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AUTHOR(S): Subhrendu K. Pattanayak, Catherine G. Corey, Yewah F. Lau and Randall A. Kramer

AFFILIATION(S): RTI International, University of North Carolina (Chapel Hill), U.S. Forest Service, and Duke University

ADDRESS FOR CORRESPONDENCE, TELEPHONE, FAX, EMAIL: 3040 Cornwallis Road, PO Box 12194, Research Triangle Park, NC 27709-2194, USA; Phone: 919-541-7355; Fax: 919-541-6683; Email: subhrendu@rti.org

ABSTRACT

In remote areas of developing countries, people's lives are closely intertwined with the condition of the natural environment. While there is a growing body of research on the social and economic impacts of natural resource use, little is known about how forest degradation and land use change impacts rural economies and livelihoods through the spread of infectious diseases such as malaria. We draw on the household production framework to specify econometric models to evaluate the links between ecosystem change and the incidence of malaria in young children. Data from a survey of households residing near a protected area in Flores, Indonesia is used to estimate multivariate logit regression models that test the correlations between the forest protection and child malaria, controlling for individual, household and community characteristics. Our results indicate statistically significant correlations between village level forest protection and the incidence of child malaria. We find that the extent of primary forest is negatively associated with child malaria, while the extent of secondary forest cover is positively correlated with child malaria. Other significant factors related to malaria in children include gender, mother's age, household wealth and house quality, village area and elevation, and level of public health infrastructure. The statistically significant correlation between the forest cover variables and child malaria rates suggest that forest protection may offer health benefits to nearby communities.

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Conservation and Health: A microeconomic study of biodiversity conservation and child malaria in Flores, Indonesia

The condition of the natural environment has important consequences for rural populations in developing countries. While there is growing body of research on the social and economic impacts of natural resource use, little is known about how environmental transformations impact public health, particularly in rural areas that lack hospitals, doctors and other public services. Perhaps the most significant environmental transformation of rural landscapes corresponds to the widespread and rapid degradation of forests in many parts of the developing world. Globally, the rate of deforestation averaged 16 million hectares from 1990 to 2000, a three percent increase in the rate of the previous decade (FAO, 2003). Co-occurring with this profound transformation of the landscape has been the reemergence of a number of infectious diseases such as malaria and pockets of acute and persistence poverty. While simple theories of causality cannot explain this co-occurrence, the literature makes it abundantly clear that this juxtaposition of deprivation, deforestation, and disease is not pure coincidence (Pattanayak, Corey, Kramer, Sills, & Murray, 2003; Guerin, Olliaro, Nosten, Druilhe, Laxminarayan, & Binka et al., 2002; Mayer, 2000; Wolman, 1995). Given malaria's expanded distribution, heightened local incidence, and increased severity, duration and resistance to treatment, it represents a critical threat to public health throughout the developing world, particularly among young children. The design and implementation of policies to improve human lives and protect forests depend critically on improving our understanding of interrelationships between forest quality, malaria incidence and human behaviors. Much of the previous economics analysis on malaria has taken a macroeconomic perspective (Sachs & Malaney, 2002; Gallup & Sachs, 2001). While global analyses indicate the seriousness of the problems, they cannot identify local determinants, causal mechanisms and consequently policy levers for highly contextual diseases such as malaria.

To better understand the link between environmental quality and human health, we draw on a household production framework to model the incidence of malaria in and around degraded forests (be they disturbed, fragmented or completely deforested). We propose that forest condition can be viewed as a direct input into production of children's health through its role in the ecology of diseases in forested rural areas. Forest condition may also have indirect impacts through behavioral responses to changes in the natural resource base. We use this conceptual framework to specify econometric models that can test for correlations between forest condition and child

malaria rates, conditional on socio-economic, public infrastructure, and individual demographic factors. The data for this analysis are drawn from a household survey in the Manggarai district of Indonesia in 1996 around a protected area, 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.

Multivariate regression models offer evidence of correlations between the quality and quantity of forest cover and child malaria rates. Primary forest cover is negatively associated with malaria incidence, while secondary forest cover is positively correlated with malaria incidence. We also find malaria incidence to be correlated with gender, mother's age, household wealth, housing quality, village area and elevation, and public health infrastructure. The significance of forest cover on malaria rates suggests public health is one of the unintended benefits of forest conservation. Thus, we contend that that microeconomic analyses of the type described in this paper can enrich our understanding of environmental determinants of disease and better identify pathways through which environmental conservation can complement public health policies.

1. MALARIA IN AND AROUND DEGRADED FORESTS

The ecological basis for disease dates back at least as far as the writing of *On Airs, Waters, and Place* by Hippocrates in 400 B.C. Today it is clear that without an "ecological perspective" on the life cycles of parasitic microorganisms and the associated infectious diseases our understanding and therefore control of diseases would be inadequate (Wilson, Levins, & Spielman, 1994; Wilson, 1995). In general, vector-borne anthroponoses such as malaria are more strongly affected by environmental factors influencing the abundance and survival of the vector (*i.e.*, specific mosquito species). Smith et al. (1999) attribute 70-90% of the risk of malaria to environmental factors. The variety and magnitude of environmental influences on this vector-borne disease is enormous; climatic factors such as precipitation and temperature affect the abundance of mosquito vectors and the development of parasites within the vectors, while anthropogenic influences operating through deforestation, agriculture, and housing construction

may influence vectorial capacity (Wilson, 2001). The impact of deforestation on temperature, precipitation, and vegetation reveals the interacting and correlated nature of these environmental influences.

A recent synthesis of the literature on forests and malaria proposes at least six pathways through which forest degradation (including disturbance, fragmentation and deforestation) can affect malaria infection and disease (Pattanayak et al., 2003).

- 1. Deforestation changes the ecology of a disease vector and its options for hosts.** Whereas the forest floor in primary growth tends to be heavily shaded and littered with a thick layer of organic matter that absorbs water and renders it quite acidic, cleared lands are generally more sunlit and prone to the formation of puddles with more neutral pH which can favor specific anopheline larvae development (Patz, Graczyk, Geller, & Vittor, 2000). Terrain affects the manner in which water collects in deforested areas – on steep inclines, streams are more common than large pools.
- 2. Deforestation can change local climate and thereby affect the spread of disease by reducing moisture held by the vegetation, and raising ground temperatures.** Higher temperatures can increase the rate at which mosquitoes develop into adults, the frequency of their blood feeding, the rate at which parasites are acquired, and the incubation of the parasite within mosquitoes (Walsh, Molyneux, & Birley, 1993). For example, deforestation and its related activities have produced new habitats for *Anopheles darlingi* mosquitoes and have caused malaria epidemics in South America (Walsh et al., 1993). The different species complexes in SE Asia (*A. dirus*, *A. minimus*, *A. balabacensis*) have been affected in different ways by forest clearance with different impacts on malaria incidence.
- 3. Forest degradation is often the beginning of a variety of land use changes.** These changes may include agriculture and livestock, plantations, human settlement, increased use of regenerating forests, road construction, and water control systems (dams, canals, irrigation systems, reservoirs). These habitat changes may not only result in mosquito populations that have higher rates of malaria transmission, but they may also lead to increased human contact and transmission (Petney, 2001). Land clearing can cause declines in populations of arboreal insectivores, leading vector populations to grow (Didham, 2001). Networks of irrigation ditches, canals and impoundments, as well as puddles from road construction, can improve vector habitats. Livestock can change vectorial capacity. Rubber plantations in Malaysia encourage *A. maculates*,

whereas *erythrina* (with their bromeliads) encouraged *A. bellator* in Trinidad (Walsh et al., 1993). Use of insecticide in follow-up agriculture can increase vector resistance (Wilson, 2001).

