Original Contribution

Causal Effect of Parental Schooling on Early Childhood Undernutrition: Quasi-Experimental Evidence From Zimbabwe

Jan-Walter De Neve* and S. V. Subramanian

* Correspondence to Dr. Jan-Walter De Neve, Institute of Public Health, Heidelberg University, Im Neuenheimer Feld 130.3, R.314, Heidelberg 69120, Germany (e-mail: janwalter.deneve@uni-heidelberg.de).

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An estimated 3.1 million children die each year because of undernutrition. Although cross-sectional and longitudinal studies have found a protective association between greater parental education and undernutrition in their children, no randomized trial has identified a causal effect, to our knowledge. Using the 1980 education reform in Zimbabwe as a natural experiment, we estimated the causal effect of additional parental schooling on the probability of anthropometric failure in their children under 5 years of age (ages 3 through 59 months). Analyzing data on 8,243 children from the 1988, 1999, 2005–2006, and 2010–2011 Demographic and Health Surveys, we found no effect of parental schooling on early childhood undernutrition at the national level in Zimbabwe. Among households in the urban and high-wealth-index subsamples, each additional year of maternal schooling led to absolute reductions in the probability of a child's being wasted of 5.2 percentage points (95% confidence interval (CI): −9.3, −1.2) and 3.6 percentage points (95% CI: −6.9, −0.4), respectively. In the subsample of children between the ages of 3 and 23 months, each additional year of paternal schooling increased the probability of a child's being stunted by 9.6 percentage points (95% CI: 1.4, 17.9). Secondary schooling alone may not be enough to improve early childhood nutrition in low-resource settings.

Abbreviations: CI, confidence interval; DHS, Demographic and Health Surveys; IV, instrumental variable; OLS, ordinary least squares; 2SLS, 2-stage least squares.

Undernutrition causes an estimated 45% of deaths among children younger than 5 years, resulting in 3.1 million deaths annually (1). Previous cross-sectional and longitudinal studies have generally found a protective association between additional formal parental education and their children’s nutritional status (2–11), health (6, 11–13), and mortality risk (13–15). Hypothesized pathways include direct knowledge transfers to future parents (16, 17); increased ability to acquire, process (17, 18), and communicate (19) information; reduced risky health behaviors during pregnancy for mothers (e.g., smoking) (20); increased labor force participation and additional resources that could be invested (e.g., in health-care services) (21–23); assortative mating with better educated and higher-income spouses (24); a general “familiarity with modern society” (17) that may make parents more receptive to modern medicine (16, 25); and the “hidden curriculum” values of discipline and obedience to authority learned in school (26). One key empirical concern regarding these previous studies is that a range of unobserved variables that are associated with both education and child health may make observational studies subject to confounding bias. Unobserved factors associated with higher economic or social prospects for a parent will lead to both higher parental education and improved child outcomes, which makes these earlier findings difficult to interpret.

In the absence of randomized trials assessing formal education (as opposed to nutrition education, for example (27–29)), a handful of studies have exploited natural experiments to address concerns about unobserved confounding (30). In the United States, investigators utilized the availability of colleges and found that additional college education among mothers led to improved health among their children (20), whereas in the United Kingdom, additional primary education resulting from a change in the minimum school-leaving-age did not (31). Conceptually, large causal effects of parental education on child health also seem more likely in a developing setting, without an established welfare system providing support to
households most at risk of early childhood undernutrition. In a recent study in Turkey, using a change in compulsory schooling law as a natural experiment, Güneş et al. (32) found that maternal primary school completion increased height-for-age z scores and weight-for-age z scores of participants’ children by 1.1 and 1.0 standard deviations, respectively. Children born to mothers with a primary school education were 2 inches (5.1 cm) taller, on average, than children born to mothers without a primary school education (32). Evidence on the causal relationship between additional education for both parents and undernutrition among children in low-resource settings, however, remains scarce. To our knowledge, this study was the first to estimate the causal effect of formal parental schooling on anthropometric failure in children in a low-resource setting.

