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Evaluating a nationwide health intervention using the intention-to-treat framework:

An application to Malawi's ITN distribution program

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Abstract

This paper develops a semi-parametric intention-to-treat estimator for a health intervention when no data for a control group is observed from the same period. This estimator is based on the assumption that the intervention has no direct effect on the outcome and influences outcomes only over its effect on an indicator variable. Conditioning on all relevant confounding variables, the intention-to-treat and the local treatment effect can be estimated. This estimator is used to evaluate Malawi's insecticide-treated net (ITN) distribution program, assuming that the distribution program affects infant mortality only over bednet ownership. It shows that the distribution scheme is associated with an infant mortality reduction of one percentage point, which corresponds to 30% of the total reduction in infant mortality over the study period.

Keywords: Treatment effect; semi-parametric estimation; malaria; health intervention

1 Introduction

Health is widely regarded as one of the key factors affecting sustainable growth in developing countries (Baldacci, et al. 2008, Cole and Neumayer 2006, Bloom and Canning 2003). Promoting good health is therefore seen as central issue in fighting poverty and promoting growth, which is also reflected in the Millennium Goals from which three are directly linked to health (MDG 4, 5, 6). This explains the substantial increase in health related spending in developing countries (Hecht and Shah 2006).

Understanding the efficiency with which these health resources are spent, is a key issue in the health and development economic literature. This paper describes an econometric framework that can be used to evaluate nationwide implemented health interventions and applies this framework to Malawi's insecticide-treated net (ITN) distribution program. This program is an interesting case to study because (1) malaria is one of the major public health problems in Malawi (NSO 2005), and (2) it was the first national ITN program in Sub Saharan Africa and can therefore serve as a model for other countries.

In recent years, a number of economic evaluations of health interventions have been published. Randomized experiments are considered as the gold standard and many pilot or small-scale programs are nowadays evaluated using an experimental design. Recent examples are the evaluation of HIV voluntary counseling and testing in rural Malawi (Thornton 2008), community-based monitoring of health service providers in Uganda (Svensson and Bjorkman 2007), a school-based HIV/AIDS intervention (Duflo, et al. 2006), a bed net distribution program (Cohen and Dupas 2008), and a school-based mass treatment with deworming drugs (Miquel and Kremer 2004) all conducted in Kenya.

Results from experimental settings however, may not be generalizable to everyday settings. The study group may not be representative for the population (because of a randomization bias for example), when scaling-up an intervention, the implementation of the intervention from the experimental setting may not be duplicable to "real" world setting (for example information of the involved personnel may be less intensive), or the experimental setting itself changes behavior of attends (because of the Hawthorne effect or Henry effect for example) (Duflo, Glennerster and M 2006, Heckman and Smith 1995). It is therefore necessary to evaluate scaled-up interventions, even if promising experimental evidence is available.

Large-scale or nationwide health interventions are usually non-experimentally evaluated.[1] Wagstaff (2007) for example studies whether Vietnam's Health Care Fund for the Poor increased health service utilization and reduced the risk of catastrophic spending, exploiting the (non-randomized) step-wise implementation of the health fund. Angel-Urdinola and Jain (2006) analyze whether a subsidized health program increased health service utilization among the poor in Armenia using individuals not eligible for free services as the control group. Lee and colleagues (2006) examine an early childhood development program in the Philippines, using a difference-in-difference approach (comparing regions where the program was implemented with region where the program was not implemented). All these studies

[1] An exception is the evaluation of the PROGESA program in Mexico, which uses a sequential implementation with treatment villages receiving benefits immediately, while benefits for the control villages were postponed for one year (Gertler 2000)

have in common that data for a control group is available for the pre- and post-intervention periods.

Many health interventions however are already scaled-up and potentially cover the full population. Malawi's ITN program for example is a national bed net distribution scheme that covers all children and pregnant women. A control group of children not covered under the scheme is therefore not available.[2] A simple before-after estimator is unfeasible in many applications because the assumption that the expected outcome in the non-treatment state is the same in the post- and the pre-treatment period is often violated (Heckman, LaLonde and Smith 1999).

In this paper an alternative to the standard before-after estimator is developed. It is based on a similar framework than the nonparametric IV estimator for the local treatment effect with covariates (Frölich 2007). The conditional IV framework requires an instrument for the treatment that has no direct effect on the outcome and influences outcomes only over its effect on the treatment variable. Conditioning on all relevant confounding variables, the intention-to-treat effect of the instrument and the local treatment effect for the compliers can be estimated. The model used in this paper in contrast, assumes that the treatment has no direct effect on the outcome and influences outcomes only over its effect on an indicator variable. If one observes this indicator variable and all relevant confounders, the average treatment effect of the indicator variable, the intention-to-treat effect of the treatment on the outcome, and local average treatment effect for the complier population can be estimated.

Applying this method to data from Malawi's Demographic and Health Surveys from 2000 and 2004, the effectiveness of the national ITN distribution scheme in reducing infant mortality is estimated. The paper finds that the distribution scheme is associated with an infant mortality reduction of one percentage point, which corresponds to almost 30% of the total reduction in yearly infant mortality over the study period.

The rest of the paper is outlined as following: Section 2 develops the empirical model. Section 3 describes Malawi's Roll Back Malaria Initiative. The model is applied to data from Malawi in section 4, and the final section concludes.

