

Economics of Environmental Epidemiology

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Infectious disease such as malaria, dengue, and diarrhea that are spread by vectors such as mosquitoes and flies are rife in the much of the developing world, potentially impacting more than two-third of the world's population. Environmental conditions that favor the transmission of such diseases are, in turn, affected by human activity such as deforestation, livestock rearing, irrigated farming, migration, road construction, dam-building, and water and sanitation infrastructure provision through their impact on the survival and abundance of disease vectors. Therefore, the prevention and control of these diseases relies on the interplay of public policies (e.g., reducing microbial pathogen load in public water supplies) and private choices (e.g., safe storage, treatment, and handling of drinking water and food inside the house). As described, externalities play important roles in disease dynamics, demonstrating the need for public policies such as subsidies/taxes, information and technical assistance to achieve socially desirable outcomes. This paper presents an application in public economics that tests analytical models of economic epidemiology. The first part of the paper reviews and synthesizes the early literature that has focused on the behavioral basis of disease control and prevention. The second part presents empirical applications where we use the analytical models to shape the econometric analyses, permitting perhaps the first tests of untested hypotheses about the effectiveness of disease control interventions and how they related to private disease control. We draw on several data sets, including a large multi-year, multi-scale on going study from rural India to build econometric models of disease outcomes and public interventions. We conclude with a discussion of policy implications and conceptual and empirical research extensions.

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Introduction

Infectious disease such as malaria, dengue, and diarrhea that are spread by vectors such as mosquitoes and flies are rife in the much of the developing world, potentially impacting more than two-third of the world's population. Environmental conditions that favor the transmission of such diseases are, in turn, affected by human activity such as deforestation, livestock rearing, irrigated farming, migration, road construction, dam-building, and water and sanitation infrastructure provision through their impact on the survival and abundance of disease vectors. Therefore, the prevention and control of these diseases relies on the interplay of public policies (*e.g.*, reducing microbial pathogen load in public water supplies) and private choices (*e.g.*, safe storage, treatment, and handling of drinking water and food inside the house). As described, externalities play important roles in disease dynamics, demonstrating the need for targeted public policies such as subsidies/taxes, information and technical assistance. This paper presents an application in public economics that tests analytical models of economic epidemiology. The first part of the paper reviews and synthesizes the early literature that has focused on the behavioral basis of disease control and prevention. The second part summarizes four empirical applications where we use the analytical models to shape the econometric analyses, permitting perhaps the first hypothesis tests about the effectiveness of disease control interventions and how they relate to private disease control. The first application combines data from four different sources to analyze the relationship between malaria prevalence and malaria prevention at the country level. Because of the level of aggregation, we are limited in what we can learn about private preventive behavior. The second and third applications address this by using cross-sectional microeconomic datasets on malaria prevention and malaria prevalence in Ethiopia and India. These analyses are subject to both recall and simultaneity bias due to the cross-sectional nature of the data. The fourth dataset overcomes these validity threats by using multi-scale panel data on *diarrhea* prevention and prevalence from an ongoing study in rural India. These analyses provide consistent evidence supporting the predictions of economic epidemiology models for both malaria and diarrhea.

Thirty percent of the global burden of disease can be attributed to environmental risks (Smith et al., 1999; Smith and Ezzati, 2005). For example, malaria transmission is affected by changes in the landscape such as deforestation, irrigated agriculture, and dams that modify the vector (mosquito) habitat. Respiratory infections are impacted by household choices of cooking fuel and house ventilation as well as transport and energy policy that affect ambient pollution. Similarly, diarrheal diseases are affected by personal hygiene choices as well as public water supply and

environmental sanitation. Collectively diseases like malaria, acute respiratory illness (ARI), and diarrhea potentially impact 4-5 billion people or most of the world's population. Unfortunately, there are no easy or transparent solutions to these environmental infections because they depend on the complicated interplay of ecology, epidemiology and economics. However, it is clear that externalities are pervasive in the transmission and prevention of these infections and public economics has a critical role in understanding and therefore controlling these diseases.

The following quotation from Spielman (2003) – one the leading malaria researchers in the world – underscores the importance of behaviors in malaria epidemiology and control:

“The vertically organized anti-malaria programs that were launched before the 1990s could be conducted largely without reference to the behavior and the belief systems of the affected populations. Indoor residual insecticides, the hallmark of the eradication era, were applied uniformly across entire continents. Although residents frequently denied the government-spray teams entry to their homes and removed the insecticidal sprays from treated walls, their active participation was largely irrelevant to the intervention. Current antimalaria programs, however, generally are organized horizontally and depend heavily on resident participation. The recently launched Roll Back Malaria (RBM) Program relies mainly on insecticide impregnated bednets (ITNs) and combination drug therapy (CT). ITNs must be hung by the people who are to sleep under them and they must be reimpregnated in a timely manner, frequently at the user's expense. Failure to sustain this intervention would result in increased sickness and death due to exposure of relatively non-immune people to new infections. CT requires a standard schedule of drug administration that relies on the cooperation and understanding of each affected person. Failure to adhere to the prescribed regimen would endanger the long-term efficacy of the regimen. Resident cooperation is even more crucial when environmental management or housing improvement becomes the intervention modality. These practical reasons drive our current need for a solid understanding of the behavioral and social factors that influence malaria risk and that may inhibit or facilitate particular intervention modalities. No longer can interventions be conducted uniformly across broad regions; the problems tend to be local.”

This paper analyzes the relationships between disease prevalence and public health agencies' behaviors as well as microeconomic behavior. The paper is organized in 3 parts. First, we introduce economic epidemiology, comparing and contrasting it with conventional economic applications to disease control, and quickly hone in on two critical concepts – externalities and prevalence elasticity. Second, we use data from a variety of sources – global and household surveys – to provide some evidence on the predictions of economic epidemiology. Finally, we discuss some of the implications for research and policy.

Conceptual Framework

In conventional applications of economics to modeling diseases, economics basically provides descriptive tools to help characterize supply side features such as technology or production of interventions such as artemisinin-based combination therapy (ACT), or demand side issues such as willingness-to-pay for drugs and vaccines. While this demand and supply logic can be used to model markets and the associated prices (i.e., institutional mechanisms for allocating resources to disease control), it is limited in two ways. First, it may not capture nonmarket behaviors and impacts. Second, demand and supply side analyses are rarely integrated, which would account for interaction.

Welfare economic tools can integrate demand and supply side issues. Prescriptive tools such as cost-effectiveness analysis (CEA) and cost-benefit-analysis (CBA) describe tradeoffs between the overall disease costs and the costs of interventions, given the effectiveness of interventions. This use of the literature has been limited by the fact that there are large knowledge gaps in the empirical evidence on disease costs and the cost-effectiveness of interventions.

The economics toolbox also includes the conceptual framework provided by public economics, which can be important for examining infectious diseases. By providing a framework for identifying institutional failures related to efficiency² and equity³ concerns, we can use public economics to design and evaluate policy instruments such as regulation and provision (of, say, health care infrastructure and immunization programs); incentive mechanisms such taxes and subsidies; and information and technical assistance such as disease surveillance, rapid diagnosis, and targeted IEC (information-education-communication) campaigns.

Economic epidemiology (EE) has emerged from the public economics tradition by focusing on the strategic behaviors of governments, households and individuals. EE applies the economic toolkit (assembled to model market exchanges) to examine non-market exchanges that underpin the development, spread, and control of infections and diseases.⁴ Although much of the early

² Efficiency failures relate to the notion of externalities, public goods, monopolistic production and sale, and a whole array of transaction costs related to information asymmetries in particular.

³ Equity concerns surround poverty (e.g., are diseases and interventions distributed unequally across socio-economic strata) as well as sustainability (e.g., inter-generational transfers) issues.