4. Deforestation is accompanied by migration that may enhance the spread of malaria. As shown in the case of gold miners in the Brazilian Amazon, migrants typically have little previous exposure and therefore lower natural immunity (Castilla & Sawyer, 1993). Moreover, migrants introduce the additional complication associated with administering health services to transient populations—inadequate medical follow up and possible side effects. Although incomplete treatment can relieve fever, the underlying malarial infection persists as the migrant moves and potentially transmits the disease to other locations, presumably on the deforestation frontier.

5. Forest degradation and ecosystem change have several putative climate impacts via the role of trees in the carbon cycle and regional weather patterns. Where the scale of deforestation is large, e.g. the Amazon basin, the effects on temperature and moisture and therefore on vector habitats can be quite significant. (Taylor, 1997). By changing temperature, humidity, availability of standing water, and vegetation, open forests can enhance malarial transmission (Bouma & van der Kaay, 1994; Martens, Rotman, & Niessen, 1995).

6. Ecosystem change can play a role in antibiotic resistance that has become a major concern for several plasmodium species (Wilson, 2001). Resistance evolves through fundamental principles of natural selection and evolution, including diverse factors such as extent of treatment, nature and site of antibiotic action, or genomic complexity of the parasite. Greater virulence results from genetic changes that occur by chance mutation, drift, or selection. While ecological change permeates the process, it is difficult to delineate the roles of specific forms of change such as deforestation.

Despite the emerging body of knowledge about the economic and ecological causes and consequences of malaria, our understanding of these complex issues remains incomplete and inadequate (McMichael, Patz, & Krovats, 1998). Consider some knowledge gaps that motivate this research (see Pattanayak et al. [2003] for further details). First, malaria is highly contextual, with incidence and transmission depending on local conditions, perturbations, and catastrophes. Thus, individual-level multi-factor research is perhaps best suited to incorporate the diversity and heterogeneity of the ecological, epidemiological, and economic phenomena surrounding malaria. Nevertheless, modeling of heterogeneity and diversity seems to be the exception rather than the rule.

Second, malaria research has lacked of a human ecology perspective, best exemplified by the insufficient and partial modeling of behaviors that include a wide variety of observable and unobservable activities related to exposure, prevention and treatment. The varied mix of factors reported in the empirical literature has found multiple socio-economic and community factors, including but not limited to wealth, knowledge and awareness of the malaria problem, age, gender, and education to be associated with malaria transmission (Vosti, 1990; Castilla & Sawyer, 1993; Perz, 1997; Lansang, Belizariob, Bustosa, Saulc, & Aguirrea, 1997; Castro & Singer, 2001). For example, Vosti (1990) showed that controlling for vector exposure, individual characteristics were only weakly related to malaria prevalence rates in miner communities of the Brazilian Amazon. On the other hand, Castilla and Sawyer (1993) found that economic status and knowledge about disease are significant factors in determining prevalence risk in endemic resettlement areas of the Amazon. In a similar study of a low endemic community in Philippines, Lansang et al. (1997) find that location, nocturnal visits to forests, migration, youth, and gender affect the likelihood of malaria. While these studies generate interesting hypotheses about the behavioral basis of malaria infections in rural areas of the developing world, we need many more household level multivariate analyses that consider human responses and reactions to changing ecological and economic conditions, before we can present an unequivocal picture of the economic and ecological determinants of malaria.

Third and perhaps most significantly, ecosystem change, including deforestation processes, is contributing to the resurgence of infectious diseases such as malaria, although the magnitude of these effects is still relatively unknown. While the role of forest degradation in the overall burden of malaria disease may indeed be small, if left ignored its contribution to the resurgence of this and many other diseases caused by vector-borne anthroponoses, may grow.

Thus, our goals in this paper are to expand this thin but critical literature on the influences of forest degradation on malaria by taking an empirical multi-factor approach and a human ecology perspective. We evaluate the relationship between forest condition and child malaria in a quasi-experimental setting of buffer zone villages around a protected area, established to conserve biodiversity, on Flores Island in eastern Indonesia. As described in the next two sections, we add to this small body of work by focusing on children, formulating our problem within the theory of household production, and using a rich mix of micro data on demographic, economic,

infrastructural and forest condition (including improved measures of forest quality, than what has been typically used).

2. A MICROECONOMIC MODEL OF FOREST CONDITION AND MALARIA

We can draw on the utility maximization and household production literature to understand how various factors relate to children's health (Popkin, 1982; Berman, Kendall, & Bhattacharyya, 1994). Health may be considered as a commodity that directly contributes to utility (i.e. sickness causes disutility). Good health can also contribute to utility by (a) increasing productivity, (b) making time available for market and non-market activities, and (c) decreasing costs of prevention and treatment of illnesses. Health outcomes, however, are not necessarily exogenously determined because households can combine time, money, and knowledge to improve and maintain their health. Thus, health can be conceptualized, like other goods, as the output of a household 'health production' function (Grossman 1972; Akin, Griffin, Guildy, & Popkin, 1985). A household can thus be viewed as maximizing utility comprising of desirable goods, including 'self-produced' health, given an income constraint and the health production constraint.

We focus on malaria in children for several reasons. First and foremost, children are most vulnerable among all sub-populations to health hazards, for example, bearing the burden of between 75 and 90 percent of the mortality and morbidity attributed to malaria (WHO, 2003). Second, relative to an adult, a child's health depends primarily on parental decisions and external factors, rather than the child's personal choices and behaviors. This translates into a relatively simpler modeling task because the estimation model does not include endogenous variables that can result in biased estimates. Third, many analyses of malaria cases cannot adequately model factors related to acquired immunity to the disease because of inadequate data. By focusing on children, we mitigate this potential source of bias, because they are unlikely to have developed immunity to malaria in their short lifespan.

