The Importance of Household Responses to Competing Disease Risks

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### Summary

Child mortality is a persistent problem in sub-Saharan Africa that policymakers have up to now been unable to solve. The Millennium Development Goals set the bar for progress at a two-thirds reduction by the year 2015 (UNDP 2011). However, there has been nowhere near sufficient progress to meet this goal. While the child mortality rate in sub-Saharan Africa had fallen from 18 percent in 1990 to 13 percent in 2009, the total number of child deaths has increased from 3,937,000 in 1990 to 3,976,000 in 2009. Additionally, the share of under-five deaths coming from Sub-Saharan Africa increased from 31 percent in 1990 to 50 percent in 2009 (UNICEF et al. 2010).

Policymakers seeking to reduce child mortality and meet this goal have long sought to identify what programs are effective and why, but up to now have been unsuccessful. A common explanation for this failure is that health production is a complicated process that depends on many interrelated health inputs that all involve choices (Rutstein 2002). I thus argue in this dissertation that understanding how households make decisions about investment in interrelated health inputs is essential to improving child health. Here I focus specifically on how households respond to competing disease risks in making health input decisions. I argue that a better understanding of this decision-making process will enable policymakers to answer key questions about why programs are effective in reducing child mortality. Here I have specifically addressed two questions: 1) why does the effect of the same program differ by program site and 2) why do families spend little on disease prevention and have high price sensitivity for it when they spend relatively large amounts on medical treatment. I developed a theoretical model of household allocation with competing disease risks for children to demonstrate that household will respond to child health programs by investing in more disease prevention for their children and that this response will differ by determinants of household allocation to children. This differential response will lead to mortality effects that vary by these determinants of household allocation. I developed an additional theoretical model of parents' decisions to invest in disease prevention or spend on medical treatment in the presence of competing disease risks. I demonstrated that high levels of competing disease risks will decrease investment in disease prevention, increase price sensitivity for prevention, and increase the use of medical treatment when sick. I applied these two models to the questions raised above and evaluate them empirically using data from sub-Saharan Africa. I find empirical support for both models.

## 1. Introduction

High levels of child mortality in sub-Saharan Africa have been an international concern for many years; however, insufficient progress has been made to improve child health and reduce mortality. In 1990, the mortality rate for children under the age of five in sub-Saharan Africa was 18 percent and while in 2009 the rate had fallen, it was still 13 percent. Further, the total number of deaths of children under five actually increased from 3,937,000 in 1990 to 3,976,000 in 2009. Most troubling is the fact that over this period, child deaths in the rest of the world declined from 8,466,000 to 4,111,000. The share of under-five deaths coming from Sub-Saharan Africa increased from 31 percent in 1990 to 50 percent in 2009 (UNICEF et al. 2010). While the exact causes of mortality declines in the rest of the world are unknown, some factors that are generally thought responsible are increased nutrition, sanitation, income, and access to health services (Rutstein 2000). Reasons for the lack of declines in Africa are unknown with certainty, but one explanation is that there are many interrelated inputs to child health production and that problems with one key input can hinder the benefits of other improvements (Rutstein 2000). For example, Victora et al. (2003) argue that low levels of child nutrition diminish the effect of any other health programs by lowering overall immunity.

Child mortality in Sub-Saharan Africa is slightly different from the rest of the world in that it stems primarily from three major diseases, diarrhea, malaria, and pneumonia, with each disease accounting for approximately 25 percent of deaths between the ages of six months and 5 years. The other 25 percent of deaths are attributable to a range of causes such as HIV/AIDS, measles, and malnutrition (WHO 2010a). While these three diseases significantly affect other regions as well, child mortality there is

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spread across a larger number of diseases. These three diseases, while endemic in sub-Saharan Africa, all have straightforward methods for both prevention and treatment, so that there is little reason for such high mortality rates.

Diarrhea is caused by bacterial and viral infections in the intestinal tract. These infections generally arise from poor sanitation or hygiene, so that one form of prevention is simply to improve sanitation and practice good hygiene (WHO 2011a). Another, less well known, method of prevention is vitamin A supplementation. Substantial clinical research has shown that vitamin A improves immune response to diarrhea infections (Villamor and Fawzi 2005). While 42 percent of children in Africa do not obtain sufficient vitamin A through diet (Aguayo and Baker 2005), clinical research has shown that high dose vitamin A supplements can supply children with enough vitamin A to reduce mortality from diarrheal infections for six months (Villamor and Fawzi 2005). Thus, while vitamin A supplementation will not prevent contracting diarrhea, it will prevent death from infections by improving immune response. In addition to these prevention measures, diarrhea can be treated with a simple solution of salt, sugar, and water. This solution treats dehydration, which is the primary cause of death from diarrhea (WHO 2011a).

Malaria is caused by a parasite that is transmitted through mosquitoes. The mosquito species that carries the parasite bite only at night, so the primary method of prevention is to sleep under and insecticide treated net (ITN), because these nets prevent being bit by mosquitoes during this critical period (WHO 2011b). Effectiveness trials in sub-Saharan Africa estimate that ITNs reduce overall child mortality from malaria by 80 percent (Lengeler 2004). The primary symptom of malaria is a strong fever, so the WHO

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recommends that individuals seek treatment for any fever contracted when in a malarial area. Treatment consists of a regiment of anti-malarial drugs (WHO 2011b)

Pneumonia is caused by a bacterial infection in the lungs. The infection affects the lungs in such a way that it is difficult and painful to breath and less oxygen is absorbed when breathing. Children are at greatest risk of pneumonia when they are immune-compromised due to factors such as HIV/AIDS and malnutrition. The primary method of prevention is with a vaccination for Hib, pneumococcus, measles and whooping cough. Additionally, adequate nutrition is an effective prevention measure. Pneumonia can be treated with common antibiotics (WHO 2011c)

The international community has made improving child survival an official priority with Millennium Development Goal number four, which calls for a two-thirds reduction in child mortality by the year 2015 (UNDP 2011). As noted above, no progress has been made toward this goal in sub-Saharan Africa. However, this is not due to a lack of effort. There are many international organizations, governments, and NGOs implementing programs that attempt to improve child survival. These programs focus on a range of issues such as nutrition, immunization, sanitation, and primary health care (UNICEF 2008). While some of these programs have been deemed successful, the persistently high level of child mortality in the region clearly indicates that the overall effect is negligible. Policy makers have long sought to understand what programs work and why, but their failures in sub-Saharan Africa demonstrate that, at least in that region, they have yet to do so. A common explanation for this lack of understanding is that health production is a complicated process that involves both biological processes and choices of many inputs, many of which are interdependent (Rutstein 2000). Thus, a key

to discovering what programs work means understanding how families make choices about interrelated health inputs. Many questions plague policymakers as they try to find programs that will effectively reduce child mortality and most would benefit from a better understanding of how families make choices about spending on health inputs.

Two specific questions are: 1) why do the effects of the same program differ by program site and 2) why do families fail to invest in effective disease prevention and have high price elasticities for prevention when they spend relatively large amounts on medical treatment. The common answers to these questions have either never been tested or failed in testing. In this research, I will answer these two questions by modeling household behavior in the context of competing disease risks. An improved understanding of how families make decisions about health inputs will make it possible to answer these questions and design policies to improve child health in Africa.

# Question 1: Differential Program Effects

There are many instances where a program aimed at reducing child mortality in one area has a vastly different effect than the same program at a different site (Beaton et al. 1993; Bishai et al. 2005; Villamor and Fawzi 2000; Kouyate et al. 2008; Stansfield et al. 1993). One example of such differential effects comes from vitamin A supplementation programs. There is a significant amount of clinical and biological evidence showing that vitamin A supplementation reduces child mortality through improvements in immune response to diarrhea infections. This evidence is summarized by Villamor and Fawzi (2005), Stephensen (2001), and Semba (1998, 1999). The clinical evidence has led to the implementation of vitamin A supplementation programs in many developing countries. In these programs, large doses of vitamin A are given to children in pill form. The doses are so large that they generally only need to be administered every six months for children to obtain the vitamin A requirements necessary to achieve the mortality reductions observed in the clinical literature.

Beaton et al. (1993) conducted the first systematic review of these large scale vitamin A supplementation programs and concluded that vitamin A supplementation is associated with a 23% decrease in child mortality. However, this conclusion obscures a wide range of findings from over 50% reduction in mortality in Tamil Nadu and Bombay to no effect in Hyderabad, Sudan, and Haiti. Further, Bishai et al. (2005) found that in a randomized trial, the effect of vitamin A supplementation varied within a region in Nepal by population characteristics such as gender and Hindu caste. There are two existing explanations for this variation in program effects. The first argues that variation in the prevalence of the targeted disease due to location or household circumstances alters the effectiveness of an intervention (for example, Bishai et al. 2005 and Bleakley 2010). By this argument, places or individuals that have low initial levels of diarrhea will see lower program effects and this will account for variation both across and within countries. The second, argues that low levels of nutrition will reduce the effectiveness of interventions because immune systems will be compromised from the start (Victora et al. 2003). Thus, variation in nutrition across places and individuals will cause variation in program effects. It is unclear much of the variation in program effects can be explained by these two theories, because they have not been empirically tested; the work focusing on these explanations puts them forward either as assumptions used for identification or as ex-post potential explanations. Thus, the reason for the large across- and within-country variation in program effects remains uncertain.

I argue that a better understanding of household decisions about health investment, specifically in response to programs will make it possible to understand this variation. In this work I propose that previous studies of vitamin A supplementation are measuring the sum of the direct effect of supplementation on diarrheal infections and the indirect effect of household responses to supplementation. Households will respond to vitamin A supplementation because a reduction in the probability of death from diarrheal infections will increase incentives for households to invest in other child health inputs (Dow et al. 1999; Becker 2007). The theory arises intuitively from the idea that if children have a high probability of dying from both diarrhea and, for example, malaria, there is little incentive for parents to invest in ITNs to reduce death from malaria because even if they do, the children will die from diarrhea. In this example, a reduction in the probability of children dying from diarrhea increases parents' incentives to invest in ITNs for malaria prevention, because children won't die from diarrhea afterward (i.e. prevention measures are complements).

There is ample reason to believe that some of the differences in previous studies' estimates of the mortality effect of vitamin A supplementation are due to different family responses to the vitamin A intervention; specifically, different responses to the increased incentive to prevent competing diseases that result from the decreased diarrheal mortality. Families will respond differently for several reasons. Families may have different preferences for the equality of child outcomes (Becker and Lewis 1973; Behrman, Pollack and Taubman 1982). Responses will also differ depending on the (shadow) prices of the quantity and quality of children and family wealth (Becker and Lewis 1973). There is an extensive literature that develops these points (See reviews by Behrman and Deolalikar 1988; Behrman 1990; and Strauss and Thomas 1995). Some of the most important determinants identified in the literature are gender, number of siblings, mother's education, and household wealth.

Models of family bargaining over the intra-household distribution of resources also predict different responses to programs based on family characteristics (Alderman, et al. 1995, Haddad et al. 1997, Thomas 2000). One of the most important characteristics identified in the literature is mother's bargaining power. Young children often have little influence over bargaining for resources so it is argued that mothers do their bargaining for them. In support of this, empirical analysis has generally found that an increase in mother's bargaining power increases household investment in child outcomes (Thomas 1990, Lundberg et al. 1997, Quisumbing and Maluccio 1999, Thomas et al. 2002).

In summary, theories of competing disease risks indicate that the decreased mortality from diarrhea disease due to vitamin A supplementation programs will increase incentives for households to invest in other health inputs. The household allocation literature indicates that household responses to incentives for investment in child health will vary by several key observable characteristics, namely gender, number of siblings, household income, mother's education, and mother's bargaining power. Combining these two findings implies that the mortality effect of vitamin A supplementation programs will vary by these household characteristics, because households will invest in complementary health inputs differentially by determinants of allocation to children. Thus, understanding the household decision-making process for spending on health inputs can explain variation program effects.

Question 2: Prevention versus Treatment

Families in Africa invest little in cost effective methods of disease prevention such as ITNs and even small price increases cause families to reduce the use of preventive methods dramatically. For example, despite the mounting body of evidence on the effectiveness of ITNs for preventing malaria (estimated at an 80 percent reduction in malaria mortality by Lengeler (2004)), only 24 percent of children under the age of 5 slept under an ITN in 2008 (WHO 2009). Further, recent studies based on randomized trials have found that use of preventative services (especially ITNs) in Africa is highly price sensitive. For example, Cohen and Dupas (2010) conducted an experiment in which women attending antenatal clinics in Kenya were offered a random price for an ITN. They found a price elasticity of -1 when increasing the price from \$0.30 to \$0.60 (a reasonable range of prices because ITNs are highly subsidized in this area). In another experiment in Kenya that used prices closer to the unsubsidized price, Dupas (2009) found an elasticity of -1.8 at the mean price of \$2.30. Hoffman et al. (2008) conducted an experiment in Uganda and found a price elasticity of -3 at the median price of \$2.72. Together these studies provide evidence of large price elasticities for ITNs that increase with price.

In contrast to the relatively low investment in prevention and high price sensitivity, families seek medical treatment for malaria in children 1.8 times per year on average (WHO 2009). Further, research over the past two decades has found that families in Africa are insensitive to the price of medical treatment. Early research by Gertler and van der Gaag (1990) in Cote D'Ivoire reported a price elasticity of demand for medical treatment of approximately -0.12. More recent work has taken advantage of the imposition of user fees as a source of exogenous price variation. For example, a body of research analyzed the effect of user fees implemented in Kenya in 1992. Using this change in user fees, Bedi et al. (2003) found an elasticity of -0.08 for public clinics. Studying the same source of variation, Mwabu, Wang'ombe, and Nganda (2003) found an overall medical treatment elasticity of -0.02. Other recent work in Tanzania and Madagascar reported similar results (Sahn, Younger, and Genicot 2002; Fafchamps and Minten 2007). With respect to malaria treatment specifically, Dzator and Asafu-Adjaye (2004) reported a price elasticity of demand of -0.23.

This literature illustrates the apparent inconsistency in family decisions between investing in disease prevention and spending on medical treatment. Families in Africa invest relatively little in cost effective methods of disease prevention such as ITNs and even small price increases cause them to reduce the use of prevention methods dramatically. In contrast, families spend substantial amounts on medical treatment and that spending is price insensitive. This pattern is inconsistent with simple cost-benefit models of prevention and treatment decisions. For example, ITN effectiveness trials in Africa estimate that an ITN has a cost per disability adjusted life year (DALY) saved from malaria of \$23 when both net and insecticide treatment are purchased and \$8 per DALY saved when only insecticide treatment is purchase for a net already owned (Goodman et. al 1999). In contrast, spending on medical treatment for the average number of episodes of malaria per year (5.4) has a cost per DALY saved of \$170 assuming that 75 percent of cases are mild and 25 percent are severe (Jha, Bangoura, and Ranson 1998).

