Lowering child mortality in poor countries:

The power of knowledgeable parents

Peter Boone$^1$

and

Zhaoguo Zhan$^2$

May 2006

What permits some poor communities to have levels of child mortality as low as those in wealthy countries, while in others more than 20% of children die before age five? We examine this question using survey data from 278,000 children in 45 low-income countries. We find that the prevalence of common diseases that kill children, such as diarrhea and respiratory infections, has little predictive power for child mortality. In fact, our work suggests that actions taken by parents and health professionals, after a child is sick, are the most important factors determining cross country differences in child survival. We calculate that improvements in treatment-seeking and education could reduce child mortality by roughly 32%, while improved sanitation and water in all households would reduce child mortality by just 3%. We also examine whether a large expansion of the public health sector, as called for by several well-publicized studies, is warranted by empirical evidence. Using a GINI coefficient we construct for treatment services, we find that public and private health systems are “equally unequal”, that is, neither type appears superior at improving mortality outcomes in very poor communities; and we show that many low-income countries have achieved large mortality declines with large private sector, or self-financed, health services. We conclude that improved education and health knowledge, targeted to the highest risk groups, is necessary, and is the most promising intervention for achieving sustainable, large declines in child mortality.

---

$^1$ Centre for Economic Performance, London School of Economics and Effective Intervention

$^2$ Centre for Economic Performance, London School of Economics.
I. Introduction

What explains the large differences in child mortality rates within and across countries? How can we design packages of reforms that lead to large declines in child mortality? These are extremely important questions and yet, due to the complex combination of socioeconomic, political and medical issues they touch, we are far from having clear answers.

Figure 1 shows the international pattern of child mortality. In middle and low-income countries, the probability of a child dying before age 5 varies from 2% to 22%. Even after controlling for income, there is large variation in mortality rates that is not well-explained. Within India, the state of Kerala has rates that approach west European levels, while other Indian states match some of the poorest Sub-Saharan African nations. The existence of poor communities which have low mortality levels suggests large reductions in mortality are feasible, even if very few poor countries have achieved large reductions.

When considering what may be effective at reducing mortality, the gold standard for evidence is that used by the medical profession and related regulatory agencies: the randomized controlled trial. This literature suggests there are several potential routes to low mortality. Neonatal deaths make up 20-40% of child deaths in poor countries, with most of these due to sepsis and birth asphyxiation. If a child survives past its first week, the main causes of death up to the age of five are diarrhea, pneumonia and malaria.

Evidence from trials suggests neonatal deaths could be prevented through better delivery techniques and improved hygiene (Darmstadt, Bhutta et al. 2005). Infant and child diseases could be sharply reduced through improved water, sanitation and hygiene alongside vector control to stop malaria. Even if a child falls ill, trials demonstrate it is possible to prevent deaths through early treatment. Diarrhea and pneumonia, if captured early enough, can usually be successfully treated through oral rehydration therapy (ORT) and antibiotics. The right anti-malarials, given early enough, can prevent malaria fatalities (Bryce, Black et al. 2005).

Trial results can be used, as was done by the Bellagio Child Survival Study Group, to come up with a list of interventions with good evidence that each individual component contributes to morbidity or mortality decline (Jones, Steketee et al. 2003). With a leap that goes beyond the evidence from most
intervention trials, we can make assumptions regarding the likely impact of multiple intervention measures on all-cause mortality, and then draw conclusions about what the likely impact of a policy package is.

However, from a policy perspective, such lists leave many questions unanswered. First, we can’t be sure that the ideal operating conditions of an intervention trial were truly equivalent to real world environments. Second, even if they were, we are also faced with institutional, political and social constraints which make many interventions difficult to implement (Hanson, Ranson et al. 2001). Finally, even if they are implemented, sustainability may be limited due to the host of problems that plague poor countries and help explain why, to date, so much remains to be accomplished.

An alternative means to examine how to reduce child mortality is to pose the question: What appears to have worked in many countries, and what has not? If we can find robust relations within and across countries, and over time, this would be useful knowledge. If these relations are consistent with health intervention trials this would boost those findings, while if they are inconsistent, we would need to question how replicable the ideal conditions of intervention trials are in the real world.

In this paper we use household survey data, from 278,000 children in 45 countries to address this agenda. We ask two basic questions: First, can we explain the large differences in child mortality rates by differences in morbidity rates, i.e. how often children get sick, or by differences in treatment patterns and other factors that could prevent mortality once a child has been exposed to disease? Second, what are the implications for the design of intervention programs aimed at sharply reducing child mortality?

The first question helps us better understand whether general morbidity levels must be tackled in order to bring down mortality rates, or whether encouraging guardians to better recognize and adequately treat their children for sickness is a more effective route to lower mortality.

The second question leads us to focus in particular on whether supply or demand-oriented measures are needed. The major intervention programs designed by the Bellagio Child Survival Study Group, and the Commission on Macroeconomics and Health, advocated large scale supply oriented projects (Commission on Macroeconomics and Health 2001). These call for a major expansion of public health facilities, personnel, and purchases of
medicines and equipment, costing $6bn to $57bn annually in donor funding, to ensure that there are available resources to cover 100% of the population with essential preventative and treatment interventions in low-income countries.

Such projects have received criticism because they require enormous government fiscal and institutional efforts which may be difficult to sustain in low-income, politically unstable countries. Further, there is a risk that, despite the best intentions of health planners, marginalized groups may still be effectively excluded from such services, or will not utilize them, because they lack the political strength that comes with greater education and wealth. These concerns, we argue, are consistent with the very strong correlation between education, treatment-seeking, and child mortality outcomes that we find in the data, and hence should not be ignored when designing schemes to improve child health.

An alternative project would be to target the education, health knowledge, and treatment-seeking of marginalized groups themselves so as to raise their demand for health services and thereby reduce mortality. This alternative strategy puts into question whether overarching public health systems need to be created before community health can be significantly improved.

We address these issues using survey data. Survey data has, admittedly, many problems, with the most important being the potential for misleading conclusions due to a lack of sufficient controls. However the advantage is that it permits us to examine outcomes in “general equilibrium”, i.e. taking into account all the socioeconomic, institutional and medical factors that may determine whether specific interventions ultimately work.

The Demographic Health Survey (DHS) data we use covers 45 low-income countries and includes over one thousand questions on individuals, households and villages that permit us to develop good control variables. We also consider instruments and alternative specifications for equations to examine robustness of outcomes. This permits us to deal with some of the important concerns about using survey data.

We motivate our empirical approach by the simple figure below.

---

3 The data is available at www.measuredhs.com
Stages to child death

In order to die a child must be exposed to a pathogen or risk, and then develop clinical disease. If the child develops clinical disease, she may be treated, and conditioned on treatment, she will either survive or die. The probability of a child dying then depends on the joint, conditional probabilities of passing through each of these stages.

To determine whether differences in the probability of child death are driven by morbidity risks or treatment patterns in our data, we rank each household according to their propensity to treat children for disease, and we also directly (and indirectly) measure morbidity for the key diseases that kill children. We run regressions within and across countries.

Our empirical results demonstrate consistently that differences in treatment indicators, followed by maternal and paternal education, are the most important variables predicting child mortality in our regressions. We calculate that if all mothers and fathers in our sample received 6.3 and 8.3 years of schooling respectively, matching the current levels in Egypt, child mortality would fall by 19%. In addition, if treatment-seeking behavior in all households was raised to the 53rd percentile of the population in our 45 countries, again matching the level in Egypt, child mortality would fall an additional 13%.

However, we find that morbidity indicators, along with improved water and sanitation, have relatively minor impacts on child survival. We estimate that if the incidence of diarrhea, acute respiratory infections, and fever were to fall by half in every region of our sample, total child mortality would fall by 3%. If all households were allocated improved water and sanitation, child mortality would fall by 2%.

---

4 This experiment assumes those with greater education, and those above the 53rd Treatment percentile as described in section 7, remain at their current levels.
If we embark on a project to improve education and treatment-seeking, do we need also to invest heavily in public health services? To answer this question we examine the relative performance of public and private systems in low-income countries.

We find no evidence that nations, or regions, with relatively large public health systems perform better or worse than regions with large private systems. We find that public-based systems are as “biased”, in terms of the distribution of health services, as private-based systems. They both tend to serve well-educated wealthy people better than less educated poor people.

There are numerous examples of countries and regions with large private, or self-financing, child healthcare services that have achieved very low mortality rates. This even includes socialist countries such as China and Vietnam, along with Egypt, Indonesia and the Indian state of Kerala. In these countries and regions, demand factors have clearly driven the supply of health services, showing that it is possible to achieve large child mortality reductions through demand-based policies, while letting the private sector expand to meet supply.

The remainder of the paper is organized as follows: Section 2 presents background information on the causes of child deaths. Section 3 describes our empirical framework and the logic behind our choice of regression equations. Section 4 describes our dataset and defines key variables. Section 5 presents our regression results. In section 6 we examine robustness and causality, and we also examine the plausibility of our key findings based on intervention trials and other literature. Section 7 examines the implications for the design of intervention packages, and evidence regarding the need for building out public health services. Section 8 presents our conclusions.

2. Basic facts on the causes of child mortality and our empirical framework

In order to interpret our empirical work, it is important to start with some basic facts regarding the causes and timing of child deaths. Figure 2 shows the probability of dying for a child from birth, up to age 10, as calculated from the DHS data in 45 countries. It is clear from this data that the

---

5 The chart records the percentage of children who were in that age category 12 months prior to the survey and who subsequently died.
greatest risk is around the time of birth, and risk stays “high” until one year of age, after which the probability of dying falls to near zero by age five.

The causes of death are not recorded in the DHS survey, although there is recent World Health Organization (WHO) data, and work by the Bellagio Child Survival Study Group, which provides consistent estimates (Black, Morris et al. 2003). Figure 3 shows these estimates for the distribution of deaths for neonates, and for non-neonates. For non-neonates, two thirds of deaths are due to diarrhea and acute respiratory infections, 14% are due to malaria and the remaining 21% to a range of smaller other causes. Note that only 0.4% of child deaths are attributed to vaccine preventable disease. Neonatal deaths are primarily attributed to birth asphyxiation, sepsis and congenital problems.

3. Empirical Framework

Our empirical framework is based on a simple statistical model that divides the probability of death between the risk of exposure to disease, and the risk of death conditioned on exposure. We then expand this to examine consumer choice, and the desired level of treatment in section 3.3.

3.1 The determinants of the probability of a child death in a household

The probability that a child dies over a short period of time, \( P^D \), can be expressed as the joint probability of two events: that the child be exposed to a risk or pathogen causing disease, and, conditional on having been exposed, that the child dies:

\[ P^D = P_{\text{exposure}} \times P_{\text{death|exposure}} \]

6 The bulk of neonatal and post-neonatal deaths are preventable with fairly simple treatments. In Section 6 we discuss the treatment regimes for the main non-neonatal causes of deaths. Neonatal diseases are also preventable, with more than half related to poor delivery techniques and infections acquired around birth. These can be prevented through improved hygiene and trained birth attendants. Congenital problems and preterm deliveries can also be reduced through better antenatal and neonatal care (see Lawn, J. E., S. Cousens, et al. 2005).
\begin{equation}
P_D(X,T) = P_E(X) \times P_D|E(X,T)
\end{equation}

\begin{itemize}
\item \(X\): Vector of household and child characteristics
\item \(T\): Index of treatment pattern in response to exposure to risks or disease
\item \(P_D\): Probability that child dies
\item \(P_E\): Probability that child is exposed to disease
\item \(P_D|E\): Probability that child dies after having been exposed to disease
\end{itemize}

Here we include a vector of conditioning variables, \(X\), such as household wealth and parent’s education, that impact each probability along with a variable \(T\), which measures the quantity of treatment that this household provides to children when they are sick.

In reality, children will be exposed to multiple pathogens during their lifetime. Suppose we prospectively plan to observe each child, \(k\), for \(M_k\) periods, and suppose there are \(J\) different pathogens that could cause deaths. Then we can write the probability that child \(k\) dies during the \(M_k\) observation periods as:

\begin{equation}
P_D^k = 1 - \prod_{j=1}^{J} \left[1 - P_E^{jk} \cdot P_{D|E}^{jk}\right]^{M_k}
\end{equation}

It is clear from (2) that the relation between morbidity levels and treatment to mortality risk could each be large or small. If some households have access to treatment technologies that permit them to cure all disease episodes, then changes in morbidity will have only a small impact on the probability of death for those households. When we compare households with access to this technology, to households without it, we’ll find that proxies for access to this technology will explain the greatest differences in mortality risk.

\footnote{We assume here for simplicity, without loss of generality, that the mortality risk from each exposure is independent of other exposures. Since our regression results are based on a linearization (or logistic transformation) of the true distribution, the coefficients we derive may reflect more complex relations in the data.}
On the other hand, if treatment regimes are poor in every household, we may find that morbidity levels explain the bulk of differences in mortality. These relations may be similar across regions and countries or highly variable, so it is an empirical question to see whether there are common trends within and across countries.

Our main regressions are based on variants of equations (2). We run regressions with child survival outcome as the dependent variable based on a logistic regression model derived from (2).

We also run regressions using regional averages. We can linearize (2) around the variables of interest and derive:

\[(3) \quad P D \approx \alpha o + \sum_{j=1}^{J} \alpha_{1j} \cdot P_{j}^{E} + \alpha_{2} \cdot P_{j}^{D|E} + \alpha_{3} \cdot M_{k} + \alpha_{4} \cdot X\]

The average observed mortality rate for a region can be approximated by summing the individual probabilities of death for each child in (3) and dividing by the total number of children:\(^8\)

\[(4) \quad \bar{D} = \alpha o + \sum_{j=1}^{J} \alpha_{1j} \cdot \bar{P}_{j}^{E} + \alpha_{2} \cdot \bar{P}_{j}^{D|E} + \alpha_{3} \cdot \bar{M}_{k} + \alpha_{4} \cdot \bar{X} + \varepsilon\]

where D is a dummy variable set to one if child k died, ε is a regression error, bars above variables denote regional averages, and we suppress regional subscripts.