Given this focus, we develop a stylized model of children's health. We assume that parents derive utility from their children and produce child health, for example, by investing time (Lch) and money on nutrition, medical care, and disease prevention. This bundle of purchased inputs for child health production is labeled as 'care giving' (CG) for the remainder of the paper. While we focus on children's health, the household production logic still holds, such that parent's

health can be included with all other commodities that contribute to household utility. The stylized model below illustrates the key insights of this approach.

A typical household maximizes utility comprising of child health (CH) and a composite consumption good (Z), for example, money. This utility is conditional on preference parameters, θ , that measure the shape of the utility curve and could be proxied by socio-economic data on parental age and education. Utility is maximized subject to two constraints. First, the household faces a child health production function that is twice-differentiable, continuous, and convex; CH is a vector of child health that depends on care giving, CG, and is conditioned by demographic factors, D, and environmental factors, F. As discussed in Section 1, parental ability to prevent diseases such as malaria by using bed nets and prophylactics is made easier or more difficult by the extent to which a child is exposed to malaria risks (e.g., mosquitoes) through deforestation and other forest disturbance, for example. Moreover, it is their perceptions of these environmental influences that dictate their actual choices. The shape of the production function, including cross-effects of F and D on CG*, are discussed below. Second, the household faces a budget constraint where expenditures on Z and CH are no greater than the sum of exogenous (I_e) and earned income. Without loss of generality, we will assume that the earned income is a function of household labor and forest quality. In rural areas adjoining forests, the primary sources of earned income are agriculture, wage labor, and forestry – all of which require labor input. Forest condition (including both quality and quantity) impacts all activities through multiple direct and indirect pathways (Sills, Lele, Holmes & Pattanayak, 2003; Pattanayak, Sills, & Kramer, 2004).

The Lagrangian for this problem, in which λ and μ are the Lagrangian multipliers, is presented in equation 1. λ is the marginal utility of child health, and μ is the marginal utility of income. The first-order conditions of this utility maximization are presented in equations 2 to 4. Simultaneous solution of these first-order conditions determines optimal consumption of CH and Z based on the optimal allocation of care giving. Time and money are allocated so that marginal opportunity costs are equal to marginal utility of consumption generated by these efforts.

$$\begin{aligned} \ell_{CH,Z,CG} &= U(CH,Z;\theta) + \\ &\lambda[G(CH,CG;D,F)] + \mu[I_c + I - p \cdot CG - Z] \end{aligned} \quad [1]$$

$$\begin{bmatrix} \ell_{CH} \\ \ell_Z \\ \ell_{CG} \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \\ 0 \end{bmatrix} = \begin{bmatrix} U_{CH} + \lambda \cdot G_{CH} \\ U_Z - \mu \\ \mu \cdot p - \lambda \cdot G_{CG} \end{bmatrix} \Rightarrow \begin{bmatrix} CH^*(p, \theta, D, F, I_c) \\ Z^*(p, \theta, F, I_c) \\ CG^*(p; CH^*, D, F, I_c) \end{bmatrix} \quad \begin{bmatrix} 2 \\ 3 \\ 4 \end{bmatrix}$$

A child's health can thus be described as a function of the cost of care giving inputs, individual demographic characteristics, environmental factors, and exogenous income. Because care giving is a household production activity and not a commodity that is entirely purchased in the marketplace at an exogenously determined price, the cost of care giving will depend on parental and household economic factors and community infrastructure – all exogenous to the child.

We are interested in how changes in forest conditions impact child malaria in rural forested areas with limited access to public health and other infrastructure. Analytically speaking, we could conduct comparative statistics in the above model, for example, by taking total derivatives of equations 2– 4 with respect to CH and F and evaluating $\partial CH/\partial F$. Unfortunately, the multiplicity of pathways and behaviors make it impossible for us to sign $\partial CH/\partial F$ *apriori*. Consider at least four complications. First, as suggested in the previous section, the ‘vector ecology’ of malaria depends on specific condition of the forests at the site of the human-vector interaction. This makes it difficult to identify the direct impact of changes in forest conditions on malaria. Second, this direct impact is moderated by caregiver perceptions of the links between forest degradation and malaria, as parents modify the level and type of averting or mitigating behaviors in response to their perceived changes in environmental exposure to malaria. Third, forests play multiple roles in household livelihoods, and therefore changes in forest conditions and or land uses generate complex indirect impacts on households. We can have income effects if, for example, changes in forest conditions increase household incomes (*e.g.*, improvements in drought mitigation and soil erosion enhancing agricultural profitability), some share of which can be used to purchase more child care and malaria protection. Fourth, we might also observe substitution effects if, for example, changes in forest conditions impact the shadow price of labor allocated to forest collection (*e.g.*, fuelwood), which causes parents to reallocate the amount of labor input into care giving. In sum, it is impossible to use a purely analytical approach to determine the net impact of these interacting ecological and economic effects. Such a conclusion strengthens the case for empirical analysis to determine how forest conditions impact child malaria.

Our model does suggest, however, that the link between malaria and forest conditions is likely to be moderated by several factors suggested by ecological and economic theory as well as prior empirical work. We contend that child malaria will be a function of factors exogenous to the child such as forest quality, child demographics, effective price of care giving, and household wealth (equation 2). Our empirical estimation must contend, however, with the reality that these factors are difficult to measure and not directly observable, and therefore operate in a second best world in which the influence of these factors are approximated by several individual, parental, household and community level variables. In Section 3, we describe a data set from the buffer zone of Ruteng Park on Flores Island in Indonesia that allows us to develop empirical surrogates for child demographics, forest quality, care giving and household wealth and test our hypotheses concerning the link between child malaria and forest quality.

We have presented a reduced form characterization of child malaria in equation 2 (instead of a structural representation) because of three reasons. First, the choice of functional form for a structural representation of all ecological and economic functions would be arbitrary. Second, the resulting analytical expressions would be sufficiently complex that the signs of most partial derivatives would be indeterminate without information on the specific functional form and the magnitudes of all of the parameters. Finally, we do not have the data to estimate the complete system (see Wolfe and Behrman [1982] for a similar reasoning).

3. RUTENG PARK IN FLORES, INDONESIA – A CASE STUDY

The setting for our empirical analysis is the buffer zone of Ruteng Park on Flores Island in Indonesia where children have experienced different rates of malaria. These villages are located in watersheds that continue to face varying degrees of forest degradation because of unequal forest protection. This quasi-experimental setting thus allows us to investigate whether there is any systematic spatial correlation between child malaria and forest condition, controlling for key individual, household and community factors.