2 Empirical model for the intention-to-treat effect

When empirically analyzing to which extend a nationwide health intervention affects health outcomes we seek to estimate an average treatment effect of the following form:

$$E[Y^1 - Y^0|T = 1]$$

with T being an indicator for receiving treatment, i.e. being exposed to the public health intervention, Y^0 being the potential health outcome without the intervention, and Y^1 being the potential health outcome with the intervention. Since every individual is potentially exposed to the health intervention, a natural choice could be the before-after estimator. In its conventional form, the before-after estimator assumes that the pre-program health outcome Y_t^0 proxies the health outcome in the post-program period $Y_{t'}^0$ (Heckman, LaLonde and Smith 1999):

[2] A regional shortage could be possible but is usually not observed in existing data sets.

$$E[Y_t^0 - Y_{t'}^0 | T = 1]$$

Longitudinal data is not necessary as long as individuals from the post-program survey are compared to similar persons from the pre-program survey. However, the assumption that the expected outcome in the non-treatment state is the same in the post- and the pre-treatment period is often violated because of trends in the outcome variable that are not associated with the health intervention (through other health programs or environmental factors for example).

This paper develops an alternative strategy to estimate the effect of a nationwide health intervention. This strategy is based on the assumption that the intervention affects outcome only over its effect on an indicator variable (with $B=b$ if the person obtains B and $B=b'$ if the person does not obtain B)*:*

$$Y^0, Y^1 \amalg T | B, X$$

The indicator variable B is the result of an individual choice. The intervention shifts the probability $P(B=b)$ but does not result in universal coverage:

$$0 < P(B = b) < 1$$

An example is a national ITN distribution scheme, which affects mortality over bed net ownership. Individuals however, voluntarily decide whether or not they obtain a bednet from any of the ITN providers.

Assume that we are able to observe all confounding variables that jointly affect potential outcomes (for example potential mortality with and without bed nets) and the indicator variable (conditional independence assumption, CIA):

$$Y_t^b, Y_t^{b'} \amalg B_t | X_t$$

The expected conditional outcomes can be written as

$$m_t^b(x) = E\left(Y_t^b \middle| X_t, B_t = b'\right) = E\left(Y_t^b \middle| X_t, B_t = b\right) = E(Y_t | X_t, B_t = b)$$
$$m_t^{b'}(x) = E\left(Y_t^{b'} \middle| X_t, B_t = b\right) = E\left(Y_t^b \middle| X_t, B_t = b'\right) = E(Y_t | X_t, B_t = b')$$

The conditional treatment effect is equal to

$$\Delta_t(x) = m_t^b(x) - m_t^{b'}(x)$$

and the average treatment effect of bed net ownership equals

$$ATE_t^B = \int \Delta_t(x) f(x) dx \tag{1}$$

The treatment effect however, is only of limited interest, as this corresponds to a hypothetic situation comparing $B=b$ for all individuals with $B=b'$ for all individuals. In reality however, some people may acquire B without the intervention, while other people do not acquire B with the intervention. To evaluate the health intervention three different groups need to be defined: always-takers with $B=b$ in any state of the world independent from health intervention, never-takers with B$=b'$ in any state of the world, and compliers with $B=b'$ in the state of the world where the intervention had not been implemented, and B=b when the intervention is implemented.[3]

The intervention has no effect on always- and never-takers, because they do not change their behavior following the implementation of the intervention. The intervention is only effective within the complier population ($C=1$) because they changed their behavior as a direct result of the intervention. The effect of the intervention (which is equivalent to the intention-to-treat estimator) is therefore equal to

$$ITT_t^T = P(C_t = 1) \int \Delta_t(x) f(x|C_t = 1) dx$$

From Bayes rule we know that $f(x|C_t = 1) = \frac{P(C_t=1|X_t)f(x)}{P(C_t=1)}$, so that the intention to treat estimator can be formulated as

$$ITT_t^T = \int \Delta_t(x) P(C_t = 1|X_t) f(x) dx$$

The conditional probability to be a complier can be written as the difference between the probability to obtain B if the intervention is active ($T=1$) and the probability to obtain B if the intervention is inactive ($T=0$):

$$P(C_t = 1|X_t) = P(B_t = b|T = 1, X_t) - P(B_t = b|T = 0, X_t)$$

If we assume mean independence of the indicator variable in post-intervention period t and pre-intervention t'

$$P(B_t = b|T = 0, X) = P(B_{t'} = b|T = 0, X)$$

the conditional probability to be a complier can be estimated from the propensity score in the post-intervention period $p_t(x) = P(B_t = b|X_t)$ and the propensity score in the pre-intervention period that is extrapolated to characteristics of the post-intervention period $p_{t',t}(x) = P(B_{t'} = b|X_t)$:

$$P(C_t = 1|X_t) = p_t(x) - p_{t',t}(x)$$

Thus, the intention to treat effect can be reformulated as

$$ITT_t^T = \int \Delta_t(x) \big[p_t(x) - p_{t',t}(x)\big] f(x) dx \tag{2}$$

Once the intention to treat estimator is identified, the local average treatment effect (i.e. the effect of the intervention within the compliers) can be estimated:

$$LATE_t^T = \frac{ITT_t^T}{P(C_t=1)} = \frac{\int \Delta_t(x)\big[p_t(x) - p_{t',t}(x)\big] f(x) dx}{\int p_t(x) - p_{t',t}(x) f(x)} \tag{3}$$

