Parental Illness Shocks and Child Health in Bangladesh

Md Shahadath Hossain* and Shaila Nazneen**

January 03, 2024

Abstract

This study examines the impact of parental illness on child health in rural Bangladesh. Using a set of health conditions that are as good as random, we find that parental illness has a significant negative effect on child height. Both Fathers' and mothers' illnesses exhibit equally detrimental effects. Exploring potential mechanisms, we find that parental illness induces financial distress, characterized by increased medical spending, diminished assets, and increased borrowing. Consequently, parents respond by substantially reducing resource allocation, manifested through decreased food intake and protein consumption. The findings of this study carry important policy implications, as mitigating the effects of parental illness could close 3.5% of the height gap between Bangladeshi children and the global average.

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

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

*Corresponding Author, Department of Economics, University of Houston, Houston, TX 77004, USA. E-mail: mhossai7@central.uh.edu.
**Environmental Science and Engineering Program, University of Texas at El Paso, El Paso, TX 79968, USA. E-mail: snazneen@miners.utep.edu

We thank Solomon Polachek, David Slichter, Subal Kumbhakar, Plamen Nikolov, Jonathan Scott, Sulagna Mookerjee, Harounan Kazianga, Elisa Taveras, Adesola Sunmoni, Jon Mansfield, Richard Daramola, Hoa Vu, Xianhua (Emma) Zai as well as the participants at the PhD seminar at Binghamton University for constructive feedback and helpful comments. Any errors in this article are our own.

1. Introduction

Child health is a crucial component of human capital development, and poor health outcomes in childhood can have long-lasting effects on educational attainment, economic productivity, and overall well-being in adulthood (Almond, Currie, and Duque, 2018; Currie and Vogl, 2013). In developing countries, where health infrastructure is weak and access to quality healthcare is often limited, child health is particularly vulnerable to various risk factors, including parental illness. Understanding the impact of parental illness on child health is crucial for informing policies and interventions aimed at improving child health outcomes in this population. However, evidence on the causal effect of parental illness on child health in a low-income context is scarce.

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 expenditures. Parental illness may also affect children's health through living in a stressful environment (Mühlenweg, Westermaier, and Morefield, 2016). Medical research indicates 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; Li, Manor, and Power, 2004).

In this study, we measure how parental illness affects child health in Bangladesh. Specifically, we investigate the impact of major illnesses of parents on under-five children's height. Child malnutrition and stunting are major concerns for Bangladesh, where 28% of children under five were two standard deviations below the World Health Organization (WHO) growth standards in 2019 (World Bank, 2022). Improving child nutrition and growth can significantly improve child survival, cognitive development, and future earnings (Almond, Currie, and Duque, 2018; Currie and Vogl, 2013).

The treatment variable in this study is major parental (i.e., father or mother, or both) illness, defined as the limitation in activities of daily living (ADLs). More specifically, we created an indicator variable "ADL limitation" if parents have at least some difficulties in walking, sitting, or carrying weight.[1] ADL limitation is a reliable indicator of long-term health status and reflects

[1] Although it would be ideal to know the specific cause of ADL limitation, it is not possible to distinguish specific health events as the underlying dataset lacks information on specific illnesses. However, a nationally representative survey indicates that falls, cuts, road traffic injuries, blunt object injuries, burns, and animal injuries collectively contribute to approximately 85% of all injury morbidities in Bangladesh leading to ADL limitation (Rahman et al., 2016).

unpredictable major illnesses (Crespo and Mira, 2014; Genoni, 2012; Gertler and Gruber, 2002). We started with a pool of healthy parents (i.e., no ADL limitation) at the baseline in 2012.[2] Parents who developed ADL limitations between 2013 and 2015 form the treatment group, and parents who remained healthy formed the control group.

We measured 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 our identification strategy is that, at the baseline, the distribution of child height is not statistically different between treated and control groups. We also show that the baseline covariates between the treatment and control groups are quite similar. Moreover, we find no significant difference in the likelihood of other household members developing an ADL limitation between treatment and control groups in 2015, 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). Furthermore, we show that the development of ADL limitation is uncorrelated with the pre-treatment health conditions such as acute and chronic conditions. As a result, for a confounder to cause bias in our 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 hard to imagine what such a confounder might be.

This finding suggests that the treatment assignment (i.e., parental illness shocks) is minimally confounded or as good as random. However, to help address any remaining endogeneity concern, we control for pre-treatment family characteristics and child health outcome using doubly robust estimation (Bang and Robins, 2005; Imbens and Wooldridge, 2009; Wooldridge, 2007, 2010). We obtain very similar results using ordinary least squared (OLS) with and without covariates, or using a doubly robust estimator.

The result shows that parental illness reduced child height by 22% of a standard deviation.[3] This effect size is comparable to children experiencing relatively large shocks, e.g., crop failure

[2] This study uses nationally representative panel data from rural Bangladesh – the Bangladesh Integrated Household Survey (BIHS). Households from 325 randomly selected villages were surveyed in 2011-12 and followed up in 2015.

[3] This result is robust to alternative definition of parental illness shocks.

(0.17 SDs) and drought (0.21 SDs) in Ethiopia (Akresh, Verwimp, and Bundervoet, 2011; Hirvonen, Sohnesen, and Bundervoet, 2020). 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. We find fathers' and mothers' illnesses have equally detrimental effects on child height. In addition, we find no heterogeneous effects by child age, gender, or birth order.

Next, we turn to understanding the mechanism. The first channel we consider is parental resource allocation. Existing empirical evidence highlights the crucial role of within-household resource allocation in determining child height (Jayachandran and Pande, 2017; Rosenzweig and Schultz, 1982). Parental illness could cause financial distress due to decreased labor supply and productivity and increased medical spending (Alam, 2015; Gertler and Gruber, 2002; Schultz and Tansel, 1997). In our data, we find that parental illness significantly reduced time allocation for both domestic work and outside work, increased medical spending, decreased assets, and increased borrowing. Furthermore, we find that parental illness significantly increased food insecurity, decreased food intake, and reduced protein consumption.

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; Currie and Vogl, 2013). Second, it contributes to the literature on adverse shocks and child health in a low-income context. Despite the prevalence of parental illnesses in developing countries, this is one of the first studies to estimate the causal effect of parental illness on child health in this population.

The rest of the paper is structured as follows. Section 2 provides the context of the study. Section 3 describes the data and method. Section 4 presents the main estimation result, robustness check, heterogeneity analysis, and mechanisms of the effect. Section 5 presents the result for another health outcome and Section 6 concludes the paper.

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 % 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%), iron (51%), and zinc (45%) ("Global Nutrition Report 2021", 2022).

Child malnutrition is present in all geographic regions and economic groups in Bangladesh. However, children living in rural areas are more likely to be stunted (28.4%), compared to their counterparts in urban areas (26.3%). Similarly, stunting is more prevalent among the lowest wealth quintile (38%) as compared to the highest wealth quintile (20%). However, stunting is equally prevalent among male and female children – 28% and 27.9%, 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% 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 (Currie and Vogl, 2013).

2.2 Illness Shocks

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

Health insurance is virtually nonexistent in Bangladesh. As a result, out-of-pocket payments are the primary way to finance healthcare in Bangladesh. About 16% 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).