⁴ Manski (1995) provides an excellent motivation and overview of the progress made by economists by examining trades and exchanges that take place between ‘buyers’ and ‘seller’ and outside the ‘textbook’

work, dating back to mid-1990s, focused on fatal diseases such as HIV-AIDS that are communicated through person-to-person contact, we show in this paper that most of the predictions and logic are generalizable to vector-borne and other ‘environmental’ diseases. EE focuses on the interplay of public policies and private responses, conditional on the disease dynamics (*i.e.*, stage, mode), and address the following kinds of questions:

- What should governments do when individuals ignore social impacts of private choices (*i.e.*, when externalities exist)?
- How does individual behavior respond to interventions that change their disease exposure?

The answer to the first question depends on the nature and size of the externality. Externalities are central to the transmission, control, and prevention of infectious diseases. In malaria control, for example, improper use of ACT or IRS can promote the emergence of drug resistance in the parasite and insecticide resistance in the mosquitoes. Similarly insufficient use of insecticide treated nets (ITNs) can increase vector capacity⁵ and effectiveness.

The answer to the second question depends on the nature and extent of prevalence elasticity, which is defined as the response of private prevention to disease prevalence. Essentially, as public intervention reduces disease prevalence, households respond by reducing key preventive and mitigating behaviors, which diminishes effectiveness of intervention. Thus, the very success of the intervention compromises its effectiveness.

While the averting behavior models in the environmental economics literature focus on similar issues, this similarity has not been noted in previous research. Unfortunately, the literature provides little or no empirical evidence on the magnitudes of externalities and prevalence elasticity. This paper presents estimates of the prevalence elasticity of demand for malaria prevention.

marketplace. Kenkel (2000) argues that Philipson (2000) has pushed the frontier in health economics by modeling the equilibrium of infections and human responses, *i.e.*, in equilibrium one individual’s preventive behavior determines and is determined by choices of other individuals such that demand for prevention is prevalence elastic. Kenkel laments health economists’ failure to capitalize on the insights provided by this approach.

⁵ Vectorial capacity measures the rate at which susceptible vectors meet infected hosts.

In an Appendix to this paper, we present a simple constrained optimization model that draws on the EE literature, the averting behavior literature in environmental economics, and the health production literature in health economics to present an analytical version of the discussion presented in the previous paragraph.

In contrast to the austere constrained optimization models used in early EE work, Gersowitz and Hammer (2003) suggest a richer set of objectives (*e.g.*, utility functions) and constraints (*e.g.*, budget and production functions) is more realistic. Health and wealth are not the sole objectives of individuals or households—individual freedoms and others’ wellbeing may also be important objectives. For example, sleeping under bednets or filtering your water before you drink imposes on individual freedoms. It is also important to determine the scope of the utility function – is this at the individual level or at the family level or does it include members of your community (immediate neighbors or virtual communities). The extent to which your objectives are altruistic affects the extent of the externalities. Similarly, to what extent are our choices dependent on outcomes in the near future (and recent past) versus things in the distant future or past. This issue is related to inter-generational altruism.

In addition to budget constraints, disease epidemiology and disease control processes impose constraints. Biology clearly imposes constraints related to the mode, transmission and progression of the disease. For example, vector characteristics such as habits and resistance to pesticides and drugs influence choices. Individual and public choices are different in situations where the disease is endemic and stable in contrast to an unstable transmission zone. The progression of the disease after infection – recovery to become susceptible, or immunity, or chronic suffering and death – impact choices. Specifying constraints also involves identifying the control options – averting or preventive behaviors (*e.g.*, vector control) and mitigating or treatment behaviors (*e.g.*, case management). Finally, knowledge and or information about all of the above constraints, including costs of the various options (what does it cost in monetary and opportunity cost terms to cope with or care for an illness) affects choices.

Given multiple objectives and numerous constraints, tradeoffs (and therefore the role of economics) are inevitable. Early work in EE focused on a shortcoming in modeling of disease dynamics: its failure to consider the idea of a “prevalence elasticity of demand” for safe (disease preventing) behaviors such as hand washing and sleeping under mosquito nets. The key to this conclusion is that the private choice to engage in “safe behaviors” depends on disease prevalence, and disease prevalence depends on safe behaviors in the community, triggering a dynamic relationship between public policies and private responses. More generally,

mathematical epidemiology models (and field epidemiology models, unfortunately) fail to use objective functions that consider the full welfare costs of infection and its offsetting interventions, and overlook the role of diminishing returns to interventions that affect the evolution of the epidemic. The literature suggests that public health interventions hit diminishing returns because of increasing opportunity costs of prevention and declining demand for prevention as prevalence decreases.

While these initial insights have helped us better understand infectious disease control, there are five reasons that these early lessons do not translate easily to the conditions in poor, tropical countries plagued with a general class of vector-borne disease such as malaria, dengue, and diarrhea. First, in contrast to fatal diseases that are transmitted person-to-person (e.g., HIV/AIDS), the choice of an effective public health intervention depends critically on what happens to a susceptible individual who becomes infected (and infectious) – does he recover (to become susceptible), become immune, or die? The intervention paths are sensitive to the targeting of different groups (uninfected, infected, or everyone) and in the susceptible-infected-susceptible case we should equally subsidize prevention and therapy that block people from being infected (Gersovitz and Hammer, 2005). Second, even for the simplest vector-borne disease, the evolution of the disease involves several state variables including dynamics of ecosystem change, vector populations, drug and pesticide resistance, and human populations with different disease statuses. Third, the medical distinction between prevention and therapy does not hold when we consider the economic properties of prevention and therapies largely because therapy can prevent future infections by reducing the pathogen load and the probability of re-infection. Fourth, we need specific information (not general information) on risk exposure (infectious people, vector, and parasites), particularly in endemic areas to design effective interventions. Fifth, purely price-based incentives are insufficient because many prevention and therapeutic behaviors are non-market and unobservable. The countervailing impacts of private behaviors could be more pronounced if we also account for similar behavioral responses in human activities that affect environmental conditions, which in turn influence disease transmissions.

In general, we see that the analytics quickly get murky if we consider the disease dynamics of a general class of vector-borne diseases. As Gersovitz and Hammer demonstrate, the models are analytically intractable, requiring assessment via numerical dynamic programming using parameters (effectiveness of preventative and therapeutic interventions, full costing of diseases) that have little or no empirical basis. Moreover, these results only hold if we assume that

individuals are rational, have perfect foresight, care about present and future household members, and can insure against adverse health outcomes.

Take for example the simple analytical model presented in the Appendix to this paper. We find that a household's optimal level of prevention depends on all exogenous variables (including prevalence) and the optimally chosen infection level. So for example if environmental modifications elevate disease prevalence, we can expect to see an impact on optimally chosen prevention. Unfortunately, as discussed in the appendix, we cannot sign this derivative because there are too many parameters of unknown signs and sizes interacting with each other in complex analytical expressions.

To offer a broad summary of the overall literature, Philipson seems to focus on the critical role of private behaviors (particularly a positive prevalence elasticity of prevention) in suggesting that public health interventions will be self-limiting. In contrast, Gersovitz and Hammer focus on the critical role of prevention and infection externalities in concluding that public intervention is critical. While both build their arguments around a model of an optimizing rational individual agent, they indicate different roles for public policies. Thus, the collective limitations of strictly analytical work call for more basic empirical studies to evaluate (a) the nature and magnitude of externalities of preventive and therapeutic choices, (b) the relative magnitudes of the net benefits of interventions, and (c) the extent of correlation among interventions, *e.g.*, the prevalence elasticity of demand for prevention. Therefore, taking a page from Gersovitz and Hammer's (2003) synthesis of the literature – "Philipson (2003) reviews the evidence from the United States that indicates that people's preventive behaviors respond to prevalence; there are no similar studies for poor countries" - we focus on testing the hypothesis of a positive prevalence elasticity of prevention of malaria in tropical countries.