In our regressions we model the risk of exposure to disease as a function of the average level of morbidity in the surrounding region. We model the probability of dying, once having been exposed to disease, as a function of household characteristics and the propensity to treat children, as measured by an index of child vaccinations and perinatal care.

When implementing these regression equations we allow for clustering of error terms within households and regions.

---

\(^8\) This equation ignores second order terms which, in the case of child morality risk, will be small.
3.2 The determinants of the propensity to treat

Treatment is ultimately a choice variable, and it is helpful to characterize the factors that determine treatment. In order to derive a regression equation for treatment, in the appendix we present a simple model where guardians maximize a utility function that expresses a trade-off between consumption and child health services, subject to a budget constraint and standard concavity assumptions needed to ensure a solution. This allows us to derive a reduced form equation which relates treatment to household characteristics, and other demand and supply variables which impact treatment outcomes:

\[ T_i = \gamma_0 + \gamma_1 \cdot X_i + \gamma_2 \cdot P^T + \lambda_i \]

where \( P^T \) is the price of treatment services, and \( X_i \) is a vector of household characteristics relevant to treatment. This latter vector will include variables such as wealth that influence the ability to buy services, along with variables such as education and health knowledge that may influence a household’s perceptions regarding the relation between treatment and the probability of a child death.

In this derivation, we assume that treatment is available at a constant price for each household, and demand determines the level of services in equilibrium for each household at that price.

However, the price of treatment, and the coefficients on key household characteristics, could be different in a public system. For example, in a theoretical command economy treatment may be allocated equally to everyone, so the supply side will determine the allocation of treatment. In a planned economy the right hand side variables may only be relevant if they correlate with the actual allocation mechanism of the planners. We can accordingly use this equation to test whether health services are allocated differently in public versus private systems.

4. Datasets and variable definitions

We use DHS survey data from all 45 available countries to address these questions. Our population is all children born within five years of the interview date. Since the DHS surveys ask questions of mothers, we often
use household data where we define households as each mother with all her children and spouse.

The DHS surveys are sponsored by USAID and are conducted in a similar manner in all countries. They use common methodologies, survey questions, and manuals for field workers. The size of the survey in each country is chosen according to the population size and the desire to measure outcomes in specific regions. In all countries the population includes all women aged 15 to 49. In India there is additional information on village data. The surveys collect a range of information on households and children based on these interviews.

During the design phase, regions are divided into primary sampling units, or clusters, and a random subset of these clusters is chosen to take part in the survey. The clusters are generally small neighborhoods and a substantial fraction of the households in each cluster will be interviewed. For example in India the average cluster size is 150 to 200 households, and averages of 30 households are interviewed in each selected cluster.

We implement our regressions using individual and cluster average data. Due to the complications that come with missing data, and varying population at national levels, we have chosen not to adjust our data for sample weights when running regressions. We are effectively assuming that child mortality is determined by a common empirical model in all clusters and regions, so that we can extrapolate from this population sample to national or global data. In every regression we limit included clusters to those where at least four observations are available.

In order to implement our regressions, we need to define key variables, and create indicators for these variables where they are not directly available. For many of the variables of interest, the DHS dataset includes several related indicators. This is valuable since each indicator provides additional information, however the multiplicity of indicators complicates the analysis when we would like to measure the specific impact of variables.

We decided to deal with this through a strategy outlined by Filmer and Pritchett (2001) when creating wealth variables from DHS data. These authors used principal components to build single indicators for wealth

---

9 See also Rutstein, S. O., K. Johnson, et al. (2004).
based on all the variables in DHS data that measure aspects of wealth. They found principal components created robust indicators for wealth that could be used for empirical analysis. DHS now provides these indicators in their datasets for a number of countries. We chose to follow this same methodology when calculating indicators for wealth, health knowledge and treatment.

The key variables we use were calculated as follows:

**Child Deaths and Child Deaths 7+:**
Our logistic version of regression equation (2) calls for a dependent variable that is set to one if a child died and zero otherwise. For each child that was born within five years of the survey, we created a dummy variable, *Child Deaths*, to indicate the child’s survival outcome during the subsequent period until the time of the interview.

In order to separate deaths due to perinatal causes, from those after birth, we also created a variable “*Child Deaths 7+” which is a dummy that records the survival outcome for all children that were alive seven days after birth. We chose seven days because this excludes almost all neonatal deaths caused by risk factors during birth. Both diarrhea- and pneumonia-related deaths start to occur after seven days. Infections that occur after birth also cause deaths in the first month.

**Treatment:**
We want our variable “treatment” to capture the propensity for children to receive treatment from modern health services when they are sick. This is calculated as a single indicator for each household, and we assume all children in the household receive similar treatment.

To calculate this we decided to take all measures of preventive healthcare used by the family which are unlikely to have a significant impact on our indicator of child mortality, *Child Deaths 7+,* and are uncorrelated with whether a child is actually diseased.

---

10 As discussed below, due to the limited number of deaths in our dataset, the power of our single country regressions is generally low. By including deaths that occur between 7 and 28 days, i.e. within the neonatal period, we increase the overall power. The point estimates for coefficients are similar regardless of the timeframe chosen.

11 Girls and boys may be treated differently; however for the purposes of this paper we have not examined this question.
The measures we used are listed in Table 1. We included standard WHO childhood vaccinations recommended within the first three months after childbirth because these are good indicators of how well children are integrated into receiving healthcare, but at the same time they have a very small impact on mortality rates. In order to create one indicator for each household, we used data for the eldest living child that was under five years of age.\textsuperscript{12,13}

We also included the only two available indicators for mother’s treatment-seeking during pregnancy: antenatal care and whether mothers delivered babies at their home or institutions. These indicators will correlate directly with neonatal mortality rates but they should have no direct impact on deaths unrelated to the birth process. Since this component of the treatment indicator should directly impact deaths, we run our regressions with \textit{Child Deaths\textsuperscript{7+}}. Since deaths after seven days of age, i.e. \textit{Child Deaths\textsuperscript{7+}}, are primarily due to diarrhea and pneumonia, such deaths would not be directly related to vaccinations and antenatal care.

We calculate our \textit{Treatment} indicator from the first principal component of the above healthcare indicators. Table 1 shows simple characteristics of our treatment indicator and its relation to the underlying components from global data. A higher treatment indicator is correlated with more antenatal care and more child vaccinations, and it also correlates with health services that were not used to calculate the indicator. We believe \textit{Treatment} provides a good proxy for the relative propensity of children to be treated by modern healthcare.

\textbf{Morbidity Indicators:}
An important goal of our study is to determine whether the large variation in national and household probabilities of child death is driven by the

\textsuperscript{12} The \textit{Bellagio Child Survival Study Group} estimated that only 0.4\% of child deaths are due to diseases which could be prevented with the standard WHO vaccinations. We have excluded measles vaccination from our indicator since it is only due nine months after a child is born, and so it would lead us to exclude families where the only available vaccine records were for children aged between 4 and 9 months. We chose children under five years of age to limit recall bias, and we picked the eldest child in order to limit any endogeneity that could arise if younger children, who are born after a recent child death, are treated differently.

\textsuperscript{13} Aaby, P. and H. Jensen (2005) argue that the non-specific impact of measles vaccines may lead to greater survival than could be explained by reduced measles fatalities alone. This finding remains controversial, as discussed by Fine, P. E. (2004), and has not been confirmed in well-designed prospective trials, but would suggest that measles vaccination may directly explain survival despite the low number of measles-attributed deaths. As discussed above, our treatment indicator does not include measles vaccines, and there is no evidence of sizable survival gains from non-specific effects of the vaccines that we do include.
exposures to disease in a child’s local environment, or by the propensity of households to treat their child in a modern health system and generally manage disease episodes. To achieve this we need measures of disease incidence.

The three main causes of child deaths are pneumonia, diarrhea and malaria. The WHO has defined conditions under which children should be treated for suspected cases of each of these diseases respectively, and a rubric is listed in Table 2.

The DHS surveys ask mothers whether their children had watery or bloody diarrhea in the previous two weeks, whether their children had coughs with rapid breathing (symptoms of acute respiratory infection and suspected pneumonia), and whether they had a fever (symptom of malaria). These questions roughly match the WHO rubric for children that require further treatment and the definitions used in intervention studies measuring the efficacy of interventions that target morbidity.\footnote{See for example Luby, S. P., M. Agboatwalla, et al. (2004) and Luby(2005) for diarrhea and acute respiratory infections. Intervention studies often define diarrhea as 3 watery stools during a 24 hour period, whereas the question in the DHS survey we use asks mothers if their child had any watery stools. This will make our disease variable more sensitive than the standard definition but less specific.}

For each child we created a dummy variable reflecting whether any child under five years of age in the household, at the time of the survey, had symptoms of disease. We then calculated the average prevalence, for all households, of each disease in each sample cluster, and used this indicator as the morbidity variable for every child in the cluster when we run regressions at the individual level.\footnote{We use the household as the indicator of disease, rather than specific children, since we assume that every child in the household is exposed to disease if one child has symptoms. This is not important for any regression results, but given the highly infectious nature of disease, it seems the most realistic assumption.}

Our disease proxy assumes that children living in clusters with greater average disease prevalence, at the time of the survey, will be exposed to diseases more often than children living in clusters where disease incidence is less. We run regressions explaining household mortality rates using this morbidity index. We also run regressions where we compare cluster level mortality to cluster level right hand side variables to check the consistency of our results.
One potential problem with this data is that the surveys take place over more than one season in some of the larger countries, and if there is seasonality in the morbidity data, this could introduce noise into the morbidity variables. In order to control for seasonal patterns of morbidity, whenever we run regressions with disease incidence we also include country specific dummies that are set to one in the month or season that the interview was completed.16

Figure 4 plots the prevalence of diarrhea, and the prevalence of symptoms of acute respiratory infections (ARI), across countries. The figure shows there is a wide variation in the average impact. We don’t have comparable data for wealthy countries; however a recent study that examined diarrhea prevalence in Australia, Canada, Ireland and the United States found respectively that the prevalence of diarrhea, defined as 3 watery stools during 24 hours, in children under 5 years of age during the previous 4 weeks was 8.2%, 11.7%, 7.6% and 11.2% respectively (Scallan, Majowicz et al. 2005). This is a more restrictive definition of diarrhea, so the figures are not comparable, although it suggests that diarrhea prevalence is substantially lower in these wealthy countries compared to low-income countries.

**Wealth:**
DHS provides a single wealth indicator for many countries based on a principal components analysis of multiple indicators of wealth. We use this variable, however we exclude sanitation and water indicators when/if these are included in the DHS indicators. When DHS does not calculate a wealth variable, we have calculated a wealth index based on their methodology including, as best possible, the same variables they generally include when calculating their wealth index.

**Education:**
It is well-understood that parents’ education plays an important independent role in predicting child mortality rates. We used the years of schooling for the mother and father separately as measures for education.

**Improved Water:**
We used the standard WHO definition of improved water. Our dummy variable assigns each individual a 1 if they use piped or covered well water,

---

16 We checked for bias due to this in our regressions by restricting our regressions to households where interviews were done in similar seasons.
and otherwise a zero. The households with zero will typically have access to surface water, uncovered wells, or other sources that are more likely to be polluted.

**Improved Sanitation:**
We used the standard WHO definition of improved sanitation. Our dummy variable is set to one if the individual use latrines, and zero otherwise.

**Time to get to the water source:**
Respondents are asked how many minutes it takes to get to their drinking source. We use this variable as an instrument for improved water in our robustness section.

**Distance to nearest public health facility:**
We include this variable to capture public health supply. Where available this measures how far it is from the household to the nearest public health center. Where this is not available, the variable captures whether mothers report: “when they are sick they do not seek healthcare for themselves because health services are ‘very far away’”. The latter question is less satisfactory since health centers may be “very far” because a mother believes they are not very helpful. To the extent this is the case the coefficient may overestimate the impact of the variable, however as discussed below we find little impact of distance on outcomes.

**Months at Risk:**
Our regression equation calls for the months at risk to be included as an explanatory variable. We need to create this variable without biasing the results if a child dies. We do this by calculating the total number of months that the child would be exposed to disease risk if the child lived to the date of the survey period. This ensures that we calculate the total ex-ante “risk” facing the child, consistent with equation (2).

**Health Knowledge:**
In order to examine the relation between health knowledge and treatment, we chose to include variables that capture whether a mother has good health knowledge related to child health in some regressions. In almost all DHS surveys they ask mothers whether they have heard of oral rehydration therapy (ORT), and whether they know of any modern methods of family-planning. We created a variable using principal components from these two indicators.
**Other Controls:**
We include national and regional dummy variables. The DHS data generally does not provide village indicators but does provide state/provinces or larger general regional indicators. We also include a dummy set to one if the household lives in an urban area. Additional control variables are described in our robustness discussion in Section 6.

Table 3 presents sample averages and standard deviations of key variables used in our regressions by region and for the 20 largest countries by population.

**4.2 Dealing with missing data and selection bias**

Our empirical model calls for a regression of survival outcomes on child specific and household specific data. An important problem here is that household data on treatment is only available when there is a living child. This missing data could bias our coefficient estimates since the explanatory variables will be missing in households where all children die, thus generating a correlation between the error term and data availability in our mortality regressions.

In order to deal with this problem we compare regression results when we do not correct for the possible bias with two alternative procedures that aim to eliminate or correct for any bias.

The first is to fill in the missing data using a “hot-deck” technique common to survey data. Under this technique we find a donor household, with an identical number of child deaths, to fill in household treatment and morbidity data in cases where all children in the household have died by the time of the interview.