In this study, we used data on Zimbabwe from the Demographic and Health Surveys (DHS), a series of surveys that collect health data in developing countries, in a first attempt to address this gap in the literature. To overcome the confounding concerns outlined above, we exploit quasi-experimental variation in educational attainment generated by a major education policy reform. In 1980, Zimbabwe reduced academic and structural barriers to secondary school attendance, which affected all parental cohorts born in 1967 or later but not parents born earlier (33, 34). As we show, the reform increased educational attainment by an average of 2.5 years for mothers fully affected by the reform and 2.4 years for fathers fully affected. Since there is no obvious reason why parents born just before 1967 would be different from parents born during or just after 1967, conditional on covariates, we were able to directly test the impact of exogenous shifts in parental educational attainment on children’s anthropometric outcomes.

METHODS

Data sources

Data were obtained from the 1988, 1999, 2005–2006, and 2010–2011 Zimbabwe DHS surveys, 4 nationally representative household surveys with anthropometric data on children who were under age 5 years at the time of the survey (35). For each survey, between 4,000 and 10,000 households were selected; all household members aged 15–49 years for women and all members aged 15–54 years for men were eligible to be interviewed. Household response rates ranged from 95% to 99%; individual participation ranged from 90% to 96% for women and from 82% to 92% for men, yielding a total sample of 146,630 individuals (35). Children were eligible for anthropometric measurements if they were between 3 and 59 months of age and living in the household at the time of the survey. Data on parental age and completed years of schooling were available for 88% of the children eligible for anthropometric measurement.

Study population

Zimbabwe has one of the highest rates of child undernutrition in the world; in 2011, 32% of children under age 5 years were stunted, 10% were underweight, and 3% were wasted (35) (see Web Appendix 1, available at https://academic.oup.com/aje, for additional details on nutrition context). The study population included all children between 3 and 59 months of age residing in Zimbabwe and living with at least 1 parent at the time of the survey. We excluded children with parents born before 1957 and after 1977 to maximize the comparability between pre- and postreform cohorts. Using a narrow subsample of pre- and postreform cohorts, there were good reasons to believe that control cohorts differed from exposed cohorts only with respect to access to secondary school. Web Figure 1 outlines the selection of study participants.

Outcomes

Our outcome of interest was anthropometric failure of the child at the time of the survey, defined as early childhood stunting (i.e., too short for age, an indicator of chronic malnutrition), wasting (i.e., too thin for height, an indicator of acute malnutrition), and underweight (i.e., weight-for-age, a composite index). As a baseline, we show anthropometric measures based on the US National Center for Health Statistics growth reference population included all children between 3 and 59 months of age residing in Zimbabwe and living with at least 1 parent at the time of the survey. We excluded children with parents born before 1957 and after 1977 to maximize the comparability between pre- and postreform cohorts. Using a narrow subsample of pre- and postreform cohorts, there were good reasons to believe that control cohorts differed from exposed cohorts only with respect to access to secondary school. Web Figure 1 outlines the selection of study participants.

Exposure

The key exposure in our analysis was total years of parental schooling completed by the time of the survey. In all models, we assessed maternal and paternal schooling separately.

Exogenous instrument

To obtain causal effects, we exploited exogenous variation in educational attainment resulting from a 1980 policy reform that reduced academic and structural restrictions limiting advancement toward secondary school (33, 34). In 1980, following independence, Zimbabwe rapidly expanded access to secondary schools for black Zimbabweans. The left-leaning politics of the new Mugabe government put an emphasis on equality through education. The government initiated a series of reforms, including automatic grade progression from primary school to secondary school, and a large secondary school construction program, focused on rural areas (38, 39). While expansions in education occurred at both the primary and secondary levels, the expansion of secondary schools was by far the most dramatic. As a result of the reform, the number of primary schools almost doubled, from 2,401 to 4,291, while the number of secondary schools increased dramatically, from 177 to 1,276. Enrollment in secondary school more than doubled, increasing from 74,746 in 1980 to 149,018 in 1981 (40) (see
This "natural experiment" provided us with an opportunity to estimate the causal impact of parental schooling on early childhood undernutrition, by comparing children of parental birth cohorts at least partially exposed to the reform with the children of those unexposed. We defined 2 mutually exclusive indicators as our instruments: an indicator—"fully exposed"—taking the value 1 if the parent was born in a cohort exposed to the 1980 education policy reform and 0 otherwise (year of birth ≥1967), and an indicator—"partially exposed"—if the parent was partially exposed to the reform (year of birth 1963–1966).

