 \\
\tau^j_i & = \tau^{j-1}_i + \exp(\mathbf{x}'_i \boldsymbol{\gamma}^j) & \text{for } j = 3, 4.
\end{align*}
\]

This model allows for individual characteristics to have different effects on the last two cut points. The results are reported in columns (2)-(3) of Table B1, and also in this case there is no evidence of gender differences.

We also tried with a model allowing for a greater flexibility, by making all cut points depend on individual characteristics as in Kapteyn et al. (2007), but the Maximum Likelihood estimation did not achieve convergence.

Overall, all these additional checks suggest the absence of a differential self-report bias by the respondent’s gender.
References


at the Council of Ministers of BiH, the Ministry of Civil Affairs of BiH, and the Project for Support and Development of Youth Structures in BiH of the German Technical Cooperation Society, GTZ.


Table 1: Sample selection criteria

<table>
<thead>
<tr>
<th>sample selection criteria</th>
<th>dropped</th>
<th>sample size</th>
</tr>
</thead>
<tbody>
<tr>
<td>children</td>
<td>7847</td>
<td>4579</td>
</tr>
<tr>
<td>age $\geq 15$ &amp; age $\leq 24$</td>
<td>794</td>
<td>3785</td>
</tr>
<tr>
<td>cohabiting with mother and father</td>
<td>1024</td>
<td>2761</td>
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<tr>
<td>self-reported health status asked in the panel wave (2002-2004)</td>
<td>242</td>
<td>2519</td>
</tr>
<tr>
<td>parental self-reported health status non-missing</td>
<td>305</td>
<td>2214</td>
</tr>
<tr>
<td>all regressors non-missing</td>
<td>154</td>
<td>2060</td>
</tr>
</tbody>
</table>

Note. The table shows the initial sample size (number of observations) and the observations lost applying our sample selection criteria.

Table 2: Children’s school attendance by parents’ health status

<table>
<thead>
<tr>
<th></th>
<th>No parent ill</th>
<th>Mother ill only</th>
<th>Father ill only</th>
<th>Both parents ill</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Full sample</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.57</td>
<td>0.36</td>
<td>0.52</td>
<td>0.37</td>
<td>0.52</td>
</tr>
<tr>
<td>(0.50)</td>
<td>(0.48)</td>
<td>(0.50)</td>
<td>(0.49)</td>
<td>(0.50)</td>
<td></td>
</tr>
<tr>
<td>15-19 age group</td>
<td>0.76</td>
<td>0.60</td>
<td>0.74</td>
<td>0.63</td>
<td>0.73</td>
</tr>
<tr>
<td>(0.43)</td>
<td>(0.50)</td>
<td>(0.44)</td>
<td>(0.49)</td>
<td>(0.44)</td>
<td></td>
</tr>
<tr>
<td>20-24 age group</td>
<td>0.52</td>
<td>0.31</td>
<td>0.48</td>
<td>0.34</td>
<td>0.48</td>
</tr>
<tr>
<td>(0.50)</td>
<td>(0.46)</td>
<td>(0.50)</td>
<td>(0.47)</td>
<td>(0.50)</td>
<td></td>
</tr>
</tbody>
</table>

Note. Standard deviations in parentheses. The sample includes 785 individuals aged 15-24 and 2060 observations.
Table 3: Sample summary statistics

<table>
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<th>Variable</th>
<th>n. obs.</th>
<th>mean</th>
<th>SD</th>
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</thead>
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<tr>
<td>child enrolled in education</td>
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<td>0.522</td>
<td>0.500</td>
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<td>2060</td>
<td>0.099</td>
<td>0.298</td>
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<tr>
<td>father only with poor health (PF)</td>
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<td>0.301</td>
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<td>both parents with poor health (PMF)</td>
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<td>poor health child</td>
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<td>poor health siblings</td>
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<td>age</td>
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<td>19.564</td>
<td>2.597</td>
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<td>male</td>
<td>2060</td>
<td>0.553</td>
<td>0.497</td>
</tr>
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<td>ethnic group (Bosniak)</td>
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<td>0.402</td>
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<td>other</td>
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<td>0.152</td>
</tr>
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<td></td>
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<tr>
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<td>2060</td>
<td>0.535</td>
<td>0.499</td>
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<tr>
<td>tertiary</td>
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<td></td>
<td></td>
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<tr>
<td>secondary</td>
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<td>0.242</td>
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<tr>
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<td>availability of water</td>
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<td>35.124</td>
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</tbody>
</table>

