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Orphanhood and Child Development: Evidence From India

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ABSTRACT This article provides the first systematic study of the short- and long-run effects of parental death on the cognitive, noncognitive (locus of control), and physical development of Indian children. Exploiting rich longitudinal data over 15 years, I use difference-in-differences with individual fixed effects to account for time-invariant unobserved heterogeneity between orphans and non-orphans and investigate the mechanisms. This method is an improvement over previous cross-sectional approaches to such explorations. I find that paternal death is negatively correlated with orphans' cognition but is not correlated with locus of control or physical health. Cognitive effects are mediated by a 10-percentage-point-lower probability of enrollment and a 20% decline in monetary investments in the child, eventually leading to one less year of schooling by age 22. These negative outcomes are concentrated among the least wealthy families, who respond to the shock by reducing consumption and increasing their labor supply.

KEYWORDS Orphanhood • Child development • India • Cognition • Locus of control

Introduction

Orphanhood is pervasive and can severely disrupt a child's life. Worldwide, there were more than 200 million orphans in 2012 (UNICEF 2012), and this figure has been trending upward. Although the rise in HIV-related deaths in sub-Saharan Africa in the early 2000s increased interest in this topic (e.g., Case et al. 2004; Evans and Miguel 2007), other parts of the developing world are at least equally affected but have remained largely understudied.

Despite ample consensus in favor of establishing national and international frameworks to help orphans, such programs tend to be ineffective (Ghanashyam 2010), perhaps because of the lack of credible and broadly scoped empirical evidence to inform these policies. Prior studies have commonly focused on orphans' school enrollment trajectories. Documenting the relative trajectories of orphans and non-orphans along other important outcomes is essential to provide a more complete picture of the effects of parental death. For instance, parental death might also affect the surviving parent's perceived returns to investments in the child, which is expected to influence outcomes such as the child's future labor market performance. Crucially, early death is generally not a random event. Therefore, analyses

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should account for potential observable and unobservable differences between orphans and non-orphans that could confound the results, as well as for the pathways through which the outcomes of interest are impacted. Information gleaned from this type of analysis could inform policies to effectively use limited public resources.

I focus on India, where the number of orphans increased from 25 million to 31 million between 2006 and 2012, making it the nation with the highest prevalence in absolute numbers (UNICEF 2012).[1] I provide the first systematic evidence on the differential evolution of child development by orphanhood status. I exploit Oxford University's Young Lives (YL), a rich survey tracking a cohort of children in Andhra Pradesh over 15 years, beginning when they were age 7. The longitudinal dimension is crucial for my analysis. First, it allows me to quantify potential *ex ante* differences between future orphans and never-orphans, better contextualizing the estimated effects of orphanhood. Moreover, although time-varying idiosyncratic factors might still be a source of bias, longitudinal tracking affords the estimation of difference-in-differences (DID) specifications with individual fixed effects to address the most pressing concerns about the presence of time-invariant omitted variables. Second, because YL was designed to track child development, it collected detailed information on cognitive abilities (standardized test scores), locus of control (a noncognitive skill),[2] physical health, and monetary investments in children. I can therefore provide a rich picture of whether and how parental death disrupts these aspects of human capital. Third, the data's long time span allows for exploring short- and long-term outcomes. Finally, attrition is a common limitation of longitudinal studies, particularly in developing countries. The YL data are unique in that only 9% of the original sample attritted over the 15-year period.

Conditional on finding significant effects of parental death, identifying the exact channel(s) through which they occur is crucial for informing policy. Theory and introspection suggest that both direct and indirect effects may be operating. The rationale for direct effects is clear: parents are crucial figures in a child's life, and the sudden disappearance of one of them can leave physical, psychological, and cognitive scars. Indirect effects may come from several sources, such as an income/wealth shock, an increase in the opportunity costs of schooling, or a change in schooling preferences.[3] All these sources would be expected to lead to lower investments in the child, which would then translate into lower human capital.

Employing a DID methodology to explore these effects, I find that orphans and non-orphans had similar cognition, noncognition, and physical health trajectories before the shock, which provides support for the parallel trends assumption required for identification. However, I found a negative and significant reduced-form effect of paternal death on cognition.[4] I show that the impact is not direct: it is mediated by lower investments in children, predominantly through lower schooling (with

[1] China is ranked second, with 20 million; Nigeria is third, with 12 million.

[2] Individuals with an internal locus of control believe they have substantial control over their outcomes (Villa 2017). Locus of control is my measure of noncognitive outcomes.

[3] I describe these mechanisms in more detail in section A.1 of the online appendix.

[4] I focus on paternal death because maternal death occurred significantly less frequently in my sample and does not have a substantial impact on child outcomes.

school enrollment declining by 10 percentage points)[5] but also through lower monetary investments in the child (an estimated 20% reduction). I argue that these effects are mainly driven by an income shock because (1) paternal death represents a large household income shock (with income declining by approximately 40% of a standard deviation), (2) most of the effects are concentrated among low-wealth households, (3) orphans are significantly more likely to work after the shock, and (4) households report that obtaining more income is their main coping strategy upon paternal death. Placebo tests and modifications of the estimating equations and the estimating sample confirm the robustness of my main results.

Literature Review

Evidence on the impacts of household health shocks on human capital accumulation is mixed. Early studies tended to find significant effects of parental death and illnesses but were cross-sectional, making it hard to argue for a causal relation (e.g., Case et al. 2004; Chen et al. 2009; Sun and Yao 2010). Subsequent work made significant progress by using short panel data. Although having multiple observations per child allows for removing biases arising from time-invariant unobserved heterogeneity, demonstrating the credibility of the parallel trends assumption requires a sufficiently long time series before the shock and access to rich data on multiple dimensions related to child development. For instance, documenting the presence of parallel trends in school enrollment, the main outcome of interest in this literature (e.g., Bratti and Mendola 2014; Case and Ardington 2006; Evans and Miguel 2007; Senne 2014) does not guarantee that this assumption also holds for other key determinants of human capital formation, such as health or monetary investments. If parallel trends were absent in these other dimensions, estimates could be biased.

Given these limitations, the disparity in prior results, even in studies exploiting longitudinal data sets, is likely partially due to the use of suboptimal data. For example, Case and Ardington (2006) and Evans and Miguel (2007) found that maternal death matters more for schooling outcomes in South Africa and Kenya, respectively. In contrast, Alam (2015) found stronger effects for paternal illness in Tanzania. Another limitation of these studies is that they focused on academic outcomes, as proxied by school enrollment and grade attainment. Not observing cognitive measures may be a substantial shortcoming because enrollment tends to be a noisy proxy for cognition, particularly in developing countries (e.g., Singh 2016). Moreover, major shocks, such as parental death, have been shown to affect individuals' attitudes and well-being in other contexts (Decker and Schmitz 2016; Kettlewell 2019). My data are rich enough to account for all these aspects and provide a more complete picture of the overall effects of parental death.[6]

[5] This effect is large. Shah and Steinberg (2017) showed that a year with a positive rain shock decreases enrollment by one percentage point in India, whereas León (2012) found the effects of exposure to civil war in Peru to be one third of my estimate for school enrollment in India.