Table 1. Selected Characteristics of Participants in a Study of the Causal Effect of Parental Schooling on Early Childhood Undernutrition, Zimbabwe, 1988–2011

<table>
<thead>
<tr>
<th>Subsample and Characteristic</th>
<th>Parental Birth Cohort</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of Persons % Mean (SD)</td>
</tr>
<tr>
<td>Living with eligible mother</td>
<td></td>
</tr>
<tr>
<td>Age, months</td>
<td>1,213 (32.7 (16.6))</td>
</tr>
<tr>
<td>Female sex</td>
<td>605 (49.6)</td>
</tr>
<tr>
<td>Stunted</td>
<td>342 (28.6)</td>
</tr>
<tr>
<td>Underweight</td>
<td>149 (11.8)</td>
</tr>
<tr>
<td>Wasted</td>
<td>42 (3.5)</td>
</tr>
<tr>
<td>Living with eligible father</td>
<td></td>
</tr>
<tr>
<td>Age, months</td>
<td>646 (33.5 (16.7))</td>
</tr>
<tr>
<td>Female sex</td>
<td>327 (50.3)</td>
</tr>
<tr>
<td>Stunted</td>
<td>175 (27.9)</td>
</tr>
<tr>
<td>Underweight</td>
<td>89 (14.1)</td>
</tr>
<tr>
<td>Wasted</td>
<td>37 (5.7)</td>
</tr>
<tr>
<td>Mothers</td>
<td></td>
</tr>
<tr>
<td>Age, years</td>
<td>1,948 (37.3 (7.3))</td>
</tr>
<tr>
<td>≥1 year of schooling</td>
<td>1,498 (77.9)</td>
</tr>
<tr>
<td>Duration of schooling, years</td>
<td>413 (4.7 (3.6))</td>
</tr>
<tr>
<td>Wealth index</td>
<td>2.6 (1.4)</td>
</tr>
<tr>
<td>Fathers</td>
<td></td>
</tr>
<tr>
<td>Age, years</td>
<td>1,138 (40.4 (6.2))</td>
</tr>
<tr>
<td>≥1 year of schooling</td>
<td>1,049 (93.2)</td>
</tr>
<tr>
<td>Duration of schooling, years</td>
<td>345 (7.5 (3.7))</td>
</tr>
<tr>
<td>Wealth index</td>
<td>3.0 (1.5)</td>
</tr>
</tbody>
</table>

Abbreviations: DHS, Demographic and Health Surveys; SD, standard deviation.

a Prior to the 1980 education reform, which increased access to secondary education.
b After the 1980 education reform, which increased access to secondary education.
c Sample weights provided by the DHS Program were used to calculate percentages.
d The child sample included children who were between the ages of 3 and 59 months at the time of the survey, were living with a parent born in 1957–1977, and had valid anthropometric data.
e A child below −2 standard deviations from the median of the National Center for Health Statistics reference population in terms of height-for-age was considered stunted. Similar calculations were performed for wasting and underweight.
f The parent sample included parents born between 1957 and 1977 who lived with a child under the age of 5 years. In the 1988 survey, men were not included and wealth index was not calculated.
g The wealth index is a composite measure of a household’s cumulative living standard. It is calculated using easy-to-collect data on a household’s ownership of selected assets, such as materials used for housing construction and types of water access and sanitation facilities. Generated based on principal components analysis, the wealth index places individual households on a continuous scale of relative wealth. In the DHS, interviewed households are separated into 5 wealth quintiles (5 being the wealthiest).
have entered secondary school in 1980 or later, and were thus not exposed to the reform in multivariable ordinary least squares (OLS) regression models (41). The assumption that underlying trends are approximately linear is more plausible when narrower windows of cohorts are included. We also assessed the robustness of our results to the exclusion of parental cohorts that were partially exposed to the reform, including only parents with at least primary schooling (i.e., those most likely to have benefitted from the reform), including the full sample of parental cohorts, including the 1994 DHS survey (which only collected anthropometric data for children aged <3 years), and modeling the outcome using a probit link function. We added indicators for child characteristics (age, sex, and birth order) and region as additional controls in our main model. We also controlled for an indicator for heap year to take into account measurement error in parental age due to age heaping. We excluded parental ages that were multiples of 5, since we still had all parental years of birth in our analysis sample (the surveys were conducted in more than 5 different years).