This apparent inconsistency has puzzled development policymakers as they work to reduce child mortality from preventable diseases such as malaria. Policymakers would like to tap into the seemingly high cost effectiveness of preventative measures and induce families to make investments in prevention, but doing so requires much higher subsidies than would be expected necessary with families continuing to rely on medical treatment for maintaining child health. There are two major explanations for this apparent inconsistency that focus on household decision-making, imperfect information and time inconsistent preferences. However, a randomized study that has empirically tested these explanations has shown them to be inadequate (Dupas 2009).

I develop an alternative model to explain the decision between investing in prevention, in this case ITNs, and waiting to seek treatment. I again draw on theories of competing disease risks from Dow et al. (1999) and Becker (2007). The decision of whether to invest in preventing malaria or wait to treat it will be influenced by the probability of dying from other diseases. Now the intuition is that if a child will die from diarrhea, there is little incentive to invest in malaria prevention, but a great incentive to see which disease is contracted and then seek treatment. As a result, factors causing mortality from competing diseases (ex. diarrhea) to be high will cause prevention (ex. ITN use) to be low, price elasticities for prevention to be high, and use of medical treatment to be high. Thus, understanding household behavior in this way can help to design policies that effectively induce families to shift from reliance on treatment to more cost effective prevention measures.

## Summary

Child mortality in sub-Saharan Africa is a persistent problem that policy makers have thus far failed to adequately combat. It is commonly thought that this failure is due to the complexity of the health production process and the many interrelated health inputs that enter it (Rutstein 2000). Based on this argument, the key to combating child mortality may lie in understanding how families make decisions about investments in interrelated heath inputs. In this research I focus on two specific unexplained policy questions and provide theoretical explanations using a model of household decisions about health spending that incorporates competing disease risks. I empirically test these theoretical explanations and find support for their hypotheses.

The rest of this dissertation is organized as follows: section 2 develops theoretical models of competing disease risks to explain household behavior in sub-Saharan Africa, focusing first on household allocation decisions and then on parental decisions between prevention and treatment for their children. Section 3 presents empirical analysis of the policy question, why do the effects of the same program differ by program site. Section 4 presents empirical analysis of the policy question, why do families fail to invest in effective disease prevention and have high price elasticities for prevention when they spend relatively large amounts on medical treatment. Section 5 concludes.

# 2. Theory

Recent theoretical work by Dow et al. (1999) and Becker (2007) on investments in health in the presence of competing disease risks has hypothesized that investment in disease specific prevention will depend on the probability of dying from other causes. Intuitively, this is because there is no incentive to invest in, for example, malaria prevention if the child will die from diarrhea regardless. In this research, I first examine the theoretical implications of this theory for household responses to child health programs. Second, I extend this theory to include the decision parents make between investing in prevention and spending on treatment for their children. The theory developed here can be used to explain the two policy questions raised in the previous section: 1) why do effects of the same program differ by program site and 2) why do families fail to invest in effective disease prevention and have high price elasticities for prevention when they spend relatively large amounts on medical treatment.

## Household Response to Health Interventions

I begin by modeling the household decision making process with a specific focus on how health resources are allocated to children. Parents have the following two period utility function:

(1) 
$$U = u_0(x_0) + B[S_1(p_1, p_2)\tau_i(\theta)u_1^n(x_1, H_1) + (1 - S_1(p_1, p_2))u_1^s(x_1)]$$

where  $u_0$  is the parent's utility in the first period, which depends on consumption in that period ( $x_0$ ),  $u_1^h$  is the parent's utility in the second period if their child survives to that period,  $u_1^s$  is the parent's utility in the second period if their child does not survive to that period,  $H_1$  signifies the health of the child in the second period,  $^1 S_1$  is the probability that

<sup>&</sup>lt;sup>1</sup> Here I will assume for simplicity that  $H_1$  is taken as given, but the model can be expanded to have  $H_1$  determined by health inputs.

the child will survive to the second period,  $p_1$  is the amount of services (effort) used in the first period to prevent disease 1 in the child,  $p_2$  is the amount of services (effort) used in the first period to prevent disease 2 in the child,  $\tau_i(\theta)$  is a weight function for the child's importance in parental utility and is a function of child and household characteristics ( $\theta$ ), and B is the discount rate.

The utility function is generalizable to n periods.<sup>2</sup> However, I use two periods here to focus on the critical period for parental investments in child health. The WHO estimates that child mortality between 6 months and 3 years is 2 percent while the mortality rate between 4 and 10 years is 0.3 percent (WHO 2010b). Thus, the first period can be thought of as the period of critical health investment and the second period can be thought of as the portion of childhood requiring less health input. The model assumes that the parent dies at the end of the second period.<sup>3</sup>

The weighting of child survival in parental utility, given by  $\tau_i(\theta)$  in equation (1), is the primary point of interest in this problem, because it indicates the level of importance that a child has in a parent's allocation decisions. These weights vary by individual and household characteristics given by  $\theta$ . The household allocation literature has identified several of these characteristics. For example, gender, mother's bargaining power, number of siblings, mother's education, and household wealth have all been demonstrated as determinants of household allocation to children (Behrman and

 $<sup>^{2}</sup>$  An n-period model produces further complementarities running from future survival to present health inputs. This will be examined in detail in extension 1 of this section. The predictions about the relationship between contemporaneous health inputs from the 2period model remain unchanged in an n-period model. <sup>3</sup> The model could also include a survival function for parents. If so, investments in prevention for children will depend on the

<sup>&</sup>lt;sup>3</sup> The model could also include a survival function for parents. If so, investments in prevention for children will depend on the probability that the parent survives, because the benefits of prevention stem from greater parental utility in the second period. Thus, there will be complementarities between parental health inputs and child health inputs. This will be examined in detail in extension 2 of this section. The predictions of the main model remain unchanged.

Deolalikar 1988; Behrman 1990; and Strauss and Thomas 1995; Thomas 1990; Lundberg et al. 1997; Quisumbing and Maluccio 1999; Thomas et al. 2002).

Assume that the child survival function can be decomposed into the effects of prevention for disease 1 and prevention for disease 2 such that

(2) 
$$S_1(p_1, p_2) = d_1(p_1)d_2(p_2)$$

where  $d_1(p_1)$  is the probability of surviving disease 1,  $d_2(p_2)$  is the probability of surviving disease,  $d_1(p_1) d_2(p_2)$  is the probability of being free from both diseases, and  $(1-d_1(p_1) d_2(p_2))$  is the probability contracting either or both diseases.

Parents maximize utility subject to the budget constraint given by:

(3) 
$$x_0 + \frac{x_1}{1+r} + q_1 p_1 + q_2 p_2 = y_0 + \frac{y_1}{1+r}$$

Where  $q_1$  is the price of preventative services for disease 1,  $q_2$  is the price of preventative services for disease 2,  $y_n$  is income in period n, and r is the real interest rate.

Maximizing utility with respect to prevention for disease 1  $(p_1)$  and prevention for disease 2  $(p_2)$  gives the following two first order conditions:

(4) 
$$\frac{\partial d_1}{\partial p_1} d_2(p_2) B(\tau_i(\theta) u_1^h - u_1^s) = \lambda q_1$$
  
(5)  $\frac{\partial d_2}{\partial p_2} d_1(p_1) B(\tau_i(\theta) u_1^h - u_1^s) = \lambda q_2$ 

The left-hand sides of equations (4) and (5) represent the marginal benefits of prevention spending for disease 1 and prevention spending for disease 2, respectively. Parents derive value from their children so these benefits stem from the added utility gained by the child surviving to the second period (i.e. the difference between  $\tau_i(\theta) u_1^h$  and  $u_1^s$ ). These survival benefits will be greater for parents with a higher weight for child

survival  $(\tau_i(\theta))$  and so will depend on the vector of determinants of this weight  $(\theta)$ . Factors that increase the utility weight for child survival will increase the marginal benefit of investment in disease prevention.

The survival benefits of each disease are positively scaled by the probability of avoiding the other disease so that an increase in disease 1 prevention will increase the marginal benefit of preventing disease 2. Intuitively, this is because there is little incentive to prevent disease 2 if the child has a high probability of dying from disease 1 regardless. Thus, the model predicts that prevention measures for the two diseases are complements. Importantly, this means that transfer programs that direct prevention measures for disease 1 to children will increase incentives for parents to invest in prevention of disease 2. Thus households will respond to programs transferring disease 1 prevention by investing more heavily in disease 2 prevention. This complementarity effect will be scaled by a child's weight in the parent's utility so that an increase in  $\tau_i(\theta)$  will increase the household response to the transfer of disease 1 prevention.

The right-hand sides of equations (4) and (5) represent the marginal costs of prevention spending for disease 1 and prevention spending for disease 2, respectively. These marginal costs are the price for each prevention measure.

From the first order conditions we see that the amount of disease 2 prevention purchased by the household is dependent most notably on the probability of surviving from disease 1 and the weight that child survival has in parental utility. Thus, one prediction of the model is that a program transferring prevention for disease 1 to children will increase the optimal amount of investment in disease 2 prevention. Another prediction is that households where child survival is given less weight in parental utility

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because of some child or household characteristic influencing allocation, the optimal amount of investment in disease prevention will be lower. Further, the response of households to transfers of disease 1 prevention will depend on the weight that child survival has in parental utility, so that households where child survival is given less weight in parental utility will not respond as strongly to transfer programs with complementary health investment.

Given the complementarities between prevention for the two diseases, to evaluate the survival effects of a program transferring disease 1 prevention to children we must identify two separate effects: first, the direct effect of disease 1 prevention increasing  $d_1(p_1)$  and second, the indirect effect of an increase in  $d_1(p_1)$  increasing the optimal amount of investment in disease 2 prevention (p<sub>2</sub>) and the subsequent increase in  $d_2(p_2)$ .

Assume now that p1 is obtained solely through transfer programs. Substituting optimal  $p_2$  into the survival equation gives optimal survival:

(6) 
$$S_1[p_2^*(p_1,\tau_i(\theta)),p_1] = d_1(p_1)d_2[p_2^*(p_1,\tau_i(\theta))]$$

In order to see the predicted survival effects of the transfer of disease 1 prevention to children, differentiate optimal survival with respect to (exogenous) p<sub>1</sub>:

(7) 
$$\frac{\partial S_1[p_2^*(p_1,\tau_i(\theta)), p_1]}{\partial p_1} = \frac{\partial d_1(p_1)}{\partial p_1} d_2[p_2^*(p_1,\tau_i(\theta))] + d_1(p_1) \frac{\partial d_2[p_2^*(p_1,\tau_i(\theta))]}{\partial p_2^*} \frac{\partial p_2^*(p_1,\tau_i(\theta))}{\partial p_1}$$

The weight of child survival in parental utility  $(\tau_i(\theta))$  will influence the effects of this transfer in two ways. First, it will directly influence the indirect effect of vitamin A supplementation on survival  $(\frac{\partial p_2^*(p_1, \tau_i(\theta))}{\partial p_1})$  through the response of households to

health complementarities. For example, parents with a lower utility weight for child survival will not increase disease 2 prevention as much in response to the program. Second, it will affect the base level of investment in disease 2 prevention (i.e. through  $p_2^*(p_1, \tau_i(\theta))$ ) and this will mitigate or magnify the direct effects of vitamin A supplementation  $(\frac{\partial d_1(p_1)}{\partial p_1})$ . These two influences mean that the effect of a transfer program depends on the weight that child survival commands in the parental utility function. Thus, the model predicts that the effect of a transfer program for disease 1 prevention will depend on the determinants of the weight that child survival has in the parental utility function.

## Prevention versus Treatment Decision

I now move on to examine the decision parents make between investing in prevention and spending on treatment for their children. Parent's now have the twoperiod utility function given by:

(8) 
$$U = u_0(x_0) + B[S_1(p_1, p_2, t_1, t_2)u_1^h(x_1, H_1) + (1 - S_1(p_1, p_2, t_1, t_2))u_1^s(x_1)]$$

where all variables are as before and the child survival function has been modified to include treatment for each disease so that  $t_1$  is the amount of medical treatment purchased to treat disease 1 in the child and  $t_2$  is the amount of medical treatment purchased to treat disease 2 in the child. For simplicity, I have omitted the explicit weight for child survival in parental utility. The inclusion of this explicit weight will not alter the predictions generated about the relationship between prevention and treatment.

Assume that the child survival function can now be decomposed into the effects of prevention for disease 1, prevention for disease 2, treatment for disease 1, and treatment for disease 2 such that

$$(9) S_1(p_1, p_2, t_1, t_2) = d_1(p_1)d_2(p_2) + [1 - d_1(p_1)T_1(t_1) + [1 - d_2(p_2)T_2(t_2) + [1 - d_1(p_1)T_1(t_1)[1 - d_2(p_2)T_2(t_2) + [1 - d_1(p_1)T_1(t_1)]] + [1 - d_2(p_2)T_2(t_2) + [1 - d_1(p_1)T_1(t_1)]] + [1 - d_2(p_2)T_2(t_2) + [1 - d_1(p_1)T_1(t_1)] + [1 - d_2(p_2)T_2(t_2) + [1 - d_1(p_1)T_1(t_1)]] + [1 - d_2(p_2)T_2(t_2) + [1 - d_1(p_1)T_1(t_1)] + [1 - d_2(p_2)T_2(t_2) + [1 - d_1(p_1)T_1(t_1)]] + [1 - d_2(p_2)T_2(t_2) + [1 - d_1(p_1)T_1(t_1)] + [1 - d_2(p_2)T_2(t_2) + [1 - d_1(p_1)T_1(t_1)]] + [1 - d_2(p_2)T_2(t_2) + [1 - d_1(p_1)T_1(t_1)] + [1 - d_2(p_2)T_2(t_2) + [1 - d_1(p_1)T_1(t_1)]] + [1 - d_2(p_2)T_2(t_2) + [1 - d_1(p_1)T_1(t_1)] + [1 - d_2(p_2)T_2(t_2) + [1 - d_1(p_1)T_1(t_1)]] + [1 - d_2(p_2)T_2(t_2) + [1 - d_1(p_1)T_1(t_1)] + [1 - d_2(p_2)T_2(t_2) + [1 - d_1(p_1)T_1(t_1)]] + [1 - d_2(p_2)T_2(t_2) + [1 - d_1(p_1)T_1(t_1)] + [1 - d_2(p_2)T_2(t_2) + [1 - d_1(p_1)T_1(t_1)]] + [1 - d_2(p_2)T_2(t_2)] + [1 - d_2$$

where  $d_1(p_1)$  is the probability of surviving disease 1,  $d_2(p_2)$  is the probability of surviving disease 2,  $T_1(t_1)$  is the probability of treatment for disease 1 leading to a successful recovery, and  $T_2(t_2)$  is the probability of treatment for disease 2 leading to a successful recovery. The term  $d_1(p_1)d_2(p_2)$  is equal to the probability of avoiding both diseases, the term  $[1 - d_1(p_1)]$  is the probability of contracting only disease 1 in a manner that would be fatal without treatment, the term  $[1 - d_2(p_2)]$  is the probability of contracting only disease 2 in a manner that would be fatal without treatment, the term  $[1 - d_1(p_1)][1 - d_2(p_2)]$  is the probability of contracting both diseases in a manner that would be fatal without treatment, and the term  $T_1(t_1)T_2(t_2)$  is the probability of treatment leading to a successful recovery if both diseases are contracted.

