Parental Health Shocks and Child Health in Bangladesh

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Abstract

I study the effect of parental illness on child health in rural Bangladesh. Using a set of health conditions that I argue are as good as random, I find that parental illness has a significant negative effect on child height. Removing the effects of parental illness would close 3.5% of the gap in height between Bangladeshi children and the global average. Fathers' and mothers' illnesses have equally detrimental effects and I find a comparable effect for children in joint families, suggesting that intra-household safety nets are ineffective in protecting children against parental illness. Finally, I explore three potential mechanisms through which parental illness may affect child health: parental resource allocation, early life stress, and parents' fertility choice.

(*JEL* D13, I12, I15, I25, J13, O12, O15)

Keywords: human capital, height, weight, health shocks, parental investments, developing countries, Bangladesh

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1. Introduction

There are various reasons why parental illness may affect the health of their children. First, parental illness could be financially costly because of increased medical spending and decreased labor supply and productivity. This may force parents to lower resource allocation toward children, e.g., by reducing food and medical expenditure. Second, parental illness may directly affect children's health through living in a stressful environment (Aaskoven, Kjær, and Gyrd-Hansen 2022; Mühlenweg, Westermaier, and Morefield 2016). For my outcome of interest, which is child height, medical research suggests that early life stress leads to stunting due to activation of the hypothalamic-pituitary-adrenal (HPA) axis and inhibition of pituitary growth hormone (GH) release (Denholm, Power, and Li 2013; Hulanicka, Gronkiewicz, and Koniarek 2001; Li, Manor, and Power 2004; Montgomery, Bartley, and Wilkinson 1997; Chrousos and Gold 1992; Pears and Fisher 2005). Finally, in response to illness and financial distress, parents may decide to have fewer children and allocate more resources to the existing children – a quality-quantity trade-off.

In this study, I measure how parental illness affects child health in Bangladesh. Specifically, I investigate the impact of major illnesses of parents on under-five children's height. Child malnutrition and stunting (i.e., severe growth deprivation) are major concerns for Bangladesh. For example, in 2019, 28 percent of children under five were two standard deviations below the World Health Organization (WHO) growth standards (World Bank 2022). Improving child nutrition and growth can significantly improve child survival, cognitive development, and future earnings (Almond, Currie, and Duque 2018; Case, Fertig, and Paxson 2005; Currie and Vogl 2013; Smith 2009; Steckel 1995).

The treatment variable in this study is major parental (i.e., father or mother, or both) illness, which is defined as the limitation in activities of daily living (ADLs). More specifically, I create an indicator variable "ADL limitation" if parents have at least some difficulties in walking, sitting, or carrying weight. ADL limitation is a reliable indicator of long-term health status and reflects unpredictable major illnesses (Bratti & Mendola, 2014; Crespo & Mira, 2014; Genoni, 2012; Gertler & Gruber, 2002).

I start with a pool of healthy parents (i.e., no ADL limitation) at the baseline in 2012. Parents who developed ADL limitations between 2013 and 2015 form the treatment group, and parents who remained healthy form the control group. About 25 percent of the sample is in the treatment group, and 75 percent is in the control group.

I measure child height based on the WHO growth standard height-for-age (HFA) z-score. HFA z-score quantifies how under-five children should grow under optimum conditions with ideal infant feeding and child health practices. In addition, HFA z-score does not considerably respond to recent dietary intake and therefore reflects long-term nutrition deficiency in a population.

A key finding motivating my identification strategy is that, at the baseline, the distribution of child height is not statistically different between treated and control groups. The average HFA z-scores of treatment and control group children in 2012 are -1.37 and -1.46, respectively, and a Kolmogorov-Smirnov (K-S) test fails to reject that the full distributions of HFA z-scores in 2012 are the same. I also show that the baseline covariates between the treatment and control groups are quite similar. Moreover, I find no significant difference in the likelihood of other household members developing an ADL limitation between treatment and control groups, which indicates that there are no household level confounders (i.e., treated households are not living in conditions that are more susceptible to injury and illness). As a result, for a confounder to cause bias in my estimates, it would need to be something (i) unobserved, (ii) specific to only one household member, and (iii) which does not affect children's height until after the ADL limitation is realized. It is difficult to imagine what such a confounder might be.

This finding suggests that the treatment assignment (i.e., parental illness) is minimally confounded or as good as random. However, to help address any remaining endogeneity, I control for family characteristics using doubly robust estimation (Bang & Robins, 2005; Imbens & Wooldridge, 2009; Robins et al., 1994; Wooldridge, 2007, 2010). I obtain very similar results with and without controls, or using alternative estimators such as propensity score matching (PSM), multivariate distance matching (MDM), and ordinary least squares (OLS).

The result shows that parental illness leads to 0.18 standard deviations (SDs) lower child height. This effect size is comparable to children experiencing relatively large shocks, e.g., crop failure (0.17 SDs) and drought (0.21 SDs) in Ethiopia (Akresh, Verwimp, and Bundervoet 2011; Hirvonen, Sohnesen, and Bundervoet 2020). Removing the effects of parental illness would close 3.5% of the gap in height between Bangladeshi children and the global average. I find fathers' and mothers' illnesses have equally detrimental effects on child height. I also find

a comparable effect for children in joint families,[1] suggesting that intra-household safety nets are ineffective in protecting children against parental illness. In addition, I do not find heterogeneous effects by child age, sex, or birth order.

Next, I turn to understanding the mechanism. I consider evidence related to the plausibility of three different channels.

The first channel I consider is parental resource allocation. There is existing empirical evidence of the crucial role of within-household resource allocation in determining child height (Attanasio et al., 2020; Jayachandran & Pande, 2017; Rosenzweig & Schultz, 1982). Parental illness could cause financial distress due to decreased labor supply and productivity and increased medical spending (Alam 2015; Bratti and Mendola 2014; Gertler and Gruber 2002; Schultz and Tansel 1997). In my data, I find that parental illness reduces time allocation for both domestic work and outside work, increases medical spending, decreases assets, and increases borrowing. Furthermore, I find that parental illness increases food insecurity, decreases food intake, and reduces protein consumption.

The second channel is early life stress. While I do not have direct measures of stress, stress appears to increase the probability of some disease conditions (Pohl, Medland, and Moeser 2015; Rosa, Lee, and Wright 2018; Taylor 2010). I use three disease conditions that have been linked with stress – fever, cough, and diarrhea – and show that parental illness increases the likelihood of having a disease condition. However, the result is imprecise, making it difficult to make a conclusive argument. Furthermore, while the absence of an effect would have suggested that stress is not important, the presence of an effect on these outcomes might be driven by some other mechanism.

Finally, I explore the fertility choice mechanism. If sick parents decide to have fewer children, their relatively small family size may lead to higher investment and better health outcomes for children – a quality-quantity trade-off which would have reduced the magnitude of the effect I am measuring. However, I do not find evidence of an effect on fertility.

This study makes two major contributions. First, it contributes to the literature on human capital accumulation by demonstrating that parental illness can cause significant loss in children's health, implying lower cognitive development and lower future earnings (Almond, Currie, and Duque 2018; Case, Fertig, and Paxson 2005; Currie 2009; Currie and Vogl 2013; Smith 2009;

[1] Joint families are households where the parents are not the household head or spouse of the head. Commonly, another male member such as father or brother of the parents is the household head in joint families.

Steckel 1995). Second, it contributes to the literature on adverse shocks and child health. Although parental illnesses are widespread in developing countries, this is one of the first studies to estimate the causal effect of parental illness on child health. There are a few qualitative and quantitative studies focusing effects of parents' cancer on children's physical functioning, such as headaches, abdominal pain, dizziness, sleeping problems, and loss of appetite (see Visser et al., 2004 for a review). However, the scope of these studies is limited: inference based on small samples – less than 100 children; focus on only cancer patients; do not use an objective measure of child health such as height; do not provide causal estimates.

The rest of the paper is structured as follows. In Section 2, I provide the context of the study. Then, I present a theoretical model explaining the conceptual framework in Section 3. Section 4 describes the data and method. In Sections 5-7, I present the main estimation result, robustness checks, heterogeneity analysis, and mechanisms of the effect. Section 8 shows the result for another health outcome (i.e., child weight). Finally, I conclude the paper in Section 9.

2. Context of the Study

2.1 Child Health

The World Health Organization (WHO) identifies malnutrition as the single greatest threat to the world's public health (WHO 2021). Globally, malnutrition is attributed to 45 percent of all child deaths (WHO 2020). Bangladesh is no exception to this trend. For example, as of 2019, more than a quarter of all children in Bangladesh were stunted, i.e., two standard deviations below the WHO growth standard (World Bank 2022). In addition, Bangladeshi children under the age of five also suffer from high rates of micronutrient deficiencies: vitamin A (21 percent), iron (51 percent), and zinc (45 percent) ("Global Nutrition Report 2021" 2022).

Child malnutrition is present in all geographic regions and economic groups in Bangladesh (Deolalikar, 2005). However, children living in rural areas are more likely to be stunted (28.4 percent), compared to their counterparts in urban areas (26.3 percent). Similarly, stunting is more prevalent among the lowest wealth quintile (38 percent) as compared to the highest wealth quintile (20 percent) ("Global Nutrition Report 2021" 2022). However, stunting is equally prevalent among male and female children– 28 percent and 27.9 percent, respectively ("Global Nutrition Report 2021" 2022).

Insufficient food intake, unhealthy diet patterns, and imbalances in nutrition intake are common reasons for high child malnutrition and stunting in Bangladesh. Around 35% of the population lacks food security in Bangladesh (NIPORT and icddr,b, 2021). Moreover, the tendency to consume cereals or staples (70 percent of total food intake) paired with an inadequate intake of protein and micronutrients is commonly responsible for reduced dietary diversity (Magnani et al. 2015). Food insecurity and poor dietary diversity in the early stage of children's life– the first 1000 days of a child's life starting from pregnancy to the second birthday– have severe consequences as they substantially affect the survival, physical growth, cognitive development, and productivity in the later life (Black et al., 2013; Currie & Vogl, 2013).

2.2 Health Shocks

Health shocks are one of the leading economic shocks in Bangladesh, as more than 25 percent of households face a health shock in a year (Hossain et al. 2019; Islam and Maitra 2012). About 24.6 percent of households spend more than 10 percent of their budget on healthcare (Ahmed et al. 2022). Major types of health shocks are accidents such as severe cuts or burn (43 percent), bone fractures (21 percent), major operation/surgery (25 percent), and non-communicable diseases (8 percent) (Hossain et al. 2019).

