CENTRE for ECONOMIC PERFORMANCE

CEP Discussion Paper No 751

October 2006

Lowering Child Mortality in Poor Countries: The Power of Knowledgeable Parents

Peter Boone and Zhaoguo Zhan

Abstract
Why do over 20% of children die in some poor countries, while in others only 2% die? We examine this question using survey data covering 278,000 children in 45 low-income countries. We find that parents' education and a mother's propensity to seek out modern healthcare are empirically important when explaining child survival, while the prevalence of common diseases, along with infrastructure such as improved water and sanitation, are not. Using a GINI coefficient we construct for treatment services, we find that public and private health systems are "equally unequal", that is, both tend to favor children in relatively well-off households, and neither appears superior at improving outcomes in very poor communities. These facts contrast with a common view that a much-expanded public health sector is necessary to reduce child mortality. Instead, we believe the empirical evidence points to the essential role of parents as advocates for their child's health. If we can provide better health knowledge and general education to parents, a private healthcare sector can arise to meet demand. We provide evidence that this alternative route to low mortality is indeed a reason behind the current success of many countries with low child mortality, including Vietnam, Indonesia, Egypt, and the Indian state of Kerala. Finally, we calculate a realistic package of interventions that target education, health knowledge and treatment seeking could reduce child mortality by 32%.

Acknowledgements
We are grateful for helpful comments and discussions with: Raj Arole, Anthony Costello, Diana Elbourne, Brigitte Granville, April Harding, Simon Johnson, and seminar participants at the Conference on Neonatal Mortality in Andhra Pradesh and the London School of Economics. Zhaoguo Zhan received financial support from Effective Intervention. Effective Intervention is a UK registered charity that designs and sponsors intervention trials to reduce child mortality in Asia and Africa.

Peter Boone is a Research Associate of the Globalisation Programme at the Centre for Economic Performance, LSE and Chair of Effective Intervention. Zhaoguo Zhan is a PhD student at the Department of Economics, Brown University.

JEL Classification Numbers: I00, I1, I12, I18

Published by
Centre for Economic Performance
London School of Economics and Political Science
Houghton Street
London WC2A 2AE

ISBN 0 7530 2057 2

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 five varies from 2% to 22%. Even after controlling for income, there is large variation in mortality rates that is not well-explained.

When considering what may be effective at reducing mortality, the gold standard for evidence is the randomized controlled trial. 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). The authors of that study argued that we need to build out large public health systems in order to ensure all children receive the 24 specified interventions.

However, from a policy perspective, such lists leave many questions unanswered. We can never be sure that the ideal operating conditions of intervention trials can be replicated closely enough when being scaled-up, and they tell us little about systemic design which is needed to ensure that interventions are implemented and sustained.

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 trial results really are.

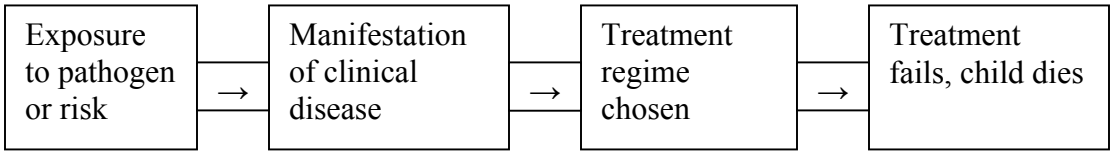
In this paper we use household survey data covering 278,000 children in 45 countries to address this agenda. We ask two basic questions: First, what is the relative importance of different socioeconomic, environmental, infrastructural and institutional

factors in explaining differences in the probability of a child surviving. Second, what implications do these findings have for the design, and in particular the systemic design, of interventions that aim to reduce child mortality.

The Demographic Health Suvey (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.[1] 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.[2]

We motivate our empirical approach by the simple figure below:

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. We run logit regressions to determine the relative empirical importance of key factors that would influence the probability of death at each stage.

Our empirical results demonstrate consistently, in both single country and global pooled data, that the prevalence of symptoms of common diseases that kill children is not a good predictor for child mortality risk. In other words, children with low mortality risk tend to get sick nearly as often as those with high risk (after conditioning on household characteristics) in poor countries.

[1] The data is available at www.measuredhs.com

[2] 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, political, institutional and other factors that may determine whether specific interventions ultimately work.

However, we find strong evidence that actions taken after a child is exposed to disease are highly important when predicting mortality. Using an index we create, we find that the propensity of a household to use modern healthcare is a good predictor for child survival outcomes. We also find that the maternal and paternal education is highly significant and equally empirically important.

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%.[3] In contrast, were we to halve the prevalence of diarrhea, fever, and cough with fast breathing, we calculate that child mortality would fall by only 3%. If all households were allocated improved water and sanitation, child mortality would fall by 2%.

It is important to understand whether general education is needed, or specific health knowledge is more important, however our surveys do not provide sufficient child-related health knowledge data across countries to measure this. We do provide evidence from India, where there are more detailed questions on health knowledge, that health knowledge alone may be a major factor explaining differences in survival outcomes.

This suggests that one route to success, amongst the countries in our dataset, is through better educated parents who seek out modern healthcare. Since parents are naturally the greatest advocates for their child's survival, and the treatments needed to prevent deaths are simple and inexpensive, it is reasonable to believe that those parents, armed with such education and knowledge, can prevent deaths. It is less clear whether or not we need to build public healthcare infrastructure, since increased demand for services that should come with knowledge may itself push the private sector to expand the main services needed to support child health.

The WHO *Commission on Macroeconomics and Health (2001)* argued against reliance on a private sector health supply in low income countries because the Commission thought it would be lower quality, and less equitable. This is plausible;

[3] This experiment assumes those with greater education, and those above the 53rd *Treatment* percentile as described in section 7, remain at their current levels.

however, public systems also tend to be biased towards the elite, so it is ultimately an empirical question. Our data includes information on the relative importance of the public and private sector in each community and nation, so we are able to analyze whether the above claims are correct.

We find no evidence that nations, or communities, with relatively large public health systems perform better or worse than regions with larger private systems. We find that public systems are as "biased", in terms of the distribution of health services, as private systems. They both tend to serve well-educated, wealthy people better than less-educated, poor people. This conclusion holds when we restrict the sample to the poorest quartile of the population in each nation.

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 includes "socialist" countries such as Vietnam, along with Egypt, Indonesia and the Indian state of Kerala. In China, approximately 70% of health service costs are paid by users. In these countries and regions, parental demand has clearly driven the supply of child 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 that demand.

This alternative route to low mortality could be especially attractive in regions where the public sector is weak, volatile, or lacks sustained finances. We believe more well-designed research efforts, including randomized controlled trials, are needed to determine the potential short-term impact of such policies, and the best means to implement them.

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.[4] It is clear from this data that the 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. [5]

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.

[4] The chart records the percentage of children who were in that age category 12 months prior to the survey and who subsequently died.

[5] 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)..

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:

$$P^D(X,T) = P^E(X) \times P^{D|E}(X,T) \quad (1)$$

X :	Vector of household and child characteristics
T:	Index of treatment pattern in response to exposure to risks or disease
P^D:	Probability that child dies
P^E:	Probability that child is exposed to disease
$P^{D\|E}$:	Probability that child dies after having been exposed to disease

Here we include a vector of conditioning variables, X, such as household wealth and parents' 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:[6]

$$P^D{}_k = 1 - \prod_{j=1}^{J} \left[1 - P^E_{jk} \cdot P^{D|E}_{jk} \right]^{M_k} \quad (2)$$

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

[6] 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.

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.

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:

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

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:[7]

$$(4) \qquad \overline{D} = \alpha_{o} + \sum_{j=1}^{J} \alpha_{1j} \cdot \overline{P}_{j}^{E} + \alpha_{2} \cdot \overline{P}^{D|E} + \alpha_{3} \cdot \overline{M}_{k} + \alpha_{4} \cdot \overline{X} + \varepsilon$$

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

[7] This equation ignores second order terms which, in the case of child mortality risk, will be small.

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

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. We derive a reduced-form regression equation for treatment based on the solution to a utility maximization problem where guardians face a trade-off between consumption and child health services, subject to a budget constraint and standard concavity assumptions needed to ensure a solution.[8]

This reduced form equation implies we should regress treatment on household characteristics, the price of health services, and a regression error term:

$$(5) \qquad T_i = \gamma_0 + \gamma_1 \cdot X_i + \gamma_2 \cdot P^T + \lambda_i$$

where p^T is the price of treatment services, and X_i is a vector of household characteristics relevant to treatment. In this derivation, we assume that treatment is available at a constant price for each household.[9]

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

[8] The model and derivation are available on request.

[9] 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.

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.

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. In every regression we limit included clusters to those where at least four observations are available.[10]

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.[11] These authors used principal components to build single indicators for wealth 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.

We describe several key variables below, while the appendix has a glossary describing all variables.

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[10] 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.

[11] See also Rutstein, S. O., K. Johnson, et al. (2004).

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.[12]

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.[13] To calculate this we take all measures of preventive healthcare used by the family which are unlikely to have a significant impact on Child Deaths 7+, and are uncorrelated with whether a child is actually diseased. 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 little or no impact on mortality rates as these diseases no longer cause many deaths. In order to create one indicator for each household, we used data for the eldest living child that was under five years of age. [14,15] We also included the only two available indictors for mother's

[12] 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.

[13] Girls and boys may be treated differently; however for the purposes of this paper we have not examined this question.

[14] The *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

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 after seven days of age, i.e. *Child Deaths7+* .

We calculate our *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. We believe *Treatment* provides a good proxy for the relative propensity of children to be treated by modern healthcare.

Morbidity Indicators:

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. [16]

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.

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any endogeneity that could arise if younger children, who are born after a recent child death, are treated differently.

[15] 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.

[16] 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.

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.[17] Figure 4 illustrates the large variation in the prevalence of diarrhea, and the prevalence of symptoms of acute respiratory infections (ARI), across countries in our dataset.[18]

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.[19]

Other variables

The remaining variables are self-explanatory and defined in Appendix 1.

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

[17] 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.

[18] 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.

[19] We checked for bias due to this in our regressions by restricting our regressions to households where interviews were done in similar seasons.

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.[20] 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.

4.3 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

[20] 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.

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. If we aim to capture the relevance of variables that have a smaller impact on Child Deaths 7+, we would need substantially more observations.[21]

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 cluster averaged data, meaning that these are adequately powered to estimate the impact of variables that have a small impact on mortality outcomes.

5. What factors predict child deaths?

We begin by examining the trends in single country regressions. We then examine results when we pool the date from all 45 countries in our global regressions. Finally, we show results for the determinants of Treatment. In section 6 we discuss the results further, and also examine robustness and the implications for causality.

[21] 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 Deathss 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 Single country regression results: What predicts child survival outcomes?

Table 4 shows detailed logit regression results for the four most populated countries in our dataset as motivated by (2). Figures 5a-g use these results to calculate the predicted percentage increase in the probability of a child death due to a one standard deviation change in right hand side variables.

There are several common trends in this data. First, Treatment, along with parents' education are generally highly significant in each regression. As illustrated in Figure 5, a one standard deviation rise in treatment, mother's and father's education predicts a 14%, 12% and 10% decline in child mortality respectively.

The morbidity indicators are rarely significant, neither individually nor jointly. As illustrated in Figure 5b, 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%.

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. Since improved sanitation and water are believed to reduce the incidence of diarrhea, and possibly pneumonia and malaria also, we included these as alternatives to morbidity indicators in our regressions.[22]

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

[22] 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).

predictions for the impact of improved water and improved sanitation based on these single country regressions.

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.

5.2 Global data: What predicts child survival?

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 4.3, 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. In general the coefficient estimates in Figures 5 and 6 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 determinants of mortality risk within and across countries. We refer to this data as the "global" data. Since our wealth variables cannot be compared across countries, we permit the coefficient on this variable to vary for each country. We also permit coefficients to vary on seasonal dummies for each country reflecting the time of year that the household was interviewed. We include country dummies for each nation. We pool all other variables in our regressions.