Out-of-pocket healthcare payments cause a financial burden to households. Around 4.50% 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% 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. Data and Method

3.1 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% 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 (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 (Genoni, 2012; Gertler and Gruber, 2002). This is because ADL limitation questions are more specific and objective and, as a result, are less prone to measurement errors.[5]

[4] We empirically investigate whether ADL limitation is difficult to predict in Section 4.2. We also empirically investigate whether ADL limitation causes financial hardship in Section 4.3.

[5] Health literature suggests that reporting of ADL limitation is less prone to measurement errors, given the specificity and objectivity of the questions, such as assessing the ability to walk, sit or lift weight.

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 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.[6]

The baseline sample consists of all healthy parents (i.e., no ADL limitations) in 2011-12.[7] The treatment group consists of parents who developed ADL limitations between 2013 and 2015, and the control group consists of parents who remained healthy.[8] About 25.3% of the children in our sample are in the treatment group, and 74.7% are in the control group. Although it would be ideal to know the specific cause of ADL limitation, it is not possible to distinguish specific health events as the underlying dataset lacks information on specific illnesses. However, a nationally representative survey indicates that falls, cuts, road traffic injuries, blunt object injuries, burns, and animal injuries collectively contribute to approximately 85% of all injury morbidities in Bangladesh leading to ADL limitation (Rahman et al., 2016).

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

[6] I check the sensitivity of my results using alternative treatment definitions – low ADL limitation and high ADL limitation – in Section 4.1. 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.

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

[8] We excluded the 2018 survey round due to estimation challenges arising from the utilization of three rounds of data. The possibility of treated parents transitioning to a healthy state and new parents developing ADL limitation in 2018 introduces the issue of treatment switching on and off. Furthermore, combining early treatment (2013-2015) and late treatment (2016-2018) groups may introduce treatment heterogeneity, leading to negative weighing problems as discussed in recent Difference-in-Differences (DiD) literature. Additionally, given our focus on the health outcomes of children under the age of 5, the natural aging process over the 7-year span between 2012 and 2018 would result in different sets of parents and children, introducing further complexities in the analysis.

attainment (Currie, 2009), occupational choice (Case, Paxson, and Islam, 2009), and labor market outcomes (Persico, Postlewaite, and Silverman, 2004).

We 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 (Jayachandran and Pande, 2017; Hirvonen, Sohnesen, and Bundervoet, 2020).[9]

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 our 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.[10]

The covariates we include are pretreatment child height-for-age z-score, 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 and Wooldridge (2009), we use a normalized difference of covariates between the treatment and control groups.[11] 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 0.16. This suggests that the baseline

[9] Anthropometric measurements, such as child height, taken by trained field workers are less prone to measurement error compared to relying on mothers' self-reported data.

[10] Similarly, child height-for-age (HFA) z-score in 2015 is -1.40.

[11] 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, $t-$ statistic $= (\bar{x}_{Treated} - \bar{x}_{Control})/\sqrt{(s^2_{Treated}/N_{Treated} + s^2_{Control}/N_{Control})}$.

covariates between treated and control groups are quite similar. The pre-treatment average HFA z-scores for treatment and control groups children are -1.46 and -1.37, respectively. The normalized difference in child height is only 0.06.[12] As a result, the data suggests that child height between the treated and the control group is balanced at the baseline.

Table 1: Baseline Covariate Balance

	Control	Treated	Normalized Difference
	(1)	(2)	(3)
Pre-treatment Outcome Variable			
Height-for-age Z-score	-1.457	-1.372	0.056
	(0.077)	(0.125)	
Pre-treatment Household Head's Characteristics			
Head's Age	39.989	39.25	-0.052
	(14.556)	(13.967)	
Head Female (=1 if yes)	0.111	0.107	-0.013
	(0.314)	(0.309)	
Head Literate (=1 if yes)	0.546	0.523	0.047
	(0.498)	(0.500)	
Head Wage-earner (=1 if yes)	0.163	0.132	-0.088
	(0.370)	(0.339)	
Head Self-employed (=1 if yes)	0.704	0.757	0.119
	(0.457)	(0.429)	
Pre-treatment Household Characteristics			
Log (Family Size)	1.438	1.496	0.157
	(0.365)	(0.374)	
Log (Income/Capita)	8.906	8.872	-0.014
	(2.373)	(2.273)	
Log (Asset/Capita)	8.580	8.534	-0.035
	(1.357)	(1.298)	
Have Loan (=1 if yes)	0.207	0.264	0.134
	(0.405)	(0.441)	
Non-health Shocks (=1 if yes)	0.115	0.125	0.032
	(0.319)	(0.331)	
Other Members' ADL limitation (=1 if yes)	0.338	0.289	-0.107
	(0.473)	(0.454)	
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.

[12] The sample mean difference test shows that the difference is not statistically significant (*t*-stat = -0.58). Bootstrapping the sample 1000 times to address small sample issue does not change the conclusion (*t*-stat = -0.58). We also conduct a nonparametric test of equality of the distribution of child height using Kolmogorov-Smirnov (K-S) test . The K-S test fails to reject the equality of the distributions of child height-for-age z-score for treated and control groups (*p*-value = 0.61).

3.2 Method

The identification strategy is driven by the key finding that the distribution of pre-treatment child height is not statistically different between treated and control groups. In addition, the pre-treatment covariates are balanced between the treatment and control groups. We can estimate the treatment effect using an ordinary least squared regression with lagged outcome and covariates.[13] The regression equation is as follows:

$$Y_{i,\tau} = \beta_0 + \beta_1 D_{i,\tau} + X'_{i,\tau-1}\,\rho + C'_{i,\tau}\,\alpha + \gamma Y_{i,\tau-1} + \epsilon_{i,\tau} \qquad (1).$$

In Eq (1), $Y_{i,\tau}$ represents the outcome variable for child i in survey round τ, where τ corresponds to the post-treatment survey round in 2015 and $\tau - 1$ corresponds to the pre-treatment survey round in 2011-12. The primary outcome variable of interest is child HFA z-score. The treatment variable $D_{i,\tau}$ is a binary indicator, taking the value of one if child i's parents have an ADL limitation at time τ, and zero otherwise. The coefficient β_1 captures the treatment effect. The vector $X_{i,\tau-1}$ comprises pre-treatment covariates, encompassing household and household head's characteristics (see Table 1 for details). In addition, $C_{i,\tau}$ is a vector of post-treatment child characteristics such as age, gender, and birth order. $Y_{i,\tau-1}$ denotes the pre-treatment outcome variable for child i at time $\tau - 1$. The error term ϵ accounts for the unobserved factors. Standard errors are clustered at the household level as treatment assignment is at that level and some households have more than one child. Furthermore, to address small sample issues, standard errors are bootstrapped with 1,000 replications.

In Eq (1) the identification of treatment effect relies on the assumption that treatment assignment is as good as random. If parents can accurately predict the likelihood of developing an ADL limitation, this assumption is violated. Moreover, if parents can take effective measures (i.e., coping strategies) against a predictable illness, the observed treatment effect may underestimate the actual effect of parental illness. To investigate this, we examine whether parents can use information about their past health conditions – acute or chronic – to predict the likelihood of developing an ADL limitation.[14] The estimation equation for this exercise is as follows:

[13] Assuming that, conditional on the pre-treatment outcome and covariates, both groups would exhibit identical expected outcomes in the absence of treatment.