We also considered a wide range of additional outcomes. We tested the sensitivity of our results to the new World Health Organization growth standards (36), using separate files released by the DHS which contain the new z scores for older DHS surveys. We assessed continuous measures for height-for-age z scores, weight-for-age z scores, and weight-for-height z scores, “severe” undernutrition (defined as z scores less than −3), and overweight (defined as z scores greater than 2). We also used an alternative identification strategy. We fitted difference-in-differences models exploiting the differential impact of the policy reform by geographic region (see Web Appendix 3 and Web Figure 3 for additional details). Finally, we assessed the nutritional outcomes of children whose parents were both fully exposed to the reform (Web Appendix 4).

### Covariates

We controlled flexibly for parental age with single-year age indicators to account for differential patterns of schooling and undernutrition by parental age. We controlled for survey year indicators to account for time-varying shocks that affected respondents differently across survey years.

### Statistical analyses

Our analysis of the education reform proceeded in 3 steps. First, we assessed whether parental birth cohorts exposed to the reform had higher educational attainment than birth cohorts not exposed to the reform in multivariable ordinary least squares (OLS) regression models (42). We also assessed the association between exposure to the reform and the probabilities of completing at least 1, 7, 9, 11, 13, and 14 or more years of schooling. Second, we assessed the intention-to-treat relationship between being in a parental reform cohort and undernutrition in one’s children. Third, we fitted 2-stage least squares (2SLS) regression models, using parental exposure to the reform as an instrumental variable (IV) for years of schooling while adjusting for covariates.

Natural experiments that change the probability of an exposure can be analyzed like randomized controlled trials with non-compliance (43). Under the assumptions discussed in Web Appendix 3 (see Web Figure 2 for a directed acyclic graph), the treatment effect among compliers is the ratio of the intention-to-treat and the difference in the probability of receiving treatment. These IV estimates are interpreted as specific to the subpopulation that complied with their treatment assignment—that is, parents who increased their duration of schooling because of the policy reform (44). Our IV analysis of the reform is akin to a “fuzzy” regression discontinuity design, where treatment is assigned probabilistically (45). As a benchmark for our 2SLS estimates, we assessed the naive association between parental schooling and undernutrition in their children. We assessed the naive relationship graphically and then adjusted for covariates. We clustered standard errors by survey and primary sampling unit to account for spatial correlations between respondents.

### Sensitivity analyses

As a first robustness check, we reduced the window of observation to a narrower set of parental birth cohorts (3-year and 5-year birth cohort windows around the reform cutoff). The assumption that underlying trends are approximately linear is more plausible when narrower windows of cohorts are included. We also assessed the robustness of our results to the exclusion of parental cohorts that were partially exposed to the reform, including only parents with at least primary schooling (i.e., those most likely to have benefitted from the reform), including the full sample of parental cohorts, including the 1994 DHS survey (which only collected anthropometric data for children aged <3 years), and modeling the outcome using a probit link function. We added indicators for child characteristics (age, sex, and birth order) and region as additional controls in our main model. We also controlled for an indicator for heap year to take into account measurement error in parental age due to age heaping. We excluded parental ages that were multiples of 5, since we still had all parental years of birth in our analysis sample (the surveys were conducted in more than 5 different years).

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### Heterogeneity

We assessed heterogeneity of the effect of parental schooling on undernutrition in their children. We applied our main model to a range of subsamples, including households with a high (quintiles 4 and 5) and low (quintiles 1 and 2) wealth index and households in urban and rural settings and restricting the sample of children to alternative age groups (3–36 months, 3–24 months).
The 1988, 1999, 2005–2006, and 2010–2011 DHS surveys included 8,243 children with data on anthropometric failure and parental characteristics—6,428 with an eligible mother and 4,175 with an eligible father (Table 1, Web Figure 1).