Ruteng Park is an Integrated Conservation and Development Project (ICDP) established in 1993 on approximately 32,000 hectares of rugged terrain encompassing seven volcanic ridges that are between 900 and 2400 meters. The park protects the best submontane and montane forests of the fragmented forests of Flores, forms a critical watershed for the population of Ruteng town and its surrounding farms, and conserves biodiversity (cave bats and Komodo rats are examples of two

known endemic species). The management plan for the park permits limited economic extraction within the park (e.g., collection of fuel, rattan, and other forest products) and emphasizes the development of nature based tourism, new agroforestry enterprises and human capital (education and health) in the 56,000 hectare buffer zone surrounding the park (Indonesian Ministry of Forestry, 1995). The Indonesian government is directly impacting forest conditions through its management of the park and protection of forests. If this ICDP based forest conservation is shown to improve child health, we have some evidence that conservation and development are complementary, rather than conflicting, objectives.

The data for this paper primarily come from three sources. Individual and household data are from a survey conducted in Ruteng in 1996 as part of a larger economic analysis project on protected areas (Kramer, Pattanayak, Sills, & Simanjuntak, 1997; Pattanayak et al., 2004). Five hundred households were selected from 13,700 farming households living in 48 village clusters (*desas*) in the buffer zone of Ruteng Park using stratified random sampling and weighted based on population density of the *desas*. The household surveys gathered detailed information on socio-economic characteristics including annual income, assets (e.g., consumer durables), and housing conditions. Additionally, the surveys collected information on age, gender, occupation, education levels and diseases for each member of the household.¹

Data on public infrastructure such as hospitals, health care clinics, and schools were collected from secondary and administrative sources. District-level (*kecamatan*) health infrastructure information included data on the number of health care workers such as doctors, nurses, midwives, and paramedics in the district, as well as the number of medical facilities such as hospitals, health centers, clinics and pharmacies. These estimates were scaled by the proportion of the district population living in a particular village to develop village level approximations of health infrastructure.

Finally, the household and administrative data was integrated with ecological data on forest cover within a geographic information system (GIS). Priyanto (1996) provides estimates of secondary (or regenerating) and primary (undisturbed) forest cover by micro-watershed of rivers and streams that emerge from Ruteng Park. The core forests of Ruteng Park have enjoyed some form of official protection for at least 80 years (Indonesian Ministry of Forestry, 1995). Secondary forests are largely the result of logging by family or commercial firms from outside our study area (Blomkvist, 1995). By overlaying environmental (e.g., watershed boundaries) and

administrative (*e.g.*, desa boundaries) data in a GIS, we obtain estimates of forest cover by desa (see Figures 2 and 3).²

Data on malaria was based on survey questions on the individual diseases in the 12 months prior to the survey. This generated a binary variable measuring whether or not an individual had malaria. Approximately one-third of the children in our sample (*i.e.*, individuals under age 16) suffered from at least one bout of malaria in the year prior to the survey. Figure 1 shows how the average incidence of malaria in children varies across the sample villages. Darker shading of the villages indicates higher disease rates in children, with a range of 0 to 88%.

In section 2, we show that malaria rates are likely to depend on forest condition, child demographics, household wealth, and costs of care giving. Here we describe the empirical surrogates for each of these factors. Starting with our key ecological indicator, we measure forest quality for each household in terms of the amount of primary (undisturbed) and secondary (regenerating) forests associated with the buffer zone *desa* that they live in.³ This estimate is developed in a GIS using the procedures described above and we find that, on average, a typical *desa* has approximately 280 hectares of primary forests and approximately 116 hectares of secondary forests.

Child specific demographic factors – age, gender and location of birth – constitute our second set of factors. Age, for example, may be correlated with exposure because as the child gets older, he or she participates in a greater number of chores, such as fuelwood and water collection, that involve going into forests. Alternatively, as a child grows, they may develop resistance to malaria through previous malaria incidences. Many cultures are preferential to boys so it is possible that girls may suffer from more diseases than boys (Akin et al., 1985). Different behaviors between boys and girls could also lead to differences in disease rates (Kondrashin & Orlov, 1989). Children born in malaria endemic areas may be less susceptible to the disease. Thus, we consider whether the children born locally are more or less likely to suffer from malaria.

Household assets and wealth proxies are our third set of factors. These reflect the influence of both ‘supply of care giving’ through better healthcare and nutrition as well as ‘demand for child health’ because wealthier families are likely to invest more in their children. The survey provides several measures of wealth - annual household expenditure, annual household income, ownership of land and livestock, consumer durables, and housing quality. Because all indicators are highly correlated, we focus on consumer durables to reduce multicollinearity

problems in our regression models (see Pattanayak et al. [2004] for further discussion of a similar approach). Our wealth index is a simple count of household possessions: radio, a television, electricity, wall clock, wrist watches, kerosene stove, and motorcycle.

The final set of factors relate to the ‘effective cost’ of care giving. We do not have exogenously determined ‘price’ of care giving because it is not a commodity or service that can be readily purchased in the market place. Instead, as shown in Section 2, it can be conceived as the outcome of a set of household production activities whose ‘effective costs’ are likely to be affected by a variety of parental, household, and community characteristics that are exogenous to the individual – particularly the child. Consider four variables that could be used to measure the role of care giving.

First, we might expect a child’s healthcare to depend on the human capital of the primary caregiver – typically, the mother. The literature suggests that the mother’s education is positively correlated with good health in children, presumably because education is a proxy for knowledge about how to best prevent and treat illness (Jalan & Ravallion, 2001; Glewwe, 1999; Perz, 1997). Similarly, the mother’s age, health condition, and occupation might also impact the effective cost of care giving because it affects how much time and effort they can spend on childcare.⁴ While there is practically no variation in the mother’s occupation in our data set (all are farmers), we include her illness history and age in our analysis.

Second, the extent of public healthcare infrastructure could also impact the effective cost and therefore extent of care giving at the household level. We describe above how we use secondary and administrative data to construct a village level proxy for the extent of healthcare services, although they do not reflect the actual number of health workers or facilities that existed in a particular village. Specifically, we use the number of health clinics as an indicator of public healthcare.

Third, housing quality can enhance disease transmission or improve care giving. We identified poor quality housing based on the condition of the walls and floor, finding that approximately 10% of our sample of houses fell into this category. Specifically, a house was deemed as poor if the walls were constructed from bamboo and the floor of the dwelling was erected on stilts.

Fourth, community level factors can influence epidemiological patterns and therefore the cost of disease prevention and care giving. Large populations and crowded living conditions can

encourage the spread of communicable diseases. So we include the size of the village to approximate these influences. We also include village elevation to represent climatic conditions such as weather. Descriptive statistics for this and all other determinants of child malaria discussed in this section are reported in Table 1.