[14] 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.

$$D_{p,\tau} = \delta_0 + \delta_1 Z_{p,\tau-1} + X'_{i,\tau-1}\,\eta + \varepsilon_{p,\tau} \quad (2)$$

This exercise uses the parents' (p) acute or chronic conditions (Z) at time $\tau - 1$ as treatment variables and parents' ADL limitation (D) at time τ as an outcome variable. Both acute and chronic condition variables are dummy indicators that equal one if parents have a condition at the time τ, and zero otherwise. Similarly, the outcome variable (D) is a dummy indicator that equals one if parents have ADL limitation at time τ, and zero otherwise. $X_{i,\tau-1}$ is the same vector of pretreatment covariates as before capturing household and household head's characteristics. Appendix Table A1 shows that having acute or chronic conditions in the pre-treatment period does not predict the likelihood of developing ADL limitation. This result suggests that the parents' ADL limitation represents an illness shock that is difficult to predict based on past health conditions.[15]

Moreover, treatment assignment is not as good as random if there exist household-level confounders (i.e., treated households live in conditions that are more susceptible to injury and illness). To assess the presence of household-level confounders, we investigate the presence of ADL limitations among other household members in treated households. The regression estimation equation for this analysis is as follows:

$$D_{o,\tau} = \lambda_0 + \lambda_1 D_{p,\tau} + \nu_\tau \quad (3).$$

In this exercise, we use parents' ADL limitation at time τ (D_p) as a treatment variable and other household members' ADL limitation in the same period (D_o) as an outcome variable. Both parents' and other household members' ADL limitations are dummy indicators, equaling one if they have an ADL limitation and zero otherwise. The results indicate that parent's ADL limitation have no significant effect on other household members' having ADL limitations ($\lambda_1 = 0.033, se_{\lambda_1} = 0.029$). Consequently, we argue that treated households are not living in conditions more susceptible to injury and illness.

So far, we have presented three lines of evidence supporting the assertion that treatment assignment (i.e., parents' ADL limitation) is as good as random: (i) balance in outcome and covariates in the pre-treatment period, (ii) the unpredictability of ADL limitation, (iii) the absence of household-level confounders. To introduce bias in our estimates of Eq (1), a

[15] This finding aligns with nationally representative survey statistics, which indicate that the majority of the injury-related morbidities result from unforeseeable accidents such as falls, cuts, road traffic injuries, blunt object injuries, burns, and animal injuries (Rahman et al., 2016).

confounder must be (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.

To address any remaining endogeneity concerns, we use doubly robust estimation (Bang and Robins, 2005; Imbens and Wooldridge, 2009; Wooldridge, 2007, 2010). This approach, based on a selection on observable approach, minimizes bias arising from misspecification by modeling both the outcome and the treatment equations. Importantly, it only requires correct specification in one of the two models to obtain an unbiased effect estimator. Consequently, it provides two opportunities to obtain unbiased estimates when addressing selection issues (Imbens and Wooldridge, 2009; Wooldridge, 2007, 2010). The doubly robust method offers advantages over OLS, including increased robustness to model misspecification and reduced sensitivity to functional form assumptions.

We use a doubly robust estimator known as the inverse-probability weighted regression adjustment (IPWRA) (Imbens and Wooldridge, 2009; Wooldridge, 2007, 2010). IPWRA combines an "inverse-probability weighted (IPW)" estimator with a "regression adjustment (RA)" estimator, effectively modeling both the treatment and outcome. 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.[16] In this study, we follow a three-step procedure to implement the IPWRA estimator, a methodology also employed in several recent empirical studies (for example, see Chhay and Yamazaki, 2021; Tani, Xu, and Zhu, 2021).

Firstly, we estimate the probability of parental illness (i.e., the treatment model) using a logistic regression model. The predicted probabilities, known as propensity scores, are then utilized to compute the inverse probability weights. The covariates included in the logit model are the same as those in Eq (1), excluding child characteristics.

Secondly, leveraging the inverse-probability weights, we fit weighted regression models of child height (i.e., outcome models) for both treatment and control groups to obtain treatment-specific predicted outcomes for each child. The outcome model includes all covariates from Eq (1). It is important to note that if the regression function is correctly specified, the weights

[16] See Appendix B for detail of the IPWRA estimator.

do not affect the consistency of the estimator. However, since the true functional form is rarely known, weighting can help reduce bias (Imbens and Wooldridge, 2009; Tani, Xu, and Zhu, 2021).

Finally, we compute the means of the predicted outcomes for both treatment and control groups. The mean difference between the treatment and control groups provides estimates of the average treatment effect (ATE) of parental illness. Similarly, we 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. ATT estimates are consistent if the models for either the treatment (step 1) or the outcome (step 2) are correctly specified (Imbens and Wooldridge, 2009; Wooldridge, 2007, 2010).

4. Results

4.1 Main Result

This section presents the main estimation result based on Eq (1). Table 2 presents the estimates of parental illness on child height-for-age (HFA) z-score. Columns (1)-(4) present the OLS estimates with and without the covariates, whereas Column (5) presents IPWRA estimates. The treatment effect in Column (1) is -0.22, and statistically significant at the 1% level. Parental illness led to a decrease in child height by 22% of a standard deviation. This point estimate is robust to the inclusion of additional control variables, as shown by the statistically identical coefficients in Column (2)-(4). This finding suggests that the treatment effect is not sensitive to observable covariates, corroborating the claim that parental illness is as good as random, and endogeneity concerns from unobservable confounders are negligible.[17]

The IPWRA estimate in Column (5) is also similar to the OLS estimates, and statistically significant at the 5% level. The IPWRA estimate shows that parental illness led to a 17.2% of a standard deviation lower child height. The overall result suggests that parental illness have a large negative effect on child height and the effect size is comparable to relatively large shocks such as crop failure, drought, and civil war.[18] A back-of-the-envelope calculation shows that

[17] Since the observed covariates do not significantly attenuate the point estimate, it is unlikely that any unobserved confounder will substantially attenuate the treatment effect.

[18] 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 height for exposure to drought in Ethiopia.

removing the effects of parental illness would close 3.5% of the gap in height between Bangladeshi children and the global average.[19]

Table 2: Effect of Parental Illness on Child Height

	(1)	(2)	(3)	(4)	(5)
Parental Illness (=1 if yes)	-0.220***	-0.178**	-0.175**	-0.170**	-0.177**
	(0.082)	(0.078)	(0.074)	(0.074)	(0.073)
Control: Pre-treatment Covariates	No	Yes	Yes	Yes	Yes
Control: Child Characteristics	No	No	Yes	Yes	Yes
Control: Pre-treatment Outcome	No	No	No	Yes	Yes
Estimation Approach	OLS	OLS	OLS	OLS	IPWRA
Observations	1,740	1,740	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). (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) Pre-treatment covariates are household head's age, head is female, head is literate, head is wage-earner, head is self-employed, family size (in log scale), income per capita, asset per capita, have a loan, household faced a non-health shock, other members have ADL limitations. Contemporaneous child characteristics include child's age, gender, and birth order. Pre-treatment outcome variable is child height-for-age z-score measured at the baseline. (e) Bootstrapped (1000 replications) standard errors are clustered at the household level and presented in parentheses. (f) Significance: ***p<0.01, **p<0.05, *p<0.1.