Equation (1) to (3) can now be semi-parametrically estimated using the propensity weighting method. From Bayes rule we know that

$$f(x) = \frac{P(B_t = b) f(x|B_t = b)}{P(B_t = b|X_t)}$$

[3] It is assumed that defiers (i.e. those with B=b in absent of the program and B=b' after implementation) do not exist.

$$f(x) = \frac{P(B_t = b')f(x|B_t = b')}{P(B_t = b'|X_t)}$$

Equation (1) to (3) can therefore be rewritten as[4]

$$ATE_t^B = \mathrm{P}(B_t = b)\, E\left[\frac{Y_t}{p_t(x)}|B_t = b\right] - \mathrm{P}(B_t = b')\, E\left[\frac{Y_t}{1-p_t(x)}|B_t = b'\right] \quad (1a)$$

$$ITT_t^T = \mathrm{P}(B_t = b)\, E\left[Y_t\frac{p_t(x)-p_{t',t}(x)}{p_t(x)}|B_t = b\right] - \mathrm{P}(B_t = b')\, E\left[Y_t\frac{p_t(x)-p_{t',t}(x)}{1-p_t(x)}|B_t = b'\right] \quad (2a)$$

$$LATE_t^T = \frac{\mathrm{P}(B_t=b)E\left[Y_t\frac{p_t(x)-p_{t',t}(x)}{p_t(x)}|B_t=b\right]-\mathrm{P}(B_t=b')E\left[Y_t\frac{p_t(x)-p_{t',t}(x)}{1-p_t(x)}|B_t=b'\right]}{P(C_t=1)} \quad (3a)$$

Equation (1a) to (3a) can be estimated by its samples analogues:

$$\widehat{ATE_t^B} = \frac{1}{N_t}\sum\frac{Y_tB_t}{\widehat{p_{t,\iota}}} - \frac{Y_t(1-B_t)}{1-\widehat{p_{t,\iota}}} \quad (1b)$$

$$\widehat{ITT_t^T} = \frac{1}{N_t}\sum\frac{Y_tB_t\left(\widehat{p_{t,\iota}}-\widehat{p_{t',t,\iota}}\right)}{\widehat{p_{t,\iota}}} - \frac{Y_t(1-B_t)\left(\widehat{p_{t,\iota}}-\widehat{p_{t',t,\iota}}\right)}{1-\widehat{p_{t,\iota}}} \quad (2b)$$

$$\widehat{LATE_t^T} = \frac{\widehat{ITT_t^T}}{P(\widehat{C_t=1})} \quad (3b)$$

with $\widehat{p_{t,\iota}}$ being the estimated propensity score of B=b in the post-intervention period $\widehat{p_{t',t,\iota}}$ being the estimated propensity score of B=b in the pre-intervention period extrapolated to data from the post-intervention period, and $P(\widehat{C_t = 1}) = \sum\widehat{p_{t,\iota}} - \widehat{p_{t',t,\iota}}$.

3 The Roll Back Malaria Initiative

Malaria is a vector-born infection caused by protozoan parasites. It is widespread in tropical and sub-tropical regions; estimated 3.3 billion people worldwide are at risk. In 2006, Malaria has caused estimated 247 million cases and nearly one million deaths, mostly in children under 5 years. From the 109 countries with a malaria epidemic, 45 are in Sub Saharan Africa (WHO 2008).

People usually get infected with malaria by a bite from the female Anopheles mosquito.[5] There is currently no vaccine against malaria, but effective prophylactic drug treatments (even though not 100% effective) are available. Other possibilities to prevent malaria are the usage of (insecticide-treated) nets and indoor residual spraying. Most adults in endemic areas are (partially) immune against an infection. Pregnancy however, reduces a woman's immunity to

[4] Note that
$$\begin{aligned} E[Y_tw_b(x)|B_t = b] &= E[E[Y_tw_b(x)|B_t = b, X]|B_t = b] \\ &= E[w_b(x)E[Y|B_t = b, X]|B_t = b] \\ &= E[m_t^b(x)(x)w_b(x)|B_t = b] \\ &= \int m_t^b(x)w_b(x)f(x|B_t = b)dx \end{aligned}$$

[5] Transmission via contaminated blood products is also possible.

malaria, making her more susceptible for an infection. Children belong to the most vulnerable group, because they have not yet developed resistance.

Malaria symptoms include fever and flu-like illnesses. Severe malaria, if untreated, can cause coma and death. Antimalarial drugs are available, which usually results in complete recovery. The most common subscribed drugs is Chloroquine but the parasites' resistance to this drug has spread widely, making this drug ineffective in many affected regions (Plowe 2008).

To provide a globally coordinated approach to fighting Malaria, the Roll Back Malaria (RBM) Partnership was launched in 1998. With the Abuja declaration from 2000, African leaders committed themselves to halve malaria death by 2010. The declaration's goals were to guarantee access to treatment within 24h after onset of symptoms, to protective measures such as insecticide-treated nets (ITNs), and to provide chemoprophylaxis to at least 60% of the population at risk (RBM 2003). The effectiveness of the RBM initiative had been unclear and the partnership had been sharply criticized for its loose association structure, its inadequate and conflicting advises given by the partner institutions, and, against the background of increasing resistance to drugs, its focus on monotherapies (Yamey 2004).