Health insurance is virtually nonexistent in Bangladesh (Rahman et al. 2013; Molla and Chi 2017). As a result, out-of-pocket payments are the primary way to finance healthcare in Bangladesh. Households spend, on average, $252 on health care per year (Ahmed et al. 2022). About 16 percent of ill-health individuals abstain from seeking health care because they cannot afford the treatment cost (BBS 2011). Those seeking healthcare are often forced to finance treatment expenses through borrowing and selling assets (Islam and Maitra 2012; Rahman et al. 2022).

Out-of-pocket healthcare payments cause a financial burden to households. Around 4.50 percent of the population (8.61 million) was pushed below the national poverty line from out-of-pocket healthcare payments in 2016 (Ahmed et al. 2022). Hamid, Ahsan, and Begum (2014) show that illness shocks drive 17 percent of households into poverty in rural Bangladesh annually. Therefore, protecting households from illness shocks has important implications for reducing poverty, human capital accumulation, and economic growth in Bangladesh.

3. Conceptual Framework

3.1 Human Capital Production Function

This section analyzes parental illness shock, intrahousehold resource allocation, and child human capital formation. I assume that a child has only one component of human capital– health (H_i). This is a sensible assumption as this study focuses on children under five. I denote parental characteristics such as age, education, and occupation by ζ_τ and child characteristics such as age, gender, and birth order by η_i. I further denote child prenatal endowment by ω_i and parental human capital investment in a child X_i. Parental illness shock is denoted by e_τ. I further assume that parental illness shocks affect all children in a household the same way (i.e., parents show no son preference) but vary across households (τ).[2] The human capital production function for child i in household τ is specified as follows:

$$H_{i,\tau} = f\left(\omega_{i,\tau}, X_{i,\tau}, e_\tau;\ \eta_{i,\tau}, \zeta_\tau\right) \quad (1)$$

where $\eta_{i,\tau}\ and\ \zeta_\tau$ are exogenous to the production function. Child human capital is determined by the child's endowments, human capital investment, and parental illness shocks. It is assumed that parental investment strictly increases child human capital ($\frac{\partial H_i}{\partial X_i} > 0$). Empirical studies provide evidence of the positive effect of parental investments on determining child human capital (Rosenzweig and Schultz 1982; Jayachandran and Pande 2017; Attanasio et al. 2020).

3.2 Parents' Preference

Parents are assumed to value child outcomes– q_i is the quality of the child.[3] Child quality varies in only one dimension– health. They also care about their own consumption (c) and leisure (l). Parents' consumption (c) includes both goods and health. Parental preferences are represented by utility function:

$$U = U(c, l, q_i) \quad (2)$$

[2] I empirically test this assumption in Section 6 and show that it holds.

[3] Parents could also value quantity of children instead of quality of children. However, Peters, Rees, and Hernández-Julián (2014) do not find evidence of a trade-off between child quantity and quality (i.e., health) in rural Bangladesh. As a result, the assumption that parents' have preference over quality (and not quantity) of children is a reasonable one in the context of Bangladesh. In Section 8, the mechanism of treatment effect, I empirically investigate whether parents' engage in a quality-quantity trade-off.

I denote parental labor supply by T. I normalize the parental time endowment to one and write the time constraint as $l + T = 1$. I also normalize the price of parental consumption to one. The household budget constraint is specified as follows:

$$\sum_i p_X X_i + c = Y + wT \tag{3}$$

where p_X is the relative price of human capital investment, w and Y are parents' wage rate and non-labor income.

3.3 Parental Illness Shock and Parents' Response

The parents' objective is to maximize their utility subject to the budget constraint and the production technology. A unique solution to the intrahousehold resource allocation problem exists if the utility function and production function are strictly concave and continuously twice differentiable. The optimal human capital investment in child i follows a functional form:

$$X_i^* = \psi(\omega_i, e_\tau, p_X, w, Y, \eta_i, \zeta_\tau) \tag{4}$$

From the human capital production function, the total effect of parental illness shock on child i's human capital can be decomposed as follows:

$$\frac{dH_{i,\tau}}{de_\tau} = \frac{\delta H_{i,\tau}}{\delta e_\tau} + \frac{\delta H_{i,\tau}}{\delta X_i}\frac{\delta X_{i,\tau}}{\delta e_\tau} \tag{5}$$

Equation (5) shows that the total effect of parental illness shock (i.e., the left-hand side term) on child human capital production ($\frac{dH_{i,\tau}}{de_\tau}$) is determined by two different effects: the direct physiological effect of parental illness ($\frac{\delta H_{i,\tau}}{\delta e_\tau}$) and behavioral (i.e., parental response) effect ($\frac{\delta H_{i,\tau}}{\delta X_i}\frac{\delta X_{i,\tau}}{\delta e_\tau}$).

A reduced-form empirical estimation can only estimate the total effect of parental illness. The direct physiological effect of parental illness ($\frac{\delta H_{i,\tau}}{\delta e_\tau}$) operates through the human capital production function and is assumed to be negative. The negative physiological effect on child health may arise from the stressful and grim household environment due to parental illness. The first part of the behavioral (i.e., parental response) effect ($\frac{\delta H_{i,\tau}}{\delta X_i}$) captures the productivity effect of parental investment. Empirical studies show that parental investments positively affect human capital production (Rosenzweig and Schultz 1982; Yi et al. 2015; Jayachandran and Pande 2017; Attanasio et al. 2020). The second part of the parental response ($\frac{\delta X_{i,\tau}}{\delta e_\tau}$) captures the parental resource allocation effect. The parental resource allocation effect is likely to be

negative if parental illness causes financial distress or makes parents physically impaired from making healthcare investments.

4. Data and Method

4.1 Data

This study uses nationally representative panel data from rural Bangladesh– the Bangladesh Integrated Household Survey (BIHS) completed by the International Food Policy Research Institute (IFPRI). A total of 5,600 households from 325 randomly selected villages were surveyed in 2011-12 and followed up in 2015. Most households are followed in both rounds as the attrition rate is only 1.26 percent per year (IFPRI, 2016). The survey provides detailed information on household demographics and economic status, including income, expenditure, savings, assets, and borrowings. It also contains household members' information, including nutrition intake, anthropometric measurements, health status, healthcare-seeking behavior, and healthcare expenditures.

The treatment variable in this study is the parental (i.e., father or mother, or both) illness, which is defined as the limitation in activities of daily living (ADLs). Limitation in ADLs is a reliable indicator of long-term health status and reflects major unanticipated illnesses that may cause severe hardships to a household (Bratti and Mendola 2014; Genoni 2012; Gertler and Gruber 2002; Crespo and Mira 2014). [4] In addition, empirical studies suggest that ADL limitation is a better indicator of illness than other self-reported indicators such as assessments of general health status and morbidities (Bratti and Mendola 2014; Genoni 2012; Gertler and Gruber 2002; Strauss et al. 1993). This is because ADL limitation questions are more specific and objective and, as a result, are less prone to measurement errors.

The surveys collect data on three ADL indicators: the extent to which one cannot walk, sit, or carry weight. The data is collected on a Likert scale to measure any difficulty in performing a task: 1 equals the person can perform the task easily, 2 equals the person can perform the task with some difficulty, 3 equals the person can perform the task with a lot of difficulties, and 4 equals the person cannot perform the task at all. Based on the responses, I create a treatment indicator that equals 1 if parents (i.e., father, mother, or both) have at least some difficulties in

[4] I empirically investigate whether ADL limitation is difficult to predict in Section 5.2 "Robustness Check". I also empirically investigate whether ADL limitation causes financial hardship in Section 7 "Mechanism".

walking, sitting, or carrying weight (i.e., responded greater than one in any of the ADL indicators) and equals 0 if parents have no issue in performing those tasks.[5]

The baseline sample consists of all healthy parents (i.e., no ADL limitations) in 2011-12.[6] The treatment group consists of parents who developed ADL limitations between 2013 and 2015, and the control group consists of parents who remained healthy. About 25.3 percent of the children in my sample are in the treatment group, and 74.7 percent are in the control group.

The primary outcome of interest in this study is child height. Child height has proven to be an informative measure of long-run nutritional status (Waterlow et al. 1977; Leonard 1988). Height at a particular age reflects the history of net nutrition– the difference between the food intake needed to sustain growth and disease and other claims on the diet. In addition, child height is strongly predictive of cognitive ability (Case and Paxson 2008), educational attainment (Currie 2009), occupational choice (Case, Paxson, and Islam 2009), and labor market outcomes (Persico, Postlewaite, and Silverman 2004; Smith 2009).

I measure child height using the WHO growth standard height-for-age (HFA) z-score. HFA z-score measures how children should grow under optimum conditions and with optimum infant feeding and child health practices. HFA z-score is a widely used health indicator for children between 0 and 5 years (Akresh, Verwimp, and Bundervoet 2011; Jayachandran and Pande 2017; Hirvonen, Sohnesen, and Bundervoet 2020; Hossain and Nikolov 2022).

In an ideal growth environment, the child height-for-age (HFA) z-score should have a normal distribution centering the mean at zero. However, in most developing countries, children grow up in a sub-optimal environment, and the median of the height distribution is often well below zero. In my data, the baseline average child HFA z-score is -1.43, which suggests that an average child in Bangladesh is 1.43 standard deviations (SDs) shorter than what they would have if they were living in an ideal growth environment.

[5] I check the sensitivity of my results using alternative treatment definitions – low ADL limitation and high ADL limitation – in Section 5.1. Low ADL limitation equals 1 if parents only have some difficulties in walking, sitting, or carrying weight (i.e., responded two in any of the ADL indicators) and equals 0 if parents have no issue in performing those tasks. And high ADL limitation equals 1 if parents have a lot of difficulty or cannot do the tasks (i.e., responded greater than two in any of the ADL indicators) and equals 0 if parents have no issue in performing those tasks.

[6] That is, all parents (including would-be parents in 2015) with ADL limitations are dropped from the baseline sample.

The baseline average HFA z-scores of treatment and control group children are -1.46 and -1.37, respectively.[7] A Kolmogorov-Smirnov (K-S) test fails to reject that the full distributions of HFA z-scores in the baseline are the same (see Appendix Table A1). As a result, the data suggests that child height between the treated and the control group is balanced at the baseline. On the contrary, the average HFA z-scores of treatment and control group children in 2015 are -1.57 and -1.35, respectively. A two-sample Kolmogorov-Smirnov (K-S) test rejects that the full distributions of HFA z-scores are the same (see Appendix Table A1).