[6] In this sense, my work aligns with recent economics findings on the multidimensionality of human capital, which emphasize the complementarities of various forms of human capital (e.g., Attanasio et al. 2020; Cunha and Heckman 2007). Little work has investigated the joint evolution of cognition, noncognition,

Research has also explored the impact of parental death on long-term outcomes in developed countries. Dupraz and Ferrara (2018) exploited paternal deaths occurring during the U.S. Civil War to show that deceased soldiers' sons performed worse in the labor market as measured by the skill level of their jobs. Using a similar strategy to examine outcomes for children of veterans from the Croatian–Serbian war, Kovac (2017) found that orphans attained worse academic outcomes and were more prone to behavioral problems and hospitalizations. Other contemporaneous research has shown that the effects of paternal absence may also operate in subtle ways. For instance, Persson and Rossin-Slater (2018) found that family ruptures during pregnancy lead to worse birth outcomes and increased perinatal complications.

Relative to most studies of developed and developing countries, I pay particular attention to the channels through which human capital accumulation is affected and the potential heterogeneity in treatment effects. This focus complements existing work on the direct and indirect determinants of human capital accumulation, socioeconomic characteristics as determinants of the degree to which shocks may distort households' decisions (e.g., Chen et al. 2009), and the determinants of child labor (e.g., Edmonds and Pavcnik 2005) and academic performance (e.g., Singh 2016).

Data

Data Overview

Oxford University's Young Lives (YL) is a panel study of two child cohorts (aged 1 and 7 in 2002) followed for five survey rounds, up to 2016.[7] I focus on the older cohort. Because these individuals were young adults (age 22) at the last interview, I can observe important outcomes that would be censored if I instead focused on the younger cohort (e.g., full academic histories).

My emphasis is on providing a rich analysis of the impact of paternal death on child development. I measure three key components of human capital: cognition, locus of control, and physical health.

Cognition

YL distributes and supervises age-specific tests measuring mathematical and language abilities in Rounds 1–4. I standardize these two subject scores to have a mean of 0 and a standard deviation of 1 among the children who completed each test in a given round. I then compute the cognitive measure as the simple average across the tests taken at each wave.

and physical health. Two exceptions are Biroli (2016) for the United Kingdom and Villa (2017) for the Philippines, but these studies did not explore the effect of parental death on the dynamic evolution of cognitive, noncognitive, and physical health.

[7] YL's sample is representative of the 95% lowest wealth *mandals* (townships) in Andhra Pradesh, the 10th most populated state in India. For sampling, 20 such mandals were randomly chosen, and approximately 50 households in each mandal were randomly selected.

Locus of Control

I obtain my measure of noncognitive skills as the principal component of the following attitudinal statements on locus of control elicited in Rounds 2–4: (1) "if I try hard, I can improve my situation in life"; (2) "I like to make plans for my future studies and work"; (3) "if I study hard at school I will be rewarded by a better job in future"; and (4) "other people in my family make all the decisions about how I spend my time." Responses to these items are coded so that higher values indicate children's greater sense of control over their lives and outcomes.[8] These statements were not introduced in the first round. Instead, YL constructed scores for problematic conduct, hyperactivity, and prosocial behavior. I compute these items' principal component to measure baseline noncognition, which is useful for conducting balance tests. However, I do not employ this variable in my longitudinal analysis of the impact of parental death because it is not comparable with subsequent measures of noncognition.

Physical Health

I focus on the first principal component from the following dimensions, which were consistently measured between Rounds 1 and 4: body mass index (BMI), BMI-for-age *z* score, height-for-age *z* score, and categorical measures for stunting and excessive thinness. The first component extracted is expected to capture good health status in terms of having an appropriate weight for a given height.[9]

In addition, children's full academic history and household members' vital status are available. Detailed information on food and nonfood investments in the child, time use, and household sociodemographic characteristics permit a detailed exploration of the mechanisms behind the effects of parental death. Moreover, this data set is unique in including information on maternal locus of control and many idiosyncratic shocks (e.g., weather, large price fluctuations, crime, and health) that minimize potential concerns about other time-varying events confounding the effects of parental death. Section A.2 in the online appendix offers a detailed description of the main variables used.

Sample Selection

Round 1 of YL included 1,008 children. Of these children, 135 had lost their father, and 55 had lost their mother by Round 5, when they were approximately age 22. For the main analysis, I use a balanced panel, producing a sample of 917 individuals. (In the robustness checks section, I discuss the determinants of attrition and show that the results are robust to using an unbalanced panel.) I discard those children whose father

[8] The eigenvalue of the first component is 1.97. The first component explains 49% of the total variation in the noncognitive variables. The weights of variables (1)–(4) in the first component are 0.53, 0.31, 0.55, and 0.56, respectively.

[9] The largest weights in the first component are for BMI-for-age (0.58), thinness (−0.5), and BMI (0.5). Fifty percent of the total variation in the physical health variables is explained by the first component.

or mother was dead by Round 1, given that such a death may have affected their baseline characteristics. This restriction reduces the number of individuals to 884. I do not remove those children for whom I do not observe at least one of the key variables at some point, causing small changes in sample size across specifications. In particular, the sample is slightly larger when I use individual fixed effects than when I include baseline or contemporaneous variables, which may be missing.

Descriptive Statistics

Descriptive statistics of the main variables of the subsample used for estimation are reported in Tables B.1–B.5 in the online appendix. Here, I highlight some key aspects. The sample is primarily rural (77%) and balanced in terms of gender (51% are female). Baseline school enrollment is almost universal (exceeding 97%), the average household size is 5.6, and the caste distribution is very similar to nationwide statistics. Some interesting dynamic considerations are children's physical development across survey rounds (i.e., their age, height, weight, and BMI), the loss of parents over time,[10] and increasing wealth among Indian households over time as reflected in the wealth index.[11]

Comparison of Orphans' and Non-Orphans' Baseline Characteristics

Before outlining my empirical strategy, I first explore whether certain characteristics are correlated with orphanhood or can predict it. This exercise is informative about potential sources of time-varying unobserved heterogeneity that might lead to biased estimates of the impact of parental death on child development. It also facilitates the interpretation of the estimated effects of orphanhood on the outcomes of interest and the mechanisms behind the effects.