Malaria is one of the major public health problems in Malawi, accounting for about 40% of all hospital deaths in children under five (NSO 2005). To control malaria, the Government has implemented several strategies through the National Malaria Control Programme. The programs included a better malaria case management, and the provision of prophylactic drug treatment to pregnant women. Its main focus however, is on malaria prevention through the distribution of ITNs.

In 1998, the Government of Malawi launched a pilot social marketing program to distribute ITNs in the Blantyre district. In 2002, the program expanded to become the first national ITN program in Sub Saharan Africa. The distribution program scheme is three-fold: Pregnant women and children under five can obtain highly subsidized ITNs (MK 50/$0.33 per net) through clinics at which health staff retains MK 10 as an incentive to sell nets. Rural families can buy nets at a cost of MK 100/$0.66 from village health committees, NGOs, and community-level health personnel. The general population (mostly in urban areas) can obtain unsubsidized nets from the commercial sector at a cost of MK 550-780/$3.86-$5.20 per net (PMI 2007).

Over the period of 2000 to 2004, Malawi managed to increase bed net coverage from 13.1% to 41.9% (NSO 2005, NSO 2001). The program had been criticized however, for its failure to reach the poor, the group which is thought to be the most vulnerable to malaria (Mathaga and Bowie 2007). The government of Malawi has therefore revisited its ITN distribution policy in 2006, which now includes the free distribution to pregnant women and children under five through the Expanded Programme on Immunization (EPI) and antenatal health care clinics, the free distribution to the "poorest of the poor", as emergency response (particularly for HIV positives), as well as a subsidized distribution through community venues, and an unsubsidized distribution over the commercial sector.

Despite improvements in the coverage of bed nets, the effectiveness of the distribution program in terms of averted malaria cases has been unclear because no decline in malaria cases and deaths reported to the WHO was observed (WHO 2008). The public malaria surveillance system however, is likely to be imprecise, as it was argued that less than 10% of the world-wide Malaria cases and deaths are being reported (Breman and Holloway 2007). It is therefore unclear, in how far the public Malaria surveillance system can accurately portray the epidemic situation. Data from infant mortality, the group which is most vulnerable to an infection, show a strong downward trend from 10.3% in 2000 to 7.6% in 2004 (NSO 2005, NSO 2001). It is unclear however, to which extend the ITN distribution program has contributed to this decline, since the Government of Malawi has launched several other programs to reduce child mortality, such as vitamin A supplementation and vaccination campaigns. Since Malawi's malaria program is a role model for other countries, a detailed analysis on the effectiveness of the distribution program in terms of averted deaths is necessary.

4 Evaluating Malawi's ITN distribution scheme

4.1 Data

The data is from Malawi's Demographic and Health Surveys conducted in the years 2000 and 2004. These are nationally representative household surveys that provide data for a wide range of indicators in the areas of population, health, and nutrition

Both surveys systematically clusters from a list of enumeration areas defined in the 1998 Malawian Census of Population and housing (560 clusters in 2000; 522 clusters in 2004). Clusters were not identical in both surveys. Geographic location of each cluster is provided (GPS codes). From the list of eligible clusters, a systematic sample of households is drawn (for a total of 14,213 households in 2000 and 15,091 households in 2004). All women age 15-49 in the selected households were eligible for the individual interview.

The individual questionnaires covered various areas, including background characteristics (age, education, religion, etc.), household characteristics, and reproductive history. Information on reproductive history is used to construct recent infant mortality rates, which includes the survival status of all children who had been born in the year previous the interview (2933 children in year 2000; 2651 children in the year 2004). This definition differs to the definition of infant mortality in the Demographic and Health Surveys, which use all birth within the past 4 years to predict infant mortality. There are three reasons why this study limits to recent births: (1) there may be a considerable recall bias in earlier births, (2) information on individual and household characteristics is retrospectively not available and represent only the current status, and (3) earlier birth had not been (fully) exposed to the national wide bednet distribution scheme that started in 2002. Infant mortality in the year previous the interview is 8.7% in 2000 and 5.1% in 2004.

This paper uses bed net ownership as indicator variable, over which the distribution program affects child mortality. All households are asked whether they own a bed net. In the year 2000, 13.1% of children in the sample live in households with a bednet and 41.9% of children in the year 2004 live in households that owned a bed net. In both surveys, mortality is higher in children who were born into households without a bed net compare to children who were born into households with a bednet (8.9% vs. 6.9% in 2000; 6.1% vs. 4.0% in 2004).[6]

[6] The remaining descriptive statistics are provided in the appendix (Table A1).

The data does not provide information on other health interventions (such as vaccinations for example) for diseased children. Environmental factors, such as rainfall, are not available. A conventional before-after estimation is therefore not feasible. Using the stepwise implementation of the programme to construct a difference-in-difference estimation is also not possible because malaria has a strong geographical pattern, and mortality rates from geographic regions are not comparable. The alternative identification strategy from section 3 seems therefore suitable for evaluating the effectiveness of the ITN distribution scheme.

4.2 Plausibility of the identification strategy

The identification strategy outlined in section 3 requires to control for all relevant variables that jointly affect child mortality and bed net ownership (i.e. CIA assumption necessary to estimate the average treatment effect of bed net ownership), as well as for all time-variant predictors of bed net ownership (necessary to get an unbiased estimator for the complier population). To make the identification strategy credible, one needs to understand, which factors are associated with child mortality and bed net ownership.

4.2.1 Child mortality

An estimated 10.5 million children under 5 years die worldwide every year; of the 20 countries with the highest child mortality, 19 are in Sub Saharan Africa. Infectious and parasitic diseases are the main causes of death and represent about 60% of child deaths (WHO 2003). Malaria is one of the biggest killers, accounting for approximately 20% of child deaths in Sub Saharan Africa (Rowe, et al. 2006, Adazu, et al. 2005), other infectious diseases such as HIV/AIDS or lower respiratory infections (Garrip, et al. 2006) and severe anemia (Adazu, et al. 2005) are also important causes of deaths.

A substantial body of evidence exists on the determinants of child health and mortality in developing countries. This literature identifies low socio-economic status as one of the most important predictors of poor health and mortality (Omariba, Beaujut and Rajulton 2007, Minujin and Delamonica 2003). Using comparable data from 24 countries, Minujin and Delamonica (2003) for example, show that children from families belonging to the bottom quintile of the wealth distribution are three times more likely to die before age five than children belonging to the top quintile. A higher susceptibility to communicable diseases, and a poor access to health care infrastructure could explain the strong disparity in child health (Galiani, Gertler and Schargodsky 2005, Shi 2000, Koenig, Bishai and Khan 2001, Maitra and Ray 2004).

A further important factor of child mortality is the mother's education (Iram and Butt 2008, Omariba, Beaujut and Rajulton 2007). Mother's education can influence child survival by different ways. Education for example, can lead to a higher efficiency in producing child health, may shape mother's preferences, and raises incomes (Schultz 1984). A particular emphasis has been placed on the role of maternal health knowledge (Kovsted, Portner and Tarp 2002, Glewwe 1999). A study conducted in Guinea-Bissau for example, found a strong and positive effect of health knowledge (measured by knowing malaria transmission modes and preventive actions) on child health and survival that ‘crowed out’ the effects of the mother’s education (Kovsted, Portner and Tarp 2002).

Maternal education however, may not be the only factor relevant for health related decision making. Health related skills, values, and information are public goods within a household. Therefore, considerable intra-household externalities of the education of other household members may exist. Lindelow (2008) for example shows that after controlling for maternal education, and income, education of other (non-spousal) household members has a significant and large effect on health care choices such as maternity services and child immunization in Mozambique.

Community effects are important determinants of child mortality. Malaria for example, is strongly associated with the distance to the next water body (Noor, et al. 2008), or to lower altitude (Bodker, et al. 2003). Furthermore, a wide gap between rural and urban areas had been observed, with rural areas having a higher level and a lower reduction in child mortality compared to urban population (Wang 2002). These community effects may be explained by a low access to health care or with poverty being mainly concentrated in rural areas.

Additionally, child characteristics, such as a low birth weight (Guilkey und Riphahn 1998), twin birth (Van der Mei, Heijstrat und Boersma 2003), and the gender of the child (Wamani, et al. 2007) are strongly associated with poor infant health and mortality.

4.2.2 Bed net ownership

Relatively little is known about the demand for bed nets and the discussion is usually centered on strategies for achieving high coverage (Stevens 2005): one the one hand it is argued that the health benefit from ITN usage is so large that the provision should be free and paid by the government. Furthermore, a positive price for bed net may prevent poor people, who may be at highest risk for malaria, from using them. On the other hand it is argued that freely distributed bed nets will be less valued and may be used for alternative purposes. Empirical evidence for both claims can be found in the literature: In a recent experimental study of free bed net distribution in Western Kenya, Cohen and Dupas (2008) found no evidence that women who received free ITNs were less likely to use them than those who paid for their bed nets; Minakawa and colleagues (2008) in contrast find a considerable misuse of bed nets as people living in a study area adjacent to Lake Victoria used bed nets for drying fish and fishing.

Up to 2006, Malawi’s government charged a (partly) subsidized price for all bed nets distributed over the social marketing program to ensure that people consider nets as valuable items. Bed nets were distributed to pregnant women and newborn children at the lowest rate. Rural household had access to heavily subsidized bed nets through community venues. Unsubsidized bednets could be obtained through the private sector (mainly in urban areas).

Relatively little empirical studies have analyzed demand factors. Most studies focus on the price of bed nets (willingness to pay), where people usually reported a positive willing to pay for bed nets. However, willingness to pay varies strongly with socio-economic status (Onwujekwe, Chima, et al. 2001, Legesse, et al. 2007). In studies that analyze the usage of bed nets, lack of access, the perception that nets cannot prevent malaria, and low education are the most important predictors for bed net usage (Belay and Deressa 2008, Pettifor, et al. 2008).

4.2.3 Selection of control variables

Bed net ownership and infant mortality are likely to be influenced by the same factors. To justify the identification strategy outlined in section 3, we need to control for all factors that are jointly associated with infant mortality and bed net ownership, as well as for all time variant factors that are associated with bed net ownership. These variables can be categorized into the following groups: (1) household characteristics, (2) characteristics of the mother, (3) characteristics of the child, and (4) regional characteristics.

The Demographic and Health surveys provide a large set of covariates that can be used to approximate these categories: education of the household head, household wealth, mother's characteristics such as age, education, ethnicity, religion, number of children ever born, health knowledge (measured by knowledge on dehydration), child's characteristics such as (hypothetical) age, twin birth, sex, size at birth, place of delivery, and regional characteristics such as region, rural area, altitude, distance to the next district capital and distance to one of the major lakes (Lake Malawi, Lake Malombe, Lake Chilwa), both estimated from GPS information.[7,8]

Unfortunately, the data does not provide information on income. However, information on labor status, education, as well as the household wealth index are likely to capture (at least part of) the missing information. A complication arises when using the DHS wealth index, which is a linear combination of household item indicator variables. Indicator weights are based on a principal component analysis (Rutstein und Johnson 2004). Neither the household item indicators nor the indicator weights are identical in the two waves. Therefore, the DHS wealth indices are not comparable. This paper re-estimates the wealth index using the following household items: electricity, radio, television, bicycle, motorcycle, car, type of toilet, type of water supply, type of house floor, and type of cooking material. The correlation coefficient between the predicted wealth index and the orginal index is 0.84 for wave 1 and 0.96 for wave 2.

Information on other health indicators, such as vaccination coverage is not available for diseased children. The inability to control for these variables could bias the results, if they proxy access to health care.[9] Conditional on all other covariates however, it seems unlikely that this causes a major bias, because the place of delivery, socio-economic characteristics and regional characteristics are strongly correlated with vaccination coverage. This hypothesis is tested

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[7] GPS codes for district capital are provided by Wikipedia; GPS boundaries for the major lakes are provided by www.sahims.net.

[8] Descriptive statistics for the variables are provided in the appendix (Table A1).

[9] EPI as further distribution channel had not been implemented before 2006. Children considered in the surveys had therefore no access to subsidized bed net over EPI services.

using data from surviving children. Conditioning on all other confounding variables, vaccinations are not significantly associated with bednet ownership.[10]

4.3 Results

To estimate the treatment effect defined by equations (1b) to (3b), the propensity scores for wave 1 and wave 2 need to be estimated. A standard Probit model is used, with the indicator that the household own any bed net as the dependent variable. The regression results are presented in table 1.

[Table 1]

Both models provide a relatively good fit of the data, while the classification based on the predicted propensity scores is slightly better for the first wave. Among the strongest predictors for bednet usage are household characteristics, such as the education of the household head and the wealth index. Mother's education seems also relevant but (at least in wave 2) to a lower extend than the household head's education. In both waves, variables that capture malaria risk are strongly associated with bed net ownership. In 2004, average bednet ownership would be 84% if everybody would live at 0 m altitude and would shrink to 45% if everybody would life at 1000 m altitude (leaving all other covariates at their current values). Similar results can be obtained for the distance to one of the main lakes where average bed net ownership would be 56% if everybody would live at the lake shore but would shrink to 16% if everybody would life 500 km away from one of the major lakes. This risk depending behavior is in line with economic theory of health seeking behavior, which should be highest if the risk imposed is highest (Geoffard und Philipson 1996). The ownership of bed nets is positively associated with increasing distance to the next city in wave 1, but this disparity vanished in wave 2. The strong positive effect of giving birth in a health facility on bednet ownership in wave 2 is expected, as antenatal health care providers were the key distribution channel for ITNs.

[Table 2]

The results from the estimation defined in equation (1b) to (3b) can be found in table 2. The average treatment effect of bednet usage is equal to 2.5%. This effect corresponds to a hypothetic, maximal effect of a distribution scheme that has a 100% complier population. In the current example however, the complier population is 33%. Within the complier population the treatment effect is higher than the average treatment effect (LATE=3%). This could be a sign for the distribution program being able to reach people at highest risk, but since the confidence intervals are rather large, there is no evidence for a significant difference between LATE and ATE. The effect of the distribution scheme on average mortality is equivalent to the intention to treat (ITT) estimator. Table 1 shows that the distribution scheme had lowered infant mortality by 1 percentage points. Given the total decline in infant mortality of 3.6 percentage points, the analysis suggests that the bednet distribution program caused almost 30% of the total decline in the study period.

[10] Results of this Probit model are provided in the appendix (Table A2).

An extrapolation bias may arise when the support of the distribution of covariates in the group with bed nets differs from that of the group with no bed nets. Fortunately, there is no evidence of common support problem as there are only 35 children with propensity scores outside the common support region (defined by the lowest propensity score of children with a bednet and the highest propensity score of children without a bed net). The sensitivity of the analysis is checked these 35 cases out of the estimation. Resulting effects are similar to ones reported in table 2.

The identification assumptions (CIA and mean independence of bed net ownership in post-intervention and pre-intervention conditional on relevant covariates) are not testable. This strategy requires controlling for all time-varying confounders. The sensitivity of the results to omitted relevant variables is analyzed, leaving blocks of covariates out of the estimation procedure (table 3).