Next, I explore the covariate balance between treated and control groups at the baseline in Table 1. The covariates I include are household characteristics (i.e., family size, income, asset, loan, non-health shocks such as crop failure, and other members with ADL limitations) and household head's characteristics (i.e., age, gender, education, and occupation). Following the suggestion of Imbens & Wooldridge (2009), I use a normalized difference of covariates between the treatment and control groups, rather than the *t*-statistics.[8] Since normalized difference compares the difference in means in units of the pooled standard division, it is not influenced by the sample size and allows for assessment of the balance of the covariates measured in different units (Imbens and Rubin 2015; Imbens and Wooldridge 2009).

Imbens & Rubin (2015) suggest as a rule of thumb that a normalized difference greater than one quarter makes linear regression methods sensitive to the specification. LaLonde (1986) shows that if the normalized difference exceeds one for several covariates, regression models are unlikely to produce credible results. Column (3) of Table 1 shows that none of the covariates have a normalized difference greater than 0.25. This suggests that the differences in baseline covariates between treated and control groups are negligible.

[7] Appendix Figure A1 shows the distribution of HFA z-score for treated and control group children. Panel A illustrates the Kernel density distribution of the baseline HFA z-scores for treated and control group children. The HFA z-score distributions for control (the dotted line) and treated group (the solid line) have no differential pattern. Conversely, Panel B illustrates that in 2015 the HFA z-scores distribution of treated children shifted to the left compared to the control children.

[8] Normalized Difference $= (\bar{x}_{Treated} - \bar{x}_{Control})/\sqrt{(s^2_{Treated} + s^2_{Control})}$ where $\bar{x}$ is the sample mean and s^2 is the sample variance of covariates. On the other hand, $t - \text{statistic} = (\bar{x}_{Treated} - \bar{x}_{Control})/\sqrt{(s^2_{Treated}/N_{Treated} + s^2_{Control}/N_{Control})}$.

Table 1: Baseline Covariate Balance

	Control	Treated	Normalized Difference
	(1)	(2)	(3)
Household head's characteristics			
Head's age	39.25	39.989	-0.052
	(13.967)	(14.556)	
Head is female (=1 if yes)	0.107	0.111	-0.013
	(0.309)	(0.314)	
Head is literate (=1 if yes)	0.523	0.546	0.047
	(0.500)	(0.498)	
Head is wage-earner (=1 if yes)	0.132	0.163	-0.088
	(0.339)	(0.370)	
Head is self-employed (=1 if yes)	0.757	0.704	0.119
	(0.429)	(0.457)	
Household characteristics			
Log (family size)	1.496	1.438	0.157
	(0.374)	(0.365)	
Log (income/capita)	8.872	8.906	-0.014
	(2.273)	(2.373)	
Log (asset/capita)	8.534	8.580	-0.035
	(1.298)	(1.357)	
Have a loan (=1 if yes)	0.264	0.207	0.134
	(0.441)	(0.405)	
Non-health shocks (=1 if yes)	0.125	0.115	0.032
	(0.331)	(0.319)	
Other members' ADL limitation (=1 if yes)	0.289	0.338	-0.107
	(0.454)	(0.473)	
Number of observations	1,300	440	1,740

Notes: (a) This table shows the baseline covariate balance between the treated (i.e., parental illness) and the control (i.e., no parental illness) groups. (b) Standard deviations are in parentheses. (c) Column (3) shows the normalized mean difference.

4.2 Method

The key finding motivating my identification strategy is that, at the baseline, the distribution of child height is not statistically different between treated and control groups. In addition, the baseline covariates are nearly balanced between the treatment and control groups. Moreover, there is no significant difference in the likelihood of other household members developing an ADL limitation between treatment and control groups. This lack of correlation indicates that there are no household level confounders biasing the treatment assignment (i.e., treated households are not living in conditions that are more susceptible to injury and illness). As a result, for a confounder to cause bias in my estimates, it has to be something (i) unobserved,

(ii) specific to only one household member, and (iii) does not affect children's height until after the ADL limitation is realized. It is difficult to imagine what such a confounder might be.

These findings suggest that the treatment assignment is minimally confounded or as good as random. However, to help address any remaining endogeneity, I use doubly robust estimation (Bang & Robins, 2005; Imbens & Wooldridge, 2009; Robins et al., 1994; Wooldridge, 2007, 2010). Doubly robust estimation is a selection on observables approach which is designed to minimize potential bias due to misspecification. It provides unbiased estimates by modeling both the outcome and the treatment equations. Doubly robust estimation requires that only at least one of the two models be correctly specified to obtain an unbiased effect estimator. As a result, it allows two opportunities to obtain an unbiased estimator when adjusting for selection issues (Emsley et al., 2008; Imbens & Wooldridge, 2009; Wooldridge, 2007, 2010).

The doubly robust estimator I use is the inverse-probability weighted regression adjustment (IPWRA) (Imbens & Wooldridge, 2009; Wooldridge, 2007, 2010). IPWRA models both treatment and outcome by combining an "inverse-probability weighted (IPW)" estimator with a "regression adjustment (RA)" estimator. The IPW estimator models the probability of treatment without any assumptions about the functional form of the outcome model. Conversely, the RA estimator models the outcome without any assumptions about the functional form of the probability of the treatment model.

The IPW estimators use weights based on the probability of receiving treatment to create a synthetic sample in which the distribution of observed baseline covariates is independent of treatment status. The inverse probability weights for the treated units are defined as $\frac{1}{P(D_i=1|X_i)}$ and for the control units as $\frac{1}{1-P(D_i=1|X_i)}$. Here, D is the treatment indicator that takes 1 if an individual i is treated and 0 otherwise; X is a multidimensional vector of baseline covariates; and $P(D_i = 1|X_i)$ is the probability of receiving treatment (propensity score) defined as $P(D_i = 1|X_i) = F\{h(X_i)\} = E(D_i|X_i)$ (Rosenbaum and Rubin 1983), where $F\{.\}$ a cumulative distribution function. On the other hand, RA estimators fit separate regression models of the outcome on the baseline covariates for treatment and control units and use the contrasts of the averages of predicted outcomes to estimate treatment effects.

Following Wooldridge (2010), the IPWRA estimator of the average treatment effect on the treated (ATT) can be expressed as

$$ATT_{IPWRA} = \frac{1}{n_T}\sum_{i=1}^{n} D_i[r_T^*(X, \delta_T^*) - r_C^*(X, \delta_C^*)], \quad (6)$$

where n_T is the number of treated units, $r_T(.)$ and $r_C(.)$ are postulated regression models of the outcome on the baseline covariates for treatment (T) and control (C) units, and model parameters are $\delta_j = (\alpha_j, \beta_j)$ and $j = (T, C)$. The estimated inverse probability weighted parameters for treated and control units (i.e., $\delta_T^* = (\alpha_T^*, \beta_T^*)$ and $\delta_C^* = (\alpha_C^*, \beta_C^*)$) are obtained from weighted regression procedures, respectively:

$$\min_{\alpha_T^*, \beta_T^*} \sum_{i=1}^{n} \frac{D_i(y_i - \alpha_T^* - X_i\beta_T^*)^2}{\hat{p}(X_i, \hat{\gamma})} \quad (7)$$

$$\min_{\alpha_C^*, \beta_C^*} \sum_{i=1}^{n} \frac{(1 - D_i)(y_i - \alpha_C^* - X_i\beta_C^*)^2}{1 - \hat{p}(X_i, \hat{\gamma})} \quad (8)$$

where $\hat{p}(X_i, \hat{\gamma})$ are the estimated probabilities of receiving treatment (i.e., propensity scores).

Weighting with propensity scores can be interpreted as removing the correlation between treatment and confounders, and regression as removing the direct effect of observed covariates. As a result, combining weighting and regression can lead to additional robustness by removing the correlation between the unobserved covariates and reducing the correlation between the unobserved and observed confounders (Imbens and Wooldridge 2009).

In this study, I follow a three-step procedure to implement the IPWRA estimator. Other recent empirical works have followed a similar approach (Chhay and Yamazaki 2021; Manda et al. 2018; Tambo and Mockshell 2018; Tani, Xu, and Zhu 2021; Walker and Zhu 2018). First, the probability of parental illness (i.e., the treatment model) is estimated using a logistic regression model, and predicted probabilities (i.e., propensity scores) are used in computing the inverse-probability weights. The baseline covariates included in the logit model are household characteristics (i.e., family size, income, asset, loan, non-health shocks such as crop failure, and other members with ADL limitations) and household head's characteristics (i.e., age, sex, education, and occupation).

Second, using the inverse-probability weights, I fit weighted regression models of child height (i.e., outcome models) for both treatment and control groups to obtain the treatment-specific

predicted outcomes for each child. In the outcome model, I also control for child characteristics such as age, gender, and birth order. This step corresponds to equations (7) and (8). It is important to note that if the regression function is correctly specified, the weights do not affect the consistency of the estimator. However, as the true functional form is rarely known in practice and misspecification is likely to arise, weighting can help reduce the bias (Imbens and Wooldridge 2009; Tani, Xu, and Zhu 2021; Walker and Zhu 2018).

Finally, I compute the means of the predicted outcomes for both treatment and control groups. The difference between the treatment and control group means provides estimates of the average treatment effect (ATE) of parental illness. Similarly, I obtain the average treatment effect on the treated (ATT) by restricting the computation of the means of the predicted outcomes to the subset of treated children. The ATT estimates are consistent if the models for either the treatment (i.e., step 1) or the outcome (i.e., step 2) are correctly specified (Emsley et al., 2008; Imbens & Wooldridge, 2009; Wooldridge, 2007, 2010).

In this study setting, the two alternative estimation techniques are ordinary least squares (OLS) and matching. Doubly robust method has several advantages over both OLS and matching methods. Relative to OLS, doubly robust method is more robust to model misspecification. Relative to matching, doubly robust estimation uses more observations and has more statistical power. Comparing OLS and Matching, estimates of matching method are more interpretable and less sensitive to functional form assumptions. Doubly robust keeps the advantages of matching method without throwing away samples and provides estimates which are more robust to misspecification and easily interpretable.