We find a donor’s treatment data according to the following hierarchy of rankings: region, cluster, antenatal visits, place of delivery, mother’s education, father’s education. Hence, donors will tend to be taken from mothers living in the same cluster, and having similar antenatal care, place of delivery and education. In order to match morbidity data, we follow the same procedure, although we use improved water and sanitation rather than antenatal care and place of delivery when finding matches. Improved water
and sanitation are correlated with diarrhea incidence, and may reflect a desire to reduce morbidity levels in households and regions.

This “hot-deck” routine has the advantage of maximizing the dataset, while maintaining the statistical distribution, and hence increasing the power of our regressions, however, by selecting donors we may be introducing bias in our dataset. An alternative procedure to achieve unbiased estimates is to follow a variant of Heckman’s estimator as outlined in Wooldridge (2002). Under this procedure we estimate the inverse Mills ratio using our selection equation for treatment, and then run an IV regression in available data to estimate our regression equation. We run this regression in our global data to test consistency of our results with the hot-deck procedure and the uncorrected regressions.

5. What factors best predict risk of child deaths?

Our empirical strategy is as follows: In section 5.1 we examine and compare within country results for all countries where data is available. We use the single country results to examine common trends across countries. We next consider improved water and improved sanitation as alternative disease indicators in section 5.2. We then examine results when we pool data for global regressions in section 5.3. We show that the pooled global regressions deliver similar results to the single country regressions, while the larger sample size substantially increases the precision of the estimates. The final section, 5.4, shows regression results measuring the determinants of treatment. After presenting the main results, in section 6 we examine robustness and the implications for causality.

In the following sections we present data showing regression results from up to 45 countries. If the same regression model is applicable to every country, then we should expect the coefficient estimates to have a Gaussian distribution with common mean and variance. However, in the single country regressions we calculate the treatment indicator separately for each nation, so we should not expect the same regression model to hold. We primarily use these single country regressions to examine whether there are common trends in the data.
**Power calculations for our regressions**

When interpreting results, it is important to keep in mind the power of our regressions since this will influence whether we should expect to find significant outcomes.

The actual power of our regressions is closely related to the mortality rate, the empirical importance of right hand side variables, and the sample size. Vaeth and Skovlund (2004) have derived a simple rule for calculating the sample size needed to power logistic regressions adequately. According to this rule, and using global sample averages from our data, we would need roughly 6,200 observations in our regression to capture the impact of a variable that generates a 15% change in Child Deaths 7+ in response to a one standard deviation change in that variable. This calculation assumes 80% power at a 5% significance level in a two-sided test. If we aim to capture the relevance of variables that have a smaller impact on Child Deaths 7+, we would need substantially more observations.\(^{17}\)

In our matched dataset we have an average 7,900 observations per country, so in many countries the power is adequate to demonstrate the significance of variables that have a large impact on mortality. However there are eleven countries with less than 3,000 observations. In these countries the power for a similar experiment will be roughly 0.50, meaning that even when the right hand side variable has a large impact on mortality, we only have a 50% chance of finding it is significant. This is important since it helps explain why outcomes are often insignificant in single country regressions.

In our global regressions we have 278,000 observations using our matched data, and 27,000 observations using our clustered data, meaning that these are adequately powered to estimate the impact of variables that have a small impact on mortality outcomes.

---

\(^{17}\) The required sample size can be approximated by the sample size needed in a randomized controlled trial. The trial is powered to measure a change in the dependent variable equal to two times the standard deviation of the right hand side variable multiplied by the regression coefficient on the right hand side variable found in the logit regression. This sample size can be adjusted for multiple regressors using a simple formula. In our case, the average value of Child Deaths 7+ is 0.06. The sample size required to measure an intervention that generates a 30% decline in Child Deaths 7+ with 80% power and 5% significance in a two-sided test is 4,624 per arm. When we make adjustments for multiple regressors, we calculate 6,165 observations are needed. We should therefore be cautious when observing insignificant outcomes in these regressions, since they could still imply that the variables do have modest or small impact on mortality, and the insignificance reflects the fact that our regressions are not powered sufficiently to measure the outcomes.
5.1 Regressions using direct measures of morbidity

Table 5 shows full regression results based on a logit transformation of equation (2), using our matched dataset, for the four most populated countries. The control variables are listed in the columns along with the variables of interest.

The first column, for each country, shows results when we include morbidity indicators as independent variables. These results are indicative of the findings in all 45 countries.

The results show that treatment is significant at the 5% level in six out of eight regressions, with the anticipated negative sign. The morbidity indicators are significant only in India, and they have a negative sign in three of ten cases. The p-value for the joint test of the null-hypothesis that all three morbidity indicators do not enter the regression ranges from 0.048-0.510. The remaining variables generally have the anticipated signs, although, apart from months of exposure, they are rarely significant.

Figures 5a-g show the predicted impact of a one standard deviation rise in treatment, education, and wealth, and the impact of a one standard deviation fall in morbidity, on mortality outcomes. Here we calculate the percentage change in mortality by linearizing each regression around the sample average for right hand side variables. We report results for all countries with a sample size of at least 3,000, i.e. those countries where the power of the regression to capture variables that have a large impact on mortality is at least 50%. The figures show there are large confidence intervals (95% bands) for most coefficients, which is consistent with the low power in many regressions; however they generally tend to be centered on a mean value with a few “outliers” as could be expected if the estimates were drawn from a random sample with a Gaussian distribution.

Despite large confidence intervals, it is apparent that the combined education variables and Treatment have the largest predicted impact, followed by wealth. The morbidity variables have negligible impact. As illustrated on Figure 5a, as we move up one standard deviation in the Treatment rank, the average probability that a child dies falls by 14.2%. When mothers’ and fathers’ education rises by one standard deviation, child mortality falls by 11.6% and 10.2% respectively. However, if the cluster where a child lives
moves down by one standard deviation in terms of the prevalence of diarrhea morbidity, the probability of a child death falls by only 3.7%. This is a common result in our regressions and implies that treatment-seeking and guardian’s education are more important than the general morbidity environment when determining health outcomes.

5.2 Improved water and sanitation as proxies for disease incidence

The above results provide evidence that the propensity to treat children, and household education, when compared to morbidity, are most important when explaining the differences in the probability of post-neonatal child deaths within countries.

However, one potential problem with these regressions is that our indicators of morbidity incidence, which are measured in our data only during the two weeks prior to an interview, may not be a very good proxy for disease incidence during the previous five years when mortality is measured. We therefore sought out alternative measures both to correlate with disease incidence and to tell us more about the importance of specific healthcare interventions.

The most common actions taken to reduce disease incidence are improved water, improved sanitation and better hygiene. A recent meta-analysis concluded that hygiene, clean water and improved sanitation can each reduce diarrhea incidence by 25-37% (Fewtrell, Kaufmann et al. 2005). A randomized controlled trial that measured the impact of hand washing in the slums of Karachi found acute respiratory infections were reduced by 50%, and diarrhea by 53%, when the intervention group received education and soap for improved hygiene (Luby, Agboatwalla et al. 2005). To the extent that improved water leads to better hygiene, it is possible that improved water will also reduce respiratory infections. Finally, since sanitation is important for insect control, these measures may also reduce the incidence of malaria (Keiser, Singer et al. 2005).

In Table 4, regression II, we substitute improved water and sanitation for morbidity variables to see whether these better predict mortality, and whether this changes the strong impact of treatment in our regressions. Figures 6a-b show the new predictions for the impact of improved water and improved sanitation in countries with greater than 3,000 population based on these estimates.
The results are very similar to those found when we used morbidity indicators directly. The regressions suggest water and sanitation have no significant predictive power for child deaths. Improved water and sanitation are each a significant predictor of mortality risk in only 16.7% and 10.2% of regressions respectively. The empirical magnitude of coefficients is generally small. The impact of treatment and education, not shown in the charts, was nearly identical to results in Figure 5. This again suggests that, within countries, the most important determinant of the probability of a child death is the general education, and the propensity for guardians to seek out modern healthcare for their children.

5.3 Cross-country differences in mortality rates

These empirical results tell us about the within-country pattern of mortality risk, but they provide no direct evidence regarding the explanation for cross country differences in mortality. As discussed in section 5.1, one problem with the single country estimates is that they are not sufficiently powered to measure the impact of variables that have modest or small impacts on overall mortality rates. This problem is illustrated in Figures 5-6 where the 95% confidence intervals around the point estimates are very wide.

Despite the low power, the results do suggest common trends. In general the coefficient estimates are consistent with a Gaussian distribution. There are a large number of coefficients near the mean estimates, and more narrow tails at either end. Given this general consistency of results, we chose to pool the data for all 45 countries and use regression equations to examine the predictive power for mortality risk within and across countries. We refer to this data as the “global” data.

Our wealth variable is defined specifically for each country, and it was not possible to create a single consistent estimator applicable to all households. So for wealth we continued to use our country specific variables derived from principal components and we permitted separate coefficients for each country in the regressions. We also allowed for a country dummy in each regression. We permitted clustering of error terms at the country and household level.

The results from the global regressions are presented in Tables 5 and 6. Table 5 presents results with morbidity indicators as regressors, while Table
6 presents results with improved water and sanitation as regressors. The five columns reflect different techniques used to deal with possible missing variable bias as discussed in section 3.

Column I shows regression results using a logit regression with the original data where we do not correct for sample selection bias. In Column II we correct for the bias by finding matching records according to our “hot-deck” procedure. In Column III we take cluster averages of variables and run OLS regressions according to (4). Column IV shows results when we run OLS regressions with the original data, and Column V shows results when we correct the regression for sample selection bias using the modified Heckman procedure.

The results in Column V show that the inverse Mills ratio is insignificant in both regressions, which suggests any selection bias is small.

Figure 7 graphs the implied impact of a one standard deviation change in treatment, morbidity and education on the probability of a child dying. We find similar results for treatment and morbidity in all regressions, with treatment being roughly twice as important as morbidity. There is greater variation in the estimates for mother’s and father’s education; however this is probably in part due to the high collinearity between the two variables leading to lower power. The joint role of education is highly significant in each regression.

Figure 8 graphs the implied empirical impact of each variable in the global data when we replace morbidity indicators with improved water and sanitation. These results are based on Table 6 and illustrate that improved water and sanitation have little predictive power for morbidity, while the impact of education and treatment remains large.

The fact that the cluster regressions have similar results to individual level regressions in Figures 7 and 8 is important. This shows that our results are not biased due to household level measurement error or other contamination, and that our decision to include cluster averages of morbidity as regressors in the individual regressions is not the reason for the low predicted impact of morbidity on mortality outcomes.
5.4 What determines treatment?

The previous regressions suggest treatment services and education explain large variations in child mortality within and across countries. This begs the further questions: why does treatment utilization vary? and are the observed differences in service utilization primarily due to supply or demand factors?

In this subsection we examine regression results that attempt to explain our treatment variable. Our regression results here are based on the equation (5). Our key demand side variables are education and wealth. Our public supply indicator is the distance to the nearest public health facility. This variable is only available in 23 countries. Due to the paucity of data and indicators, we also turn to a different analysis of the role of demand and supply by looking at where households seek out treatment in Section 7.

We ran regressions both in the global data, and as single country regressions (not reported). The results were similar in both. The single country regressions implied that mother’s and father’s education, followed by wealth, were empirically the most important explanatory variables, while the dummy variable representing distance to public health centers was slightly less important.\(^\text{18}\)

Table 7 presents results from our global regression. Column I shows an OLS regression in all available countries when we exclude “distance to nearest public health center”. Column II shows the regression results when we include this variable in the reduced sample of 23 available countries. The results are similar in both regressions.

These global regressions also suggest education is the single most important explanatory variable. We calculate that a one standard deviation rise in mother’s and father’s education would generate an 0.29 rise in treatment (measured in units of one standard deviation), while a one standard deviation fall in distance to public health center generates an 0.06 rise in treatment. Since we have included individual wealth components for each country in

\(^{18}\) When measured in terms of standard deviations of treatment, the average single country impact of a one standard deviation rise in wealth, mother’s education and father’s education was an: 0.10, 0.11, and 0.08 rise in treatment. A one standard deviation fall in distance to the nearest health facility generated an 0.06 rise in treatment. These variables were significant at the 5% level in 78%, 78%, 70% and 65% of the single country regressions.
the regression, and permitted coefficients to vary by country, we do not measure the impact of wealth in this regression.

In the regressions listed in Table 7 we did not include health knowledge as a regressor because there is good reason to believe it is endogenous, i.e. health knowledge may be higher if you frequently seek out treatment, or it may lead you to better seek out treatment. In Figure 9 we show the coefficient estimates on health knowledge when we add this variable into each single country regression. The figure shows there is a large and robust, positive relation between health knowledge and treatment across most countries.

We present this result because it could have important policy implications. If this is a causal relationship, then improving health knowledge could substantially raise treatment, and this in turn could reduce mortality rates. Given that general education and wealth levels take much longer to change, and building out public infrastructure is time consuming, costly and difficult, then focusing on improving health knowledge could be the most effective means to substantially change treatment-seeking and reduce child mortality.\footnote{We also ran treatment regressions in global data (not reported here) and found similar results to the single country regressions.}

In Section 7 we return to the issue of what determines treatment by looking at where households gain their treatments – in the private or public sector – in order to better understand routes to increase treatment provision. Before returning to this issue, in the next section we examine robustness and causality.

6. Robustness and causality

The regression results from Section 5 demonstrate, at the least, that the propensity to treat children, and parent’s education, are very good predictors of child mortality, while morbidity measures and access to water and sanitation are poor predictors. We’ve shown the results are robust at the cluster and household level, and selection bias does not have a strong impact.

