Prevention versus Treatment with Competing Disease Risks

March 2011

Benjamin Yarnoff
Department of Economics
University of Illinois at Chicago
601 South Morgan Street
Chicago, IL 60607
Phone: (224)766-6014
Fax: (312)996-1404
byarno2@uic.edu
Abstract

Three diseases, diarrhea, malaria, and pneumonia, account for 75% of all child deaths in Africa. Thus, it is possible that investments to prevent any one of these diseases depends on the prevalence of the other two; for example, there is little incentive for a family to invest in malaria prevention if their child will die from diarrhea regardless. Using this notion, I develop a model to explain the apparent inconsistent behavior of families in Africa spending relatively large amounts on medical treatment, but investing little in disease prevention and exhibiting substantial price sensitivity for prevention. In my model, investments in prevention for two diseases are complements, and disease prevention and medical treatment are substitutes. Thus, factors causing low levels of diarrhea prevention will cause low levels of malaria prevention, high price sensitive for malaria prevention, and high levels of treatment spending. I empirically test these predictions in the context of malaria and diarrhea in Africa using child vitamin A supplementation programs as an exogenous increase in prevention of diarrhea mortality. The results of this analysis support the predictions of the model, demonstrating that vitamin A supplementation leads to increased investment in malaria prevention, decreased price sensitivity for malaria prevention, and decreased treatment spending.
Introduction

Families in Africa invest little in cost effective methods of disease prevention such as mosquito bed nets and even small price increases cause families to reduce the use of preventive methods dramatically (WHO 2009; Cohen and Dupas 2008; Dupas 2009; Hoffman et al. 2008). However, these families spend substantial amounts on medical treatment and that spending has been shown to be price insensitive (WHO 2009; Gertler and van der Gaag 1990; Bedi et al. 2003; Dzator and Asafu-Adjaye 2004). 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). Despite the greater cost effectiveness of ITNs, in 2008 only 24 percent of children under the age of five slept under an ITN in sub-Saharan Africa, but children received treatment for malaria an average of 1.8 times per year (WHO 2009).

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 with families continuing to rely on medical treatment for maintaining child health. Potential explanations of the apparent inconsistent behavior that focus on imperfect information
or time inconsistent preferences have been shown by empirical analysis to be inadequate (Dupas 2009).

In this article, I develop a model to explain the decision between investing in prevention, in this case ITNs, and waiting to seek treatment. 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. Intuitively, 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.

This model is particularly relevant for Africa because child illness and mortality is dominated by three diseases: diarrhea, malaria, and pneumonia. These three diseases account for 75 percent of child mortality between the ages of 6 months and 5 years (approximately 3.75 million children each year) (WHO 2010a). In this context, each disease makes up a large portion of the competing risk of death and thus significantly influences the risk environment for families making decisions about investments in child health.

I empirically test the predictions of the model using variation in Vitamin A supplementation, which substantial clinical evidence has shown to improve the immune response to diarrheal infections and reduce mortality (Villamor and Fawzi 2005). Unfortunately, it has been estimated that 42 percent of children in Africa are vitamin A deficient because diets are lacking and supplements are not available for purchase (Aguayo and Baker 2005). Thus, to reduce mortality from diarrhea, governments, international organizations, and NGOs have implemented supplement distribution programs. These programs provide a plausible, exogenous
decrease in diarrhea mortality that allows me to examine the effect of this change on malaria prevention and medical treatment spending. I find that, as predicted by the model, a decrease in diarrhea mortality increases investment in malaria prevention (i.e. children sleeping under an ITN), decreases the price elasticity of demand for ITNSs, and decreases the use of medical treatment.

Background

Despite the mounting body of evidence on the effectiveness of ITNs for preventing malaria (estimated at a 20 percent mortality reduction 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 (2008) 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 bed nets 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 its 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.

The literature just reviewed 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. However, these families spend substantial amounts on medical treatment and that spending is price insensitive.