I use matching methods as a robustness check following the recent literature (Alam 2015; Bø, Halvorsen, and Thoresen 2019; Gao, Zhai, and Garfinkel 2010; Kandpal 2011; Litzow, Pattanayak, and Thinley 2019; Manda et al. 2018; Tambo and Mockshell 2018; Tani, Xu, and Zhu 2021). The identification strategy here is to find a statistically comparable group in the pre-treatment (i.e., baseline) variables who have no illness (i.e., control) to match those who have an illness (i.e., treated). Then, estimate the treatment effect of parental illness using the matched sample.

I implement two popular matching methods – multivariate distance matching (MDM) and propensity score matching (PSM). Multivariate distance matching (MDM) works by pairing close units based on a distance metric– Mahalanobis (normalized Euclidean) distance, which measures the proximity between observations in the multivariate space of covariates. Two units

with identical covariate values will have a Mahalanobis distance of zero, and the distance increases the more different the covariate values are. On the other hand, propensity score matching (PSM) pairs units with similar propensity scores (i.e., likelihood of treatment) instead of computing multivariate distance.

I apply matching methods following a three-step process. First, for each treated observation, find "statistical twins" from the untreated observations. Second, compute the counterfactual outcome for the observation at hand using the outcome values of the matched observations. Finally, estimate the average causal effect by taking the mean difference between the observed and the "imputed" counterfactual values (Jann 2017). For average treatment effect on the treated (ATT), I need to estimate counterfactual outcomes ($\hat{Y}_i^0$) using the untreated matches. It is shown below using the treatment effect terminology–

$$\widehat{ATT} = \frac{1}{N^{D=1}} \sum_{i|D=1} \left[Y_i - \hat{Y}_i^0 \right] = \frac{1}{N^{D=1}} \sum_{i|D=1} \left[Y_i - \sum_{j|D=0} w_{ij} Y_j \right] \quad (9)$$

where, D is the treatment indicator that takes 1 if an individual i is treated and 0 otherwise. And w_{ij} is the matching weight, which depends on the matching algorithms.

Several matching algorithms are available, such as nearest neighbor, radius, caliper, and kernel. I use the kernel matching algorithm,[9] which uses a weighted average of all untreated observations to construct the counterfactual outcome. It assigns a relatively larger weight to untreated observations for which the Mahalanobis distance (or difference in propensity score) is smaller.

Before moving to the treatment effect estimation, the final point to note is that the doubly robust and the matching method rely on the conditional independence assumption (CIA) (Lechner 2001; 2002; Angrist and Pischke 2008).[10] CIA implies that given a set of pre-treatment observable covariates, potential outcomes are independent of treatment assignment (Caliendo and Kopeinig 2008; Imbens and Rubin 2015). However, to satisfy the CIA, the matching method requires a credible belief that there are no unobservable confounders that may bias the impact estimates (Litzow, Pattanayak, and Thinley, 2019; Cunningham, 2021). In this study context, I argue that the CIA is likely to hold as both the outcome and the pre-treatment covariates are quite similar at the baseline.[11] As a result, I argue that the treatment assignment

[9] Using nearest neighbor, radius and caliper matching algorithms provides similar results.
[10] This assumption is also referred to as unconfoundedness (Rosenbaum and Rubi, 1983) and selection on observables (Heckman and Robb, 1985; Dale and Kruger (2002).
[11] Table 1 shows the covariate balance without matching and Appendix Table 2 shows the post-matching balance of covariates using both MDM and PSM methods.

is as good as random, and the unobservable confounders are not a concern for estimating the effect of parental illness.

As a further robustness check, I run OLS regressions with and without the baseline covariates. The OLS estimations with and without covariates are very similar, suggesting that the observed covariates do not significantly influence the treatment effect. In addition, the ATT estimates of IPWRA, MDM, and PSM are very similar to the OLS estimates. This finding provides further confidence in the reliability of the causal estimates of the doubly robust method.

5. Results

5.1 Main Result

I present the average treatment effect on the treated (ATT) of parental illness on child height-for-age (HFA) z-score in Table 2. Columns (1) and (2) present the OLS estimates with and without the covariates. The coefficients of parental illness with and without the covariates in OLS estimation are similar, which suggests that the treatment effect is not sensitive to observable covariates. Column (3) shows the ATT estimate from IPWRA (i.e., doubly robust) method, which is also very similar to the OLS estimates. This finding reaffirms my claim that parental illness is as good as random, and endogeneity concerns from unobservable confounders are negligible.

The ATT estimate from IPWRA shows that parental illness leads to a 0.18 standard deviation lower HFA z-score. The result suggests that parental health shocks have a significant negative effect on child height. Akresh, Verwimp, and Bundervoet (2011) find a similar magnitude of negative effect for children in Rwanda born during a crop failure and children living during a civil war, 0.173 standard deviations and 0.234 standard deviations, respectively. Similarly, Hirvonen et al., (2020) find a 0.21 lower HFA z-score for exposure to drought in Ethiopia. Parental illness is more prevalent than larger shocks such as drought and civil war but has a similar magnitude of negative effect on child health.

Table 2: Effect of Parental Illness on Child Height

	OLS Without Control	OLS With Controls	IPWRA	MDM	PSM
	(1)	(2)	(3)	(4)	(5)
Parental illness (=1 if yes)	-0.220***	-0.175**	-0.177**	-0.201**	-0.198**
	(0.078)	(0.074)	(0.074)	(0.090)	(0.089)
Control	No	Yes	Yes	Yes	Yes
Observations	1,740	1,740	1,740	1,611	1,605

Notes: (a) This table presents the average treatment effect on the treated (ATT) based on Ordinary Least Squares (OLS), Inverse-probability-weighted Regression Adjustment (IPWRA), Multivariate Distance Matching (MDM), and Propensity Score Matching (PSM) methods. (b) The treatment variable is the parental illness that equals one if parents have any ADL limitation, and zero otherwise. (c) The outcome variable is the children's height-for-age z-score. (d) Control variables are household head's age, head is female, head is literate, head is wage-earner, head is self-employed, family size, income per capita, asset per capita, have a loan, household faced a non-health shock, other members have ADL limitations, and child's age, gender, and birth order. (e) Robust standard errors are in parentheses. (f) Significance: ***p<0.01, **p<0.05, *p<0.1.

A back-of-the-envelope calculation shows that removing the effects of parental illness would close 3.5% of the gap in height between Bangladeshi children and the global average. I compute this number by multiplying the treatment effect by the fraction of children who are treated and dividing it by the average HFA z-score of Bangladeshi children.

I check the sensitivity of the primary estimation results using alternative definitions of the treatment variable – low ADL limitation and high ADL limitation. The low ADL indicates that parents have some difficulty performing ADL activities, whereas the high ADL limitation indicates that parents have a lot of difficulties or cannot perform ADL activities. The result of this exercise is presented in Appendix Table A3. The results show that greater ADL limitations are associated with somewhat greater child growth deprivation (i.e., greater decline in HFA z-score). However, the difference is not statistically significant.

5.2 Robustness Check

In this sub-section, I test the robustness of the main result. Particularly, I conduct two robustness checks: estimate the treatment effect using alternative methods and check predictability of parents' ADL limitation. I briefly explain these tests and show the empirical findings.

First, I use matching methods as a robustness check following the recent literature (Alam, 2015; Bø et al., 2019; Gao et al., 2010; Kandpal, 2011; Litzow et al., 2019; Manda et al., 2018; Tambo & Mockshell, 2018; Tani et al., 2021). I implement two popular matching methods –

multivariate distance matching (MDM) and propensity score matching (PSM). Columns (4) and (5) of Table 2 show the estimates of MDM and PSM estimates with kernel matching algorithms.[12] The ATT estimates of MDM and PSM are very similar to the OLS and IPWRA estimates, which, once again, suggests that the unobserved confounding factors do not affect the main estimates in any meaningful way. As a result, I argue that the treatment effect of parental illness is robust to alternative identification strategies.

In the second robustness check, I test whether parents could use information about their past health conditions – acute and chronic – to predict the likelihood of having ADL limitation.[13] If parents could successfully predict the likelihood of having an ADL limitation, the treatment assignment is not as good as random. Besides, parents could take effective measures (i.e., coping strategies) against a predictable illness that will likely reduce the effect of parental illness. In such a scenario, the observed treatment effect will be an underestimation of the true treatment effect.

This exercise uses the parents' acute and chronic conditions at the baseline as treatment variables and parents' ADL limitation in 2015 as an outcome variable. Both acute and chronic condition variables are dummy indicators that equal one if parents have a condition at the baseline, and zero otherwise. Similarly, the outcome variable is a dummy indicator that equals one if parents have ADL limitation in 2015, and zero otherwise. Table 3 shows that having acute or chronic conditions at the baseline does not predict the likelihood of developing ADL limitation in 2015. This result suggests that the treatment variable of this study– parents' ADL limitation – represents an illness shock that is difficult to predict based on their past health conditions.

[12] Other matching algorithams such as nearest neighbors, radius, and caliper provide similar results. Hence, the matching estimation results are robust to the choice of matching algorithm.

[13] Acute condition is an episode of illness that lasts less than a month, and include health conditions such diarrhea, fever, injury, pain, headache, malaria, pneumonia, and typhoid. On the other hand, chronic condition is an episode of illness that lasts more than months and include health conditions such as heart disease, gastric ulcer, asthma (respiratory disease), diabetic, cancer, and epilepsy.

Table 3: Predicting Parents' ADL Limitation

	Outcome: ADL Limitation in 2015 (=1 if yes)		
	OLS Without Control	OLS With Controls	IPWRA
	(1)	(2)	(3)
Panel A			
Acute condition at baseline (=1 if yes)	0.012	-0.004	-0.009
	(0.030)	(0.030)	(0.031)
Control	No	Yes	Yes
Observations	1,019	1,019	1,019
Panel B			
Chronic condition at baseline (=1 if yes)	-0.021	-0.033	-0.039
	(0.055)	(0.054)	(0.054)
Control	No	Yes	Yes
Observations	1,019	1,019	1,019

Notes: (a) This table presents the average treatment effect on the treated (ATT) based on Ordinary Least Squares (OLS) and Inverse-probability-weighted Regression Adjustment (IPWRA) methods. (b) The explanatory variable in Panel A is the parents, acute condition (at the baseline) that equals one if parents had any disease that lasted less than a month, and zero otherwise. The explanatory variable in Panel B is the parents' chronic condition (at the baseline) that equals one if parents had any disease that lasted more than 3 months, and zero otherwise. (c) The outcome variable is parents' ADL limitation (in 2015) that equals one if parents have any ADL limitation, and zero otherwise. (d) Control variables are household head's age, head is female, head is literate, head is wage-earner, head is self-employed, family size, income per capita, asset per capita, have a loan, household faced a non-health shock, and other members have ADL limitations. (e) Robust standard errors are in parentheses. (f) Significance: ***p<0.01, **p<0.05, *p<0.1.