The results from performing tests of differences in the means of observable characteristics at baseline (Round 1) between students who became orphans after Round 1 and those who did not are provided in Table 1. Overall, the differences are not statistically significant and are generally of small economic size. For example, the gap in enrollment rates is only 0.6 percentage points, and the difference in height (an important measure of health) is only 0.5 centimeters and favors future orphans. Importantly, there are no sizable differences in parents' and children's cognition or noncognition or in household wealth.[12]

[10] All children have parents alive at Round 1. By Round 5, approximately 15% of fathers and 6% of mothers had died.

[11] YL constructs the wealth index as the simple average of three subindices: the Housing Quality Index (housing-related comfort based on the quality of housing materials and housing size), the Consumer Durables Index (capturing the ownership of household items, such as cars or televisions), and the Services Index (measuring access to electricity, safe drinking water, safe sanitation, and fuel for cooking). The index ranges from 0 to 1, with larger values reflecting higher socioeconomic status. For more information, see Briones (2017).

[12] Premature death is not exclusive of those who are poor. Approximately 40% of all paternal deaths occur to households above median wealth. Indeed, exploiting possibly exogenous, unexpected causes of deaths

Table 1 Balance check

Variable (at baseline)	Mean, Non-Orphans	Mean, Orphans	Difference
Enrolled (proportion)	.974	.980	–.006
Current School Grade	2.816	2.891	–0.075
Parental Noncognitive Ability (PCA 1)	0.022	0.031	–0.009
Child's Cognitive Ability	0.009	–0.127	0.136
Child's Physical Health (PCA 1)	–0.395	–0.471	0.076
Child's Age (in months)	95.551	95.970	–0.419
Father's Age (in years)	36.315	40.384	–4.068***
Child's Height (in cm)	117.979	118.474	–0.495
Child's Weight (in kg)	19.420	19.625	–0.205
Child's BMI	13.892	14.142	–0.250
Child Not Stunted (proportion)	.667	.710	–.043
Child Moderately Stunted (proportion)	.261	.230	.031
Child Severely Stunted (proportion)	.073	.060	.013
Mother's BMI	21.155	20.302	0.853†
Rural (proportion)	.774	.752	.022
Household Size (number of individuals)	5.542	5.772	–0.230
Household Head Is Female (proportion)	.051	.059	–.009
Female Caregiver (proportion)	.988	1.000	–.012
0–20th Percentile Wealth Index (proportion)	.200	.250	–.048
20th–40th Percentile Wealth Index (proportion)	.196	.267	–.071†
40th–60th Percentile Wealth Index (proportion)	.213	.168	.044
60th–80th Percentile Wealth Index (proportion)	.197	.139	.059
80th–100th Percentile Wealth Index (proportion)	.195	.178	.016
Household Owns Land (proportion)	.870	.822	.049
Household Has Access to Water (proportion)	.824	.802	.022
Household Has Access to Toilet (proportion)	.297	.287	.010
No Parent Can Read (proportion)	.573	.651	-.078
Child Enrolled in Preprimary Education (proportion)	.572	.584	-.012
Female (proportion)	.519	.436	.083
Low Caste (proportion)	.787	.832	-.044

Notes: Tests for differences in means between children who became orphans at some point during Rounds 2–5 of the YL survey ($n = 101$) and children who did not become orphans in that period ($n = 771$). All variables are measured at baseline (i.e., YL's Round 1, when children were age 7). For caste, I construct an indicator variable equal to 1 if the child belongs to Scheduled Caste, Scheduled Tribe, or Other Backward Caste (i.e., "low caste") and 0 otherwise. For religion, I construct five indicators denoting whether the child identifies as Hindu (almost 90% of my sample), Christian, Muslim, Buddhist, or Sikh. I do not find any statistical difference in religious affiliation between orphans and non-orphans.

†$p < .10$; ***$p < .001$

The two dimensions on which the differences are statistically significant are maternal BMI and paternal age. Maternal BMI is unlikely to be an important concern, given that the average maternal BMI for both groups lies comfortably within widely

is not of marginal relevance in Andhra Pradesh, given medical studies (e.g., the Public Health Foundation of India's State-Level Disease Burden Initiative) showing that they are fairly common in the region. See online appendix section A.3 for more detailed information on the main causes of death in the region and related aspects.

accepted healthy thresholds in the medical literature: BMIs below 18 and above 23 are considered unhealthy for Indian standards (Misra et al. 2009). Moreover, although paternal age may suggest that some deaths in my sample are nonrandom, parents' average age in Round 1 is only 40, far from the male life expectancy of age 64 in Andhra Pradesh. Even at the last survey round, fathers of orphans would have been, on average, 55 years old. Still, to account for potential differences between future orphans and never-orphans, in specifications that do not include individual fixed effects, I control for maternal BMI, parental age at baseline, and a vector of additional dimensions, such as household composition and wealth (detailed later).

A potential concern, however, is that the relatively small sample size and the low percentage of children who became orphans (15%) might limit the statistical power to find differences. For instance, Table 1 suggests that although the differences are not statistically significant, future orphans live in somewhat less wealthy households and have less educated parents. My empirical analysis addresses this issue by exploiting the longitudinal data to net out time-invariant differences between future orphans and never-orphans through the inclusion of individual fixed effects. Therefore, the unbiasedness of my estimates relies on the assumption that the *trends* in all the outcomes of interest, which I show to evolve in parallel for the two groups before the shock, would have continued doing so in the absence of the treatment.

Further discussion on this initial exploration, including an analysis showing that the timing of death is generally unpredictable (at least using the rich observable information available in YL), is provided in section B.1 of the online appendix.

Main Analysis

Reduced-Form Effects on Cognition

I employ two main strategies to explore whether paternal death disrupts child development: estimating models accounting for observable baseline heterogeneity and conducting a DID analysis.

Models Accounting for Observable Baseline Heterogeneity

Given that paternal death is not likely allocated randomly, cross-sectional differences between orphans and non-orphans might determine both treatment status and the outcomes of interest. The richness of my data allows me to account for baseline differences in gender, household composition, the wealth index, and indicators for the mother being literate and alive. I also control for the two dimensions in which future orphans and never-orphans differ initially (as shown in Table 1), maternal BMI and paternal age:

$$cognition_{i,j,t} = \beta_1 fatherdead_{i,j,t} + \beta_2 \mathbf{X}_{i,j,1} + \alpha_j + \alpha_t + \varepsilon_{i,j,t}, \quad (1)$$

where the cognition level of individual i living in *mandal* (township) j at survey round t is regressed on an indicator of being a paternal orphan at time t and on $\mathbf{X}$,

the vector of observable characteristics measured at baseline (as indicated by the subscript 1).[13] α_j and α_t represent mandal and round fixed effects, respectively. They allow me to control for time-invariant mandal characteristics (e.g., different attitudinal values) and trends common to all mandals (e.g., a growing economy in Andhra Pradesh). ε is an error term. For inference, I cluster standard errors at the mandal level. Given the small number of mandals (20), I apply wild bootstrapping (Roodman et al. 2019).