The results from the global regressions are presented in Tables 5 and 6, while Figures 7 and 8 calculate the implied impact of a one standard deviation change in key variables on child mortality. The five columns in Tables 5 and 6 reflect different

techniques used to deal with possible missing variable bias as discussed in section 3, and regressions where we use cluster averages instead of individual child data.

Figures 7 and 8 show that the results do not change when we use cluster averages or individual level regressions, and they are also robust to alternative methods to deal with missing variable bias. The results in column V of Tables 5 and 6 show that the inverse Mills ratio is insignificant in both regressions, which suggests any selection bias is small.

In our global data we once again find that treatment is roughly twice as important as morbidity when predicting survival outcomes. Our estimates of the empirical importance of morbidity in Figures 7 and 8 assume that all three measures of morbidity simultaneously decline by one standard deviation, something that would be very hard to engineer in practice.

The role of parents' education is highly significant in each regression and the combined sum of mother's and father's education is roughly as important as treatment when predicting mortality outcomes. The variation in the point estimates on mother's and father's education probably reflects the high collinearity between these indicators.

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 mortality.

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.

5.3 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.

Table 7 presents results from our global regression.[23] 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 regressions suggest education is the single most important explanatory variable when determining treatment. 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 the regression, and permitted coefficients to vary by country, we do not measure the impact of wealth in this regression.

In section 6.1.4 we discuss whether education per se, or health knowledge more specifically, is important for reducing mortality. In Figure 9 we illustrate that there is a strong relation between specific health knowledge, in this case an index calculated using answers to questions on AIDS knowledge and family planning, and treatment in many countries. However, the causality here is not clear: perhaps households that know more

[23] 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.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.

go to clinics, however it could be that they learn their health knowledge because they visit clinics more often.

We present this result because it could have important policy implications. If households get treatment because they are better informed about the need and benefits, then improving health knowledge could substantially raise treatment, and this in turn could reduce mortality rates. Given that general education 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 an effective means to substantially change treatment-seeking and reduce child mortality.[24]

6. Further discussion, robustness and causality

The regression results from Section 5 demonstrate, at the least, that the propensity to treat children, and parents' 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 and measurement error; (ii) if endogeneity of right hand side variables could be biasing results for improved water and sanitation; and (iii) if the large impact is plausible given results from intervention trials and other sources.

[24] We also ran treatment regressions in global data (not reported here) and found similar results to the single country regressions.

6.1 Disaggregating our principal components and expanding 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 are 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, and only one question asking "have you heard of ORT" relates to child health. This variable is not significant when we include it in our mortality regressions, but as illustrated in Figure 10, it and other health knowledge variables are highly correlated with our treatment indicator. We'd ideally like 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 Indian survey 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.

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.

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 that can be used to check for measurement error. They ask each mother how much time is spent each day fetching 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.[25]

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

[25] Luby et. al.(2004) show that diarrhea incidence can be sharply reduced in a region with poor water supply through hand washing.

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.[26] 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.

Table 2 outlines the WHO approved protocol for treatment of the main diseases that cause child deaths. The first-line treatments listed here are inexpensive and easy to administer.[27] 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

[26] 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.

[27] The cost of oral antibiotics is roughly £0.25 per course. The cost of anti-malarials ranges from £0.25 to £1.50 depending on the type.

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, shown in Table 10, 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 parents' adherence to several of these measures.

7. Implications for child health programs targeting reduced child mortality

In this section, we expand our discussion to calculate the impact of targeted packages of measures aimed at reducing child mortality. 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 whether a large expansion of public health infrastructure is truly needed to reduce child deaths.

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 the 24 interventions recommended by the Bellagio Child Survival Group which they estimate would reduce child mortality by 63%. 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 measures in Table 10 are not rocket science: they 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.

This is an extremely important issue with ramifications for how policy should be designed to reduce mortality. Education could be essential for success for two key reasons: 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).[28]

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. Since the parent is invariably the most important advocate for a child, and is the first one to observe or be able to prevent risk factors, it is natural that they, rather than the public health sector, play the essential role in determining child survival outcomes.

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 Table10, 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

[28] Education will probably also change the distribution of services within the household, especially for girls.

empirical evidence to justify this argument.[29] 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 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 wealth quartile of the population, 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

29 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).

private spending accounts for 71% of total health spending (Adams 2005).[30] 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.

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. Figure 11 suggests a package of education, health knowledge and treatment seeking measures could reduce child mortality by 32%.

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).[31]

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[30] 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.

[31] 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 necessarily need to build out a large public health system in order to achieve major declines in child mortality. In fact, it seems les risky, 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 parents to seek out the best care for their children, 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). Loevinsohn and Harding (2005) discuss promising experiments with contracting out health services to the private sector. It is also important to understand how best to target measures to ensure the extremely poor receive services. Gwatkin, Bhuiya et al. (2004) discusses several experiments with different delivery systems that aim to target services to the poor. Finally, there is a need to better understand what services are unlikely to be provided in the private sector and the role of regulatory bodies.[32]

8. Conclusions

Despite years of effort to reduce child mortality, with sound scientific knowledge of the causes of disease, simple and inexpensive treatments that prevent deaths, and substantial foreign assistance and goodwill, there are several million preventable child deaths each year. Our main goal for this paper was to examine why child mortality varies so much across poor countries, and what can be done to reduce mortality.

The evidence we have presented suggests there is a route to low mortality that may achieve more, in regions where mortality remains high, than "traditional" programs which call for aggressive expansion of the public health sector. In order for these traditional programs to succeed, we need to ensure that sick children are brought to

[32] Our experience in trials to reduce child mortality in several remote regions of India and Africa is that the private sector covers most of the primary, secondary and tertiary services needed for maternal and child healthcare. However, some services, such as blood banks, would need to be coordinated, and there is a role for greater regulation and pricing transparency.

clinics in time, we need to structure the public system to ensure it provides adequate care, we need to raise large amounts of new financing, and we must sustain such healthcare despite volatile public finances, bouts of political instability, and a local elite that is often not interested in helping those communities that suffer from high mortality.

Our evidence suggests an alternative route to low mortality. The biological bond between a parent and child ensures parents are every child's greatest advocate. If we can help the parent, by providing better health knowledge and general education, we can direct that powerful advocacy to tackle the causes of child deaths. If markets respond to parents who seek out appropriate care, a private healthcare sector could arise to provide adequate services. This alternative route to low mortality characterizes the impetus for notable improvement in health system in many low income nations today, and it could prove a highly effective and sustainable path for other countries where child mortality is still too high.

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Appendix 1:
Data glossary and descriptions not included in section 4

Child Deaths, Child Deaths 7+:
See Section 4 for description. Child deaths is a dummy equal to one if the child died by the time of the interview, zero otherwise. Child deaths 7+ is a dummy set to one if the child survived to seven days of age, but died subsequently, and zero if it survived past seven days of age.

Treatment:
See Section 4 for description. The first principal component of a set of variables listed in Table 1.

Morbidity indicators:
See section 4 for description. Each morbidity indicator is the cluster average for the household level data for the following symptoms/diseases:

- Diarrhea: A dummy set to one if a child had diarrhea in the household in the previous two weeks
- Cough with fast breathing: A dummy set to one if a child had cough with fast breathing in the household in the previous two weeks
- Fever: A dummy set to one if a child had fever in the household in the previous two weeks

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:
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, 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:
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 variabl.

Months at Risk:
Our regression equation calls for the months at risk to be included as an explanatory variable. We calculate 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 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 1

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

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

Source: Authors calculations

Table 2

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

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

Source:(World Health Organization 2006)

Table 3

Summary statistics for selected variables

		Child Deaths		Child Deaths 7+		Treatment		Diarrhea		Cough & Fast Breathing		Fever		Improved Water		Improved Sanitation		Mother's years of schooling		Father's years of schooling	
	Pre5>0	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
All countries	244278	0.058	0.200	0.036	0.158	0.000	1.568	0.22	0.17	0.20	0.17	0.37	0.21	0.64	0.48	0.60	0.49	4.5	4.6	5.7	4.9
Africa	121991	0.075	0.224	0.050	0.183	-0.114	1.640	0.24	0.17	0.19	0.16	0.41	0.20	0.61	0.49	0.60	0.49	3.3	4.1	4.4	4.8
Asia	78691	0.046	0.180	0.024	0.132	-0.048	1.580	0.18	0.17	0.18	0.17	0.31	0.20	0.67	0.47	0.54	0.50	5.3	4.9	7.0	4.8
Latin America	43596	0.034	0.155	0.020	0.117	0.371	1.273	0.21	0.18	0.26	0.21	0.35	0.21	0.66	0.47	0.73	0.44	6.1	4.5	6.8	4.6
Bangladesh	5421	0.049	0.188	0.025	0.132	0.187	1.194	0.09	0.08	0.22	0.13	0.45	0.16	0.96	0.19	0.89	0.31	3.9	3.8	4.3	4.5
Brazil	3808	0.032	0.146	0.020	0.116	0.960	1.062	0.17	0.18	0.27	0.22	0.32	0.22	0.92	0.26	0.82	0.38	5.8	3.8	5.4	4.0
Colombia	3656	0.019	0.124	0.008	0.078	0.773	1.079	0.17	0.19	NA	NA	0.30	0.24	0.89	0.31	0.89	0.31	7.3	3.9	7.2	4.1
Egypt	4761	0.035	0.158	0.020	0.118	0.714	0.934	0.27	0.21	0.14	0.16	0.40	0.24	0.82	0.38	0.92	0.27	6.3	5.8	8.3	5.6
Ethiopia	7276	0.083	0.237	0.054	0.193	-1.517	1.632	0.30	0.16	0.28	0.16	0.37	0.18	0.62	0.49	0.20	0.40	1.2	2.8	2.3	3.9
Ghana	2789	0.058	0.205	0.027	0.139	0.394	1.298	0.21	0.17	0.13	0.15	0.27	0.19	0.39	0.49	0.60	0.49	4.1	4.5	6.0	5.7
India	39601	0.056	0.196	0.030	0.143	-0.455	1.751	0.21	0.17	0.21	0.17	0.32	0.19	0.77	0.42	0.40	0.49	3.9	4.7	6.4	5.0
Indonesia	15542	0.032	0.159	0.018	0.120	0.132	1.460	0.11	0.13	0.09	0.13	0.28	0.21	0.39	0.49	0.57	0.49	7.9	3.9	8.4	4.1
Kenya	3996	0.060	0.205	0.036	0.156	0.241	1.376	0.21	0.17	0.23	0.16	0.49	0.21	0.38	0.49	0.75	0.43	6.6	4.2	7.6	4.6
Morocco	4813	0.032	0.152	0.016	0.107	0.825	0.980	0.14	0.13	0.14	0.14	0.30	0.19	0.73	0.44	0.75	0.43	2.6	4.3	4.0	5.0
Mozambique	7035	0.087	0.244	0.063	0.209	0.307	1.526	0.18	0.13	0.13	0.13	0.33	0.19	0.62	0.48	0.55	0.50	2.5	2.8	4.1	3.3
Nepal	4766	0.053	0.185	0.028	0.135	-0.040	1.275	0.25	0.13	0.28	0.18	0.38	0.15	0.38	0.48	0.25	0.43	1.5	3.0	4.5	4.2
Nigeria	3791	0.087	0.232	0.055	0.184	-1.228	1.650	0.24	0.19	0.14	0.14	0.40	0.20	0.49	0.50	0.71	0.45	4.2	4.8	5.8	5.6
Peru	10604	0.030	0.150	0.017	0.112	0.495	1.153	0.20	0.17	0.22	0.18	0.31	0.19	0.70	0.46	0.66	0.47	7.4	4.4	8.9	4.0
Phillippines	4962	0.026	0.138	0.013	0.098	0.512	1.283	0.14	0.16	0.13	0.15	0.30	0.21	0.55	0.50	0.83	0.37	9.0	3.9	8.7	4.1
South Africa	4187	0.043	0.191	0.031	0.162	0.631	1.147	0.16	0.18	0.21	0.21	NA	NA	0.78	0.41	0.83	0.38	8.3	3.8	7.6	4.4
Tanzania	2137	0.089	0.244	0.060	0.200	0.760	1.034	0.17	0.12	0.20	0.13	0.45	0.19	0.79	0.40	0.80	0.40	5.1	3.7	NA	NA
Turkey	2695	0.032	0.147	0.015	0.098	0.156	1.404	0.36	0.25	NA	NA	NA	NA	0.69	0.46	0.97	0.17	5.0	3.7	7.0	3.9
Uganda	4275	0.072	0.210	0.049	0.173	0.056	1.363	0.27	0.16	0.28	0.16	0.53	0.23	0.38	0.48	0.83	0.38	4.4	3.8	6.6	4.2
Vietnam	1906	0.011	0.093	0.005	0.060	0.590	1.302	0.11	0.14	0.20	0.18	0.28	0.21	0.74	0.44	0.78	0.41	7.1	3.8	7.8	3.7

