Deforestation and malaria: Revisiting the human ecology perspective

Subhrendu K. Pattanayak **and** ***Junko Yasuoka***[1]

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1. Introduction

The ecological basis for disease dates at least as far back as 400 B.C. to Hippocrates's writing of On Airs, Waters, and Place. As Wilson (1995) clarifies, our understanding and therefore control of diseases would be inadequate without an "ecological" perspective on the life cycles of parasitic microorganisms and the associated infectious diseases. As Smith et al. (1999; p 583) contend, "many of the critical health problems in the world today cannot be solved without major improvement in environmental quality." In this chapter we focus on malaria because its transmission (and control) has clear links to ecosystem changes that result from natural resource policies such as land tenure, road building, and agricultural subsidies. The resulting ecosystem change has a tremendous influence on the pattern of diseases such as malaria (Martens 1998; Molyneux 1998; Grillet 2000). This is partly because, of all the forest species that transmit diseases to human beings, mosquitoes are among the most sensitive to ecosystem change: their survival, density, and distribution have been altered by environmental changes caused by different land transformations. While we agree that 'ecological lenses' can help improve our understanding of disease prevention, we use this chapter to articulate a particular ecological perspective – a human ecology viewpoint that puts human behavior change front and center.

In the last decade, we have seen a series of widely cited papers drawing out the connections between ecosystem change and diseases, many of which are synthesized in the 2005 Millennium Ecosystem Assessment (Corvalan et al., 2005a; Corvalan et al. 2005b; Campbell-Lendrum, 2005; Patz et al., 2005; McMichael et al., 1998). This renewed interest in the more distal causes of disease reflects in part the emergence of new fields such as 'sustainability science' (Kates et al., 2001) and 'biocomplexity' (Wilcox and Colwell, 2003) that argue for "a more realistic view [requiring] a holistic perspective that incorporates social as well as physical, chemical, and biological dimensions of our planet's systems." The resurgence also reflects the growing importance of fields of social epidemiology (e.g., Berkman and Kawachi, 2000; Oakes and Kaufman, 2006) that draw on Rose's (1985) call to examine the *cause of cause* and resolve the *prevention paradox* in developing a population strategy for health.

In joining this growing chorus, we focus on an older human ecology tradition (Wessen, 1972; McCormack, 1984), which posits that (a) we humans modify our natural environment, sometimes increasing disease risks, and (b) we ultimately adapt to the new disease risk environment. Two stylized, yet complicating, facts emerge from this viewpoint (Pattanayak et al., 2006a). First, disease prevention behaviors (including ecosystem changes that modify disease risks) respond to disease levels, suggesting a dynamic feedback exposure and control. Second, individuals and households typically will not consider how their private actions affect public health outcomes and therefore will make socially inappropriate and

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[1] Address correspondence to Subhrendu K. Pattanayak, Fellow and Senior Economist in Public Health and Environment, RTI International and Research Associate Professor at North Carolina State University, subhrendu@rti.org or Tel (919) 541-7355. Junko Yasuoka is a Post-Doctoral Fellow Harvard School of Public Health, Department of Population and International Health. Many ideas reflected in this paper are based on discussions with Erin Sills, Keith Alger, Gene Brantley, Kelly Jones, Christine Poulos, and George Van Houtven. We are grateful to Carol Colfer for her encouragement and, most of all, patience with the development of this chapter. This chapter was completed while Pattanayak was a Visiting Scholar at the University of California, Berkeley.

sub-optimal choices, unless convinced otherwise. Typically, some combination of government laws (e.g., regulation), community norms (information), and markets prices (compensation) help narrow this wedge between private and 'optimal' social behaviors. This modification of domain to now more systematically human behavior is consistent with complaints that the ecology-and-health approach takes a predominantly biophysical approach that can easily overlook the social, cultural, and economic driving forces that are crucial to understanding anthropogenic ecosystem disruptions and their human health impacts (McMichael, 2001; Parkes et al. 2003).

In this chapter, we focus on malaria and deforestation, rather than a sweeping review of broad links between infectious diseases and ecosystem change to keep things manageable and present somewhat in-depth arguments. We restrict ourselves to malaria not only because its transmission is clearly linked to ecological changes, but because it is a major (if not the major) health concern in the tropics (Hay et al., 2004). We focus on deforestation because it is a major development policy concern and often heralds many other 'malaria-causing' land use changes (Pattanayak et al., 2006c).

The remainder of the chapter is organized as follows. In Section II, we briefly review the literature on ecology of infectious diseases. In Section III, we re-introduce the human ecology perspective for better understanding the role of humans in land use change as well as in a variety of behaviors to prevent (e.g., sleep under nets, take prophylaxis) and treat (e.g., seek medical care, follow the drug regimen) malaria. In Section IV, we draw out the empirical implications of such a strategy, using our own fieldwork and secondary data sets. Finally, we conclude with a call for systematic environmental and health impact assessments that rely on inter-disciplinary longitudinal studies.