Model

Recent theoretical work by Dow et al. (1999) and Becker (2007) on investments in health in the presence of competing disease risk 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 extend this line of reasoning to include the decision
parents make between investing in prevention and spending on treatment for their children. I develop a model in which parents have the following two-period utility function:

\[
U = u_0(x_0) + B[S_1(p_1, p_2, t)u^h_1(x_1, H_1)] + [(1 - S_1(p_1, p_2, t))u^s_1(x_1)]
\]

where \( u_0 \) is the parent’s utility in the first period, which depends on consumption in that period \( (x_0) \), \( u^h_1 \) is the parent’s utility in the second period if their child survives to that period, \( u^s_1 \) 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, \( S_1 \) is the probability that the child will survive to the second period, \( B \) is the discount rate, \( p_1 \) is the amount of services (effort) used in period 0 to prevent disease 1 in the child, \( p_2 \) is the amount of services (effort) used in period 0 to prevent disease 2 in the child, and \( t \) is the amount of medical treatment purchased or produced for the child.

The utility function is generalizable to \( n \) periods. However, I use two periods here to focus on the critical period for parental investments in child health. For example, the WHO estimates that child mortality between 6 months and 3 years is 2 percent while the mortality rate between 4 and 10 years old 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 parental health input. The model assumes that the parent dies at the end of the second period.\(^1\)

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

---

\(^1\) 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.
(2) \( S_1(p_1, p_2, t) = d_1(p_1)d_2(p_2) + [1 - d_1(p_1)d_2(p_2)]T(t) \)

where \( d_1(p_1) \) is a factor relating to the probability of surviving disease 1, \( d_2(p_2) \) is a factor relating to the probability of surviving disease 2, and \( T(t) \) is the probability of treatment leading to a successful recovery after contracting either or both diseases. The factors \( d_1(p_1) \) and \( d_2(p_2) \) are constructed such that \( d_1(p_1)d_2(p_2) \) is equal to the probability of surviving both diseases and \( [1 - d_1(p_1)d_2(p_2)] \) is the probability of contracting either or both diseases in a manner that would be fatal without treatment.

Parents maximize utility subject to the budget constraint given by:

(3) \( x_o + \frac{x_i}{1+r} + q_1 p_1 + q_2 p_2 + [1 - d_1(p_1)d_2(p_2)] \frac{q_1 T}{1+r} = y_o + \frac{y_1}{1+r} \)

Where \( q_1 \) is the price of preventative services (effort) for disease 1, \( q_2 \) is the price of preventative services (effort) for disease 2, \( q_t \) is the price of treatment and is scaled by the probability of illness, \( y_i \) is income, and \( r \) is the real interest rate. Maximizing utility with respect to parental investments, prevention for disease 1 \( (p_1) \), prevention for disease 2 \( (p_2) \), and treatment \( (t) \) gives the following four first order conditions:

(4) \( \frac{\partial d_1}{\partial p_1}d_2(p_2) \left[(1-T(t))B(u_1 - u_1^*) + \frac{q_1 T}{1+r}\right] = \lambda q_1 \)

(5) \( d_1(p_1) \frac{\partial d_2}{\partial p_2} \left[(1-T(t))B(u_2^h - u_2^*) + \frac{q_2 T}{1+r}\right] = \lambda q_2 \)

(6) \( \frac{\partial T}{\partial t} B(u_1^h - u_1^*) = \lambda \frac{q_t}{1+r} \)

(7) \( x_o + \frac{x_i}{1+r} + q_1 p_1 + q_2 p_2 + [1 - d_1(p_1)d_2(p_2)] \frac{q_1 T}{1+r} = y_o + \frac{y_1}{1+r} \)

The left-hand sides of equations (4), (5), and (6) represent the marginal benefit of an increase in prevention spending for disease 1, prevention spending for disease 2, and treatment
spending 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 \(u^b_i\) and \(u^t_i\)). The benefits of prevention also come from the lowered expected costs of treatment resulting from a lower probability of illness. Note that the benefits of additional prevention in each specific disease are scaled by the probability of contracting the other disease. Intuitively this is because there is less incentive to invest in, for example, malaria prevention if the child will die from diarrhea. Additionally, the utility benefits of prevention are scaled by the effectiveness of treatment. Intuitively, this is because the utility benefits of prevention lie in avoiding fatal sickness and therefore depend on the probability that the current level of treatment will lead to a recovery if a potentially fatal sickness occurs. The right-hand sides of equations (4), (5), and (6) correspond to the marginal costs of prevention for disease 1, prevention for disease 2, and treatment, respectively. Equation (7) is the budget constraint and illustrates the tradeoffs involved in the decision between investing in prevention and spending on treatment that stem from limited resources.