In this section we focus on whether it is reasonable to believe these are causal relations, i.e. should we conclude that if we raised education and the
propensity to treat, child mortality would fall by the amounts predicted by our regression equations?

Our approach is to consider: (i) if we have adequately controlled for potential confounding factors; (ii) if endogeneity of right hand side variables could be biasing results; and (iii) if the large impact is plausible given results from intervention trials and other sources.

6.1 Disaggregating our principal components to expand the list of controls

One problem with survey data is that we can never be sure we have sufficient and adequate controls to prevent biased coefficient estimates. We’ve attempted to deal with this by adding multiple control variables, along with regional and urban dummies, and seasonal dummies to our regression equations.

6.1.1 Controls and interaction terms for malaria endemic regions

Our global regressions assume that the coefficients on morbidity variables are constant across countries. If the morbidity indicators reflect different underlying diseases and mortality risks in different regions, then our results may be biased. This could particularly be a problem for fever, which is a symptom for malaria in endemic regions, but would not indicate malaria in regions where there is no malaria.

In order to test whether this is biasing our results, we created a dummy variable for each country that was set to one or zero depending on whether the country suffered from endemic malaria according to data published by the WHO and Roll Back Malaria. We included this term, interacted with fever, in our matched-data global regressions to test whether there is a differential implication for fever in the malaria endemic zones.

The impact of this change on our main regression results is illustrated in Figure 10. The p-value for the interaction term was 0.056, while fever on its own was insignificant, so the coefficient estimates do suggest that fever is only important in malaria endemic nations. However, the implied impact of a one standard deviation change in morbidity indicators is unaffected by these changes, as shown in Figure 10, and the implied impact of fever remained small.
6.1.2 Controls for possible ethnic elites

Our next test is to examine whether there is a third factor which is driving treatment, education and low mortality in our regressions. In particular, if some regions or households receive preferential access to public services, say because they tend to be elite ethnic groups, then our regression results may actually be picking up correlations with this preferential status rather than causality of specific right hand side variables.

The DHS data includes measures of ethnic groups and castes for 26 countries which we can add to regressions to determine whether they change our main results. Due to the large number of variables (for example in Zambia there 57 groups), we restricted the analysis to single country regressions. For each country where data is available, we have created a dummy variable equal to one for each ethnic group, and then run our single country regressions as described in Table 4, column I, with these additional variables.

The regression results suggest class may be an important indicator of mortality risk, but it does not bias our estimates. We found that the ethnicity variables were jointly significant in 15 out of 24 regressions. The calculated impact of a one standard deviation rise in treatment on mortality was –13.6% in the original regressions for these countries, and changed to -13.7% when we added class variables. The impact of a one standard deviation rise in mother’s education changed from -10.4% to -9.9%, and the impact of father’s education moved from -11.3% to -10.8%. A one standard deviation fall in morbidity predicted a 2.4% fall in mortality compared to 3.6% in the original regressions. None of the new estimates were outside the confidence intervals of the original estimates.

6.1.3 Low birth weight and age of mother

There is a substantial literature which demonstrates that low birth weight babies, and children of young mothers, are at greater risk of death than older children. The causality of this relation is not clear, however it could reflect poor nutrition and issues related to women’s rights.

We included a dummy variable set to one if the mother was under 18 when the child was born, and a dummy variable set to one if the child was reported
to be “small or very small” at birth, in our logit matched regressions. The impact of adding these controls on our main regression findings are illustrated in Figure 10. Each of these variables was significant in the regression, with the anticipated sign, however they did not substantially affect the significance or empirical importance of treatment, education and morbidity indicators.

6.1.4 Health knowledge versus education

One issue which is highly important but difficult to measure in this data is the relative importance of general education as compared to health knowledge. It could be that health knowledge is the main factor needed to lower mortality, or it may be that general reasoning skills, literacy, and the empowerment that comes with education are essential.

Unfortunately there are few common indicators of health knowledge across countries. As described in Section 3, we have compiled country level health knowledge indicators based on family planning knowledge and knowledge of ORT. These are strongly correlated with treatment as shown in Figure 9, but it is difficult to sort out the direction of causality. We included these as controls in our global data, allowing coefficients to vary for each country on the health knowledge term, and as Figure 10 illustrates this did not affect our main findings.

However, we don’t believe these two variables properly capture health knowledge that matters for child survival. We’d prefer to have a comprehensive list of variables that covers issues such as beliefs regarding clean delivery practices during birth, and knowledge of risk signs and treatments for diarrhea, pneumonia and malaria. The only common variable we have related to child health for all countries is whether mothers had heard of ORT. This does not tell us whether mothers know how to use it properly, nor whether they actually implement it.

The Indian survey (unfortunately this variable is not available for all mothers in other countries) has one interesting variable that is closer to what we would like to measure. They ask every mother whether one “should increase, maintain, or decrease fluids” given to a child when the child is sick with diarrhea. The correct answer is to increase fluids since death from diarrhea is almost always a result of rapid dehydration. However, increasing fluids is counter-intuitive since children often vomit what little fluid they are
given, and it may seem appropriate to reduce fluids until the child can take fluids without vomiting.

To see whether this form of health knowledge was correlated with mortality, we created a dummy variable equal to one if the mother believed fluids should be decreased, an answer that is clearly wrong and would pose risks to the child, and zero otherwise. Approximately 30% of mothers in India believe fluids should be decreased. We then ran our mortality regressions using the matched data in India. The results from those regressions showed that this variable was highly significant, with a p-value of 0.005. The coefficient estimate implied that a child whose mother believed fluids should be decreased during diarrhea episodes had a 15.2% greater risk of death than one whose mother did not believe this. The inclusion of this variable had little impact on the other estimates.

This result in our Indian data suggests that, were we to properly measure health knowledge specifically, we may find it plays a very important role in explaining child deaths. Unfortunately we cannot measure this adequately in all countries with our data, but we will return to this issue in Section 7.

6.2 Feedback and endogeneity

Even if we assume that our regressions have adequate controls and there is little evidence of measurement error generating bias, our coefficients could be biased if there are feedback effects from right hand side variables to the dependent variable. In this case the error term in our regression equations would be correlated with dependent variables, hence biasing results.

6.2.1 Could endogeneity of improved water and sanitation explain the low impact of these on mortality?

Our regression results suggest clean water and sanitation have a minimal impact on child mortality. It is worth exploring potential reasons for this somewhat surprising finding.

As discussed above, a recent meta-analysis of improved water, sanitation and hygiene interventions found that these interventions can improve diarrhea incidence by 25-37% (Fewtrell, Kaufmann et al. 2005). Further, they found that the benefits of combined interventions, such as hygiene with
clean water and sanitation together, were no greater than the benefits from a single measure such as clean water.

If we assume that diarrhea related diseases cause 31% of total child deaths, and we assume a linear relation between deaths and morbidity, then such measures could reduce diarrhea mortality by 8-12%.

There is also potential for clean water to reduce acute respiratory infections if it contributes to better hygiene. A recent trial of hand-washing in Karachi found hand-washing contributed to a 50% decline in acute respiratory infections when compared to control regions (Luby, Agboatwalla et al. 2005).

Hence the empirical relevance of water and sanitation could be large, or negligible, depending on the sum of these parts and the relation between the different morbidities and mortality.

We ran OLS regressions using the global clustered data to examine the correlation between improved water or sanitation and our morbidity indicators. In these regressions we controlled for wealth, mother and father’s education, urban living, seasonal dummies and country dummies. The point estimates from the regressions implied that if both water and sanitation were improved, diarrhea would fall by 3% and fever by 4%. Each was significant at the 10% level. There was no significant impact on cough symptoms of acute respiratory infections.

So why do intervention studies suggest improved water and sanitation reduces morbidity quite substantially, while our results imply only a modest impact? Let’s start with a potential fault with our methodology. It may be that improved water and sanitation have been supplied to some of the worst off regions, and since we do not take this into account in our regressions, our coefficients are biased to show less impact than is actually the case.

This does not seem obvious. Indeed, a priori we would have thought the bias would go in the opposite direction. Political realities often mean that influential groups, regardless of relative need, actually receive improved water first. This would bias the impact of water on morbidity and mortality in favor of clean water programs rather than against.
6.2.1(a) Instrument for improved water from Indian village data

To examine this further, we consider a potential instrument from the Indian data that permits us to identify whether better quality water impacts health. The Indian DHS dataset includes a dummy variable for each village specifying whether the village has access to surface water for agricultural irrigation. Surface water is typically polluted since it has not gone through natural filtration similar to groundwater, but it makes for a ready supply of water and so reduces the demand for an improved water supply. To the extent that clean water is important for health, villages with readily available surface water should therefore actually be worse off if they are less likely to get an improved water supply.

We ran a first stage regression with improved water as the dependent variable, and “access to surface water for irrigation” plus our controls as explanatory variables. The coefficient on the irrigation term was negative, as expected, with a p-value of .005.

We then used this as an instrument for an IV regression across Indian villages. The results, presented in table 8 for mortality, were nearly identical to our OLS regressions. The improved water variable remained insignificant.

6.2.1(b) Instrument for improved water using time taken to fetch water

There is a second potential instrument available in most countries reported by DHS. They ask each mother how much time is spent each day getting water. Those households who are far from a water source are likely to use less water, and may, from time to time, substitute lower quality water instead of making the long trip to fetch water. Further, since they may leave water in containers for long periods between fetching new water, it may become more contaminated than someone who has a water source nearby.

We ran a first stage regression of improved water on time to fetch water, where the latter was measured by three dummy variables set to one if it took longer than 15, 30 or 60 minutes to fetch water. In our data 10.1% of families report it takes more than 60 minutes to fetch water.

These estimated coefficients for all three dummy variables were negative as expected, and they were jointly highly significant with a p-value of 0.000.
We then ran an IV regression in our global, matched data. The results from this regression for mortality in our global matched data are shown in Table 8. The coefficient on improved water is still insignificant and has the wrong sign. This regression also implies that improved water has little or no impact of mortality outcomes.

So what else could explain the weak impact of water and sanitation in our regressions? Our guess is several factors:

First, the intervention trials are typically done in the worst off regions, where water quality improvements are probably important, and yet these regions may not be representative of regions without improved water in our samples. The substantial increase in improved water and sanitation over the last 40 years may mean that the major gains from this intervention are now past. This is the explanation chosen by Sastry and Burgard (2005) when discussing why improved water no longer correlates with Brazilian prevalence of diarrhea.

A second, and related reason, is that the availability of medicines to treat common diseases may make improved water less important for mortality outcomes. The major improvements in improved water in Europe and the United States occurred when typhoid and cholera were still major causes of death, and when antibiotics and ORT were unavailable or unknown (Culter and Miller 2005). The availability of vaccinations and antibiotics, and knowledge of ORT, has dramatically reduced death rates.

A third reason is that water quality at the source does not ensure good water quality in the home, or lower morbidity, unless households store water in clean conditions and generally maintain good hygiene (Trevett, Carter et al. 2005). There could also be problems with the source if wells, pumps and pipes are not maintained properly. In our morbidity regressions, mother’s education was highly significant when predicting household diarrhea. This suggests hygiene behavior may be essential, rather than the source and plentifulness of the water, for preventing diarrhea.

Finally, intervention studies, and our regressions, focus on diarrhea as a symptom of lethal disease. However, diarrhea is in reality a syndrome caused by many different pathogens having varying mortality risk, and these pathogens can be transmitted in many ways. One of the most lethal pathogens is rotavirus, which is common to high and low-income countries,
and is believed to be transmitted through a fecal-oral route. Better hygiene alone, even when the water source is not improved, can eliminate the causes of these types of diarrhea incidence.

6.2.2 Endogeneity of wealth

A second bias would occur if child mortality directly influenced wealth. There are several potential channels here. Fatal child illness could reduce family wealth if households had to raise funds to finance healthcare, or, it could perversely raise wealth if families were able to save more due to fewer children.

While these are each plausible, we doubt they play a significant role here. We have examined child deaths which occurred in the previous five years to the study. Over half of these deaths occur in the first year of life. As discussed in Section 2, and further discussed in Section 7, the vast majority of diseases would be easy and cheap to treat, hence prohibitively expensive treatment is unlikely to be the main reason children died. With the maximum timeframe of five years, we doubt child deaths contribute much to higher savings.

6.3 Reality check: Is such a large decline in mortality due to treatment and education plausible?

As a final robustness test, let’s consider whether it is reasonable to believe that parental education and raising the “propensity to treat” could truly generate the empirical declines in mortality risk that we estimate here. According to our regression equations, holding fixed wealth, a one standard deviation rise in mother’s and father’s education (roughly 4 years each), along with a one standard deviation improvement in treatment, can generate a 40% decline in post-neonatal child mortality.

To see if this is plausible we focus on two questions: is the WHO recommended treatment regime for the common diseases that kill children feasible for a large enough fraction of very poor households that we can

---

20 Acemoglu, D. and S. Johnson (2005) estimate, using the discovery of vaccines and antibiotics as instruments, that the impact of increased life expectancy on income per capita is near to zero. Weil. D. (2005) estimates a larger impact of health on incomes, but his focus is on diseases that leave people sick during their working years, rather than factors that impact the first months of life.
make inroads into child mortality without changing wealth and incomes?, and would the WHO recommended treatment regime, and other simple interventions to improve child health, sharply reduce child mortality if implemented?

Table 1 outlines the treatment regimes for the three main diseases that cause child deaths. For diarrhea, the most important early treatment is to provide fluids, and WHO recommends providing oral rehydration therapy. The cost of this is minimal, but it requires effort and time by guardians to prepare and administer. WHO recommends oral antibiotics at home for suspected pneumonia, and if severe, hospitalization. The cost of a typical full regime of oral antibiotics is around $0.25, and hospitalization will cost roughly $6 per child in low-income countries. Finally, a regime of anti-malarials can be administered at home and costs $0.25, but in regions where artemisinin-based combination therapy is required costs rise to $1.25-$2.50 per treatment (Breman, 2004).