Difference-in-Differences

Although the previous approach allows me to account for some of the most pressing forms of individual heterogeneity, there could be omitted variables that correlate with both the death shock and the outcomes of interest. The longitudinal data allow me to implement a DID strategy that includes individual and survey round fixed effects. Because one cannot observe what the human capital outcomes of orphans would have been had the parental death not occurred, never-orphans serve as counterfactual for the orphans' outcomes. Formally, I estimate the following:

$$cognition_{i,t} = \beta(treated_i \times post_{i,t}) + \alpha_i + \alpha_t + \varepsilon_{i,t}, \quad (2)$$

where the cognition level of individual i at survey round t is regressed on individual and round fixed effects (α_i and α_t, respectively). The estimate of interest, β, is attached to the interaction between an indicator signaling whether a child becomes an orphan at any time ($treated_i$) and an indicator taking the value of 1 if that event has already happened by round t ($post_{i,t}$). One aspect worth emphasizing is that β is estimated from within-individual variation. Never-orphans, who do not feature such variation in the interaction term (which is always 0 for them), serve as controls for orphans, the same role that future orphans take before experiencing the shock.

Note that any additive time-invariant individual characteristic, whether observable or not, will not bias my estimates. However, individual, time-varying shocks are still potential confounders. Therefore, the detailed information on the occurrence of other contemporaneous events, such as a drought or land redistribution (see the full list in section A.2, online appendix), is valuable in increasing the credibility of my identification.

Starting with estimates of Eq. (1) in column 1 of Table 2, I find that orphans score 22% of a standard deviation lower than non-orphans. This effect is significant at the 10% confidence level; for inference, one should focus on the wild bootstrapped p values provided in brackets. This result is virtually unchanged after the inclusion in column 2 of the set of indicators for important contemporaneous shocks to the household. Column 3 presents estimates of my preferred specification,

[13] All time-varying variables (except maternal vital status) are measured at Round 1 because measures at later rounds may have also been influenced by paternal death (and β_1 would then yield a partial estimate of the overall effect of paternal death on the outcome).

Table 2 Effects of paternal death on cognition

	(1)	(2)	(3)	(4)
Father Is Dead	−0.220†	−0.209†	−0.086	−0.317†
	(0.119)	(0.118)	(0.111)	(0.178)
	[.079]	[.096]		
Quartile 2 Wealth × Father Is Dead				0.371
				(0.283)
Quartile 3 Wealth × Father Is Dead				0.050
				(0.343)
Quartile 4 Wealth × Father Is Dead				0.439†
				(0.255)
Maintained Controls	Yes	Yes	No	No
Individual Fixed Effects	No	No	Yes	Yes
Contemporaneous Shocks	No	Yes	Yes	Yes
Number of Observations	3,270	3,270	3,446	3,446
R^2	.186	.193	.591	.592

Notes: Columns 1 and 2 estimate pooled ordinary least-squares regressions featuring a set of maintained controls that are measured in the first round of the YL survey: gender, household composition, wealth index, maternal BMI, literacy, vital status (time-varying), paternal age, mandal fixed effects, and round fixed effects. Column 2 additionally controls for the vector of contemporaneous shocks: weather, price, regulatory shocks, etc. (listed in section A.2 of the online appendix). Columns 3 and 4 instead control for individual (and round) fixed effects. Column 4 features the interaction of the treatment dummy variable with indicator variables for the household belonging to the second, third, or fourth quartile of the wealth index (ref. = the first quartile). The sample size increases in columns 3 and 4 because the availability of time-varying controls is not required. The level effects of the quartile indicators are absorbed by the individual fixed effects. Standard errors, clustered at the mandal level in columns 1 and 2 and at the individual level in columns 3 and 4, are shown in parentheses. Wild bootstrapped p values are shown in brackets.

†$p < .10$

Eq. (2). The point estimate remains negative but decreases in magnitude and is no longer significant. This result, however, masks large heterogeneity. In column 4, I interact my treatment with indicators for the household's baseline wealth quartile (reference = the lowest quartile). I find that paternal death has a sizable level effect among children in the leftmost part of the wealth distribution (−0.317), whereas wealthier children do not experience cognitive decline, on average.

For completeness, in panel a of Figure B.1 (online appendix), I present the point estimates and 95% confidence intervals from an extension of Eq. (2) in which I interact a treatment indicator with the *distance* to paternal death.[14] This approach is useful in validating the parallel trends assumption. I find that this assumption holds: no

[14] The horizontal axis measures the distance to parental death in terms of rounds. "b" and "a" indicate rounds before and after the shock takes place, respectively. "X" indicates the round when the shock happens. The accompanying numbers refer to the distance to the shock. For instance, if a child is observed to be an orphan in Round 3 but not Round 2, that child's value for "b2" will be 1 in Round 1 (and 0 in every other round); in Round 2, the only indicator taking value 1 will be "b1"; in Round 3, only "X" will take the value 1; and in Round 4, it will be "a1."

Table 3 Effects of paternal death on locus of control and on physical health

	Locus of Control		Physical Health	
	(1)	(2)	(3)	(4)
Father Is Dead	–0.076	–0.017	–0.127	0.002
	(0.085)	(0.192)	(0.255)	(0.130)
	[.392]		[.617]	
Maintained Controls	Yes	No	Yes	No
Individual Fixed Effects	No	Yes	No	Yes
Contemporaneous Shocks	Yes	Yes	Yes	Yes
Number of Observations	3,081	3,222	2,889	3,043
R^2	.249	.504	.228	.771

Notes: Columns 1 and 3 replicate column 2 in Table 2. Columns 2 and 4 replicate column 3 in Table 2. Standard errors, clustered at the mandal level in columns 1 and 3 and at the individual level in columns 2 and 4, are shown in parentheses. Wild bootstrapped *p* values are shown in brackets.

statistical differences are seen before the shock relative to the baseline category (the round before the event, "b1").[15]

Reduced-Form Effects on Other Human Capital: Locus of Control and Physical Health

Following recent literature on the multidimensionality of human capital and the importance of socioemotional skills in late-life outcomes, I proceed as in Table 2 to explore the impact of paternal death on locus of control and physical health.

Locus of Control

The first two columns of Table 3 estimate Eqs. (1) and (2), respectively. The results show that paternal death has a negative but not statistically significant effect on locus of control. Panel c in Figure B.1, which includes individual and round fixed effects, confirms that paternal death does not have a significant impact on locus of control while demonstrating parallel trends before the shock.