6. Heterogeneity

6.1 Illness of Household vs. Non-Household Head Parents

In this sub-section, I explore the heterogeneity of treatment effects by the characteristics of parents and children. First, I assess whether parental illness has differential effects on child height by the parents' household head status. This exercise will help us to explore whether the within-household safety net effectively protects children. Household head parents have greater control over household resources than non-household head parents,[14] and their illness may make a household more economically vulnerable. On the other hand, as the non-head parents are less likely to be the household's main earner, their illness may not put the household in financial distress. As a result, given the within-household safety net feature of a joint family, children of non-head parents may not suffer a large loss in height.

Panel A of Table 4 shows the average treatment effect of household head parents' illness on child HFA z-score.[15] The IPWRA estimate shows that head parents' illness leads to 0.17

[14] Commonly, another male member such as father or brother of the parent is the household head in joint families.

[15] A Kolmogorov-Smirnov (K-S) test fails to reject that the full distributions of HFA z-scores in the baseline are the same (see Appendix Table A4).

standard deviations lower HFA z-score. Panel B of Table 4 shows the average treatment effect of non-household head parents' illness on child HFA z-score. The IPWRA estimate shows that non-head parents' illness leads to -0.19 standard deviations lower HFA z-score. Although the treatment effect for non-head parents is not statistically significant, the ATT estimates for head and non-head parents are not statistically different. This result suggests that the within-household safety net is ineffective in protecting children's health against parental illness.

Table 4: Heterogenous Effect of Parental Illness by Parents' Head Status

	OLS Without Control	OLS With Controls	IPWRA
	(1)	(2)	(3)
Panel A: Household head parents			
Parental illness (=1 if yes)	-0.211**	-0.176**	-0.171**
	(0.089)	(0.086)	(0.085)
Control	No	Yes	Yes
Observations	1,263	1,263	1,263
Panel B: Non-household head parents			
Parental illness (=1 if yes)	-0.209	-0.177	-0.193
	(0.163)	(0.165)	(0.179)
Control	No	Yes	Yes
Observations	477	477	477

Notes: (a) This table presents the average treatment effect on the treated (ATT) based on Ordinary Least Squares (OLS) and Inverse-probability-weighted Regression Adjustment (IPWRA) methods. (b) The treatment variable is the parental illness that equals one if parents have any ADL limitation, and zero otherwise. (c) The outcome variable is the children's height-for-age z-score. (d) Panel A shows the average treatment effect on the treated (ATT) for household head parents, and Panel B shows the ATT for non-household head parents. (e) Control variables are household head's age, head is female, head is literate, head is wage-earner, head is self-employed, family size, income per capita, asset per capita, have a loan, household faced a non-health shock, other members have ADL limitations, and child's age, gender, and birth order. (f) Robust standard errors are in parentheses. (g) Significance: ***p<0.01, **p<0.05, *p<0.1.

6.2 Illness of Father vs. Mother

Studies show that illnesses of father and mother have a differential effect on children's educational outcomes (Alam 2015; Bratti and Mendola 2014; Lim 2020). In Table 5, I explore whether illness of the father have any differential effect on child height compared to the illness of the mother.[16] Panel A of Table 5 shows the average treatment effect of father's illness on child HFA z-score. The IPWRA estimate shows that father's illness leads to 0.16 standard deviation lower HFA z-score. On the other hand, Panel B of Table 5 shows the average treatment effect of the mother's illness on child HFA z-score. The IPWRA estimate shows that mother's illness leads to 0.17 standard deviation lower HFA z-score. Although the treatment

[16] I conduct equality of distribution tests separately for only father's illness and only mother's illness. The Kolmogorov-Smirnov (K-S) tests fail to reject that the full distributions of HFA z-scores in the baseline are the same (see Appendix Table A5).

effect for father's illness is not statistically significant, the effect size for father's illness is not statistically different from the mother's illness.

Table 5: Heterogenous Effect of Father's and Mother's Illness

	OLS Without Control	OLS With Controls	IPWRA
	(1)	(2)	(3)
Panel A: Father only			
Father's illness (=1 if yes)	-0.234	-0.152	-0.163
	(0.183)	(0.173)	(0.172)
Control	No	Yes	Yes
Observations	1,214	1,214	1,214
Panel B: Mother only			
Mother's illness (=1 if yes)	-0.140*	-0.169**	-0.166**
	(0.084)	(0.078)	(0.078)
Control	No	Yes	Yes
Observations	1,697	1,697	1,697

Notes: (a) This table presents the average treatment effect on the treated (ATT) based on Ordinary Least Squares (OLS) and Inverse-probability-weighted Regression Adjustment (IPWRA) methods. (b) The treatment variable in Panel A is the father's illness that equals one if father has any ADL limitation, and zero otherwise. The treatment variable in Panel B is the mother's illness that equals one if mother has any ADL limitation, and zero otherwise. (c) The outcome variable is the children's height-for-age z-score. (d) Control variables are household head's age, head is female, head is literate, head is wage-earner, head is self-employed, family size, income per capita, asset per capita, have a loan, household faced a non-health shock, other members have ADL limitations, and child's age, gender, and birth order. (e) Robust standard errors are in parentheses. (f) Significance: ***p<0.01, **p<0.05, *p<0.1.

6.3 Heterogeneity by Child Age Groups:

Next, I explore whether the effect of parental illness varies with the age of the children. The WHO breastfeeding guidelines suggest that children should be breastfed for at least the first 24 months along with other complementary foods**.** Besides, children grow faster between 0-24 months than between 25-59 months. Therefore, I divide the children into two groups based on feeding practices and growth phase: children aged 0-24 months and 25-59 months**.** Table 6, Panel A and B, show the effect of parental illness on child HFA z-score for ages 0-24 months and 25-59 months, respectively. The IPWRA estimate shows parental illness has a similar effect on both age groups– -0.21 standard deviations for children ages 0-24 months and -0.16 standard deviations for children ages 25-59 months. This finding suggests that parental illness is equally detrimental to all children between 0-5 years of age.

Table 6: Heterogenous Effect of Parental Illness by Child Age Groups

	OLS Without Control	OLS With Controls	IPWRA
	(1)	(2)	(3)
Panel A: Aged 0-24 months			
Parental illness (=1 if yes)	-0.196	-0.201	-0.213
	(0.149)	(0.148)	(0.150)
Control	No	Yes	Yes
Observations	701	701	701
Panel B: Aged 25-59 months			
Parental illness (=1 if yes)	-0.197**	-0.167**	-0.159**
	(0.078)	(0.079)	(0.079)
Control	No	Yes	Yes
Observations	1,039	1,039	1,039

Notes: (a) This table presents the average treatment effect on the treated (ATT) based on Ordinary Least Squares (OLS) and Inverse-probability-weighted Regression Adjustment (IPWRA) methods. (b) The treatment variable is the parental illness that equals one if parents have any ADL limitation, and zero otherwise. (c) The outcome variable is the children's height-for-age z-score. (d) Panel A and B show the average treatment effect on the treated (ATT) for age groups 0-24 and 25-59, respectively. (e) Control variables are household head's age, head is female, head is literate, head is wage-earner, head is self-employed, family size, income per capita, asset per capita, have a loan, household faced a non-health shock, other members have ADL limitations, child's gender, and child's birth order. (f) Robust standard errors are in parentheses. (g) Significance: ***p<0.01, **p<0.05, *p<0.1.

6.4 Heterogeneity by Child's Gender:

Empirical studies from developing countries provide evidence of differential parental investments and height outcomes based on a child's gender (Barcellos, Carvalho, and Lleras-Muney 2014; Jayachandran and Pande 2017). Girls display a substantial height disadvantage in contexts where son preference is prevalent (Dasgupta 2016). In the context of financial distress caused by parental illness, parents may favorably invest limited household resources in boys if they exhibit son preference.

In Table 7, I investigate whether parental illness leads to differential height outcomes based on the child's gender. Panels A and B show the average treatment effect of parental illness for boys and girls, respectively. The IPWRA estimates show that parental illness leads to 0.26 standard deviations lower HFA z-score for boys and 0.13 standard deviations lower HFA z-score for girls. However, the treatment effect is not statistically significant for girls, and the ATT estimate for girls is not statistically different from boys. As a result, I argue that parental illness does not lead to differential health outcomes for daughters due to son preference in Bangladesh. Other studies find similar results in Bangladesh. For instance, Sultana, Rahman, and Akter (2019) find that girls are 11% less likely to be stunted than boys.

Table 7: Heterogenous Effect of Parental Illness by Child Gender

	OLS Without Control	OLS With Controls	IPWRA
	(1)	(2)	(3)
Panel A: Boys			
Parental illness (=1 if yes)	-0.258**	-0.254**	-0.263**
	(0.110)	(0.102)	(0.102)
Control	No	Yes	Yes
Observations	921	921	921
Panel B: Girls			
Parental illness (=1 if yes)	-0.181	-0.120	-0.127
	(0.111)	(0.111)	(0.110)
Control	No	Yes	Yes
Observations	819	819	819

Notes: (a) This table presents the average treatment effect on the treated (ATT) based on Ordinary Least Squares (OLS) and Inverse-probability-weighted Regression Adjustment (IPWRA) methods. (b) The treatment variable is the parental illness that equals one if parents have any ADL limitation, and zero otherwise. (c) The outcome variable is the children's height-for-age z-score. (d) Panels A and B show the average treatment effect on the treated (ATT) for boys and girls, respectively. (e) Control variables are household head's age, head is female, head is literate, head is wage-earner, head is self-employed, family size, income per capita, asset per capita, have a loan, household faced a non-health shock, other members have ADL limitations, child's age, and child's birth order. (f) Standard errors are in parentheses. (g) Significance: ***p<0.01, **p<0.05, *p<0.1.