We assess the robustness of our primary results by examining alternative definitions of the treatment variable – low and high ADL limitations. The low ADL limitation 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 results presented in Appendix Table A2 show that increased ADL limitation corresponds to a more pronounced decline in child height. Notably, these estimates are not statistically significantly different from our main estimates.

4.2 Heterogeneity

In this subsection, we explore the heterogeneity of treatment effects by characteristics of parents and children. First, we investigate how treatment effects vary by illness shocks to fathers and mothers.[20] Then, we examine whether the effect of parental illness varies with a child's age, gender, and birth order. Studies have highlighted the presence of differential parental investments and height outcomes based on various child characteristics (Barcellos, Carvalho, and Lleras-Muney, 2014; Jayachandran and Pande, 2017).

[19] This number is computed 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.
[20] Studies show that illnesses of father and mother have a differential effect on children's educational outcomes (Alam, 2015).

4.2.1 Heterogeneity by Illness of Father and Mother

Table 3 investigates whether illnesses of fathers and mothers have differential effects on child height.[21] Panel A and Panel B present the ATT estimates of fathers' and mothers' illnesses on child height, respectively. The estimates in Column (3) show that father's illness reduced child height by 12% of a standard deviation, while mother's illness led to an 18% reduction. Although the treatment 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.[22] This result suggests that fathers' and mothers' illnesses have equally detrimental effects on child height.

Table 3: Heterogenous Effect of Father's and Mother's Illness

	(1)	(2)	(3)
Panel A: Father Only			
Father's Illness (=1 if yes)	-0.234	-0.122	-0.127
	(0.178)	(0.174)	(0.174)
Control	No	Yes	Yes
Estimation Approach	OLS	OLS	IPWRA
Observations	1,214	1,214	1,214
Panel B: Mother Only			
Mother's Illness (=1 if yes)	-0.140	-0.179**	-0.181**
	(0.089)	(0.080)	(0.083)
Control	No	Yes	Yes
Estimation Approach	OLS	OLS	IPWRA
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 (in log scale), 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. Pre-treatment child height-for-age z-score is also included as a control. (e) Bootstrapped (1000 replications) standard errors are clustered at the household level and presented in parentheses. (f) Significance: ***p<0.01, **p<0.05, *p<0.1.

4.2.2 Heterogeneity by Child Characteristics

Moving forward, we explore whether the effect of parental illness varies with the age of the children, categorizing them into two groups: 0-24 months and 25-59 months. This categorization aligns with the WHO breastfeeding guidelines and the child growth phase.

[21] we conducted 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 identical.

[22] The overlapping confidence interval suggests that the point estimates are not significantly different from each other.

Appendix Table A3, Panel A and B, show the effect of parental illness on these age groups. The point estimates show parental illness has a similar effect on both age groups, suggesting that parental illness is equally detrimental to all children between 0 and 5 years of age.

In Appendix Table A4, we investigate whether parental illness leads to differential height outcomes based on the child's gender. Panels A and B show the estimates of parental illness for boys and girls, respectively. We find parental illness led to a relatively larger height loss for boys. However, the ATT estimate for girls is not statistically different from boys.

Finally, we explore how parental illness affects children of different birth orders in Appendix Table A5. Panels A-C present the estimates for first-born, second-born, and third-or-higher-born children, respectively. The ATT estimates 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 compared to first-born and second-born children.

4.3 Mechanisms

In this section, we turn to understanding the mechanisms. We explore the viability of two channels: parental resource allocation and fertility choice. We briefly discuss these channels and examine their empirical support by re-estimating the main estimation equation with these proposed channels as outcome variables.

4.3.1 Parental Resource Allocation

The primary channel we explore is parental resource allocation. If parental illness induces financial hardship within the household, parents might adjust the allocation of resources, consequently affecting child height. Empirical studies underscore the pivotal role of within-household resource allocation in determining child height (Jayachandran and Pande, 2017; Rosenzweig and Schultz, 1982).

Parental illness can lead to financial distress through reduced labor supply and productivity, coupled with increased medical spending (Alam, 2015; Gertler and Gruber, 2002; Schultz and Tansel, 1997). Particularly in developing countries with less established credit markets and social protection systems, parents may resort to healthcare financing strategies like borrowing and selling productive assets. These strategies may alleviate immediate financial burdens but potentially reduce future earnings and intensify the financial challenges (Islam and Maitra,

2012). Understanding these mechanisms is crucial for devising targeted interventions that mitigate the adverse effects of parental illness on child height.

In Table 4, we 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. Productivity is measured based on parents' time allocation to domestic and outside work. In the survey, the time use data is only available for household head parents. Mothers' 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 over the past 12 months.

Table 4: 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**
		(11.058)	(11.277)	(11.511)
Outside Work (Minutes/Day)	410.97	-53.879**	-47.927**	-47.726**
		(23.602)	(24.896)	(24.157)
Observations		1,074	1,074	1,074
Panel B: Budget Constraint				
Log (Income/Capita)	9.39	0.031	0.031	0.037
		(0.120)	(0.117)	(0.116)
Log (Medical Spending/Capita)	6.43	0.180**	0.247***	0.244***
		(0.082)	(0.079)	(0.083)
Log (Savings/Capita)	6.99	-0.043	-0.024	-0.027
		(0.215)	(0.212)	(0.211)
Log (Asset/Capita)	8.57	-0.120	-0.101*	-0.103*
		(0.080)	(0.061)	(0.059)
Log (Loans/Capita)	8.17	0.437*	0.428**	0.432*
		(0.246)	(0.228)	(0.238)
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 estimation. (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. (f) Control variables are household head's age, head is female, head is literate, head is wage-earner, head is self-employed, family size (in log scale), income per capita, asset per capita, have a loan, household faced a non-health shock, and other members have

ADL limitations. (g) Bootstrapped (1000 replications) standard errors are clustered at the household level and presented in parentheses. (h) Significance: ***p<0.01, **p<0.05, *p<0.1.

The ATT estimates in Panel A of Table 4 show that parental illness reduced time allocation for both domestic work by 23 minutes per day and outside work by 48 minutes per day. The ATT estimates in Panel B show that parental illness led to a 24% higher medical spending per capita, a 10% lower assets per capita, and a 43% higher loans per capita. These findings underscore the substantial financial burden imposed by parental illness, stemming from reduced productivity, increased medical spending, and reliance on informal healthcare financing.