2. A brief synthesis of mosquito ecology and malaria epidemiology

While the impacts of ecosystem change on health are diverse and longstanding, its rate and geographical range have increased markedly over the last few decades. Different kinds of environmental changes have resulted from a wide variety of human activities, including deforestation, agricultural activities, plantations, logging, fuel wood collection, road construction, mining, hydropower development and urbanization (Walsh et al. 1993, Patz et al. 2000, Patz et al. 2004). It is the process of clearing forests and subsequent land transformation that alters every element of local ecosystems, including microclimate, soil and aquatic conditions, and most significantly, the ecology of local fauna and flora. These in turn have profound impact on the survival, density and distribution of human disease vectors and parasites (Martens 1998, Grillet 2000), including influences on breeding places, daily survival probability, density, human-biting rates, and incubation period. Thus, the altered vector/parasite ecology modifies the transmission of vector-borne diseases such as malaria, Japanese encephalitis and filariasis (Sharma and Kondrashin 1991, Walsh et al. 1993).

Numerous country and area studies have described how the density and distribution of local vector species have been altered due to ecosystem change, and some longitudinal studies have shown that the change in vector ecology has altered local disease incidence and prevalence (Sharma and Kondrashin 1991, Patz 2000). However, the mechanism linking ecosystem change, vector ecology and vector-borne diseases is still unclear. We draw on a paper by Yasuoka and Levins (forthcoming) that examines the mechanisms linking deforestation, anopheline ecology, and malaria epidemiology by drawing together 60 examples of changes in anopheline ecology as a consequence of deforestation and agricultural development in Latin America, Africa, and South and Southeast Asia.

Massive clearing of forests has enormous impacts on local ecosystems and human disease pattern. It alters microclimates by reducing shade, altering rainfall patterns, augmenting air movement, and changing the humidity regime (Reiter 2001). It also reduces biodiversity and increases surface water availability through the loss of topsoil and vegetation root systems that absorb rain water (Chivian ed. 2002). For anopheline species that breed in shaded water bodies, deforestation can reduce their breeding habitats, thus affecting their propagation. On the other hand, some environmental and climatic changes due to deforestation can facilitate the survival of other anopheline species, resulting in prolonged seasonal malaria transmission (Kondrashin et al. 1991).

As shown in Table 1 (drawn from Yasuoka and Levins), different land transformations have different impacts on local ecosystem and disease pattern. For example, rubber plantation increased local major malaria vectors in all four cases in Malaysia and Thailand. In Malaysian hilly areas, forest clearance for rubber plantation, which started early in the 1900s, exposed the land and streams to the sun and created breeding places for *An. maculatus*, which led to an increase in this species and a marked rise in the incidence and severity of malaria (Cheong 1983). Cyclic malaria epidemics in Malaysia over 50 years are correlated with rubber replanting in response to market fluctuations (Singh and Tham 1990). Another example is in Chantaburi, Thailand, where the land was transformed to rubber plantation and other fruit tree cultivations, such as rambutan, durian and mangosteen spurred by high markets between 1974 and 1984. The consequent ecological changes favored *An. dirus*, which demonstrated its greatest capability for adaptation in circumstances of rubber and fruit tree cultivations. As a result, local malaria reemerged and malaria transmission was established at high levels (Rosenberg et al. 1990).

All papers on the development of irrigation systems reported an increase in the density of major vectors and following increase in malaria incidence. For example, irrigation schemes developed by the Mahi-Kadan Project across the River Mahi in India in 1960 had typical management problems, including over-irrigation, lack of proper drainage, weedy channels, leaking sluice gates, and water-logged fallow fields. These created extended breeding habitats for *An. culicifacies*, which resulted in an increase of the vector and malaria transmission.

In some cases, different anopheline species responded differently to the same land transformation. For example, due to deforestation for rice cultivation and irrigation development in Sri Lanka, *An. annularis, An. barbirostris*, *An. culicifacies*, and *An. varuna* decreased, while *An. jamesii* and *An. subpictus* increased, and *An. nigerrimus* and *An. vagus* did not change substantially (Amerasinghe et al. 1991; Konradsen et al. 2000). Not only species abundance, but also species involvement in malaria transmission changed markedly during the land transformation. *Anopheles annularis*, *An. culicifacies* and *An. vagus* were the main vectors during the construction phase and the first irrigation year. *Anopheles subpictus* was playing a major role in the second and third years, when rice fields were fully irrigated. Throughout the process, *An. culicifacies* demonstrated continuous involvement in malaria transmission.

Other cases demonstrated species replacement. Land use such as cassava and sugarcane cultivations, which need little water and provide little shade, often create unfavorable environment for anophelines, especially those which require shade. In Thailand, the transformation from forest to cassava or sugarcane cultivations eliminated shady breeding habitats for the primary vector species, *An. dirus*, but created widespread breeding grounds for *An. minimus*, which have greater sun preference and was the predominant species throughout the year. Consequently, malaria transmission among resettled cultivators became high (Prothero 1999).