The reform increased the average duration of schooling by 2.6 years (95% confidence interval (CI): 2.2, 2.9) for mothers fully exposed to the reform and 2.5 years (95% CI: 2.0, 3.0) for fathers fully exposed, and by 0.9 years (95% CI: 0.7, 1.2) for mothers partially exposed to the reform and 1.8 years (95% CI: 0.7, 3.0) for fathers partially exposed.

**RESULTS**

The 1988, 1999, 2005–2006, and 2010–2011 DHS surveys included 8,243 children with data on anthropometric failure and parental characteristics—6,428 with an eligible mother and 4,175 with an eligible father (Table 1, Web Figure 1).

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Table 3. Ordinary Least Squares and 2-Stage Least Squares Regression Results for the Causal Effect of Parental Duration of Schooling on Early Childhood Undernutrition, Zimbabwe, 1988–2011a

<table>
<thead>
<tr>
<th>Parental Exposure Category and Model</th>
<th>Child’s Anthropometric Outcome</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>β</td>
<td>95% CI</td>
<td>β</td>
</tr>
<tr>
<td>Model 1: OLS</td>
<td></td>
<td>−1.6</td>
<td>−1.9, −1.2</td>
<td>−1.1</td>
</tr>
<tr>
<td>Model 2: 2SLS (IV)</td>
<td></td>
<td>0.8</td>
<td>−2.9, 4.5</td>
<td>0.9</td>
</tr>
<tr>
<td>Maternal Schooling (n = 6,428)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 1: OLS</td>
<td></td>
<td>−1.0</td>
<td>−1.5, −0.5</td>
<td>−1.0</td>
</tr>
<tr>
<td>Model 2: 2SLS (IV)</td>
<td></td>
<td>2.6</td>
<td>−1.5, 6.8</td>
<td>0.2</td>
</tr>
<tr>
<td>Paternal Schooling (n = 4,175)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviation: CI, confidence interval; IV, instrumental variable; OLS, ordinary least squares; 2SLS, 2-stage least squares.

a Model 1 was an OLS linear probability model. Model 2 was a 2SLS linear probability model in which exposure to the reform was used as an IV for parental duration of schooling (in years). All models included an indicator for parental age and an indicator for survey year. In model 2, we used 2 instruments: an indicator for partial exposure to the 1980 education reform (parental year of birth 1963–1966) and an indicator for full exposure to the reform (parental year of birth ≥1967). The sample included children who were between the ages of 3 and 59 months at the time of the survey and were living with a parent born in 1957–1977. F-statistics were 29.7 for maternal schooling and 21.2 for paternal schooling.

Sensitivity analyses

Table 4 and Web Table 3 display the results of sensitivity analyses for the intention-to-treat results; Web Tables 4–7 display the results of sensitivity analyses for the 2SLS results. In general, our results were not sensitive to accounting for parental age heaping, different specifications and definitions of the outcome, alternate sample specifications (using a narrower birth cohort window around the reform cutoff, excluding parents who were partially exposed to the reform, including only those parents with at least 7 years of schooling, and including the full sample of parents), the inclusion of additional controls for a child’s age, sex, and birth order, region indicators, use of an alternative identification strategy, or having both parents fully exposed to the reform. Using alternate sample definitions, additional maternal schooling slightly decreased the probability of a woman’s child being wasted (0.8–1.4 percentage points) (Web Table 4, columns 5, 6, and 9), whereas, using the new World Health Organization criteria, additional maternal schooling increased the probability of her child’s being stunted (3.5 percentage points, 95% CI: 0.1, 6.9) (Web Table 5). A small effect was seen for additional paternal schooling on weight-for-age z scores and weight-for-height z scores, but these coefficients did not reach conventional benchmarks for statistical significance (Web Table 6). Similar to our main results, the overall impression of these estimates was that parental schooling had no effect on early childhood undernutrition at the national level.

Heterogeneity

Table 5 shows 2SLS results for the effect of parental schooling on undernutrition in their children in subsamples (see Web Table 8 for intention-to-treat results). Results were consistent across children in households that were most at risk of early childhood undernutrition: those located in rural settings, those in the lowest wealth quintiles (quintiles 1 and 2), and those with

1.4, 2.2) for fathers partially exposed (Table 2; see Web Table 1 and Web Figure 4 for results by schooling level).