Continued on next page
Table 3 – continued from previous page

<table>
<thead>
<tr>
<th>Variable</th>
<th>n. obs.</th>
<th>mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>CES-D mother &gt; 5.6(^{(c)})</td>
<td>940</td>
<td>0.180</td>
<td>0.384</td>
</tr>
<tr>
<td>CES-D father &gt; 5.6(^{(c)})</td>
<td>940</td>
<td>0.052</td>
<td>0.222</td>
</tr>
<tr>
<td>CES-D both parents &gt; 5.6(^{(c)})</td>
<td>940</td>
<td>0.318</td>
<td>0.466</td>
</tr>
</tbody>
</table>

\(^{(a)}\) Means and standard deviations refer only to the samples with positive salaries. Salaries are expressed in hundreds of convertible marks (KM) at the 1996 value.

\(^{(b)}\) Summary statistics refer to the estimation sample used in Table 6.

\(^{(c)}\) Summary statistics refer to the estimation sample used in Table 7.

Note. Summary statistics are reported for the estimation sample in Table 4. Reference categories for categorical variables are shown in parenthesis and the other categories in italics.
Table 4: Effect parental self-reported poor health on child school enrollment

<table>
<thead>
<tr>
<th></th>
<th>OLS</th>
<th>RE</th>
<th>FE</th>
<th>OLS</th>
<th>RE</th>
<th>FE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(1)</td>
<td>(2)</td>
<td>(3)</td>
<td>(4)</td>
<td>(5)</td>
<td>(6)</td>
</tr>
<tr>
<td>mother only with poor health</td>
<td>-0.141***</td>
<td>-0.092***</td>
<td>-0.069***</td>
<td>-0.140***</td>
<td>-0.091***</td>
<td>-0.068**</td>
</tr>
<tr>
<td></td>
<td>(0.032)</td>
<td>(0.024)</td>
<td>(0.027)</td>
<td>(0.032)</td>
<td>(0.024)</td>
<td>(0.026)</td>
</tr>
<tr>
<td>father only with poor health</td>
<td>-0.053</td>
<td>-0.031</td>
<td>-0.017</td>
<td>-0.053</td>
<td>-0.031</td>
<td>-0.016</td>
</tr>
<tr>
<td></td>
<td>(0.033)</td>
<td>(0.024)</td>
<td>(0.025)</td>
<td>(0.033)</td>
<td>(0.024)</td>
<td>(0.025)</td>
</tr>
<tr>
<td>both parents with poor health</td>
<td>-0.072**</td>
<td>-0.020</td>
<td>0.001</td>
<td>-0.070*</td>
<td>-0.018</td>
<td>0.003</td>
</tr>
<tr>
<td></td>
<td>(0.036)</td>
<td>(0.025)</td>
<td>(0.026)</td>
<td>(0.036)</td>
<td>(0.025)</td>
<td>(0.026)</td>
</tr>
<tr>
<td>child with poor health</td>
<td>0.012</td>
<td>-0.026</td>
<td>-0.048</td>
<td>0.012</td>
<td>-0.026</td>
<td>-0.048</td>
</tr>
<tr>
<td></td>
<td>(0.048)</td>
<td>(0.040)</td>
<td>(0.042)</td>
<td>(0.048)</td>
<td>(0.040)</td>
<td>(0.042)</td>
</tr>
<tr>
<td>at least one sibling with poor health</td>
<td>-0.059</td>
<td>-0.022</td>
<td>-0.008</td>
<td>-0.059</td>
<td>-0.022</td>
<td>-0.008</td>
</tr>
<tr>
<td></td>
<td>(0.068)</td>
<td>(0.038)</td>
<td>(0.041)</td>
<td>(0.068)</td>
<td>(0.038)</td>
<td>(0.041)</td>
</tr>
</tbody>
</table>

N. observations 2060 2060 2060 2060 2060 2060
N. individuals 785 785 785 785 785 785

*, **, *** statistically significant at the 10%, 5% and 1% level, respectively.