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+*)

	Bangladesh		Brazil		India		Indonesia	
	I	**II**	**I**	**II**	**I**	**II**	**I**	**II**
Treatment	-0.156***	-0.157***	-0.201***	-0.198***	-0.031	-0.030	-0.182***	-0.190***
	(0.036)	(0.036)	(0.062)	(0.062)	(0.022)	(0.022)	(0.041)	(0.042)
Diarrhea	0.620		0.301		0.363**		-0.134	
	(0.828)		(0.645)		(0.178)		(0.663)	
Cough &	-0.455		-0.624		0.051		1.338**	
Fast Breathing	(0.582)		(0.599)		(0.190)		(0.578)	
Fever	1.035*		-0.315		0.141		-0.591	
	(0.537)		(0.545)		(0.176)		(0.450)	
		-0.393		0.233		0.039		0.052
Improved Water		(0.322)		(0.378)		(0.104)		(0.149)
		-0.094		-0.196		-0.029		-0.045
Improved Sanitation		(0.190)		(0.286)		(0.081)		(0.147)
	-0.074**	-0.071**	-0.089**	-0.090**	-0.043***	-0.043***	-0.047**	-0.045**
Mother's education	(0.029)	(0.029)	(0.037)	(0.038)	(0.010)	(0.010)	(0.025)	(0.025)
	0.000	-0.001	-0.158***	-0.158***	-0.018***	-0.019***	-0.060**	-0.061**
Father's Education	(0.023)	(0.023)	(0.051)	(0.051)	(0.007)	(0.007)	(0.025)	(0.026)
Urban	0.190	0.194	0.270	0.293	0.038	0.050	-0.549***	-0.539***
	(0.161)	(0.162)	(0.280)	(0.289)	(0.079)	(0.082)	(0.193)	(0.186)
	0.015***	0.015***	0.020***	0.020***	0.037***	0.037***	0.014***	0.014***
Months at risk	(0.004)	(0.004)	(0.006)	(0.005)	(0.001)	(0.001)	(0.005)	(0.005)
Wealth	-0.056	-0.058	-0.037	0.000	-0.108***	-0.106***	-0.032	-0.034
	(0.041)	(0.041)	(0.065)	(0.062)	(0.017)	(0.018)	(0.031)	(0.031)
Seasonal dummies	Yes		Yes		Yes		Yes	
State/Region dummies	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Diagnostic statistics:								
P-value for joint test of Significance of Morbidity/water& sanitation indicators	0.182	0.434	0.510	0.699	0.048	0.877	0.113	0.900
N	6753	6746	3504	3495	41508	41504	17596	17537
Probability > F	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000
F	7.76	9.14	4.93	5.64	31.47	34.77	7.47	7.14

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
Dependent Variable: Child Deaths 7+

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

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+

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

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

	Dependent variable:				
	Treatment (OLS regression, original data, household level)			Child Deaths 7+ (Logit regression, matched data, child outcomes)	
	I	II	III	IV	V
Treatment				-0.112***	-0.107***
				0.006	0.008
Treatment* (Share of child health services sought in the private sector)					-0.022
					0.020
Share of child health services sought in the private sector			0.069		0.019
			0.064		0.044
Mother's years of schooling	0.062***	0.068***	0.068***	-0.031***	-0.031***
	0.008	0.010	0.010	0.004	0.004
Father's years of schooling	0.030***	0.029***	0.029***	-0.020***	-0.020***
	0.005	0.006	0.006	0.003	0.003
Urban	0.284***	0.209***	0.208***	-0.026	-0.026
	0.052	0.038	0.040	0.027	0.027
Distance to nearest public health center		-0.214***	-0.208***		
		0.069	0.068		
Diarrhea				0.308***	0.300***
				0.061	0.061
Cough & Fast Breathing				-0.034	-0.030
				0.066	0.066
Fever				0.144**	0.124**
				0.055	0.055
Number of children in household at start of observation period	-0.014	-0.020	-0.020		
	0.011	0.016	0.016		
Months at risk				0.024***	0.024***
				0.000	0.000
Wealth indicators for each country	Yes	Yes	Yes	Yes	Yes
	Yes	Yes	Yes	Yes	Yes
Seasonal dummies by country				Yes	Yes
Country dummies	Yes	Yes	Yes	Yes	Yes
Number of observations	191453	127036	123721	278601	273937
Prob > F	0.0000	0.0000	0.0000	0.0000	0.0000

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

Dataset	**Indian Villages**		**Global matched**	
Dependent variable:	**Child Deaths 7+**		**Child Deaths 7+**	
Instrument	**Access to surface water for agriculture**		**Time needed to get drinking water**	
	OLS	**IV**	**OLS**	**IV**
Treatment	-0.0056	-0.0051	-0.0057***	-0.0057***
	(0.0037)	(0.0040)	(0.0007)	(0.0007)
Improved Water	0.0241	0.0727	0.0000	-0.0049
	(0.0172)	(0.1623)	(0.0009)	(0.0071)
Improved sanitation	0.0171	0.0170	-0.0010	-0.0005
	(0.0161)	(0.0163)	(0.0014)	(0.0011)
Mother's years of schooling	-0.0034	-0.0033	-0.0004***	-0.0004***
	(0.0023)	(0.0023)	(0.0001)	(0.0001)
Father's years of schooling	-0.0010	-0.0008	-0.0007***	-0.0007***
	(0.0015)	(0.0016)	(0.0001)	(0.0001)
Months at Risk	0.0008	0.0007	0.0013***	0.0013***
	(0.0006)	(0.0007)	(0.0002)	(0.0002)
Wealth	-0.0036	-0.0042	Yes	Yes
	(0.0027)	(0.0032)	Yes	Yes
Seasonal dummies	No	No	No	No
Country dummies	Yes	Yes	Yes	Yes
Number of observations	508	508	270929	270929
F	2.5900	2.4900	.	
R-squared	0.14	0.13	0.03	0.03

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)

	Ranking of household according to global Treatment indicator		
	Bottom 10%	**Middle 10%**	**Top 10%**
For a child with the following symptoms of illness during the last two weeks, did you seek out any treatment?			
Diarrhea	32	49	53
Cough or fever	36	65	67
The percentage of mothers that followed WHO recommended practices related to breast feeding:			
Breastfed child within one hour after birth	40	60	59
Breastfed child for first six months	98	99	88
Percentage of households where the youngest son aged 10 months or over has been vaccinated for measles	6	89	92

Table 10

The 24 Intervention Measures proposed by the Bellagio Child Survival Study Group

Exclusive breastfeeding (0-6 months)	Counseling to give only breastmilk to children from birth to 6 months of age
Breastfeeding (6-11 months)	Counseling to continue breastfeeding, on demand if possible, from 6 to 11 months of age
Complementary feeding	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.
Zinc	Four rounds of zinc supplements given to caretaker when child is between 2 and 23 months of age
Vitamin A	One dose delivered six months from 7 to 59 months; 9 doses total
Measles vaccine	One dose at 9 months.
Hib vaccine	Three doses within 1st year, delivered with DPT
Tetanus toxoid	Two doses during pregnancy
Clean delivery (skilled attendant at birth)	"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
Newborn temperature management	Thermal protection for all newborns and provision of extra care for low birthweight babies, including "kangaroo mother care" which entails nursing the stable, low birthweight baby skin-to-skin and tied to the mother's front
Antibiotics for preterm premature rupture of membranes (PPROM)	Oral erythromycin 250 mg 8 hourly x 7 days for PPROM before delivery
Antenatal Steroids	Two injections of betamethasone after onset of premature labour
Nevirapine and replacement feeding	Anti-retroviral drugs for the mother (Nevirapine) and breastmilk substitute (formula) for 12 months.
Insecticide-treated materials	One impregnated net every three years for each HH with ≥ 1 child under five
Antimalarial intermittent preventive treatment in pregnancy	Minimum 3 doses of sulfadoxine-pyrimethamine (SP, or Fansidar) within the 2nd & 3rd trimesters of each pregnancy. Dose = 3 tablets of 500 S +25 mg P
Water, sanitation, hygiene	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
Zinc for diarrhea	Zinc suspension or dispersable tablet for 10 days (20mg)
Vitamin A for tx of measles	Vitamin A (200,000 units)
Oral rehydration therapy	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
Antibiotics for dysentery	Ciprofloxacin (150mg/day for 3 days) + zinc suspension or dispersable table for 10 days + ORT (3 sachets)
Antibiotics for pneumonia	Amoxicillin (500mg/day for 3 days)
Antibiotics for sepsis	Tx course of injectable gentimicin and injectable ampicillin for 7 days
Newborn resuscitation	Resuscitation of newborn who is not breathing using bag and mask
Antimalarials	Effective antimalarial