The primary point of interest in these first order conditions is their interrelationship. From these first order conditions, I can illustrate these interrelationships as well as identify the exogenous determinants of demand. The general form of the conditional demand functions are:

\[
\begin{align*}
(8) \quad p_1^* &= f(t, p_2, y_0, y_1, w_i, q_1, q_2, q_i) \\
(9) \quad p_2^* &= f(t, p_1, y_0, y_1, w_i, q_2, q_i, q_1) \\
(10) \quad t^* &= f(p_1, p_2, y_0, y_1, w_i, q_i, q_1, q_2)
\end{align*}
\]
To obtain predictions about how the three demands are related, I will make the following basic assumptions about the functional forms of \( T(t) \), \( d_1(p_1) \), and \( d_2(p_2) \):

\[
\begin{align*}
T(t) &= \sqrt{r}, t \in [0,1] \\
d_1(p_1) &= \sqrt{p_1}, p_1 \in [0,1] \\
d_2(p_2) &= \sqrt{p_2}, p_2 \in [0,1]
\end{align*}
\] (11)

The relationship between \( p_1 \) and \( p_2 \) can be seen clearly in the first order conditions. In equation (5), an increase in \( p_1 \) increases \( d_1 \) and thus raises the benefits 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 extremely low because of a lack knowledge about prevention or a lack of availability (i.e., effective marginal product of prevention is low), 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 individuals will have a magnified effect on survival in that they will also increase private investment in prevention of disease 2.

The complementarity effect will be hindered by higher prices for prevention of disease 2. This can be seen by inserting the assumptions in (11) into equation (5) and rearranging:

\[
p_2^* = \frac{p_1}{\lambda^2 q_2^2} \left[ (1-\sqrt{r})B(u_i-h-u_i') + \lambda \frac{q_it}{1+r} \right]^2
\]

In equation (12) an increase in \( p_1 \) raises optimal \( p_2 \), but is scaled by the price of \( q_2 \), so that the complementarity effect is limited by higher prices.

\[\text{2 These assumptions are made for computational ease due to the complexity of the interactions in the first order conditions. The implications drawn from the following comparative statics are robust to several different specifications such as } (x/(1+x)).\]
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, does a policy increasing prevention for disease 1, such as distributing vitamin A supplements, also reduce price responsiveness for disease 2? To answer this, differentiate equation (12) with respect to \( q_2 \) and rearrange to get an expression for price elasticity:

\[
E = \frac{\partial p_2}{\partial q_2} \frac{q_2}{p_2} = -\frac{p_1}{p_2} \left[ \frac{(1-\sqrt{t})B(u_1^b - u_1^t) + \lambda \frac{q_1 t}{1+r}}{\lambda^2 q_2^2} \right]^2
\]

Now, to see the differential price response by levels of \( p_1 \), differentiate the price elasticity with respect to \( p_1 \) and simplify using equation (6):

\[
\frac{\partial E}{\partial p_1} = \left[ \frac{p_1 \frac{\partial p_2}{\partial p_1} - p_2}{p_2^2} \right] \left[ \frac{(1-\sqrt{t})B(u_1^b - u_1^t) + \lambda \frac{q_1 t}{1+r}}{\lambda^2 q_2^2} \right]^2
\]

Equation (14) demonstrates that \( \frac{\partial E}{\partial p_1} \) will be greater than zero if

\[
\frac{p_1}{p_2} \frac{\partial p_2}{\partial p_1} > 1
\]

The inequality in (15) can be interpreted as requiring the elasticity of demand of prevention for disease 2 with respect to the prevention of disease 1 to be elastic. Thus, if complementarity effects are strong, changes in \( p_1 \) will mitigate the effects of price on demand for \( p_2 \) (i.e. decrease the price elasticity of demand.