Since parental illness causes a significant financial burden, we explore its potential effect on the quality and quantity of food intake within the household. Table 5 provides insights into two broad indicators: food insecurity (i.e., no food to eat at least for a meal in the past four weeks) and food intake index based on protein and carbohydrate consumption frequency in a week.[23] In Panel A, we observe that parental illness increased the likelihood of a household experiencing food insecurity by 4 percentage points, accompanied by a 9% of a standard deviation reduction in food intake. Analyzing individual components in Panel B, we find that parental illness significantly reduced protein consumption. Particularly, the frequency of egg consumption is reduced by 0.18 days a week, an 11% reduction from the weekly average of 1.6 days. Similarly. meat consumption is reduced by 0.08 days a week, a 25% reduction from the weekly average of 0.32 days. However, carbohydrate (i.e., cereal, wheat, and potato) consumption remained unaffected by parental illness. Evidence from experimental studies highlights the crucial role of protein consumption as an important predictor of child height in Bangladesh (Dasgupta, 2016; Mahfuz et al., 2019).

[23] 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 5: 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 yes)	0.079	0.049**	0.044**	0.044**
		(0.021)	(0.020)	(0.020)
Food Intake index	0.000	-0.112*	-0.086	-0.086
		(0.060)	(0.056)	(0.057)
Observations		1,727	1,727	1,727
Panel B: Individual Components				
Protein Eating Frequency/Week				
Lentil/bean	1.136	-0.068	-0.068	-0.060
		(0.115)	(0.109)	(0.110)
Eggs	1.601	-0.226*	-0.188*	-0.175
		(0.117)	(0.109)	(0.117)
Dairy Products	2.075	-0.130	-0.054	-0.050
		(0.189)	(0.186)	(0.196)
Meat (Beef or Goat)	0.322	-0.084**	-0.081**	-0.084**
		(0.037)	(0.039)	(0.041)
Poultry (Chicken or Duck)	0.444	0.017	0.041	0.040
		(0.063)	(0.064)	(0.062)
Protein Intake Index	0.000	-0.109*	-0.084	-0.081
		(0.063)	(0.056)	(0.054)
Carbohydrates Eating Frequency/Week				
Cereal	0.153	-0.057	-0.041	-0.044
		(0.087)	(0.087)	(0.091)
Wheat Flour	1.091	-0.107	-0.080	-0.097
		(0.138)	(0.134)	(0.137)
Potato	5.209	-0.039	-0.055	-0.050
		(0.104)	(0.105)	(0.105)
Carbohydrate Intake Index	0.000	-0.029	-0.011	-0.018
		(0.062)	(0.064)	(0.064)
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 estimation. (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 (in log scale), income per capita, asset per capita, have a loan, household faced a non-health shock, and other members have ADL limitations. (f) Bootstrapped (1000 replications) standard errors are clustered at the household level and presented in parentheses. (g) Significance: ***p<0.01, **p<0.05, *p<0.1.

Alongside the financial strain, parental illness might 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.[24] We examine the effect of parental illness on health inputs and childcare in Panel A and B of Appendix Table A6, respectively.[25] However, we do not find evidence supporting a significant reduction in parental investment in child health inputs or a decrease in time spent with children due to parental illness.[26] Consequently, these factors do not appear to explain the observed decline in child height associated with parental illness.

4.3.2 Quality-Quantity Trade-off

Extensive literature explores the trade-offs between child quantity and quality within households (S. E. Black, Devereux, and Salvanes, 2005; Millimet and Wang, 2011; Peters, Rees, and Hernández-Julián, 2014). If parents facing ADL limitations choose to have fewer children, their relatively small family size may lead to increased investment in children and improved child health outcomes. In Appendix Table A7, we investigate whether parental illness influences their fertility choices measured by the number of children.

If parental illness prompted adjustments in fertility choices favoring existing children, we would expect a significant negative effect on the number of children. However, the ATT estimates show positive or statistically insignificant small negative effects, suggesting that parental illness did not influence fertility decisions. This aligns with the findings of Peters, Rees, and Hernández-Julián (2014) in rural Bangladesh, providing further support that, in this context, fertility choice adjustment does not seem to account for the observed decline in child height associated with parental illness.

5. Other Health Outcome

We have previously observed that parental illness causes households to reduce protein consumption without significantly affecting carbohydrate consumption. Such a dietary shift may sustain weight but could severely impede a child's growth potential. Experimental studies

[24] Information on prenatal and postnatal inputs is only available for children under the age of 2 years, whereas parental time allocation is available for only household head parents. As a result, the number of observations in this analysis is smaller than the main estimation sample.
[25] We 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.
[26] Since many health care inputs in Bangladesh are provided by health care workers through free-of-cost home visits, it is not surprising to observe insignificant effects on these inputs.

emphasize the significance of carbohydrate intake in child weight (Kirk et al., 2012; Sondike, Copperman, and Jacobson, 2003), while protein consumption in child height (Das et al., 2020; Mahfuz et al., 2019). Studies also indicate a higher prevalence of obesity among children facing poverty and food insecurity (Drewnowski and Specter, 2004; Griffith, 2022). Consequently, child weight may not capture the effect of parental illness shocks. We test this hypothesis in this section and present the result in Appendix Table A8.

The treatment variable is parental illness, and the outcome variable is child weight-for-age z-score. Columns (1) and (2) present the OLS estimates with and without the covariates, while Column (3) presents the ATT estimates of the IPWRA. In all three specifications, the point estimates are close to zero and statistically insignificant. This finding suggests that parental illness did not have a discernable effect on child weight. As discussed earlier, this result is consistent with the notion that households adopt diets sustaining weight amid the financial strain posed by parental illness.

6. 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. Despite the prevalence and severity of illnesses in developing countries, this is one of the first studies to estimate the causal effect of parental illness on child health in a low-income context.

Our findings reveal a significant negative effect of parental illness on child height, akin to the impact of major shocks such as droughts or civil wars. Notably, conventional shock mitigation strategies such as selling assets and borrowing appear ineffective in safeguarding children from these consequences. Moreover, our analysis indicates that eliminating the effects of parental illness could potentially narrow the height gap between Bangladeshi children and the global average by 3.5%.

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. 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.

Reference

Ahmed, Sayem, Mohammad Wahid Ahmed, Md Zahid Hasan, Gazi Golam Mehdi, Ziaul Islam, Clas Rehnberg, Louis W Niessen, and Jahangir A M Khan. 2022. "Assessing the Incidence of Catastrophic Health Expenditure and Impoverishment from Out-of-Pocket Payments and Their Determinants in Bangladesh: Evidence from the Nationwide Household Income and Expenditure Survey 2016." *International Health* 14 (1): 84–96. https://doi.org/10.1093/inthealth/ihab015.

Akresh, Richard, Philip Verwimp, and Tom Bundervoet. 2011. "Civil War, Crop Failure, and Child Stunting in Rwanda." *Economic Development and Cultural Change* 59 (4): 777–810. https://doi.org/10.1086/660003.

Alam, Shamma Adeeb. 2015. "Parental Health Shocks, Child Labor and Educational Outcomes: Evidence from Tanzania." *Journal of Health Economics* 44 (December): 161–75. https://doi.org/10.1016/j.jhealeco.2015.09.004.

Almond, Douglas, Janet Currie, and Valentina Duque. 2018. "Childhood Circumstances and Adult Outcomes: Act II." *Journal of Economic Literature* 56 (4): 1360–1446. https://doi.org/10.1257/jel.20171164.