Empirical Evidence

This paper reports empirical assessments of the prevalence elasticity of disease prevention behaviors using four different datasets. Econometric analysis using global data on malaria control show that national prevention is clearly responsive to the extent of historical malaria prevalence in the country. This finding is confirmed in two microeconomic data sets – 800 households from the Tigray region of Ethiopia, and 600 households from the Keonjhar region of India. We further probe this issue by examining the case of diarrhea prevention and control with data from an on-going evaluation of a large scale rural water supply and sanitation program in Maharashtra, India

involving approximately 65,000 individuals from 11,000 households in 250 communities. These applications are described next.

Case I: Global or ‘Macro’ Evidence of Prevalence Elasticity

We constructed a global malaria dataset by combining data from four sources. First, we collected data on malaria preventive behaviors and incidence levels from the World Health Organization’s Global Health Atlas. The Global Health Atlas has data for up to 195 countries from 1990 to 2004 on a range of malaria variables, including the number of cases, intervention coverage, and service delivery by country. We use the annual number of reported malaria cases from the Global Health Atlas as a measure of prevalence in our analysis. To measure country-wide preventive activities, we use data from two additional variables in the Global Health Atlas: the percent of households who used one or more bednets (treated or untreated) for their child under five and the total annual number of bednets (treated or untreated) sold or distributed. The number of countries with data available in this dataset varies by year.

Second, we use data from the 2001 Human Development Report (HDR) to measure socioeconomic characteristics at the country level. The HDR measures on economic conditions using per capita GDP and social conditions using adult literacy rates, educational enrollment rates, and life expectancy for 162 countries.

Third, we use the malaria ecology index created by Kiszewski et al. (2004) to capture vector ecology and climatic factors. This index combines climatic factors (*e.g.*, rainfall and precipitation), the presence of different mosquito vectors, and the human biting rates of these vectors to proxy for mosquito transmission. This index captures the ecological conditions with the strongest influence on the intensity of malaria prevalence and can therefore predict the actual and potential stability of transmission. It is reported on a scale of 0 (low transmission potential) to 39 (high transmission potential). Data is available for 160 countries.

Finally, we use several of Kaufmann et al. (2003)’s measures as indicators of political conditions. We use data on political stability, voice and accountability, and control of corruption from 1998 for this analysis. Political stability measures the likelihood that the government in power will be destabilized; voice and accountability measures the amount of civil liberties and political rights afforded to citizens; and control of corruption measures the exercise of public power for private gain within a country. All measures are reported on a scale of -2.5 (lowest) to 2.5 (highest). This data is available for 195 countries and island states. Because this dataset has the largest number of observations, we match data from other sources to the Kaufmann et al. (2003) data, giving us a total sample size of 195.

The combination of data from different sources with different levels of coverage also required some assumptions. We assume that those countries not appearing in the Global Health Atlas's malaria data represent countries with no malaria cases or preventive behavior in the recent past. A quick inspection suggests that this is a safe assumption because countries not appearing on the list are from the West and/or from outside the tropics, where there is no malaria transmission or malaria prevention. Second, reporting schedules for preventive behavior information – total net use by children under five and total nets sold – vary by country and were not annual. These reporting schedules leave information gaps by year, so we average information over the six-year period from 1999 to 2004. Our estimation strategy, however, considers the influence of malaria prevalence (*i.e.*, averaged from 1990 to 2000) on recent preventive behaviors (*i.e.*, in years 1999-2004). We also test the robustness of our findings by applying an instrument for this index of prevalence.

Table 1 presents results from regressing preventive behaviors (net use and nets sold) on malaria prevalence, a per capita GDP index, educational enrollment, control of corruption and political stability. The results show that preventive behaviors are prevalence elastic at the macroeconomic scale: there is less prevention in countries with lower malaria prevalence.

These results must be interpreted cautiously for several reasons. First, since these data are highly aggregated, one cannot distinguish prevalence elasticity in the public's demand for prevention from prevalence elasticity in the health system's supply of prevention (*i.e.*, the targeting of interventions to areas with high prevalence). Despite this ambiguity, this analysis supports our assertion/hypothesis that behaviors—of individuals and/or health systems—are important in the transmission and efficient control of malaria. Second, since our data originate from disparate sources, we were concerned about imprecision in the measures of some concepts – and specifically overlap between measures. To address concerns about both concept validity and multi-collinearity, we conducted pair-wise correlation tests between various independent variables to develop a specification that includes a reasonable mix of ecological, institutional and economic determinants of preventive behaviors. The specifications in Table 1 take these test results into account.

Finally, the Global Health Atlas's malaria data reflect data from countries' public health surveillance systems, which we know to be imperfect measures of prevalence due to self-treatment, mis-diagnosis, and reporting problems. Because of this potential errors-in-variables problem, we use Kiszewski et al.'s (2004) index of malaria ecology as an instrument. As shown in Table 2, there is a strong positive association between malaria prevalence (malaria ecology) and preventive behaviors at the cross-country level.

Case II: Malaria Prevention in Tigray, Ethiopia

There could be all kinds of aggregation concerns with cross-country regressions, not the least of which is the scale mismatch with our model of preventive behaviors. Thus, we test this hypothesis with two micro datasets from Tigray, Ethiopia and Orissa, India.

In the late 1990s, malaria incidence was increasing in the Tigray province of Ethiopia due to the construction of microdams that created large pools of stagnant water. Malaria transmission follows a seasonal pattern, with peak transmission occurring at the end of the rainy season (October through November) and, to a lesser extent, during the rainy season (June through September). Both *Plasmodium falciparum* and *Plasmodium vivax* are present in Tigray, with *Plasmodium falciparum* predominating (Ghebreyesus, 1996). Unlike highly endemic areas, where cyclical exposure allows children to develop a resistance to malaria over time, in Tigray, malaria affects children and adults. This has significant implications for labor productivity and results in high per household costs. As shown in Cropper et al. (2004), the annual per household cost of illness in Tigray was estimated between US\$9 and US\$31, which is about 14 percent of household income.

In an attempt to alleviate the burden of malaria, the government of Tigray has initiated several malaria control activities. These include spraying in outbreak areas, particularly in the early part of the peak transmission season; encouraging environmental control by communities, such as draining marshes and filling in ditches having standing water; and training volunteer community health workers to recognize and treat malaria with chloroquine. In addition to the government-encouraged draining program, individuals engage in other control methods, including indoor residual spraying, burning dung and leaves, taking malaria prophylaxis, and using bednets.

We test our hypothesis of the prevalence elasticity of prevention by using data from a 1997 study conducted in the Tembien sector of Tigray province in northern Ethiopia (Figure 1). Information about the original 1997 study can be found in Cropper et al. (1999), Cropper et al. (2004) and Poulos et al. (2006). Eighteen villages in two districts (Tangua Abergelle and Kola Tembien) were sampled in this study. Villages were selected to obtain sufficient variation in malaria incidence; lacking official statistics on incidence, information on chloroquine distribution and village altitudes were used as proxies for incidence levels. A total of 889 individual interviews were completed; however, 41 respondents (4.6 percent of the sample) were not familiar with malaria. These households were dropped from the sample, leaving 848 respondents.⁶

⁶ The sampling target of the study was 50 households in each of the 18 study villages. Because up-to-date household lists did not exist, random sampling within each village was difficult. Therefore, each

The survey asked about household's current health status, knowledge of malaria, and past expenditures on malaria prevention and treatment. This included information on the adoption of the preventive behaviors being explored in this research: draining water, spraying, burning, taking medicine, and using a bednet. The survey also collected data on the socioeconomic characteristics, including education, income, assets, occupation, and housing construction.