Parents maximize utility subject to the budget constraint given by:

(10) 
$$x_0 + \frac{x_1}{1+r} + q_1 p_1 + q_2 p_2 + [1 - d_1(p_1)] \frac{q_{t1}t_1}{1+r} + [1 - d_2(p_2)] \frac{q_{t2}t_2}{1+r} = y_0 + \frac{y_1}{1+r}$$

Where  $q_1$  is the price of preventative services for disease 1,  $q_2$  is the price of preventative services for disease 2,  $q_{t1}$  is the price of treatment for disease 1 and is scaled by the probability of contracting disease 1,  $q_{t2}$  is the price of treatment for disease 2 and is scaled by the probability of contracting disease 2,  $y_n$  is income in period n, and r is the real interest rate. Maximizing utility with respect to prevention for disease 1 ( $p_1$ ), prevention for disease 2 ( $p_2$ ), treatment for disease 1 ( $t_1$ ), and treatment for disease 2 ( $t_2$ ) gives the following four first order conditions:

$$(11) \frac{\partial d_1}{\partial p_1} \bigg[ \bigg[ d_2(p_2) - T_1(t_1) \big[ 1 + (1 - d_2(p_2)) T_2(t_2) \big] \bigg] B(u_1^h - u_1^s) + \lambda \frac{q_{t_1} t_1}{1 + r} \bigg] = \lambda q_1$$

$$(12) \frac{\partial d_2}{\partial p_2} \bigg[ \bigg[ d_1(p_1) - T_2(t_2) \big[ 1 + (1 - d_1(p_1)) T_1(t_1) \big] \bigg] B(u_1^h - u_1^s) + \lambda \frac{q_{t_2} t_2}{1 + r} \bigg] = \lambda q_2$$

(13) 
$$\frac{\partial T_1}{\partial t_1} \left[ 1 + (1 - d_2(p_2))T_2(t_2) \right] B(u_1^h - u_1^s) = \lambda \frac{q_{t_1}}{1 + r}$$
  
(14) 
$$\frac{\partial T_2}{\partial t_2} \left[ 1 + (1 - d_1(p_1))T_1(t_1) \right] B(u_1^h - u_1^s) = \lambda \frac{q_{t_2}}{1 + r}$$

The left-hand sides of equations (11) and (12) represent the marginal benefits of prevention spending for disease 1 and prevention spending for disease 2, respectively. Parents derive value from their children so these benefits stem primarily from the added utility gained by the child surviving to the second period (i.e. the difference between  $u_1^h$  and  $u_1^s$ ). These survival benefits will be greater for parents with higher preferences for children. The benefits of prevention also come from the decreased expected costs of treatment that result from a lower probability of illness.

The survival benefits of each disease are positively scaled by the probability of avoiding the other disease so that an increase in disease 1 prevention will increase the marginal benefit of preventing disease 2. Intuitively, this is because there is little incentive to prevent disease 2 if the child has a high probability of dying from disease 1 regardless. Thus, the model predicts that prevention measures for the two diseases are complements. The survival benefits of prevention are also negatively scaled by the effectiveness of treatment so that an increase in treatment for either disease will lower the marginal benefit of prevention. Intuitively, this is because there is little incentive to invest in prevention if the current level of treatment will lead to a successful recovery. Thus, the model predicts that prevention and treatment are substitutes.

The left-hand sides of equations (13) and (14) represent the marginal benefits of treatment spending for disease 1 and treatment spending for disease 2, respectively. As with prevention, these benefits stem from the added utility parents gain by the child

surviving to the second period. The survival benefits of treatment for each disease are negatively scaled by the probability of contracting the other disease so that a decrease in disease 1 prevention will raise the marginal benefit of spending on disease 2 treatment. Intuitively, this is because there is a greater incentive to wait to seek treatment for both diseases if the child has a high probability of contracting disease 1. This again illustrates the substitution between prevention and treatment. The survival benefits of each disease are also positively scaled by the effectiveness of treatment for the other disease so that an increase in treatment for disease 1 will raise the marginal benefit of treatment for disease 2. Intuitively, this is because there is greater incentive to spend on treatment for disease 2 if the child has a low probability of dying from disease 1 when he contracts both diseases.

The right-hand sides of equations (11), (12), (13), and (14) represent the marginal costs of prevention for disease 1, prevention for disease 2, treatment for disease 1, and treatment for disease 2, respectively. These costs are the prices of each input. Note that the marginal costs of treatment for each disease are the actual price rather the expected. This is because the probability of contracting each disease scales both marginal benefits and marginal costs of treatment and so falls out of the first order conditions for treatment.

In order to examine the effect of competing disease risks on prevention and treatment spending, I will construct conditional demand functions for each input. The general form of the conditional demand functions are:

- (15)  $p_1^* = f(t_1, t_2, p_2, y_0, y_1, q_1, q_2, q_{t1}, q_{t2})$
- (16)  $p_2^* = f(t_1, t_2, p_1, y_0, y_1, q_2, q_1, q_{t1}, q_{t2})$
- (17)  $t_1^* = f(t_2, p_1, p_2, y_0, y_1, q_1, q_2, q_{t1}, q_{t2})$

(18) 
$$t_2^* = f(t_1, p_1, p_2, y_0, y_1, q_1, q_2, q_{t1}, q_{t2})$$

The signs of the competing disease risk effects can be clearly seen in the first order conditions. In equation (12) (i.e. the optimal amount of investment in disease 2 prevention), an increase in  $p_1$  increases  $d_1$  and thus raises the marginal benefit of investing in  $p_2$ . Thus, factors causing high levels of investment in  $p_1$  will cause high levels of investment in  $p_2$  and factors causing low levels of  $p_1$  will cause low levels of  $p_2$  (i.e. prevention measures are complementary). Importantly, if investment in  $p_1$  is low because of a lack of knowledge about prevention or a lack of availability, there will also be little investment in  $p_2$  even if  $p_2$  is known as an effective prevention measure and is readily available. Additionally, transfer programs that direct prevention measures for disease 1 to children will have a magnified effect on survival in that they will also increase private investment in prevention of disease 2.

The effect of competing disease risks on treatment can also be seen in the first order conditions. In equation (14) (i.e. the optimal amount of investment in disease 2 treatment), an increase in  $p_1$  increases  $d_1$ , which lowers the marginal benefit of spending on  $t_2$ . Thus, factors causing high levels of investment in  $p_1$  will cause low levels of spending on  $t_2$  and factors causing low levels of  $p_1$  will cause high levels of  $t_2$ . Additionally, transfer programs that direct prevention measures for disease 1 to children will lower spending on treatment for disease 2.

Another important question in examining the apparent inconsistency between investment in prevention and spending on treatment relates to the effect of complementary prevention measures on price responses. Namely, do high levels of a competing disease risk increase price sensitivity for disease prevention? The answer can again be seen in the first order conditions. Solving equation (12) for  $\frac{\partial d_2}{\partial p_2}$  gives the

following:

(19) 
$$\frac{\partial d_2}{\partial p_2} = \frac{\lambda q_2}{\left[d_1(p_1) - T_2(t_2)(1 + (1 - d_1(p_1))T_1(t_1))\right]B(u_1^h - u_1^s) + \lambda \frac{q_{12}t_2}{1 + r}}$$

An increase in q<sub>2</sub> raises the right hand side of equation (19) and, assuming that there are decreasing returns to prevention (i.e.  $\frac{\partial^2 d_2}{\partial p_2^2} < 0$ ), this will cause a decrease in investment

in  $p_2$ . A decrease in  $p_1$  will lower the denominator of the right hand side of equation (19) and will increase the magnitude of the price effect. Thus, factors causing low levels of investment in  $p_1$  will cause high price sensitivity for disease 2 prevention. Additionally, transfer programs that direct prevention for disease 1 to individuals will cause lower levels of price sensitivity for disease 2 prevention.

To summarize, the model developed here leads to three clear predictions about the effect of competing disease risks on prevention and treatment spending:

- (1) Factors causing a low level of disease 1 prevention such as lack of availability or a lack of knowledge about benefits of prevention will cause low levels of investment in the prevention of disease 2. A policy that provides prevention measures for disease 1 to children will induce families to invest more heavily in prevention for disease 2.
- (2) Factors causing a low level of disease 1 prevention will increase price sensitivity for measures used to prevent disease 2. A policy that provides disease 1 prevention measures to children will decrease price sensitivity for measures used to prevent disease 2.

(3) Factors causing a low level of disease 1 prevention will cause high levels of spending on treatment for disease 2. A policy that provides prevention for disease 1 to children will cause a decrease in treatment spending for disease 2 when sick.

### Extension 1: Three Period Child Survival Function

Assume now there are three periods so that utility is given by

(20) 
$$U = u_0(x_0) + B\{S_1(p_{11}, p_{12}, t_{11}, t_{12})u_1^h(x_1, H_1) + [(1 - S_1(p_{11}, p_{12}, t_{11}, t_{12}))u_1^s(x_1)]\} + B^2(1 - S_1(p_{11}, p_{21}, t_{11}, t_{12}))u_2^s(x_2)$$

$$+B^{2}S_{1}(p_{11}, p_{21}, t_{11}, t_{12})\{S_{2}(p_{21}, p_{22}, t_{21}, t_{22})u_{2}^{n}(x_{2}, H_{2}) + [(1 - S_{2}(p_{21}, p_{22}, t_{21}, t_{22}))u_{2}^{s}(x_{2})]\}$$

where  $u_n^h$  is utility in period n if the child survives to that period,  $u_n^s$  is utility in period n if the child does not survive to that period, and  $S_n$  is the probability of the child surviving to period n, which depends on the prevention of disease 1 for period n ( $p_{n1}$ ), the prevention of disease 2 for period n ( $p_{n2}$ ), spending on treatment for disease 1 in period n ( $t_{n1}$ ), and spending on treatment for disease 2 in period n ( $t_{n2}$ ). The survival function for each period is the same as before (equation (9)).

The budget constraint is now given by:

$$(21) \quad x_{0} + \frac{x_{1}}{1+r} + \frac{x_{2}}{(1+r)^{2}} + q_{11}p_{11} + q_{12}p_{12} + [1 - d_{11}(p_{11})]\frac{q_{t11}t_{11}}{1+r} + [1 - d_{12}(p_{12})]\frac{q_{t12}t_{12}}{1+r} \\ + \frac{1}{1+r}S_{1}\left[q_{21}p_{21} + q_{22}p_{22} + [1 - d_{21}(p_{21})]\frac{q_{t21}t_{21}}{1+r} + [1 - d_{22}(p_{22})]\frac{q_{t22}t_{22}}{1+r}\right] = y_{0} + \frac{y_{1}}{1+r} + \frac{y_{2}}{(1+r)^{2}}$$

Where  $q_{n1}$  is the price of health inputs to prevent disease 1 in period n,  $q_{n2}$  is the price of health inputs to prevent disease 2 in period n,  $q_{tn1}$  is the price of treatment for disease 1 in period n, and  $q_{tn2}$  is the price of treatment for disease 2 in period n. Maximizing utility with respect to prevention for disease 1 in period 1 ( $p_{11}$ ), prevention for disease 2 in

period 1 ( $p_{12}$ ), treatment for disease 1 in period 1 ( $t_{11}$ ), treatment for disease 2 in period 1 ( $t_{12}$ ), prevention for disease 1 in period 2 ( $p_{21}$ ), prevention for disease 2 in period 2 ( $p_{22}$ ), treatment for disease 1 in period 2 ( $t_{21}$ ), and treatment for disease 2 in period 2 ( $t_{22}$ ) gives the following eight first order conditions:

$$(22) \frac{\partial d_{11}}{\partial p_{11}} \Big[ d_{12}(p_{12}) - T_{11}(t_{11}) \Big[ 1 + (1 - d_{12}(p_{12})) T_{12}(t_{12}) \Big] \Big[ B\Big[ (u_1^h - u_1^s) + BS_2(u_2^h - u_2^s) \Big] + \lambda \frac{q_{t11}t_{11}}{1 + r} \Big] \\ - \frac{\partial d_{11}}{\partial p_{11}} \lambda \Big( \frac{1}{1 + r} (1 - T_{11}(t_{11})) \Big[ Cost_2 \Big] \Big] = \lambda q_{11}$$

$$(23) \frac{\partial d_{12}}{\partial p_{12}} \Big[ d_{11}(p_{11}) - T_{12}(t_{12}) \Big[ 1 + (1 - d_{11}(p_{11})) T_{11}(t_{11}) \Big] \Big[ B\Big[ (u_1^h - u_1^s) + BS_2(u_2^h - u_2^s) \Big] + \lambda \frac{q_{t12}t_{12}}{1 + r} \Big] \\ - \frac{\partial d_{12}}{\partial p_{12}} \lambda \Big( \frac{1}{1 + r} (1 - T_{12}(t_{12})) \Big[ Cost_2 \Big] \Big] = \lambda q_{12}$$

$$(24) \ \frac{\partial T_{11}}{\partial t_{11}} \Big[ 1 + (1 - d_{12}(p_{12}))T_{12}(t_{12}) \Big] \Big[ B(u_1^h - u_1^s) + S_2 B(u_2^h - u_2^s) - \frac{\lambda}{1 + r} \Big[ Cost_2 \Big] \Big] = \lambda \frac{q_{t11}}{1 + r}$$

$$(25) \frac{\partial T_{12}}{\partial t_{12}} \Big[ 1 + (1 - d_{11}(p_{11}))T_{11}(t_{11}) \Big] \Big[ B(u_1^h - u_1^s) + S_2 B(u_2^h - u_2^s) - \frac{\lambda}{1 + r} \Big[ Cost_2 \Big] \Big] = \lambda \frac{q_{t12}}{1 + r}$$

$$(26) \ \frac{\partial d_{21}}{\partial p_{21}} \left[ \left[ d_{22}(p_{22}) - T_{21}(t_{21}) \left[ 1 + (1 - d_{22}(p_{22})) T_{22}(t_{22}) \right] \right] B^2(u_2^h - u_2^s) + \lambda \frac{q_{121}t_{21}}{1 + r} \right] = \lambda q_{21}$$

$$(27) \quad \frac{\partial d_{22}}{\partial p_{22}} \left[ \left[ d_{21}(p_{21}) - T_{22}(t_{22}) \left[ 1 + (1 - d_{21}(p_{21})) T_{21}(t_{21}) \right] \right] B^2(u_2^h - u_2^s) + \lambda \frac{q_{122}t_{22}}{1 + r} \right] = \lambda q_{22}$$

$$(28) \ \frac{\partial T_{21}}{\partial t_{21}} \Big[ 1 + (1 - d_{22}(p_{22})) T_{22}(t_{22}) \Big] B(u_2^h - u_2^s) = \lambda \frac{q_{t21}}{1 + r}$$

(29) 
$$\frac{\partial T_{22}}{\partial t_{22}} [1 + (1 - d_{21}(p_{21}))T_{21}(t_{21})] B(u_2^h - u_2^s) = \lambda \frac{q_{122}}{1 + r}$$

Where Cost<sub>2</sub> is the cost of prevention and treatment in the second period.