Bang, Heejung, and James M. Robins. 2005. "Doubly Robust Estimation in Missing Data and Causal Inference Models." *Biometrics* 61 (4): 962–73. https://doi.org/10.1111/j.1541-0420.2005.00377.x.

Barcellos, Silvia Helena, Leandro S. Carvalho, and Adriana Lleras-Muney. 2014. "Child Gender and Parental Investments in India: Are Boys and Girls Treated Differently?" *American Economic Journal: Applied Economics* 6 (1): 157–89. https://doi.org/10.1257/app.6.1.157.

BBS. 2011. "Report of the Household Income & Expenditure Survey 2010." Dhaka: Bangladesh Bureau of Statistics. http://203.112.218.65:8008/WebTestApplication/userfiles/Image/LatestReports/HIES-10.pdf.

Black, Sandra E., Paul J. Devereux, and Kjell G. Salvanes. 2005. "The More the Merrier? The Effect of Family Size and Birth Order on Children's Education." *The Quarterly Journal of Economics* 120 (2): 669–700. https://doi.org/10.1093/qje/120.2.669.

Case, Anne, and Christina Paxson. 2008. "Stature and Status: Height, Ability, and Labor Market Outcomes." *Journal of Political Economy* 116 (3): 499–532. https://doi.org/10.1086/589524.

Case, Anne, Christina Paxson, and Mahnaz Islam. 2009. "Making Sense of the Labor Market Height Premium: Evidence from the British Household Panel Survey." *Economics Letters* 102 (3): 174–76. https://doi.org/10.1016/j.econlet.2008.12.011.

Chhay, Panharoth, and Koji Yamazaki. 2021. "Rural Electrification and Changes in Employment Structure in Cambodia." *World Development* 137 (January): 105212. https://doi.org/10.1016/j.worlddev.2020.105212.

Crespo, Laura, and Pedro Mira. 2014. "Caregiving to Elderly Parents and Employment Status of European Mature Women." *The Review of Economics and Statistics* 96 (4): 693–709. https://doi.org/10.1162/REST_a_00426.

Currie, Janet. 2009. “Healthy, Wealthy, and Wise: Socioeconomic Status, Poor Health in Childhood, and Human Capital Development.” *Journal of Economic Literature* 47 (1): 87–122. https://doi.org/10.1257/jel.47.1.87.

Currie, Janet, and Tom Vogl. 2013. “Early-Life Health and Adult Circumstance in Developing Countries.” *Annual Review of Economics* 5 (1): 1–36. https://doi.org/10.1146/annurev-economics-081412-103704.

Das, Subhasish, J. Johanna Sanchez, Ashraful Alam, Ahshanul Haque, Mustafa Mahfuz, Tahmeed Ahmed, and Kurt Z. Long. 2020. “Dietary Magnesium, Vitamin D, and Animal Protein Intake and Their Association to the Linear Growth Trajectory of Children from Birth to 24 Months of Age: Results From MAL-ED Birth Cohort Study Conducted in Dhaka, Bangladesh.” *Food and Nutrition Bulletin* 41 (2): 200–210. https://doi.org/10.1177/0379572119892408.

Dasgupta, Shatanjaya. 2016. “Son Preference and Gender Gaps in Child Nutrition: Does the Level of Female Autonomy Matter?” *Review of Development Economics* 20 (2): 375–86. https://doi.org/10.1111/rode.12231.

Denholm, Rachel, Chris Power, and Leah Li. 2013. “Adverse Childhood Experiences and Child-to-Adult Height Trajectories in the 1958 British Birth Cohort.” *International Journal of Epidemiology* 42 (5): 1399–1409. https://doi.org/10.1093/ije/dyt169.

Drewnowski, Adam, and SE Specter. 2004. “Poverty and Obesity: The Role of Energy Density and Energy Costs.” *The American Journal of Clinical Nutrition* 79 (1): 6–16. https://doi.org/10.1093/ajcn/79.1.6.

Genoni, Maria Eugenia. 2012. “Health Shocks and Consumption Smoothing: Evidence from Indonesia.” *Economic Development and Cultural Change* 60 (3): 475–506. https://doi.org/10.1086/664019.

Gertler, Paul, and Jonathan Gruber. 2002. “Insuring Consumption Against Illness.” *American Economic Review* 92 (1): 51–70. https://doi.org/10.1257/000282802760015603.

“Global Nutrition Report 2021.” 2022. https://globalnutritionreport.org/resources/nutrition-profiles/asia/southern-asia/bangladesh/.

Griffith, Rachel. 2022. “Obesity, Poverty and Public Policy.” *The Economic Journal* 132 (644): 1235–58. https://doi.org/10.1093/ej/ueac013.

Hamid, Syed Abdul, Syed M. Ahsan, and Afroza Begum. 2014. “Disease-Specific Impoverishment Impact of Out-of-Pocket Payments for Health Care: Evidence from Rural Bangladesh.” *Applied Health Economics and Health Policy* 12 (4): 421–33. https://doi.org/10.1007/s40258-014-0100-2.

Hirvonen, Kalle, Thomas Pave Sohnesen, and Tom Bundervoet. 2020. “Impact of Ethiopia’s 2015 Drought on Child Undernutrition.” *World Development* 131 (July): 104964. https://doi.org/10.1016/j.worlddev.2020.104964.

Hossain, Md. Shahadath, Malabika Sarker, Shaila Nazneen, Bharati Basu, Fatema Binte Rasul, and Hossain I. Adib. 2019. “How Health Shocks and Its Relationship to Repayments of Loans from Microfinance Institutions Can Affect Migrant Households in Bangladesh.” *Migration and Development* 8 (3): 370–93. https://doi.org/10.1080/21632324.2019.1565088.

Imbens, Guido W., and Donald B. Rubin. 2015. *Causal Inference for Statistics, Social, and Biomedical Sciences: An Introduction.* Cambridge: Cambridge University Press. https://doi.org/10.1017/CBO9781139025751.

Imbens, Guido W., and Jeffrey M. Wooldridge. 2009. "Recent Developments in the Econometrics of Program Evaluation." *Journal of Economic Literature* 47 (1): 5–86. https://doi.org/10.1257/jel.47.1.5.

International Food Policy Research Institute (IFPRI). 2016. "Bangladesh Integrated Household Survey (BIHS) 2015." Harvard Dataverse. https://doi.org/10.7910/DVN/BXSYEL.

Islam, Asadul, and Pushkar Maitra. 2012. "Health Shocks and Consumption Smoothing in Rural Households: Does Microcredit Have a Role to Play?" *Journal of Development Economics* 97 (2): 232–43. https://doi.org/10.1016/j.jdeveco.2011.05.003.

Jayachandran, Seema, and Rohini Pande. 2017. "Why Are Indian Children So Short? The Role of Birth Order and Son Preference." *American Economic Review* 107 (9): 2600–2629. https://doi.org/10.1257/aer.20151282.

Kirk, Shelley, Bonnie Brehm, Brian E. Saelens, Jessica G. Woo, Elizabeth Kissel, David D'Alessio, Christopher Bolling, and Stephen R. Daniels. 2012. "Role of Carbohydrate Modification in Weight Management among Obese Children: A Randomized Clinical Trial." *The Journal of Pediatrics* 161 (2): 320-327.e1. https://doi.org/10.1016/j.jpeds.2012.01.041.