The relationship between disease prevention and treatment in equation (10) is more complicated than the relationship between prevention measures because of the two pathways that stem from equation (7): (1) increased spending on \( p_2 \) leaves fewer resources available for
spending on treatment; (2) decreased probability of illness decreases the expected costs of
treatment. Pathway (1) will reduce the optimal amount of treatment spending, while pathway (2)
will increase optimal treatment spending, so it is unclear what the sign of the net effect will be.
To determine the sign of the effect, begin by dividing equation (4) by equation (5), inserting the
assumptions in (11), and then simplifying:

\[ q_2 p_2 = q_1 p_1 \]

Then totally differentiate equation (7) (for simplicity ignoring the effect of \( x_1 \))\(^3\) and rearrange to
get an expression for \( \frac{\partial t}{\partial p_1} \):

\[ \frac{\partial t}{\partial p_1} = \left( \frac{1 + r}{q_1 \sqrt{p_1 p_2}} \right) \left[ \frac{q_1 t}{1 + r} \left( \sqrt{\frac{p_1}{4 p_2}} \frac{\partial p_2}{\partial p_1} + \sqrt{\frac{p_2}{4 p_1}} \right) - q_1 - q_2 \frac{\partial p_2}{\partial p_1} \right] \]

The expression for the effect of \( p_1 \) on \( t \) is a complicated function of parameters, but the sign will
be negative if:

\[ \frac{q_1 t}{1 + r} \left( \sqrt{\frac{p_1}{4 p_2}} \frac{\partial p_2}{\partial p_1} + \sqrt{\frac{p_2}{4 p_1}} \right) < q_1 + q_2 \frac{\partial p_2}{\partial p_1} \]

Rearranging, multiplying both sides by \( \frac{p_1}{p_2} \), and inserting equation (16) gives:

\[ \frac{\partial p_2}{\partial p_1} \frac{p_1}{p_2} > -1 \]

This inequality will always hold, because it has been shown that \( \frac{\partial p_2}{\partial p_1} > 0 \). This demonstrates
that the net effect of a change in \( p_1 \) on \( t \) is negative, implying that factors causing low levels of

\(^3\) Allowing changes in \( x_1 \) creates the possibility of tradeoffs between consumption and treatment, reducing effect (1). The comparative statics for this scenario are even more complex than those presented here and so I leave \( x_1 \) fixed here and equation (17) thus serves as an upper bound for the effect of \( p_1 \) on \( t \). I leave it to the data to show if changes in \( x_1 \) reverse the sign of the effect.
prevention for disease 1 such as a lack of availability or knowledge of benefits of prevention will cause individuals to spend more on treatment. Additionally, policies transferring prevention for disease 1 to individuals will result in decreased levels of treatment spending.

To summarize, the model developed here leads to three clear implications:

(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 individuals will induce them to invest more heavily in prevention of disease 2.

(2) Factors causing a low level of disease 1 prevention will increase price sensitivity for goods (effort) used to prevent disease 2. A policy that provides goods to prevent disease 1 to individuals will decrease price sensitivity for goods used to prevent disease 2.

(3) Factors causing a low level of disease 1 prevention will cause high levels of spending on treatment. A policy that provides goods to prevent disease 1 to individuals will cause a decrease in treatment spending.

**Empirical Testing**

The competing diseases of diarrhea and malaria provide an opportunity for testing the model developed above. 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). 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, programs that provide these vitamin A supplements will cause a decrease in mortality from diarrhea and this change in competing risk of death can be used to assess the predictions of my model.

Over the past two decades, the importance of vitamin A supplements and their low level of availability for purchase have motivated governments, international organizations, and NGOs to distribute supplements 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 lasting effects are possible because vitamin A is fat soluble and thus mega-doses remain in the body for extended periods.

Vitamin A supplementation offers a perfect example of the type of prevention transfer discussed in the model. All available evidence suggests that parents do not invest in preventing child death from diarrhea with vitamin A supplements because of a lack of availability, a lack of knowledge, or both (Klemm et al. 2007; Sserunji and Harvey 2005; MOST Project 2010). Accordingly, many public, or quasi-public, programs have targeted distribution of vitamin A as a way to decrease mortality. Parents are likely to understand the expected benefits of vitamin A supplementation as well as expect future rounds of supplementation due to education components associated with supplementation program (Sserunji and Harvey 2005; National Nutrition Program 2008; MOST Project 2010). However, this is not a prerequisite for the model to be accurate because the benefits of supplementation will reveal themselves to parents over time due to the frequency 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 child medical exams and found that parents accurately observe severity of illness, especially for diarrhea (Kalter et al. 1991).