6.5 Heterogeneity by Birth Order:

Studies show large birth order differences in nutritional status and height of children (Horton 1988; Hatton and Martin 2010; Jayachandran and Pande 2017). The argument is that, unlike first-born children, later-born children need to share household resources with their siblings. This sibling rivalry for household resources puts later-born children at a disadvantage and leads to birth-order inequality in intrahousehold resource allocation.

In Table 8, I investigate how parental illness affects children of different birth orders. Panels A-C show the average treatment effect of parental illness on child HFA z-score for first-born, second-born, and third-or-higher-born children, respectively. The IPWRA estimate shows that the parental illness leads to 0.22 standard deviations lower HFA z-score for the first-born children, whereas 0.23 standard deviations lower HFA z-score for third-or-higher-born children. The ATT estimates of parental illness for different birth orders are not statistically different. This result suggests that later-born children, particularly third-or-higher-born children, do not experience a greater loss in height due to parental illness as compared to first-born and second-born children.

Table 8: Heterogenous Effect of Parental Illness by Child's Birth Order

	OLS Without Control	OLS With Controls	IPWRA
	(1)	(2)	(3)
Panel A: First-born			
Parental illness (=1 if yes)	-0.268**	-0.214*	-0.218*
	(0.131)	(0.127)	(0.128)
Control	No	Yes	Yes
Observations	560	560	560
Panel B: Second-born			
Parental illness (=1 if yes)	0.021	0.047	0.023
	(0.158)	(0.142)	(0.143)
Control	No	Yes	Yes
Observations	544	544	544
Panel C: Third or higher born			
Parental illness (=1 if yes)	-0.336***	-0.264**	-0.233*
	(0.127)	(0.120)	(0.124)
Control	No	Yes	Yes
Observations	634	634	634

Notes: (a) This table presents the average treatment effect on the treated (ATT) based on Ordinary Least Squares (OLS) and Inverse-probability-weighted Regression Adjustment (IPWRA) methods. (b) The treatment variable is the parental illness that equals one if parents have any ADL limitation, and zero otherwise. (c) The outcome variable is the children's height-for-age z-score. (d) Panels A-C show the average treatment effect on the treated (ATT) for first-born, second-born, and third or higher-born children, respectively. (e) Control variables are household head's age, head is female, head is literate, head is wage-earner, head is self-employed, family size, income per capita, asset per capita, have a loan, household faced a non-health shock, other members have ADL limitations, child's age, and child's gender. (f) Robust standard errors are in parentheses. (g) Significance: ***p<0.01, **p<0.05, *p<0.1.

7. Mechanisms

Until now, I have presented evidence that parental illness shocks negatively affect child height. In this section, I turn to understanding the mechanism. I consider evidence related to the plausibility of three different channels: parental resource allocation, early life stress, and parents' fertility choice. I briefly discuss these three channels and examine their empirical support by re-estimating the main estimation equations with these proposed channels as outcome variables.

7.1 Parental Resource Allocation

The first and potentially the most critical channel I explore is parental resource allocation. If parental illness causes financial hardship for the household, parents may alter within-household resource allocations which may affect child height. There is growing empirical evidence of the

crucial role of within-household resource allocation in determining child height (Attanasio et al. 2020; Jayachandran and Pande 2017; Rosenzweig and Schultz 1982).

Parental illness can cause financial distress due to decreased labor supply and productivity and increased medical spending (Alam 2015; Bratti and Mendola 2014; Gertler and Gruber 2002; Schultz and Tansel 1997). Besides, in developing countries with less well-established credit markets and social protection systems, parents may be compelled to use health care financing strategies such as borrowing and selling productive assets that may reduce future earnings and deepen the financial burden (Islam and Maitra 2012; Mitra et al. 2016; Rahman et al. 2022; Sauerborn, Adams, and Hien 1996).

In Table 9, I present the effect of parental illness on the household's financial burden. The treatment variable is parental illness, and the outcome variables are productivity and financial constraint indicators in Panel A and B, respectively. I measure parents' productivity based on the time they spend on domestic and outside work. In the survey, the time use data is only available for household head parents. Mother's time use is considered for domestic work, which includes activities such as sewing, cooking, and other housework. Father's time use is considered for outside work, which includes activities such as farming and employment. The survey collects the amount of household income, medical spending, savings, assets, and loans for the prior 12 months.

IPWRA estimates in Panel A of Table 9 show that parental illness reduces time allocation for both domestic work (22.6 minutes per day) and outside work (47.7 minutes per day). IPWRA estimates in Panel B show that parental illness leads to a 24.4 percent higher medical spending per capita. In addition, parental illness leads to 10 percent lower assets per capita and 43 percent higher loans per capita. These findings show that parental illness creates a significant financial burden due to lower productivity, large medical spending, and informal health care financing.

Table 9: Parental Illness and Financial Burden

Outcome Variables	Mean at the Baseline	OLS Without Control	OLS With Controls	IPWRA
	(1)	(2)	(3)	(4)
Panel A: Productivity				
Domestic work (minutes/day)	420.10	-18.414*	-21.646**	-22.591**
		(10.073)	(9.979)	(10.181)
Outside work (minutes/day)	410.97	-53.879**	-47.927**	-47.725**
		(21.292)	(21.325)	(21.490)
Observations		1,074	1,074	1,074
Panel B: Budget Constraint				
Log (income/capita)	9.39	0.031	0.031	0.037
		(0.109)	(0.104)	(0.104)
Log (medical spending/capita)	6.43	0.180**	0.247***	0.244***
		(0.075)	(0.072)	(0.074)
Log (savings/capita)	6.99	-0.043	-0.024	-0.028
		(0.192)	(0.190)	(0.191)
Log (asset/capita)	8.57	-0.120*	-0.101*	-0.103*
		(0.071)	(0.054)	(0.054)
Log (loans/capita)	8.17	0.437**	0.428**	0.432**
		(0.210)	(0.207)	(0.207)
Observations		1,740	1,740	1,740

Notes: (a) This table presents the average treatment effect on the treated (ATT) based on Ordinary Least Squares (OLS) and Inverse-probability-weighted Regression Adjustment (IPWRA) methods. (b) The treatment variable is the parental illness that equals one if parents have any ADL limitation, and zero otherwise. (c) Each cell of this table comes from a different regression equation. (d) Productivity (i.e., domestic and outside work) data is available only for parents who are household head or spouse of the head. Domestic work includes mother's time use in a day on cooking, sewing, and other housework. Outside work includes father's time use in a day on farm work and employment. (e) All households reported positive income, medical spending, and asset amounts. For savings and loans, some households reported zero amounts. For zero value responses in savings and loans, I use natural log transformation assuming a small positive number (i.e., ln(x+1)) to avoid dropping observations. Using inverse hyperbolic sine transformation gives similar results. (e) Control variables are household head's age, head is female, head is literate, head is wage-earner, head is self-employed, family size, income per capita, asset per capita, have a loan, household faced a non-health shock, and other members have ADL limitations. (f) Robust standard errors are in parentheses. (g) Significance: ***p<0.01, **p<0.05, *p<0.1.

Since parental illness causes a significant financial burden, it may reduce the quality and quantity of food intake in the household. I use two broad indicators of food intake: food insecurity (i.e., no food to eat at least for a meal in the past four weeks) and an index of food intake comprised of frequency of protein and carbohydrate consumption in a week.[17] Panel A of Table 10 shows the effect of parental illness on the two broad indicators of food intake, whereas Panel B shows the effects on each individual component of the food intake index.

[17] Food intake index is the first principal component of the protein and carbohydrate intake variables and normalized it to have zero mean and one standard deviation.

Table 10: Food Insecurity Mechanism of the Treatment Effect

Outcome Variables	Mean at the Baseline	OLS Without Control	OLS With Controls	IPWRA
	(1)	(2)	(3)	(4)
Panel A				
Food insecurity (=1)	0.079	0.049***	0.044**	0.044**
		(0.018)	(0.018)	(0.018)
Food intake index	0.000	-0.112**	-0.084*	-0.086*
		(0.053)	(0.049)	(0.049)
Observations		1,727	1,727	1,727
Panel B: Individual components				
Protein eating frequency in a week				
Lentil/bean	1.136	-0.068	-0.068	-0.060
		(0.100)	(0.099)	(0.099)
Eggs	1.601	-0.226**	-0.188*	-0.175*
		(0.105)	(0.103)	(0.104)
Dairy products	2.075	-0.130	-0.054	-0.050
		(0.171)	(0.165)	(0.166)
Meat (beef and goat)	0.322	-0.084**	-0.081**	-0.084**
		(0.035)	(0.035)	(0.036)
Poultry (chicken and duck)	0.444	0.017	0.041	0.040
		(0.056)	(0.056)	(0.056)
Protein intake index	0.000	-0.109**	-0.084*	-0.081*
		(0.052)	(0.049)	(0.049)
Carbohydrates eating frequency in a week				
Cereal	0.153	-0.057	-0.041	-0.044
		(0.075)	(0.076)	(0.075)
Wheat flour	1.091	-0.107	-0.080	-0.097
		(0.122)	(0.122)	(0.123)
Potato	5.209	-0.039	-0.055	-0.050
		(0.095)	(0.095)	(0.096)
Carbohydrate intake index	0.000	-0.028	-0.011	-0.018
		(0.055)	(0.055)	(0.056)
Observations		1,727	1,727	1,727

Notes: (a) This table presents the average treatment effect on the treated (ATT) based on Ordinary Least Squares (OLS) and Inverse-probability-weighted Regression Adjustment (IPWRA) methods. (b) The treatment variable is the parental illness that equals one if parents have any ADL limitation, and zero otherwise. (c) Each cell of this table comes from a different regression equation. (d) Food intake index is the first principal component of the protein and carbohydrate intake variables and normalized it to have zero mean and one standard deviation. Similarly, protein and carbohydrate indices are created with protein and carbohydrate intake variables, respectively. (e) Control variables are household head's age, head is female, head is literate, head is wage-earner, head is self-employed, family size, income per capita, asset per capita, have a loan, household faced a non-health shock, and other members have ADL limitations. (f) Robust standard errors are in parentheses. (g) Significance: ***p<0.01, **p<0.05, *p<0.1.