In China, Kerala, Vietnam, Indonesia and elsewhere, very poor households do seek out such healthcare when needed, and they apparently pay a substantial fraction of the total costs, and of their incomes, on such care (Varatharajan, D. et. al. 2004 and Adams, S. 2005). As a result, these nations have achieved very low child mortality rates despite poverty.

The question of affordability is closely linked to perceived value, so to the extent that many people do not perceive high value for healthcare, they will not be willing to pay for healthcare, and they may not adequately seek healthcare out, even if the price is low. A recent study in Nepal that examined the impact of health knowledge on neonatal mortality demonstrated that, even with free neonatal health services to all households in the trial, neonatal mortality was 28% lower in the intervention groups that benefited from improved health knowledge when compared to the controls who had access to free care only (Morrison, J. et. al. 2005).

Given these general facts, we believe our regression findings are plausible, i.e. we should not conclude that material poverty per se is a major obstacle to achieving a large reduction in child mortality. Poverty will be an obstacle for some households, and it does mean that child health in poor nations will be worse than in wealthy nations, but it is not a reason to believe that we cannot make substantial improvements. We return to the issue of financing healthcare for the extremely poor in section 7.
Next let’s consider what the probability of survival would be if a child is treated according to these regimes. There are no trials that permit us to measure exactly what happens when a child receives a “poor” treatment regime compared to one that receives a “better” treatment regime. However, we only need a modest absolute change in survival probabilities in order to make dramatic changes in mortality rates. For example, if children are sick with disease ten times per year, and they face a case fatality rate of 0.025%, then the child mortality rate will be 117. If you can reduce this case fatality rate to 0.005%, then the child mortality rate falls to 25. We need to reduce the risk of death from each episode by just 0.02%.

Some studies have pointed out the potential empirical importance of treatment-seeking and general health knowledge. A recent WHO-sponsored study that reviewed the evidence in favor of twelve family- and community-based interventions that promote child survival, provides a summary of available evidence. This study concludes that uncomplicated diarrhea, malaria and local infections can be cured at home with efficacious treatments. They argue that ORT can prevent death from watery diarrhea in all but the most severe cases. They state that improved home treatment of malaria has “the potential to have a large impact with one well-conducted study reporting a 41% mortality reduction”. Finally, they note that “poor care-seeking has been implicated in 6-70% of [child] deaths” (Hill, Kirkwood et al. 2004).

A broader analysis of the potential to reduce mortality comes from the Bellagio Child Survival Study Group. They calculated that the introduction of 24 basic health interventions could generate a 63% decline in child mortality. In Table 9 we show, where data is available, that our Treatment indicator is indeed correlated with parent’s adherence to several of these measures. As we discuss further in section 7, well-educated parents who seek out treatments when their children are sick, could easily implement most of the important measures listed in that survey.

7. Implications for child health programs targeting reduced child mortality

The results from our regressions suggest that education and treatment-seeking, which are closely linked to health knowledge, are the most critical factors explaining differences in mortality risk in poor regions.
We argued that this is likely to be a causal relation rather than a simple correlation. We found little evidence of feedback relations that distort the data, we controlled for potentially confounding factors including wealth and social/tribal status, we argued it is plausible that inexpensive treatments and improved health knowledge could substantially reduce child mortality based on intervention trials, we showed that health knowledge and treatment-seeking is highly variable and hence different enough across groups that it could generate large mortality differences, and our treatment indicator and education are both highly correlated with specific activities of mothers that reflect good treatment patterns.

In this section, we expand our discussion to calculate the impact of targeted programs that change education, treatment, morbidity, improved water and improved sanitation. We then examine the key differences and issues that arise when we compare the solutions put forward by the Bellagio Child Survival Study Group with our findings. We also discuss specifically whether a large expansion of public health infrastructure and investment is truly needed to reduce child deaths sharply.

7.1 What would be the impact of targeted programs to reduce morbidity, improve water and sanitation, raise education, or raise treatment levels?

In order to predict the impact of major intervention programs, we need to consider not only the empirical impact of the intervention when we increase or add the services, but also the pattern of existing services to see how much room there is for further increases. Figure 11 shows the impact of several alternative intervention programs on Child Deaths based on our logit, matched data regression results.

We calculate that expanding clean water and sanitation to 100% of the population would reduce child mortality by just 2%. The low impact of these variables reflects the generally high coverage of water and sanitation in many regions, along with the low predicted impact of improved water, based on our regression results and discussed in section 5.2.

The second experiment measures the impact of halving the incidence of diarrhea, fever and ARI throughout all countries in our sample. We’re not sure how this could be achieved, but even if it were achieved, we calculate
that the impact would be to reduce mortality by just 3%. This reflects the weak empirical relation between symptoms of common childhood diseases and mortality in our regression outcomes.

In our third experiment, we consider the impact of raising the propensity to treat children in all households to the 53rd percentile of our global treatment indicator. This is the level in Egypt today. This may be achievable through targeted health education projects, such as a Nepalese study referred to below. We forecast child mortality would fall by 13% if this was achieved.

Finally, the fourth experiment illustrates the impact of greater education. Here we examine the predicted outcomes if we raise the years of schooling for mothers and fathers to 6.3 and 8.3 respectively. This is once more the level currently in Egypt. We calculate that child mortality would fall by 19% if this were achieved.

**7.2 Do we need to build out the public health sector in order to reduce mortality?**

The empirical results in section 7.1 suggest that an aggressive program aimed at improving education (with health knowledge) and treatment-seeking could achieve very large declines in child mortality.

Table 10 presents a list of the recommendations by the Bellagio Child Survival Group. At first glance, the main interventions promoted by the *Bellagio Child Survival Study Group* appear completely unrelated to the results from our regressions. However, we believe the relation is closer than it may appear. The specific measures are generally simple and can be achieved by parents at modest cost to themselves, through treatment at home and access to very basic child health services. Our regression results probably reflect that well educated households with high treatment indicators do implement a similar package of measures as listed in Table 10 to keep their children alive.

However, there is a potentially much more important difference between our interpretation of the correlation between education and outcomes, and the list of evidence-based interventions. The following two factors could explain why so much remains to be accomplished, despite the relatively simple and well-known nature of the measures listed in Table 10.
First, to the extent that political institutions respond to the demands of vocal and powerful citizens, these institutions probably do not strongly reflect the interests of marginal groups. Education may be essential to ensure that marginalized groups have enough voice to receive public services (including better education itself).  

Second, it is probably easier for an educated person, and surely for a literate person, to learn about improved health practices, to perceive the value of such services, and to seek out adequate healthcare.

We believe these two factors probably play a critical role in explaining why education is so important in our regression results. This could also explain why, despite general consensus amongst professionals in all countries, and the simplicity of the measures needed to reduce child mortality as outlined in Table 10, the rates of child death remain very high in many poor countries.

This then raises the question: if we embark on a program to change education, health knowledge and treatment-seeking, so that parents follow or seek out the measures outlined in Table 10, do we really need to build out the supply of public infrastructure, or can we rely on market forces to ensure that private supply expands to meet demand?

The Commission on Macroeconomics and Health argued that a large public build out was needed because the private sector could not be relied on to provide equitable, affordable services sufficient to reduce mortality. However, they did not present empirical evidence to justify this argument. There is substantial variation in the size of the private sector in the 45 countries we examine, so it is possible for us to use this data to compare basic performance measures of public and private systems.

We start by examining whether public systems provide a more equal distribution of healthcare across groups. We do this using our treatment indicator and mortality outcomes. Figures 12 and 13 plot the average level of treatment, and a GINI coefficient we calculated for treatment, against the

---

21 Education will probably also change the distribution of services within the household, especially for girls.
22 The Bellagio Child Survival Study Group was less decisive, although their general assumption was that the public sector would be responsible for most services, while the private sector “should be involved whenever possible, especially in monitoring and ensuring quality and equity” (Bryce, J., S. el Arifeen, et al. 2003).
size of the private health sector in the country. The chart shows clearly that neither public nor private systems perform better in terms of levels, or equity of the allocation of treatment. Figure 14 shows the same conclusion can be drawn when examining mortality outcomes.

One criticism that could be directed at our argument is that Figure 14 represents the average outcomes for the whole population, but it does not show us where the poor are receiving their healthcare. It could be that low mortality is achieved only in countries where the public sector provides services to the very poor. Figure 15 addresses the question directly and shows that even when we limit the analysis to the bottom quartile of the population, in terms of wealth, we find a similar pattern. The very poor can have low mortality rates in both public and private regimes, and there is no correlation between the ownership status of the health sector and mortality outcomes.

In order to properly control for confounding factors, we also ran regressions where we included the relative size of the private sector in our treatment and mortality regressions. We also interacted the size of the private sector with treatment to learn whether there was a different predicted impact of treatment according to the public or private nature of the health system. These results are reported in Table 7 column III-V. They show that the size of the private sector is not significant in mortality or treatment regressions, implying again that the public and private sectors perform equally well.

Figure 14-15 allow us to draw one final conclusion. It is interesting to note that there are many successful countries with both small and large private systems. Vietnam has a large private sector despite being viewed as an example of a socialist success, and private spending accounts for 71% of total health spending (Adams 2005). The same is true of Kerala, a state in India where healthcare success has often been attributed to many years of “communist” governments, where the poor reportedly seek out 60-70% of health care services in the private sector (Varatharajan, Thankappan et al. 2004). Indonesia and Egypt are other countries with unusually low child mortality and a large private sector. Kazakhstan, Armenia and Peru are examples of countries with virtually exclusive public sectors and low mortality.

---

23 Adams, S. J. (2005) reports that the private sector pays 63% of healthcare spending in China. The high private percentage in part reflects user costs in the public health system.
### 7.3 Designing interventions to reduce child mortality

What does this imply for the design of intervention programs aimed at reducing child mortality?

Our first conclusion is that parental and child education, including efforts to rapidly improve health knowledge, should be a key component of any project, and, given the strong evidence that education matters, it would be a major gamble to assume that we can make large inroads without education.

There is a surprising paucity of well-designed research trials examining how health-related education could impact survival outcomes, and the best means to achieve this. A recent, large community-based intervention to reduce neonatal mortality in Nepal is an exception. This project examined whether the formation of women’s groups to improve health knowledge and practices ahead of child birth would affect neonatal mortality. They found neonatal mortality fell by 28% in the intervention areas compared to controls (Morrison, Tamang et al. 2005).

One additional example is a long-running project in rural Maharashtra called the Comprehensive Rural Health Project. In this project, a self-financing private clinic was created to service a village population of 150,000 people since the early 1970s. The clinic was established to build credibility with the population; however the major intervention was the selection and training of village health workers to educate households on the causes of disease and child deaths, and to improve treatment-seeking and general health-related behavior. While there were no prospective controls in the region to compare outcomes with, the health outcomes do appear very promising when compared to rural regions in Maharashtra and the country as a whole. Child mortality rates fell 68% during the first five years of the project, and today, despite general poverty, child mortality rates are near to levels in wealthy countries (Arole and Arole 1994).

Our second conclusion is that we don’t need to build out a large public health system in order to achieve major declines in child mortality. In fact, it seems safer, and probably more sustainable in most poor countries, to concentrate on improving education and health knowledge which can be passed on within communities and across generations. This empowers individuals to seek out the best care for themselves, and means they don’t
need to rely on political elites, public sector capacity, and national financial stability, in order to gain better mortality outcomes.

However, there are still important issues with ensuring proper supply. Communities may resist health education efforts when health services are not made available (Arole and Arole 1994). So, a health education project may need to be combined in a partnership with a service provider (private or public) for an initial period, and it may prove worthwhile to partially subsidize health costs initially. Loevinsohn and Harding (2005) discuss promising experiments with contracting out health services to the private sector.

Under either a public or private-based system, it is important to understand how best to target measures to ensure the extremely poor receive services, and in regions where limited services exist, there is probably a case for at least temporary subsidization to encourage rapid changes in health behavior and experience. Gwatkin, Bhuiya et al. (2004) discusses several experiments with different delivery systems that aim to target services to the poor.

8. Conclusions

Our main goal for this paper was to examine “what works” in terms of routes to reduce child mortality. We have shown that education, improved health knowledge and treatment-seeking are highly correlated with low child mortality, and conclude that it should therefore be possible dramatically to reduce child mortality through concerted efforts to improve these. This is consistent with intervention studies, which demonstrate that fairly simple measures can lead to dramatic health improvements.

The importance of education and health knowledge takes the conclusions from intervention studies one step further, since it helps explain stark regional differences in health standards despite general consensus on how to achieve better outcomes. We’ve argued that education is probably important both because the public sector is more responsive to educated groups, and because educated households are able to best take actions that can sharply reduce case fatality rates from disease.

We have also reexamined some traditional assumptions about the role of public sector contributions, arguing that given the importance we find of
parental education, it may be a mistake to focus primarily on large public health infrastructure when designing intervention programs to reduce child mortality. The most common justification for preferring public healthcare — that it best serves the poorest and leads to a more equitable distribution of services — does not hold up to scrutiny. This leads us to conclude that a focus on the oft-recommended “if-you-build-it,-they-will-come” strategy is probably misguided; and because, for such a strategy to succeed, we need dedicated political elites, firm public finances, and political stability, this is certainly a risky route to improvement in many countries.

While more evidence is needed in this discussion, our assessment of data underscores the relative importance of demand over supply factors in catalyzing positive change in health for the poor. We believe the evidence suggests that empowering individuals, by educating them and improving their healthcare knowledge, so that they can seek appropriate healthcare in every environment, is the most fundamental step needed to improve the health of communities which need improvement the most.
Appendix I:

A.1. Deriving regressions for treatment

In this appendix we outline the derivation for our treatment equations.