We rely on self-reported malaria experience to measure malaria prevalence. Like the public health statistics used above, self-reported malaria may be an inaccurate measure of true malaria due to misdiagnosis. However, self-reports are appropriate for this analysis because it is concerned with private preventive behavior – which is motivated by perceived rather than actual illness. Use of self-reported rather than actual malaria will not impact our hypothesis unless errors in self-reports are unbalanced across levels of prevention.

Self-reported malaria incidence indicates that this disease is widespread in the sample, with more than 50 percent of all adults and children experiencing a malaria case in the past two years. On average, a household experiences two cases of malaria per year. In addition, about seven percent of all households have had a family member die of malaria. In terms of avoiding malaria, 66 percent of the sample reported practicing at least one preventive behavior, less than 15 percent practice two or more preventive behaviors, and less than 4 percent practice more than three preventive behaviors. Draining areas of standing water around the house is the most common preventive activity, with 83 percent of the sample reporting this behavior. Other activities include burning dung or leaves to keep mosquitoes away (10 percent), residual indoor spraying (8 percent), taking malaria prophylaxis (7 percent), and sleeping under a bednet (2 percent).⁷

Village prevalence is measured by the village-specific average of self-reported malaria cases per person per year. To explore the relationship between preventive behavior and disease prevalence, we plotted the probability that a sample household engages in a preventive behavior by their quartile in which their village malaria prevalence falls. Figure 2 shows that for all four preventive behaviors, the likelihood of household preventive behavior increases monotonically with prevalence quartile. We also conducted multivariate regression analyses of the likelihood of engaging in the four preventive activities: draining, burning, spraying, or taking prophylaxis.

interviewer was placed at a different location in the village and instructed to walk in a specified direction, interviewing every other household.

⁷ Due to the small number of households that reported sleeping under a bednet, we only consider draining, burning, spraying, and taking prophylaxis in our exploration of prevalence-elastic behaviors.

Table 3 reports estimates of the coefficients, p-values, and marginal effects of increases in village and household characteristics on the likelihood that a household engages in each of the preventive activities. These results suggest an increase in village-specific prevalence by one case per year per person is correlated with a 25 percent increase in the likelihood of draining water, a 13 percent increase in the likelihood of spraying indoors, an 11 percent increase in the likelihood of burning dung or leaves, and a 10 percent increase in the likelihood of taking prophylactics.⁸

Case III: Malaria Prevention in Keonjhar, Orissa, India

To test the robustness of these findings, we analyzed a data set from the Eastern Ghats of India (Keonjhar district in the state of Orissa) that contains household level information on malaria prevention and village prevalence. The study was designed to examine the links between mining, forest biodiversity, and rural livelihoods. Because of its location in a rural forested and malarious region of eastern India, we are able to examine the links between malaria prevalence and prevention activities. Instead of presenting details on design and findings of the study (see Saha et al., 2006), here we focus on the key aspects of the data collection and variables that are relevant to our application.

The household survey was designed and implemented between August and November of 2006. We interviewed approximately 600 randomly households from 20 villages (stratified by presence of/distance from a mine) in the blocks of Joda and Keonjhar Sadar of Keonjhar district. We also collected administrative records (e.g., population characteristics), and GIS layers on topography and infrastructure. The survey contains modules designed to elicit information on household access to public utilities, household perceptions of environmental change, forest resource use, health, education, demography, current consumption, forest products, agricultural activities, employment, asset ownership and participation in community activities. Specifically it included

⁸ To further explore these relationships and establish an empirical measure of prevention externality, we regress individual malaria incidence on village-level and household preventive behaviors. Village prevention is the average number of preventive activities [drain, burn, spray, medicine] in a village; and private prevention includes the average number of preventive activities (drain, burn, spray, medicine) by a household. We find that village-level preventive behaviors have a significant and negative impact on malaria incidence; whereas individual behaviors have a much smaller and positive impact. One may interpret this as evidence of prevention externality, however, caution should be taken when interpreting these results because of the contemporaneous nature of the dataset precludes a better understanding of the dynamics.

information on individual level incidence and prevalence of malaria, knowledge regarding the illness, and a variety of malaria-related prevention and treatment behaviors.

Self-reported malaria prevalence data suggests that 34% of the sample population experienced malaria in the past 5 years (2001-2005). Approximately 75% of households had someone suffer malaria in the past 2 years, with household averaging at least 1 case of malaria in 2005. 81% of malaria cases were diagnosed by a blood test. Approximately 75% of the cases received medical treatment at a health facility, of which 70% went a public facility. Among those who received medical attention, an average of approximately 340 Rupees (8 US dollars) was spent on treatment, 6 nights were spent in a hospital or in-patient clinic, and 6 days were spent being unproductive (e.g., missing school or work). Furthermore, family members lost about 10 work days taking care of the sick.

Turning to preventive behaviors, approximately 73% of households practice at least 1 preventive behavior. 41% sleep under a bednet; 33% use repellants (mostly traditional, rather than commercial); 5% rely public health spraying (IRS as well as outdoors); and only 4% clean drains and avoid standing water.

Results of regression model of prevention behavior are reported in Table 4, where malaria prevalence is defined as number of cases per 1000 villagers in a village who suffered the disease prior to 2005.⁹ The results indicate that prevention is positively and significantly correlated with malaria prevalence, *ceteris paribus*. That is, controlling for demographic characteristics, caregiver characteristics, malaria knowledge, and socio-economic factors, the decision to engage in prevention (or just sleeping under a bednet, the most popular prevention activity) is prevalence elastic.

Again, data limitations provide three reasons for cautious interpretation of the Tigray and Keonjhar results (cases 2 and 3). First, the cross-sectional nature of the micro data cases forces us to rely on recall data. Thus, our self-reported malaria measures are subject to potential recall bias. Second, while these data are good measures of private behavior, our dataset provides limited information on how public supply of prevention (via the health system) varies across our sample. Targeted public health interventions will change the relative prices of alternative prevention methods relative to one another and relative to treatment. Future analyses will introduce controls

⁹ Malaria prevalence is defined as the number of cases prior to 2005 divided by village sample size and times 1000.

for access to markets and health care facilities. Third, there may be simultaneity bias in these analyses since this is a cross-sectional dataset and we cannot break the simultaneity between prevalence and prevention. A panel dataset would allow us to distinguish the prevalence elasticity of demand for prevention from the effect of prevention on prevalence.

Case IV: Diarrhea Prevention in Maharashtra, India

We turn to a panel data to address the potential recall and simultaneity biases that may exist in the Tigray and Orissa analyses. We also wish to explore whether prevalence elasticity of prevention holds for other infectious diseases. Thus, we examine the case of diarrhea prevention and control with data from an on-going evaluation of a large scale rural water supply and sanitation program (*Jalswarajya*) in India involving approximately 65,000 individuals from 10,000 households in 250 communities.

The study is collecting multi-scale multivariable data on 10,000 households in 230 communities at four different times – in the dry season and monsoon seasons before the water and sanitation interventions and in the dry season and monsoon seasons after the interventions. Data on biological, socioeconomic, environmental, cultural, and institutional factors that affect child health outcomes are being collected at the individual-, household-, and community-levels. The household and community questionnaires were developed over 12 months and were informed by existing survey instruments in the public domain, our previous surveys, a literature review, feedback from technical advisors and fellow researchers. Survey instruments were extensively tested using focus group discussions and pretests. The final household survey instrument collected about 3000 variables per household, including multiple measures of disease outcomes (*e.g.*, diarrhea by age in the last six months, one month, two weeks, and 48 hours), prevention activities (*e.g.*, use of improved water and sanitation, hand washing, safe water handling), therapeutic behaviors (*e.g.*, home remedies and treatment, doctor and hospital visits), environmental sanitation (*e.g.*, community and household water quality, drainage) and a full accounting of socioeconomic variables (*e.g.*, socio-economic status, health literacy, and demographics). For details on the full study design, see Pattanayak et al. (2005).