Malaria is a perfect 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 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 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 (9). 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 height-for-age and mother’s education. Specifically, I estimate the following regression equation:
(21) \[ ITN_i = \beta_0 + \beta_1 A_i + \beta_2 \text{Price}_m + \beta_3 A_i \ast \text{Price}_m + \beta_4 X_i + \beta_5 X_m + \eta_j + \epsilon_i \]

In this equation, \( A \) equals one if the child received a vitamin A supplement and zero otherwise, \( \text{Price}_m \) 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, age, and gender, \( X_m \) is a vector of household specific determinants of health production such as mother’s education, number of children that have died, and wealth, \( \eta_j \) is a community fixed effect, and \( \epsilon_i \) is a random error term.

The coefficients \( \beta_1 \) and \( \beta_2 \) in equation (21) indicate the extent to which households respond to the provision of vitamin A 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 hindered by higher prices.\(^4\)

The coefficients \( \beta_2 \) and \( \beta_3 \) in equation (21) indicate the level of price sensitivity for those with and without vitamin A supplementation. The coefficient \( \beta_2 \) is the base price response for ITNs and \( \beta_3 \) is the additional price response for individuals who received vitamin A supplementation. To determine whether the price response 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. This is done as follows:

\[ E_i = \frac{(\beta_3 + \beta_2 \ast A_i) \ast \text{Mean(Price}_m)}{\text{mean}(ITN \mid A_i = 0) + \beta_1 \ast A_i} \]

It is important to note that in equation (22) vitamin A supplementation enters in both the numerator and denominator. This is because from the model we know 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

\(^4\) This is a similar idea to that of Yarnoff (2011), which found that higher shadow prices for ITNs decreased household response to complementarities.
effect of vitamin A to the denominator, meaning that the sign of $\beta_2$ 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 $\beta_2$ will be negative because price will hinder household response to complementarities. At the same time however, the price elasticity may be lower for those with vitamin A depending on the other parameters. The difference in price response is thus jointly determined by $\beta_1$ and $\beta_2$.

While concerns over the endogeneity of vitamin A supplementation are diminished in this context because vitamin A supplementation is generally not available for purchase and is distributed by an external group, there is still concern over any vitamin A supplements obtained outside of a national distribution day. For example, some clinics are equipped with vitamin A supplements to give 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 effects of receiving a supplement on a national distribution day or from a clinic.

There also may be concern over vitamin A program targeting (administrative selection) or factors that influence a household’s decision to participate in a supplementation program. Additionally, concern over the endogeneity of prices is strong, because regional prices vary based on supply and demand factors within the region. For example, regions with higher ITN use will likely have higher prices due to higher demand from some third factor such as health knowledge. This assumes an upward sloping supply curve, a point argued for by Simon et al. (2001). In fact, regressions (not reported here) show that prices are positively correlated with household wealth, mother’s education, and literacy rates, all factors that will be positively correlated with demand.
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. 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 bed net 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. 

I estimate the following regression equation:

$\Pi T N_i = \beta_0 + \beta_1 A_i + \beta_2 A_i \ast Price_m + \beta_3 X_i + \eta_m + \epsilon_i$ 

Where all variables are as before and $\eta_m$ is a mother fixed effect. Note that $Price_m$ is omitted from equation (23) because it does not vary within a household. Thus, while this equation produces more reliable estimates of the effect of vitamin A and the extent to which this effect is offset by higher prices, it will not produce estimates of the base price response. In order to evaluate the difference in price elasticities for supplemented individuals, I will evaluate equation (22) at a range of values for $\beta_2$ (the base price effect).

While the mother fixed effect 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 height-for-age) and mother fixed effects on vitamin A supplementation indicate that only age is a significant predictor of

---

5 However, factors that are child specific may affect family decisions and these remain a possible source of bias. I will test for the presence of this bias with sensitivity analysis by child characteristics.
participation in supplementation programs with older children more likely to receive supplementation. This relationship is likely purely mechanical because older children have been in program target groups longer than their younger siblings. This argument is supported by the insignificance of gender and height-for-age in explaining vitamin A supplementation (two factors that would not be mechanically related due to increased time in the target group). Thus, based on this lack of observable correlation, it is plausible that unobserved child characteristics will be uncorrelated with the effect of vitamin A supplementation and mother fixed effects will be sufficient to control for all confounding factors.