Physical Health

I replicate the analysis, this time for physical health, in columns 3 and 4 of Table 3 and in panel d of Figure B.1. Once again, I find no noticeable change contemporaneously to paternal death. If anything, I find a slight but nonsignificant improvement in health. Importantly, parallel trends are also present.

[15] A similar analysis for maternal death is reported in panel b of Figure B.1 (online appendix). As mentioned earlier, this event is significantly less frequent, and its reduced-form effects under this two-way fixed effects approach are zero.

Mechanisms

Main Mechanism: Effects on Enrollment

In a formal test of whether orphanhood has an impact on school enrollment,[16] I estimate the following model:

$$enrolled_{i,t} = \sum_{g=-18}^{12} \beta_g (D_{i,t}(g) \times treated_i) + \alpha_i + \alpha_t + \varepsilon_{i,t}. \quad (3)$$

This model exploits the retrospective observation of *yearly* academic enrollment (vs. only at the time of the survey) and the exact year of paternal death. I reconstruct each child's academic trajectory between 1998 and 2015, which I index by t. For children who became orphans, I may observe them up to 18 years before paternal death (when it occurs in 2015) and up to 12 years after paternal death (when it occurs in 2003, the first year after the first round of YL, Round 1).

The estimates of interest are those attached to the multiple interactions between an indicator signaling whether a child became an orphan at any time ($treated_i$) and various indicators for whether, at any given year t, the distance to paternal death is g years. For instance, $D_{i,t}(-1)$ will take the value of 1 if, at time t, the child is one year away from becoming an orphan, and 0 otherwise.

The graphical representation of the point estimates for the interactions and their 95% confidence intervals are provided in panel a of Figure 1. It shows that the enrollment patterns of future orphans and never-orphans evolved similarly until the academic year of the shock (inclusive). However, the probability of enrollment in the academic year following the shock fell by 10 percentage points. This rapid decline is useful for credible identification because it reduces the potential that other contemporaneous factors are confounding the effect.[17] Moreover, consistent with the heterogeneity analysis in column 4 of Table 2, a DID regression in which the treatment is simply a post-shock dummy variable yields a level effect of −0.125 (significant at the 1% confidence level), but its interactions with indicators for belonging to the third and fourth wealth quartiles (ref. = belonging to the least wealthy households) take values of 0.212 and 0.160, respectively. Thus, the least wealthy households are the most prone to withdraw children from school in response to a paternal death.

Long-Run Effects on Academic Achievement

Because survey participants were already aged 22 by the last round, it is possible to explore late-life impacts of paternal death by looking at the highest grade ever achieved. This examination is helpful to determine whether school withdrawal is a

[16] More information on India's education system is provided in section A.4 of the online appendix.

[17] I also test the equality of coefficients across years. As expected, it is rejected when comparing before and after the death (e.g., the test of equality between a2 and b1 has a p value of .01), but not within pre- and post-death periods.

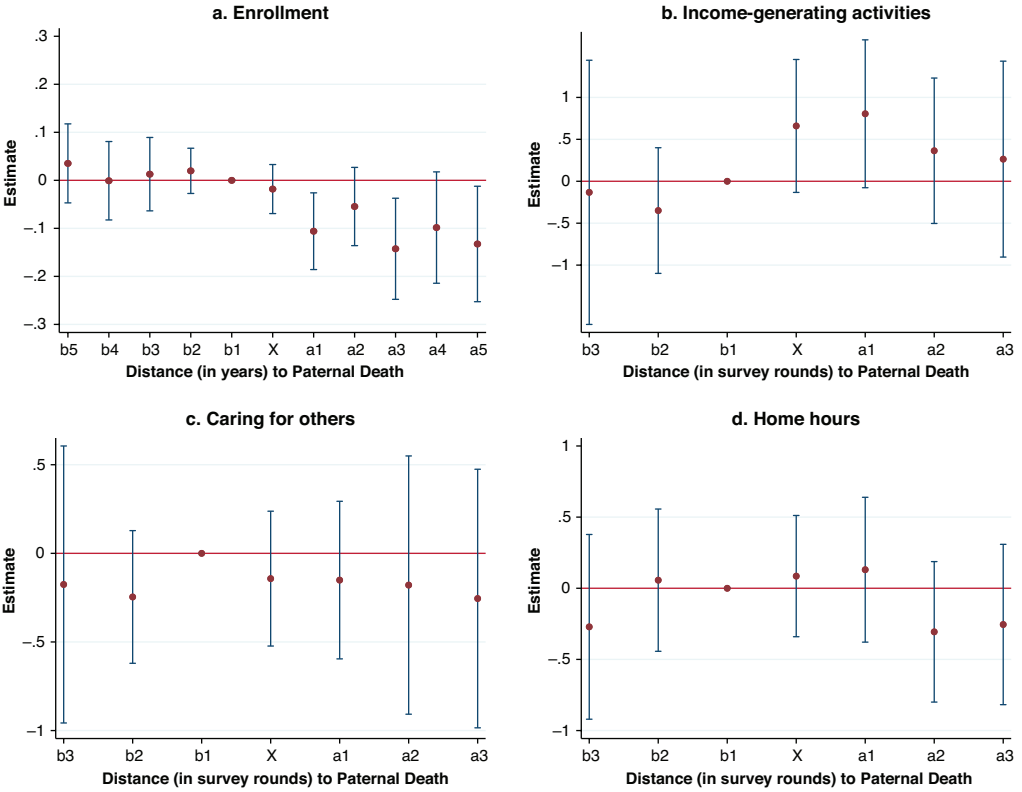

Fig. 1 Effects of paternal death by distance to death. Point estimates and 95% confidence intervals (computed using standard errors clustered at the individual level) of multiple indicators of distance to paternal death (β_g) in a regression of the indicated outcome on individual (α_i) and round (in panels b–d) or year (in panel a) (α_t) fixed effects of the following form: $outcome_{i,t} = \sum_{g=T_L}^{T_H} \beta_g (D_{i,t}(g) \times Treated_i) + \alpha_i + \alpha_t + \varepsilon_{i,t}$. The horizontal axis measures the distance to parental death in terms of survey rounds or years. "b" and "a" indicate rounds before and after the shock takes place, respectively. "X" indicates the round when the shock happens. The accompanying numbers refer to the distance to the shock. The omitted category is one survey round/year before the shock ("b1").

temporary adjustment, with children perhaps returning to school at some point after the shock.