Self-reported diarrhea incidence data suggests that 6% of the overall sample and 14% of the children under 5 had diarrhea in the 2 weeks prior to our rainy season (August – September) survey. In the 6 months prior to our dry season survey (April – May 2006), which is our measure of diarrhea prevalence in the study area, we find approximately 2% of the overall sample and 3%

of the under-5 children had suffered from diarrhea.¹⁰ Approximately 8% of households had someone suffer from diarrhea in the past 6 months. Seventy-two percent of diarrhea cases were diagnosed by doctor or nurse while 26% were self-diagnosed.

The costs of diarrhea in a community extend far beyond the costs of treating diarrhea cases, and include the costs of acquiring, treating and storing water as well as the costs of improving sanitation and hygiene. Typical households spend 15 and 30 minutes walking to and waiting at their water source, and 30 minutes walking to the village commons for defecation. About 80% of the community water sources have total coliforms in excess of national standards (national standard is less than 10 total coliforms per 100 mL) and about 40% have e. coli, with an average count of 16 (national standard is zero E.coli). The contamination level is 10 times more in the drinking water vessel inside the house, in spite of the fact that about 67% of the households filter their drinking water, 15% only store it in narrow-mouth containers, 7% store in vessels elevated more than 3 feet off the ground, and 93% wash their vessels on a daily basis. Households also incur costs because they buy and maintain storage tanks outside the house. Furthermore, the typical child who falls ill with diarrhea misses 5 days of school or household chores and spends 4 days in hospital, whereas her primary care giver loses 5 days of work. Of the 3% of that received treatment at a health facility, they spent approximately \$25 on treatment. Home remedies for child diarrhea cases results in expenditures on pills (80%) and or injections (45%).

Our survey generated data on 8 preventive behaviors that are viewed as critical for diarrhea control (Kleinau et al., 2003): using an improved water supply, using a latrine (in lieu of open defecation), boiling water before drinking, filtering water before drinking, storing water in narrow mouthed container, elevating water storage above 3 feet, washing water storage containers on a daily basis, and hand-washing by caregivers at all critical times. Approximately 91% of households engage in at least 2 preventive behaviors. Sixty-seven percent obtain water from improved water sources which include covered public and private wells and public and private taps. Fifteen percent store their drinking water in narrow mouthed containers, and 7 percent elevate the containers above 3 feet. Almost all households wash their water storage on a daily basis. While two thirds of the households filter their water, only 3 percent boil it before drinking. Majority (86%) of the households do not use a sanitation facility, and only 6 percent state that caregivers wash their hands at all 4 critical times that include before preparing food/cooking,

¹⁰ Diarrhea prevalence is defined as number of cases per 1000 villagers in a village who experienced diarrhea 6 months prior to May 2005.

before eating, before feeding children, after changing baby/handling child's feces, and after defecation. Out of 5 averting behaviors of our particular interest (i.e., obtain water from improved source, do not openly defecate, boil water before drinking, store water in narrow mouthed containers, and elevate water containers above 3 feet) - we find that on average households only do one.

We test for prevalence elasticity of prevention by estimating multivariate regression models for each of these behaviors, controlling for socio-demographic, caregiver, economic, and caregiver characteristics. The results are reported in Table 5. We find that households facing higher levels of diarrhea prevalence are more like to obtain water from improved source, use a sanitation facility, boil water before drinking, elevate water storage above 3 feet. However, diarrhea prevalence is negatively correlated with filtering water before drinking, storing water in narrow mouthed containers, washing these containers on a daily basis, and caregivers who wash their hands at all critical times.

Instead of looking at specific preventive behaviors, we might wish to view overall prevention behaviors. A Poisson model of the number of preventive behaviors (inflated for zeros) suggests that behaviors are prevalence elastic *ceteris paribus* (Table 6). A second way of considering overall prevention behaviors lies in using the data we collected on point-of-use microbial water quality, measured in terms of *E.coli* counts.¹¹ The *E.coli* count can be considered as behavioral index of prevention and regressed on prevalence and other controls using a zero-inflated Poisson model. By including preventive behaviors in this model and testing the statistical significance of estimated parameters, we confirm the causal role of behaviors in household production of potable water. As shown in Table 7, we find a negative and significant correlation between diarrhea prevalence and microbial water quality at the point-of-use – suggesting that household respond with lower prevention when prevalence declines, *ceteris paribus*.

¹¹ A microbial water quality test costs about 20% more than the entire household survey, partly because the water sample must be transported in an ice-pack to laboratories in a major industrial center (in this case Pune) and tested all within 24 hours. This restricted our water quality sampling to every other house – or approximately 5000 households – in our overall sample.

Discussion

The analyses reported in this paper are still preliminary. While they will be refined through a whole battery of specification tests, the inherent limitations of the data used as well as the state of the art in economic epidemiology imply that our findings will remain exploratory. Nevertheless, these are among the first empirical analyses based on economic epidemiology and are important in testing and refining this model. Such an iterative exchange between theory and empirics is consistent with some of the major developments in economics (Udry, 2003; Mookherjee, 2005), and is vital if we are to substantially improve our understanding of an inter-disciplinary problem like infectious diseases and the environment.

Despite the preliminary nature of our analysis of four data sets, the findings are robust across scales, regions, and infections – preventive behaviors are prevalence elastic. The fact that households will modify their preventive choices in response to disease prevalence suggests that it may be necessary to accept endemic disease – at a level lower than no intervention – as a second best outcome because of tradeoffs between private and social costs. While public interventions are not as effective as desired, there will be potential savings in private (prevention) costs. Naturally, the dynamic nature of the public-private relationship suggests applying a package of prevention and therapy that offset externalities (risky private behaviors) on an ongoing basis.

While the estimates reported in this paper provide a starting point in a decision-analysis toolkit for infectious disease control, it is critical to assemble empirical data on several other important parameters. For example, meta-analyses on the effectiveness and costs of interventions would be a useful starting point. However, the specificity of diseases such as malaria and diarrhea suggests a significant need for primary analysis from representative countries and regions to (a) estimate size of externalities, (b) examine nature of relationship between preventive and curative behaviors, and (c) estimate the private and public costs of interventions and disease.

We also identify at least four sets of conceptual interdisciplinary research questions that can serve as a platform for extending this research program. First, the prevalence elasticity logic pivots on the assumption of rational choice. As recent advances in behavioral economics suggest, it might be helpful to reconsider the socio-psychological basis of our model of individual behaviors, with rational choice as a special case (Mullainathan and Thaler, 2001).¹² Second, infectious diseases

¹² The basic premise is that neo-classical economics has accomplished less than its promise because it has not fully accounted for: (a) bounded rationality - limited cognitive abilities that constrain human problem

are concentrated in countries and regions in which multiple markets are thin, incomplete or missing. This suggests that we must use household specific shadow prices in understanding behavioral responses (Poulos, 2000). Moreover, if household or community changes to the natural environment affect infection probabilities (*e.g.*, deforestation through fires which affects ARI and malaria), the shadow prices would both depend on environmental variables and now capture the full opportunity costs of prevention (*e.g.*, farm income given up because one must reduce deforestation to reduce malaria). The operational implication for empirical work is fewer exogenous variables and a smaller set of econometric instruments.

Finally, it is imperative to identify key elements of the inter-related dynamics of immunity development, vector ecology, resistance development, and human responses to actual and perceived changes in disease prevalence and the natural environment (Hammer, 1993). The specification challenges of parameterizing such an inter-related system are formidable and were partly responsible for our desire to focus on some tractable concepts such as prevalence elasticity. Nevertheless, more work is needed for specifying tractable models, estimating essential parameters, and relying on numerical simulation methods (in the context of dynamic optimization). In the interim, the research described in this paper presents a stepping stone in understanding how economics can help understand the transmission and potential control of infectious diseases that originate in the environment. To our knowledge, the evidence discussed in this paper represents the first set of rigorous empirical results from developing countries in the analytics-heavy field of economic epidemiology.

solving, (b) bounded willpower - people sometimes make choices that are not in their long-run interest, and (c) bounded self-interests - the comforting fact that humans are often willing to sacrifice their own interests to help others.