Suppose a household maximizes expected utility which is derived from own consumption, \( U(C) \) and lump-sum utility, \( v \), that they gain if their child survives. A child’s survival is affected by how much the household spends on treatment, \( T \), which has a price, \( p^T \), where all prices are normalized in terms of units of consumption. Assuming the household has total income \( W \), the maximization problem for the household is:

\[
\text{Max}_{C \in E} \left[ U(C) + D \cdot v \right]
\]
subject to:
\[
C + p^T \cdot T \leq W
\]
\[
\Pr(D = 0) = 1 - \Pi(T; N)
\]
\[
\Pr(D = 1) = \Pi(T; N)
\]

In order to derive simple closed-form solutions for our empirical work, we assume that the level of Treatment, \( T \), is committed to before a child is born, so we do not need to consider dynamics. Further, we assume utility and the probability of survival can be approximated by quadratic functions derived from Taylor expansions:

\[
U(C) = \theta_0 + \theta_1 C - \frac{\theta_2}{2} C^2 \tag{A1}
\]

\[
\Pi(T, N) = \tau_0 + \tau_1 T - \frac{\tau_2}{2} T^2 + \tau_3 N - \frac{\tau_4}{2} N^2 + \tau_5 N \cdot T \tag{A2}
\]

The closed form solution to this problem is easy to derive given the above assumptions and quadratic approximations. Assuming an interior solution, the optimal level of treatment, \( T^* \) is a function of the incidence of disease, the cost of healthcare, and wealth of the household. The solution shows that treatment rises with wealth and falls with the price of health services.

The impact of higher morbidity on treatment will generally be negative, since higher morbidity, at the margin, lowers total expenditures available for
consumption, so households will tend to reduce expenditures on treatment and reduce consumption also.

There is an important role here for each household’s perceptions of the benefit of treatments when deciding how much to spend on healthcare. Equation (A2) presents an explicit function that relates the probability of death to key parameters. Health knowledge and education may affect a household’s perceptions of these parameters. It is natural to assume that education would lead one to discard traditional beliefs, and learn non-intuitive treatments, so that health knowledge would raise the perceptions of the likelihood of survival if a child is treated. However, the bias may go the other way, if uneducated people overestimate the ability of modern healthcare to prevent deaths. In our empirical regressions we make no assumptions regarding how education and other variables will matter, but we do include these in our regression equations to explain treatment.

In order to define our regression equation, we linearize the solution to the above maximization problem, add a regression error $\lambda$, and include a vector of household control variables $X$ that include education, health knowledge and other household characteristics. Our treatment indicator only measures treatments for a child at the time of birth, which are one time events, so we do not introduce morbidity terms to explain the empirical indicator we use. The regression equation is:

$$
T_i = \gamma_0 + \gamma_1 \cdot X_i + \gamma_2 \cdot P_T + \lambda_i
$$

This equation assumes households are free to purchase as much treatment, $T$, as they chose at a price $P_T$. In many health systems in low-income countries this is a reasonable assumption since, at the margin, households buy services in the private sector. However, in countries where there is a large public system, then public supply, rather than household demand, may determine outcomes.

In a public-based system we should expect a weaker relation between treatment and household characteristics to the extent that these systems are better able to achieve universal coverage. Public systems may rely less on individual finance and initiative to seek out healthcare, and this improved universal coverage could lead to lower mortality rates. This is something that we can examine further in our empirical work.
Table 1

Proportion of mothers and children that received the individual services used to generate Treatment

<table>
<thead>
<tr>
<th>Ranking of household according to global Treatment indicator</th>
<th>Bottom 10%</th>
<th>Middle 10%</th>
<th>Top 10%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percentage of mothers that had at least one antenatal visit during their last pregnancy</td>
<td>17%</td>
<td>78%</td>
<td>100%</td>
</tr>
<tr>
<td>Percentage of mothers whose last child was delivered at a hospital or health clinic</td>
<td>3%</td>
<td>82%</td>
<td>95%</td>
</tr>
<tr>
<td>Did your most recent living child receive these vaccinations?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BCG</td>
<td>0%</td>
<td>97%</td>
<td>100%</td>
</tr>
<tr>
<td>DPT1</td>
<td>7%</td>
<td>96%</td>
<td>100%</td>
</tr>
<tr>
<td>Poio1</td>
<td>28%</td>
<td>97%</td>
<td>100%</td>
</tr>
<tr>
<td>DPT2</td>
<td>3%</td>
<td>92%</td>
<td>100%</td>
</tr>
<tr>
<td>Polio2</td>
<td>16%</td>
<td>92%</td>
<td>100%</td>
</tr>
<tr>
<td>DPT3</td>
<td>0%</td>
<td>83%</td>
<td>100%</td>
</tr>
<tr>
<td>Polio3</td>
<td>0%</td>
<td>57%</td>
<td>100%</td>
</tr>
</tbody>
</table>

Source: Authors calculations
Table 2

Simplified WHO classification rubric for sick children presenting to a clinic with symptoms of disease

<table>
<thead>
<tr>
<th>Primary Symptom:</th>
<th>Additional Symptoms:</th>
<th>Classify as:</th>
<th>Treatment:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cough</td>
<td>Fast breathing, and/or, chest indrawing, and/or stridor in calm child</td>
<td>Pneumonia or severe pneumonia</td>
<td>Antibiotics and urgent hospitalization if severe</td>
</tr>
<tr>
<td>Fever</td>
<td>No signs of pneumonia, measles, or severe febrile disease</td>
<td>Malaria</td>
<td>Oral anti-malarial</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>Lethargic or unconscious, sunken eyes, not able to drink, skin pinch goes back slowly, irritable</td>
<td>Severe or some dehydration</td>
<td>Fluid (ORT) and food. Refer to hospital if severe.</td>
</tr>
<tr>
<td></td>
<td>Blood in stool</td>
<td>Dysentery</td>
<td>Treat with oral antibiotic</td>
</tr>
</tbody>
</table>

Source: (World Health Organization 2006)
Table 3

Summary statistics for selected variables

<table>
<thead>
<tr>
<th>Country</th>
<th>Child Deaths</th>
<th>Child Deaths 7+</th>
<th>Treatment</th>
<th>Diarrhea</th>
<th>Cough &amp; Fast Breathing</th>
<th>Fever</th>
<th>Improved Water</th>
<th>Improved Sanitation</th>
<th>Mother's years of schooling</th>
<th>Father's years of schooling</th>
</tr>
</thead>
<tbody>
<tr>
<td>All countries</td>
<td>0.058 0.200</td>
<td>0.036 0.158</td>
<td>0.000 1.568</td>
<td>0.22 0.17</td>
<td>0.20 0.17 0.37 0.21</td>
<td>0.64 0.48</td>
<td>0.60 0.49</td>
<td>4.5 4.6</td>
<td>5.7 4.9</td>
<td></td>
</tr>
<tr>
<td>Africa</td>
<td>0.075 0.224</td>
<td>0.050 0.183</td>
<td>-0.114 1.640</td>
<td>0.24 0.17</td>
<td>0.19 0.16 0.41 0.20</td>
<td>0.61 0.49</td>
<td>0.60 0.49</td>
<td>3.3 4.1</td>
<td>4.4 4.8</td>
<td></td>
</tr>
<tr>
<td>Asia</td>
<td>0.046 0.180</td>
<td>0.024 0.132</td>
<td>-0.048 1.580</td>
<td>0.18 0.17</td>
<td>0.18 0.17 0.31 0.20</td>
<td>0.67 0.47</td>
<td>0.54 0.50</td>
<td>5.3 4.9</td>
<td>7.0 4.8</td>
<td></td>
</tr>
<tr>
<td>Latin America</td>
<td>0.034 0.155</td>
<td>0.020 0.117</td>
<td>0.371 1.273</td>
<td>0.21 0.18</td>
<td>0.26 0.21 0.35 0.21</td>
<td>0.66 0.47</td>
<td>0.73 0.44</td>
<td>6.1 4.5</td>
<td>6.8 4.6</td>
<td></td>
</tr>
<tr>
<td>Bangladesh</td>
<td>0.049 0.188</td>
<td>0.025 0.132</td>
<td>0.187 1.194</td>
<td>0.09 0.08</td>
<td>0.22 0.13 0.45 0.16</td>
<td>0.96 0.19</td>
<td>0.89 0.31</td>
<td>3.9 3.8</td>
<td>4.3 4.5</td>
<td></td>
</tr>
<tr>
<td>Brazil</td>
<td>0.032 0.146</td>
<td>0.020 0.116</td>
<td>0.960 1.062</td>
<td>0.17 0.18</td>
<td>0.27 0.22 0.32 0.22</td>
<td>0.92 0.26</td>
<td>0.82 0.38</td>
<td>5.8 3.8</td>
<td>5.4 4.0</td>
<td></td>
</tr>
<tr>
<td>Colombia</td>
<td>0.019 0.124</td>
<td>0.008 0.078</td>
<td>0.773 1.079</td>
<td>0.17 0.19</td>
<td>NA 0.24 0.89 0.31</td>
<td>0.73 0.39</td>
<td>7.3 0.4</td>
<td>7.2 4.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Egypt</td>
<td>0.035 0.158</td>
<td>0.020 0.118</td>
<td>0.714 0.934</td>
<td>0.27 0.21</td>
<td>0.14 0.16 0.40 0.24</td>
<td>0.82 0.38</td>
<td>0.92 0.27</td>
<td>6.3 5.8</td>
<td>8.3 5.6</td>
<td></td>
</tr>
<tr>
<td>Ethiopia</td>
<td>0.083 0.237</td>
<td>0.054 0.193</td>
<td>-1.517 1.632</td>
<td>0.30 0.16</td>
<td>0.28 0.16 0.37 0.18</td>
<td>0.62 0.49</td>
<td>0.20 0.40</td>
<td>1.2 2.8</td>
<td>2.3 3.9</td>
<td></td>
</tr>
<tr>
<td>Ghana</td>
<td>0.058 0.205</td>
<td>0.027 0.139</td>
<td>0.394 1.298</td>
<td>0.21 0.17</td>
<td>0.13 0.15 0.27 0.19</td>
<td>0.39 0.49</td>
<td>0.60 0.49</td>
<td>4.1 4.5</td>
<td>6.0 5.7</td>
<td></td>
</tr>
<tr>
<td>India</td>
<td>0.056 0.196</td>
<td>0.030 0.143</td>
<td>-0.455 1.751</td>
<td>0.21 0.17</td>
<td>0.21 0.17 0.32 0.19</td>
<td>0.77 0.42</td>
<td>0.40 0.49</td>
<td>3.9 4.7</td>
<td>6.4 5.0</td>
<td></td>
</tr>
<tr>
<td>Indonesia</td>
<td>0.032 0.159</td>
<td>0.018 0.120</td>
<td>0.132 1.460</td>
<td>0.11 0.13</td>
<td>0.09 0.13 0.28 0.21</td>
<td>0.39 0.49</td>
<td>0.57 0.49</td>
<td>7.9 3.9</td>
<td>8.4 4.1</td>
<td></td>
</tr>
<tr>
<td>Kenya</td>
<td>0.060 0.205</td>
<td>0.036 0.156</td>
<td>0.241 1.376</td>
<td>0.21 0.17</td>
<td>0.23 0.16 0.49 0.21</td>
<td>0.38 0.49</td>
<td>0.75 0.43</td>
<td>6.6 4.2</td>
<td>7.6 4.6</td>
<td></td>
</tr>
<tr>
<td>Morocco</td>
<td>0.032 0.152</td>
<td>0.016 0.107</td>
<td>0.825 0.980</td>
<td>0.14 0.13</td>
<td>0.14 0.14 0.30 0.19</td>
<td>0.73 0.44</td>
<td>0.75 0.43</td>
<td>2.6 4.3</td>
<td>4.0 5.0</td>
<td></td>
</tr>
<tr>
<td>Mozambique</td>
<td>0.087 0.244</td>
<td>0.063 0.209</td>
<td>0.307 1.526</td>
<td>0.18 0.13</td>
<td>0.13 0.13 0.33 0.19</td>
<td>0.62 0.48</td>
<td>0.55 0.50</td>
<td>2.5 2.8</td>
<td>4.1 3.3</td>
<td></td>
</tr>
<tr>
<td>Nepal</td>
<td>0.053 0.185</td>
<td>0.028 0.135</td>
<td>-0.040 1.275</td>
<td>0.25 0.13</td>
<td>0.28 0.18 0.38 0.15</td>
<td>0.38 0.48</td>
<td>0.25 0.43</td>
<td>1.5 3.0</td>
<td>4.5 4.2</td>
<td></td>
</tr>
<tr>
<td>Nigeria</td>
<td>0.087 0.232</td>
<td>0.055 0.184</td>
<td>-1.228 1.650</td>
<td>0.24 0.19</td>
<td>0.14 0.14 0.40 0.20</td>
<td>0.49 0.50</td>
<td>0.71 0.45</td>
<td>4.2 4.8</td>
<td>5.8 5.6</td>
<td></td>
</tr>
<tr>
<td>Peru</td>
<td>0.030 0.150</td>
<td>0.017 0.112</td>
<td>0.495 1.153</td>
<td>0.20 0.17</td>
<td>0.22 0.18 0.31 0.19</td>
<td>0.70 0.46</td>
<td>0.66 0.47</td>
<td>7.4 4.4</td>
<td>8.9 4.0</td>
<td></td>
</tr>
<tr>
<td>Philippines</td>
<td>0.026 0.138</td>
<td>0.013 0.098</td>
<td>0.512 1.283</td>
<td>0.14 0.16</td>
<td>0.13 0.15 0.30 0.21</td>
<td>0.55 0.50</td>
<td>0.83 0.37</td>
<td>9.0 3.9</td>
<td>8.7 4.1</td>
<td></td>
</tr>
<tr>
<td>South Africa</td>
<td>0.043 0.191</td>
<td>0.031 0.162</td>
<td>0.631 1.147</td>
<td>0.16 0.18</td>
<td>0.21 0.21 0.79 0.40</td>
<td>0.80 0.40</td>
<td>5.1 3.7</td>
<td>NA 0.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tanzania</td>
<td>0.089 0.244</td>
<td>0.060 0.200</td>
<td>0.760 1.034</td>
<td>0.17 0.12</td>
<td>0.20 0.13 0.45 0.19</td>
<td>0.79 0.40</td>
<td>0.80 0.40</td>
<td>5.1 3.7</td>
<td>4.0 5.0</td>
<td></td>
</tr>
<tr>
<td>Turkey</td>
<td>0.032 0.147</td>
<td>0.015 0.098</td>
<td>0.156 1.404</td>
<td>0.36 0.25</td>
<td>NA NA NA 0.69 0.46</td>
<td>0.97 0.17</td>
<td>5.0 3.7</td>
<td>7.0 3.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Uganda</td>
<td>0.072 0.210</td>
<td>0.049 0.173</td>
<td>0.056 1.363</td>
<td>0.27 0.16</td>
<td>0.28 0.16 0.53 0.23</td>
<td>0.38 0.48</td>
<td>0.83 0.38</td>
<td>4.4 3.8</td>
<td>6.6 4.2</td>
<td></td>
</tr>
<tr>
<td>Vietnam</td>
<td>0.011 0.093</td>
<td>0.005 0.060</td>
<td>0.590 1.302</td>
<td>0.11 0.14</td>
<td>0.20 0.18 0.28 0.21</td>
<td>0.74 0.44</td>
<td>0.78 0.41</td>
<td>7.1 3.8</td>
<td>7.8 3.7</td>
<td></td>
</tr>
</tbody>
</table>