Note. The dependent variable is a dichotomous indicator for being enrolled in education. The samples include individuals aged 15-24 in the Bosnian LSMS (2002-2004) cohabiting with both their parents. The table reports OLS, child random effects (RE) and child fixed effects (FE) estimates of the effect of parents’ self-reported poor health on the probability of child school enrollment using a linear probability model. All models also control for the variables listed in Section 5. Heteroskedasticity-robust standard errors in parentheses. OLS standard errors are clustered by child.
Table 5: Effect of parental self-reported poor health on child school enrollment controlling for parents’ salaries

<table>
<thead>
<tr>
<th></th>
<th>OLS (1)</th>
<th>RE (2)</th>
<th>FE (3)</th>
<th>OLS (4)</th>
<th>RE (5)</th>
<th>FE (6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>mother only with poor health</td>
<td>-0.134***</td>
<td>-0.088***</td>
<td>-0.065**</td>
<td>-0.134***</td>
<td>-0.089***</td>
<td>-0.069***</td>
</tr>
<tr>
<td></td>
<td>(0.032)</td>
<td>(0.024)</td>
<td>(0.027)</td>
<td>(0.032)</td>
<td>(0.024)</td>
<td>(0.027)</td>
</tr>
<tr>
<td>father only with poor health</td>
<td>-0.047</td>
<td>-0.029</td>
<td>-0.015</td>
<td>-0.044</td>
<td>-0.027</td>
<td>-0.014</td>
</tr>
<tr>
<td></td>
<td>(0.033)</td>
<td>(0.024)</td>
<td>(0.025)</td>
<td>(0.033)</td>
<td>(0.024)</td>
<td>(0.025)</td>
</tr>
<tr>
<td>both parents with poor health</td>
<td>-0.064*</td>
<td>-0.014</td>
<td>0.004</td>
<td>-0.059</td>
<td>-0.013</td>
<td>0.004</td>
</tr>
<tr>
<td></td>
<td>(0.036)</td>
<td>(0.025)</td>
<td>(0.026)</td>
<td>(0.036)</td>
<td>(0.025)</td>
<td>(0.026)</td>
</tr>
<tr>
<td>last monthly salary mother</td>
<td>0.026**</td>
<td>0.016</td>
<td>0.006</td>
<td>0.033**</td>
<td>0.036**</td>
<td>0.035</td>
</tr>
<tr>
<td></td>
<td>(0.013)</td>
<td>(0.014)</td>
<td>(0.017)</td>
<td>(0.015)</td>
<td>(0.016)</td>
<td>(0.024)</td>
</tr>
<tr>
<td>last monthly salary father</td>
<td>0.004</td>
<td>0.003</td>
<td>0.001</td>
<td>0.011*</td>
<td>0.007</td>
<td>0.003</td>
</tr>
<tr>
<td></td>
<td>(0.005)</td>
<td>(0.003)</td>
<td>(0.004)</td>
<td>(0.006)</td>
<td>(0.005)</td>
<td>(0.005)</td>
</tr>
<tr>
<td>usual net monthly salary mother</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>usual net monthly salary father</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N. observations</td>
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<td>2060</td>
<td>2060</td>
<td>2060</td>
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<td>785</td>
<td>785</td>
<td>785</td>
<td>785</td>
</tr>
</tbody>
</table>

*, **, *** statistically significant at the 10%, 5% and 1% level, respectively.