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Table 1. Prevalence Elasticity

Variables	Log (Net Use)		Log (Nets Sold)	
	Coef.	p-value	Coef.	p-value
Log (Prevalence)	0.159	0.000	0.520	0.000
GDP index	-7.648	0.004	-8.390	0.031
Enrollment ratio	-0.001	0.976	-0.029	0.251
Corruption	0.347	0.341	1.457	0.002
Political stability	0.407	0.304	0.244	0.676
Number of obs	153		153	
F(5,147)	21.84		43.65	
Prob > F	0.000		0.000	
R ²	0.412		0.546	
Root MSE	2.56		3.53	

Table 2. Prevalence Elasticity (Malaria Ecology)

Variables	Log (Net Use)		Log (Nets Sold)	
	Coef.	p-value	Coef.	p-value
Log (Malaria Ecology)	0.414	0.000	0.532	0.001
GDP index	-8.502	0.005	-13.689	0.002
Enrollment ratio	0.010	0.642	-0.034	0.302
Corruption	0.202	0.644	1.753	0.009
Political stability	0.271	0.501	-0.206	0.751
Number of obs	130		130	
F(5,124)	20.35		21.14	
Prob > F	0.000		0.000	
R ²	0.413		0.427	
Root MSE	2.70		4.16	

Table 3. Tigray, Ethiopia: Effect of Prevalence on Preventive Behaviors

Characteristics	Draining standing water			Indoor spraying			Burning dung/leaves			Taking medicine		
	Coef.	p-value	M.E.	Coef.	p-value	M.E.	Coef.	p-value	M.E.	Coef.	p-value	M.E.
1USD increase in annual household income	-0.00004	0.52	<1%	3.32E-06	0.96	<1%	-0.0002	0.01	<1%	-0.00009	0.24	<1%
1 case per person increase in village-specific annual prevalence	1.1208	0.00	+25%	1.1009	0.00	+13%	0.6822	0.02	+11%	0.8773	0.01	+10%
A household member died from malaria*	1.0337	0.01	+14%	0.0379	0.88	<1%	0.3072	0.15	<1%	0.0033	0.99	<1%
One person increase in household size	0.0087	0.77	<1%	-0.0636	0.11	<1%	0.0546	0.13	<1%	-0.0771	0.06	-1%
One year increase in schooling	-0.0206	0.60	<1%	0.1153	0.02	+1%	-0.0559	0.29	<1%	0.0687	0.17	+1%
Single female head of household	-0.0755	0.67	<1%	-0.0835	0.71	<1%	-0.1282	0.55	<1%	-1.1869	0.01	<1%
Number of obs	792			773			754			767		
Wald X ²	32.93			19.69			17.28			25.33		
Prob > X ²	0.000			0.003			0.008			0.000		
Pseudo R ²	0.048			0.050			0.036			0.06		
Log pseudolikelihood	-328.887			-185.932			-231.154			-182.679		

Table 3. Tigray, Ethiopia: Effect of Prevalence on Preventive Behaviors (continued)

LHS = Take Any Prevention Activity	Coef.	p-value
1USD increase in annual household income	-0.00003	0.554
1 case per person increase in village-specific annual prevalence	1.23	0.000
A household member died from malaria*	0.54	0.058
One person increase in household size	0.01	0.741
One year increase in schooling	0.002	0.782
Single female head of household	0.01	0.932
Constant	0.48	0.021
Number of obs	803	
Wald $\chi^2(6)$	23.96	
Prob > χ^2	0.0005	
Pseudo R ²	0.04	
Log pseudolikelihood	-332.70	

Table 4. Keonjhar, Orissa – Malaria Prevention

Variable	Take Any Prevention Activity			Sleep Under Bed Nets		
	Coef.	p-value	M.E.	Coef.	p-value	M.E.
Village level malaria prevalence prior to 2005	0.002	0.020	0.0007	0.004	0.021	0.002
Belong to scheduled caste / tribe	-0.67	0.000	-0.16	-0.59	0.000	-0.23
Caregiver's age	-0.00009	0.987	-0.00002	0.008	0.007	0.003
Caregiver can read newspapers	0.90	0.008	0.18	0.69	0.002	0.27
Own land	0.56	0.000	0.16	0.70	0.000	0.26
Own radio	-0.18	0.119	-0.05	0.45	0.000	0.18
Live in a good house	1.09	0.000	0.21	0.47	0.025	0.19
Distance to nearest health facility	0.002	0.218	0.0007	-0.001	0.632	-0.0004
Had major illness / death in the family last year	-0.13	0.581	-0.04	0.09	0.637	0.04
Id mosquitoes as key cause for malaria	0.74	0.022	0.26	0.54	0.112	0.19
Id stagnant water as key cause for malaria	-0.05	0.839	-0.02	-0.33	0.163	-0.12
Constant	-0.28	0.557	---	-1.86	0.000	---
Number of obs	587			587		
Wald X^2	178.22			262.38		
Prob > X^2	0.0000			0.0000		
Pseudo R^2	0.15			0.23		
Log pseudolikelihood	-288.66			-307.03		

Table 5. Maharashtra, India – Diarrhea Prevention Behaviors

Variable	Improved Water Supply		No Open Defecation		Boil Water Before Drinking		Filter Water Before Drinking	
	Coef.	p-value	Coef.	p-value	Coef.	p-value	Coef.	p-value
Historical diarrhea prevalence 6 months before survey at the village level	12.57	0.000	10.67	0.002	10.55	0.000	-19.24	0.000
Household size	-0.02	0.032	0.001	0.911	-0.02	0.219	-0.03	0.000
Belong to scheduled caste / tribe	-0.20	0.015	-0.23	0.005	-0.12	0.148	-0.13	0.019
Caregiver's age	-0.002	0.258	0.01	0.001	0.006	0.127	0.007	0.003
Caregiver can read newspapers	0.30	0.000	0.48	0.000	0.44	0.000	0.25	0.000
Had major illness / death in the family last 3 months	-0.17	0.062	0.08	0.381	0.17	0.139	0.03	0.713
Live in a best house	0.21	0.013	0.62	0.000	0.17	0.054	0.12	0.096
Own land	-0.05	0.413	0.02	0.845	-0.03	0.659	-0.06	0.222
Own radio	0.04	0.411	0.19	0.000	0.03	0.766	-0.05	0.400
Distance to nearest health facility	-0.003	0.000	-0.002	0.068	-0.002	0.321	-0.002	0.003
Constant	0.46	0.000	-1.89	0.000	-2.46	0.000	0.72	0.000
Number of obs	8600		8600		8594		8594	
Wald $X^2(10)$	93.88		153.72		86.29		127.67	
Prob > X^2	0.0000		0.0000		0.0000		0.0000	
Pseudo R^2	0.07		0.11		0.07		0.05	
Log pseudolikelihood	-5086		-3081		-953		-5201	

Table 5. Maharashtra, India – Diarrhea Prevention Behaviors (continued)