Web Table 2 presents intention-to-treat results, in which childhood undernutrition was regressed directly on the instrument and covariates. We found no significant differences in the probability of early childhood stunting, wasting, or underweight for children of parents who were fully exposed to the reform compared with children of parents who were not exposed. Figure 1 displays undernutrition by parental year of birth and full exposure to the education reform, separately for maternal and paternal exposure.

The crude association between additional parental schooling and undernutrition in their children was nonlinear, particularly among fathers: Children of fathers with 6–7 years of education had the highest risk, with risk declining sharply thereafter for children of fathers with 8 or more years of education (Web Figure 5). The association between parental schooling and anthropometric failure persisted in multivariable ordinary least squares regression models (model 1, Table 3). Each additional year of maternal schooling was associated with a decrease of 1.6 percentage points (95% CI: −1.9, −1.2) in the probability of a woman’s child being stunted, 1.1 percentage points (95% CI: −1.4, −0.8) in the probability of the child’s underweight, and 0.3 percentage points (95% CI: −0.4, −0.1) in the probability of the child’s being wasted. Each additional year of paternal schooling was associated with a decrease of 1.0 percentage points (95% CI: −1.5, −0.5) in the probability of a man’s child being stunted, 1.0 percentage points (95% CI: −1.3, −0.6) in the probability of the child’s being underweight, and 0.3 percentage points (95% CI: −0.5, −0.1) in the probability of the child’s being wasted. In contrast to the negative association between maternal schooling and all 3 measures of anthropometric failure in ordinary least squares regression analyses, we found no evidence of a causal effect of parental schooling in 2SLS (IV) regression analyses (model 2, Table 3).
Table 4. Intention-to-Treat Regression Results for the Causal Effect of Parental Duration of Schooling on Early Childhood Undernutrition (Sensitivity Analyses), Zimbabwe, 1988–2011

<table>
<thead>
<tr>
<th>Parental Exposure Category and Child’s Anthropometric Outcome</th>
<th>Sensitivity Analysis Model</th>
<th>Sensitivity Analysis Model</th>
<th>Sensitivity Analysis Model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Model 1: Region Indicator</td>
<td>Model 2: Probit Link Function</td>
<td>Model 3: 3-Year Cohort Window</td>
</tr>
<tr>
<td>No. of Persons</td>
<td>β</td>
<td>95% CI</td>
<td>No. of Persons</td>
</tr>
<tr>
<td>Maternal exposure</td>
<td>6,428</td>
<td>3.3</td>
<td>−5.2, 11.7</td>
</tr>
<tr>
<td>Stunted</td>
<td>6,428</td>
<td>2.2</td>
<td>−4.4, 8.8</td>
</tr>
<tr>
<td>Underweight</td>
<td>−3.0</td>
<td>−0.9</td>
<td>−3.0</td>
</tr>
<tr>
<td>Wasted</td>
<td>4,175</td>
<td>7.7</td>
<td>−1.9, 17.3</td>
</tr>
<tr>
<td>Paternal exposure</td>
<td>4,175</td>
<td>0.0</td>
<td>−7.2, 7.2</td>
</tr>
<tr>
<td>Wasted</td>
<td>−1.9</td>
<td>−5.7, 2.0</td>
<td>−2.5</td>
</tr>
<tr>
<td>Model 4: 5-Year Cohort Window</td>
<td>3,612</td>
<td>14.1</td>
<td>−5.0, 33.3</td>
</tr>
<tr>
<td>Paternal exposure</td>
<td>2,262</td>
<td>18.4</td>
<td>−2.1, 39.0</td>
</tr>
<tr>
<td>Wasted</td>
<td>−5.2</td>
<td>−4.1, 14.4</td>
<td>−4.7</td>
</tr>
<tr>
<td>Model 5: Excluding the Partially Exposed</td>
<td>3,592</td>
<td>18.2</td>
<td>−12.2, 4.4</td>
</tr>
<tr>
<td>Model 6: Full Sample</td>
<td>6,832</td>
<td>18.2</td>
<td>−12.2, 4.4</td>
</tr>
</tbody>
</table>