Source: Calculated from DHS survey data for each country, see text for definitions
Table 4
Examples of Regression results from the four largest countries by population
(Dependent variable: Child Deaths 7+)

<table>
<thead>
<tr>
<th></th>
<th>Bangladesh</th>
<th>Brazil</th>
<th>India</th>
<th>Indonesia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>I</td>
<td>II</td>
<td>I</td>
<td>II</td>
</tr>
<tr>
<td>Treatment</td>
<td>-0.156***</td>
<td>-0.157***</td>
<td>-0.201***</td>
<td>-0.198***</td>
</tr>
<tr>
<td></td>
<td>(0.036)</td>
<td>(0.036)</td>
<td>(0.062)</td>
<td>(0.062)</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>0.620</td>
<td>0.301</td>
<td>0.363**</td>
<td>-0.134</td>
</tr>
<tr>
<td></td>
<td>(0.828)</td>
<td>(0.645)</td>
<td>(0.178)</td>
<td>(0.663)</td>
</tr>
<tr>
<td>Cough &amp; Fast Breathing</td>
<td>-0.455</td>
<td>-0.624</td>
<td>0.051</td>
<td>1.338***</td>
</tr>
<tr>
<td></td>
<td>(0.582)</td>
<td>(0.599)</td>
<td>(0.190)</td>
<td>(0.578)</td>
</tr>
<tr>
<td>Fever</td>
<td>1.035</td>
<td>-0.315</td>
<td>0.141</td>
<td>-0.591</td>
</tr>
<tr>
<td></td>
<td>(0.537)</td>
<td>(0.545)</td>
<td>(0.176)</td>
<td>(0.450)</td>
</tr>
<tr>
<td>Improved Water</td>
<td>-0.393</td>
<td>-0.315</td>
<td>0.141</td>
<td>-0.591</td>
</tr>
<tr>
<td></td>
<td>(0.322)</td>
<td>(0.545)</td>
<td>(0.176)</td>
<td>(0.450)</td>
</tr>
<tr>
<td>Improved Sanitation</td>
<td>-0.094</td>
<td>-0.196</td>
<td>-0.029</td>
<td>-0.045</td>
</tr>
<tr>
<td></td>
<td>(0.190)</td>
<td>(0.286)</td>
<td>(0.081)</td>
<td>(0.147)</td>
</tr>
<tr>
<td>Mother’s education</td>
<td>-0.074**</td>
<td>-0.071**</td>
<td>-0.089**</td>
<td>-0.090**</td>
</tr>
<tr>
<td></td>
<td>(0.029)</td>
<td>(0.029)</td>
<td>(0.037)</td>
<td>(0.038)</td>
</tr>
<tr>
<td>Father’s Education</td>
<td>0.000</td>
<td>0.001</td>
<td>-0.158**</td>
<td>-0.158**</td>
</tr>
<tr>
<td></td>
<td>(0.023)</td>
<td>(0.023)</td>
<td>(0.037)</td>
<td>(0.038)</td>
</tr>
<tr>
<td>Urban</td>
<td>0.190</td>
<td>0.194</td>
<td>0.270</td>
<td>0.293</td>
</tr>
<tr>
<td></td>
<td>(0.161)</td>
<td>(0.162)</td>
<td>(0.280)</td>
<td>(0.289)</td>
</tr>
<tr>
<td>Months at risk</td>
<td>0.015***</td>
<td>0.015***</td>
<td>0.020***</td>
<td>0.020***</td>
</tr>
<tr>
<td></td>
<td>(0.004)</td>
<td>(0.004)</td>
<td>(0.006)</td>
<td>(0.005)</td>
</tr>
<tr>
<td>Wealth</td>
<td>-0.056</td>
<td>-0.058</td>
<td>-0.037</td>
<td>-0.000</td>
</tr>
<tr>
<td></td>
<td>(0.041)</td>
<td>(0.041)</td>
<td>(0.065)</td>
<td>(0.062)</td>
</tr>
<tr>
<td>Seasonal dummies</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>State/Region dummies</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Diagnostic statistics:</td>
<td>P-value for joint test of Significance of Morbidity/water&amp; sanitation indicators</td>
<td>0.182</td>
<td>0.434</td>
<td>0.510</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>6753</td>
<td>6746</td>
<td>3504</td>
</tr>
<tr>
<td></td>
<td>Probability &gt; F</td>
<td>0.0000</td>
<td>0.0000</td>
<td>0.0000</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>7.76</td>
<td>9.14</td>
<td>4.93</td>
</tr>
</tbody>
</table>

Note: See section 5.1 for a description of the regressions. Child Deaths 7+ is a dummy variable set to zero if the child is alive, and one if the died more than seven days after birth.

***: denotes coefficient significant at 1% level
** : denotes coefficient significant at 5% level
* : denotes coefficient significant at 10% level
Table 5:
Results from global regressions

<table>
<thead>
<tr>
<th></th>
<th>Logit Unadjusted data</th>
<th>Logit Matched data</th>
<th>OLS Cluster averages, matched data</th>
<th>OLS Unadjusted data</th>
<th>IV Heckman</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment</td>
<td>-0.1164*** (0.0073)</td>
<td>-0.1123*** (0.0065)</td>
<td>-0.0079*** (0.0011)</td>
<td>-0.0057*** (0.0003)</td>
<td>-0.0043*** (0.0009)</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>0.3475*** (0.0691)</td>
<td>0.3083*** (0.0611)</td>
<td>0.0150*** (0.0042)</td>
<td>0.0151*** (0.0027)</td>
<td>0.0154*** (0.0034)</td>
</tr>
<tr>
<td>Cough &amp; Fast Breathing</td>
<td>0.0136 (0.0748)</td>
<td>-0.0344 (0.0659)</td>
<td>-0.0048 (0.0037)</td>
<td>-0.0003 (0.0029)</td>
<td>0.0000 (0.0033)</td>
</tr>
<tr>
<td>Fever</td>
<td>0.1569* (0.0624)</td>
<td>0.1444*** (0.0548)</td>
<td>0.0101** (0.0040)</td>
<td>0.0062*** (0.0024)</td>
<td>0.0063* (0.0033)</td>
</tr>
<tr>
<td>Mother’s Education</td>
<td>-0.0257*** (0.0044)</td>
<td>-0.0306*** (0.0038)</td>
<td>-0.0015*** (0.0004)</td>
<td>-0.0003** (0.0001)</td>
<td>-0.0003 (0.0002)</td>
</tr>
<tr>
<td>Father’s Education</td>
<td>-0.0210*** (0.0034)</td>
<td>-0.0203*** (0.0029)</td>
<td>-0.0009*** (0.0004)</td>
<td>-0.0007** (0.0001)</td>
<td>-0.0007*** (0.0001)</td>
</tr>
<tr>
<td>Urban</td>
<td>-0.0641** (0.0315)</td>
<td>-0.0265 (0.0270)</td>
<td>0.0014 (0.0015)</td>
<td>-0.0021** (0.0010)</td>
<td>-0.0026 (0.0015)</td>
</tr>
<tr>
<td>Months at risk</td>
<td>0.0325*** (0.0006)</td>
<td>0.0242*** (0.0005)</td>
<td>0.0005*** (0.0001)</td>
<td>0.0013*** (0.0000)</td>
<td>0.0013*** (0.0002)</td>
</tr>
<tr>
<td>Wealth variable for each country</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Seasonal Dummies</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Country Dummies</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Inverse Mills Ratio</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.1295 (0.0727)</td>
</tr>
<tr>
<td>N</td>
<td>271639</td>
<td>278601</td>
<td>27092</td>
<td>271641</td>
<td>271641</td>
</tr>
<tr>
<td>Prob &gt; F</td>
<td>0.0000</td>
<td>0.0000</td>
<td>0.0000</td>
<td>0.0000</td>
<td>0.0000</td>
</tr>
</tbody>
</table>

Note: See section 5.2 for a description of regressions. Child Deaths 7+ is a dummy variable set to zero if the child is alive, and one if the died more than seven days after birth.

***: denotes coefficient significant at 1% level
**: denotes coefficient significant at 5% level
*: denotes coefficient significant at 10% level
Table 6
Results from global regressions
Dependent Variable: Child Deaths 7+

<table>
<thead>
<tr>
<th></th>
<th>Logit</th>
<th></th>
<th>OLS</th>
<th></th>
<th>IV</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Unadjusted data</td>
<td>Matched Data</td>
<td>Cluster averages, matched data</td>
<td>Unadjusted data</td>
<td>Heckman</td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>-0.1195</td>
<td>-0.1150</td>
<td>-0.0084</td>
<td>-0.0057</td>
<td>-0.0045</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.0073)</td>
<td>(0.0065)</td>
<td>(0.0010)</td>
<td>(0.0003)</td>
<td>(0.0009)</td>
<td></td>
</tr>
<tr>
<td>Improved water</td>
<td>-0.0023</td>
<td>-0.0171</td>
<td>0.0006</td>
<td>0.0000</td>
<td>-0.0001</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.0221)</td>
<td>(0.0193)</td>
<td>(0.0019)</td>
<td>(0.0009)</td>
<td>(0.0009)</td>
<td></td>
</tr>
<tr>
<td>Improved sanitation</td>
<td>-0.0124</td>
<td>-0.0224</td>
<td>-0.0018</td>
<td>-0.0010</td>
<td>-0.0013</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.0265)</td>
<td>(0.0234)</td>
<td>(0.0035)</td>
<td>(0.0010)</td>
<td>(0.0014)</td>
<td></td>
</tr>
<tr>
<td>Mother’s Education</td>
<td>-0.0273</td>
<td>-0.0318</td>
<td>-0.0017</td>
<td>-0.0004</td>
<td>-0.0003</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.0044)</td>
<td>(0.0038)</td>
<td>(0.0004)</td>
<td>(0.0001)</td>
<td>(0.0002)</td>
<td></td>
</tr>
<tr>
<td>Father’s Education</td>
<td>-0.0193</td>
<td>-0.0189</td>
<td>-0.0008</td>
<td>-0.0007</td>
<td>-0.0006</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.0034)</td>
<td>(0.0029)</td>
<td>(0.0004)</td>
<td>(0.0001)</td>
<td>(0.0001)</td>
<td></td>
</tr>
<tr>
<td>Urban</td>
<td>-0.0584</td>
<td>-0.0152</td>
<td>0.0019</td>
<td>-0.0019</td>
<td>-0.0023</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.0315)</td>
<td>(0.0270)</td>
<td>(0.0016)</td>
<td>(0.0011)</td>
<td>(0.0015)</td>
<td></td>
</tr>
<tr>
<td>Months at risk</td>
<td>0.0325</td>
<td>0.0242</td>
<td>0.0004</td>
<td>0.0013</td>
<td>0.0013</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.0006)</td>
<td>(0.0005)</td>
<td>(0.0001)</td>
<td>(0.0000)</td>
<td>(0.0002)</td>
<td></td>
</tr>
<tr>
<td>Wealth variable</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>for each country</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Country Dummies</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>Inverse Mills Ratio</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.1075</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(0.0763)</td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>270929</td>
<td>277862</td>
<td>27092</td>
<td>270929</td>
<td>270929</td>
<td></td>
</tr>
<tr>
<td>Prob &gt; F</td>
<td>0.0000</td>
<td>0.0000</td>
<td>0.0000</td>
<td>0.0000</td>
<td>0.0000</td>
<td></td>
</tr>
</tbody>
</table>

Note: See section 5.2 for a description of the regressions. Child Deaths 7+ is a dummy variable set to zero if the child is alive, and one if the died more than seven days after birth.