Variable	Drinking Water Storage with Narrow Mouth		Elevate Drinking Water Storage Above 3ft		Wash Drinking Water Storage Containers Daily		Adults Wash Hands At All Critical Times	
	Coef.	p-value	Coef.	p-value	Coef.	p-value	Coef.	p-value
Historical diarrhea prevalence 6 months before survey at the village level	-11.37	0.000	3.07	0.119	-9.04	0.001	-3.92	0.139
Household size	-0.007	0.425	0.03	0.002	-0.006	0.527	0.003	0.763
Belong to scheduled caste / tribe	-0.03	0.601	0.006	0.932	-0.02	0.755	-0.10	0.136
Caregiver's age	-0.006	0.018	0.0002	0.947	-0.0001	0.970	0.0003	0.920
Caregiver can read newspapers	-0.09	0.081	0.04	0.592	-0.03	0.563	0.16	0.007
Had major illness / death in the family last 3 months	0.46	0.000	-0.15	0.206	0.02	0.812	-0.05	0.657
Live in a best house	-0.39	0.000	0.24	0.006	-0.03	0.676	0.06	0.430
Own land	-0.16	0.001	0.01	0.809	0.08	0.131	-0.06	0.325
Own radio	-0.06	0.418	0.04	0.530	-0.07	0.276	-0.009	0.898
Distance to nearest health facility	0.0007	0.191	0.0007	0.387	0.0009	0.324	-0.002	0.025
Constant	-0.53	0.000	-1.82	0.000	1.65	0.000	-1.49	0.000
Number of obs	8323		8594		8538		8600	
Wald $X^2(10)$	125.92		24.33		19.22		22.90	
Prob > X^2	0.0000		0.0068		0.0375		0.0111	
Pseudo R^2	0.04		0.01		0.02		0.01	
Log pseudolikelihood	-3367		-2077		-2110		-1974	

Table 5. Maharashtra, India – Diarrhea Prevention Behaviors (continued)

LHS = Take Any Prevention Activity	Coef.	p-value
Historical diarrhea prevalence 6 months before survey at the village level	7.22	0.014
Household size	-0.01	0.229
Belong to scheduled caste / tribe	-0.23	0.002
Caregiver's age	-0.003	0.146
Caregiver can read newspapers	0.32	0.000
Had major illness / death in the family last 3 months	0.06	0.532
Live in a best house	0.32	0.000
Own land	-0.04	0.549
Own radio	0.02	0.781
Distance to nearest health facility	-0.002	0.001
Constant	0.76	0.000
Number of obs	8600	
Wald $X^2(10)$	109.17	
Prob > X^2	0.0000	
Pseudo R^2	0.05	
Log pseudolikelihood	-4391	

Table 6. Poisson Regression Results of Count of Averting Behaviors

Variable	Coef.	p-value
Historical diarrhea prevalence 6 months before survey at the village level	4.63	0.000
Household size	-0.004	0.320
Belong to scheduled caste / tribe	-0.13	0.001
Caregiver's age	0.0001	0.921
Caregiver can read newspapers	0.20	0.000
Had major illness / death in the family last 3 months	0.06	0.152
Live in a best house	0.19	0.000
Own land	-0.05	0.087
Own radio	0.05	0.020
Distance to nearest health facility	-0.002	0.001
Constant	-0.05	0.406
Number of obs	8600	
Wald chi2(10)	150.17	
Prob > chi2	0.0000	
Log pseudolikelihood	-10160	

Table 7. Poisson Regression Results of Count of *E. coli* present at point of use

Variable	Coef.	p-value	Coef.	p-value
Use improved water supply	-0.34	0.005		
Don't openly defecate	-0.52	0.035		
Drinking water storage with narrow mouth	-0.30	0.064		
Boil water before drinking	-0.43	0.409		
Filter water before drinking	0.18	0.170		
Elevate drinking water storage above 3ft	-0.26	0.262		
Adults wash hands at critical times	0.44	0.017		
Count of averting behaviors			-0.35	0.000
Household size	-0.06	0.044	-0.06	0.032
Belong to scheduled caste / tribe	0.29	0.034	0.26	0.055
Caregiver's age	0.01	0.312	0.01	0.159
Caregiver can read newspapers	-0.19	0.120	-0.15	0.231
Had major illness / death in the family last 3 months	0.24	0.309	0.20	0.383
Live in a best house	0.13	0.604	0.14	0.575
Own land	0.37	0.004	0.37	0.003
Own radio	-0.01	0.936	-0.05	0.758
Distance to nearest health facility	0.00	0.966	0.00	0.864
Historical diarrhea prevalence 6 months before survey at the village level	-6.38	0.182	-8.95	0.066
Count of <i>E. coli</i> present at point of distribution	0.00	0.002	0.00	0.002
Constant	2.87	0.000	3.01	0.000
Number of obs	4015		4148	
Wald $X^2(18) / (12)$	81.42		65.7	
Prob > X^2	0.0000		0.0000	
Log pseudolikelihood	-131446		-135958	

Figure 1. Map of Tigray

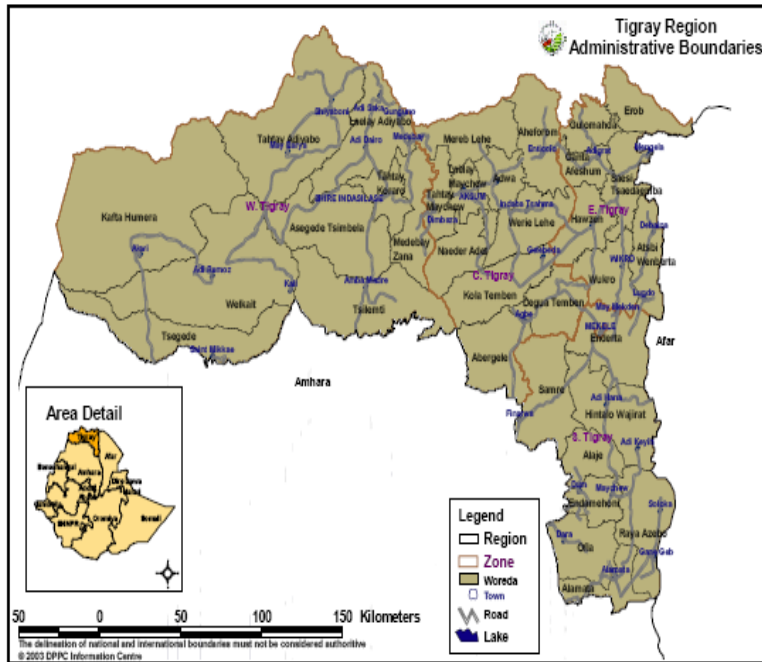
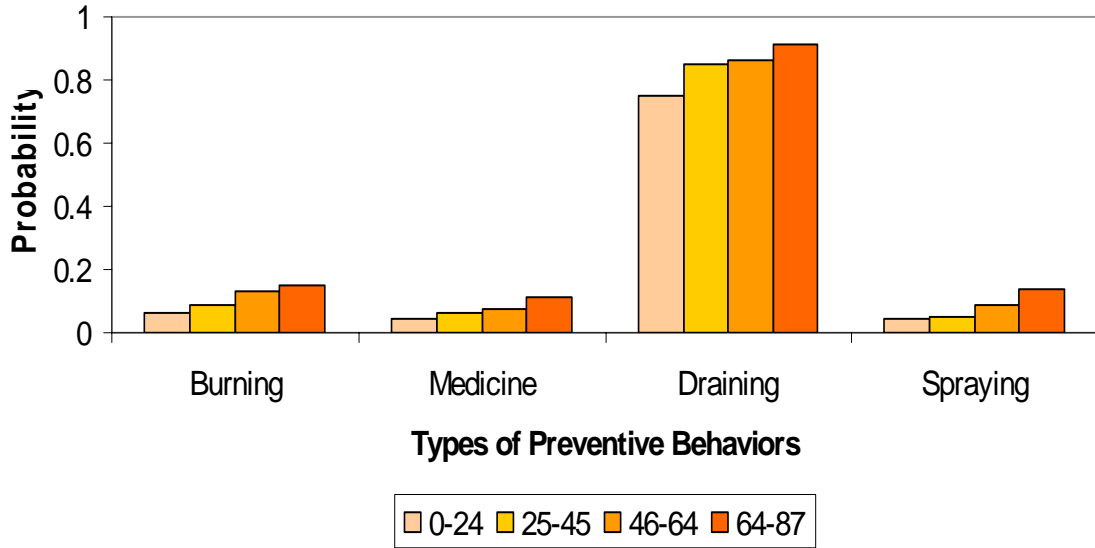


Figure 2. Preventive Behaviors by Prevalence



Appendix: Simple Analytics of Infectious Disease & the Environment

Philipson (1999) posits that the demand for goods that prevent illness, a , depend on both prevalence, I , and the price of the preventive good, p : $a(I, p)$. Demand decreases with price ($\frac{\partial a}{\partial p} < 0$) and increases with prevalence ($\frac{\partial a}{\partial I} > 0$). He also posits that prevalence is increasing in price, so $a = a(p, I(p))$. He goes on to show that the positive prevalence response of demand dampens the price response of demand. This prevalence response is shown to affect the epidemiology of disease, the effectiveness of public economic policy tools, and the appropriate measure of welfare effects.