IPWRA estimates show that parental illness makes a household four percentage points more likely to be food insecure. Further, these treated households have a 0.09 standard deviation lower food intake. Looking at the individual components of the food intake index in Panel B, I find that parental illness reduces household protein consumption. Particularly, parental illness

reduces the frequency of egg consumption by 0.175 days a week– an 11 percent reduction from the weekly average of 1.6 days. Moreover, parental illness reduces meat consumption by 0.084 days a week– a 27.4 percent reduction from the weekly average of 0.32 days. However, I find parental illness does not affect the frequency of carbohydrate (i.e., cereal, wheat, and potato) consumption. Evidence from experimental studies shows that protein consumption is an important predictor of child height in Bangladesh (Dasgupta 2016; Mahfuz et al. 2019).

Table 11: Parental Input Mechanism of Treatment Effect

Outcome Variables	Mean at the Baseline	OLS Without Control	OLS With Controls	IPWRA
	(1)	(2)	(3)	(4)
Panel A: Health inputs				
Child is breastfeeding (=1)	0.969	0.005	0.003	0.002
		(0.016)	(0.016)	(0.016)
Total vaccination given to a child	6.204	-0.100	-0.051	0.101
		(0.255)	(0.260)	(0.275)
Number of antenatal visits	2.245	-0.070	0.032	0.016
		(0.201)	(0.193)	(0.193)
Delivery at health facility (=1)	0.237	-0.018	0.002	0.007
		(0.040)	(0.039)	(0.039)
Child given vitamin-A (=1)	0.681	0.001	-0.002	0.001
		(0.042)	(0.043)	(0.043)
Mother took iron tablet (=1)	0.534	-0.010	-0.001	-0.008
		(0.043)	(0.043)	(0.044)
Health input index	0.000	-0.039	-0.003	-0.005
		(0.085)	(0.085)	(0.087)
Observations		727	727	727
Panel B: Childcare input				
Childcare (minutes/day)	86.078	12.417	15.32**	14.986**
		(7.645)	(7.596)	(7.582)
Observations		1,074	1,074	1,074

Notes: (a) This table presents the average treatment effect on the treated (ATT) based on Ordinary Least Squares (OLS) and Inverse-probability-weighted Regression Adjustment (IPWRA) methods. (b) The treatment variable is the parental illness that equals one if parents have any ADL limitation, and zero otherwise. (c) Each cell of this table comes from a different regression equation. (d) Health input index is the first principal component of the six parental health investment variables (i.e., breastfeeding, vaccination, antenatal visits, delivery at a facility, vitamin A, and iron tablet), and normalized it to have zero mean and one standard deviation. (e) Childcare data is available only for parents who are household head or spouse of the head. (f) Control variables are household head's age, head is female, head is literate, head is wage-earner, head is self-employed, family size, income per capita, asset per capita, have a loan, household faced a non-health shock, and other members have ADL limitations. (g) Child inputs are available for children up to 24 months. (h) Robust standard errors are in parentheses. (i) Significance: ***p<0.01, **p<0.05, *p<0.1.

Along with creating financial distress, parental illness may make parents physically impaired from making prenatal and postnatal healthcare investments such as antenatal doctor visits, breastfeeding, and vaccination. Besides, parental illness may affect child health by lowering

the time spent with children. I present the effect of parental illness on health inputs and childcare input in Panel A and B of Table 11, respectively.

I consider several health inputs such as whether a child is breastfed, total number of vaccinations, delivery at a health facility, vitamin-A intake, mother took iron tablet, and mother took a calcium supplement. However, I do not find evidence that parental illness significantly reduces investment in child health inputs. Similarly, I do not find evidence of parental illness reducing the time spent with children. Instead, ill parents spend more time with children. As a result, parental investment in health and childcare inputs does not explain the lower child height.

7.2 Early Life Stress

The second channel I explore is early life stress. Parental illness may directly affect children's health through living in a stressful environment (Mühlenweg, Westermaier, and Morefield 2016; Aaskoven, Kjær, and Gyrd-Hansen 2022). Studies show that parental illness is associated with children's low emotional, social, and physical functioning (Visser et al., 2004). Moreover, studies suggest that early life stress leads to a lower child height due to activation of the hypothalamic-pituitary-adrenal (HPA) axis and inhibition of pituitary growth hormone (GH) release (Denholm, Power, and Li 2013; Hulanicka, Gronkiewicz, and Koniarek 2001; Li, Manor, and Power 2004; Montgomery, Bartley, and Wilkinson 1997; Chrousos and Gold 1992; Pears and Fisher 2005).

While I do not have direct measures of stress, stress appears to increase the probability of some disease conditions (Pohl, Medland, and Moeser 2015; Rosa, Lee, and Wright 2018; Taylor 2010). The survey collects data on three disease conditions – fever, cough, and diarrhea – in the last two weeks, which have been linked with stress. Child health conditions are available for only children up to 24 months. I also create a standardized measure – child health condition index– by taking the first principal component of three child health indicators and normalized it to have zero mean and one standard deviation.

The IPWRA estimates in Table 12 show that parental illness increases the likelihood of having a disease condition, but the treatment effect is only statistically significant for diarrhea. I also find that parental illness increases the child health condition index by 0.12 standard deviations, but the effect is not statistically significant. The imprecise result makes it difficult to assert a conclusive argument. Furthermore, while the absence of an effect would have suggested that

stress is not important, the presence of an effect on these outcomes might be driven by some other mechanism.

Table 12: Early Life Stress Mechanism of Treatment Effect

Outcome Variables	Mean at the Baseline	OLS Without Control	OLS With Controls	IPWRA
	(1)	(2)	(3)	(4)
Had fever in last two weeks (=1 if yes)	0.349	0.017	0.023	0.023
		(0.045)	(0.045)	(0.046)
Had cough in last two weeks (=1 if yes)	0.391	0.054	0.070	0.066
		(0.045)	(0.045)	(0.045)
Had diarrhea in last two weeks (=1 if yes)	0.254	0.036	0.044*	0.045*
		(0.025)	(0.026)	(0.025)
Child health condition index	0.000	0.098	0.127	0.122
		(0.088)	(0.090)	(0.089)
Observations		685	685	685

Notes: (a) This table presents the average treatment effect on the treated (ATT) based on Ordinary Least Squares (OLS) and Inverse-probability-weighted Regression Adjustment (IPWRA) methods. (b) The treatment variable is the parental illness that equals one if parents have any ADL limitation, and zero otherwise. (c) Each cell of this table comes from a different regression equation. (d) Outcome variables are whether the child had a health condition such as fever, cough, or diarrhea in last 2 weeks. Child health conditions are available for children up to 24 months. (e) Child health condition index is the first principal component of the three child health indicators (i.e., fever, cough, and diarrhea), and normalized it to have zero mean and one standard deviation. (f) Control variables are household head's age, head is female, head is literate, head is wage-earner, head is self-employed, family size, income per capita, asset per capita, have a loan, household faced a non-health shock, and other members have ADL limitations. (g) Robust standard errors are in parentheses. (h) Significance: ***p<0.01, **p<0.05, *p<0.1.

7.3 Quality-Quantity Trade-off

There is extensive literature studying the trade-offs between child quantity and quality within a household (Horton 1986; S. E. Black, Devereux, and Salvanes 2005; Millimet and Wang 2011; Peters, Rees, and Hernández-Julián 2014). However, Peters, Rees, and Hernández-Julián (2014) do not find evidence of a trade-off between child quantity and child health in rural Bangladesh.[18] If parents with ADL limitations decide to have fewer children, their relatively small family size may lead to higher investment in children and improved child health outcomes. In Table 13, I test whether parental illness affects their fertility choice (i.e., the number of children). If parents alter their fertility choice to benefit the existing children, we will find a negative effect of parental illness on the total number of children in 2015. The IPWRA estimate in column (3) shows that the coefficient is positive and statistically

[18] Similarly, Mccarthy and Pearlman (2022) do not find evidence of a trade-off between child quantity and child education in rural Bangladesh.

insignificant. The result shows that parental illness does not affect their fertility choice, which is consistent with the finding of Peters, Rees, and Hernández-Julián (2014) in rural Bangladesh.

Table 13: Effect of Illness on Fertility Choice

Outcome Variable	OLS Without Control	OLS With Controls	IPWRA
	(1)	(2)	(3)
Total number of children	0.166**	0.025	0.016
	(0.068)	(0.057)	(0.059)
Mean at the Baseline		2.171	
Control	No	Yes	Yes
Observations	1,416	1,416	1,416

Notes: (a) This table presents the average treatment effect on the treated (ATT) based on Ordinary Least Squares (OLS) and Inverse-probability-weighted Regression Adjustment (IPWRA) methods. (b) The treatment variable is the parental illness that equals one if parents have any ADL limitation, and zero otherwise. (c) The outcome variable is the number of children in 2015. (d) Control variables are household head's age, head is female, head is literate, head is wage-earner, head is self-employed, family size, income per capita, asset per capita, have a loan, household faced a non-health shock, and other members have ADL limitations. (e) Robust standard errors are in parentheses. (f) Significance: ***p<0.01, **p<0.05, *p<0.1.

8. Other Health Outcome

As discussed in section 7.1, parental illness cause households to reduce consumption of protein without significantly affecting carbohydrate consumption. Such a diet may help sustain a child's weight but cause severe damage to a child's growth potential. Experimental studies show carbohydrate intake is an important predictor of child weight (Kirk et al. 2012; Sondike, Copperman, and Jacobson 2003), whereas protein consumption is an important predictor of child height (Das et al. 2020; Mahfuz et al. 2019). Studies also show that the prevalence of overweight (i.e., obesity) is higher among individuals with poverty and food insecurity (Drewnowski and Specter 2004; Griffith 2022). As a result, child weight may not capture the effect of parental health shocks. I test this hypothesis in this section and present the result in Table 15.

The treatment variable is parental illness, and outcome variable is child weight-for-age z-score. Columns (1) and (2) present the OLS estimates with and without the covariates, and column (3) presents the ATT estimates of the IPWRA. The coefficients of all three specifications are close to zero and statistically insignificant. These results suggest that parental illness does not affect child weight. As argued above, this result is not surprising as the households adopt daily diets that are weight-sustaining.

Table 14: Effect of Parental Illness on Child Weight

	OLS Without Control	OLS With Controls	IPWRA
	(1)	(2)	(3)
Parental illness (=1 if yes)	-0.029	0.010	0.002
	(0.064)	(0.061)	(0.061)
Control	No	Yes	Yes
Observations	1,740	1,740	1,740

Notes: (a) This table presents the average treatment effect on the treated (ATT) based on Ordinary Least Squares (OLS) and Inverse-probability-weighted Regression Adjustment (IPWRA) methods. (b) The treatment variable is the parental illness that equals one if parents have any ADL limitation, and zero otherwise. (c) The outcome variable is the children's weight-for-age z-score. (d) Control variables are household head's age, head is female, head is literate, head is wage-earner, head is self-employed, family size, income per capita, asset per capita, have a loan, household faced a non-health shock, other members have ADL limitations, and child's age, gender, and birth order. (e) Robust standard errors are in parentheses. (f) Significance: ***p<0.01, **p<0.05, *p<0.1.