The model also predicts that vitamin A supplementation will reduce the amount of spending on medical treatment. To test this hypothesis, I estimate the demand for medical treatment given by equation (10). I measure treatment as whether the child received medical treatment during an episode of malaria or diarrhea 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 a mother fixed effect 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 where the child received the supplement. Specifically, I estimate the following system of regression equations:

\begin{align*}
(24) \quad &Fever_i = \alpha_0 + \alpha_1 A_i + \alpha_2 ITN_i + \alpha_3 X_i + \eta_m + \varepsilon_i \\
(25) \quad &Treatment_i \mid Fever_i = 1 = \gamma_0 + \gamma_1 A_i + \gamma_2 X_i + \gamma_3 IMR_i + \eta_m + \varepsilon_i
\end{align*}

Where \(Fever_i\) equals one if the child contracted a fever (the primary symptom of malaria) in the past two weeks and zero otherwise, \(Treatment_i\) equals one if the child received treatment for the
fever, $A_i$ equals one if the child received a vitamin A supplement and zero otherwise and is divided by where the supplement was received, $ITN_i$ equals one if the child slept under an ITN and zero otherwise, $IMR_i$ is the inverse mills ratio generated from the predicted probability of contracting a fever in equation (24), $X_i$ is a vector of other child specific determinants of demand for health inputs such as height-for-age, age, and gender, $\eta_m$ is a mother fixed effect, and $\varepsilon_i$ is a random error term.

Equation (24) is a latent variable model of the probability of contracting a fever (Malaria). Equation (25) will only be observed if equation (24) 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 except through the joint effect of vitamin A. $\gamma_1$ is the parameter of interest in equation (25), because it will indicate how households change their treatment spending in response to vitamin A supplementation. The model predicts that $\gamma_1$ will be negative.

Data

I use data on child ITN use, 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 bed net use and prices 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 a fever (the primary symptom of malaria) in the past two weeks, and received medicine as treatment for that fever (the WHO-recommended treatment for malaria). Additionally, the survey contains data on the price households paid for ITNs. Households that did not own an ITN (and thus did 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 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 household 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 800 children for the second stage of treatment analysis.

**Results**

Table 1 presents results from regression analysis of ITN use. Columns 4 through 5 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 analyses by child age and health status (proxied by height-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 for 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 largely exogenous. The results also show that this estimate is moderated by the price of nets 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 for price.

Columns 1 though 3 of Table 1 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 child age. The estimates are virtually identical for all three columns, demonstrating that results are not sensitive to observable child factors and implying that they will be insensitive to unobservable factors 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 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. The results also show that this estimate is moderated 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.

Table 2 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 analyses by child age and health status (proxied by height-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 for 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 7 a percentage point increase in ITN use.

Columns 1 though 3 of Table 2 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 child age. 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 (22) at a range of values for the parameter $\beta_2$ (the base price effect). Table 3 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 supplementation (68 percent), the second column contains elasticity estimates for un-supplemented children, and the third column contains estimates for supplemented children.
These estimates show that price elasticities are significantly smaller for supplemented children. For example, at the overall elasticity of 1 ($\beta_2 = -0.02$) found by Cohen and Dupas (2008), the elasticity for supplemented children is 20 percent lower than the elasticity for un-supplemented children. At the elasticity of 1.8 ($\beta_2 = -0.04$) found by Dupas (2009), the elasticity for supplemented children is 25 percent lower than the elasticity for un-supplemented children. At the elasticity of approximately 3 ($\beta_2 = -0.07$) found by Hoffman et al (2008), the elasticity for supplemented children is 27 percent lower than the elasticity for un-supplemented children. These results demonstrate that ITN price elasticity is significantly lower for children who received vitamin A supplementation.

Table 4 presents results from a mother fixed effect analysis of the effect of vitamin A supplementation on the probability of contracting a fever. This is the first stage in the 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 corresponds to a model with a full set of controls and columns 2 and 3 show sensitivity analyses by height-for-age and age. In all cases the effect of vitamin A is separated by place of receipt. The estimated effect of vitamin A received at national distribution days is virtually zero. The effect of vitamin A received from a clinic however is positive and not that small (6 percent of mean fever probability). Further, ITN use reduces probability of contracting a fever by 13 percent of mean fever. However, none of these estimates are statistically significant.

Table 5 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 a fever. These results demonstrate that vitamin A supplementation received from national distribution
days reduces the probability of medical treatment. The point estimates from the model with full controls (column 1) imply that supplementation reduces the probability of treatment by 0.12 percentage points representing a 32 percent reduction in mean treatment. These results are insensitive to the inclusion of both age and height-for-age, implying that results are not sensitive to observable or unobservable child factors. Additionally, as expected 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 unobserved likelihood of going to a clinic biasing estimates upward for these children.

**Conclusions**

The model developed in this article provides important insight into the manner in which families in Africa decide between investing in disease prevention and spending on medical treatment for their children. Previously, researchers and policymakers have puzzled over the apparent inconsistency that families in Africa spend little on disease prevention, but relatively large amounts on medical treatment. Here I have demonstrated that high levels of competing disease risks will lower incentives to invest in disease prevention and increase incentives to wait for diseases to be realized and then spend on treatment. The inconsistency between prevention and treatment spending can thus be explained by high levels of competing disease risks in Africa.

I have tested this hypothesis using the competing diseases diarrhea and malaria, with vitamin A supplementation programs serving as an exogenous decrease in diarrhea mortality. I find strong evidence that the decrease in diarrhea mortality from vitamin A supplementation causes increased investment in ITNs, decreased price elasticity for ITNs, and decreased spending on medical treatment for children when sick.
This research has important implications for policy. Policymakers want to induce greater investment in disease prevention and reduce reliance on treatment in order to reduce the cost of child survival. This model has two implications for this effort. First, subsidies for prevention measures such as ITNs will be ineffective at increasing use if there are competing diseases for which prevention is unknown or unavailable. Second, programs targeting diseases for which prevention is unknown or unavailable will produce magnified prevention effects because they will induce families to invest more in preventing other diseases for which prevention methods are known and available. A policy agenda structured along these lines has the potential to significantly reduce child mortality from preventable diseases.
References


MOST Project. 2010. “Starter Kit for Vitamin A capsule Distribution.” USAID.


Table 1: Effect of Vitamin A Supplementation on ITN Use

<table>
<thead>
<tr>
<th></th>
<th>Mother Fixed Effects</th>
<th>Community Fixed Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(1)</td>
<td>(2)</td>
</tr>
<tr>
<td>Vitamin A</td>
<td>0.039**</td>
<td>0.039**</td>
</tr>
<tr>
<td></td>
<td>(0.019)</td>
<td>(0.019)</td>
</tr>
<tr>
<td>Vitamin A*Price</td>
<td>-0.003*</td>
<td>-0.003*</td>
</tr>
<tr>
<td></td>
<td>(0.002)</td>
<td>(0.002)</td>
</tr>
<tr>
<td>Price</td>
<td>0.003</td>
<td>0.003</td>
</tr>
<tr>
<td>Household Resources</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Child age</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Child height for age</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Mean (unsupplemented)</td>
<td>0.07</td>
<td>0.07</td>
</tr>
<tr>
<td>Observations</td>
<td>7017</td>
<td>7017</td>
</tr>
</tbody>
</table>

Notes:
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 models include controls for child gender, number of children in household, and number of sick adults in household,
4. *** p<0.01, ** p<0.05, * p<0.1
Table 2: Vitamin A Supplementation on ITN Use, by Source of Vitamin A

<table>
<thead>
<tr>
<th></th>
<th>Mother Fixed Effects</th>
<th>Community Fixed Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(1)</td>
<td>(2)</td>
</tr>
<tr>
<td>Vitamin A from Nation Distribution Day</td>
<td>0.039*</td>
<td>0.039*</td>
</tr>
<tr>
<td>Vitamin A from Health Facility</td>
<td>0.037</td>
<td>0.037</td>
</tr>
<tr>
<td>Vitamin A from Nation Distribution Day *Price</td>
<td>-0.003</td>
<td>-0.003</td>
</tr>
<tr>
<td>Vitamin A from Health Facility*Price</td>
<td>-0.004</td>
<td>-0.004</td>
</tr>
<tr>
<td>Price</td>
<td>0.003</td>
<td>0.003</td>
</tr>
</tbody>
</table>