The left-hand sides of equations (22) through (29) represent the marginal benefit of an increase in spending on prevention spending for disease 1 in period 1, prevention spending for disease 2 in period 1, treatment spending for disease 1 in period 1, treatment spending for disease 2 in period 1, prevention spending for disease 1 in period 2, prevention spending for disease 2 in period 2, treatment spending for disease 1 in period 2, treatment spending for disease 2 in period 2, respectively. As in the two-period model, the marginal benefits stem primarily from the added utility parents gain from the child surviving to the next period and secondarily from the reduced expected costs of treatment that result from increased disease prevention. In the three-period model, the marginal benefits of prevention and treatment for children in period 1 are now increased by the additional expected utility gained from the increased probability of surviving in period 2. Additionally, the marginal benefits are reduced by the increased expected costs of paying for child health inputs in period 2. These two effects will be a net positive, so the marginal benefits of disease prevention and treatment spending in period 1 will be greater than in the two-period model.

Importantly, the size of the increased marginal benefits will depend on the level of child survival in period 2. This means that a transfer of disease prevention or treatment for period 2, increasing the probability of survival in period 2, will increase the marginal benefit of investing in disease prevention and the marginal benefit of spending on treatment for period 1. There will thus be complementarities running from child health inputs in period 2 to child health inputs in period 1. The first order conditions for period 2 health inputs show that this complementarity will not run the other way, because period

1 survival falls out of the optimal decision since it is a multiplier for both marginal cost and marginal benefit.

It is also clear from the first order conditions that the complementarity observed in the two-period model between disease prevention within period 1 as well as the substitution between prevention and treatment within period 1 remain. Additionally, the second period has the same complementarity between disease prevention within period as well as the same substitution between prevention and treatment within period

To summarize, including a third period with a separate survival function adds one additional prediction to those from the original model: Factors causing a low level of child survival in the second period will cause low levels of investment in both disease prevention and medical treatment for children in the first period. Thus, a policy increasing child survival in the second period will increase investment in disease prevention and spending on medical treatment for children in the first period. *Extension 2: Parental Survival* 

Returning to the two-period model, assume now that parents have a survival function so that utility is given by

(30) 
$$U = u_0(x_0) + BS_p(h_p) \{S_1(p_1, p_2, t_1, t_2)u_1^n(x_1, H_1)\} + [(1 - S_1(p_1, p_2, t_1, t_2))u_1^s(x_1)]\}$$

Where  $S_p$  is the probability of the parent surviving to the second period and is an increasing function of parental health inputs  $h_p$ . All other variables are as before and the survival function remains the same as before (given by equation (9)).

The budget constraint is now given by:

$$(31) \ x_0 + \frac{S_p(h_p)x_1}{1+r} + q_ph_p + q_1p_1 + q_2p_2 + [1-d_1(p_1)]\frac{q_{t1}t_1}{1+r} + [1-d_2(p_2)]\frac{q_{t2}t_2}{1+r} = y_0 + \frac{S_p(h_p)y_1}{1+r}$$

Where  $q_p$  is the price of parental health inputs. Maximizing utility with respect to parental health inputs ( $h_p$ ), prevention for disease 1 ( $p_1$ ), prevention for disease 2 ( $p_2$ ), treatment for disease 1 ( $t_1$ ), and treatment for disease 2 ( $t_2$ ) gives the following five first order conditions:

$$(32) \frac{\partial S_{p}}{\partial h_{p}} \bigg[ B(S_{1}(p_{1}, p_{2}, t_{1}, t_{2})u_{1}^{h} - (1 - S_{1}(p_{1}, p_{2}, t_{1}, t_{2}))u_{1}^{s}) + \lambda \frac{y_{1} - x_{1}}{1 + r} \bigg] = \lambda q_{p}$$

$$(33) \frac{\partial d_{1}}{\partial p_{1}} S_{p}(h_{p}) \bigg[ [d_{2}(p_{2}) - T_{1}(t_{1})[1 + (1 - d_{2}(p_{2}))T_{2}(t_{2})]] B(u_{1}^{h} - u_{1}^{s}) + \lambda \frac{q_{n}t_{1}}{1 + r} \bigg] = \lambda q_{1}$$

$$(34) \frac{\partial d_{2}}{\partial p_{2}} S_{p}(h_{p}) \bigg[ [d_{1}(p_{1}) - T_{2}(t_{2})[1 + (1 - d_{1}(p_{1}))T_{1}(t_{1})]] B(u_{1}^{h} - u_{1}^{s}) + \lambda \frac{q_{n}t_{2}}{1 + r} \bigg] = \lambda q_{2}$$

$$(35) \frac{\partial T_{1}}{\partial t_{1}} S_{p}(h_{p}) [1 + (1 - d_{2}(p_{2}))T_{2}(t_{2})] B(u_{1}^{h} - u_{1}^{s}) = \lambda \frac{q_{n}}{1 + r}$$

$$(36) \frac{\partial T_{2}}{\partial t_{2}} S_{p}(h_{p}) [1 + (1 - d_{1}(p_{1}))T_{1}(t_{1})] B(u_{1}^{h} - u_{1}^{s}) = \lambda \frac{q_{n}}{1 + r}$$

Again, the left-hand sides of equations (32), (33), (34), (35), and (36) represent the marginal benefit of an increase in spending on parental health inputs, prevention spending for disease 1, prevention spending for disease 2, treatment spending for disease 1, and treatment spending for disease 2, respectively. The marginal benefits of prevention and treatment for children are now scaled by the probability that the parent survives to the second period. Intuitively, this is because parents will only derive benefit from their children if they themselves are alive. The marginal benefit of parental health inputs depends on expected utility in the second period. This means that the marginal benefit is increasing in child survival to the extent that parents gain utility from their children (i.e. the magnitude of the difference between  $u_1^h$  and  $u_1^s$ ). The relationship running from parental health investments to child disease prevention and treatment can be clearly seen from the first order conditions. In equations (33), (34), (35), and (36) an increase in the probability of parental survival will increase the marginal benefit of investment in child disease prevention and treatment. Thus, an increase in parental survival probability will increase incentives to invest in prevention and spend on treatment for children. Intuitively, this is because parents will only gain benefit from their child surviving to the second period if they survive themselves.

The relationship running in the opposite direction (from child disease prevention and treatment to parental health investments) can also be clearly seen from the first order conditions. In equation (32) an increase in child survival from greater child disease prevention or medical treatment will increase the marginal benefit of investment in parental health inputs. Intuitively, this is because higher child survival to the second period increases parental utility in the second period and thus the benefits of parental survival. Thus, an increase in child disease prevention or medical treatment will increase incentives to invest in parental survival.

The predictions from the main model remain unchanged. The only difference is that now the complementarity effects will be scaled by the probability of parental survival so that parents with low survival rates will have a lower response of investment in  $p_2$  following an increase in  $p_1$ .

To summarize, in the extension to the model developed here, including parental survival, the predictions from the main model are unchanged and there are three additional predictions:

- Factors causing a low level of parental survival will cause low levels of investment in both disease prevention and medical treatment for children. A policy increasing parental survival will increase investment in disease prevention and spending on medical treatment for children.
- (2) Factors causing low levels of child survival, due to either low levels of disease prevention or medical treatment, will cause low levels of parental health investment. A policy increasing child survival will increase investment in parental health inputs.
- (3) Factors causing a low level of parental survival will mitigate the complementarity effect between prevention measures for the two diseases so that parents with a higher probability of survival will have a greater investment in p<sub>2</sub> in response to increases in p<sub>1</sub>.

## 3. Empirical Analysis: Heterogeneous Program Effects

The model of household responses to health programs developed in section 2 implies that decreased mortality from diarrhea infections due to vitamin A supplementation will increase incentives for households to invest in child health and households will respond to these incentives differentially by determinants of a child's weight in parental utility. This differential investment suggests that the effect of vitamin A supplementation on mortality is dependent on the determinants of a child's weight in parental utility. To test this hypothesis, I estimate the following:

(37) 
$$Mort_i = \beta_0 + \beta_1 A_i + \beta_2 \theta_i + \beta_3 A_i * \theta_i + \beta_4 X_i + \eta_k + \varepsilon_i$$

(38) ITN<sub>i</sub> = 
$$\alpha_0 + \alpha_1 A_i + \alpha_2 \theta_i + \alpha_3 A_i * \theta_i + \alpha_4 X_i + \omega_i + \varepsilon_i$$

Equation (37) is intended to be the reduced form demand function for child mortality (*Mort*) (corresponding to the optimal survival equation in section 2) and equation (38) is intended to be a model of the demand for ITNs for malaria prevention (*ITN*) (corresponding to the optimal investment in  $p_2$  equation in section 2). In these equations, *A* equals one if the child received a vitamin A supplement and zero otherwise,  $\theta$  is a vector of determinants of the household's weight for the child's welfare, *X* is a vector of other determinants of demand for health inputs and mortality,  $\eta$  is a community specific effect,  $\omega$  is a household specific effect, and  $\varepsilon$  is a random error term. The coefficient of interest in equation (37) is  $\beta_3$  since this will indicate if the effect of vitamin A supplementation on child health inputs and mortality depend on these observable determinants of a child's weight in parental utility. This corresponds to the variation in the indirect survival effect observed in equation (7) of section 2. If this coefficient is
significantly different from zero, then the effect of vitamin A supplementation on survival varies by the weight for child welfare in parental utility.

The variation in the effect of vitamin A on survival in equation (37) could also be accounted for by traditional explanations of differential program responses (i.e. different initial levels of the targeted disease or different innate immunity). Thus, to test whether differential survival effects are due to differing household responses to competing disease risks, equation (38) estimates these household responses. This corresponds to the optimal amount of investment in  $p_2$  in section 2 (equation (5)). The coefficient of interest in equation (38) is  $\alpha_3$  because this will indicate the extent to which responses to competing disease risks vary by observable determinants of household allocation to children. If this coefficient is significantly different from zero, then the effect of vitamin A supplementation on investment in prevention for competing disease risks varies by observable determinants of allocation to children.

As is common, available data are not sufficient to estimate correctly specified models, because there will be important missing variables. Regression models control for observable determinants of household health production and community health resources. In regression models of the effect of vitamin A on mortality I include the following household characteristics as controls for the health production function: mother's education, mother's literacy, mother's age, mother's age at first birth, number of living children, number of dead sons, number of dead daughters, whether there was a birth in the past year, household wealth, and marital status. I also include the following child characteristics: age, age squared, gender, birth order, and birth weight. As a control for community health resources I include community fixed effects. Concerns over the endogeneity of vitamin A supplementation are not so strong here, because vitamin A supplementation is not a health input readily available for purchase. Instead, it is primarily received through community-based programs initiated by governments, international aid organizations, and NGOs. Indeed, the inability to purchase vitamin A supplements has led organizations such as the World Bank to promote large scale supplementation initiatives at the community level (World Bank 2004). This circumstance implies that the primary source of endogeneity will relate to targeting of these supplementation campaigns at the community level. This is an issue that is effectively addressed by community fixed effects.

There may still be omitted factors that are correlated with both the decision to participate in vitamin A supplementation programs and child mortality, biasing estimates of the causal effect. However, much of these remaining factors are controlled for in the analysis of differential effects (the effects of principal interest for this research), because it is in essence a difference-in-difference (DD) analysis with differentially affected groups serving as controls. This procedure will purge the estimates of bias from all omitted factors affecting both vitamin A supplementation and mortality that are constant across the groups.

In the case of estimating the effect on ITN use, there is greater concern about endogeneity of vitamin A status due to household factors relating to health resources and health behaviors. Fortunately, in the case of ITNs, I am able to estimate a model for the effect of vitamin A on ITN use that includes mother (household) fixed effects instead of community fixed effects. I cannot conduct a similar analysis for mortality because, as discussed in more detail below, vitamin A status had to be imputed for deceased children based on the average status of their living siblings.<sup>4</sup> Mother fixed effects address much of the concern over endogeneity as they control for all family-specific, unmeasured factors such as health input prices, health resources, and health behaviors that may confound the estimated effect of vitamin A status on ITN use. These include access to and prices for prevention and treatment services, parental health knowledge, preferences for health care, as well as other potentially confounding factors.

While within household analysis covers most confounding factors, there may still be concern that child specific health factors will bias estimates. However, regressions (not reported here) of child characteristics (gender, age, birth order, and birth weight) on vitamin A supplementation indicate that only age and birth order are significant predictors of the decision to participate in supplementation programs. This is likely a mechanical relationship due to older children being in the program target group longer. Evidence for this lies in the fact that birth weight and gender—two factors that would not be mechanically related to supplementation—do not significantly influence program participation. Further, this is again a difference-in-difference analysis, so the only omitted factors that can confound estimates are child specific factors that vary systematically by group. To this end I include as controls the following child characteristics: age, age squared, gender, birth order, and birth weight.

Due to problems identifying or obtaining data on all of the components of the vector of household weight determinants ( $\theta$ ), I cannot include all of the components in the estimation of these equations. Thus, my estimation strategy will be to include

<sup>&</sup>lt;sup>4</sup> This method of construction means that estimating deviations from mean household vitamin A status and mean household mortality will require observations for three or more living children in the household within the age range being analyzed. Since this is not feasible, I cannot perform a household fixed effects analysis for the mortality analysis. I can use household fixed effects for the analysis of ITN provision, because in this analysis I only examine living children and thus no imputation of vitamin A status is necessary.

determinants that have been clearly identified in the literature and are available in the data. In this case, I will include gender, number of siblings, mother's education, mother's bargaining power, and household wealth. These five factors have been shown, in the literature reviewed in section 1, to influence household allocation to children. If omitted weighting factors are correlated with included factors, their omission will bias the estimates of the effect that these included weighting factors have on the effect of vitamin A supplementation. However, even in this case we can determine if the effect of the intervention varies by household weights and this is an improvement over past work that has failed entirely to address differential household responses.