9. Conclusion

Major illnesses are quite prevalent, unpredictable, and costly events that cause a substantial burden to the household in Bangladesh. Substantial medical expenditure combined with reduced labor supply arising from the major illness of parents can severely affect children's human capital accumulation. Although a handful of studies find a negative effect of parental illness on children's educational outcomes (Sun and Yao 2010; Bratti and Mendola 2014; Alam 2015; Dhanaraj 2016; Lim 2020; Aaskoven, Kjær, and Gyrd-Hansen 2022), causal evidence on child health outcomes is nonexistent. To fill the gap in the literature, I provide causal estimates on how parental illness affects child health outcomes.

I find that parental illness has a significant negative effect on child height. The effect size is similar to children experiencing large shocks such as drought or civil war. I also find that the usual shock mitigation strategies such as within-household safety net and informal health care financing strategies such as selling assets and borrowing are ineffective in protecting children. Furthermore, I show that removing the effects of parental illness would close 3.5% of the gap in height between Bangladeshi children and the global average.

The findings of this study have important policy implications as child growth deprivation has a significant negative effect on child survival, cognitive development, and adult-life outcomes such as earnings (Almond, Currie, and Duque 2018; Case, Fertig, and Paxson 2005; Currie and Vogl 2013; Smith 2009; Steckel 1995). Furthermore, in revealing the ineffectiveness of informal shock mitigation strategies, this study highlights the importance of designing and implementing formal safety net mechanisms to protect children. Protecting children against

parental illness is critical in achieving global sustainable development goals (SDGs), particularly 1 (no poverty), 2 (zero hunger), and 3 (good health and well-being).

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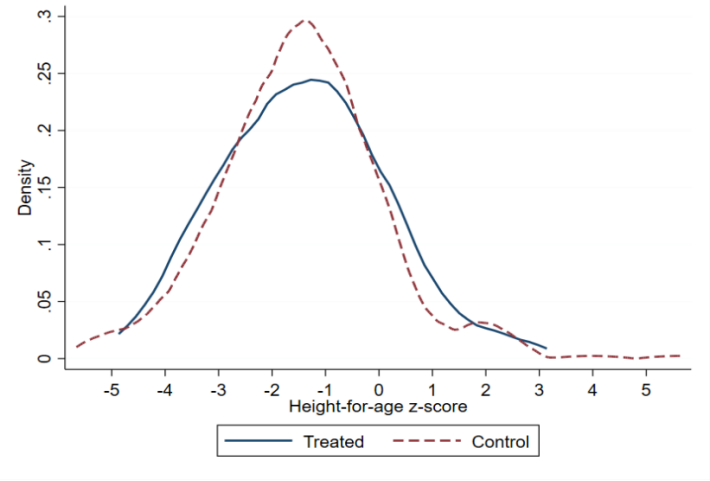
Appendix:

Panel A. Kernel density of HFA z-score in 2012



Panel B. Kernel density of HFA z-score in 2015

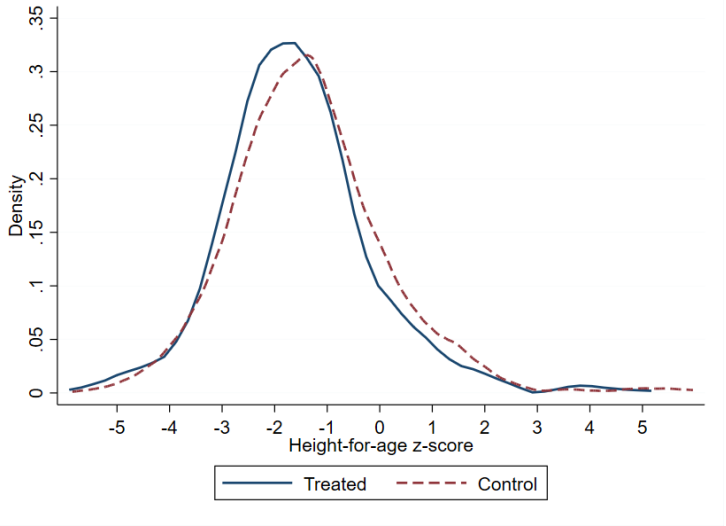


Figure A1: Child Health Outcome of Treated and Untreated Groups

Table A1: Kolmogorov-Smirnov (K-S) Tests of the Equality of Distributions

	2012		2015	
	Difference	p-value	Difference	p-value
	(1)	(2)	(3)	(4)
Untreated group contains smaller values than treated group	0.073	0.313	0.005	0.993
Untreated group contains larger values than treated group	-0.036	0.756	-0.088	0.006
Combined K-S	0.073	0.607	0.088	0.012

Notes: (a) The two-sample Kolmogorov-Smirnov (K-S) test determines whether there are any differences in the distribution of child height-for-age (HFA) z-score for treated and untreated groups. Null hypotheses: equality of the distributions of child height-for-age for treated and untreated groups. Alternative hypothesis: the distributions are different for treated and untreated groups. (b) The largest difference between the distribution functions is 0.073 in 2012 and the approximate asymptotic p-value for this is 0.603. (c) The largest difference between the distribution functions is 0.088 in 2015 and the approximate asymptotic p-value for this is 0.012.

Table A2: Baseline Covariate Balance After Matching

	MDM			PSM		
	Treated	Control	Normalized Difference	Treated	Control	Normalized Difference
	(1)	(2)	(3)	(1)	(2)	(3)
Household head's characteristics						
Head's age	38.943	39.627	-0.048	38.870	39.879	-0.071
Head is female (=1)	0.092	0.092	0.000	0.092	0.092	0.000
Head is illiterate (=1)	0.534	0.542	-0.017	0.533	0.544	-0.021
Head is wage-earner (=1)	0.123	0.123	0.000	0.123	0.123	0.000
Head is self-employed (=1)	0.773	0.771	0.004	0.775	0.783	-0.018
Household characteristics						
Log (family size)	1.481	1.481	0.000	1.479	1.479	0.000
Log (income/capita)	8.918	9.042	-0.053	8.916	8.940	-0.010
Log (asset/capita)	8.525	8.553	-0.021	8.525	8.538	-0.009
Loan (=1)	0.251	0.251	0.000	0.249	0.249	0.000
Non-health shocks (=1)	0.116	0.116	0.000	0.116	0.116	0.000
Other members' ADL limitation (=1)	0.284	0.284	0.000	0.282	0.282	0.000
Number of observations	1,185	423	1,608	1,180	422	1,602

Notes: (a) This table shows the covariate balance between the treated (ADL limitation) and the control group (no ADL limitation) before and after the matching. (b) All covariate values are at the baseline. (c) Matching uses Multivariate (i.e., Mahalanobis) distance matching (MDM) with kernel algorithm.

Table A3: Effect of Parental Illness on Child Height (Using Alternative Definitions)

	Low ADL Limitation			High ADL Limitation		
	OLS Without Control	OLS With Controls	IPWRA	OLS Without Control	OLS With Controls	IPWRA
	(1)	(2)	(3)	(4)	(5)	(6)
Parental illness (=1 if yes)	-0.232***	-0.165**	-0.153*	-0.201	-0.205*	-0.216*
	(0.082)	(0.081)	(0.081)	(0.135)	(0.121)	(0.123)
Control	No	Yes	Yes	No	Yes	Yes
Observations	1,567	1,567	1,567	1,471	1,471	1,471

Notes: (a) This table presents the average treatment effect on the treated (ATT) based on Ordinary Least Squares (OLS) and Inverse-probability-weighted Regression Adjustment (IPWRA) methods. (b) The treatment variable in Columns 1-3 is the parental illness that equals one if parents have low ADL limitation, and zero otherwise. The treatment variable in Columns 4-6 is the parental illness that equals one if parents have high ADL limitation, and zero otherwise. (c) The outcome variable is the children's height-for-age z-score. (d) Control variables are household head's age, head is female, head is literate, head is wage-earner, head is self-employed, family size, income per capita, asset per capita, have a loan, household faced a non-health shock, other member have ADL limitations, and child's age, gender, and birth order. (e) Robust standard errors are in parentheses. (f) Significance: ***p<0.01, **p<0.05, *p<0.1.

Table A4: Kolmogorov-Smirnov (K-S) Tests of the Equality of Distributions (Head Parents' Illnesses)

	2012		2015	
	Difference	p-value	Difference	p-value
	(1)	(2)	(3)	(4)
Untreated group contains smaller values than treated group	0.080	0.303	0.009	0.960
Untreated group contains larger values than treated group	-0.051	0.620	-0.095	0.012
Combined K-S	0.080	0.589	0.095	0.024

Notes: (a) The two-sample Kolmogorov-Smirnov (K-S) test determines whether there are any differences in the distribution of child height-for-age (HFA) z-score for treated and untreated groups. Null hypotheses: equality of the distributions of child height-for-age for treated and untreated groups. Alternative hypothesis: the distributions are different for treated and untreated groups.

Table A5: Kolmogorov-Smirnov (K-S) Tests of the Equality of Distributions (Father's and Mother's Illnesses)

	2012		2015	
	Difference	p-value	Difference	p-value
	(1)	(2)	(3)	(4)
Panel A: Father's Illness				
Untreated group contains smaller values than treated group	0.152	0.384	0.019	0.951
Untreated group contains larger values than treated group	-0.152	0.384	-0.137	0.081
Combined K-S	0.152	0.725	0.137	0.161
Panel B: Mother's Illness				
Untreated group contains smaller values than treated group	0.121	0.107	0.022	0.782
Untreated group contains larger values than treated group	-0.005	0.996	-0.068	0.101
Combined K-S	0.121	0.214	0.068	0.201

Notes: (a) The two-sample Kolmogorov-Smirnov (K-S) test determines whether there are any differences in the distribution of child height-for-age (HFA) z-score for treated and untreated groups. Null hypotheses: equality of the distributions of child height-for-age for treated and untreated groups. Alternative hypothesis: the distributions are different for treated and untreated groups.