LaLonde, Robert J. 1986. "Evaluating the Econometric Evaluations of Training Programs with Experimental Data." *The American Economic Review* 76 (4): 604–20.

Leonard, William. 1988. "Review of Human Growth: A Comprehensive Treatise (Volumes 2 and 3) Edited by Frank Falkner and James M Tanner." *American Journal of Physical Anthropology* 77: 403–4.

Li, Leah, Orly Manor, and Chris Power. 2004. "Early Environment and Child-to-Adult Growth Trajectories in the 1958 British Birth Cohort." *The American Journal of Clinical Nutrition* 80 (1): 185–92. https://doi.org/10.1093/ajcn/80.1.185.

Magnani, Rich, Lesley Oot, Kavita Sethuraman, Golam Kabir, and Setara Rahman. 2015. "USAID OFFICE OF FOOD FOR PEACE FOOD SECURITY COUNTRY FRAMEWORK FOR BANGLADESH FY 2015–2019." AID-OAA-A-12-00005.

Mahfuz, Mustafa, Mohammed Ashraful Alam, Subhasish Das, Shah Mohammad Fahim, Md Shabab Hossain, William A Petri Jr, Per Ashorn, Ulla Ashorn, and Tahmeed Ahmed. 2019. "Daily Supplementation With Egg, Cow Milk, and Multiple Micronutrients Increases Linear Growth of Young Children with Short Stature." *The Journal of Nutrition*, October, nxz253. https://doi.org/10.1093/jn/nxz253.

Millimet, Daniel L., and Le Wang. 2011. "Is the Quantity-Quality Trade-Off a Trade-Off for All, None, or Some?" *Economic Development and Cultural Change* 60 (1): 155–95. https://doi.org/10.1086/661216.

Mühlenweg, Andrea M., Franz G. Westermaier, and Brant Morefield. 2016. "Parental Health and Child Behavior: Evidence from Parental Health Shocks." *Review of Economics of the Household* 14 (3): 577–98. https://doi.org/10.1007/s11150-015-9284-y.

National Institute of Population Research and Training (NIPORT), and International Centre for Diarrhoeal Disease Research, Bangladesh (icddr,b). 2021. "Bangladesh Adolescent

Health and Wellbeing Survey 2019-20." https://www.data4impactproject.org/publications/bangladesh-adolescent-health-and-wellbeing-survey-2019-20-summary-findings/.

Persico, Nicola, Andrew Postlewaite, and Dan Silverman. 2004. "The Effect of Adolescent Experience on Labor Market Outcomes: The Case of Height." *Journal of Political Economy* 112 (5): 1019–53. https://doi.org/10.1086/422566.

Peters, Christina, Daniel I Rees, and Rey Hernández-Julián. 2014. "The Trade-off between Family Size and Child Health in Rural Bangladesh." *Eastern Economic Journal* 40 (1): 71–95. https://doi.org/10.1057/eej.2012.36.

Rahman, Aminur, Salim Mahmud Chowdhury, Saidur Rahman Mashreky, Michael Linnan, AKM Fazlur Rahman, Mohammad Jahangir Hossain, Tahera Ahmed, Kamran Ul Baset, Abu Talab, and Sakander Ali. "Bangladesh Health and Injury Survey 2016 (BHIS 2016): Summary Report." (2016).

Rosenzweig, Mark R., and T. Paul Schultz. 1982. "Market Opportunities, Genetic Endowments, and Intrafamily Resource Distribution: Child Survival in Rural India." *The American Economic Review* 72 (4): 803–15.

Schultz, T. Paul, and Aysit Tansel. 1997. "Wage and Labor Supply Effects of Illness in Côte d'Ivoire and Ghana: Instrumental Variable Estimates for Days Disabled." *Journal of Development Economics* 53 (2): 251–86. https://doi.org/10.1016/S0304-3878(97)00025-4.

Sondike, Stephen B., Nancy Copperman, and Marc S. Jacobson. 2003. "Effects of a Low-Carbohydrate Diet on Weight Loss and Cardiovascular Risk Factor in Overweight Adolescents." *The Journal of Pediatrics* 142 (3): 253–58. https://doi.org/10.1067/mpd.2003.4.

Tani, Massimiliano, Lei Xu, and Yu Zhu. 2021. "The Impact of an Un(Der)Funded Inclusive Education Policy: Evidence from the 2013 China Education Panel Survey." *Journal of Economic Behavior & Organization* 190 (October): 768–84. https://doi.org/10.1016/j.jebo.2021.08.024.

Waterlow, J. C., R. Buzina, W. Keller, J. M. Lane, M. Z. Nichaman, and J. M. Tanner. 1977. "The Presentation and Use of Height and Weight Data for Comparing the Nutritional Status of Groups of Children under the Age of 10 Years." *Bulletin of the World Health Organization* 55 (4): 489–98.

WHO. 2020. "Children: Improving Survival and Well-Being." WHO Fact Sheets. 2020. https://www.who.int/news-room/fact-sheets/detail/children-reducing-mortality.

———. 2021. "Fact Sheets - Malnutrition." 2021. https://www.who.int/news-room/fact-sheets/detail/malnutrition.

Wooldridge, Jeffrey M. 2007. "Inverse Probability Weighted Estimation for General Missing Data Problems." *Journal of Econometrics* 141 (2): 1281–1301. https://doi.org/10.1016/j.jeconom.2007.02.002.

———. 2010. Econometric Analysis of Cross Section and Panel Data, Second Edition. MIT Press.

World Bank. 2022. "Prevalence of Stunting, Bangladesh." World Bank. 2022. https://data.worldbank.org/indicator/SH.STA.STNT.MA.ZS?locations=BD.

Appendix A:

Table A1: Predicting Parents' ADL Limitation

	Outcome: ADL Limitation in 2015 (=1 if yes)		
	(1)	(2)	(3)
Panel A			
Pre-treatment Acute Condition (=1 if yes)	0.010	-0.004	-0.009
	(0.035)	(0.033)	(0.035)
Control	No	Yes	Yes
Estimation Approach	OLS	OLS	IPWRA
Observations	1,019	1,019	1,019
Panel B			
Pre-treatment Chronic Condition (=1 if yes)	-0.021	-0.033	-0.039
	(0.068)	(0.069)	(0.065)
Control	No	Yes	Yes
Estimation Approach	OLS	OLS	IPWRA
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 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 (in log scale), income per capita, asset per capita, have a loan, household faced a non-health shock, and other members have ADL limitations. (e) Bootstrapped (1000 replications) standard errors are clustered at the household level and presented in parentheses. (f) Significance: ***p<0.01, **p<0.05, *p<0.1.

Table A2: Effect of Parental Illness on Child Height (Using Alternative Definitions)

	Low ADL Limitation			High ADL Limitation		
	(1)	(2)	(3)	(4)	(5)	(6)
Parental Illness (=1 if yes)	-0.232***	-0.175**	-0.166**	-0.201	-0.205	-0.190
	(0.082)	(0.082)	(0.081)	(0.140)	(0.127)	(0.132)
Controls	No	Yes	Yes	No	Yes	Yes
Estimation Approach	OLS	OLS	IPWRA	OLS	OLS	IPWRA
Observations	1,567	1,567	1,567	1,469	1,469	1,469

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 (in log scale), 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. Pre-treatment child height-for-age z-score is also included as a control. (e) Bootstrapped (1000 replications) standard errors are clustered at the household level and presented in parentheses. (f) Significance: ***p<0.01, **p<0.05, *p<0.1.