4. EMPIRICAL RESULTS

The variables used in our analysis exhibit wide distributions, but indicate a rural lifestyle with high levels of poverty. The average household has approximately 6 members, including two children. The typical household owns less than one consumer durable. The female caregiver in the household is nearly 35 years old. Nearly 70 percent of caregivers received some formal education and of those that did attend school, the average level of educational attainment was approximately 5 years, the equivalent of primary school. Caregivers typically experienced one illness during the 12 months prior to the survey. The mean age of children in the survey is 8 years. Approximately 86 percent of the child sample was born locally. On average, there is less than one medical clinic per village, while village populations around the park consist of 2,500 inhabitants and cover approximately as many hectares.

Table 2 presents statistics on the average illness rate among children in the community. Since the health of young children may be particularly sensitive to environmental and other factors, we conduct separate analyses for three cohorts of children: less than 5 years, 5 to 10 years, and over 10 years to less than 16 years. We also report results for the full sample of children younger than 16 years. The occurrence of malaria generally increases with age. Approximately 27 percent in those less than 5 years were reported to have experienced malaria in the 12 months prior to the survey, while nearly 42 percent of children over 10 and under 16 years were reported to have experienced malaria over the same period. Males in the youngest cohort were reported to have experienced approximately 33 percent higher rates of malaria than females, though in the older cohorts females had slightly higher rates of malaria than males.

Recognizing that a child's malaria incidence, as recorded in the survey response, M_i , is a binary variable that takes values 0 or 1, we implement a discrete choice model. That is, we can formulate *a priori* that $\text{Prob}[M_i=1] = F(\beta'x)$, where F is any function of the index $\beta'x$ that satisfies the axioms of probability, and x is a vector of individual, household, community and environmental factors related to child malaria.⁵ This implies that

$$E(M_i | x) = F(\beta'x) = 0 * F(M_i = 0 | x) + 1 * F(M_{AFi} = 1 | x) \quad [5]$$

where $F(\beta'x)$ is the conditional mean function that can be treated as a nonlinear regression or as a binary choice model amenable to maximum likelihood estimation. Specifically, we estimate a logit regression model using maximum likelihood methods.

All the variables listed in Table 1 and discussed in the previous section are included in estimating the logit model. We take the natural log of the forest cover and village area variables to reduce scale differences (which can cause convergence problems in maximum likelihood estimation), improve linearity, and pull in outliers (Hamilton, 1992). All the t-statistics are corrected to account for the fact that many of the observations belong to sub-groups or ‘clusters’ with shared factors (*i.e.*, children within the same household) and may therefore violate the assumption of identical and independent regression errors across observations. These adjustments for error correlation and heteroskedasticity among and across these clusters of observations were implemented through clustered robust regression methods.⁶ This approach adjusts the coefficients’ standard errors and level of significance, but does not affect the size and sign of the estimated coefficients.

Table 3 presents regression results for three child cohorts and the full sample of all those under 16 years of age. Our key variables of interest are primary and secondary forest cover, proxies for vector ecology. The signs and statistical significance on the coefficients are different across all cohorts, offering some evidence of the confounding (or effect modification) role of behavior. We see that the amount of primary forests in a village is negatively associated with malaria incidence among children in the two youngest cohorts. In contrast, we see that the extent of secondary forest in a village is positively associated with occurrence of malaria only in the youngest cohort. The lack of statistical significance in the model of malaria in older children suggests possibly confounding effects of various behavioral patterns that are difficult to fully characterize – older children may have developed resistance, take precautionary actions, work in the fields and forests, and are otherwise exposing and protecting themselves in many more complicated ways.

Specific child demographic factors appear to have weak correlations with malaria rates. In the youngest cohort, males are more likely to experience malaria, relative to females. However, in the two older cohorts, gender is not associated with disease at a high level of statistical significance. Children in the younger cohorts are somewhat less likely to have malaria, relative to

children in the oldest cohort. Children born locally are more likely to have malaria (although this is a weak relationship). Without information on previous location of residence, we are unable to determine whether the migrant children came from an area of endemic malaria and thus had acquired immunity prior to moving to the region.

Household wealth generally has a strong negative correlation with malaria. We see that the caregiver's age (but not illness history during in the 12 months prior to the survey) is positively associated with malaria occurrence in the youngest and oldest cohorts, respectively. Older caregivers may be able to dedicate less time and effort to childcare. Caregiver's level of education was also included in the logit models but was found to be statistically insignificant in the full specification. Parsimonious specifications including only the caregiver's education showed a statistically weak negative relationship, possibly because formal education is not correlated with health literacy concerning malaria prevention and or treatment. Poor housing quality, serving as a proxy for both environmental factors such as housing materials and wealth, is positively correlated with malaria in the youngest cohort.

Our indicator of public healthcare infrastructure, clinics per person, is negatively correlated with the occurrence malaria among all cohorts. The negative correlation could be because more clinics per capita facilitate rapid diagnosis and treatment of malaria and thereby lower the effective cost of care giving. We also find that the size of the village is negatively associated with malaria rates. Finally, village elevation is negatively correlated with malaria - capturing the impact of climatic factors such as temperature and rainfall.

5. DISCUSSION: THE UNINTENDED IMPACTS OF FOREST CONSERVATION

The pace of economic development in recent decades has resulted in dramatic changes to the natural environment, including rapid rates of global deforestation, without necessarily enhancing the level or quality of public health infrastructure. Co-occurring with these transformations has been the resurgence of a number of infectious diseases, malaria among the most pernicious and most prevalent. While the co-occurrence of forest changes and malaria expansion cannot be explained by simple theories of causality, the literature suggests that the juxtaposition of deprivation, deforestation and disease is not pure coincidence. By enhancing our understanding of the interrelationships between forest degradation and malaria incidence, we can

begin identifying local determinants, causal mechanisms and consequently policy levers for highly contextual diseases such as malaria.

In this paper, we take a microeconomic approach and human ecology perspective, drawing on the household production framework, to better understand the links between ecosystem change and the incidence of malaria in young children. Children are both especially vulnerable to changes in environmental risks and key players in the future growth and prosperity of a society. Forest condition can be considered a direct input into the production of children's health through its role in the ecology of diseases in forested rural areas, where it profoundly impacts malaria transmission by the mosquito vector. Additionally, forest condition may have indirect impacts through behavioral responses of humans to changes in the natural resource base.

A survey of households living in the buffer zone of Ruteng Park, on Flores Island, Indonesia provides data on child malaria by age-cohorts, individual characteristics, and parental resources and constraints. This information is combined with administrative data on local public health infrastructure and ecological data on the extent and type of forests at the village level. Multivariate logit regressions are used to test hypothesis about the correlation of child malaria and forest quality, conditional on critical demographic, socio-economic, and public infrastructure factors.