***: denotes coefficient significant at 1% level
**: denotes coefficient significant at 5% level
*: denotes coefficient significant at 10% level
### Table 7
The determinants of treatment, and the relation between child mortality, treatment and the share of child health services sought in the private sector

<table>
<thead>
<tr>
<th>Dependent variable:</th>
<th>Treatment (OLS regression, original data, household level)</th>
<th>Child Deaths 7+ (Logit regression, matched data, child outcomes)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>I</td>
<td>II</td>
</tr>
<tr>
<td>Treatment</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment* (Share of child health services sought in the private sector)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Share of child health services sought in the private sector</td>
<td>0.062***</td>
<td>0.068***</td>
</tr>
<tr>
<td></td>
<td>0.008</td>
<td>0.010</td>
</tr>
<tr>
<td>Mother’s years of schooling</td>
<td>0.030***</td>
<td>0.029***</td>
</tr>
<tr>
<td></td>
<td>0.005</td>
<td>0.006</td>
</tr>
<tr>
<td>Father’s years of schooling</td>
<td>0.284***</td>
<td>0.209***</td>
</tr>
<tr>
<td></td>
<td>0.052</td>
<td>0.038</td>
</tr>
<tr>
<td>Urban</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distance to nearest public health center</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diarrhea</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cough &amp; Fast Breathing</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fever</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of children in household at start of observation period</td>
<td><strong>-0.014</strong></td>
<td>-0.020</td>
</tr>
<tr>
<td></td>
<td>0.011</td>
<td>0.016</td>
</tr>
<tr>
<td>Months at risk</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wealth indicators for each country</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Seasonal dummies by country</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Country dummies</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Number of observations</td>
<td>191453</td>
<td>127036</td>
</tr>
<tr>
<td>Prob &gt; F</td>
<td>0.0000</td>
<td>0.0000</td>
</tr>
</tbody>
</table>

Note: See section 5.4 and 7.2 for a description of the regressions. *Child Deaths 7+* is a dummy variable set to zero if the child is alive, and one if the died more than seven days after birth.

***: denotes coefficient significant at 1% level, **: denotes coefficient significant at 5% level, *: denotes coefficient significant at the 10% level.
Table 8
Robustness tests for the impact of improved water in Indian Village Data and Global Data

<table>
<thead>
<tr>
<th>Dataset</th>
<th>Indian Villages</th>
<th>Global matched</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dependent variable:</td>
<td>Child Deaths 7+</td>
<td>Child Deaths 7+</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Instrument</th>
<th>Access to surface water for agriculture</th>
<th>Time needed to get drinking water</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OLS</td>
<td>IV</td>
</tr>
<tr>
<td>Treatment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>-0.0056</td>
<td>-0.0051</td>
</tr>
<tr>
<td></td>
<td>(0.0037)</td>
<td>(0.0040)</td>
</tr>
<tr>
<td>Improved Water</td>
<td>0.0241</td>
<td>0.0727</td>
</tr>
<tr>
<td></td>
<td>(0.0172)</td>
<td>(0.1623)</td>
</tr>
<tr>
<td>Improved sanitation</td>
<td>0.0171</td>
<td>0.0170</td>
</tr>
<tr>
<td></td>
<td>(0.0161)</td>
<td>(0.0163)</td>
</tr>
<tr>
<td>Mother’s years of schooling</td>
<td>-0.0034</td>
<td>-0.0033</td>
</tr>
<tr>
<td></td>
<td>(0.0023)</td>
<td>(0.0023)</td>
</tr>
<tr>
<td>Father’s years of schooling</td>
<td>-0.0010</td>
<td>-0.0008</td>
</tr>
<tr>
<td></td>
<td>(0.0015)</td>
<td>(0.0016)</td>
</tr>
<tr>
<td>Months at Risk</td>
<td>0.0008</td>
<td>0.0007</td>
</tr>
<tr>
<td></td>
<td>(0.0006)</td>
<td>(0.0007)</td>
</tr>
<tr>
<td>Wealth</td>
<td>-0.0036</td>
<td>-0.0042</td>
</tr>
<tr>
<td></td>
<td>(0.0027)</td>
<td>(0.0032)</td>
</tr>
<tr>
<td>Seasonal dummies</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Country dummies</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Number of observations</td>
<td>508</td>
<td>508</td>
</tr>
<tr>
<td>F</td>
<td>2.5900</td>
<td>2.4900</td>
</tr>
<tr>
<td>R-squared</td>
<td>0.14</td>
<td>0.13</td>
</tr>
</tbody>
</table>

Note: See section 6.2.1 for a description of the regressions. Child Deaths 7+ is a dummy variable set to zero if the child is alive, and one if the died more than seven days after birth.

***: denotes coefficient significant at 1% level
** : denotes coefficient significant at 5% level
*: denotes coefficient significant at 10% level
Table 9

Proportion of mothers and children that received specific maternal and child healthcare services
(these services were not used to compute the treatment index)

<table>
<thead>
<tr>
<th>Ranking of household according to global Treatment indicator</th>
<th>Bottom 10%</th>
<th>Middle 10%</th>
<th>Top 10%</th>
</tr>
</thead>
<tbody>
<tr>
<td>For a child with the following symptoms of illness during the last two weeks, did you seek out any treatment?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diarrhea</td>
<td>32</td>
<td>49</td>
<td>53</td>
</tr>
<tr>
<td>Cough or fever</td>
<td>36</td>
<td>65</td>
<td>67</td>
</tr>
<tr>
<td>The percentage of mothers that followed WHO recommended practices related to breast feeding:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breastfed child within one hour after birth</td>
<td>40</td>
<td>60</td>
<td>59</td>
</tr>
<tr>
<td>Breastfed child for first six months</td>
<td>98</td>
<td>99</td>
<td>88</td>
</tr>
<tr>
<td>Percentage of households where the youngest son aged 10 months or over has been vaccinated for measles</td>
<td>6</td>
<td>89</td>
<td>92</td>
</tr>
<tr>
<td>Intervention Measure</td>
<td>Description</td>
<td></td>
<td></td>
</tr>
<tr>
<td>--------------------------------------------------------------------------------------</td>
<td>-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exclusive breastfeeding (0-6 months)</td>
<td>Counseling to give only breastmilk to children from birth to 6 months of age</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breastfeeding (6-11 months)</td>
<td>Counseling to continue breastfeeding, on demand if possible, from 6 to 11 months of age</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Complementary feeding</td>
<td>Counseling on proper feeding of infants 6-23 months of age with adequate and safe foods in addition to breastmilk, and growth monitoring and promotion.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Zinc</td>
<td>Four rounds of zinc supplements given to caretaker when child is between 2 and 23 months of age</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vitamin A</td>
<td>One dose delivered six months from 7 to 59 months; 9 doses total</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Measles vaccine</td>
<td>One dose at 9 months.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hib vaccine</td>
<td>Three doses within 1st year, delivered with DPT</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tetanus toxoid</td>
<td>Two doses during pregnancy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clean delivery (skilled attendant at birth)</td>
<td>“Essential newborn care”: clean hands, clean perineum, nothing unclean introduced into vagina, clean delivery surface, cleanliness in cutting the umbilical cord, cleanliness for cord care of newborn</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Newborn temperature management</td>
<td>Thermal protection for all newborns and provision of extra care for low birthweight babies, including &quot;kangaroo mother care&quot; which entails nursing the stable, low birthweight baby skin-to-skin and tied to the mother's front</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antibiotics for preterm premature rupture of membranes (PPROM)</td>
<td>Oral erythromycin 250 mg 8 hourly x 7 days for PPROM before delivery</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antenatal Steroids</td>
<td>Two injections of betamethasone after onset of premature labour</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nevirapine and replacement feeding</td>
<td>Anti-retroiral drugs for the mother (Nevirapine) and breastmilk substitute (formula) for 12 months.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insecticide-treated materials</td>
<td>One impregnated net every three years for each HH with ≥ 1 child under five</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antimalarial intermittent preventive treatment in pregnancy</td>
<td>Minimum 3 doses of sulfadoxine-pyrimethamine (SP, or Fansidar) within the 2nd &amp; 3rd trimesters of each pregnancy. Dose = 3 tablets of 500 S + 25 mg P</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Water, sanitation, hygiene</td>
<td>Provision of equipment and materials needed to move from UNICEF category of access to safe water “not improved” to least costly country-specific “improved” access to safe water</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Zinc for diarrhea</td>
<td>Zinc suspension or dispersable tablet for 10 days (20mg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vitamin A for tx of measles</td>
<td>Vitamin A (200,000 units)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oral rehydration therapy</td>
<td>For children with at least some dehydration, ORS in facility (1 sachet administered and 2 sent home with mother); at home, ORT/increased fluids and continued feeding</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antibiotics for dysentery</td>
<td>Ciprofloxacin (150mg/day for 3 days) + zinc suspension or dispersable tablet for 10 days + ORT (3 sachets)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antibiotics for pneumonia</td>
<td>Amoxicillin (500mg/day for 3 days)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antibiotics for sepsis</td>
<td>Tx course of injectable gentimcin and injectable ampicillin for 7 days</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Newborn resuscitation</td>
<td>Resuscitation of newborn who is not breathing using bag and mask</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antimalarials</td>
<td>Effective antimalarial</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Figure 1: Child mortality and GDP per capita by country, 2003
Figure 2: The probability of a child dying at different ages

Source: calculated from Demographic Health Surveys in 45 countries
Figure 3: Estimated causes of neonatal and post-neonatal child deaths (2003)

(a) Non-neonatal Child Deaths:

- Diarrhoea 34%
- Pneumonia 34%
- Malaria 14%
- Other 14%
- AIDS 4%

Source: Black et. al. (2003)
(b) Neonatal Deaths

Source: Black et. al. (2003)
Figure 4: Average morbidity prevalence
Figure 5a: The impact of a one standard deviation rise in treatment on the probability of Child Deaths 7+ in regressions with morbidity indicators

Note: Dotted lines show +/- 2 standard deviation bands around point estimates.

Figure 5b: The impact of a one standard deviation fall in the prevalence of diarrhea on the probability of Child Deaths 7+.

Note: Dotted lines show +/- 2 standard deviation bands around point estimates.
Figure 5c: The impact of a one standard deviation fall in the prevalence of cough with fast breathing on the probability of Child Deaths 7+.

Note: Dotted lines show +/- 2 standard deviation bands around point estimates.

Figure 5d: The impact of a one standard deviation fall in the prevalence of fever on the probability of Child Deaths 7+.

Note: Dotted lines show +/- 2 standard deviation bands around point estimates.
Figure 5e: The impact of a one standard deviation rise in years of mothers’ education on the probability of Child Deaths 7+

![Figure 5e](image)

Note: Dotted lines show +/- 2 standard deviation bands around point estimates.

Figure 5f: The impact of a one standard deviation rise in years of father’s education on the probability of Child Deaths 7+

![Figure 5f](image)

Note: Dotted lines show +/- 2 standard deviation bands around point estimates
Figure 5g: The impact of a one standard deviation rise in wealth on the probability of Child Deaths 7+.

Note: Dotted lines show +/- 2 standard deviation bands around point estimates
Figure 6a: The impact of a one standard deviation rise in improved water on the probability of Child Deaths 7+

Note: Dotted lines show +/- 2 standard deviation bands around point estimates

Figure 6b: The impact of a one standard deviation rise in improved sanitation on the probability of Child Deaths 7+

Note: Dotted lines show +/- 2 standard deviation bands around point estimates
Figure 7: The average impact of a one standard deviation change in explanatory variables on Child Death 7+ in global data

Note: The chart shows the predicted impact of a one standard deviation rise in Treatment, a one standard deviation rise in mother’s years of schooling, and a one standard deviation rise in father’s years of schooling. “Morbidity” shows the combined impact of a one standard deviation fall in diarrhea, cough with fast breathing and fever. Source: Calculations based on regression results from Table 5.
Figure 8: The average impact of a one standard deviation change in explanatory variables on Child Death 7+ in global data

Note: The chart shows the predicted impact of a one standard deviation rise of Treatment, the combined impact of a one standard deviation increase in improved water and sanitation, and the impact of a one standard deviation rise in years of mother’s and father’s education respectively.

Source: Calculations based on regression results from Table 6.
Figure 9: The impact of a one standard deviation rise in “Health Knowledge on Treatment (measured as a percentage of one standard deviation of treatment)
Figure 10: The average impact of a one standard deviation change in explanatory variables on Child Death 7+ under alternative robustness tests in global data

Note:
1. The chart shows the predicted impact of a one standard deviation rise of treatment, the combined impact of a one standard deviation increase in improved water and sanitation, and the impact of a one standard deviation rise in years of mother’s and father’s education respectively. “Morbidity” shows the combined impact of a one standard deviation fall in diarrhea, cough with fast breathing and fever.
2. These results are calculated from the following regressions:
   - **Logit-base case**: Global matched data as in Table 5, column 2
   - **Health knowledge added**: Health knowledge variable added as control to base case
   - **Malaria endemic regions**: Interaction term for malaria endemic zones with fever added as control to base case
   - **Size of private sector**: Proportion of child health services in the private sector for each cluster interacted with treatment and added as a regressor to base case
   - **Mother’s age and low birth weight**: Mother’s age and a dummy variable equal to one if child was “small or very small” at birth added to regression equation
   - **Child Deaths as dependent variable**: Child Deaths instead of Child Deaths 7+, i.e. including children that died during the first seven days after birth, as the dependent variable in base case.
Figure 11: The impact of four alternative packages aimed at reducing child mortality in global data

Note:
The chart shows the point estimates for the impact of changes to right hand side variables based on the regression results using logit matched-data as reported in Table 5, and the similar regression in Table 6 when we calculate the impact of improved water and sanitation.
Figure 12: Treatment and the share of child health services sought in the private sector
Figure 13: Inequality of treatment and the share of child health services sought in the private sector
Figure 14: Child Deaths 7+ compared to the share of child health services sought in the private sector

![Graph showing the relationship between child deaths 7+ and the share of child health services sought in the private sector across various countries.](image)
Figure 15: Child Deaths 7+ compared to the share of child health services sought in the private sector (households in the bottom wealth quartile only)
References:


