Kanako Ishida
4770 Buford Hwy. NE
Mail stop K-23
Atlanta, GA 30341 U.S.A.
Tel: +1-770-488-5981
kishida@cdc.gov

Evaluation of mortality impact of mass-scale disease-prevention intervention:

A case of nationwide distribution of insecticide-treated nets in Togo, West Africa

AUTHORS:

Ishida, Kanako, Paul Stupp, and Howard Goldberg

Division of Reproductive Health, Centers for Disease Control and Prevention

Marcy Erskine

International Federation of Red Cross

Kodjo Morgah

Independent consultant

Abstract

Evaluation of the mortality impact of a national-scale disease-prevention intervention is complicated by the endogeneity—those individuals who receive the intervention may be healthier than those who did not. This population-based study assesses the mortality impact of Togo's 2004 nationwide child health campaign that distributed insecticide-treated nets (ITNs) to mothers of children ages 9–59 months. We first demonstrate that a one-time campaign that restricts programme eligibility to households with a surviving child inadvertently excludes some households with a high child-mortality risk, by comparing mortality rates before and after the campaign according to households' eligibility status. We then estimate a two-equation model for ITN receipt and mortality risk in order to address potential endogeneity of ITN receipt. Results show that among households with a child eligible to receive an ITN, living in a household with ITNs significantly reduced mortality among children ages 20–59 months even after controlling for endogeneity. (149 words)

Keywords:

programme evaluation, disease prevention, child mortality, malaria, insecticide-treated net (ITN), Sub-Saharan Africa, Togo

Introduction

With increased funding, international cooperation, and advancement of technology, disease-prevention intervention efforts in less developed countries are expanding in their scale and scope, and the need for adequately evaluating the mortality impact of large-scale programmes is increasingly recognized by global health policy makers (Bertrand et al. 2006, Nahlen & Low-Beer 2007). For national disease-prevention programmes, randomized controlled groups do not exist because they target anyone within the national boundary (Kurth et al. 2006, Shadish et al. 2002), and since programme participation is not random in most cases, a common alternative, comparisons between those who were and were not exposed to the programme and between pre- and post-programme (Bonell et al. 2009), are often confounded by endogeneity, regression to the mean, and secular trends. For example, when participants are selected for their measurable characteristics that are associated with morbidity and mortality risks, such as higher socioeconomic status or geographic advantages of the participants, such as urban residence or proximity to where the programme is executed, multiple regression models that control for these observed characteristics will produce an unbiased estimate of the effect of programme participation. However, if participants are different from non-participants in unobserved characteristics, such as interest, motivation, and attitude toward health that are associated with the receptiveness and readiness to the programme participation as well as actual health status, then programme participation is endogenous and the estimate of the programme participation is biased in this method, unless these unobserved characteristics are controlled for (Hutchinson & Wheeler 2006). Furthermore, regression to the mean is observed when participants screened for certain eligibility criteria which are associated with an increased or decreased risk of the morbidity or mortality that the programme is design to reduce, the risk then returns to a less

extreme level with the passage of time, regardless of intervention (Allison et al. 2009, Morton & Torgerson 2005). Finally, morbidity and mortality may be changing over time as a result of other contemporaneous factors, including changes in political environments, infrastructure, and health service delivery.

The objective of this population-based study is to highlight potential bias as a result of endogeneity in a mass disease-prevention programme evaluation and address it methodologically. We use a simultaneous estimation approach and assess community-level effects in a discrete-time hazard modeling framework, using household-survey data collected three years after the 2004 National Integrated Child Health Campaign in Togo, West Africa, which included first-time nationwide distribution of insecticide-treated nets (ITNs) in Africa. Eaqually importantly, we also provide programmatic implication for mass public-health intervention efforts, particularly, we demonstrate that a one-time disease-control campaign that defines implicit and obvious eligibility—presence of surviving child during its execution period in this case—inadvertently fails to include a high risk segment of the population.

Methods

Background and data

During the African Roll Back Malaria summit held in Abuja, Nigeria in 2000, the use of ITNs was identified as one of the most effective measures for controlling malaria, and high ITN coverage for those most at risk of malaria mortality, particularly children under age five, was agreed to be an important goal of malaria control initiatives. In malaria endemic countries, 70 per cent coverage of ITNs is expected to bring about a 50 per cent reduction in malaria mortality and about 15–19 per cent reduction in all-cause childhood mortality (Korenromp et al. 2004, Rowe et al. 2007). Over the last decade, nationwide distribution of ITNs, often in conjunction with other

intervention programmes such as mass drug administration, has been carried out in many sub-Saharan African countries with high malaria prevalence.

The first nationwide distribution was conducted in 2004 in Togo, West Africa through partnership among the Togolese Ministry of Health, the Canadian Red Cross, the Global Fund to Fight AIDS, Tuberculosis and Malaria (GFATM), the World Health Organization (WHO), and the United Nations Children's Fund (UNICEF) (Takpa et al. 2005). Distribution of free long-lasting insecticide-treated net was part of a national integrated child health campaign, which also included measles and polio vaccinations and presumptive treatment (mebendazole) for helminthes infection. All three services were administered to all children aged 9–59 months who arrived at the local health care provider, regardless of vaccination history or whether they possessed an ITN. The original plan set by the Ministry was to distribute one ITN per child. A total of approximately 905,000 ITNs were distributed nation-wide.

The 2007–08 Togo Survey on Mortality Impact and Utilization of Insecticide-Treated Nets Three Years After an Integrated Campaign was conducted between December 2007 and February 2008. A nationally representative two-stage cluster sample design was used with 300 census enumeration districts (ED) randomly selected in the first stage and 42 households per ED randomly selected in the second stage. For the purpose of the study, EDs were defined as "communities." Household interviews were conducted in all households in the sample, and a separate interview was conducted with all women ages 15–49 years, who resided in these households. The household interview collected information on current mosquito net ownership, including ITN received during the 2004 campaign, and other household characteristics, such as assets and housing materials. The questionnaire for women collected a complete birth history for each respondent, including dates of birth, survival status, and age at death if the child died. The

sample contains 10,944 households with completed interviews, 11,860 women ages 15–49 years living in these households, and 30,291 live births to these women. We adjusted for stratification, clustering at the ED level, and unequal probability of selection throughout the analysis.

Analytic approach

We first estimate pre- and post-campaign mortality rates and their differentials by eligibility status and whether the household owned an ITN among eligible households. We use child-months between January 2002 and November 2007. The analytic sample consists of 539,680 child-months, corresponding to 17,193 live births from both eligible and non-eligible households. Although ITN distribution targeted children ages 9–59 months, mortality rates among children ages 1–8 months are included in the models because in the baseline study conducted in September 2004, malaria morbidity was found to be similarly high in this age group of children (Eliades et al. 2006). In addition, ITNs could potentially protect these younger children if sleeping spaces are shared. Infants under age one month are not included because neonatal deaths are generally attributed to endogenous factors, including congenital abnormalities, immaturity, and birth injuries.

In the estimation of mortality impact, we use two strategies to address the potential endogeneity bias. The first is to simultaneously estimate the receipt of an ITN and mortality impact in the same model, taking into account unobserved characteristics that affect these two outcomes. We use bivariate probit regression analyses to estimate discrete-time hazard models of child mortality and simultaneously assess the effects of ITN ownership on the hazards of dying, and the effects of household and community characteristics on the likelihood of the receipt of an ITN during the campaign. The simultaneous estimation approach assumes a joint normal distribution for the errors of the two equations, in other words, takes into account the potential

correlations in the unobservable affecting mortality and the programme participation. We expect a negative correlation of error terms, which would indicate that the unobserved characteristics that predispose the household to receive an ITN might decrease child mortality, independently of ITN ownership.

In order to estimate the mortality impact of a campaign ITN, we consider all exposures at ages 1–59 months to children who were born during or after 1997 and lived in households eligible to receive a campaign ITN. The dependent variable for the first equation is a dichotomous and time-varying indicator of whether the child was alive or died in any given month. Campaign participation is captured by the household-level ITN ownership, in other words, residence in a household that had a campaign ITN. It is a dichotomous variable coded 1 if the month of exposure is after the December 2004 campaign *and* the child lived in the household that received an ITN. Since the effectiveness of ITNs is well established in the randomized controlled studies across various settings (Fegan et al. 2007, Lengeler 2004, Phillips-Howard et al. 2003, ter Kuile et al. 2003), this simple binary indicator is rather designed to ask whether ITN distribution had significant, discernable effects of reducing child mortality *at the national level*. Therefore, we do *not* control for the potential heterogeneity in the number of ITNs or children in the household which received an ITN or the retention or conditions of ITNs, the frequency of use, or the level of adherence to the instructions regarding the use, and we estimate the effect of ITN ownership on all-cause childhood mortality. Since the ITN distribution campaign coincided with measles and polio vaccination and treatment for helminthes infection, a very small portion of the protective effect of ITN ownership may be attributable to mortality reduction as a result of these other interventions.

The second strategy to address the potential endogeneity bias is to estimate the effect of ITN coverage at the community level in addition to direct protective effects of ITNs in the households. This covariate captures potentially protective effects of community ITN coverage and is constructed based on all 42 households in each sampling cluster, regardless of whether the households were eligible to get an ITN. Estimating the effect of ITN coverage at the community level has both methodological advantage and programmatic importance. First, the mortality impact of community-level ITN coverage is more likely to be exogenous to the unobserved characteristics of mothers than that of ITN ownership measured at the individual level. Second, results from past studies regarding the community-wide effects of ITN ownership and use have been inconsistent, and we need further evidence based on a large sample. There might be community-level effects of ITN ownership of reducing malaria prevalence if the number of blood-fed mosquitoes in a community is reduced, thereby reducing transmission risk even for children not directly protected by ITNs in their households (Cuzin-Ouattara et al. 1999, Gimnig et al. 2003, Hawley et al. 2003, Hii et al. 2001, Howard et al. 2000, Magesa et al. 1991, Maxwell et al. 2002, Mbogo et al. 1996, Smith et al. 2001). On the other hand, there might be no community-level effects when ITN coverage is incomplete because mosquitoes might be diverted from households with ITNs to neighboring households without ITNs, increasing the risk of malaria transmission among children in the latter households (Lindsay et al. 1993, Lines et al. 1987, Quinones et al. 1998, Snow et al. 1987, Thomson et al. 1995). In this case, malaria transmission risk might remain unchanged *at the community level*. We calculate the proportion of households with an ITN in the sample cluster, and code any month of exposure that precedes December 2004 0 and 1 otherwise.

We include a number of independent variables that are potentially related to mortality in the first equation. Child's characteristics included age categorized into three groups (ages 1–8 months, 9–19 months, and 20–59 months), birth year, sex, birth order, and birth interval length (less than 24 months and 24 months or longer). Birth year is designed to capture the secular trend of mortality over years. Mother's characteristics are age at birth of the child and marital status (whether they have never been in a union, currently in a consensual or marital union, or separated/divorced/widowed), and educational attainment (no schooling, incomplete elementary schooling, and complete elementary or above) at the time of the survey. Household characteristics include number of children under age five at the time of the campaign to capture the potential sibling competition for household and parental resources, whether there were any pre-campaign nets in the household, and wealth status. Community-level variables are region and proportion of households with pre-campaign nets. We subsequently add interactions between campaign ITN ownership and child age in this mortality equation to examine whether the expected protective effects of ITNs vary by the age of the child. Of note, these characteristics were recorded at the time of the survey and might not accurately reflect the characteristics at the time of the campaign.

The dependent variable in the second equation is the receipt of a campaign ITN, and is time-invariant, coded 1 for all the households that received a campaign ITN and 0 otherwise, regardless of time. This assumes that the unobserved characteristics that predispose mothers to receive an ITN were also correlated with unobserved characteristics that improve child survival chance throughout the period considered for this study. There two equations have the same set of covariates, except that we omit, from the first mortality equation, the community-level variable of proportion of households with electricity to avoid the potential identification problem in the

simultaneous estimation model. This covariate was found to be a significant determinant of the receipt of an ITN, but not to be significantly associated with child mortality in a one-equation model in an auxiliary analysis (results not shown).

Results

Mortality rates among children ages 1–59 months

Table 1 presents mortality rates for children ages 1–59 months during two time periods: three years before and three years after the December 2004 campaign. Nationally, the estimated annual mortality rate declined from 15.6 deaths per 1,000 child-years before the campaign to 12.9 deaths per 1,000 child-years after the campaign. Since the campaign targeted only those households with children ages 9–59 months in December 2004, we also calculate mortality rates before and after the campaign by whether or not children lived in a household eligible to receive a campaign ITN. Pre-campaign mortality for children in eligible households was substantially lower than pre-campaign mortality of children from non-eligible households (10.6 versus 73.8). This is because the eligibility criterion excluded households with children that died immediately before the campaign were not eligible to receive an ITN unless they also had surviving children ages 9–59 months. After the campaign, the mortality of these groups changed in opposite directions: the mortality of children from eligible households slightly *rose* to 11.8 while the mortality of children from non-eligible households precipitously *dropped* to 17.4, reducing the differential by eligibility status after the campaign. The eligibility criterion used during the campaign, the presence of a surviving child, omitted a group of households with unrepresentatively high child mortality before the campaign. That is, the absence of children in those households was often a result of mortality among young children before the distribution occurred. Following the campaign however, the mortality rates for both groups rapidly regressed

to the national mean, but the rate for ineligible households remained significantly higher than that for eligible households, even after the campaign. This suggests that the siblings of children who died immediately before the campaign were also at a higher risk of mortality. The higher mortality among children in the ineligible households may also be explained by that the children, who were either not yet born or under 9 months of age at the time of the campaign, were likely to have first-time, young mothers, whose risk of experiencing the child death was higher than their high-parity and older mothers. In other words, the ITN distribution campaign inadvertently failed to include a group of households with high mortality risk for young children.

In Table 2, we restrict the sample to the child-months of exposure in the eligible households and calculate pre- and post-campaign mortality rates for children who lived in eligible households that received an ITN during the campaign and those lived in similarly eligible households which nonetheless did not receive an ITN. In this way, we eliminate from the analysis the confounding effect of the distribution scheme on mortality and examined the potential protective effects of ITNs against child mortality. The pre-campaign annual mortality rate of children from households that received an ITN during the campaign was 9.7 deaths per 1,000 child-years, while the rate among children from households that did not receive an ITN was 12.2. Although the difference between these figures is not statistically significant because of the insufficient sample size, the lower mortality for households that received ITNs might reflect the endogenous campaign participation. That is, households with lower pre-campaign mortality could have had a higher likelihood of receiving an ITN because mothers with characteristics favorable to child survival were also more likely to participate in the campaign. Thus, the expected protective effects of ITNs against mortality might be confounded by the protective

effects of other unmeasured practices, indicating the need to control for the unobserved characteristics in the estimation of the mortality impact of ITNs.

After the campaign, the mortality rates remained virtually the same among children in households with ITNs (9.7 to 9.9), but *increased* among children in households without ITNs (12.2 to 15.6). Although this increase is not statistically significant, the pattern of change in mortality between two periods by ITN receipt is consistent with the expected protective effects of ITNs. The difference in pre-campaign mortality by ITN receipt is not significant; however, the difference became significantly large after the campaign.

Household characteristics

Since only households with children ages 9–59 months were eligible to receive an ITN, we restrict the sample to the 4,589 households that contained at least one living child ages 9–59 months in December 2004, which constitute 40.4 per cent of the total sample of households.[1] Of these households, 68 per cent of these households actually received at least one ITN during the campaign as shown in Table 3. The same table also compares characteristics of households eligible to receive an ITN to the full sample which is representative of all households in Togo. The average number of children ages 9–59 months in the household at the time of the campaign was 1.4 in eligible households compared to 0.8 in all households. The average age of mothers[2] with children ages 9–59 months was 31.9 years old. The proportion of eligible households with at least one mother in a marital or cohabiting union at the time of the survey was 94.1 per cent. The breakdown of eligible households is 50.6 per cent with all mothers with no schooling; 32.2 per cent with the most educated mother in the household with incomplete elementary; and 17.2 per cent with the most educated mother with at least complete elementary schooling. Household wealth status is constructed based on wealth index scores, which is weighted sums of a wide

range of the housing characteristics and assets at the time of the survey and used to classify households into four wealth quartiles, with 1st being the lowest and 4th being the highest. Higher percentage of eligible than all households are found in the two lowest quartiles (32.3 per cent in the first and 27.7 per cent in the second categories) probably because of the association between lower economic status and higher fertility. There are six geographic units; the national capital city, Lomé, and five administrative regions (see Figure 1). Of note, each region is characterized by different climatologic conditions, ranging from the humid tropical and coastal south (Maritime) to the semiarid north (Savanes). Economic status also varies by region: Maritime is the most developed and modernized region that hosts the capital city, Lomé, and Savanes is the poorest region in the country. Compared to all households nationally, the eligible households are less likely to be found in Lomé (22.9 per cent versus 17.8 per cent).

[Figure 1 about here]

Mortality impact of ITN ownership: Multivariate models

Finally, we use a simultaneous estimation approach to examine whether the protective effects of ITN ownership are observed after controlling for both measured and unmeasured characteristics that might affect the receipt of ITNs. In Table 4, we present coefficient estimates of child mortality from two-equation probit regression models. The sample is restricted to children ages 1–59 months from eligible households. In order to control for the overall upward trend in mortality as a result of regression to the mean shown in Table 1, we include a time-varying, dichotomous variable coded 0 when the discrete child-month belongs to the period before the campaign and 1 when it belongs to the period after the campaign.

The residual covariance (–0.004) shown in the last row of the table is negative as expected, yet small and non-significant. The effect of ITNs on mortality is only slightly smaller

in this two-equation probit model than in the single-equation probit model (results not shown). Nonetheless, we present these conservative results from the simultaneous estimation.

The coefficient estimate for the time-varying variable of the receipt of an ITN at the household level is –0.109, which is statistically significant, suggesting that children living in a household with a campaign ITN were significantly less likely to die, controlling for other child, mother, household, and community-level characteristics.

The second model assesses interactions between presence of ITNs and child age, in order to investigate whether the protective effects of ITNs might have varied in significance and strength according to the age of the child. The interaction coefficients for having an ITN at ages 1–9 months and ages 10-19 months are small and not statistically significant. The interaction coefficient for having an ITN at ages 20–59 months is negative and significant.

In order to highlight the protective and particularly interactive effect of ITNs by age, predicted mortality rates are presented in Figure 2. Here, all other independent variables included in the simultaneous equation model are set to their respective means to represent typical children in eligible households. The estimated annual number of deaths per 1,000 children ages 1–59 months is 8.9 among those living in a household with ITNs and 12.9 without ITNs based on the first model where the effect of ITNs is constrained to be the same for all children ages 1–59 months. The second part of the figure shows age-specific mortality rates for children in households with and without ITNs based on the second bivariate probit model. As expected, mortality rates decline with age in general, but the estimated protective effect of having an ITN increases with age. The predicted annual death rate at ages 1–9 months is 29.0 per 1,000 child-years in households with campaign ITNs and 26.9 per 1,000 in households without them, and the difference is not significant. The age-specific annual rate at ages 10–19 months is 18.4 per 1,000

child-years with ITNs, and 22.5 per 1,000 without ITNs. The difference of 4.1 per 1,000 child-years is marginally significant. The protective effect of ITNs on mortality is most clearly evident at ages 20–59 months where the rate is 9.0 per 1,000 child-years in households without an ITN compared to 4.6 per 1,000 child-years in households with an ITN: 4.6 per 1,000 child-years; this difference is statistically significant.

[Figure 2 about here]

We find no evidence that the proportion of households in communities with a campaign ITN reduced mortality, holding constant the ITN ownership at the household level. Similarly, the proportion of households with a pre-campaign net in the sampling cluster did not have a discernible, protective effect on mortality, either alone or jointly with the proportion of households with ITNs (adjusted Wald test results not shown). Thus, our results do not support the hypothesis that coverage of ITNs at the community level provides protection against child mortality in addition to household-level effects.

We summarize additional findings from the second two-equation model. First, the coefficient for the variable indicating the period after the campaign is positive and significant (0.176) as expected, capturing the overall increase in mortality in the eligible households shown in Table 3. The coefficient for birth year is significantly negative, mirroring the secular trend of decreasing mortality since 1997 in Togo. Children born within 24 months after their mothers' previous birth were more likely to die than are first-borns. Children of mothers who have never been in union had a higher likelihood of dying than children of women in a union at the time of the interview; however, children of separated, divorced, and widowed mothers were not significantly disadvantaged in their survival. Mothers' educational attainment had expected linear negative effects on mortality; however, a significant protective effect is limited to children

of mothers with completed elementary education or higher. Household wealth status is not a significant predictor of child mortality, except for a marginally significant protective effect of being in a household in the second highest quartile compared to the lowest. Finally, children who resided in Plateaux, Centrale, and particularly Savanes were at a higher risk of dying compared to their counterparts in Lomé, which is consistent with the high incidence of severe poverty in Savanes.

Discussion and conclusions

The significance of mass disease-prevention efforts has not been paid much attention in the studies of populations despite its unique contribution to the course of mortality trend at the national level. We demonstrated that a distribution scheme based on the presence of a child, set forth by the first-ever nationwide ITN distribution in Togo, was closely and negatively associated with mortality that the campaign attempted to reduce, highlighting the important caveat of one-time public-health intervention. Mortality before the 2004 campaign among children in households that were not eligible to participate in the campaign was substantially higher than that among children in eligible households. This is because eligibility was implicitly conditional on survival of children to 9–59 months of age; households that had a recent child death were disproportionately excluded from the campaign. Children in non-eligible households continued to have a significantly higher risk of mortality than children from eligible households even after the campaign, although the difference precipitously decreases as a result of regression to the mean, suggesting that the most vulnerable children in the population were selected *out* of the campaign target.

Furthermore, we also showed that among eligible households, those which received an ITN had a slightly lower mortality rate than those which did not receive it even *before* the

campaign. Children who have already benefited from their mothers' other health-enhancing characteristics that are either observed, such as higher wealth status, or unobserved, such as better hygienic practices and nutritional status, were more likely to receive an ITN during the campaign. In other words, campaign exposure was endogenous to the mortality that it was designed to reduce.

We also demonstrated application of several strategies to address this last endogenous exposure to the campaign to isolate the effect of the campaign on child mortality. The first is the use of simultaneous estimation. The results indicated that the contribution of endogenous exposure to the mortality reduction is minimal, however. This may be because in addition to the breadth of the variables considered in this analysis, which captured a large portion of characteristics favorable to both receipt of ITNs and child survival, the campaign included measles vaccination. Some mothers might have participated in a campaign because they have failed to have their children vaccinated in a regular health provider visit as a result of the *lack* of unobserved characteristics favorable to child survival, cancelling out the otherwise negative correlation of the unobservable. The second is estimation of the community effects of ITN coverage. The results indicated there was no protective effect that was additional to the effect of ITN ownership at the household level.

Our findings provide robust evidence that nationwide ITN distribution appreciably reduced mortality among children ages 20–59 months in Togo, a malaria-endemic country, which solidly justifies current policy efforts to use mass ITN distribution to protect young children from malaria-related mortality. The limited success among younger children, however, should be taken with caution and should not be used to justify their exclusion in malaria prevention efforts in Togo or elsewhere, considering the results from the baseline study that

showed that malaria burden is comparable among younger children in Togo. The smaller effect among young children may be attributable to the relatively smaller proportion of malaria-related deaths at younger ages. In addition, since the ITN distribution campaign did not include children in this age group, mothers may have used the ITN for the children for whom they received the ITNs, and sharing of sleeping spaces and ITNs might have been limited.

This study provided encouraging findings about mortality impact of ITN distribution but also raised important issues related to the strategies of mass distribution schemes and their evaluations and implication beyond the Togo ITN distribution campaign and malaria-prevention efforts. First, eligibility needs to be carefully considered. One-time campaign may be cost effective and improve the coverage substantially in a short period of time; however, unless the mechanisms of routine replacement and redistribution are set in place, the presence of potential beneficiaries required by the programme may inadvertently exclude those at most risk because of the implied survival of the beneficiaries at the time of programme execution. Previous morbidity and mortality should be cautiously assess in defining the target population in order to increase the impact. In the case of this Togo campaign, inclusion of younger children and expectant mothers may have reduced such selection. Second, the potential bias in estimating the programme impact has been largely neglected in the literature. Although we found such bias to be minimal in our study, programme analysts need to consider this issue when evaluating the impact of disease-prevention efforts.

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Table 1 Mortality rates (deaths per 1,000 child-years) for children ages 1–59 months by whether the household was eligible to receive an ITN during the campaign, full-sample of exposure between January 2002 and November 2007

Eligibility status	Before campaign (January 2002–November 2004)	After campaign (January 2005–November 2007)	Difference between before and after campaign
Eligible[a]	10.6 (8.8–12.3)	11.8 (10.1–13.5)	+1.2
Not eligible	73.8 (60.9–86.7)	17.4 (13.3–21.6)	–56.4***
Difference by eligibility status	+63.2***	+5.6**	
Total	15.6 (13.5–17.6)	12.9 (11.2–14.6)	–2.7

Note: n=539,680 child-months All figures are weighted. 95 per cent confidence intervals are in the parenthesis.
$^{\dagger}p<0.10$; $*p<0.05$; $**p<0.01$; $***p<0.001$.