Figure 1: Child mortality and GDP per capita by country, 2003

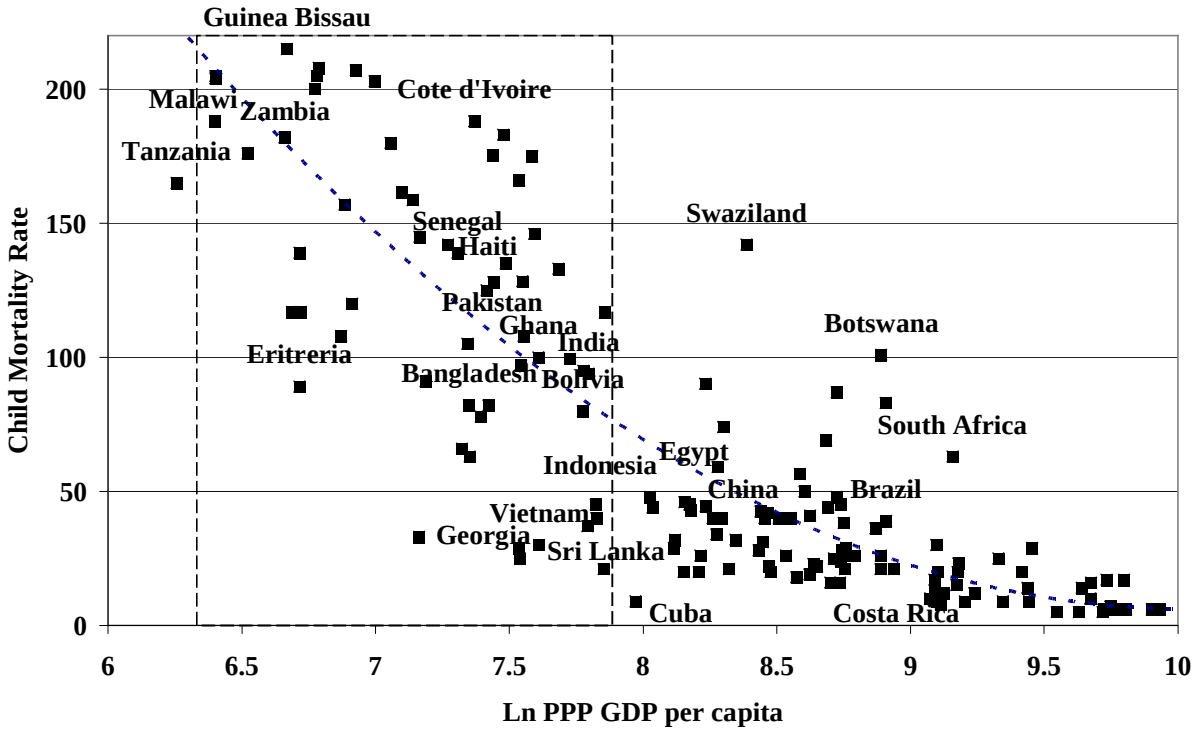
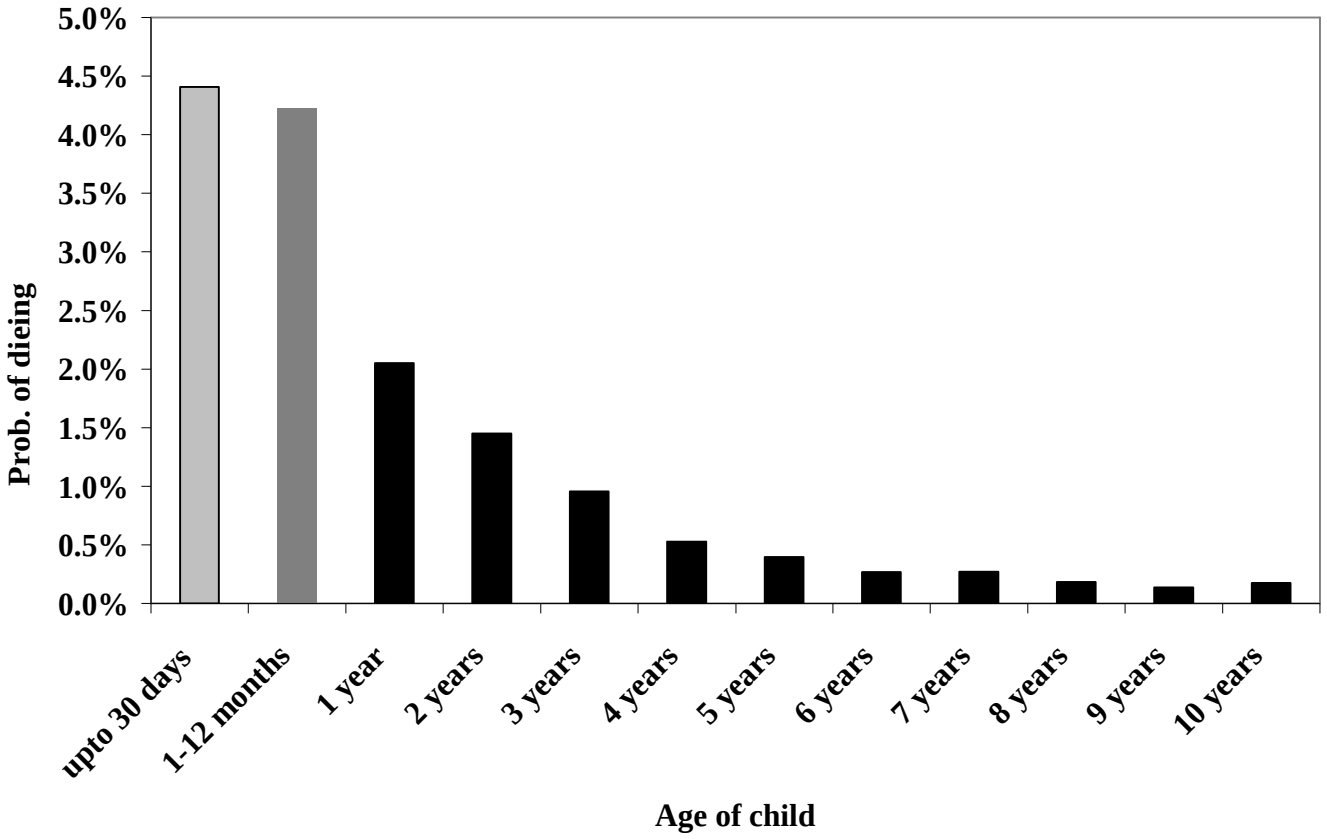


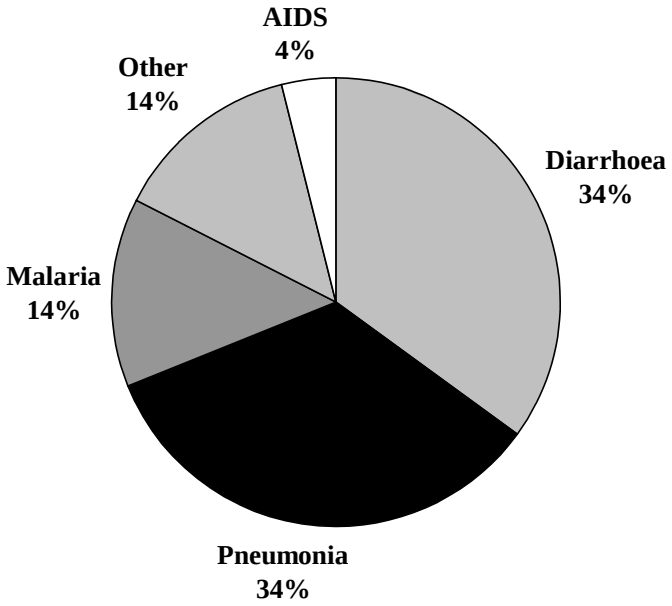
Figure 2: The probability of a child dying at different ages



Source: calculated from Demographic Health Surveys in 45 countries

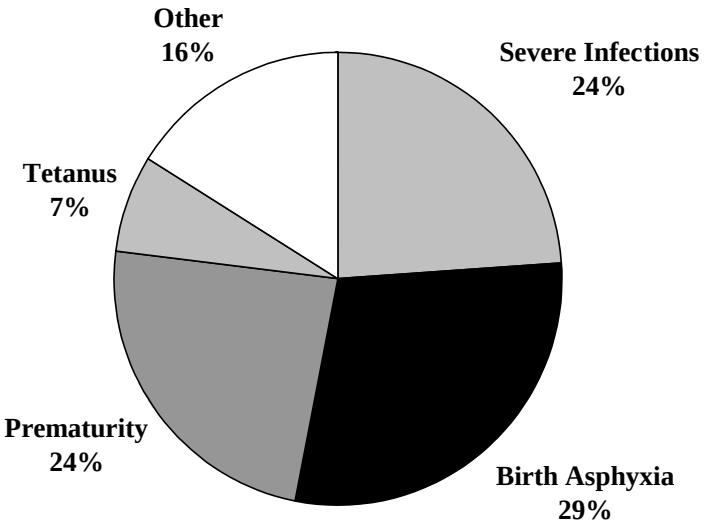
Figure 3: Estimated causes of neonatal and post-neonatal child deaths(2003)

(a) Non-neonatal Child Deaths:



Source: Black et. al. (2003)

(b) Neonatal Deaths



Source: Black et. al. (2003)

Figure 4: Average morbidity prevalence

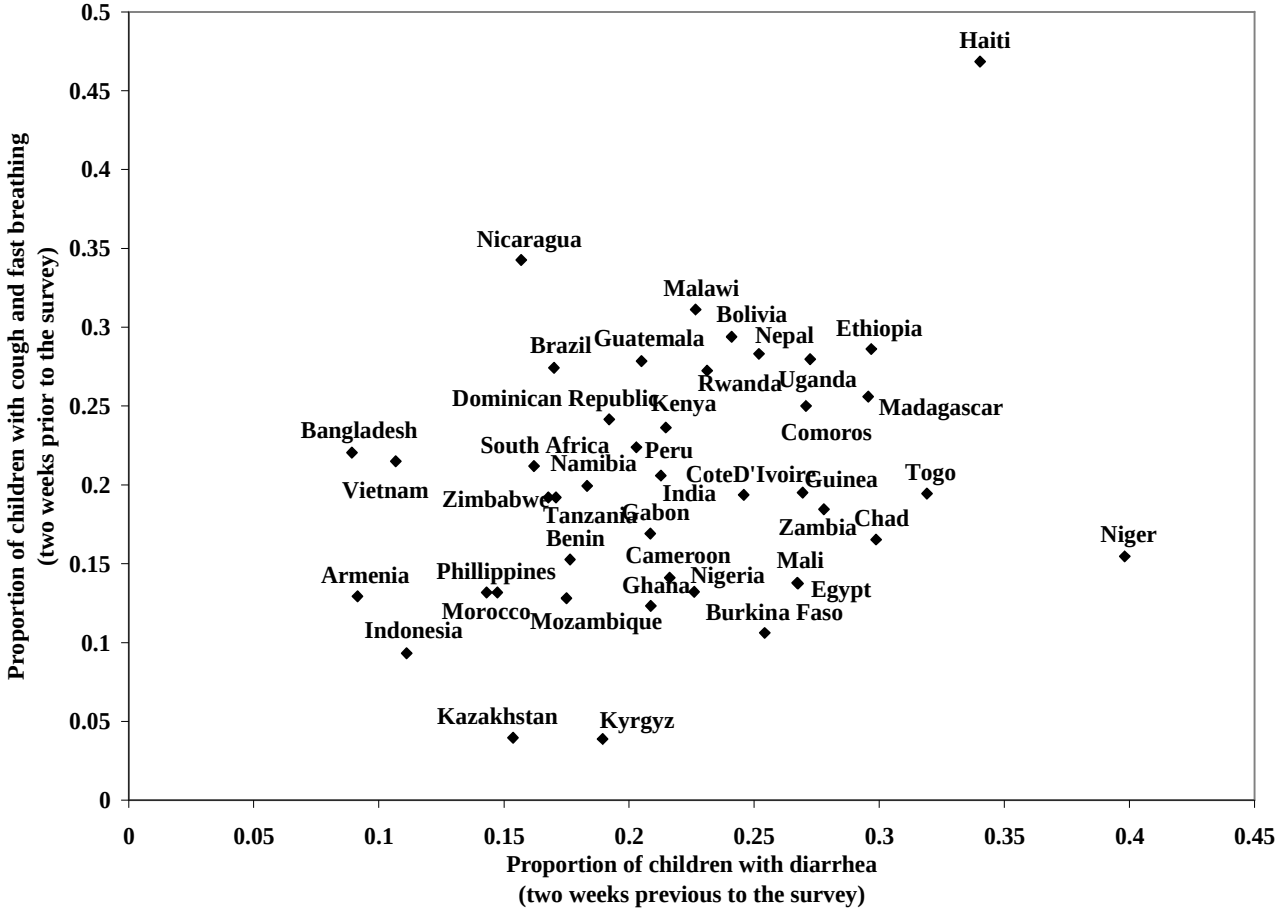
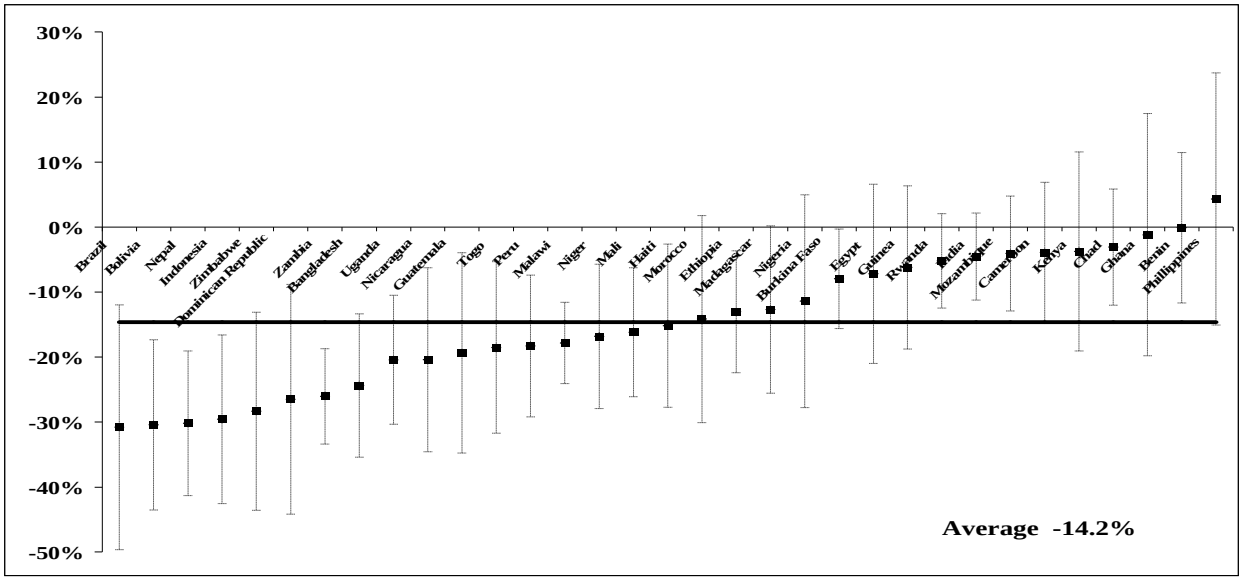
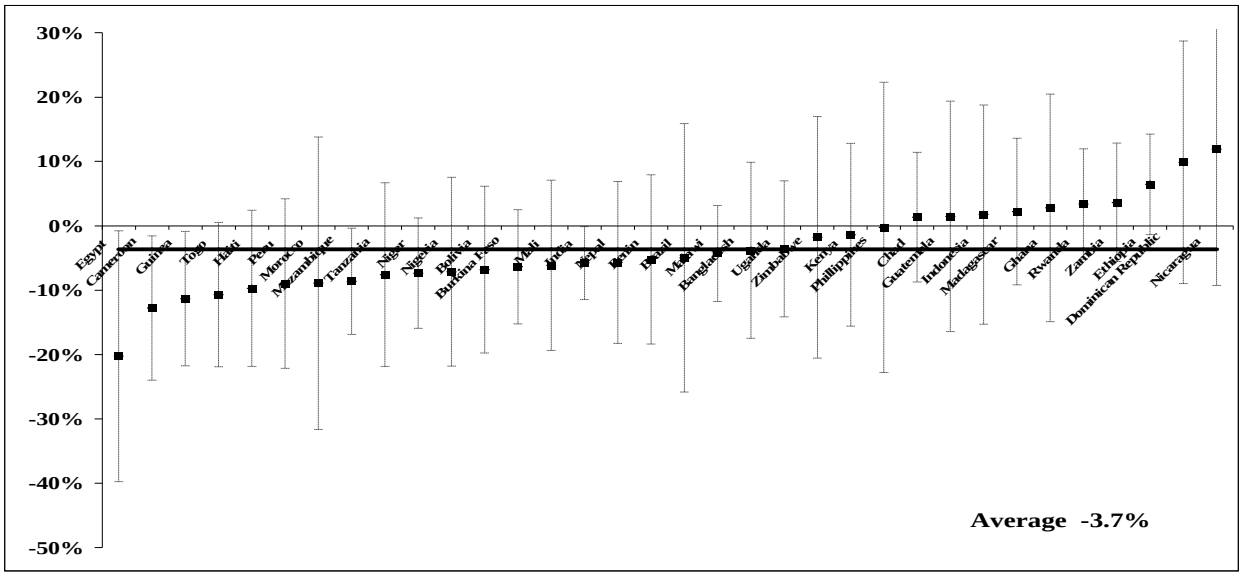