Note. The dependent variable is a dichotomous indicator for being enrolled in education. The samples include individuals aged 15-24 in the Bosnian LSMS (2002-2004) cohabiting with both their parents. The table reports OLS, child random effects (RE) and child fixed effects (FE) estimates of the effect of parents’ self-reported poor health on the probability of child school enrollment using a linear probability model. All models also control for the variables listed in section 5. Heteroskedasticity-robust standard errors in parentheses. OLS standard errors are clustered by child.
Table 6: Alternative health measures: limitations in activities of daily living (ADLs)

<table>
<thead>
<tr>
<th></th>
<th>ADLs score (continuous)</th>
<th></th>
<th>ADLs score (dichotomous)</th>
<th></th>
<th>poor health status</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OLS (1)</td>
<td>RE (2)</td>
<td>FE (3)</td>
<td>OLS (4)</td>
<td>RE (5)</td>
<td>FE (6)</td>
</tr>
<tr>
<td>ADLs score mother</td>
<td>-0.020</td>
<td>-0.028*</td>
<td>-0.032*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.019)</td>
<td>(0.015)</td>
<td>(0.017)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ADLs score father</td>
<td>-0.021</td>
<td>-0.021</td>
<td>-0.013</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.020)</td>
<td>(0.016)</td>
<td>(0.018)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ADLs score mother × ADLs score father</td>
<td>0.004</td>
<td>0.005*</td>
<td>0.004</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.003)</td>
<td>(0.003)</td>
<td>(0.003)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ADLs score mother ≥ 6</td>
<td></td>
<td></td>
<td></td>
<td>-0.046</td>
<td>-0.068*</td>
<td>-0.091**</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(0.056)</td>
<td>(0.039)</td>
<td>(0.042)</td>
</tr>
<tr>
<td>ADLs score father ≥ 6</td>
<td></td>
<td></td>
<td></td>
<td>-0.031</td>
<td>0.008</td>
<td>0.046</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(0.052)</td>
<td>(0.041)</td>
<td>(0.045)</td>
</tr>
<tr>
<td>ADLs score both parents ≥ 6</td>
<td>0.010</td>
<td>-0.012</td>
<td>-0.025</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.005)</td>
<td>(0.003)</td>
<td>(0.003)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mother only with poor health</td>
<td></td>
<td></td>
<td></td>
<td>-0.161***</td>
<td>-0.127***</td>
<td>-0.092**</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(0.047)</td>
<td>(0.041)</td>
<td>(0.047)</td>
</tr>
<tr>
<td>father only with poor health</td>
<td></td>
<td></td>
<td></td>
<td>-0.119**</td>
<td>-0.098**</td>
<td>-0.053</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(0.052)</td>
<td>(0.042)</td>
<td>(0.047)</td>
</tr>
<tr>
<td>both parents with poor health</td>
<td></td>
<td></td>
<td></td>
<td>-0.081</td>
<td>-0.070</td>
<td>-0.044</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(0.081)</td>
<td>(0.044)</td>
<td>(0.052)</td>
</tr>
<tr>
<td>N. observations</td>
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<td>946</td>
<td>946</td>
<td>946</td>
<td>946</td>
<td>946</td>
</tr>
<tr>
<td>N. individuals</td>
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<td>473</td>
<td>473</td>
<td>473</td>
<td>473</td>
<td>473</td>
</tr>
</tbody>
</table>

*, **, *** statistically significant at the 10%, 5% and 1% level, respectively.