We also see that same kind of land transformation could result in totally different malaria situations, depending on locality and ecological characteristics of local vector species. For example, deforestation followed by development of coffee plantations in southeast Thailand favored the breeding of *An. minimus* and made the previously malaria-free region to hyperendemic (Suvannadabba 1991). On the contrary, in Karnataka, India, large-scale deforestation for coffee plantations reduced seepages, which were the principal breeding sites for An. fliviatilis, a vector responsible for hyper-endemic malaria in the region. As a result, this vector population completely collapsed, and malaria disappeared from the area (Karla 1991).

Deforestation for mine development is one of the examples that not only create breeding sites, but also significantly increase human contacts with vectors. Where settlement and mining activities took place in the Amazon, *An. darlingi* increased because of the increase in breeding sites, including borrow pits after road or settlement constructions, drains, and opencast mine workings. As a result, malaria, which was present in the Amazon's indigenous population, was spread to immigrants and miners (Conn et al. 2002).

In summary, the changes in anopheline density and malaria incidence are both varied and complex, depending on the kind of land transformation, ecological characteristics of local mosquitoes, and altered human behavior (to be discussed further). Some key findings include:

- Some anopheline species were directly affected by deforestation and/or subsequent land use, some favored or could adapt to the different environmental conditions that were created, and some invaded and/or replaced other species in the process of development and cultivation.
- Malaria incidence fluctuated according to different stages of development, changes in vector density, and altered human contact patterns with vectors.
- More mosquitoes (vector density or variety) were neither a necessary nor a sufficient condition for increases in malaria incidence. In fact, inverse relationships between the vector abundance and disease incidence have been reported from different regions (Ijumba and Lindsay 2001, Amerasinghe 2003), presumably because of human adaptations (see next).
- In general, a complex set of macroeconomic (changes in terms of trade), demographic (*e.g.*, migration), policy (e.g., colonization of forest frontiers) and behavioral (e.g., 'malaria literacy and knowledge') factors underlie the ecosystem changes and land transformations that influence mosquito ecology and malaria epidemiology (Sharma and Kondrashin 1991; Molyneux 1998). We turn to these considerations in some detail next.

3. Revisiting the human ecology perspective

If ecosystem changes impact mosquito density and activity, and possibly malaria incidence, then environmental management (e.g., vegetation management, modification of river boundaries, drainage of swamps, reduction of standing water, oil application etc.) could reverse these trends. Even though insecticide-treated bed nets and indoor residual spraying of insecticides are the predominant vector control tools, there is growing support for the management of vegetation and water bodies in light of increasing resistance to insecticides and antimalarials (Lindsay and Birley, 2004). Keiser et al.'s (2005) review of 24 environmental management studies suggests that environmental management can reduce malaria risk ratio by 88% (compared to 79.5% for human habitation modifications, for example).

Furthermore, if these are indeed modifiable behavioral causes, it should be possible to induce these behaviors. Yasuoka et al. (2006a) conducted a 20-week pilot education program to improve community knowledge and mosquito control with participatory and non-chemical approaches in Sri Lanka. They evaluated their program effectiveness using pre-educational and post-educational surveys in two intervention and two comparison villages. Their controlled intervention shows that participatory education program led to improved knowledge of mosquito ecology and disease epidemiology, changes in agricultural practices, and an increase in environmentally sound measures for mosquito control and disease prevention. The success of the intervention was attributed to four 'human ecology' characteristics: a community-based education that enhanced residents' understanding of the mosquito-borne disease problems in their own community, a participatory approach that allowed participants to gain hands-on experiences with actions to be taken, using non-chemical measures that decreased environmental and health risks in residential areas and paddy fields, and an approach that required no cost or extensive instruments. Furthermore, this community-based approach suppressed the density of adult Anopheles in the southwest monsoon season, though little impact was detected on Culex and Aedes densities (Yasuoka et al., 2006b).

Vegetation and water management, however, are just one class of human behaviors that impact the transmission and control of malaria. The links between ecosystem change, vector ecology and disease epidemiology all depend critically on human density, gender ratio, immigration of non-immune people, and knowledge, attitudes and practices primarily because they alter the pattern and frequency of human contacts with vectors. Furthermore, a recent special colloquium of the International Society of Ecosystem Health (Patz et al., 2004) suggests that malaria can be exacerbated by a broad array of land use drivers and underlying human behavioral factors beyond changes to the biophysical environment. These include movement of populations, pathogens, and trade; agriculture; and urbanization. Deforestation features prominently in this review and is closely linked to many of these mechanisms.