Figure 5a: The impact of a one standard deviation rise in treatment on the probability of Child Deaths 7+ in regressions with morbidity indicators



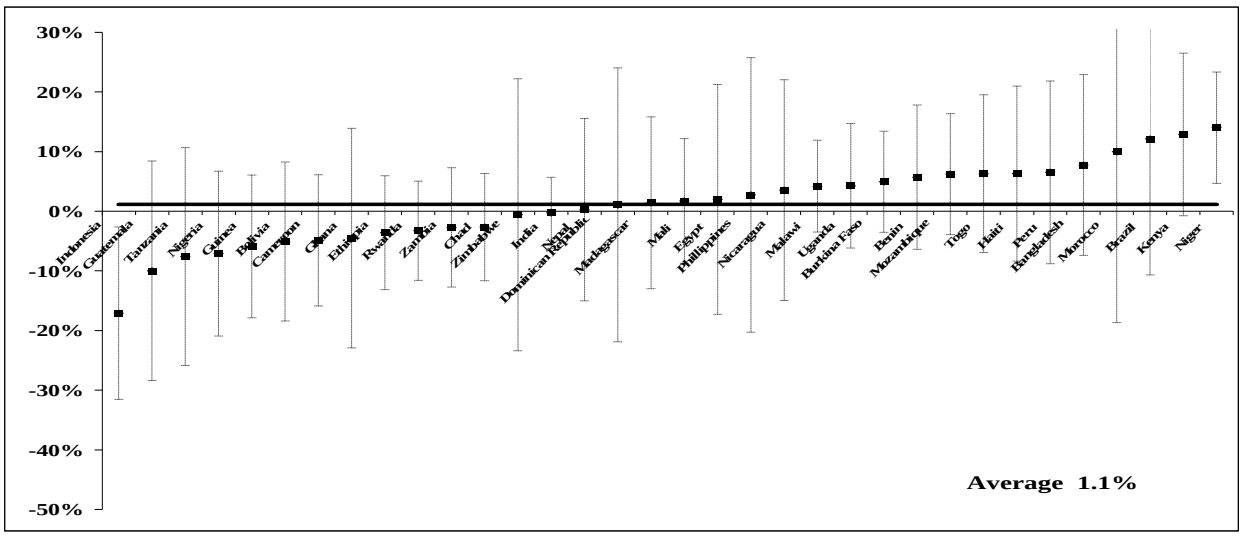
Note: Dotted lines show +/- 2 standard deviation bands around point estimates.

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



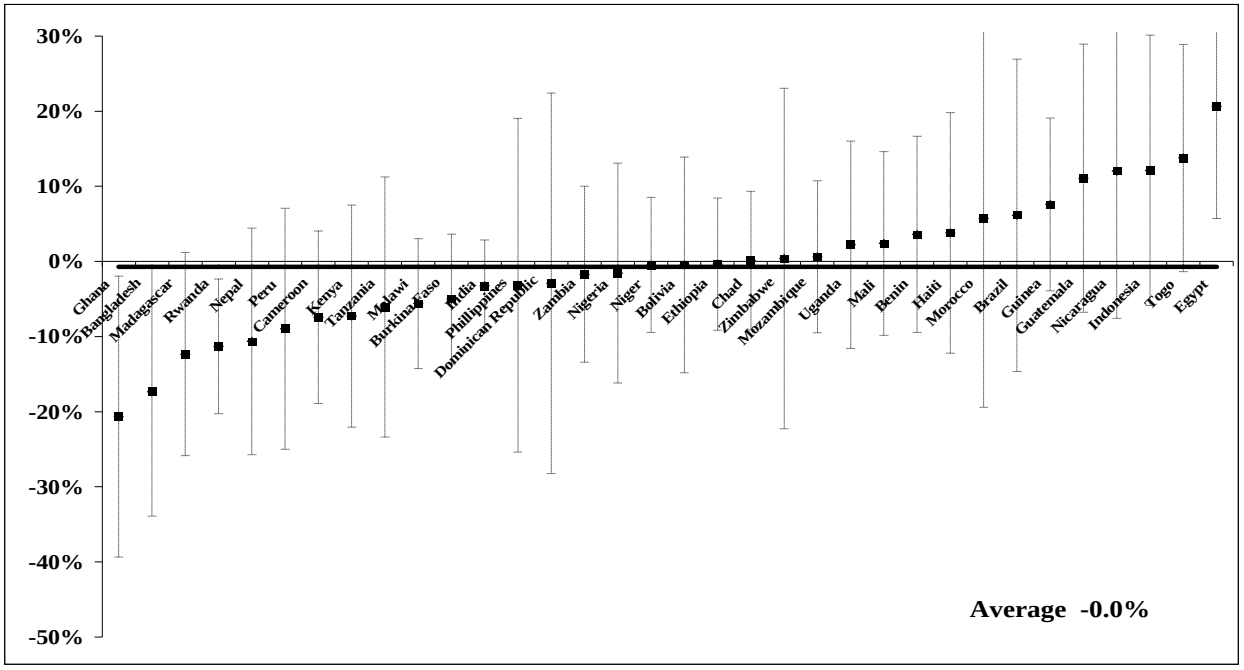
Note: Dotted lines show +/- 2 standard deviation bands around point estimates.

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+.



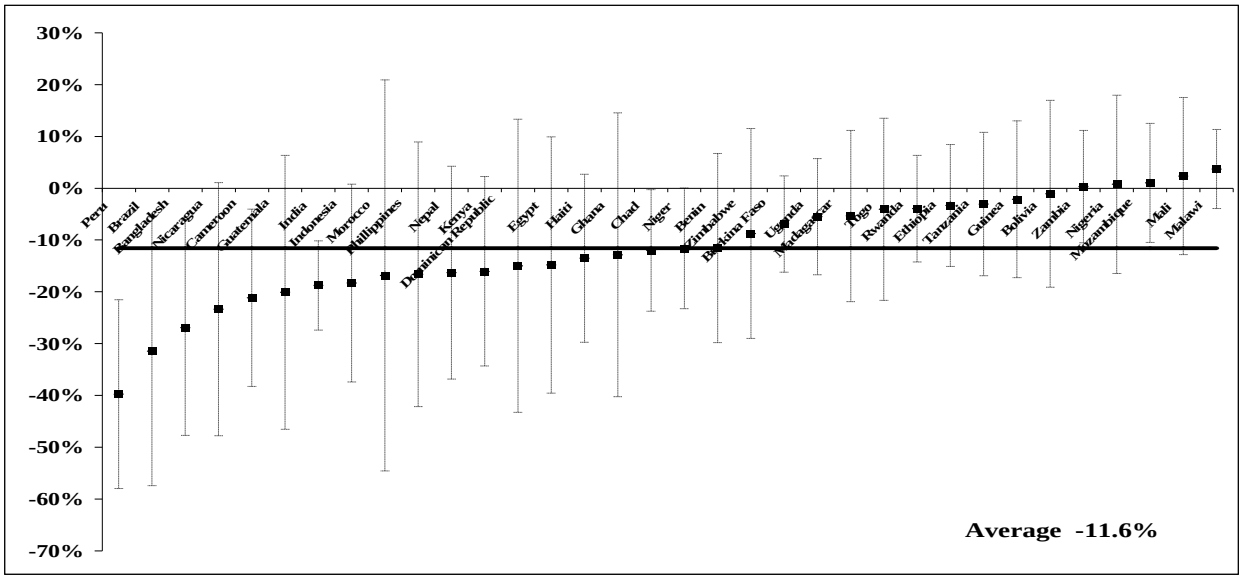
Note: Dotted lines show +/- 2 standard deviation bands around point estimates.