I regress the highest grade achieved at Round 5 (measured in years of education) on whether the father was dead by that round (i.e., whether the child experienced paternal death at some point during the study) on the maintained controls:

$$Higest\ grade\ achieved_{i,j,5} = \beta_1 father\ is\ dead_{i,j,5} + \beta_2 \mathbf{X}_{i,j,1} + \alpha_j + \varepsilon_{i,j,5}. \quad (4)$$

Table 4 reports the results estimated by ordinary least-squares (OLS) and Tobit regressions; the results are also presented graphically in section B.3 of the online appendix. The Tobit regressions take into account that the lower bound of years of education is 0. The results show that children whose father died completed approximately one less year of education (columns 1 and 2) and that the probability of reaching higher

Table 4 Long-run effects on enrollment and academic achievement

	Highest Education		Reached Grade 5	Reached Grade 12	Reached Postsecondary	Highest Education
	(1)	(2)	(3)	(4)	(5)	(6)
Father Died in Rounds 1–5	−1.150*	−1.151**		−0.153†	−0.130*	−1.123*
	(0.441)	(0.440)		(0.069)	(0.055)	(0.406)
	[.021]			[.070]	[.037]	[.020]
Father Died in Round 2			−0.045			
			(0.050)			
			[.449]			
Estimator	OLS	Tobit	OLS	OLS	OLS	OLS
Region Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes
Only Children Ever Enrolled	No	No	No	No	No	Yes
Baseline Controls	Yes	Yes	Yes	Yes	Yes	Yes
Number of Observations	824	824	824	824	824	814
R^2	.273		.120	.235	.195	.275

Notes: Indicators of individual academic achievement (in columns 3–5) or the number of years of education achieved (columns 1, 2, and 6) are regressed on orphanhood indicators. All regressions are cross-sectional, with the outcome measured at Round 5. The estimation method is ordinary least-squares (OLS) except for column 2, which takes into account the lower-bound censoring of education at 0 through a Tobit model. Column 6 replicates column 1 to show that the results are not an artifact of including in the sample children never enrolled (those with 0 years of education). Controls are as in Table 2's column 1. Standard errors, clustered at the mandal level, are shown in parentheses. Wild bootstrapped p values are shown in brackets.

†$p < .10$; *$p < .05$; **$p < .01$

academic levels decreased for orphans relative to non-orphans. The attainment of lower grades, such as grade 5, does not differ significantly between orphans at that round and children who are not orphans at that round (column 3).[18] However, these two groups of children start diverging before finishing high school (column 4), and the gap in the probability of ever attending higher education remains (column 5). The results in column 1 are robust to restricting the sample to children enrolled in formal education at some point in their lives (column 6).[19]

Further Discussion on Mechanisms

I complement the previous findings on lower post-shock cognition and enrollment by showing that (1) school enrollment is crucial to human capital accumulation, which is not obvious in the Indian context; and (2) paternal death negatively affects monetary investments in children, another determinant of cognition. These two factors account for the gap in cognitive ability between orphans and non-orphans.

[18] In this comparison, the main independent variable is an indicator of paternal death up to Round 3.

[19] I explored heterogeneity of the effects on enrollment by child's gender (reported in Table B.12, online appendix) and by household composition, but I did not find any.

Table 5 Effects of paternal death on annual investments (in 2006 rupees) in the child

	(1)	(2)
Father Is Dead	−6,540.858*	−7,746.322**
	(1,904.855)	(2,515.282)
Sample	All	Only enrolled
Individual Fixed Effects	Yes	Yes
Contemporaneous Shocks	Yes	Yes
Number of Observations	2,433	1,025
R^2	.573	.562

Notes: Regressions use information on annual investments in the child in Rounds 3–5 (the only ones for which YL collected this information). I therefore remove from my estimating sample children who were orphans by Round 3. Both regressions control for individual and round fixed effects, as well as for the set of shocks. Column 2 restricts the sample to students contemporaneously enrolled. Standard errors, clustered at the individual level, are shown in parentheses.

*$p < .05$; **$p < .01$

One pending question is why families remove their children from school and invest less in them. In this section, I also outline a novel finding in the orphanhood literature uncovering significant increases in child's hours worked following paternal death. This finding suggests that the income channel is the most likely precursor.[20] Moreover, using longitudinal data on total household earnings, I formally confirm that households experience a large negative income shock after a paternal death. I then show that the mothers of paternal orphans desire lower educational attainment for their children only after the shock.

Effects on Parental Investment

Parental investments of time and money have been shown to significantly improve child outcomes (e.g., Foster and Gehrke 2017). YL provides detailed information on monetary investments in children, such as on food, clothing, and school materials. Similar to in my main analysis, I first show that future orphans and never-orphans do not differ in their received investments (measured in 2006 USD) before the shock. Figure B.5 (online appendix) shows that a test of equality of means in Round 2 (the first round for which information on investments in children is available) is not rejected ($p = .14$).[21] Table 5 shows that orphans' and non-orphans' investment paths (measured in 2006 Indian rupees) start diverging after the event. This finding holds when all children are included in the estimating sample (column 1) and

[20] My finding of effects for deaths of only fathers, households' main income earners, is suggestive of the income channel.

[21] I redefine treated children as those whose parents died between Rounds 3 and 5 so that the units of observation are comparable at the new baseline, which is now Round 2. The results hold in Table B.22 (online appendix), which displays results for a more disaggregated measure of investments (between food and nonfood consumption).

Table 6 Channels for the impact of orphanhood on cognition

	(1)	(2)	(3)
Father Is Dead	–0.317†	–0.223	–0.113
	(0.178)	(0.165)	(0.137)
Child Is Currently Enrolled		0.488***	0.159***
		(0.044)	(0.040)
Log Investments in Child			0.014*
			(0.008)
Quartile 2 Wealth × Father Is Dead	0.371	0.372	0.170
	(0.283)	(0.271)	(0.213)
Quartile 3 Wealth × Father Is Dead	0.050	0.007	–0.203
	(0.343)	(0.315)	(0.408)
Quartile 4 Wealth × Father Is Dead	0.439†	0.335	0.211
	(0.255)	(0.237)	(0.233)
Number of Observations	3,446	3,446	2,560
R^2	.592	.610	.813

Notes: For convenience, column 1 replicates the fourth column in Table 2. Columns 2 and 3 incrementally control for enrollment status and investments in the child in the year before the survey round, respectively. Standard errors, clustered at the individual level, are shown in parentheses.

†$p < .10$; *$p < .05$; ***$p < .001$

when only children still enrolled in school (who have higher investment requirements linked to schooling) are included (column 2).

Returning to the Determinants of Cognition

I conduct a mediation analysis to understand which channels account for a larger fraction of the overall impact on cognition. The objective is to study how the inclusion of endogenous variables previously found to have been affected by paternal death (investments in the child and school enrollment) modifies the point estimate and statistical significance of the treatment indicator from the baseline specification.