Table continues
Table 4. Continued

<table>
<thead>
<tr>
<th>Model 7: Child Age, Sex, and Birth Order</th>
<th>Model 8: ≥7 Years of Schooling</th>
<th>Model 9: Including the 1994 DHS&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of Persons</td>
<td>β</td>
<td>95% CI</td>
</tr>
<tr>
<td>Maternal exposure</td>
<td>6,182</td>
<td>4,228</td>
</tr>
<tr>
<td>Stunted</td>
<td>4.3</td>
<td>−4.4, 12.9</td>
</tr>
<tr>
<td>Underweight</td>
<td>3.7</td>
<td>−3.2, 10.5</td>
</tr>
<tr>
<td>Wasted</td>
<td>−1.9</td>
<td>−5.8, 2.1</td>
</tr>
<tr>
<td>Paternal exposure</td>
<td>3,879</td>
<td>3,580</td>
</tr>
<tr>
<td>Stunted</td>
<td>7.4</td>
<td>−2.1, 17.0</td>
</tr>
<tr>
<td>Underweight</td>
<td>−0.0</td>
<td>−7.3, 7.3</td>
</tr>
<tr>
<td>Wasted</td>
<td>−1.7</td>
<td>−5.5, 2.2</td>
</tr>
</tbody>
</table>

Model 10: Parental Heap Year Indicator

| No. of Persons | β | 95% CI | No. of Persons | β | 95% CI | No. of Persons | β | 95% CI |
| Maternal exposure | 6,428 | 5,046 | 6,428 |
| Stunted | 3.3 | −5.2, 11.8 | 1.2 | −7.9, 10.3 | −13.0 | −38.7, 12.7 |
| Underweight | 2.2 | −4.6, 9.0 | 0.4 | −6.6, 7.4 | −10.5 | −29.5, 8.5 |
| Wasted | −2.9 | −6.9, 1.1 | −3.2 | −7.8, 1.4 | 1.3 | −7.8, 10.4 |
| Paternal exposure | 4,175 | 3,267 | 4,175 |
| Stunted | 7.0 | −2.5, 16.5 | 10.5 | 0.1, 21.0 | 8.9 | −42.4, 60.3 |
| Underweight | −1.8 | −5.8, 2.1 | 0.9 | −6.8, 8.6 | 2.2 | −36.6, 41.0 |
| Wasted | −0.6 | −7.8, 6.6 | −1.2 | −5.5, 3.0 | 8.4 | −15.0, 31.7 |

Abbreviations: CI, confidence interval; DHS, Demographic and Health Survey; N/A, not available.

<sup>a</sup> Robustness checks for the intention-to-treat results presented in Web Table 2. Models 1 and 3–12 show regression results from intention-to-treat ordinary least squares models controlling for indicators for parental age and survey year. Model 1 additionally controlled for a region indicator. Model 2 was a probit model. Model 10 additionally controlled for a parental heap year indicator. Model 12 additionally controlled for an indicator for reform exposure and a region indicator. Full exposure to the reform was defined as 1 if parental year of birth was 1967 or later and as 0 otherwise. The sample included children who were between the ages of 3 and 59 months at the time of the survey and were living with a parent born in 1957–1977.

<sup>b</sup> The 1994 DHS only collected anthropometric data for children aged <3 years (as opposed to <5 years) who lived with their mothers.

<sup>c</sup> Exposure in model 12 was the interaction between an indicator for being exposed to the 1980 education reform and a continuous variable for the percentage of respondents with zero years of schooling in each region during the prereform period.
children aged 3–36 months (Web Figure 6). For households in the urban and high-wealth-index subsamples, each additional year of maternal schooling induced by the policy reform led to absolute reductions in the probability of a woman’s child being wasted of 5.2 percentage points (95% CI: −9.3, −1.2) and 3.6 percentage points (95% CI: −6.9, −0.4), respectively; for children between the ages of 3 and 23 months, each additional year of paternal schooling increased the probability of a man’s child being stunted by 9.6 percentage points (95% CI: 1.4, 17.9).