Pattanayak et al. (2006b) underscore this behavioral aspect of malaria control and present four reasons why it is important to understand the role of deforestation from a policy and planning perspective. These include:

1. Deforestation is not merely the exogenous (remote control) removal of forest cover. It is the beginning of an entire chain of activities, including forest clearing, farming, irrigation, livestock, and non-timber forest product collection, that have ecological (vector habitat) as well as behavioral (exposure and transmission) consequences for malaria.
2. Deforestation is an integral part of life and the landscape in many parts of the world with high malaria rates (Donohue, 2003; Wilson, 2001). Consequently, sustainable forest management has become an important policy goal, as donor agencies and local policy makers take a more integrated view of people in the natural landscape. The resulting changes in land cover, as well as changes in how people interact with the forest, have implications for malaria. Thus, conservation policies aimed at slowing deforestation will impact malaria (Taylor, 1997; and Walsh et al., 1993).
3. Millions of rural households depend directly on a wide variety of forest products and services (Byron and Arnold, 1999). By lowering local people’s natural wealth, deforestation can reduce household capacity to invest in health care and pay for malaria prevention and treatment. At the same time, deforestation may increase the wealth of other households, who will then be better able to avoid and cure malaria.
4. Deforestation and malaria are central elements of the vicious cycle of poverty in rural areas of developing countries. In simplistic terms, malaria could be considered to “cause” deforestation, because malaria can make people poorer and poverty has been found to “cause” deforestation under some conditions. In reality, the linkages are more complex and site-specific.

These ideas lead us to a human ecology framework for understanding the links between deforestation and malaria. Human ecology involves the study of human–environment interactions and extends notions of ecology and health by explicitly traversing boundaries between “nature and culture” and “environment and society” (Parkes et al., 2003). Others have labeled these the ‘environmental health’ or the ‘ecology and health’ (Aron and Patz, 2001) perspectives. As Parkes et al. (2003) clarify, ultimately all these fields converge on three themes:

(a) integrated approaches to research and policy,

(b) methodological acknowledgment of the synergies between the social and biophysical environments,

(c) incorporation of core ecosystem principles into research and practice

Specific to malaria, we need a shift in the view of humans as passive or constant factors in malaria epidemiology to a view in which people are very active factors (actors) in causing significant changes in epidemiological patterns (Wessen, 1972; MacCormack, 1987). The centrality of human behavior is confirmed by the number of instances in which human behaviors show up in Figure 1 in this chapter and in the Patz et al. (2004) review.

4. Empirics of human ecology: Approach and evidence

In this section we present an initial attempt to examine the importance of human behaviors in malaria transmission and control, and recognize the “active” (dynamic) aspects of human behavioral response. Omitting behavioral responses from any analysis of malaria and ecosystem change would result in a classic case of confounding. Human behavior in this case has all attributes of a confounding factor because it is (a) correlated with the outcome and the risk factor, (b) not necessarily in the causal chain, and (c) very likely to be unbalanced across the different levels of risks. As such behavioral confounders can mimic the risk factor and mask the ecological relationship we are attempting to discover.

What does this mean in practical terms? If we are, for example, using cross-sectional or time series variation in data on deforestation and malaria only, we will face what is labeled an “omitted variable” problem in statistics/econometrics. This problem leads to biased inferences and inconsistent estimates of policy parameters because the real cause is an omitted variable, e.g., the in-migration of susceptible sub-populations. A second related and possibly more pernicious issue is that of endogeneity or reverse causality (or simultaneity). Consider an example from Sawyer (1993) to better understand this ‘endogeneity’ bias. High rates of malaria can encourage forms of land use in which men work as day laborers (in logging or ranching), allowing their wives and children to live in towns with relatively lower threat of malaria, rather than establishing family farms. It in such a situation is often difficult to disentangle the causal role of deforestation in malaria transmission.

To further investigate the empirical implications of these ‘behavioral’ or ‘human ecology’ models, we offer two simple tests that are conducted at three different scales. First, we compare a simple regression model of malaria and deforestation (‘naïve model’) to model including linear behavioral controls (‘linear controls model’). Second, we compare the same naïve model to one where the behavioral factors are used as determinants of deforestation or the ‘endogenous’ risk exposure. Behavior in this case is an instrument for the deforestation risk (the instrumental variable [IV] model). Economic theory provides one basis for identifying variables that can explain deforestation and thus serve as instruments (Sills and Pattanayak, 2006).

Arguably the naïve model is a bit of straw man, but it allows us to investigate the importance of a human ecology strategy. We conduct these evaluations at three scales: a micro analysis of child malaria and community deforestation (case from Indonesia), a meso analysis of regional malaria and regional deforestation (case from Brazil), and a macro analysis of national malaria and deforestation. Data limitations preclude the use of accurate behavioral indicators and force us to use proxy variables.[2] Thus, our analysis should be considered as preliminary, and therefore illustrative of the overarching human ecology approach proposed here.