### Data

I use data from the Demographic and Health Survey (DHS) in ten Sub-Saharan African countries over the period 2000 to 2006. Specifically I use data from Burkina Faso, Cameroon, Chad, Guinea, Mali, Malawi, Mozambique, Nigeria, Niger, and Rwanda. This data is well suited for analyzing the hypothesis proposed above, because it contains information on child health inputs and outcomes as well as detailed information on household characteristics. The Demographic and Health Surveys are nationally representative surveys of ever-married women aged 15 to 49. The surveys collect retrospective data on the health of all children born in the last five years as well as data on household and family characteristics.

The data contains information on vitamin A supplementation over the past six months, so to be in keeping with this time frame I will measure child mortality over the past six months. I will restrict my sample to children between the ages of 6 months and 3 years, because these children are at the highest risk for mortality. This sample consists of approximately 50,000 children. The countries used in this analysis all have high levels of mortality among children age six months to three years old. Column 1 of table 1 shows mean mortality rates for the six month period prior to the interview. Mortality rates in this period were 3.96 percent overall and range from 3.27 percent in Guinea to 5.13 percent in Nigeria.

	(1)	(2)	(3)
	Mortality	ITNs	Vitamin A
Total	3.96	7.40	59.99
Burkina Faso	4.28	2.22	35.94
Cameroon	3.70	1.16	39.16
Guinea	3.27	1.45	68.11
Malawi	3.94	16.66	70.49
Nigeria	5.13	1.04	35.84
Rwanda	3.98	14.70	78.33
Mozambique	3.33		54.33
Chad	4.98		45.22
Mali	4.05		58.00
Niger	3.57		73.77

 Table 1: Percentage of Child Mortality, ITN Use and Vitamin A Supplementation in the Past Six

 Months for Children Ages 6 Months to 3 Years, by Country

In measuring the demand for health inputs, I will use information on whether the child slept under an ITN the previous night. The survey modules containing questions about ITN use were only asked in six of the ten countries, so the sample used for the ITN analysis consists of approximately 29,500 children. In this analysis I will use only living children, because the DHS only contains information on the health inputs of living children. Column 2 of table 1 shows the levels of ITN use for the six countries in which data was collected. The level of ITN use is 7.4 percent overall and ranges from 1.04 percent in Nigeria to 16.66 percent in Malawi. These levels are low given the prevalence of malaria in these countries.

All countries used in this analysis have implemented vitamin A supplementation programs to varying degrees. Column 3 of Table 1 shows the mean rate of vitamin A

supplementation in the past six months. Supplementation rates in this period were 59.99 percent overall and range from 35.84 percent in Nigeria to 78.33 percent in Rwanda.

There is substantial variation in both mortality and ITN use between communities in each country and also within those communities. For example, regressions of mortality on country fixed effects produces an  $R^2$  of 0.002 and regressions of mortality on community fixed effects produces and  $R^2$  of 0.101. Additionally, regressions of ITN use on country fixed effects produces an  $R^2$  of 0.07 and regressions of ITN use on community fixed effects produces and  $R^2$  of 0.34.

The primary drawback of the DHS is that despite gathering information on those children that have died (a major benefit of the dataset), it does not record information on vitamin A supplementation or other health inputs for those children. Other researchers (for example Thapa et al 2005) have adjusted for this shortcoming by measuring the average coverage of vitamin A supplementation in a community and assigning this average level of coverage to all children in the community. The problem with this approach is that it potentially confounds the impact of vitamin A coverage with other unobservable community factors such as community health services.

In my analysis of mortality effects, I limit my sample to households where there are multiple children recorded in the data and assign children who have died the vitamin A status of their living siblings This restriction eliminates only 10 percent of the sample. In the case where the living siblings differ in their coverage of vitamin A supplementation, I will assign the average coverage across siblings to the dead child. As a test for the validity of proxying the vitamin A status of deceased children with the status of their living siblings, I examine the correlation in vitamin A status between living siblings. In 71 percent of households all members received the same vitamin A treatment (either all children received it or no children did). In regressions (not reported here) of child characteristics (gender, age, birth order, and birth weight) on vitamin A supplementation only age and birth order significantly predictors the decision to participate in supplementation programs. This method of assigning vitamin A receipt to deceased children may result in estimates biased toward zero because it is likely that dead children will have a lower probability of vitamin A supplementation than currently living children. This means that the estimates here may be lower bounds of the true effects. This sample restriction is unnecessary for the analysis of effects on ITN provision, because in that analysis the sample consists of only living children.

The determinants of a child's household weight in decision making to be examined in the empirical analysis are gender, number of siblings, mother's education, mother's bargaining power, and household wealth. Measuring the first three factors is fairly straightforward, but measuring mother's bargaining power and wealth merits some explanation. Mother's bargaining power will be measured using information about who controls daily household purchases. The intuition behind this approach is that whether a woman makes decisions about daily expenditures reflects her bargaining power and her influence over household allocation to children. Household wealth will be measured using the DHS wealth index. This index is consistent across countries and measures wealth by resources and amenities in a household.

#### Results

Table 2 presents a descriptive analysis of the effect of vitamin A supplementation on child mortality and the use of ITNs and the extent to which these effects differ by determinants of household allocation to children. This analysis first demonstrates the strong mortality reduction associated with vitamin A supplementation. It also demonstrates the variation in this reduction by observable determinants of allocation to children. The descriptive analysis also demonstrates that vitamin A is associated with higher levels of ITN use and that this association also varies by observable determinants of allocation to children.

		Morta	lity	ITN			
	Rec	eived V	<u>itamin A</u>	Receiv	ed Vitar	<u>nin A</u>	
	No Yes Difference		No	Yes	Difference		
All	0.044	0.038	-0.006	0.037	0.089	0.052	
Child Gender							
Female	0.046	0.038	-0.008	0.036	0.090	0.054	
mMale	0.042	0.037	-0.005	0.038	0.087	0.049	
Decision Maker							
Mother Decides	0.033	0.036	0.003	0.042	0.093	0.051	
Father Decides	0.045	0.038	-0.006	0.035	0.087	0.052	
Mother's Education							
Less than Primary Education	0.046	0.041	-0.006	0.018	0.033	0.015	
Primary Education	0.041	0.037	-0.005	0.070	0.121	0.051	
Secondary Education	0.032	0.022	-0.010	0.059	0.162	0.103	
Household Wealth							
Poor	0.044	0.039	-0.005	0.019	0.049	0.030	
Average	0.035	0.041	0.005	0.021	0.066	0.045	
Wealthy	0.048	0.036	-0.011	0.081	0.140	0.060	
Number of Siblings							
1	0.045	0.037	-0.007	0.048	0.115	0.067	
2	0.041	0.037	-0.004	0.034	0.093	0.059	
3	0.045	0.038	-0.007	0.032	0.072	0.040	

 Table 2: Descriptive Analysis of Differential Vitamin A Effects

Table 3 presents results from the estimation of seven models of the reduced form effect of vitamin A supplementation on mortality in the past six months for children age 6 months to 3 years. Column 1 presents results from a simple model of the mean effect of vitamin A on child mortality. These results demonstrate the significant effects of vitamin A supplementation, with supplementation leading to a 0.8 percentage point decrease in child mortality. This decrease represents a 20 percent decline in mortality from the mean mortality rate of 4 percent and is statistically significant.

Columns 2 and 3 present results from models where the effect of vitamin A supplementation on child mortality is allowed to vary by gender and mother's bargaining power respectively. These results indicate that the effect of supplementation does not differ by either of these characteristics as both differential effects are small and statistically insignificant.

Column 4 presents results from a model where the effect of vitamin A supplementation is allowed to vary by mother's education. These results demonstrate that children of higher educated mothers see a greater reduction in mortality than do those of low educated mothers. Specifically, children of mother's with less than a primary school education see a 0.5 percentage point decrease in mortality (12.5 percent of mean mortality) while children of mothers with a primary school education see a 1.3 percentage point decrease in mortality (30 percent of mean mortality) and children of mothers with a secondary school education see a 1.7 percentage point decrease in morality (42.5 percent of mean mortality). These differences from the low educated are statistically significant.

Column 5 presents the results of a model where the effect of vitamin A supplementation is allowed to vary by the number of siblings that a child has. Since all of the children in this sample have at least one sibling, the base case is children with only one sibling and the number of siblings is measured as the number above one. These results indicate that vitamin A causes a reduction of 1 percentage point (25 percent of mean mortality) for children with one sibling and that each additional sibling decreases

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this effect by 0.1 percentage points. This effect of additional siblings is significant at the 10 percent level.

Column 6 presents the results of a model where the effect of vitamin A supplementation is allowed to vary by household wealth. These results demonstrate that the effect of vitamin A supplementation is significantly higher for high wealth households. However, the effect does not differ in a statistically significant way for households with average wealth. Supplementation reduces mortality for children in the poorest households by 0.6 percentage points (15 percent of mean mortality) and 1.2 percentage points in high wealth households (30 percent of mean mortality). This difference is statistically significant at the 10 percent level.

Column 7 presents results from a model that incorporates all of the factors of the previous models (gender, mother's bargaining power, mother's education, number of siblings, and wealth). The only differential effect that remains statistically significant in this model is that for number of siblings. These variables are collinear. However, the F-test for the joint significance of the interaction terms is not significant. Thus, we cannot conclude that the effect differs by all household characterstics. The lack of joint significance is primarily driven by the steep decline in the effect of household wealth. The F-test of the joint significance of mother's education and number of siblings is significant however, so we can conclude that the effect differs by these two characteristics.

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Vitamin A	-0.008**	-0.009**	-0.008**	-0.005**	-0.010**	-0.006**	-0.008**
	(0.001)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.004)
Vitamin A*Female		0.001					-0.001
		(0.003)					(0.003)
Vitamin A*Mother Decides			-0.002				-0.000
			(0.004)				(0.004)
Vitamin A*Primary Education				-0.008**			-0.005
				(0.003)			(0.004)
Vitamin A*Secondary Education				-0.012**			-0.008
				(0.005)			(0.006)
Vitamin A*Siblings					0.001*		0.001*
(after the first sibling)					(0.001)		(0.001)
Vitamin A*Average Wealth						-0.002	0.001
						(0.004)	(0.004)
Vitamin A*High Wealth						-0.006*	0.001
-						(0.004)	(0.004)
Observations	49764	49764	46513	49764	49764	49764	46513
Mean	0.04	0.04	0.04	0.04	0.04	0.04	0.04

Table 3: Effect of Vitamin A Supplementation on Mortality in the Past 6 Months of Children Aged 6 Months to 3 Years

Notes:

1. All models include controls for mother's education, mother's literacy, mother's age, mother's age at first birth, birth in the last year, marital status, if father is present, household wealth index, number of living children, number of dead sons, number of dead daughters, age of the child, whether the child is first born, gender of the child, and the birth weight of the child.

2. All models include community fixed effects

3. Standard (clustered on DHS primary sampling units) errors are given in parentheses

4. \* indicates statistical significance at the 10% level, \*\* indicates statistical significance at the 5% level

The estimates from these models provide strong evidence that there are differences in the effect of vitamin A supplementation on child mortality by determinants of a child's weight in household decision making. Specifically, there is clear evidence in support of a differential effect by mother's education, number of siblings, and household wealth.

After determining that the mortality effects of vitamin A supplementation vary by determinants of household allocation, I will evaluate whether these differences are due to different behavioral responses by households. Specifically, I examine whether households respond to supplementation differentially in their investment in ITNs. Table 4 presents results from the estimation of seven models of the effect of vitamin A supplementation on ITN provision for children age six months to 3 years. Column 1 presents results from a simple model of the mean household response to complementarities between vitamin A and ITNs. These results demonstrate a strong household response to vitamin A supplementation. Supplementation leads to a 1.7 percentage point increase in ITN provision. This increase represents a 23 percent increase in ITN provision from the mean rate of 7.4 percent and is statistically significant.

Columns 2 and 3 present results from models where the response to vitamin A supplementation is allowed to vary by gender and mother's bargaining power respectively. Results indicate that the response to supplementation does not differ by either of these characteristics as both differential effects are small and statistically insignificant.

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Vitamin A	0.017**	0.015**	0.016**	-0.014**	0.026**	-0.016**	-0.030**
	(0.005)	(0.007)	(0.005)	(0.005)	(0.006)	(0.006)	(0.008)
Vitamin A*Female		0.004					0.005
		(0.008)					(0.008)
Vitamin A*Mother Decides			0.002				0.001
			(0.008)				(0.008)
Vitamin A*Primary Education				0.035**			0.027**
				(0.007)			(0.007)
Vitamin A*Secondary Education				0.096**			0.073**
				(0.013)			(0.013)
Vitamin A*Siblings					-0.006**		-0.004**
(after the first sibling)					(0.002)		(0.002)
Vitamin A*Average Wealth					. ,	0.023**	0.020**
C						(0.008)	(0.008)
Vitamin A*High Wealth						0.073**	0.060**
e						(0.010)	(0.010)
Observations	29500	29500	29488	29500	29500	29500	29488
Mean	0.074	0.074	0.074	0.074	0.074	0.074	0.074

Table 4: Effect of Vitamin A Supplementation on the Provision of Treated ITNs to Children Aged 6 Months to 3 Years

Notes:

1. All models include controls for age of the child, age of the child squared, whether the child is first born, gender of the child, and the birth weight of the child.

2. All models include household fixed effects

Standard (clustered on Household) errors are given in parentheses
 \* indicates statistical significance at the 10% level, \*\* indicates statistical significance at the 5% level

Column 4 presents results from a model where the response to vitamin A supplementation is allowed to vary by mother's education. These results demonstrate that households with higher educated mothers have a greater response to vitamin A supplementation than do those with low educated mothers. Households where mothers have less than a primary school education are found to reduce ITN provision in response to vitamin A supplementation. In contrast, households where mothers have a primary school education increase ITN provision to supplemented children by 2.1 percentage points (28 percent of mean provision) and households where mothers have a secondary school education increase provision by 8.2 percentage points (111 percent of mean provision). These differences are statistically significant.

Column 5 presents the results of a model where the response to vitamin A supplementation is allowed to vary by the number of siblings that a child has. These results indicate that vitamin A supplementation increases the provision of ITNs by 2.6 percentage points (35 percent of mean provision) to children in households with no siblings and that each additional sibling decreases this effect by 0.6 percentage points. This effect of additional siblings is statistically significant.