Table 2 Mortality rates (deaths per 1,000 child-years) for children ages 1–59 months by whether the household received an ITN during the campaign, sub-sample of exposure between January 2002 and November 2007 in households eligible to receive an ITN

Whether received an ITN	Before campaign (January 2002–November 2004)	After campaign (January 2005–November 2007)	Difference between before and after Campaign
Yes	9.7 (7.8–11.6)	9.9 (8.3–11.6)	+0.2
No	12.2 (9.2–15.3)	15.6 (11.9–19.4)	+3.4
Difference by whether received an ITN	+2.5	+5.7**	
Total	10.6 (8.8–12.3)	11.8 (10.1–13.5)	+1.2

Note: n=463,042 child-month. All figures are weighted. 95 per cent confidence intervals are in the parenthesis.
$^{\dagger}p<0.10$; *$p<0.05$; **$p<0.01$; ***$p<0.001$.

Table 3 Summary statistics of key variables among eligible and all households

Variables	Eligible households	All households
Per cent households with campaign ITN	68.2	-
Per cent households with net before campaign	16.2	15.8
Mean # of children ages 9–59 months in household at campaign (standard error)	1.4 (0.013)	0.8 (0.015)
Mean age in years of mothers[1] in household (standard error)	31.9 (0.118)	–
Per cent mothers[1] in union in household	94.1	–
Educational attainment of the most educated mothers[1] in household (per cent distribution)		
No schooling	50.6	–
Incomplete elementary	32.2	–
Complete elementary and above	17.2	–
Column total	100.0	–
Wealth status (per cent distribution)		
Quartile 1 (Lowest)	32.3	27.6
Quartile 2	27.7	23.8
Quartile 3	20.2	24.1
Quartile 4 (Highest)	19.9	24.5
Column total	100.0	100.0
Region (per cent distribution)		
Lomé	17.8	22.9
Maritime	24.1	25.3
Plateaux	25.4	23.2
Centrale	10.6	8.3
Kara	11.6	11.6
Savanes	10.6	8.7
Column total	100.0	100.0
Total number of households	4,589 (40.4 per cent)	10,918 (100.0 per cent)

Note: All figures, except total number of households, are weighted.
[1]Mothers refer to those who had at least one child ages 9–59 months at the time of the campaign.

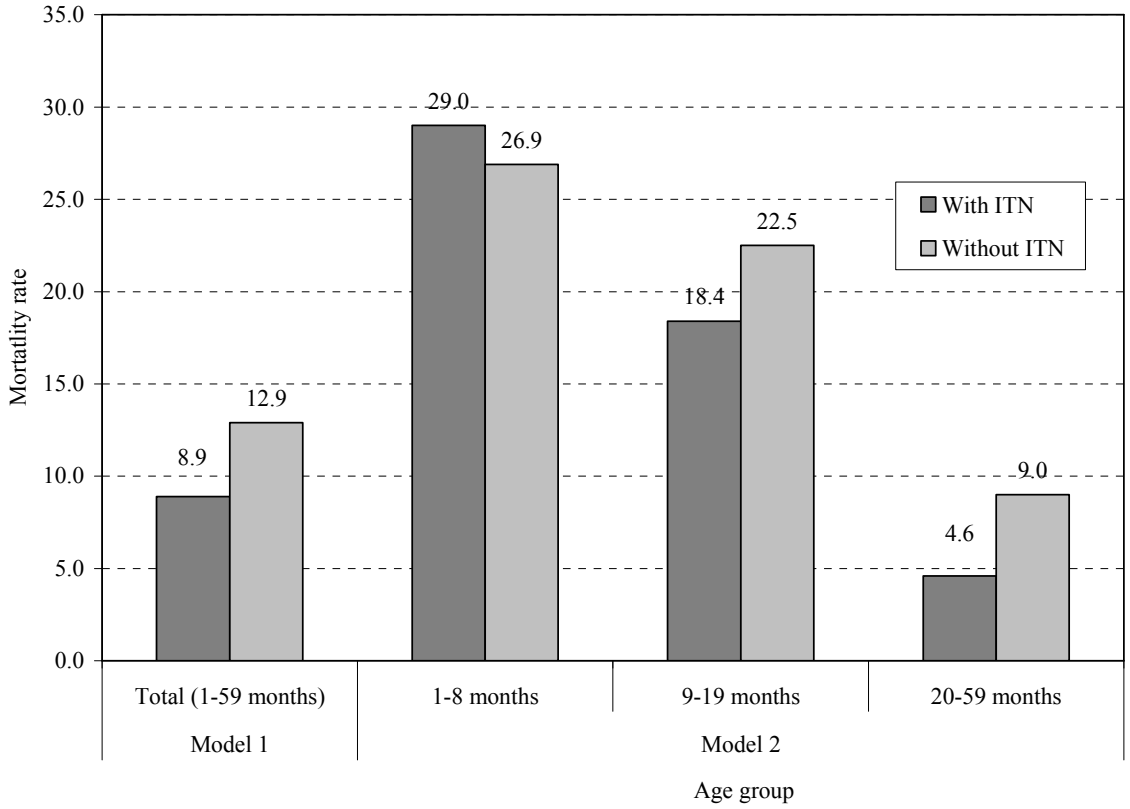
Table 4 Coefficient estimates for mortality from discrete-time bivariate probit regression models, sub-sample of exposure at ages 1–59 months for children who were born in or after 1997 in eligible households