4a. Macro analysis using global data from 120 countries

In this case study, we examine the macro level correlation of malaria and forest using a global data set. Pattanayak et al. (2006) describe the combination of data from 5 sources to produce a global malaria dataset and use it to examine how disease prevention behaviors respond to disease levels. The World Health Organization’s Global Health Atlas provides data on a range of malaria variables, including the number of cases, for up to 195 countries from 1990 to 2004. The World Development Reports provide data on forest cover in 1990 and the annual rate of increase from 1990 to 2000. We obtain behavioral

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[2] If the measurement error (because of the use of proxy variables) is of the classical variety – i.e., uncorrelated with the regression error – then we would face an attenuation bias (make the correlation seem smaller than it is). Data weakness is not the main problem here. Instead, we would argue that the paucity of good data is ultimately because of inadequate attention to the human ecology perspective in empirical analysis – both statistical estimation and numerical simulation.

proxies from three other sources. First, data from the 2001 Human Development Report (HDR) provides measures of economic conditions (per capita GDP) and social conditions (adult literacy rates, educational enrollment rates, and life expectancy). Second, Kaufmann et al. (2003) provide data on political stability, voice and accountability, and control of corruption. Additionally, we also include a malaria ecology index to capture vector ecology and climatic factors (Kiszewski et al., 2004). 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. Descriptive statistics and other details of the data compilation and synthesis are included in Pattanayak et al. (2006).

Our key variable is the number of malaria cases in a country in the 1996-2000 period. Various variables (malaria cases, malaria ecology index, and GDP index) are converted into natural logarithms to reduce scale differences, improve linearity and pull in outliers. Median regression methods are used. Results of the three models – naïve, linear controls, and IV are presented in columns 2, 3 and 4 of Table 2 (Panel 1). We report the coefficient on the deforestation variable, the probability value (p.value) associated with this coefficient and the overall significance of the model. The regression coefficient reflects the size and sign of the correlation with malaria incidence. The p.value reflects the statistical significance of the correlation (i.e., less than 0.1 is suggestive of a relationship).

The naïve model is statistically significant and explains about 41% of the variation. We also find confirmation of our key hypothesis: annual rate of forest cover increase (during the 1990 to 2000 period) is negatively correlated with malaria incidence in the 1996-2000 period: more deforestation is positively correlated with higher levels of malaria.

The linear-controls model (where we account for potential confounding due to GDP, school enrollment, voice and accountability, and stability of the governmental institutions) is also statistically significant and explains about 52% of the variation in the malaria cases. We also find that deforestation is positively correlated with malaria, except now the size of this correlation is twice as big.

Finally, the IV model is also significant and explains about 54% of the variation. In this model, first behavioral variables are used to predict deforestation, and then the predicted deforestation is used to explain malaria. Again we see that the deforestation variable is positive correlated with malaria, but now the size of this coefficient is almost 4 times as big as the naïve model – providing a statistically significant evidence of a much stronger correlation between the disease and exposure change due to deforestation.

4b. Meso (regional) analysis using the case of 480 Brazilian micro-regions

In this case study, we examine the hypothesis regarding the regional level correlation of malaria and forest cover. We use a cross-sectional data set of approximately 490 Brazilian micro-regions, which in the Brazilian context is anything between one to twelve counties. The malaria data comes from DATASUS (website). It is reported in terms of 1000 inhabitants, and represents hospital morbidity over the 1992-2000 period. Climate is represented by long run temperature and rainfall (averaged over several years) in the 490 micro-regions, based on weather stations that are located approximately one per micro-region. Census data (website address) on housing, population, education levels, income, medical care (proxied by number of doctors and hospital beds) and infrastructure (percent of the households connected to water, sanitation, and all-weather roads) is for 1991. Forest cover and vegetation data of the same vintage are from IPEA and protected area data is from INPE, both Brazilian data agencies. Pattanayak et al. (2006b) present additional detail on the compilation and use of this data in analysis.

Instead of dwelling on the details on the analysis, we focus on the key results using the structure from the previous case study. The naïve model (including some ecological controls for weather) is statistically significant and explains 46% of the variation. First, we see that micro-regions with higher forest cover have lower rates of malaria, all things considered. Second we find that micro-regions with higher deforestation (in the 1985 to 1995 time period) have greater rates of malaria.

The linear controls model (where we account for potential confounding due to demographics, income, infrastructure, and institutions such as protected areas) is statistically significant and explains 56% of the variation. First, we find that micro-regions with higher deforestation have greater rates of malaria – with the correlation that is significantly larger than the naïve model coefficients (almost twice as large). Second, micro-regions in the Amazon with conservation units have lower malaria rates for a given level of deforestation.

Finally, the IV model uses a variety of regional factors – presence of protected area, distance to highway and to state capital, population, size and location of the micro-region – as instruments for deforestation in the micro-regions. The overall model is significant. Now the size of the deforestation coefficient is almost 3 times as big as the linear-controls model. The results are consistent across the three models (i.e., deforestation is correlated with more malaria), but the sizes of the estimated coefficient are much larger (3-6 fold) in the models that include proxies for human behavior.

4c. Micro analysis using data on 340 children from Flores, Indonesia

Malaria is highly contextual, with incidence and transmission depending on local conditions, perturbations, and catastrophes. Thus, household or community-level multi-factor research is perhaps best suited to incorporate the diversity and heterogeneity of the ecological, epidemiological, and economic phenomena surrounding malaria. This case study examines the evidence on whether deforestation causes child malaria in the setting of Ruteng Park on Flores Islands in eastern Indonesia.