The results are reported in Table 6. The first column replicates column 4 in Table 2 for convenience. In the second column, I additionally control for enrollment status. As expected, this variable is strongly positively correlated with performance. Moreover, the point estimate for orphanhood status largely decreases (in absolute value) and becomes nonsignificant. In column 3, I further introduce the log of monetary investments in the child in the previous year. This variable is also positively correlated with cognition and further reduces the point estimate of the treatment. To estimate the relative importance of these mechanisms, I employ Gelbach's (2016) decomposition, which apportions the amount of decline in the treatment effect estimates from columns 1 to 3 due to each of the two mediators and is independent of the order in which the mediators are added to the baseline regression. I estimate that 85% of the decline in cognition after paternal death is explained by lower enrollment.

Effects of Paternal Death on Time Allocation

YL asked participants to report their time spent on various activities on a normal weekday: sleeping, playing, caring for relatives, doing chores, doing domestic tasks, working for a wage, attending school, and studying outside school.

I estimate the DID specification for income-earning activities (self-employed and market hours) and for home production. Panel b of Figure 1 shows that although orphans devoted approximately the same amount of time to income-generating activities in the household (e.g., farming or running their own businesses) before the shock, they contributed significantly more afterward. This finding, coupled with the greater likelihood of school dropout among these children, suggests that orphans substitute school time with work time.

Complementing these results, panel c of Figure 1 shows that time devoted to caring for other household members did not differ before the shock and remained nonsignificant after it. This finding reinforces the idea that orphans did not have different trends in time allocations before the event and that the paternal death was likely unexpected, given the absence of systematic pre-shock differences in care of the father. I find similar results for home production, shown in panel d.

Impacts on Household Income and Maternal Noncognition

Although the preceding results provide important evidence supporting the central role of the income channel, a key component of the argument is that paternal death represents a large income shock for families. Household income was recorded only in Rounds 2 and 3, but these data allow me to explore whether parental death affects total household income when I control for child (i.e., household) fixed effects. The results in column 1 of Table 7 show that paternal death leads to an average income decline of approximately 32,000 rupees—a substantial decline of approximately 40% of the standard deviation of income.

This sizable reduction in income is likely to make it unaffordable for a substantial fraction of households to continue sending their children to school. If so, mothers would be expected to lower their aspirations for their children's educational attainment after a paternal death. Column 2 in Table 7 exploits mothers' responses to the YL Round 2 question, "ideally, what level of formal education would you like your child to complete?" The results show that widows have lower academic aspirations for their children after the shock. I next verify that mothers of children who became orphans in later rounds did not differ before the paternal death from those whose children never became orphans. Column 3 of Table 7 shows that mothers of children who became orphans in Rounds 3, 4, or 5 did not respond systematically differently from the mothers whose children never lost their father.

Finally, columns 4 and 5 test for evidence that maternal locus of control might be affected by her spouse's death. Again, because this outcome is observable only in Round 2, I first investigate whether current widows display a lower locus of control (column 4). I do not find evidence for this. Column 5 shows that this result is unlikely to be an artifact of Round 2 widows having already experienced a noncognition

Table 7 Effects on household income and maternal academic aspirations for the child

	Household Earnings	Desired Education		Maternal Noncognition	
	(1)	(2)	(3)	(4)	(5)
Father Is Dead	−31,814.819**				
	(9,988.126)				
Father Died in Round 2		−1.369†		0.425	
		(0.716)		(0.328)	
		[.070]		[.252]	
Father Died in Rounds 3–5			−0.128		0.183
			(0.393)		(0.259)
			[.775]		[.477]
Individual Fixed Effects	Yes	No	No	No	No
Maintained Controls	No	Yes	Yes	Yes	Yes
Contemporaneous Shocks	Yes	No	No	No	No
Number of Observations	1,660	800	771	649	636
R^2	.679	.191	.185	.166	.154

Notes: Column 1 regresses household income in Rounds 2 and 3 on an indicator for orphanhood and round and individual (equivalent to household) fixed effects. The outcome is mothers' desired educational level for their children in columns 2–3 and maternal noncognition (locus of control) in columns 4–5, both measured in Round 2; these columns use the same main controls as in column 1 of Table 3. In columns 2 and 4, the outcome is regressed on an indicator for the father being dead by Round 2. Columns 3 and 5 proceed similarly but discard those who were orphans by Round 2 and explore instead whether children who became orphans in Rounds 3–5 differ from those who did not. Standard errors, clustered at the individual level in column 1 and at the mandal level in columns 2–5, are shown in parentheses. Wild bootstrapped p values are shown in brackets.

†$p < .10$; **$p < .01$

impact because, at Round 3, future widows did not display any statistical difference before the event. This finding suggests that the lower maternal educational aspirations for their children after a parental death are more likely driven by the decline in household resources than by a change in the value attached to education through surviving mothers' locus of control.

Robustness

Alternative Channels

Several alternative pathways could feature an intermediate step between paternal death and school withdrawal. For example, paternal death could alter participation in social programs, lead to early marriage, change attitudes toward schooling, and affect children's health. In turn, these outcomes could lead to school dropout. In section B.4 of the online appendix, I show that the following channels do not seem to be operating: (1) postponing enrollment or moving marriage forward; (2) increased participation in social programs; and (3) increased incidence of child illnesses.

Placebo Tests

I conduct two placebo tests. The first one is a randomized-based inference (Imbens and Wooldridge 2009). I start by discarding the students that truly became orphans during the study. Then, to the remaining individuals, I randomly assign orphanhood status so that their share at any round is the same as that represented by the truly treated children in the data (i.e., I let the actual data inform the counterfactual distribution of orphans that I use). I repeat this process 500 times. The results, reported in Figure B.6 (online appendix), show that the actual baseline estimates for enrollment, academic achievement, monetary investments, and cognition lie outside the 95% empirical confidence interval obtained from the distribution of randomly assigned treatment effects.

The second placebo test, unlike the first, does not rely on simulating data. Instead, I examine whether actual outcomes that should not have been influenced by the treatment are indeed not affected. In section B.5 in the online appendix, I show that height at Round 5, an outcome largely determined by age 7 (Martorell and Habicht 1986), does not systematically differ between orphans and non-orphans.

Additional Robustness Checks

I further explore the stability of the reduced-form effects on cognition to different estimating samples. In particular, I (1) work with an unbalanced panel, (2) include only those children who were enrolled in school, (3) control for whether the father was working for a wage at Round 1 as a proxy for household income, and (4) include only children whose parents were aged 40 or younger at Round 1. The goal of (4) is to make the sample even more homogeneous and to reduce the concerns about the statistical difference in Round 1 age between parents who died and those who did not during the sample period. The results in Table B.23 (online appendix) are consistent with those in Table 2.