These models confirm that the extent of primary (protected) forests in a village is negatively correlated with malaria in the younger children (aged 0 – 10). This is corroborated by the finding that the extent of secondary (regenerating) forests is positively associated with malaria in the youngest age cohort (0-5 years). We also find a number of individual and household factors to be correlated with malaria rates in children. The impact of demographic factors—gender and location of birth—on disease may be related to household responsibilities, preferential treatment based on gender, or other socio-demographic factors. We see that wealth, maternal characteristics, and housing quality are statistically associated with malaria among children, suggesting that those with greater resources may be better able to protect their children from malaria. At the community level, health infrastructure, village size and elevation are negatively correlated with malaria.

Although the size and statistical significance of each of these factors varies across cohorts, the sign is consistent across the three cohorts and the overall population. Generally, the coefficients on models for the youngest cohort (under 5 years of age) have the largest sizes,

indicating that the very young may be particularly vulnerable to ecosystem changes, parental inputs, and household and community factors.

As discussed in Section 1, the statistical results regarding the correlation of forest condition and child malaria are consistent with the multiple pathways through which deforestation can increase malaria infection and disease, including vector ecology, local climate, land use change and human behaviors. These ecosystem changes may not only result in mosquito populations that have higher rates of malaria transmission, but also lead to increased human contact and transmission. Collectively, these results suggest that Ruteng Park may be providing the unintended health benefit in surrounding communities.

Although this study is an important contribution to a thin empirical literature on forest malaria, there are some caveats in interpreting these findings. While our spatial mapping of forest quality and village location is orders of magnitude more precise than the macro analysis, it represents an approximate characterization of how forests, mosquitoes, households, and children interact. Spatial activity diaries, richer characterization of human interactions, and household level forest mapping would undoubtedly enhance the precision of this type of model building and our inferences regarding ecology and economics of child malaria. Furthermore, we are examining a single cross-section of the data, with limited ability to understand the dynamics of disease transmission and prevention. In general, it is difficult, if not impossible, to establish causality with a cross-sectional observational data set. While a spatially precise multi-period cohort data set would be ideal for answering the questions we pose, we believe that the statistical correlations reported in this paper present a useful starting point for beginning to think about the role of forest management in malaria control.

Despite these limitations, this study addresses some of the current knowledge gaps concerning the ecological and economic causes and consequences of malaria. First, we depart from the traditional macro-analyses of the consequences of malaria by considering a mix of socio-economic, behavioral, and ecological determinants. Second, by taking a micro-economic perspective, we consider household and individual factors related to exposure, prevention and treatment and thereby account for aspects of heterogeneity and diversity missing from previous research on this topic that have relied on a limited set of meso-scale variables. Finally, while vector ecology suggests that forests have an important impact on the incidence and transmission of malaria, this study is one of the few that has brought data to bear on the question regarding the

role of forest degradation on malaria. It is critical that additional multi-variable household level analyses be undertaken to improve our understanding of the interrelationships between forests, human health and development.

Although both health and forest policies ultimately aspire to improve the lives of people, it remains unclear whether these policies complement or conflict with each other due to the complex and dynamic relationship between deforestation, malaria and poverty. Our results indicate possibilities for designing complementary policies that simultaneously protect the environment and human health.

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Table 1. Characteristics of child population in study area

Variable	Mean	Std. Dev.	Min.	Median	Max.
Household & individual data ^a					
Family size	5.6	1.7	1.0	6.0	10.0
Number of children	2.2	1.4	0.0	2.0	6.0
Male (children)	0.53	0.50	0.0	1.0	1.0
Percentage households native born	0.86	0.35	0.0	1.0	1.0
Child age	7.7	4.5	0.0	8.0	15.0
Caregiver age	34.6	10.2	18.0	33.0	75.0
Caregiver number illnesses	1.0	0.7	0.0	1.0	4.0
Caregiver education (avg. years)	4.6	3.2	0.0	6.0	12.0
Household wealth index (number of consumer durables)	0.9	1.3	0.0	0.0	7.0
Poor housing quality	0.08	0.28	0.0	0.0	1.0
Village-level data					
Village-weighted share of clinics ^b	0.10	0.07	0.02	0.07	0.30
Village population	2,504	1,379	897	2,219	6,344
Village area (hectares)	2,881	1,903	439	2,322	9,174
Village elevation (meters)	926	264	500	825	1700
Environmental statistics ^c					
Primary forest cover (hectares)	280	263	9	223	1414
Secondary forest cover (hectares)	116	93	1	95	394

^a Individual and household data are from a survey conducted in Ruteng in 1996 as part of a larger economic analysis project on protected areas (Kramer et al., 1997; Pattanayak and Kramer, 2001; Pattanayak et al., 2004).

^b Data on public infrastructure such as hospitals, health care clinics, and schools were collected from secondary and administrative sources. District-level (*kecamatan*) health infrastructure information on the number health clinics in the district was scaled by the proportion of the district population living in a particular village to develop village level approximations of health infrastructure.

^c Environmental (*e.g.*, watershed boundaries) and administrative (*e.g.*, desa boundaries) data were integrated in a GIS to obtain estimates of forest cover by desa. For example, if two watersheds overlap with a particular desa, we use GIS to first calculate the fraction of the watersheds that lies in the desa, and then use this fraction multiplied by the total primary forest cover in a watershed to estimate a proportion of primary forest cover in the desa. Proportions are similarly computed for each overlapping watershed and summed to generate an aggregate measure of primary forest cover in the vicinity of households in the desa.

Table 2. Malaria rates of cohorts

	Average Total Population	Boys	Girls
Full sample - under 16 years n = 1,118	34.7% (47.6%)	35.1% (47.8%)	34.4% (47.6%)
0 to 5 years n = 342	27.3% (44.6%)	31.1% (46.4%)	22.6% (42.0%)
5 to 10 years n = 433	34.9% (47.7%)	34.6% (47.7%)	35.1% (47.8%)
10 years to 16 years n = 343	41.9% (49.4%)	40.4% (49.2%)	43.6% (49.7%)