Household Resources: No Yes No Yes Yes No
Child age: Yes Yes No Yes No No
Child height 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:
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 models include controls for child gender, number of children in household, and number of sick adults in household,
4. *** p<0.01, ** p<0.05, * p<0.1
Table 3: Range of Elasticities from Mother Fixed Effect Analysis

<table>
<thead>
<tr>
<th>Base Price Response</th>
<th>Overall</th>
<th>Vitamin A supplementation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No</td>
</tr>
<tr>
<td>-0.01</td>
<td>-0.55</td>
<td>-0.50</td>
</tr>
<tr>
<td>-0.02</td>
<td>-1.10</td>
<td>-0.88</td>
</tr>
<tr>
<td>-0.03</td>
<td>-1.65</td>
<td>-1.27</td>
</tr>
<tr>
<td>-0.04</td>
<td>-2.20</td>
<td>-1.65</td>
</tr>
<tr>
<td>-0.05</td>
<td>-2.76</td>
<td>-2.04</td>
</tr>
<tr>
<td>-0.06</td>
<td>-3.31</td>
<td>-2.42</td>
</tr>
<tr>
<td>-0.07</td>
<td>-3.86</td>
<td>-2.81</td>
</tr>
<tr>
<td>-0.08</td>
<td>-4.41</td>
<td>-3.19</td>
</tr>
<tr>
<td>-0.09</td>
<td>-4.96</td>
<td>-3.58</td>
</tr>
<tr>
<td>-0.10</td>
<td>-5.51</td>
<td>-3.96</td>
</tr>
</tbody>
</table>
Table 4: Effect of Vitamin A on Contracting a Fever

<table>
<thead>
<tr>
<th></th>
<th>Mother Fixed Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(1)</td>
</tr>
<tr>
<td></td>
<td>(2)</td>
</tr>
<tr>
<td></td>
<td>(3)</td>
</tr>
<tr>
<td>Vitamin A from Nation Distribution Day</td>
<td>-0.002</td>
</tr>
<tr>
<td></td>
<td>(0.022)</td>
</tr>
<tr>
<td>Vitamin A from Health Facility</td>
<td>0.013</td>
</tr>
<tr>
<td></td>
<td>(0.025)</td>
</tr>
<tr>
<td>ITN</td>
<td>-0.030</td>
</tr>
<tr>
<td></td>
<td>(0.031)</td>
</tr>
<tr>
<td>Household Resources</td>
<td>No</td>
</tr>
<tr>
<td>Child age</td>
<td>Yes</td>
</tr>
<tr>
<td>Child height for age</td>
<td>Yes</td>
</tr>
<tr>
<td>Mean (unsupplemented)</td>
<td>0.23</td>
</tr>
<tr>
<td>Observations</td>
<td>7017</td>
</tr>
</tbody>
</table>

Notes:
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 models include controls for child gender, number of children in household, and number of sick adults in household,
4. *** p<0.01, ** p<0.05, * p<0.1
Table 5: Effect of Vitamin A on Receiving Anti-Malarial Drugs When Sick

<table>
<thead>
<tr>
<th></th>
<th>Mother Fixed Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(1)</td>
</tr>
<tr>
<td>Vitamin A from Nation Distribution Day</td>
<td>-0.12*</td>
</tr>
<tr>
<td></td>
<td>(0.07)</td>
</tr>
<tr>
<td>Vitamin A from Health Facility</td>
<td>0.21</td>
</tr>
<tr>
<td></td>
<td>(0.14)</td>
</tr>
<tr>
<td>Child age</td>
<td>Yes</td>
</tr>
<tr>
<td>Child height for age</td>
<td>Yes</td>
</tr>
<tr>
<td>Mean (unsupplemented)</td>
<td>0.37</td>
</tr>
<tr>
<td>Observations</td>
<td>861</td>
</tr>
</tbody>
</table>

Notes:
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 models include controls for child gender, number of children in household, and number of sick adults in household,
4. All models include inverse mills ratio constructed from estimates in table 4
5. *** p<0.01, ** p<0.05, * p<0.1