The environmental economics literature's attention to averting behavior (e.g., Bartik 1988, Courant and Porter 1981, Hartford 1984, Harrington and Portney 1987) may offer additional insights by bridging the gap between Philipson's stylized treatment of averting behavior on the one hand and Gersovitz & Hammer's (2003) need for empirically based parameters for numerical solutions on the other. In particular, we develop a static model of individual (or unitary household) decisionmaking regarding averting behavior and environmental quality to explore the reasons why we may observe prevalence elasticity at the aggregate level. While both Philipson and GH use dynamic models of the social perspective, the importance of dynamics at the individual level are likely to vary with extent to which health risk factors are endogenous or exogenous. In future model extensions, this preliminary model will be expanded to include other time periods.

We adapt GH's equation (24) to be an individual health risk function:

$$(1) \quad i = \alpha_1 \alpha_2 n m(I)$$

where i is an indicator of whether the individual has malaria or not in the given time period; n is the number of mosquitoes that the individual encounters; α_1 is the number of bites by the average mosquito the individual encounters per unit time; $m(I)$, the proportion of mosquitoes that are infected, depends on the prevalence of malaria, and α_2 is the proportion of bites by infected mosquitoes that lead to a human infection. $n m(I)$ is the number of number of infected mosquitoes that the individual encounters each time period. $\alpha_1 \alpha_2$ is the proportion of bites that are infectious.

Malaria risk is endogenous because private behaviors can affect the risk in numerous ways. First, private averting activities can affect malaria risk by reducing α_1 or α_2 , depending on the type of averting behavior they engage in. Private averting behaviors that reduce α_1 , including sleeping under an insecticide-treated or untreated net, are a_1 . Averting behaviors that reduce α_2 , including intermittent preventive therapy with artemisin combination therapy or another effective therapy, are a_2 . *Ceteris paribus*, increased averting behavior reduces malaria risk: $\frac{\partial i}{\partial a_1} < 0$ and $\frac{\partial i}{\partial a_2} < 0$.

In this model, a_1 and a_2 are substitutes for lowering I .

Malaria risk is also affected by private choices affecting the mosquito habitat. a_3 include private vector control activities, such as using insect repellent and draining areas of standing water that may serve as breeding areas could reduce the number of mosquitoes that the individuals is exposed to, m . If infected mosquitoes are evenly distributed over space, the number of infected mosquitoes that the individual is exposed to will also decrease. These private vector control activities are assumed to have a smaller impact on the mosquito population than public vector control activities because they are implemented over a relatively small area and thus affect local mosquito populations only. The individual is assumed to be maximizing utility, which is simply the leisure, L , that she can consume subject to a income constraint. Malaria affects the quality of L and causes the individual to miss work, S . Thus, the full-income constraint is

$$(2) \quad y = [T - L(i) - S(i)] - p_1 a_1 - p_2 a_2 - p_3 a_3 - p_i i$$

where w is the wage rate, T is total time available, L is leisure, and S is the number of days of work missed due to an episode of malaria. p_1 , p_2 , and p_3 are the prices of a_1 , a_2 , and a_3 , respectively. p_i is the cost of treating an episode of malaria.

The individual chooses a_1 , a_2 , and a_3 to maximize utility:

$$(3) \quad \begin{aligned} & \text{Max} \ell = U(L) \\ & + \lambda [y - w\{T - L(i) - S(i)\} \\ & + \sum_{k=1}^3 p_k a_k - p_i i \{\alpha_1(a_1) \alpha_2(a_2) n(a_3) m(I)\}] \end{aligned}$$

The first order conditions are:

$$(4) \quad \frac{\partial \ell}{\partial L} = \frac{\partial U}{\partial L} - \lambda w = 0$$

$$(5) \quad \frac{\partial \ell}{\partial a} = \lambda [p_j - \frac{\partial i}{\partial a_j} \{p_i + w(\frac{\partial L}{\partial i} + \frac{\partial S}{\partial i})\}] = 0, \quad \forall j \in (1,2,3)$$

Equation (4) shows that individuals will choose L to equate the marginal benefit of increasing consumption with the marginal cost, which is measured in terms of wages. Similarly, equation (5) shows that the individual will choose averting activities to equate their marginal cost to the marginal benefit of reducing the malaria, measured in terms of cost-of-illness, pain and suffering, and work loss.

The solution to (3) implies an indirect utility function, $V^* = V(i, p_1, p_2, p_3, p_i, w, i)$. Previous analyses of averting behavior (see Larson and Gnedenko 1998), show that the optimal choice of each of the averting behaviors (indexed by $j \in (1,2,3)$), a_j^* , is the expenditure minimizing level of averting activity, given the optimal level of endogenous malaria risk, i^* :

$$(6) \quad a_j^* = -\frac{\partial V / \partial p_j}{\partial V / \partial y} = a_j^*(p_j, I, i^*[\alpha_1, \alpha_2, n, m, I])$$

We can use this general function to explore the relationship between malaria prevalence, I , and the optimally chosen level of averting behaviors, a_j^* . Differentiating equation (6) with respect to I yields:

$$(7) \quad \frac{\partial a_j^*}{\partial I} = \frac{\partial a}{\partial I} \Big|_{i^* \text{ constant}} + \frac{\partial a}{\partial i} \frac{\partial i^*}{\partial n} \frac{\partial n}{\partial I}$$

The sign of this term may be positive or negative, depending on several factors. Prevalence elastic behavior would suggest a strong positive effect; therefore, it is of particular interest to understand what factors contribute to a positive sign. By the structure of the model, the first term in equation (7) is positive, as is the term $\frac{\partial a}{\partial i}$. The sign of equation (7) thus depends importantly

on the sign of $\frac{\partial i^*}{\partial n} \frac{\partial n}{\partial I}$. As discussed by Courant and Porter (1981) [see equation 13] and Larson and Gnedenko (1999) [see footnotes 20-22] in similar contexts, the sign of this last combined term depends importantly on how prevalence affects the marginal productivity of preventive behavior. If an increase in malaria prevalence has no effect on this marginal productivity, then the overall sign of Equation (7) should be positive, as long the marginal utility of reducing malaria risk is positively related to the consumption of other goods (which is required for it to be a normal good). If an increase in prevalence has a *positive* effect on the marginal productivity of averting behaviors, then other things equal, the magnitude of $\frac{\partial a_j^*}{\partial I}$ should be even more positive (i.e., more prevalence elastic). It is not hard to imagine situations where the productivity of averting activities, such as using bed nets, is greater when malaria is more prevalent in the community. Van Houtven and Pattanayak (1999) suggest that the analytics are trickier than either author suggests because the sign depends on a multiplicative and additive combination of at least a half-a-dozen terms with unknown signs and sizes.