Note. The dependent variable is a dichotomous indicator for being enrolled in education. The estimation samples include individuals aged 15-24 in the Bosnian LSMS (2003 and 2004) cohabiting with both their parents. The table reports OLS, child random effects (RE) and child fixed effects (FE) estimates of the effect of parents’ having reported limitations in ADLs (both continuous and dichotomized) on the probability of child school enrollment using a linear probability model. The continuous ADLs score ranges between 3 (no limitation) and 9 (all three limitations listed in section 6.2 for more than 3 months). All models also control for the variables listed in Section 5. Heteroskedasticity-robust standard errors in parentheses. OLS standard errors are clustered by child.
Table 7: Alternative health measures: CES-D depression scale

<table>
<thead>
<tr>
<th></th>
<th>mental health (continuous)</th>
<th>mental health (dichotomous)</th>
<th>poor health status</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OLS (1)</td>
<td>RE (2)</td>
<td>FE (3)</td>
</tr>
<tr>
<td>CES-D mother</td>
<td>-0.018**</td>
<td>-0.025***</td>
<td>-0.030***</td>
</tr>
<tr>
<td>CES-D father</td>
<td>0.007</td>
<td>-0.002</td>
<td>-0.002</td>
</tr>
<tr>
<td>CES-D mother × CES-D father</td>
<td>0.000</td>
<td>0.002*</td>
<td>0.003**</td>
</tr>
<tr>
<td>CES-D mother &gt; 5.6</td>
<td>-0.067*</td>
<td>-0.085**</td>
<td>-0.091**</td>
</tr>
<tr>
<td>CES-D father &gt; 5.6</td>
<td>-0.030</td>
<td>-0.027</td>
<td>0.017</td>
</tr>
<tr>
<td>CES-D both parents &gt; 5.6</td>
<td>-0.050</td>
<td>-0.026</td>
<td>0.008</td>
</tr>
<tr>
<td>mother only with poor health</td>
<td>-0.161***</td>
<td>-0.127***</td>
<td>-0.092**</td>
</tr>
<tr>
<td>father only with poor health</td>
<td>-0.121**</td>
<td>-0.098**</td>
<td>-0.053</td>
</tr>
<tr>
<td>both parents with poor health</td>
<td>-0.081</td>
<td>-0.070</td>
<td>-0.043</td>
</tr>
<tr>
<td>N. observations</td>
<td>940</td>
<td>940</td>
<td>940</td>
</tr>
<tr>
<td>N. individuals</td>
<td>470</td>
<td>470</td>
<td>470</td>
</tr>
</tbody>
</table>

*, **, *** statistically significant at the 10%, 5% and 1% level, respectively.

Note. The dependent variable is a dichotomous indicator for being enrolled in education. The estimation samples include individuals aged 15-24 in the Bosnian LSMS (2003 and 2004) cohabiting with both their parents. The table reports OLS, child random effects (RE) and child fixed effects (FE) estimates of the effect of parents’ Center of Epidemiological Studies Depression (CES-D) scale on the probability of child school enrollment using a linear probability model. The CES-D scale we consider ranges between 0 (no depression symptoms) and 21 (maximum depression symptoms). All models also control for the variables listed in Section 5. Heteroskedasticity-robust standard errors in parentheses. OLS standard errors are clustered by child.
Table 8: Effect of parental health on child school enrollment by child age

<table>
<thead>
<tr>
<th></th>
<th>age 15-19</th>
<th>age 20-24</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OLS (1)</td>
<td>RE (2)</td>
</tr>
<tr>
<td>mother only with poor health</td>
<td>-0.145*** (0.043)</td>
<td>-0.125*** (0.039)</td>
</tr>
<tr>
<td>father only with poor health</td>
<td>-0.018 (0.036)</td>
<td>-0.028 (0.032)</td>
</tr>
<tr>
<td>both parents with poor health</td>
<td>-0.085* (0.048)</td>
<td>-0.055 (0.041)</td>
</tr>
<tr>
<td>N. observations</td>
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<td>977</td>
</tr>
<tr>
<td>N. individuals</td>
<td>369</td>
<td>369</td>
</tr>
</tbody>
</table>

*, **, *** statistically significant at the 10%, 5% and 1% level, respectively.