The data for this analysis are drawn from a household survey in the Manggarai district of Flores, Indonesia in 1996 around a protected area (Ruteng Park), established to conserve biodiversity. The survey and accompanying secondary data collection generated household data on wealth, housing quality, and number of adults, as well as individual data on age, gender, occupation, education and disease history during the twelve months prior to the survey. GIS is used to combine environmental statistics, including the amount and extent of primary and secondary forest cover at the village level, with the survey data and secondary data on public infrastructure such as sub-regional health care facilities. The sample includes approximately 340 kids under the age of 5. Given the binary nature of the data on malaria in kids under the age of 5, we estimate and report probit models of child malaria. Pattanayak et al. (2005) include details.

Starting with the naïve model, we find that the overall model is significant and, this being micro data, explains only about 6% of the variation. We find that the extent of protected (primary) forest cover is not statistically related to malaria, whereas the extent of disturbed (secondary) forest is positively correlated with malaria rates.

The linear-controls model accounts for potential confounding due to various individual, household and village characteristics. The overall model is significant, now explaining about 15% of the variability in the malaria data. As in the naïve model, the extent of disturbed forests is positively correlated with malaria (although now the coefficient is twice as big as before). Most interesting, we now confirm our key hypothesis that the extent of protected forests is indeed negatively correlated with malaria incidence.

Finally, the IV model uses a variety of community level factors – distance to highway, population, village size, elevation, and rainfall – as instruments for protected and disturbed forest cover around the villages. The overall model is highly significant. Most crucially, now the sizes of the coefficients are almost 3 times as big as the linear-controls model. Malaria in little children is highly positively correlated with the extent of disturbed forests and negatively correlated with the extent of protected forests.

5. Concluding thoughts

Vector-borne diseases such a malaria wreak havoc on the lives of many millions of people in poor, tropical countries, partly because these regions are exposed to environmental conditions such deforestation, livestock rearing, irrigated farming, road construction, and dam-building that encourage vector abundance and disease transmission. We argue that it is critical to focus on the deforestation linkage because it is the beginning of an entire chain of activities that affect malaria risks; can trigger behavioral changes due to accompanying increases or decreases in wealth; can lock communities into a vicious cycle of poverty, illness and environmental degradation; and is an integral part of the landscape and therefore of donor agencies and policy maker focus. Recognizing that deforestation often precedes many other relevant land use changes (particularly conversion to agriculture), taking deforestation as a starting point allows us to look at the impact of other elements in the “matrix of transformations.” As such it serves as a broad indicator of change in the ecology of infectious disease paradigm. This lead us to recommend a human ecology that focuses on the role of humans in land use change as well as in a variety of behaviors to prevent (e.g., sleep under nets, take prophylaxis) and treat (e.g., seek medical care, follow the drug regimen) malaria. We then review the implications of this framework change for empirical research and application – both in data collection and analysis and inference.

The empirical case studies draw attention to the role of socio-economic determinants of malaria and importance of including behavioral variables in empirical models of malaria incidence and prevalence. They illustrate how omitting behavioral factors from the analysis can lead to erroneous and biased interpretations regarding the nature of ecosystem changes and disease transmission – the size, sign, and statistical significance of regression coefficients can be wrong. In general, they are intended to highlight different elements of human-induced ecosystem change, disease outcomes, and economic causes and consequences.

What we have not discussed is the inherent dynamics of coupled natural and social systems. In a recent paper, for example, Pattanayak et al. (2006a) analyze global and micro data to show that malaria prevention behaviors depend on malaria prevalence. They find that households and countries engage in greater degree of prevention if they face high rates of malaria and fewer prevention behaviors if they confront low rates of malaria. That is, the causal arrow can also flow in the other direction (such an arrow is shown as a dotted arrow in Figure 1, typically missing from most assessments). This logical feedback and dynamic between prevention and prevalence suggests that it is insufficient and inappropriate to model and consider socio-economic behaviors as something outside the malaria infection and transmission process. Behavior and its determinants are part and parcel of the ecology and epidemiology and must be built into the analysis and planning.

In fact, it is safe to say that many of these findings hold for a general class of vector-borne infectious diseases such as dengue, leishmaniasis, hantavirus pulmonary syndrome, schistosomiasis, filariasis, lyme disease, onchocerciasis and loiasis. Space limitations preclude a comprehensive discussion of these diseases (for additional details, see Wilson [2001] and tables 4 and 5 in Colfer et al. [2006], for example). As suggested in Figure 1, ecosystem changes influence the emergence and proliferation of these diseases by altering the ecological balance and context within which disease hosts or vectors and parasites breed, develop and transmit diseases (Patz et al 2000). For example, deforestation is often followed by water resources development and livestock management, which open up numerous possibilities for disease risks.