Conclusion

Employing a long and rich panel of children and their households from the Indian state of Andhra Pradesh, this study is the first to quantify the impact of parental death on the evolution of cognition, locus of control, and physical health. Accounting for individual heterogeneity through a DID strategy, I find that experiencing a paternal death after age 7 has a significant effect on orphans' cognition. These impacts are mediated by lower investments in children. One year after the shock, orphans' probability of school enrollment is approximately 10 percentage points lower. Moreover, monetary investments in children also decline by approximately 20%.

These findings suggest the presence of a wealth/income channel and are supported by other empirical results: (1) heterogeneous effects are present in terms of wealth (the effects are concentrated mostly among families at the bottom of the wealth distribution), and (2) orphans devote more time to income-generating activities (and reduce their investments and consumption) after paternal death. These two findings

are not surprising given that paternal death represents a large income shock to the households in my sample.

My work constitutes a step toward informing public policy attempting to address the impact that parental health shocks have on human capital formation—policy that has been largely ineffective. The lower human capital observed in this study comes from declines in monetary and schooling investments in the child resulting from budget constraints of the less wealthy households (rather than through a direct effect). Therefore, policy-sourced monetary aid should be channeled toward such low-wealth households to prevent school dropout and a decline in investments in the children. Such policy is expected to yield further intergenerational benefits as these children form their own households. ■

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References

Alam, S. A. (2015). Parental health shocks, child labor and educational outcomes: Evidence from Tanzania. *Journal of Health Economics, 44,* 161–175.

Attanasio, O., Meghir, C., & Nix, E. (2020). Human capital development and parental investment in India. *Review of Economic Studies, 87,* 2511–2541.

Biroli, P. (2016). *Health and skill formation in early childhood* (UBS Center Working Paper No. 17). Zurich, Switzerland: UBS International Center of Economics in Society, University of Zurich.

Bratti, M., & Mendola, M. (2014). Parental health and child schooling. *Journal of Health Economics, 35,* 94–108.

Briones, K. (2017). *"How many rooms are there in your house?": Constructing the Young Lives wealth index* (Technical Note No. 43). Oxford, UK: Young Lives.

Case, A., & Ardington, C. (2006). The impact of parental death on school outcomes: Longitudinal evidence from South Africa. *Demography, 43,* 401–420.

Case, A., Paxson, C., & Ableidinger, J. (2004). Orphans in Africa: Parental death, poverty, and school enrollment. *Demography, 41,* 483–508.

Chen, S. H., Chen, Y.-C., & Liu, J.-T. (2009). The impact of unexpected maternal death on education: First evidence from three national administrative data links. *American Economic Review: Papers & Proceedings, 99,* 149–153.

Cunha, F., & Heckman, J. (2007). The technology of skill formation. *American Economic Review: Papers & Proceedings, 97,* 31–47.

Decker, S., & Schmitz, H. (2016). Health shocks and risk aversion. *Journal of Health Economics, 50,* 156–170.

Dupraz, Y., & Ferrara, A. (2018). *Fatherless: The long-term effects of losing a father in the U.S. Civil War* (CAGE Working Paper No. 538). Coventry, UK: CAGE Research Centre, University of Warwick.

Edmonds, E. V., & Pavcnik, N. (2005). Child labor in the global economy. *Journal of Economic Perspectives, 19*(1), 199–220.

Evans, D. K., & Miguel, E. (2007). Orphans and schooling in Africa: A longitudinal analysis. *Demography, 44,* 35–57.

Foster, A. D., & Gehrke, E. (2017). *Start what you finish! Ex ante risk and schooling investments in the presence of dynamic complementarities* (NBER Working Paper 24041). Cambridge, MA: National Bureau of Economic Research.

Gelbach, J. B. (2016). When do covariates matter? And which ones, and how much? *Journal of Labor Economics, 34*(2, part 1), 509–543.
Ghanashyam, B. (2010). India failing children orphaned by AIDS. *Lancet, 375,* 363–364.
Imbens, G. W., & Wooldridge, J. M. (2009). Recent developments in the econometrics of program evaluation. *Journal of Economic Literature, 47,* 5–86.
Kettlewell, N. (2019). Risk preference dynamics around life events. *Journal of Economic Behavior & Organization, 162,* 66–84.
Kovac, D. (2017). *Do fathers matter?: Paternal mortality and children's long-run outcomes* (Working Paper No. 609). Princeton, NJ: Industrial Relations Section, Princeton University. Retrieved from https://dataspace.princeton.edu/bitstream/88435/dsp01td96k5012/3/609.pdf
León, G. (2012). Civil conflict and human capital accumulation: The long-term effects of political violence in Perú. *Journal of Human Resources, 47,* 991–1022.
Martorell, R., & Habicht, J.-P. (1986). Growth in early childhood in developing countries. In F. T. Falkner & J. M. Tanner (Eds.), *Human growth: A comprehensive treatise* (Vol. 3, 2nd ed., pp. 241–262). New York, NY: Plenum Publishing.
Misra, A., Chowbey, P., Makkar, B., Vikram, N., Wasir, J., Chadha, D., . . . Gulati, S. (2009). Consensus statement for diagnosis of obesity, abdominal obesity and the metabolic syndrome for Asian Indians and recommendations for physical activity, medical and surgical management. *Journal of the Association of Physicians of India, 57*(2), 163–170.
Persson, P., & Rossin-Slater, M. (2018). Family ruptures, stress, and the mental health of the next generation. *American Economic Review, 108,* 1214–1252.
Roodman, D., Nielsen, M. Ø., MacKinnon, J. G., & Webb, M. D. (2019). Fast and wild: Bootstrap inference in Stata using boottest. *Stata Journal, 19,* 4–60.
Senne, J.-N. (2014). Death and schooling decisions over the short and long run in rural Madagascar. *Journal of Population Economics, 27,* 497–528.
Shah, M., & Steinberg, B. M. (2017). Drought of opportunities: Contemporaneous and long-term impacts of rainfall shocks on human capital. *Journal of Political Economy, 125,* 527–561.
Singh, A. (2016). Learning more with every year: School year productivity and international learning divergence. *Journal of the European Economic Association, 18,* 1770–1813.
Sun, A., & Yao, Y. (2010). Health shocks and children's school attainments in rural China. *Economics of Education Review, 29,* 375–382.
UNICEF. (2012). *The state of the world's children 2012: Children in an urban world* (Report). New York, NY: United Nations Children's Fund.
Villa, K. M. (2017). Multidimensional human capital formation in a developing country: Health, cognition and locus of control in the Philippines. *Economics & Human Biology, 27,* 184–197.

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