Table A3: Heterogenous Effect of Parental Illness on Child Height by Child Age Groups

	(1)	(2)	(3)
Panel A: Aged 0-24 Months			
Parental Illness (=1 if yes)	-0.196	-0.201	-0.213
	(0.151)	(0.148)	(0.156)
Control	No	Yes	Yes
Estimation Approach	OLS	OLS	IPWRA
Observations	701	701	701
Panel B: Aged 25-59 Months			
Parental Illness (=1 if yes)	-0.197**	-0.180**	-0.179**
	(0.080)	(0.081)	(0.081)
Control	No	Yes	Yes
Estimation Approach	OLS	OLS	IPWRA
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 (in log scale), 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. Pre-treatment child height-for-age z-score is also included as a control. (f) Bootstrapped (1000 replications) standard errors are clustered at the household level and presented in parentheses. (g) Significance: ***p<0.01, **p<0.05, *p<0.1.

Table A4: Heterogenous Effect of Parental Illness on Child Height by Child Gender

	(1)	(2)	(3)
Panel A: Boys			
Parental Illness (=1 if yes)	-0.258**	-0.254**	-0.263**
	(0.108)	(0.105)	(0.107)
Control	No	Yes	Yes
Estimation Approach	OLS	OLS	IPWRA
Observations	921	921	921
Panel B: Girls			
Parental Illness (=1 if yes)	-0.181	-0.120	-0.127
	(0.113)	(0.116)	(0.118)
Control	No	Yes	Yes
Estimation Approach	OLS	OLS	IPWRA
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 (in log scale), 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. Pre-treatment child height-for-age z-score is also included as a control. (f) Bootstrapped (1000 replications) standard errors are clustered at the household level and presented in parentheses. (g) Significance: ***p<0.01, **p<0.05, *p<0.1.

Table A5: Heterogenous Effect of Parental Illness on Child Height by Child's Birth Order

	(1)	(2)	(3)
Panel A: First-born			
Parental Illness (=1 if yes)	-0.268**	-0.214	-0.218*
	(0.127)	(0.132)	(0.128)
Control	No	Yes	Yes
Estimation Approach	OLS	OLS	IPWRA
Observations	560	560	560
Panel B: Second-born			
Parental Illness (=1 if yes)	0.021	0.047	0.023
	(0.160)	(0.144)	(0.144)
Control	No	Yes	Yes
Estimation Approach	OLS	OLS	IPWRA
Observations	544	544	544
Panel C: Third or Higher Born			
Parental Illness (=1 if yes)	-0.336***	-0.264**	-0.233*
	(0.130)	(0.120)	(0.128)
Control	No	Yes	Yes
Estimation Approach	OLS	OLS	IPWRA
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 (in log scale), 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. Pre-treatment child height-for-age z-score is also included as a control. (f) Bootstrapped (1000 replications) standard errors are clustered at the household level and presented in parentheses. (g) Significance: ***p<0.01, **p<0.05, *p<0.1.

Table A6: 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	0.003	0.003	0.002
		(0.016)	(0.016)	(0.016)
Total Vaccination Given	6.204	-0.100	-0.051	0.101
		(0.255)	(0.260)	(0.287)
Number of Antenatal Visits	2.245	-0.070	0.032	0.016
		(0.193)	(0.195)	(0.201)
Delivery at Health Facility (=1)	0.237	-0.018	0.002	0.007
		(0.040)	(0.038)	(0.042)
Child Given Vitamin-A (=1)	0.681	0.001	-0.002	0.001
		(0.043)	(0.042)	(0.042)
Mother Took Iron Tablet (=1)	0.534	-0.010	-0.001	-0.008
		(0.042)	(0.043)	(0.044)
Health Input Index	0.000	-0.039	-0.003	-0.005
		(0.083)	(0.085)	(0.088)
Observations		727	727	727
Panel B: Childcare Input				
Childcare (Minutes/Day)	86.078	12.417	15.32*	14.986*
		(8.462)	(8.866)	(8.755)
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 estimation. (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 (in log scale), 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) Bootstrapped (1000 replications) standard errors are clustered at the household level and presented in parentheses. (i) Significance: ***p<0.01, **p<0.05, *p<0.1.

Table A7: Effect of Illness on Fertility Choice

	Outcome: Total Number of Children		
	(1)	(2)	(3)
Parental Illness (=1 if yes)	0.127*	-0.037	-0.051
	(0.070)	(0.058)	(0.059)
Mean at the Baseline		2.171	
Control	No	Yes	Yes
Estimation Approach	OLS	OLS	IPWRA
Observations	1,065	1,065	1,065

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 (in log scale), income per capita, asset per capita, have a loan, household faced a non-health shock, and other members have ADL limitations. (e) Bootstrapped (1000 replications) standard errors are clustered at the household level and presented in parentheses. (f) Significance: ***p<0.01, **p<0.05, *p<0.1.

Table A8: Effect of Parental Illness on Child Weight

	(1)	(2)	(3)
Parental Illness (=1 if yes)	-0.029	-0.003	-0.012
	(0.067)	(0.060)	(0.060)
Control	No	Yes	Yes
Estimation Approach	OLS	OLS	IPWRA
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 (in log scale), 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. Pre-treatment child height-for-age z-score is also included as a control. (e) Bootstrapped (1000 replications) standard errors are clustered at the household level and presented in parentheses. (f) Significance: ***p<0.01, **p<0.05, *p<0.1.

Appendix B:

IPWRA Estimator:

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

$$ATT_{IPWRA} = \frac{1}{n_T}\sum_{i=1}^{n} D_i[r_T^*(X, \delta_T^*) - r_C^*(X, \delta_C^*)], \tag{4}$$

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:

$$\min_{\alpha_T^*, \beta_T^*} \sum_{i=1}^{n} \frac{D_i(y_i - \alpha_T^* - X_i\beta_T^*)^2}{\hat{p}(X_i, \hat{\gamma})} \tag{5}$$

$$\min_{\alpha_C^*, \beta_C^*} \sum_{i=1}^{n} \frac{(1 - D_i)(y_i - \alpha_C^* - X_i\beta_C^*)^2}{1 - \hat{p}(X_i, \hat{\gamma})} \tag{6}$$

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).

Doubly robust method rely on the conditional independence assumption (CIA) (Lechner, 2001; 2002; Angrist and Pischke, 2008). CIA implies that given a set of pre-treatment observable covariates, potential outcomes are independent of treatment assignment (Imbens and Rubin, 2015). However, to satisfy the CIA, the doubly robust method requires a credible belief that there are no unobservable confounders that may bias the impact estimates (Litzow, Pattanayak, and Thinley, 2019). In this study context, we argue that the CIA is likely to hold as both the pre-treatment outcome and covariates are quite similar at the baseline.