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



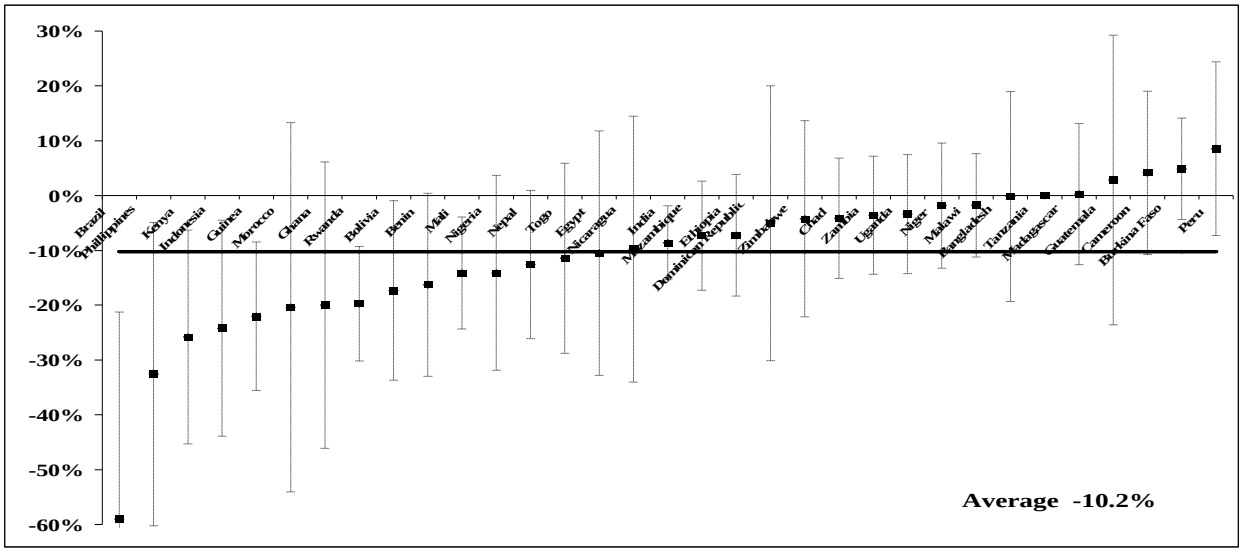
Note: Dotted lines show +/- 2 standard deviation bands around point estimates.

Figure 5e: The impact of a one standard deviation rise in years of mothers' education on the probability of Child Deaths 7+



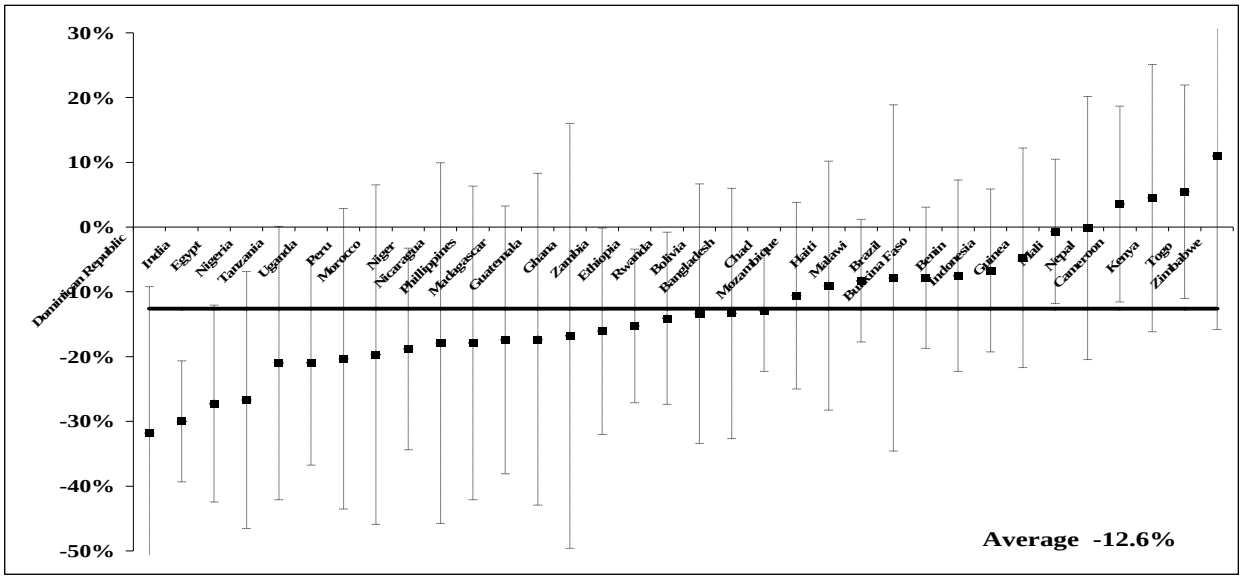
Note: Dotted lines show +/- 2 standard deviation bands around point estimates.

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



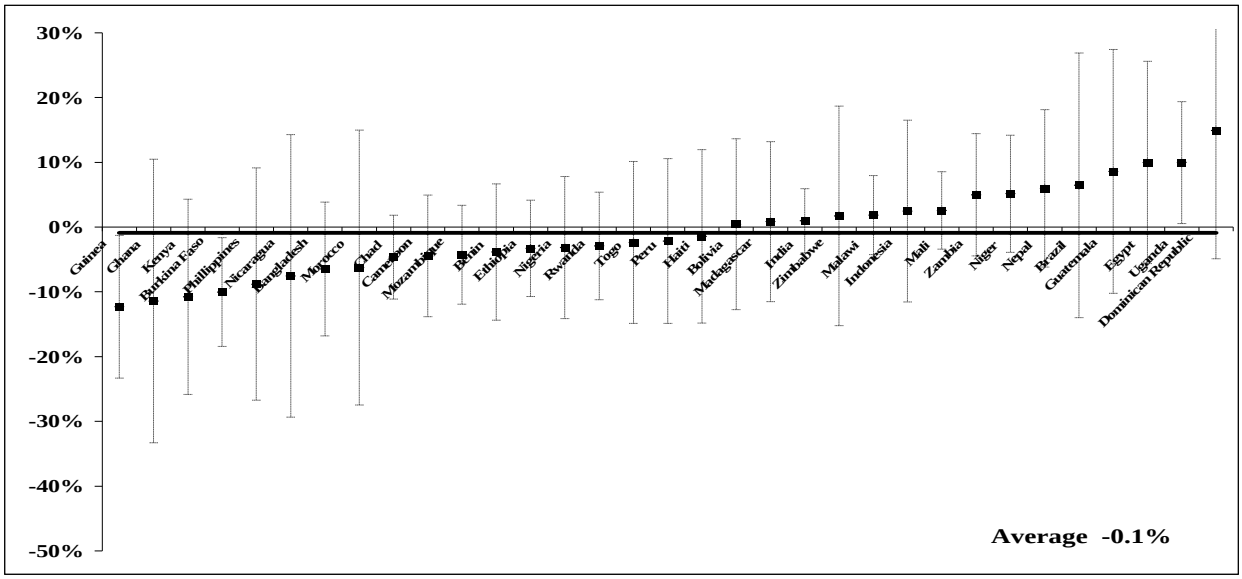
Note: Dotted lines show +/- 2 standard deviation bands around point estimates

Figure 5g: The impact of a one standard deviation rise in wealth on the probability of Child Deaths 7+.



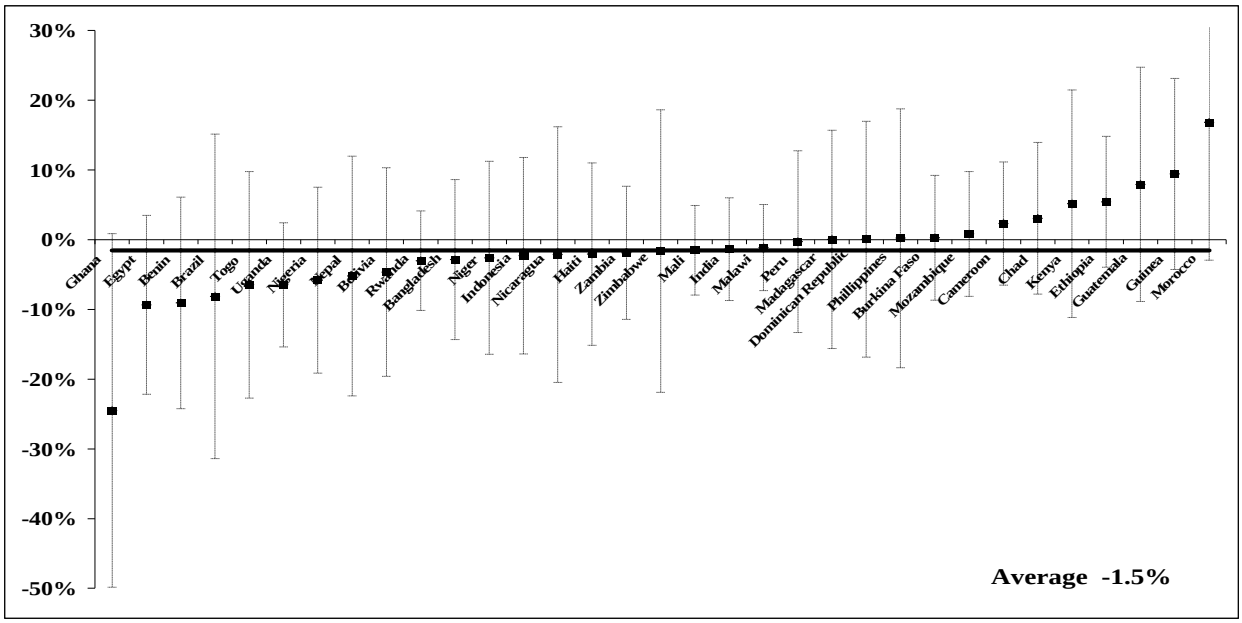
Note: Dotted lines show +/- 2 standard deviation bands around point estimates

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



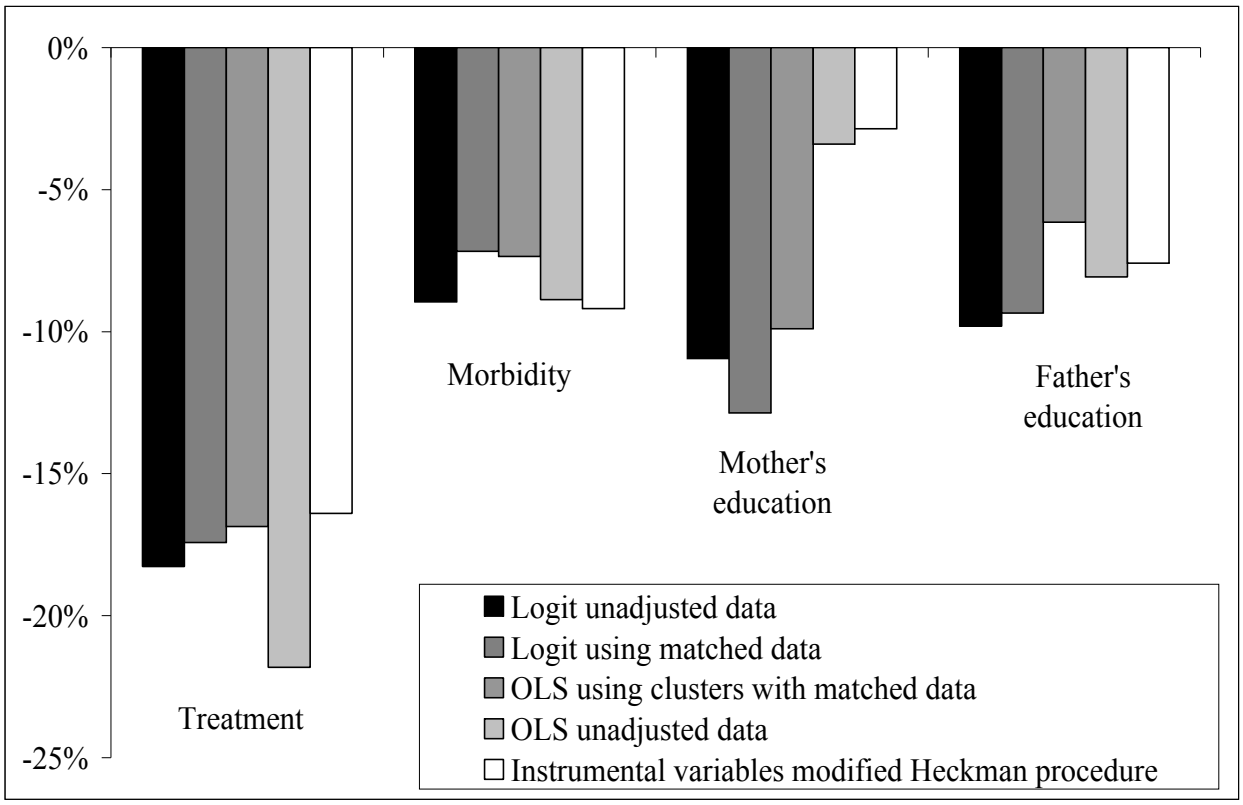
Note: Dotted lines show +/- 2 standard deviation bands around point estimates