**DISCUSSION**

Using an education policy reform in Zimbabwe as a natural experiment, we found that additional parental schooling had no effect on the probability of stunting, wasting, or underweight in participants’ children under age 5 years at the national level. Findings were generally consistent across a wide range of sensitivity analyses. For households in the urban and high-wealth-index subsamples, each additional year of maternal schooling had a protective effect against the probability of being wasted in children, whereas among children aged 3–23 months, each additional year of paternal schooling increased the probability of being stunted. We interpreted our 2SLS (IV) estimates as causal under the assumptions outlined in Web Appendix 3. While the education reform allowed us to identify the causal effects of interest, relying on changes generated by the policy reform came with some limitations in terms of external validity. First, most of the variation explored reflected variation in

secondary schooling. Effects of schooling may be qualitatively and quantitatively different in primary school (46, 47). Indeed, in our descriptive analysis, we found the association between parental schooling and childhood undernutrition to be nonlinear, particularly among fathers: An additional year of primary school was associated with an increase in the probability of stunting and underweight, but secondary school and beyond was associated with a reduction in the probability of stunting and underweight (Web Figure 5). Second, the causal effects that we estimated were specific to the subpopulation of compliers—that is, those induced to increase schooling because of the reform. Third, the results were specific to the context and years of exposure under study (the 1970s through the 1990s). Nevertheless, one advantage of using Zimbabwe’s supply-side school reform was the important size of its impact (over 2 years of additional schooling at the national level for those fully exposed) and the fact that it affected a very large segment of the population who would otherwise not have enrolled in school.

Schooling may not affect all subpopulations equally, for several reasons. Parents might not invest in behaviors that are protective of child health if they are unable to anticipate the important labor market (48) or nonpecuniary (49) returns of schooling. Stunting and wasting also represent distinct biological processes, and the pathways by which parental schooling may influence these outcomes are probably different (50). Wasting is typically acute and is associated with more important muscle and fat loss than is stunting, possibly alerting parents more quickly to change their feeding practices or seek health care to correct undernutrition in their children (51). In settings where health services are limited, better-educated parents are more likely to rely on their ability to adequately evaluate their child’s growth and to take corrective action when needed (47). In further analysis, we found protective effects of maternal schooling on early childhood wasting in urban and wealthier households (but not on early childhood stunting or underweight), suggesting that in settings with increased resources, maternal schooling can be a determinant of early childhood wasting. Additional maternal schooling and access to resources may be complements in reducing the risk of childhood wasting, rather than substitutes as has been argued elsewhere (52). Another reason for these positive results could be that the margin of schooling affected by the reform in this subgroup was a key margin for spillover effects to children. In urban settings, the reform increased access to higher levels of schooling (such as middle secondary school and beyond). These higher levels of schooling may be a “critical period” for future parents (53), when increased access to education can reduce wasting in their children.

Our findings have a number of key implications for population health. First, this study contributes to the broader literature on the causal effect of education on health (54–59). Education and health are strongly correlated, but there is no consensus on whether more schooling is a causal determinant of better health. Our findings suggest that parental schooling may play a more muted role in parents’ decisions about whether and how much to invest in their children’s health than previously suggested (2–11). Second, in addition to the question of whether education causally improves health, it is unclear which levels of schooling are most important. Our findings contribute to knowledge about the causal effects of secondary schooling in particular. Third, while much of the literature has focused on the benefits of education for children themselves (60, 61), our study contributes to knowledge about the extent to which these educational benefits are shared (or not) across generations. Finally, we found a protective effect of maternal schooling on wasting among wealthier households (but not among the poor), suggesting that the reform may have widened existing health disparities (62–67).

Expanding access to secondary school had no effect on childhood stunting, wasting, or underweight at the national level in Zimbabwe. This study suggests that while secondary schooling is important in its own right, it may not be enough to improve early childhood anthropometric measures in low-resource settings.

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Author affiliations: Institute of Public Health, Heidelberg University, Heidelberg, Germany (Jan-Walter De Neve); Department of Global Health and Population, Harvard T.H. Chan School of Public Health, Boston, Massachusetts (Jan-Walter De Neve); Department of Social and Behavioral Sciences, Harvard T.H. Chan School of Public Health, Boston, Massachusetts (S. V. Subramanian); and Center for Population and Development Studies, Harvard T.H. Chan School of Public Health, Boston, Massachusetts (S. V. Subramanian).

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