Column 6 presents the results of a model where the response to vitamin A supplementation is allowed to vary by household wealth. These results demonstrate that households with more wealth have a greater response to vitamin A supplementation than do those with low wealth. Households with low wealth are found to reduce ITN provision in response to vitamin A supplementation. In contrast, households an average level of wealth increase ITN provision to supplemented children by 0.7 percentage points (10 percent of mean provision) and households with a high level of wealth increase provision by 5.7 percentage points (77 percent of mean provision). These differences are statistically significant.

Column 7 presents results from a model that incorporates all of the factors of the previous models (gender, mother's bargaining power, mother's education, number of siblings, and wealth). The effects of mother's education, number of siblings, and household wealth on household responses to vitamin A supplementation are again statistically significant.

The estimates from these models provide strong evidence that households respond to complementarities between vitamin A supplementation and ITN provision. Additionally, there is evidence that this response varies by determinants of a child's weight in household decision making. Specifically, there is evidence in support of a differential response by mother's education, number of siblings, and household wealth; the same determinants that influenced the effect on mortality.

### Robustness Check

It was argued above first that vitamin A supplementation is likely exogenous because it is provided solely by intervention programs and second that fixed effects and the difference-in-difference procedure would be sufficient to purge any remaining confounding factors from the estimation. This argument can be tested to some extent. First, there may be concern that community fixed effects in the analysis of child mortality are insufficient to address the endogeniety of vitamin A supplementation. To examine the strength of community fixed effects I conduct the analysis of ITN use first with community fixed effects and then with mother fixed effects and compare the estimates.

	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)
Vitamin A	0.007*	0.017**	0.009	0.015**	0.008*	0.016**	-0.005	-0.014**	0.015**	0.026**	-0.001	-0.016**
	(0.004)	(0.005)	(0.005)	(0.007)	(0.004)	(0.005)	(0.004)	(0.005)	(0.006)	(0.006)	(0.005)	(0.006)
Vitamin A*Female			-0.003	0.004								
			(0.007)	(0.008)								
Vitamin A*Mother Decides					-0.002	0.002						
					(0.010)	(0.008)						
Vitamin A*Primary Education							0.020**	0.035**				
·							(0.009)	(0.007)				
Vitamin A*Secondary Education							0.046***	0.096**				
·							(0.016)	(0.013)				
Vitamin A*Siblings							, , ,	. ,	-0.005**	-0.006**		
(after the first sibling)									(0.002)	(0.002)		
Vitamin A*Average Wealth										. ,	0.018**	0.023**
e											(0.008)	(0.008)
Vitamin A*High Wealth											0.013	0.073**
C											(0.010)	(0.010)
Community Fixed Effects	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No
Household Fixed Effects	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Observations	29500	29500	29500	29500	29488	29488	29500	29500	29500	29500	29488	29488
Mean	0.074	0.074	0.074	0.074	0.074	0.074	0.074	0.074	0.074	0.074	0.074	0.074

Table 5: Comparison of Community and Household Fixed Effects for ITN Provision

Notes:

1. Models with community fixed effects include controls for mother's education, mother's literacy, mother's age, mother's age at first birth, birth in the last year, marital status, if father is present, household wealth index, number of living children, age of the child, whether the child is first born, and gender of the child.

2. Models with household fixed effects include controls for age of the child, age of the child squared, whether the child is first born, gender of the child, and the birth weight of the child.

3. Standard (clustered on Community for community fixed effects and Household for household fixed effects) errors are given in parentheses

4. \* indicates statistical significance at the 10% level, \*\* indicates statistical significance at the 5% level

Any differences between these estimates will relate to confounding factors for ITN use at the household level. Assuming that these factors will have a similar effect on child mortality, these comparisons will provide insight into the bias remaining in the child mortality analysis. Table 5 presents these comparisons. These demonstrate that any selection that remains after controlling for community effects is negative. This can be seen in the fact that estimated ITN effects are higher when mother fixed effects are used. This implies that estimates of the effect of vitamin A on child mortality using community fixed effects serve as lower bounds of the true effect.

The primary threat to the validity of the difference-in-difference approach relates to unobserved child health that may vary systematically across groups (i.e. child gender, mother's bargaining power, mother's education, number of siblings, and household wealth), but within vitamin A status. The most likely confounder from a theoretical view is unmeasured child health. It may be that vitamin A is correlated with child mortality through other aspects of health. If this unmeasured health varies systematically by group, then the difference-in-difference procedure will not control for this bias. To address this issue, I estimate the model of child mortality with and without observable measures of health status: the number of other boy and girl children that have ever died in the household and child birth weight. Table 6 reports results from this sensitivity analysis. These results demonstrate that the inclusion or exclusion of these controls does not significantly affect the estimates of the effect of vitamin A on child mortality. More importantly, both measures of health are strong predictors of mortality, implying that their inability to affect the estimates of vitamin A impacts stems from not being correlated with vitamin A responses rather than not having an effect on mortality at all.

	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)
Vitamin A	-0.008**	-0.008**	-0.009**	-0.009**	-0.008**	-0.008**	-0.004**	-0.005**	-0.010**	-0.010**	-0.006**	-0.006**
	(0.001)	(0.001)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Vitamin A*Female			0.001	0.001								
			(0.003)	(0.003)								
Vitamin A*Mother Decides					-0.002	-0.002						
					(0.004)	(0.004)						
Vitamin A*Primary Education					` ´		-0.010**	-0.008**				
,							(0.003)	(0.003)				
Vitamin A*Secondary Education							-0.014**	-0.012**				
							(0.005)	(0.005)				
Vitamin A*Siblings									0.001	0.001*		
(after the first sibling)									(0.001)	(0.001)		
Vitamin A*Average Wealth									()		-0.001	-0.002
											(0.004)	(0.004)
Vitamin A*High Wealth											-0.005	-0.006*
											(0.004)	(0.004)
Child Health Status Controls	No	Yes										
Observations	49764	49764	49764	49764	49764	46513	49764	49764	49764	49764	49764	49764
Mean	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04

Table 6: Sensitivity Analysis of the Effect of Vitamin A Supplementation on Mortality

Notes:

1. All models include controls for mother's education, mother's literacy, mother's age, mother's age at first birth, birth in the last year, marital status, if father is present, household wealth index, number of living children, age of the child, whether the child is first born, and gender of the child.

2. Child health status controls are number of dead sons, number of dead daughters, and the birth weight of the child.

3. All models include community fixed effects

4. Standard (clustered on Community) errors are given in parentheses

5. \* indicates statistical significance at the 10% level, \*\* indicates statistical significance at the 5% level

This is evidence that there is little selection on observable variables, strengthening the plausibility of the difference-in-difference assumption that there is no selection on unobservable variables. The differential effect by mother's education is significant both with and without controls for health status and the point estimates are comparable. The differential effect by number of siblings is only significant when controls for health status are included, but this is primarily the result of a decrease in precision as the point estimates are identical with and without controls. The differential effect by household wealth is slightly smaller when controls for health are not included, which leads the estimate to be insignificant, but the estimates are still very similar in magnitude. In any case, this is evidence of negative selection that would bias estimates downward, implying that they are a lower bound.

In the analysis of ITN use, there again may be concern over bias resulting from unobserved child health status that differs systematically by group. To test this I again perform sensitivity analysis on the inclusion of controls for child health status, in this case birth weight. Table 7 presents results from this sensitivity analysis. These results demonstrate that the inclusion of birth weight does not alter the estimated effect of vitamin A on child ITN use. The estimates of the differential effects of vitamin A status on ITN use by mother's education, number of siblings, and household wealth are all identical with and without controls for birth weight. More importantly, the estimated effect of birth weight on ITN provision is statistically significant. This is strong evidence that unobserved child health status will not bias estimates of the effect of vitamin A on ITN use.

	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)
Vitamin A	0.017**	0.017**	0.015**	0.015**	0.016**	0.016**	-0.014**	-0.014**	0.026**	0.026**	-0.016**	-0.016**
	(0.005)	(0.005)	(0.007)	(0.007)	(0.005)	(0.005)	(0.005)	(0.005)	(0.006)	(0.006)	(0.006)	(0.006)
Vitamin A*Female			0.004	0.004								
			(0.008)	(0.008)								
Vitamin A*Mother Decides					0.002	0.002						
					(0.008)	(0.008)						
Vitamin A*Primary Education							0.035**	0.035**				
							(0.007)	(0.007)				
Vitamin A*Secondary Education							0.096**	0.096**				
							(0.013)	(0.013)				
Vitamin A*Siblings									-0.006**	-0.006**		
(after the first sibling)									(0.002)	(0.002)		
Vitamin A*Average Wealth											0.023**	0.023**
-											(0.008)	(0.008)
Vitamin A*High Wealth											0.073**	0.073**
C C											(0.010)	(0.010)
Child Birth Weight Controls	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Observations	29500	29500	29500	29500	29488	29488	29500	29500	29500	29500	29488	29488
Mean	0.074	0.074	0.074	0.074	0.074	0.074	0.074	0.074	0.074	0.074	0.074	0.074

Table 7: Sensitivity Analysis of the Effect of Vitamin A Supplementation on ITN Provision, Birth Weight

Notes:

1. All models include controls for mother's education, mother's literacy, mother's age, mother's age at first birth, birth in the last year, marital status, if father is present, household wealth index, number of living children, age of the child, whether the child is first born, and gender of the child.

2. All models include household fixed effects

3. Standard (clustered on Household) errors are given in parentheses

4. \* indicates statistical significance at the 10% level, \*\* indicates statistical significance at the 5% level

In regressions (not reported) I find that the within household participation decision is influenced by age and birth order, so to guarantee that systematic variation in these characteristics is not biasing estimated vitamin A effects, I perform sensitivity on the inclusion of these controls. Table 8 presents results from this analysis. These results demonstrate that the inclusion of age and birth order controls does not alter the estimated effects of vitamin A status on ITN use. Importantly, age and birth order significantly affect ITN use. This is strong evidence that unobserved child characteristics in general will not bias estimates of the effect of vitamin A on ITN use.

	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)
Vitamin A	0.017**	0.017**	0.015**	0.015**	0.017***	0.016**	-0.014**	-0.014**	0.028**	0.026**	-0.015**	-0.016**
	(0.005)	(0.005)	(0.007)	(0.007)	(0.005)	(0.005)	(0.005)	(0.005)	(0.006)	(0.006)	(0.006)	(0.006)
Vitamin A*Female			0.004	0.004								
			(0.008)	(0.008)								
Vitamin A*Mother Decides					-0.000	0.002						
					(0.008)	(0.008)						
Vitamin A*Primary Education							0.036**	0.035**				
-							(0.007)	(0.007)				
Vitamin A*Secondary Education							0.098**	0.096**				
2							(0.013)	(0.013)				
Vitamin A*Siblings							· · · ·	· · · ·	-0.007**	-0.006**		
(after the first sibling)									(0.002)	(0.002)		
Vitamin A*Average Wealth									( )	()	0.023**	0.023**
· · · · · · · · · · · · · · · · · · ·											(0.008)	(0.008)
Vitamin A*High Wealth											0.073**	0.073**
											(0,010)	(0.010)
Child Age and Birth Order Controls	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Observations	29500	29500	29500	29500	29488	29488	29500	29500	29500	29500	29488	29488
Mean	0.074	0.074	0.074	0.074	0.074	0.074	0.074	0.074	0.074	0.074	0.074	0.074

Table 8: Sensitivity Analysis of the Effect of Vitamin A Supplementation on ITN Provision, Age and Birth Order

Notes:

1. All models include controls for mother's education, mother's literacy, mother's age, mother's age at first birth, birth in the last year, marital status, if father is present, household wealth index, number of living children, age of the child, whether the child is first born, and gender of the child.

2. All models include household fixed effects

3. Standard (clustered on Household) errors are given in parentheses

4. \* indicates statistical significance at the 10% level, \*\* indicates statistical significance at the 5% level

# 4. Empirical Analysis: Prevention versus Treatment

The model of parental decisions between disease prevention and medical treatment developed in section 2, predicts that high levels of mortality from a competing disease risk will cause prevention of other diseases to be low, price elasticities for that prevention to be high, and spending on medical treatment to be high. The competing diseases of diarrhea and malaria provide an opportunity for testing the predictions of the model. The WHO estimates that deaths from diarrhea account for 25 percent of total child mortality in sub-Saharan Africa (WHO 2010a). Substantial, clinical research has shown that vitamin A improves immune response to diarrhea infections (Villamor and Fawzi 2005).<sup>5</sup> While 42 percent of children in Africa do not obtain sufficient vitamin A through diet (Aguayo and Baker 2005), clinical research has shown that mega-dose vitamin A supplements can supply children with enough vitamin A to reduce mortality from diarrhea infections for six months (Villamor and Fawzi 2005). All available evidence suggests that parents do not invest in preventing child death from diarrhea with vitamin A supplements outside of programs because of a lack of availability, a lack of knowledge, or some other factor (Klemm et al. 2007; Sserunji and Harvey 2005; MOST Project 2010). This has led governments, NGOs, and international organizations to implement vitamin A distribution programs in developing countries (World Bank 2004). For example, the WHO has included distribution of vitamin A supplements with its national immunization days (WHO 1999) and USAID has implemented stand alone distribution programs (Houston 2003). In these distribution programs, health workers administer a pill containing a dose of vitamin A that will last for 6 months. The long-

<sup>&</sup>lt;sup>5</sup> Villamor and Fawzi (2005) note that there is no biological basis for vitamin A improving immune response to malaria and in general, studies have found vitamin A to be ineffective at reducing malaria mortality. They cite six field studies where vitamin A has no effect on malaria outcomes and one field study that found vitamin A to reduce malaria incidence.

lasting effects are possible because vitamin A is fat soluble and thus mega-doses remain in the body for extended periods. These distribution programs will cause an exogenous decrease in mortality from diarrhea and this change in competing risk of death can be used to assess the predictions of the model.

Parents are likely to understand the expected benefits of vitamin A supplementation due to education components associated with supplementation programs (Sserunji and Harvey 2005; National Nutrition Program 2008; MOST Project 2010). However, this is not a prerequisite for supplementation programs to identify the model, because the benefits of supplementation will reveal themselves to parents over time due to the frequency and severity of diarrhea in Africa. For example, in the sample used in this analysis, 20 percent of children had an episode of diarrhea in the past two weeks. Extrapolating from this, the average child will have more than 2 episodes of diarrhea over a 6 month period (the time period covered by a vitamin A supplement). Thus, children will likely contract diarrhea more than once after receiving supplementation and when they do, their parents will be able to observe the improved immune response (whether they know that it is due to vitamin A or not) because of the extreme symptoms of severe diarrhea such as blood in stools. Previous research has tested the accuracy of parent observations of child symptoms by comparing parental reports from interviews to medical exams and found that parents accurately observe severity of illness in their children, especially for diarrhea (Kalter et al. 1991).