Moreover, the simultaneity between prevalence and prevention discussed previously Pattanayak et al. (2006a) only points to the proverbial tip of the dynamic that is inherent in coupled natural and social systems. As Hammer (1993) suggests, in the case of malaria, very little is known about the inter-related dynamics of ecosystem changes, vector density and infectivity, development of immunity and resistance (to pesticides and drugs) and human response. Wiemer’s (1987) case of schistosomiasis in China and Gersovitz and Hammer’s (2005) model of malaria prevention and treatment are early attempts to examine these dynamics through mathematical simulations. Much more conceptual work is needed before ecosystem change dynamics can be incorporated into such models. Empirical research must test hypotheses about the nature and magnitude of these relationships and generate statistical parameters that can then be used for policy scenario analysis.

In the interim, however, the human ecology approach to public health can take root and thrive through the conduct of systematic economic and health impact assessments of forest policies. Such evaluations need to be inter-disciplinary longitudinal studies, with at least the following features:

1. It is impossible to design and implement a rigorous study and make credible inferences without a clear understanding of the policy scenario. Specificity of the policy scenario – be it a project at a site, a program that includes a collection of projects, or a nation/region-wide policy – allows the analyst to understand the mechanism of disease transmission and economic impacts in terms of ‘modifiable causes’.
2. With a clear scenario, it is then possible to design rigorous evaluations to infer ‘causal policy impacts’. These are typically through randomized assignment of the program or a quasi-experimental design that includes data collection in program and control (non-program) sites during various stages of program implementation, including baseline (pre-program) and endline (post-program) data.
3. The credibility of the resulting evaluation will ultimately ride on the quality of the data and the rigor and care in data analysis. For a study of this type, outcomes variables include indicators of health, wealth, and the environment. Extent of forest cover and forest condition are among the key explanatory variables. Other explanatory variables include socio-economic, demographic, environmental, health, and public health policy indicators. The challenge in empirical work is to identify robust measures of these variables and separate independent and dependent variables. The multiple channels for feedback between malaria, deforestation and poverty suggest that these variables would be dependent variables in some specifications, and independent variables in other specifications and data sets.
4. Although researchers can employ an array of sophisticated techniques to remedy defects in available data, clearly “prevention” in the form of careful data collection is superior to “cure” in the form of ad hoc statistical fixes. Longitudinal data sets – and particularly panel data sets – are key to addressing at least three critical issues in the types of research proposed here: heterogeneity, endogeneity, and dynamics or mobility (Ezzati et al., 2005). Ideally, data should be collected at several scales, ranging from individual level health and demographic data, to household level economic information, to community and regional level environmental statistics and policy factors.

The human ecology approach proposed in this chapter that is built on these conceptual and empirical roots can be used for at least two practical purposes (Pattanayak et al. 2006c). First, it can help organize the conceptual links between coupled natural and socio-economic systems and serve as a platform for generating testable hypothesis and policy parameters. Such efforts are critical for understanding the ecological, entomological, epidemiological and economic aspects of deforestation, malaria, and their behavioral underpinnings. Second, it will be vital for building decision analysis and scenario simulation tools (Kramer et al., 2006), which rely on estimated parameters, for formulating integrated strategies that cut across health, environment and economic sectors to address the broad idea of ecosystem change and disease control. Scenario simulation can for example inform the design of surveillance and monitoring framework necessary to detect changes in the environment, vector density, human migration and behavior, and incidence of diseases in order to both contain vector-borne diseases and prevent epidemics.

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Table 1. Ecosystem change and malaria

Deforestation/ Agricultural development	**Country/ Region**	**Density decrease**		**Density increase**		**Increased human contacts**		**References**
		Species	**Malaria**	**Species**	**Malaria**	**Species**	**Malaria**	
Deforestation	Thailand	*An. dirus*	-					Taylor 1997
	Nepal	*An. minimus*		*An. fluviatilis*				Sharma VP. 2002
	India			*An. fluviatilis*				Kalra 1991
	Sri Lanka	*An. barbirostris*		*An. annularis*	+			Amerasinghe and Ariyasena 1990, Konradsen et al. 2000
				An. jamesii	+			
				An. nigerrimus	+			
				An. subpictus	+			
				A peditaeniatus	?			
	Sahel, Africa	*An. funestus*						Mouchet et al. 1996
Land exploitation/ pollution	Mediterranean	*An. labranchiae*	-					Coluzzi 1992
		An. sacharovi	-					
		An. superpictus	-					
Cacao plantation	Trinidad			*An. bellator*				Downs and Pittendrigh 1946, Ault 1989
Cassava	Thailand	*An. dirus*	-	*An. minimus*	+			Bunnag et al. 1978, Sornmani 1987, Prothero 1999
	Thailand	*An. dirus*	-					Rosenberg et al. 1990
Sugarcane	Thailand	*An. dirus*		*An. minimus*	+			Sornmani 1972, Sornmani 1974, Bunnag et al.1978
Coffee plantation + irrigation dams	India	*An. fluviatilis*	-					Kalra 1991
+ tree crops	Thailand			*An. minimus*	+			Suvannadabba 1991