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



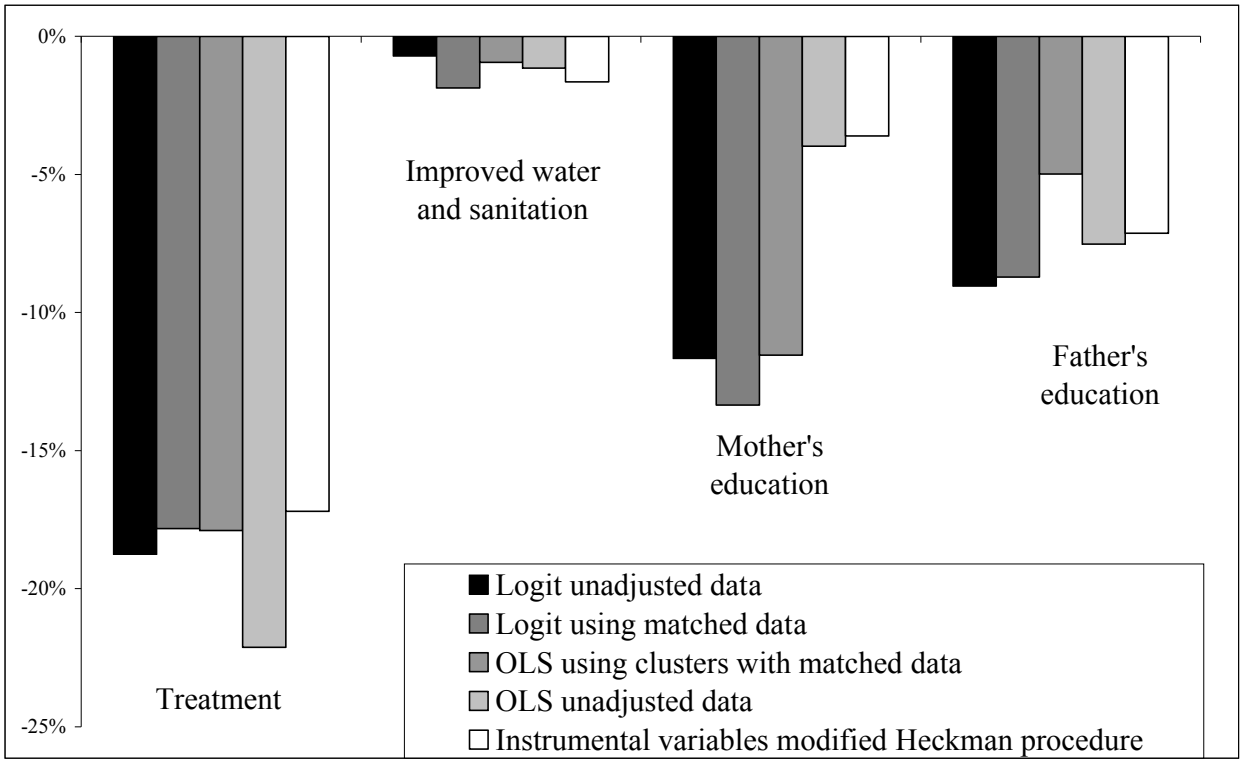
Note: Dotted lines show +/- 2 standard deviation bands around point estim

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 year's 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)

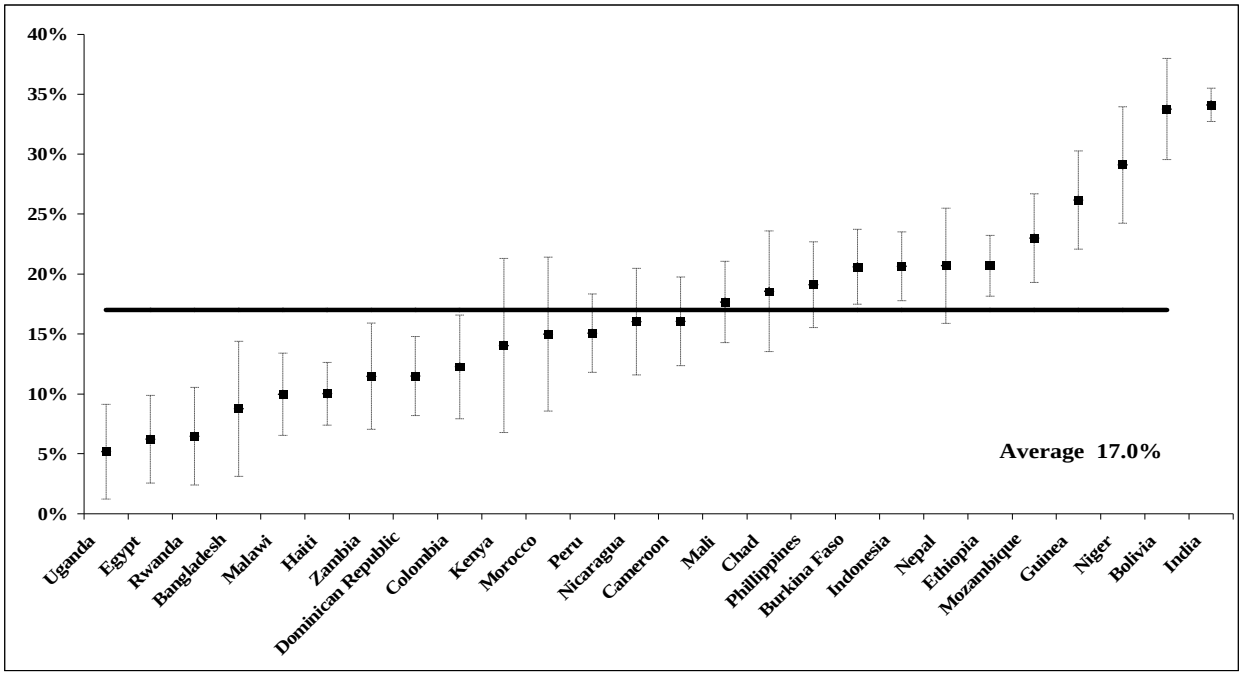
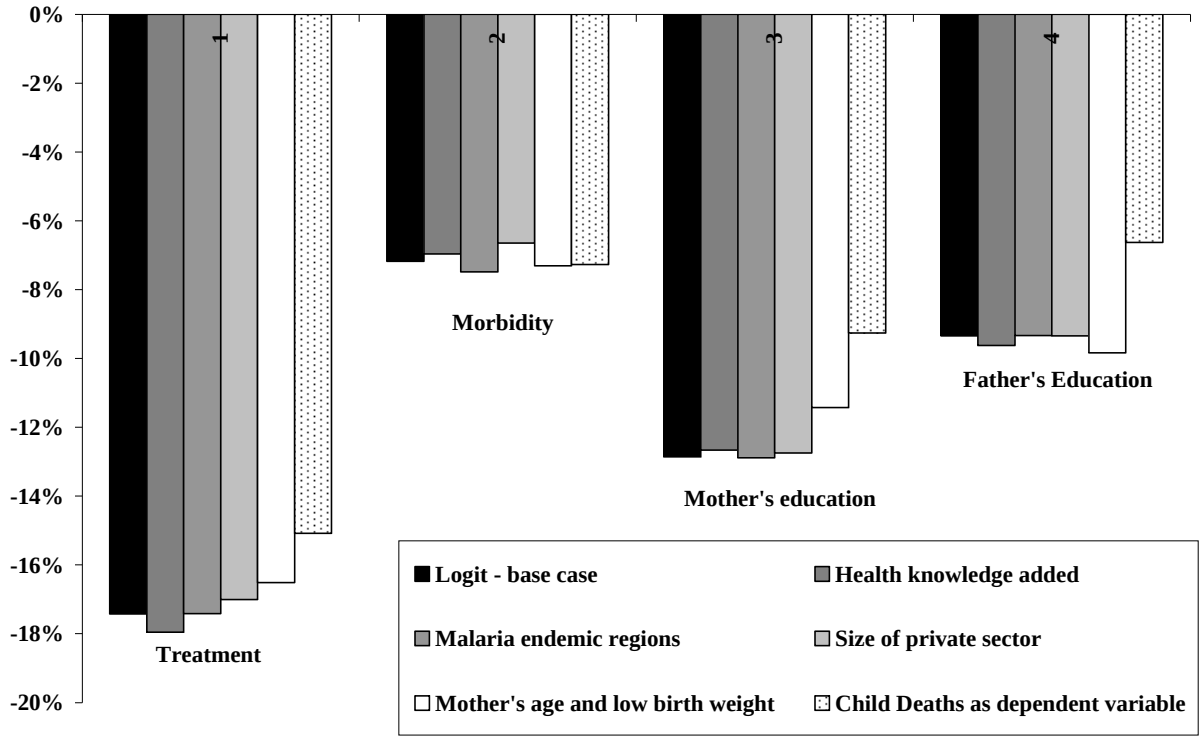


Figure 10: The average impact of a one standard deviation change in explanatory variables on Child Death 7+ under alternative robustness tests in global data



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

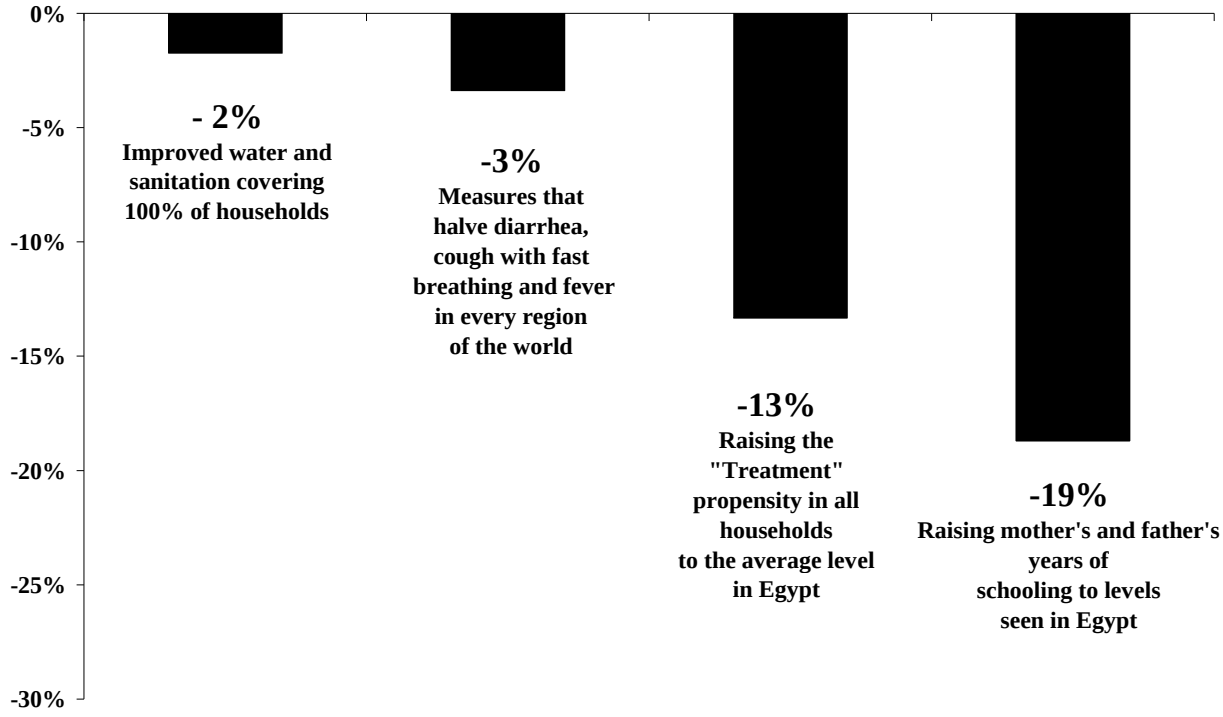
Malaria endemic regions: Interaction term for malaria endemic zones with fever added as control to base case