Malaria is an example of a large competing disease risk for diarrhea. The WHO estimates that deaths from malaria account for 23 percent of total child mortality in sub-Saharan Africa (WHO 2010a). In most cases, ITNs are not distributed freely like vitamin

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A supplements, but are instead purchased through private or public centers, likely because, in contrast to vitamin A, the benefits of ITNs are well known among the population and nets are available for purchase.

Based on the model, the transfer of vitamin A supplements will induce an increase in investment in malaria prevention and reduce price sensitivity for prevention. To test these hypotheses, I estimate the demand for ITNs given by equation (16). A fully specified conditional demand function of this type includes all other prices and production parameters (Pollack 1969). However, as is common, I do not have all necessary values, most notably the price of treatment and the parameters of the health production function for malaria prevention. Thus, in estimating this demand function, I include controls for general prices within a community with community fixed effects and observable determinants of health production such as child anthropometrics and mother's education. Specifically, I estimate the following regression equation:

(39)  $ITN_i = \beta_0 + \beta_1 A_i + \beta_2 \operatorname{Price}_m + \beta_3 A_i * \operatorname{Price}_m + \beta_4 X_i + \beta_5 X_m + \eta_i + \varepsilon_i$ 

In this equation,  $ITN_i$  equals one if child i slept under an ITN the previous night and zero otherwise,  $A_i$  equals one if child i received a vitamin A supplement in the past six months and zero otherwise,  $Price_m$  is the price of ITNs faced by household m,  $X_i$  is a vector of child specific determinants of health production such as height-for-age, weight-for-age, age, and gender,  $X_m$  is a vector of household specific determinants of health production such as mother's education, sanitation resources, and wealth,  $\eta_j$  is a community fixed effect, and  $\varepsilon_i$  is a random error term.

The coefficients  $\beta_1$  and  $\beta_3$  in equation (39) indicate the extent to which households respond to the vitamin A supplementation by investing in complementary disease prevention (ITNs). The coefficient  $\beta_1$  is the base level of response to complementarities when the price of ITN equals zero and  $\beta_3$  is the extent to which this response is lower at higher prices.<sup>6</sup>

The coefficients  $\beta_2$  and  $\beta_3$  in equation (39) indicate the price effect for those with and without vitamin A supplementation. The coefficient  $\beta_2$  is the base price effect for ITNs and  $\beta_3$  is the additional price effect for children who received vitamin A supplementation. To determine whether the price effect for those with vitamin A is lower than for those without, I construct price elasticities from the coefficients  $\beta_1$ ,  $\beta_2$ , and  $\beta_3$  at the mean level of ITN use:

(40) 
$$E_{i} = \frac{(\beta_{2} + \beta_{3} * A_{i}) * mean(Price_{m})}{mean(ITN \mid A_{i} = 0) + \beta_{1} * A_{i}}$$

Note that in equation (40) vitamin A supplementation enters in both the numerator and denominator. From the model, it is clear that vitamin A supplementation will increase the level of ITN use as well as affect the level of price response. Thus, to compute the percent change in ITN use from a price increase, we must add the level effect of vitamin A to the denominator, meaning that the sign of  $\beta_3$  alone does not indicate whether those with vitamin A are less price sensitive than those without. In fact, from the discussion above, we expect that  $\beta_3$  will be negative because higher prices will lower household responses to complementarities. At the same time however, the price elasticity may be lower for those with vitamin A depending on the other parameters. Thus, the difference in price sensitivity is jointly determined by  $\beta_1$ ,  $\beta_2$ , and  $\beta_3$ .

While concerns over the endogeneity of vitamin A supplementation are diminished in this context because vitamin A supplementation is generally not available

<sup>&</sup>lt;sup>6</sup> This is a similar idea to that of Yarnoff (2011), which found that higher shadow prices for ITNs decreased household response to complementarities.

for purchase and is obtained from an external group on a national distribution day, there is still concern over any vitamin A supplements obtained outside of a national distribution day. Most notably, some clinics are equipped with vitamin A supplements to distribute to children that were missed on the national distribution day when they come in for treatment or a check up. In this case, supplementation may be correlated with health seeking behavior or overall child health. I am able to test for these confounders by estimating the differential effect of receiving a supplement on a national distribution day versus receiving it from a clinic.

There also may be concern over vitamin A program targeting (administrative selection) as well as factors that influence a household's decision to participate in a supplementation program. Additionally, concern over the endogeneity of prices is strong, because prices vary based on supply and demand factors within an area. For example, areas with higher ITN use will likely have higher prices due to higher demand from some third factor such as health knowledge.<sup>7</sup> In fact, regressions (not reported here) show that prices are positively correlated with household wealth, mother's education, and literacy, all factors that will be positively correlated with demand for health.

To account for the endogeneity of price as well as any vitamin A endogeneity relating to administrative selection or participation decisions, I estimate a model with mother fixed effects. Specifically, I estimate the following regression equation:

(41) 
$$ITN_i = \beta_0 + \beta_1 A_i + \beta_3 A_i * \operatorname{Pr} ice_m + \beta_4 X_i + \eta_m + \varepsilon_i$$

Where all variables are as before and  $\eta_m$  is a mother fixed effect. Note that  $\beta_2 Price_m$  is omitted from equation (41) because it does not vary within a household. Thus, while this

<sup>&</sup>lt;sup>7</sup> This assumes an upward sloping supply curve, a point argued for by Simon et al. (2001).

equation produces more reliable estimates of the effect of vitamin A and the extent to which this effect differs by price, it will not produce estimates of the base price effect. In order to evaluate the difference in price elasticities for supplemented children, I will evaluate equation (40) (the price elasticity of demand) at a range of values for  $\beta_2$  (the base price effect).

Controlling for mother fixed effects addresses much of the concern over price endogeneity as this specification controls for unmeasured, family-specific demand and supply factors as well as all relevant complement and substitute prices. Mother fixed effects also control for any vitamin A endogeneity by controlling for all family-specific, unmeasured factors such as health input prices, health resources, and health behaviors that may confound the estimated effect of vitamin A supplementation on ITN use. These include access to and prices for prevention and treatment services, parental health knowledge, and preferences for health care as well as other potentially confounding factors.

The mother fixed effect analysis is identified by households where one child received a vitamin A supplement and a sibling did not (10 percent of the sample). For the analysis to be unbiased, it must be the case that supplementation is missed randomly after controlling for mother effects. The only confounding factors that remain are those that are child specific, because while mother fixed effect analysis covers all confounding factors relating to household resources and health behaviors, there may still be child specific factors that will bias estimates. However, regressions (reported in table 9) of vitamin A supplementation on child characteristics (gender, age, height-for-age, and weight-for-age) and mother fixed effects indicate that no observable child characteristics are significant predictors of receiving a vitamin A supplement. All coefficients are small and statistically insignificant. This lack of observable correlation provides evidence that unobserved child characteristics will be uncorrelated with the effect of vitamin A supplementation since unobserved measures of important characteristics such as child health will be correlated with observable health characteristics (i.e. height and weight). Further, past analysis of nutrition programs provides evidence that children randomly miss distribution days. For example, in a study where researchers administered micronutrient supplements to children in their homes daily, a given child was randomly not present 15 percent of the time (Begin et al. 2008).

Male	0.003
	(0.01)
Age	0.001
	(0.001)
Height-for-Age Z-score	0.0003
	(0.0034)
Weight-for-Age Z-score	-0.003
	(0.004)
Mean	0.68
Observations	7017
Notes:	
1. Standard errors (clustered on the m	other) are given in parentheses
<ol><li>Model includes mother fixed effect</li></ol>	ts
3. *** p<0.01, ** p<0.05, * p<0.1	

 Table 9: Determinants of Vitamin A Supplementation Controlling for

 Mother Fixed Effects

The third prediction of the model is that vitamin A supplementation will reduce the amount of spending on medical treatment for malaria. To test this hypothesis, I estimate the demand for malaria treatment given by equation (18). I measure treatment as whether the child received anti-malarial drugs during an episode of malaria in the past two weeks. Again, a fully specified conditional demand function includes all relevant prices and production parameters and I do not have all of these values. Thus, I again include mother fixed effects to control for all prices and health production parameters constant within a family. Additionally, whether the child received the supplement from a clinic is of greater concern here, because it may be correlated with a higher propensity to go to clinics, biasing estimates on seeking treatment. Thus, I will control for the location where the child received the supplement. Specifically, I estimate the following system of regression equations:

(42) 
$$Malaria_i = \alpha_0 + \alpha_1 A_i + \alpha_2 ITN_i + \alpha_3 X_i + \eta_m + \varepsilon_i$$

(43) (*Treatment*<sub>i</sub> | *Malaria*<sub>i</sub> = 1) =  $\gamma_0 + \gamma_1 A_i + \gamma_2 X_i + \gamma_2 IMR_i + \eta_m + \varepsilon_i$ 

Where *Malaria*<sup>*i*</sup> equals one if child i had malaria symptoms in the past two weeks and zero otherwise, *Treatment*<sup>*i*</sup> equals one if child i received treatment for the malaria symptoms,  $A_i$  equals one if child i received a vitamin A supplement in the past six months and zero otherwise and is divided by where the supplement was received (national distribution day or health clinic), *ITN*<sup>*i*</sup> equals one if child i slept under an ITN the previous night and zero otherwise, *IMR*<sup>*i*</sup> is the inverse Mills ratio generated from the predicted probability of having malaria symptoms in equation (42),  $X_i$  is a vector of other child specific determinants of demand for health inputs (height-for-age, weight-for-age, age, and gender),  $\eta_m$  is a mother fixed effect, and  $\varepsilon_i$  is a random error term.

Equation (42) is a latent variable model of the probability of having malaria symptoms. Equation (43) will only be observed if equation (42) is greater than some critical value. In order to incorporate mother fixed effects in this analysis, I estimate both stages linearly, with ITN serving as an identifier. ITN is used as an identifier here, because it will affect the probability of getting malaria, but not getting treatment.  $\gamma_1$  is the parameter of interest in equation (43), because it indicates how households change their treatment spending when a child is sick in response to vitamin A supplementation. The model predicts that  $\gamma_1$  will be negative.

# Data

I use data on ITN use, ITN price, vitamin A supplementation, and medical treatment from the Multiple Indicator Cluster Survey (MICS) from seven sub-Saharan African countries. Specifically I use the following countries and years: Burkina Faso 2006, Burundi 2005, Cameroon 2006, Central African Republic 2006, Cote D'Ivoire 2006, Gambia 2006, and Guinea-Bissau 2006. I use these countries and years, because data on ITN use and price are available in these places at these times.

The MICS is a nationally representative survey of ever-married women aged 15 to 49. The survey collects retrospective data on the health of all living children under the age of five as well as data on household and family characteristics. Most importantly, it contains information on whether a child received a vitamin A supplement in the past six months, slept under an ITN the previous night, had malaria symptoms in the past two weeks, and received anti-malarial drugs as treatment for those malaria symptoms (the WHO-recommended treatment for malaria symptoms). Additionally, the survey contains data on the price households paid for ITNs. Households that did not own an ITN (and thus do not have data recorded for price paid), were assigned the average price paid in their community. I convert these prices to dollars using exchange rates from the relevant time period and adjust for inflation with the CPI. I perform all analysis on a sample of children between the ages of 6 months and 3 years. I limit the sample in this way because this is the age group for which vitamin A supplementation and ITN use are most effective and for which diarrhea and malaria are most deadly. For comparability between community-fixed-effect and mother-fixed-effect analysis, I further limit the sample to households with two or more children in this age group. This results in a sample of approximately 7,000 children for the ITN analysis and first stage of the treatment analysis and a sample of approximately 860 children for the second stage of treatment analysis.

	Vitamin A in past 6 month					
	No	Yes	Difference			
Dependent Variables						
ITN	0.09	0.11	0.02**			
Malaria	0.21	0.19	-0.02**			
Anti-Malarials	0.44	0.39	-0.05**			
Independent Variables						
ITN Price	5.49	4.73	-0.76**			
Male	0.50	0.52	0.02			
Age in Months	20.18	20.82	0.64**			
Height-for-Age Z-score	-0.78	-0.92	-0.14*			
Weight-for-Age Z-score	-0.54	-0.89	-0.36**			
Wealth Category 1 (Lowest)	0.002	0.002	0.000			
Wealth Category 2	0.23	0.21	-0.02*			
Wealth Category 3	0.21	0.21	-0.004			
Wealth Category 4	0.25	0.24	-0.01			
Wealth Category 5 (Highest)	0.18	0.20	0.02**			
Number of Kids Under 5	2.38	2.45	0.08**			
Flush Toilet	0.16	0.29	0.14**			
Pit Latrine	0.01	0.01	0.00			
Bucket or Similar for Toilet	0.83	0.70	-0.14**			
No Education	0.68	0.64	-0.04**			
Primary Education	0.22	0.21	-0.01			
Secondary Education or Greater	0.10	0.15	0.05**			
Notes: 1. ** p<0.05, * p<0.1						

**Table 10: Descriptive Analysis** 

Table 10 presents descriptive statistics from this sample by vitamin A status. This descriptive analysis shows that, as the model predicts, children that received a vitamin A supplement in the past 6 months are more likely to have slept under an ITN and less likely to have received anti-malarial drugs when sick. However, these summary statistics raise some concern over confounding factors biasing this relationship because many household and child characteristics are also significantly associated with vitamin A supplementation. This demonstrates the necessity of regression controls for producing unbiased estimates of the effects of vitamin A supplementation. As table 9 (above) demonstrated, no child characteristics are associated with vitamin A supplementation after controlling for mother fixed effects.