Note. The dependent variable is a dichotomous indicator for being enrolled in education. The estimation samples include individuals aged 15-19 and 20-24, respectively, in the Bosnian LSMS (2002-2004) cohabiting with both their parents. The table reports OLS, child random effects (RE) and child fixed effects (FE) estimates of the effect of parents’ self-reported poor health on the probability of child school enrollment using a linear probability model. All models also control for the variables listed in section 5. Heteroskedasticity-robust standard errors in parentheses. OLS standard errors are clustered by child. The sum of the two subsample sizes is different from the one reported in Table 4 since there are some individuals who turn 20 and change subsample during the 2002-2004 period, and they are dropped from the subsamples if they have less than 2 time observations.
Table 9: Effect of parental health on child school enrollment by child gender

<table>
<thead>
<tr>
<th></th>
<th>daughters</th>
<th></th>
<th></th>
<th>sons</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OLS (1)</td>
<td>RE (2)</td>
<td>FE (3)</td>
<td>OLS (4)</td>
<td>RE (5)</td>
<td>FE (6)</td>
<td></td>
</tr>
<tr>
<td>mother only with poor health</td>
<td>-0.139***</td>
<td>-0.072**</td>
<td>-0.040</td>
<td>-0.097**</td>
<td>-0.088***</td>
<td>-0.084**</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.052)</td>
<td>(0.035)</td>
<td>(0.037)</td>
<td>(0.038)</td>
<td>(0.034)</td>
<td>(0.041)</td>
<td></td>
</tr>
<tr>
<td>father only with poor health</td>
<td>-0.067</td>
<td>-0.049</td>
<td>-0.039</td>
<td>-0.000</td>
<td>-0.007</td>
<td>0.010</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.046)</td>
<td>(0.031)</td>
<td>(0.033)</td>
<td>(0.044)</td>
<td>(0.035)</td>
<td>(0.038)</td>
<td></td>
</tr>
<tr>
<td>both parents with poor health</td>
<td>0.053</td>
<td>0.066</td>
<td>0.062</td>
<td>-0.122***</td>
<td>-0.065**</td>
<td>-0.026</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.059)</td>
<td>(0.044)</td>
<td>(0.047)</td>
<td>(0.039)</td>
<td>(0.030)</td>
<td>(0.033)</td>
<td></td>
</tr>
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</tr>
<tr>
<td>N. individuals</td>
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<td>352</td>
<td>433</td>
<td>433</td>
<td>433</td>
<td></td>
</tr>
</tbody>
</table>

*, **, *** statistically significant at the 10%, 5% and 1% level, respectively.

Note. The dependent variable is a dichotomous indicator for being enrolled in education. The estimation samples include individuals aged 15-24 in the Bosnian LSMS (2002-2004) cohabiting with both their parents. The table reports OLS, child random effects (RE) and child fixed effects (FE) estimates of the effect of parents’ self-reported poor health on the probability of child school enrollment using a linear probability model. All models also control for the variables listed in Section 5. Heteroskedasticity-robust standard errors in parentheses. OLS standard errors are clustered by child.

Table A1. Robustness checks: Effect of parents’ self-reported poor health on child school enrollment

<table>
<thead>
<tr>
<th></th>
<th>FE (1)</th>
<th>FE (2)</th>
<th>FE (3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>mother only with poor health</td>
<td>-0.079***</td>
<td>-0.074***</td>
<td>-0.077***</td>
</tr>
<tr>
<td></td>
<td>(0.028)</td>
<td>(0.027)</td>
<td>(0.027)</td>
</tr>
<tr>
<td>father only with poor health</td>
<td>-0.020</td>
<td>-0.022</td>
<td>-0.025</td>
</tr>
<tr>
<td></td>
<td>(0.025)</td>
<td>(0.025)</td>
<td>(0.025)</td>
</tr>
<tr>
<td>both parents with poor health</td>
<td>0.011</td>
<td>0.006</td>
<td>-0.002</td>
</tr>
<tr>
<td></td>
<td>(0.026)</td>
<td>(0.026)</td>
<td>(0.026)</td>
</tr>
<tr>
<td>city by year FE</td>
<td>yes</td>
<td>no</td>
<td>no</td>
</tr>
<tr>
<td>quadratic in parents’ ages</td>
<td>no</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>lagged school enrollment status</td>
<td>no</td>
<td>no</td>
<td>yes</td>
</tr>
<tr>
<td>N. observations</td>
<td>2060</td>
<td>2060</td>
<td>2042</td>
</tr>
<tr>
<td>N. individuals</td>
<td>785</td>
<td>785</td>
<td>785</td>
</tr>
</tbody>
</table>

*, **, *** statistically significant at the 10%, 5% and 1% level, respectively.