	Model 1		Model 2	
Independent variable	Coefficient	Standard error	Coefficient	Standard error
Discrete time-level				
After campaign (time-varying)	0.176*	0.079	0.180*	0.079
Child-level				
Age in months [20–59 omitted]				
1–8	0.372***	0.032	0.333***	0.034
9–19	0.302***	0.031	0.277***	0.033
Birth year	–0.027***	0.007	–0.030***	–0.007
Male sex	0.022	0.023	0.021	0.023
Parity/birth interval [1st birth omitted]				
Birth interval less than 24 months	0.150***	0.024	0.152***	0.024
2nd–4th birth	–0.000	0.033	–0.004	0.033
5th birth and above	0.027	0.045	0.025	0.045
Mother-level				
Age in years at birth of the child	–0.004†	0.02	–0.004†	0.002
Marital status [in union omitted]				
Never in union	0.304**	0.104	0.303**	0.104
Separated/divorced/widowed	0.016	0.073	0.017	0.072
Educational attainment [no schooling omitted]				
Incomplete elementary	–0.047	0.031	–0.047	0.031
Complete elementary and above	0.153**	0.050	–0.152**	0.050
Household-level				
ITN (time-varying)	**–0.109***	**0.054**		
Interaction with child ages 1–9 months (time-varying)			0.025	0.072
Interaction with child ages 10–19 months (time-varying)			–0.062	0.072
Interaction with child ages 20–59 months (time-varying)			–0.188**	0.061
Ownership of pre-campaign net	0.023	0.036	–0.024	0.036

# of children ages 0–59 months	–0.012	0.012	–0.012	0.012
Wealth status [Quartile 1 omitted]				
Quartile 2	–0.000	0.028	–0.001	0.028
Quartile 3	–0.068†	0.040	–0.069†	0.040
Quartile 4 (Wealthiest)	–0.060	0.050	–0.059	0.050
Community-level				
Region [Lomé omitted]				
Maritime	0.125†	0.073	0.125†	0.073
Plateaux	0.184*	0.072	0.183*	0.072
Centrale	0.186*	0.072	0.184*	0.072
Kara	–0.007	0.089	–0.009	0.089
Savanes	0.229**	0.075	0.228**	0.075
Proportion of households with ITN in ED (time-varying)	**–0.018**	**0.200**	**–0.014**	**0.202**
Proportion of households with pre-campaign net in ED	–0.199	0.174	–0.196	0.174
σ (residual covariance)	–0.004	0.017	–0.004	0.017

Note: n= 524,837 child-month. All figures are weighted.
†$p<0.10$; *$p<0.05$; **$p<0.01$; ***$p<0.001$.

Figure 1 The map of Togo



Note: Other independent variables included in the probit regression model are set to their respective means.

Figure 2 Predicted mortality rate per 1,000 child-years based on two bivariate probit models with and without age-specific effects

Endnotes

[1]Approximately 6 per cent of all children ages 1–59 months and 7 per cent of children ages 9–59 months *at the time of the campaign* did not reside with their biological mothers *at the time of the survey*. However, since the time when these children began to live away from their mothers cannot be determined from the data, all children are assumed to have lived with their biological mothers until the time of the survey. Thus, whether the household contained a child ages 9–59 months at the time of the campaign and was thus eligible to receive an ITN, as well as the number of children ages 9–59 months per household are determined based on this assumption. Likewise, we have no information about any children who lived in the household, but were not biological children of female respondents ages 15–49 years; therefore, they are not included in the analysis.

[2] If there was more than one mother in a household (often because of polygamy), the average age of all the mothers is used as a household value. In these cases, the household value for proportion of mothers in union was 1 if at least one mother was in a union and 0 otherwise, and the education level of the most educated mother was used to represent for mothers' educational attainment.