### Results

Table 11 presents results from regression analysis of ITN use. Columns 4 through 6 contain results from community fixed effect regressions. Column 4 contains results from a model with a full set of controls while columns 5 and 6 contain results from models that provide sensitivity analysis by child characteristics (age, gender, height-for-age, and weight-for-age) and household resources (proxied by household wealth, mother's education, and household sanitation facilities). Estimates in column 4 demonstrate that the base effect of vitamin A is to increase ITN use by 3.7 percentage points, representing a 41 percent increase from the mean ITN use of children who do not receive supplementation. This effect does not differ in the sensitivity analyses, providing support for the argument that vitamin A supplementation is exogenous. The results also show that this estimate is moderated by the price of ITNs to the extent that a one dollar price increase decreases ITN use by 0.2 percentage points (5 percent of the base effect of

vitamin A), although this effect is not statistically significant. The endogeneity of price can be seen here in the positive, but small and statistically insignificant, coefficient on price.<sup>8</sup>

	Mot	her Fixed Eff	fects	Comm	unity Fixed	<b>Effects</b>
	(1)	(2)	(3)	(4)	(5)	(6)
Vitamin A	0.039**	0.039**	0.039**	0.034**	0.036**	0.038**
	(0.019)	(0.019)	(0.019)	(0.016)	(0.016)	(0.016)
Vitamin A*Price	-0.003*	-0.003*	-0.003*	-0.002	-0.002	-0.002
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Price				0.003	0.003	0.004
				(0.003)	(0.003)	(0.003)
Household Resources	No	No	No	Yes	Yes	No
Child age and gender	Yes	Yes	No	Yes	No	No
Child height-for-age and weight-for-age	Yes	No	No	Yes	No	No
Mean (unsupplemented)	0.07	0.07	0.07	0.07	0.07	0.07
Observations	7017	7017	7017	7017	7017	7017
Notes:						

### Table 11: Effect of Vitamin A Supplementation on ITN Use

1. Standard errors (clustered on the mother) are given in parentheses

2. Household characteristics include: wealth, mother's education, type of sanitation facilities, type of water source

3. All community fixed effect models include controls for number of children in household, and number of sick adults in household

4. \*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Columns 1 through 3 of Table 11 present results from a mother fixed effect analysis. Column 1 contains estimates from a model with a full set of child specific controls while columns 2 and 3 contain estimates from sensitivity analyses by child specific health status (proxied by height-for-age and weight-for-age) and child age and gender. The estimates are identical for all three columns, demonstrating that results are not sensitive to observable child characteristics and implying that they will be insensitive to unobservable characteristics as well. The results demonstrate that the base effect of vitamin A is to increase ITN use by 3.9 percentage points (43 percent of mean ITN use

<sup>&</sup>lt;sup>8</sup> This may also be the result of low price variation within community. The mean within-community standard deviation is 0.47 and the median within-community standard deviation is zero.

for the unsupplemented). This is similar to the estimates from the community fixed effect analysis, implying that unobserved household resources and health behaviors do not bias the estimated effect of vitamin A supplementation (further evidence of the exogeneity of vitamin A supplementation). The results also show that this estimate is reduced by the price of nets to the extent that a one dollar price increase decreases ITN use by 0.31 percentage points (8 percent of the base effect of vitamin A) and this effect is statistically significant.

Moth	her Fixed Ef	ffects	Com	Community Fixed Effects		
(1)	(2)	(3)	(4)	(5)	(6)	
0.039*	0.039*	0.039*	0.025*	0.027*	0.030**	
(0.020)	(0.020)	(0.020)	(0.016)	(0.016)	(0.016)	
0.037	0.037	0.037	0.068***	0.070***	0.071***	
(0.026)	(0.026)	(0.026)	(0.023)	(0.023)	(0.023)	
-0.003	-0.003	-0.003	-0.001	-0.001	-0.001	
(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	
-0.004	-0.004	-0.004	-0.007***	-0.007***	-0.007***	
(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	
			0.003	0.003	0.004	
			(0.003)	(0.003)	(0.003)	
No	No	No	Yes	Yes	No	
Yes	Yes	No	Yes	No	No	
Yes	No	No	Yes	No	No	
0.07	0.07	0.07	0.07	0.07	0.07	
7017	7017	7017	7017	7017	7017	
	Moti (1) 0.039* (0.020) -0.003 (0.002) -0.004 (0.003) No Yes Yes Q.07 7017	Mother Fixed Ef           (1)         (2)           0.039*         0.039*           (0.020)         (0.020)           0.037         0.037           (0.026)         (0.026)           -0.003         -0.003           (0.002)         (0.002)           -0.004         -0.004           (0.003)         (0.003)	Mother Fixed Effects           (1)         (2)         (3)           0.039*         0.039*         0.039*           (0.020)         (0.020)         (0.020)           0.037         0.037         0.037           (0.026)         (0.026)         (0.026)           -0.003         -0.003         -0.003           (0.002)         (0.002)         (0.002)           -0.004         -0.004         -0.004           (0.003)         (0.003)         (0.003)           No         No         No           Yes         Yes         No           Yes         No         No           0.07         0.07         0.07           7017         7017         7017	Mother Fixed Effects         Comm           (1)         (2)         (3)         (4)           0.039*         0.039*         0.039*         0.025*           (0.020)         (0.020)         (0.020)         (0.016)           0.037         0.037         0.037         0.068***           (0.026)         (0.026)         (0.023)         -0.003           -0.003         -0.003         -0.001         (0.002)           (0.002)         (0.002)         (0.002)         (0.002)           -0.004         -0.004         -0.004         -0.003           -0.003         (0.003)         (0.003)         (0.003)           0.003         (0.003)         (0.003)         0.003           0.003         (0.003)         (0.003)         0.003           0.003         (0.003)         0.003         0.003           0.003         No         Yes         Yes           Yes         Yes         No         Yes           Yes         No         No         Yes           0.07         0.07         0.07         0.07           7017         7017         7017         7017	Mother Fixed Effects         Community Fixed Effects           (1)         (2)         (3)         (4)         (5)           0.039*         0.039*         0.039*         0.025*         0.027*           (0.020)         (0.020)         (0.020)         (0.016)         (0.016)           0.037         0.037         0.037         0.068***         0.070***           (0.026)         (0.026)         (0.023)         (0.023)         (0.023)           -0.003         -0.003         -0.003         -0.001         -0.001           (0.002)         (0.002)         (0.002)         (0.002)         (0.002)           -0.004         -0.004         -0.007***         -0.007***           (0.003)         (0.003)         (0.003)         (0.003)           .         .         .         .         .           .         .         .         .         .         .           .         .         .         .         .         .         .           .         .         .         .         .         .         .         .           .         .         .         .         .         .         .         .	

Table 12: Vitamin A Supplementation on ITN Use, by Source of Vitamin A

1. Standard errors (clustered on the mother) are given in parentheses

2. Household characteristics include: wealth, mother's education, type of sanitation facilities, type of water source

All community fixed effect models include controls for number of children in household, and number of sick adults in household,
 \*\*\* p<0.01, \*\* p<0.05, \* p<0.1</li>

Table 12 presents estimates of the effect of vitamin A supplementation by place of receipt. Columns 4 through 6 present estimates from community fixed effect regressions. Column 4 contains results from a model with a full set of controls while columns 5 and 6 contain results from models that provide sensitivity analysis by child characteristics (age, gender, height-for-age, and weight-for-age) and household resources (proxied by household wealth, mother's education, and household sanitation facilities). These estimates demonstrate a significant heterogeneity in the effect of vitamin A by place of receipt with the effect of supplements received from a national distribution day estimated as a 2.8 percentage point increase in ITN use and the effect of supplements received at a clinic estimated at a 7 percentage point increase in ITN use.

Columns 1 though 3 of Table 12 present results from a mother fixed effect analysis and demonstrate that this vitamin A heterogeneity is being driven entirely by unobserved household health behaviors. Column 1 contains estimates from a model with a full set of child specific controls while columns 2 and 3 contain estimates from sensitivity analyses by child specific health status (proxied by height-for-age and weightfor-age) and child age and gender. The estimated effect of vitamin A supplementation is virtually identical whether received from a national distribution day (3.9 percentage points) or a clinic (3.7 percentage points), although only the effect for national distribution days is statistically significant due to low statistical power.

While mother fixed effect models produce plausibly unbiased estimates of the effect of vitamin A supplementation and the interaction between vitamin A and price, they cannot produce estimates of the base price effect. Thus, to estimate the differential elasticities for supplemented and un-supplemented children, I evaluate equation (40) at a range of values for the parameter  $\beta_2$  (the base price effect). Table 13 presents estimates of elasticities for a range of  $\beta_2$  values. The first column contains the overall average elasticity weighted by the proportion of the sample who received supplemented children, (68 percent), the second column contains elasticity estimates for un-supplemented children,
and the third column contains estimates for children who received a vitamin A supplement. These estimates show that price elasticities are significantly smaller for supplemented children. For example, at the overall price elasticity of -1 ( $\beta_2 = -0.02$ ) found by Cohen and Dupas (2010), the price elasticity for supplemented children is 20 percent lower than the price elasticity for un-supplemented children. At the price elasticity of -1.8 ( $\beta_2 = -0.04$ ) found by Dupas (2009), the price elasticity for supplemented children. At the price elasticity of -3.8 ( $\beta_2 = -0.07$ ) found by Hoffman et al (2008), the price elasticity for supplemented children. At the price elasticity of -3.8 ( $\beta_2 = -0.07$ ) found by Hoffman et al (2008), the price elasticity for unsupplemented children is 27 percent lower than the price elasticity for unsupplemented children. These results demonstrate that ITN price elasticity is significantly lower for children who received vitamin A supplementation.

	Frice Elusicity			
		Vitamin A supplementation		
Base Price Response	Overall	No	Yes	
-0.01	-0.52	-0.55	-0.50	
-0.02	-0.95	-1.10	-0.88	
-0.03	-1.39	-1.65	-1.27	
-0.04	-1.83	-2.20	-1.65	
-0.05	-2.26	-2.76	-2.04	
-0.06	-2.70	-3.31	-2.42	
-0.07	-3.14	-3.86	-2.81	
-0.08	-3.57	-4.41	-3.19	
-0.09	-4.01	-4.96	-3.58	
-0.10	-4.45	-5.51	-3.96	

 Table 13: Range of Elasticities from Mother Fixed Effect Analysis

Drigo Elasticity

Table 14 presents results from a mother fixed effect analysis of the effect of vitamin A supplementation on the probability of having malaria symptoms. This is the first stage in a two stage model of the effect of vitamin A supplementation on the decision to seek medical treatment when a child is ill and will be used to construct the

inverse Mills ratio for the second stage. Column 1 contains estimates from a model with a full set of controls and columns 2 and 3 contain sensitivity analyses by child specific health status (proxied by height-for-age and weight-for-age) and child age and gender. In all cases the effect of vitamin A is separated by place of receipt. The estimated effect of vitamin A received on national distribution days is virtually zero. The effect of vitamin A received from a clinic however is positive, but small (6 percent of mean malaria probability). Additionally, ITN use reduces the probability of malaria by 13 percent of the mean. However, none of these estimates are statistically significant.

	Mother Fixed Effects		
	(1)	(2)	(3)
Vitamin A from National Distribution Day	-0.002	-0.001	-0.005
	(0.022)	(0.022)	(0.022)
Vitamin A from Health Facility	0.013	0.013	0.021
	(0.025)	(0.025)	(0.025)
ITN	-0.030	-0.024	-0.025
	(0.031)	(0.031)	(0.031)
Child age and gender	Yes	Yes	No
Child height-for-age and weight-for-age	Yes	No	No
Mean (unsupplemented)	0.23	0.23	0.23
Observations	7017	7017	7017
Notes:			

Table 14: Effect of Vitamin A on Having Malaria Symptoms

Standard errors (clustered on the mother) are given in parentheses
 \*\*\* p<0.01, \*\* p<0.05, \* p<0.1</li>

Table 15 presents results from a mother fixed effect analysis of the effect of vitamin A supplementation on the probability of receiving anti-malarial drugs after contracting malaria. These results demonstrate that vitamin A supplementation received from national distribution days reduces the probability of medical treatment when sick. The point estimates from the model with full controls (column 1) imply that supplementation reduces the probability of treatment by 0.13 percentage points,

representing a 35 percent reduction in mean treatment. These results are insensitive to the inclusion of age, gender, height-for-age, and weight for age, implying that results are not sensitive to observable or unobservable child factors. Additionally, the effect of vitamin A received from a clinic has a positive, but statistically insignificant, effect on medical treatment. This is most likely due to an unobserved higher likelihood of going to a clinic biasing estimates upward for these children.

	Mother Fixed Effects		
	(1)	(2)	(3)
Vitamin A from National Distribution Day	-0.13*	-0.11*	-0.18**
	(0.07)	(0.07)	(0.09)
Vitamin A from Health Facility	0.20	0.20	0.30
	(0.15)	(0.17)	(0.23)
Child age and gender	Yes	Yes	No
Child height-for-age and weight-for-age	Yes	No	No
Mean (unsupplemented)	0.37	0.37	0.37
Observations	861	861	861
Notes:			

Table 15: Effect of Vitamin A on Receiving Anti-Malarial Drugs When Sick

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Standard errors (clustered on the mother) are given in parentheses 2. All models include inverse mills ratio constructed from estimates in table 5

3. \*\*\* p<0.01, \*\* p<0.05, \* p<0.1

## 5. Conclusion

Child mortality is a persistent problem in sub-Saharan Africa that policymakers have up to now been unable to solve. The Millennium Development Goals set the bar for progress at a two-thirds reduction by the year 2015 (UNDP 2011). However, there has been nowhere near sufficient progress to meet this goal. While the child mortality rate in sub-Saharan Africa had fallen from 18 percent in 1990 to 13 percent in 2009, the total number of child deaths has increased from 3,937,000 in 1990 to 3,976,000 in 2009. Additionally, the share of under-five deaths coming from Sub-Saharan Africa increased from 31 percent in 1990 to 50 percent in 2009 (UNICEF et al. 2010).

Policymakers seeking to reduce child mortality and meet this goal have long sought to identify what programs are effective and why, but up to now have been unsuccessful. A common explanation for this failure is that health production is a complicated process that depends on many interrelated health inputs that all involve choices (Rutstein 2002). I thus argue in this dissertation that understanding how households make decisions about investment in interrelated health inputs is essential to improving child health. Here I focus specifically on how households respond to competing disease risks in making health input decisions. I argue that a better understanding of this decision-making process will enable policymakers to answer key questions about why programs are effective in reducing child mortality. Here I have specifically addressed two questions: 1) why does the effect of the same program differ by program site and 2) why do families spend little on disease prevention and have high price sensitivity for it when they spend relatively large amounts on medical treatment.

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I developed a theoretical model of household allocation with competing disease risks for children to demonstrate that household will respond to child health programs by investing in more disease prevention for their children and that this response will differ by determinants of household allocation to children. This differential response will lead to mortality effects that vary by these determinants of household allocation. I developed an additional theoretical model of parents' decisions to invest in disease prevention or spend on medical treatment in the presence of competing disease risks. I demonstrated that high levels of competing disease risks will decrease investment in disease prevention, increase price sensitivity for prevention, and increase the use of medical treatment when sick. I applied these two models to the questions raised above and evaluate them empirically using data from sub-Saharan Africa. I find empirical support for both models.

The importance of competing disease risks in household decision-making about health inputs informs policy more generally as well. First, it implies that an understanding of the overall disease environment is imperative, since high levels of a disease for which prevention is either unknown or unavailable will deter households from investment in known and available forms of prevention for other diseases. Second, it implies that program targeting diseases where prevention is either unknown or unavailable will have magnified effects, since they will induce families to make more private investment in preventing competing diseases. A policy agenda structured with this knowledge of household decision-making could go far in improving child health in Africa.

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