Note. The dependent variable is a dichotomous indicator for being enrolled in education. The estimation samples include individuals aged 15-24 in the Bosnian LSMS (2002-2004) cohabiting with both their parents. The table reports child fixed effects (FE) estimates of the effect of parents’ self-reported poor health on the probability of child school enrollment using a linear probability model. In column (3) we loose 18 observations as past student status is missing. All models also control for the variables listed in Section 5. Heteroskedasticity-robust standard errors in parentheses.
Table B1. Evidence on reporting bias in self-reported health

<table>
<thead>
<tr>
<th></th>
<th>Ordered probit</th>
<th>Flexible-thresholds ordered probit</th>
<th>Flexible-thresholds ordered probit</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>cut point 3</td>
<td>cut point 4</td>
</tr>
<tr>
<td>female</td>
<td>0.0107 (0.054)</td>
<td>-0.0217 (0.0717)</td>
<td>0.0372 (0.0599)</td>
</tr>
<tr>
<td>age</td>
<td>-0.0002 (0.0018)</td>
<td>0.0026 (0.0026)</td>
<td>-0.0008 (0.0022)</td>
</tr>
<tr>
<td>education</td>
<td>-0.0006 (0.0069)</td>
<td>0.0078 (0.0116)</td>
<td>-0.0155 (0.0105)</td>
</tr>
<tr>
<td>constant</td>
<td>-0.3136* (0.1904)</td>
<td></td>
<td>0.4297** (0.1695)</td>
</tr>
<tr>
<td>cut point 1</td>
<td>-2.3584*** (0.1663)</td>
<td></td>
<td>-2.3469*** (0.0894)</td>
</tr>
<tr>
<td>cut point 2</td>
<td>-1.3841*** (0.1467)</td>
<td></td>
<td>-1.3730*** (0.0523)</td>
</tr>
<tr>
<td>cut point 3</td>
<td>-0.5036*** (0.1490)</td>
<td></td>
<td>(see above)</td>
</tr>
<tr>
<td>cut point 4</td>
<td>0.8000*** (0.1500)</td>
<td></td>
<td>(see above)</td>
</tr>
<tr>
<td>Log likelihood</td>
<td>-4830</td>
<td>-4823</td>
<td></td>
</tr>
<tr>
<td>Wald test $\theta'$s ($\chi^2(19)$)</td>
<td>1107.78 [0.00]</td>
<td>1294.59 [0.00]</td>
<td></td>
</tr>
<tr>
<td>Wald test female = 0</td>
<td>0.04 [0.84]</td>
<td>0.38 [0.83]</td>
<td></td>
</tr>
<tr>
<td>N. observations</td>
<td>5238</td>
<td>5238</td>
<td></td>
</tr>
<tr>
<td>N. individuals</td>
<td>262</td>
<td>262</td>
<td></td>
</tr>
</tbody>
</table>

*, **, *** statistically significant at the 10%, 5% and 1% level, respectively.

Note. The dependent variable is an ordered categorical variable (1. None; 2. Mild; 3. Moderate; 4. Severe; 5. Extreme) for vignettes for health state descriptions in Set-A (‘Mobility and affect’) of the Bosnia-Herzegovina’s WHS (WHO). Coefficient estimates are reported in the table. Heteroskedasticity-robust standard errors clustered by individual are reported in parentheses and p-values in brackets. For two observations vignettes’ responses are missing.