Note: standard deviation in parentheses

Table 3. Logit regression model of child malaria

	Cohort: (> 5 years)		Cohort: (5 to 10)		Cohort: (10 to 16)		Cohort: (< 16 years)	
	Coeff	p-value	Coeff	p-value	Coeff	p-value	Coeff	p-value
Regression constant	4.075	0.078	2.438	0.230	-0.902	0.651	2.052	0.153
Primary (protected) forest	-0.284	0.097	-0.276	0.061	0.143	0.405	-0.107	0.336
Secondary (regenerating) forest	0.693	0.000	0.162	0.214	0.037	0.819	0.213	0.045
Male (0 = no; 1 = yes)	0.665	0.019	0.064	0.762	-0.086	0.709	0.130	0.345
Born in village (0 = no; 1 = yes)	0.393	0.574	0.447	0.506	0.197	0.695	0.215	0.607
Wealth index	-0.127	0.318	-0.232	0.036	-0.093	0.334	-0.159	0.038
Mother's health index	-0.065	0.334	-0.003	0.947	-0.086	0.099	-0.053	0.159
Mother's age	0.032	0.026	0.005	0.702	0.017	0.265	0.012	0.179
Health care facility index	-10.039	0.000	-4.667	0.015	-2.726	0.223	-5.280	0.001
Poor quality house (0 = no; 1 = yes)	1.476	0.009	0.122	0.762	-0.020	0.961	0.374	0.230
Village area	-0.713	0.001	-0.181	0.354	0.037	0.851	-0.214	0.134
Village elevation	-0.002	0.008	-0.001	0.056	-0.001	0.268	-0.001	0.013
0-5 cohort (0 = no; 1 = yes)	--	--	--	--	--	--	-0.666	0.001
5-10 cohort (0 = no; 1 = yes)	--	--	--	--	--	--	-0.311	0.048
pseudo R2	0.15		0.04		0.04		0.06	
N	337		431		338		1,106	

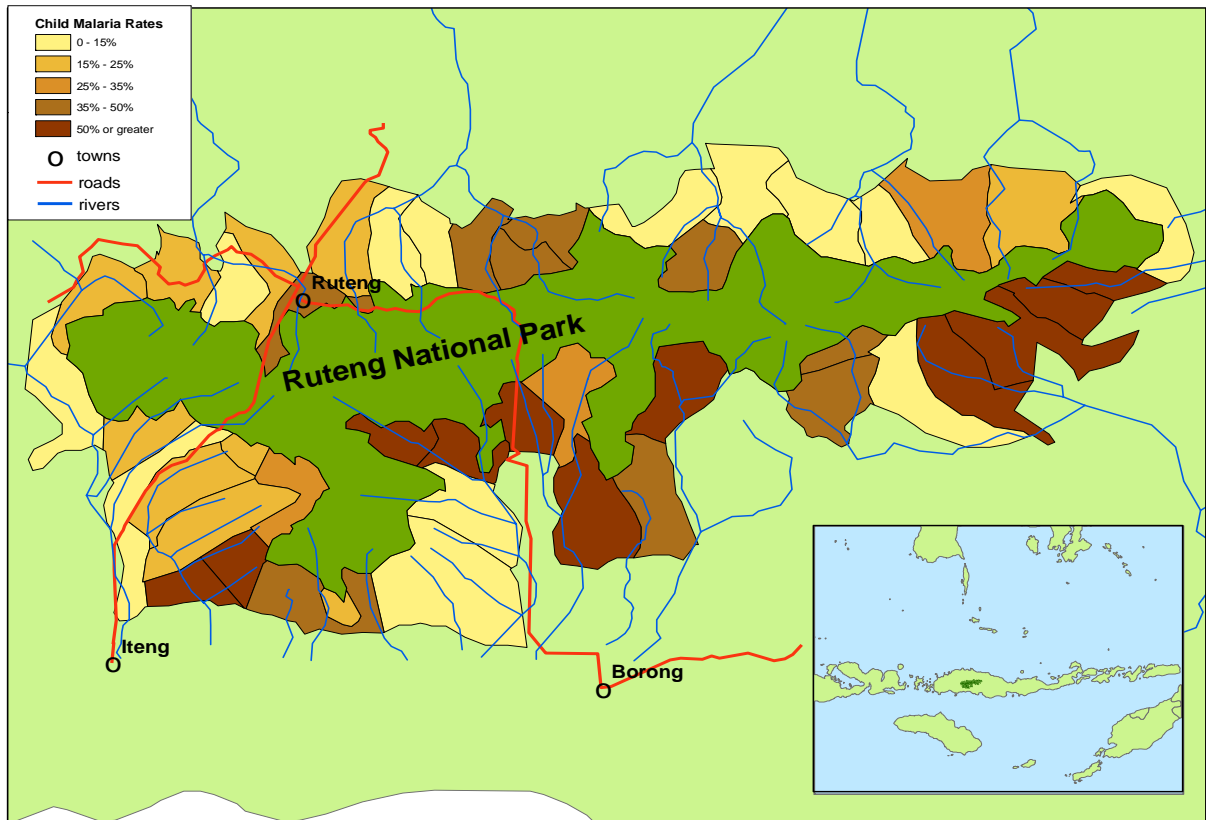


Figure 1. Average malaria rates in children by village.