[Table 3]

When omitting community characteristics estimated effects are not significant on the 5% level anymore. This is expected since geographic information, particularly the distance to the next water body and altitude, is a strong predictor of bednet ownership and is also strongly associated with malaria related mortality. The sensitivity of omitting these variables demonstrates the importance of using geographic information when evaluating health policies (McKenzie and Gibson 2007). All other results however, are fairly robust to omitting relevant variables, which puts faith on the results presented in this paper.

5 Conclusions

This paper develops an empirical method to estimate the treatment effect of a large-scale intervention when data from a control group is not available. The identification of this effect requires that the intervention affects outcome only over a single indicator variables, which can be observed in the data. Conditioning on all relevant confounding variables, the intention-to-treat and the local treatment effect can be estimated.

The estimator is applied to data from Malawi to evaluate the effectiveness of Malawi's ITN distribution program in terms of averted infant death. It is shown that the distribution program was effective in reducing infant death, with about 30% of the total reduction in infant mortality over the study period attributable to the net distribution program. This demonstrates the effectiveness of Malawi's ITN social marketing program, which could serve as role model for other countries also.

The study demonstrates the usefulness of Demographic and Health Surveys for evaluating large-scale health interventions in developing countries. Particularly, the provision of GPS data can greatly improve our understanding of health seeking behavior as well as the effectiveness of policies in different geographic settings. The geographic information used in the current paper is altitude, distance to the next water body, and the distance to the next city. Including further information, such as the distance to the next health care provider for example, could further sharpen the results.

This study was able to link the ITN distribution scheme to recent declines in infant mortality, the group that is most severely affected by malaria. The lack of observing a similar trend in pubic surveillance data provided by the WHO indicates that this data may be inaccurate. Claims on effectiveness of malaria programs should therefore not be made on basis of the pubic surveillance data.

The study cannot identify the effectiveness of other components of Malawi's national malaria strategy (i.e. malaria case management, and the provision of prophylactic drug treatment to pregnant women). Furthermore, the effectiveness of the revision of the ITN distribution policy from 2006 has been unclear as data is only available for the years 2000 and 2004. Future research is needed to evaluate these factors.

TABLES

TABLE 1: Propensity scores (Probit model)

	Wave 1			Wave 2		
	Coef.		z	Coef.		z
Household characteristics						
Highest education HH: primary	0.31	***	2.82	0.23	***	2.99
Highest education HH: secondary, higher	0.71	***	5.03	0.59	***	5.34
Wealth index	0.54	***	9.63	0.54	***	8.23
Child's characteristics						
Twin	-0.17		-1.45	-0.08		-0.72
Age	0.09		0.72	-0.06		-0.56
Sex	0.04		0.58	-0.01		-0.23
Size at birth: average	-0.02		-0.18	-0.03		-0.47
Size at birth: larger than average	-0.12		-1.01	-0.20	**	-2.32
Place of birth: public health facility	0.13		1.60	0.24	***	3.48
Place of birth: private health facility	0.09		0.83	0.20	***	2.66
Mother's characteristics						
Age of mother	0.02	*	1.81	0.00		0.07
Highest education mother: primary	0.30	***	3.05	0.16	**	2.09
Highest education mother: secondary, higher	0.80	***	4.98	0.36	***	2.81
Ethnicity: Tumbuka	-0.12		-0.79	-0.22		-1.51
Ethnicity: lomwe	-0.47	***	-3.06	-0.18		-1.63
Ethnicity: Yao	0.18		1.24	0.03		0.25
Ethnicity: Ngoni	-0.29	**	-2.05	-0.08		-0.68
Ethnicity: other	0.10		0.79	-0.14		-1.22
Religion: Muslim	0.22	*	1.70	0.50	***	4.09
Religion: Other	-0.11		-0.33	0.00		-0.02
No. of children ever born	-0.02		-0.64	0.00		-0.03
Heard of oral rehydration	0.11		0.80	0.28	**	2.09
Uses oral rehydration	-0.08		-0.76	0.17		1.43
Married	0.07		0.56	0.17	*	1.83
Working	0.08		1.14	0.15	***	2.64
Regional characteristics						
Distance to lake	-0.007	***	-5.81	-0.002	***	-2.73
Altitude	-0.001	***	-8.18	-0.001	***	-10.48
Region: Central	-0.714	***	-4.98	-0.167		-1.26
Region: Southern	-1.000	***	-7.21	-0.458	***	-3.33
Rural	-0.068		-0.64	0.184		1.48
Distance to city	0.010	***	3.39	0.000		0.12
_cons	-0.49		-1.39	0.44		1.37
Number of observations			2615			2338
Pseudo R2			0.33			0.15
Classified + if predicted Pr>= .5			86.92%			69.25%
Hosmer-Lemeshow chi2(8)			12.14			12.36

TABLE 2: Treatment effects

	Effect	SE	95% Confidence interval	
ATE	-0.025	0.011	-0.046	-0.005
LATE	-0.030	0.012	-0.053	-0.007
ITT	-0.010	0.004	-0.017	-0.002

Note: Standard errors and confidence intervals are based on the bootstrap method with 1000 replications

TABLE 3: Sensitivity analysis

	w/o community		w/o household		w/o child		w/o mother	
	eff.	SE	eff.	SE	eff.	SE	eff.	SE
ATE	-0.026	0.013	-0.019	0.010	-0.026	0.011	-0.028	0.011
LATE	-0.019	0.010	-0.020	0.010	-0.029	0.011	-0.038	0.012
ITT	-0.006	0.003	-0.007	0.003	-0.009	0.004	-0.013	0.004