Deforestation/ Agricultural development	**Country/ Region**	**Density decrease**		**Density increase**		**Increased human contacts**		**References**
		Species	**Malaria**	**Species**	**Malaria**	**Species**	**Malaria**	
Tea plantation	Sri Lanka			*A. culicifacies*				Jones 1951
Rubber	Malaysia			*An. maculatus*	+			Cheong 1983, Singh and Tham 1990, Walsh et al. 1993
+ Fruits	Thailand			*An. dirus*	+			Rosenberg and Maheswary 1982, Prasittisuk et al. 1989, Rosenberg et al. 1990
	Thailand			*An. dirus*				Prasittisuk et al. 1989
+ Orchards	Thailand			*An. dirus*	+			Taylor 1997
Rice	China			*An. sinensis*				Baolin 1988, Service 1989, van der Hoek et al. 2001
	Malaysia	*An. umbrosus*	-	*A. campestris*	+			Ooi 1959, Sandosham 1970
	Indonesia			*An. aconitus*	+			Marwoto and Arbani 1991
	Southeast Asia	*An. dirus*						Kondrashin et al. 1991
	Nepal	*An. fluviatilis*		*A. culicifacies*	+			Walsh et al. 1993, Sharma et al. 1984, Subedi et al. 2000, Reuben 1989
	Sri Lanka	*An. annularis* *An. barbirostris* *An. culicifacies* *An. varuna*		*An. jamesii* *An. subpictus*				Amerasinghe et al. 1991, Konradsen et al. 2000
	Africa			*An. funestus* *An. gambiae*				Mouchet et al. 1996, Reiter 2001
Rice + maize	Thailand	*An. dirus*		*An. minimus*				Prasittisuk et al. 1989, Konradshin et al. 1991

Deforestation/ Agricultural development	Country/ Region	Density decrease		Density increase		Increased human contacts		References
		Species	**Malaria**	**Species**	**Malaria**	**Species**	**Malaria**	
Irrigation system	India			*An. culicifacies*	+			Ault 1989, Amerasinghe et al. 1991
	Afghanistan	*An. superpictus*		*An. pulcherrimus*	+			Service 1989, Amerasinghe et al. 1991
	Africa			*An. arabiensis* *An. gambiae*	+ +			Service 1989, Amerasinghe et al. 1991
	Sahara			*An. gambiae*	+			Coluzzi 1984, Coluzzi 1992
	Guyana	*An. darlingi*		*An. aquasalis*	+			Ault 1989, Amerasinghe et al. 1991
Hydropower dam	Sri Lanka			*An. culicifacies*	+			Wijesundera 1988, Konradsen et al. 2000
Clearing of mangroves/ swamps for fish pond or mining	Malaysia			*An. sundaicus*	+			Ooi 1959, Walsh et al. 1993
	Indonesia			*An. sundaicus*	+			Marwoto and Arbani 1991
	Indonesia			*An. sundaicus*				Ooi 1959, Sandosham 1970
Mining + settlement	Thailand					*An. dirus*		Kondrashin et al. 1991
	Amazon			*An. darlingi*	+			Marques 1987, Conn et al. 2002
Settlements + urbanization or highway construction	Amazon	*An. darlingi*						Tadei et al. 1998, Tadei and Thatcher 2000, Conn et al. 2002
	Indonesia					*A. balabacensis*	+	Marwoto and Arbani 1991
	Indonesia	*A. balabacensis* *A. leucosphyrus*						Marwoto and Arbani 1991
	India			*An. stephensi*	+			Kalra 1991

Source: Yasuoka, J., Levins, R. 2007. Impact of deforestation and agricultural development on anopheline ecology and malaria epidemiology. Am. J. Trop. Med. Hyg.

Table 2. Empirics of ‘human ecology’ modeling of malaria and deforestation links

4a. MACRO	**naïve**	**linear controls**	**IV**
annual forest increase	-0.049	-0.089	-0.168
p.value	(0.065)	(0.013)	(0.038)
ecology controls	yes	yes	yes
behavioral controls	no	yes	as IV
Pseudo RSq.	0.407	0.519	0.540

4b. MESO	**naïve**	**linear controls**	**IV**
deforestation	7.89e-07	2.25e-06	6.99e-06
p.value	(0.047)	(0.000)	(0.004)
ecology controls	yes	yes	yes
behavioral controls	no	yes	as IV
Pseudo RSq.	0.461	0.555	0.235

4c. MICRO	**naïve**	**linear controls**	**IV**
log (primary forests)	-0.062	-0.163	-0.382
p.value	(0.497)	(0.106)	(0.046)
log (secondary forests)	0.234	0.401	0.609
p.value	(0.006)	(0.000)	(0.008)
ecology controls	yes	yes	yes
behavioral controls	no	yes	as IV
Pseudo RSq.	0.055	0.153	0.153

Figure 1. Ecology of vector-borne diseases - impact of human activities