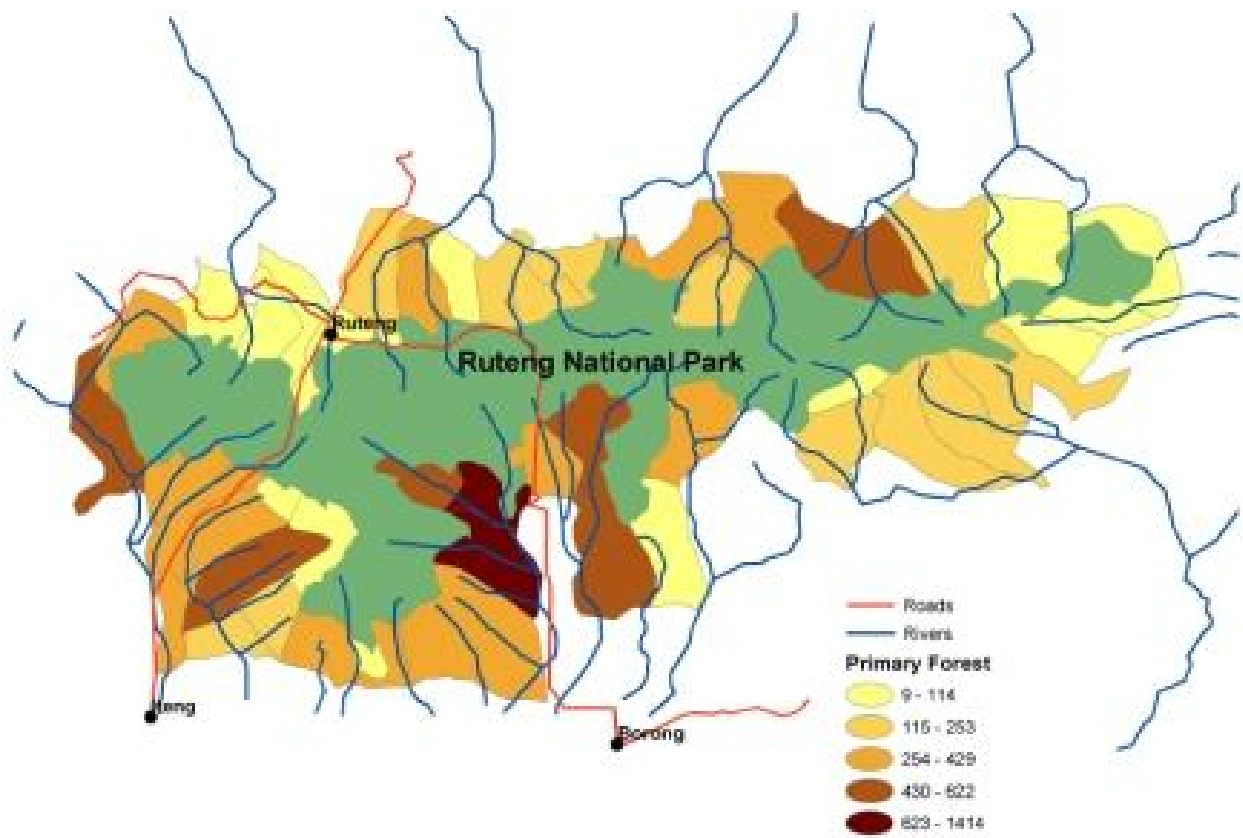


Figure 2. Levels of primary forest cover by village

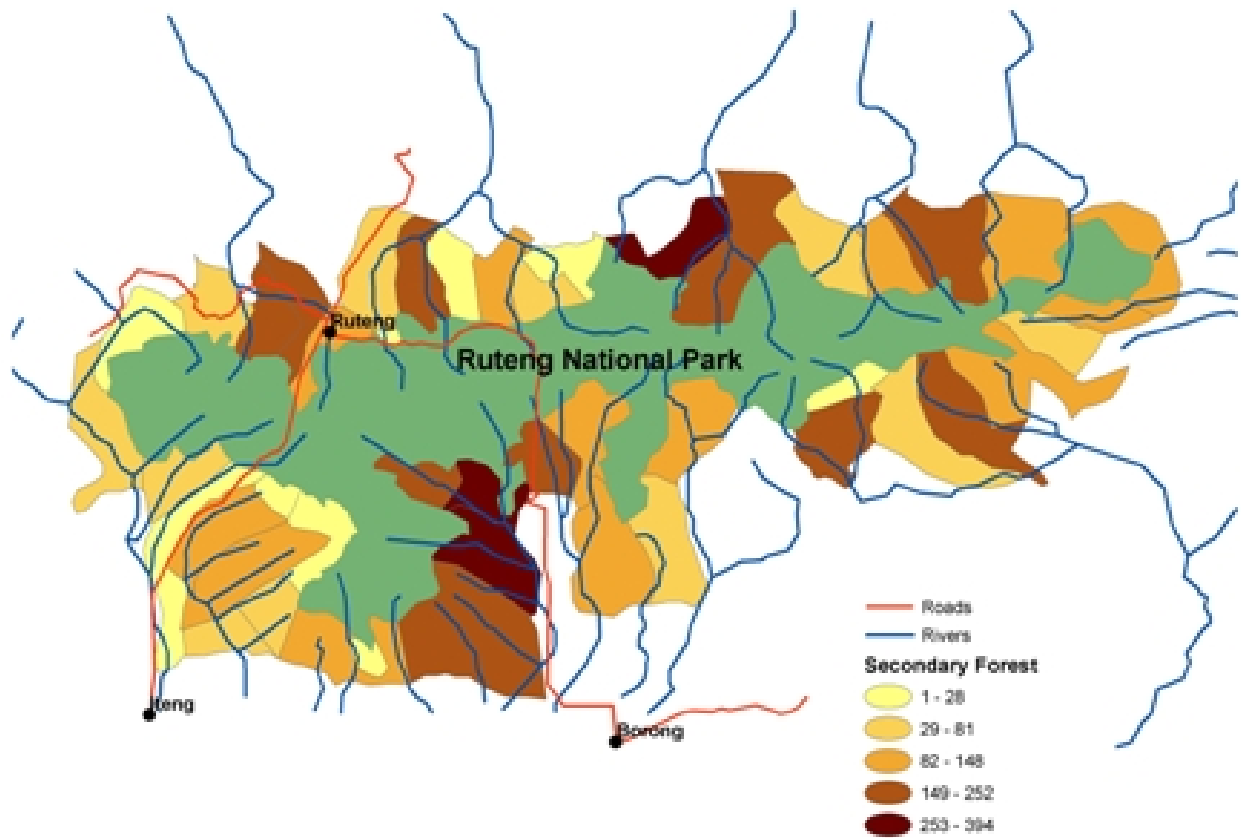


Figure 3. Levels of secondary forest cover by villa

ENDNOTES

¹ Child illness history was typically reported by female household caregiver. Previous studies have reported sensitivity of maternal diagnosis of malaria ranging from approximately 40 to 75 percent and specificity ranging from 50 to 60 percent (see Lubanga et al. 1997; Aly Thera et al. 2000; Bailo Diallo et al., 2001).

² For example, if two watersheds overlap with a particular desa, we use GIS to first calculate the fraction of the watersheds that lies in the desa, and then use this fraction multiplied by the total primary forest cover in a watershed to estimate a proportion of primary forest cover available to the desa. Proportions are similarly computed for each overlapping watershed and summed to generate an aggregate measure of primary forest cover available to desa households.

³ In the long run, intensive forest use could also lead to degradation of forest, which might then be categorized as secondary (Moeliono, 1995). We find that population density is inversely correlated with primary forest cover. For our household model, however, we treat quality of forest available to households as exogenous to their current year decisions about collection, because that quality is generated by longer-term activities in the entire desa and not by an individual household's decisions in a given year.

⁴ Perhaps more directly, mothers without malaria are less likely to transmit malaria to their children. Unfortunately, mother's malaria is likely to be correlated with several other variables included in the regression models, causing severe multi-collinearity problems and arguing for its exclusion as a potential regressor.

⁵ The three axioms of probability are: (a) $0 \leq F(\beta'x) \leq 1$; (b) $F(\beta'x) > 0$; and (c) F approaches 0 and 1 as $(\beta'x)$ approaches $-\infty$ and $+\infty$.

⁶ This approach uses the Huber/White/sandwich estimator of variance based on the assumption that observations are not independent but rather can be divided into M groups, G_1, G_2, \dots, G_M that are

independent. The formula for the robust estimator of variance is $\hat{V} = \hat{V} \left(\sum_{k \neq}^M u_k^{(G)'} u_k^{(G)} \right) \hat{V}$ where

$\hat{V} = (\partial^2 \ln L / \partial \beta^2)^{-1}$ (the conventional estimator of variance) and $u_k^{(G)}$ is the contribution of the k th group to the scores $\partial \ln L / \partial \beta$.