Note: Standard error and are based on the bootstrap method with 1000 replications

APPENDIX

TABLE A1: Descriptive statistics (sample)

	Wave 1		Wave 2	
	Net = 0	Net = 1	Net = 0	Net = 1
Household characteristics				
Highest education HH: primary	0.66	0.53	0.66	0.59
Highest education HH: secondary, higher	0.10	0.38	0.10	0.25
Wealth index	-0.20	0.54	-0.31	0.08
Child's characteristics				
Twin	0.06	0.05	0.05	0.04
Age	0.51	0.51	0.51	0.51
Sex	1.50	1.49	1.48	1.50
Size at birth: average	0.58	0.62	0.51	0.51
Size at birth: larger than average	0.18	0.14	0.20	0.14
Place of birth: public health facility	0.38	0.49	0.35	0.45
Place of birth: private health facility	0.13	0.16	0.28	0.30
Mother's characteristics				
Age of mother	26.15	25.70	26.33	25.80
Highest education mother: primary	0.64	0.62	0.66	0.64
Highest education mother: secondary, higher	0.05	0.25	0.06	0.16
Ethnicity: Tumbuka	0.08	0.17	0.10	0.09
Ethnicity: lomwe	0.22	0.05	0.19	0.16
Ethnicity: Yao	0.14	0.17	0.13	0.21
Ethnicity: Ngoni	0.11	0.07	0.08	0.08
Ethnicity: other	0.16	0.32	0.12	0.14
Religion: Muslim	0.16	0.18	0.11	0.22
Religion: Other	0.02	0.01	0.02	0.01
No. of children ever born	3.59	3.19	3.68	3.35
Heard of oral rehydration	0.13	0.12	0.18	0.18
Uses oral rehydration	0.72	0.75	0.74	0.77
Married	0.89	0.91	0.86	0.91
Working	0.53	0.57	0.56	0.57
Regional characteristics				
Distance to lake	58.30	34.83	60.81	50.21
Altitude	850.74	724.52	933.79	798.72
Region: Central	0.37	0.27	0.42	0.35
Region: Southern	0.52	0.35	0.46	0.52
Rural	0.86	0.66	0.94	0.87
Distance to city	18.77	18.37	22.20	20.52
Infant mortality rate	0.08	0.07	0.06	0.04

Omitted categories: Highest education: none; size at birth: below average; place of birth: home, other; ethnicity: Chewa; religion: Christian; has never heard of oral rehydration; unmarried; not working; region: Northern; urban

TABLE A2: Sensitivity check for omitted other health interventions (Probit model)

	Wave 1			Wave 2		
	Coef.		z	Coef.		z
Household characteristics						
Highest education HH: primary	0.31	***	2.82	0.23	***	3.00
Highest education HH: secondary, higher	0.71	***	5.01	0.58	***	5.32
Wealth index	0.54	***	9.63	0.54	***	8.25
Child's characteristics						
Twin	-0.17		-1.42	-0.07		-0.65
Age	0.08		0.57	-0.12		-1.07
Sex	0.04		0.59	-0.02		-0.28
Size at birth: average	-0.02		-0.18	-0.03		-0.45
Size at birth: larger than average	-0.12		-1.00	-0.19	**	-2.18
Place of birth: public health facility	0.13		1.55	0.23	***	3.32
Place of birth: private health facility	0.09		0.79	0.19	***	2.61
Mother's characteristics						
Age of mother	0.02	*	1.82	0.00		0.02
Highest education mother: primary	0.30	***	3.05	0.16	**	2.08
Highest education mother: secondary, higher	0.80	***	4.98	0.36	***	2.77
Ethnicity: Tumbuka	-0.13		-0.80	-0.22		-1.55
Ethnicity: lomwe	-0.47	***	-3.06	-0.18		-1.63
Ethnicity: Yao	0.18		1.23	0.04		0.26
Ethnicity: Ngoni	-0.29	**	-2.04	-0.08		-0.71
Ethnicity: other	0.10		0.77	-0.14		-1.22
Religion: Muslim	0.22	*	1.70	0.50	***	4.09
Religion: Other	-0.11		-0.33	-0.01		-0.04
No. of children ever born	-0.02		-0.65	0.00		0.01
Heard of oral rehydration	0.11		0.78	0.27	**	2.02
Uses oral rehydration	-0.08		-0.77	0.17		1.40
Married	0.07		0.57	0.16	*	1.76
Working	0.08		1.15	0.15	***	2.62
Regional characteristics						
Distance to lake	-0.01	***	-5.82	0.00	***	-2.72
Altitude	0.00	***	-8.19	0.00	***	-10.53
Region: Central	-0.71	***	-4.98	-0.17		-1.25
Region: Southern	-1.00	***	-7.22	-0.46	***	-3.35
Rural	-0.07		-0.62	0.19		1.49
Distance to city	0.01	***	3.40	0.00		0.24
Other health interventions						
Had any vaccination	0.03		0.32	0.11		1.49
_cons	-0.50		-1.42	0.41		1.26
Number of observations			2615			2338
Likelihood-ratio test (model 1 nested in model 2)			0.1			2.23

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