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

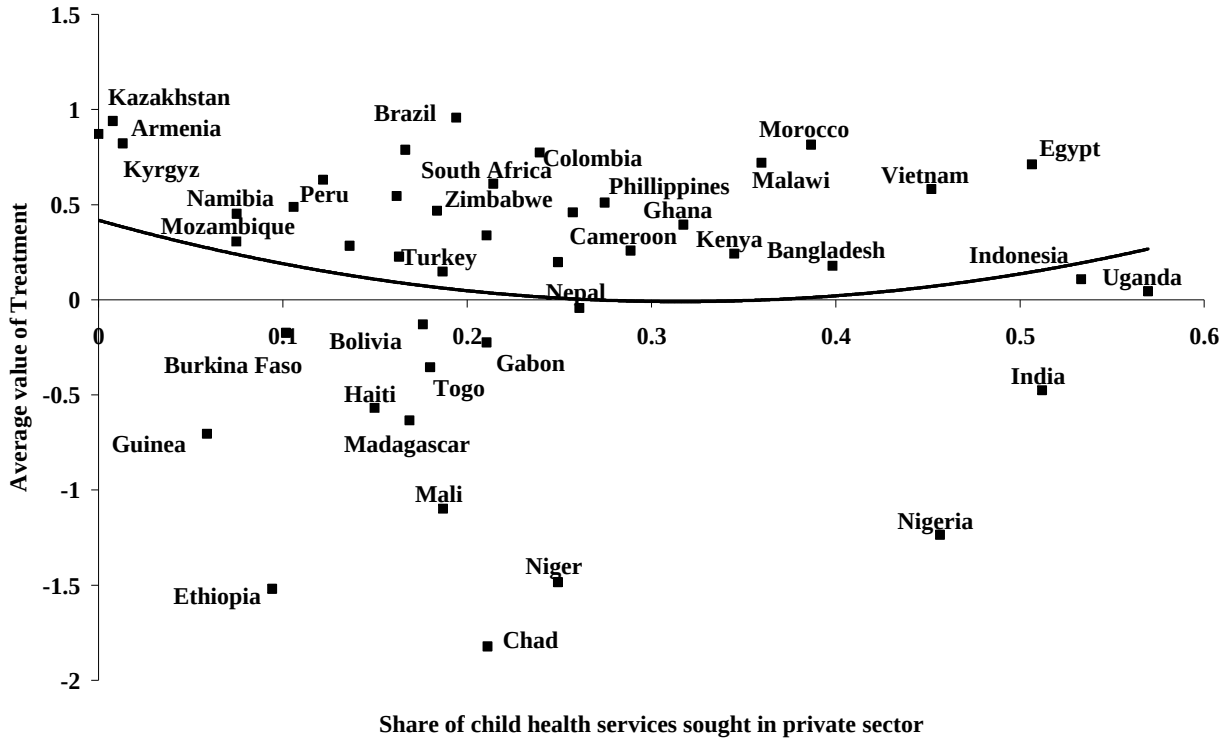


Figure 13: Inequality of treatment and the share of child health services sought in the private sector

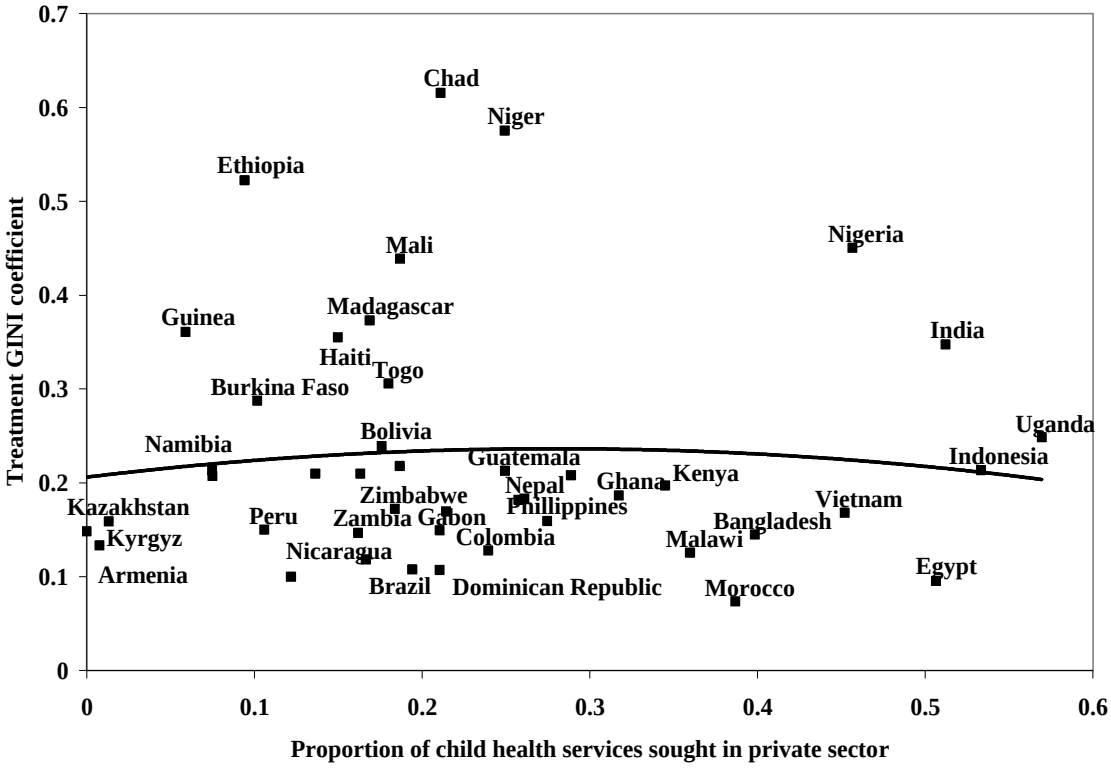


Figure 14: Child Deaths 7+ compared to the share of child health services sought in the private sector

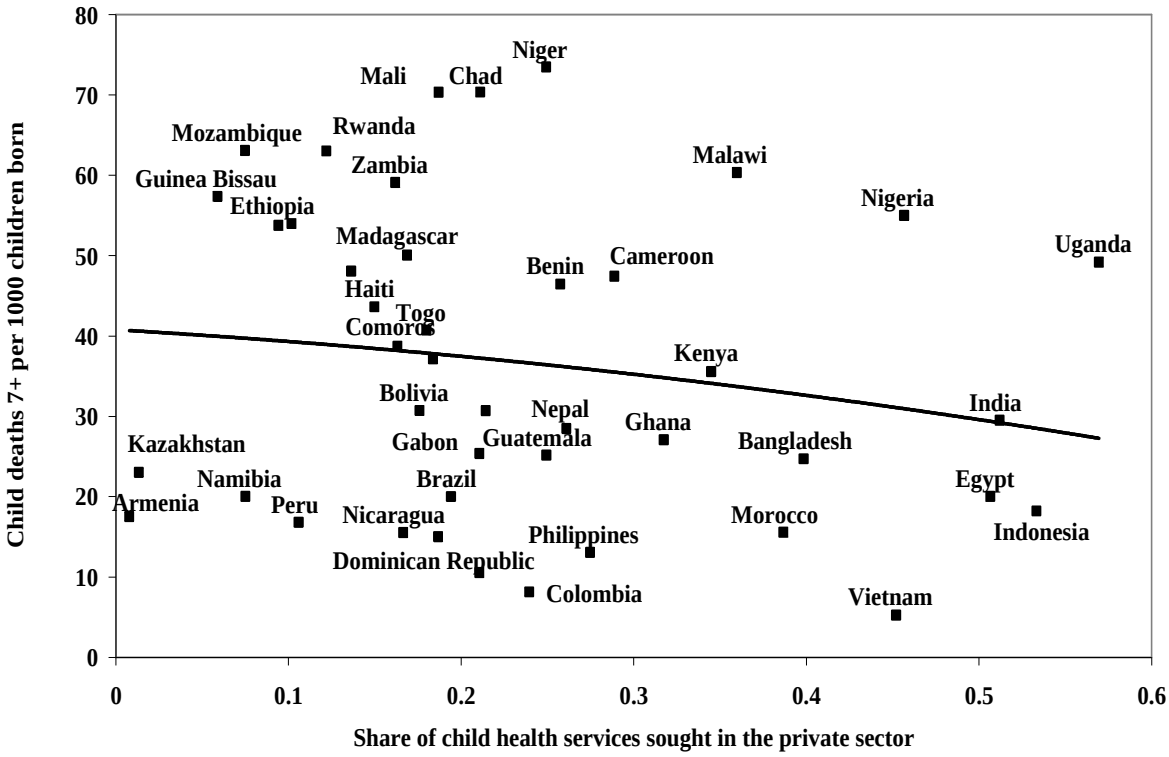
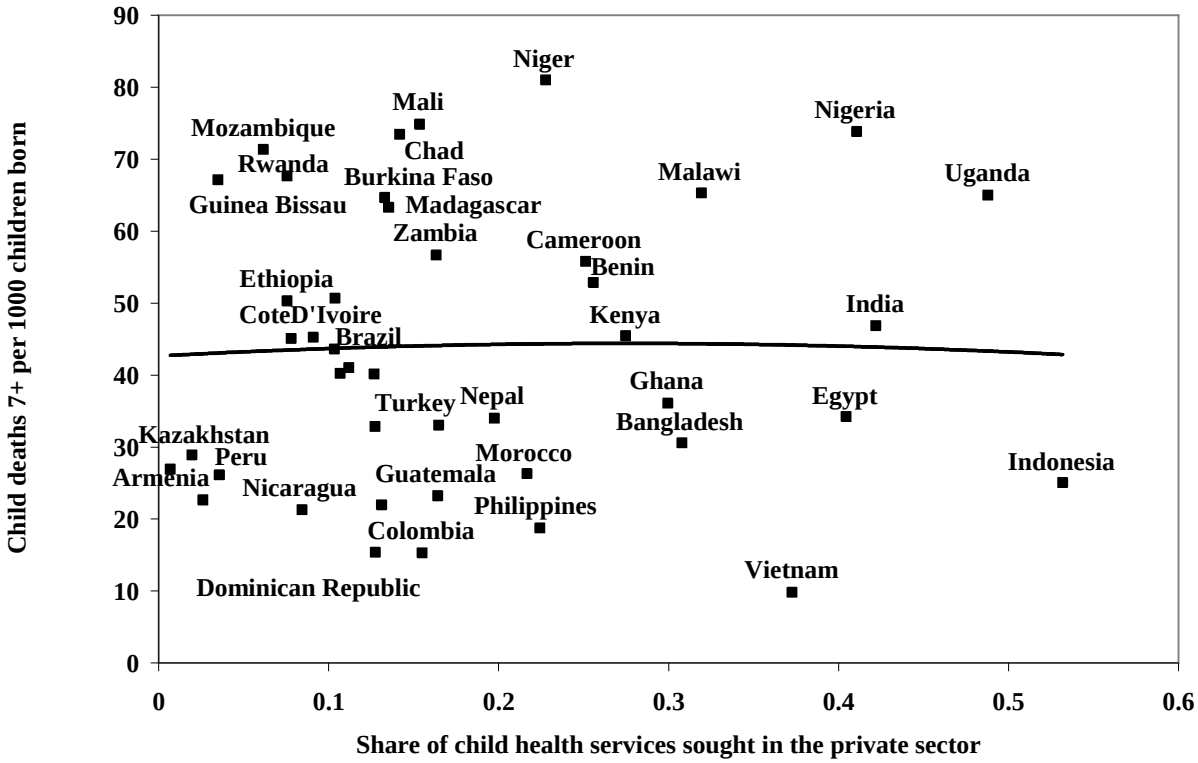


Figure 15: Child Deaths 7+ compared to the share of child health services sought in the private sector (households in the bottom wealth quartile only)



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