Public Health Effects of Ecosystem Degradation: Evidence from Deforestation in Indonesia *

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Abstract

Despite growing concern about the effect of declining environmental quality on human health, little effort has been made to quantify the effect of ecosystem degradation on the incidence and burden of infectious diseases. Using village-level administrative panel data and district-level satellite data on forest cover, I find that deforestation from 2001-2008 in Indonesia can explain over one million additional malaria infections. The evidence is consistent with an ecological response and the effect of deforestation on malaria cannot be explained by post-deforestation land use change, anti-malarial programs or migration. The effect is specific to malaria, with deforestation having no discernible effect on other diseases with disease ecologies different from that of malaria. Back of the envelope calculations suggest that the local health benefits from avoided deforestation are equal to or greater than the global carbon benefits, underscoring a large, yet previously ignored and unquantified cost of deforestation with major implications for the design of payments for ecosystem services.

JEL Codes: Q53, Q56, Q57, Q20, O13

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1 Introduction

In addition to the large costs of morbidity and mortality, infectious diseases result in lower economic growth (Bleakley, 2007; Well, 2007; Gallup and Sachs, 2001), lower educational outcomes (Barreca, 2010; Cutler et al., 2010; Lucas, 2010; Miguel and Kremer, 2004) and declining fertility (Lucas, 2013). The most widespread of these diseases, malaria, affects 300-500 million people each year, half of whose victims are children under the age of five (Lucas, 2010; Sachs and Malaney, 2002), disproportionately affecting poor and vulnerable populations (Ngonghala et al., 2014). Many papers have discussed interventions that can reduce the incidence of such diseases in poor countries (Ashraf, Berry and Shapiro, 2010; Cohen and Dupas, 2010; Hoffmann, 2009; Hoffmann, Barrett and Just, 2009). Yet, even as quantifying the links between environmental factors (eg. air pollution) and human health and well-being has become a central component of the modern research agenda in environmental economics (Graff Zivin and Neidell, 2013; Greenstone and Jack, 2013), little effort has been made to understand the effect of ecosystem degradation on the spread of infectious diseases. This void in research is important to fill since increasingly, interventions dependent on insecticides and anti-malarial drugs are likely to be limited in their effect as a result of increasing resistance of disease-carrying vectors and pathogens, and environmental and natural resource management will be essential in controlling the incidence of infectious diseases (Lindsay and Birley, 2004).

In this paper, I provide the first causal evidence linking deforestation to malaria. In theory, deforestation can affect malaria through several channels. First, post-deforestation land use change in the form of agricultural activity and urbanization has been associated

with malaria. Second, migrants can act as latent hosts of malaria since they typically have lower incomes and less access to medical facilities than native populations and deforestation often drives migration in poor countries. Third, deforestation can alter the disease ecology of malaria. In particular, deforestation results in accumulation of water puddles and reduction in biodiversity, both of which favor the growth of anophelene larvae (Pattanayak and Pfaff, 2009; Patz and Olson, 2006; Patz et al., 2000; Walsh, Molyneux and Birley, 1993). While the first two mechanisms are socio-economic in nature, the third is purely ecological. In this paper, I provide evidence that there is a pure ecological response in the effect of deforestation on the incidence of malaria.

Using district-level variation in deforestation and a rich village census data set from Indonesia, I find that deforestation has a statistically significant impact on the incidence of malaria [1]. In particular, there are three key findings. First, for the average within-sample loss in forest cover (1000 Ha) in a district, there is a 2% - 7.2% increase in the probability of a malaria outbreak in each village in that district, which translates to about 45,000 - 162,000 additional infected individuals in Indonesia each year. Second, the effect of deforestation on malaria is largely concentrated in villages within or near forests, emphasizing the local nature of the health externalities, in sharp contrast to the global nature of the carbon externalities. Back of the envelope calculations suggest that the health cost of deforestation is between \$58 - \$208 per hectare compared to the carbon cost of \$32 - \$160 per hectare[2]. Third, the

[1]Indonesia is an ideal setting for such a study. It contains the third largest stand of tropical forest in the world experiences the highest rate of deforestation in the world (Margono et al., 2014). Between 2001-2008, Indonesia saw 4.8 million hectares of forest cover loss. In fact, deforestation in Indonesia makes it the third largest producer of greenhouse gasses worldwide, behind the US and China (Burgess et al., 2012). At the same time, malaria poses a major public health risk. The WHO estimates that 44% of the population in Indonesia, over 130 million people, is at risk of malaria infection (Elyazar, Hay and Baird, 2011)

[2]See Nature (2009); Honorine (2010). The World Bank estimates that Indonesia's 1 million square kilometers of forest cover could earn Indonesia between \$400 million - \$2 billion through carbon credits. That

observed effect is much more pronounced in forests intended to preserve biodiversity, than in forests which are for the purpose of logging and land use change. Furthermore, the effect can not be explained by common reasons provided in the public health literature, such as post-deforestation land use change, anti-malarial programs or migration, suggesting that the underlying mechanism is an ecological as opposed to a socio-economic response.

Despite forests forming a critical part of the environment and ecosystems in poor communities, achieving causal identification of the effect of deforestation on infectious diseases has been difficult (Pattanayak et al., 2006)[3]. There are two major identification concerns. First, there could omitted variables bias associated with road access, occupational choice[4], poverty levels[5], geographic features, sanitation facilities, medical facilities, and agricultural activity. Second, deforestation could be endogenous to malarial outbreak through reverse causality or simultaneity if malarial outbreak affects workers' choice to clear forest through reduced availability of healthy labor.

A major contribution of this paper is to overcome these challenges and provide causal identification of the effect of deforestation on the incidence of malaria by building on important prior research in the public health literature (Bauch, Pattanayak and Sills, 2014; Laporta et al., 2013; Myers et al., 2013; De Silva and Marshall, 2012; Pattanayak and Pfaff, 2009; Yasuoka and Levins, 2007; Patz and Olson, 2006; Patz et al., 2000). I employ panel

translates to \$4-\$20 per hectare. I assume a 5% discount rate and estimate a 10-year present discounted value of carbon benefits.

[3]Reviews of the epidemiology and public health literature by Keesing et al. (2010) and Pongsiri et al. (2009) examine case studies on the links between biodiversity loss and local disease ecology. Both reviews illustrate and recognize that aside from lab experiments that produce limited results, field studies in this area have been largely correlational case studies.

[4]Individuals working in the forestry sector could have increased exposure to vectors in the event of deforestation.

[5]Deforestation could either result in exploitation of local population or provide local economic windfalls and could thus influence vulnerability to diseases through income and access to medical facilities.

methods by exploiting satellite data on forest cover and a rich village census data on disease outbreaks to control for time-invariant geographic, climatic and demographic factors that could be correlated with both deforestation and diseases. This allows me to identify the effect of deforestation on malaria off of the timing of deforestation (and not whether or not a village experienced deforestation) that is plausibly exogenous to local disease outcomes. Administratively most deforestation decisions are made at the district level or at the national level in consultation with district heads.[6]. All forested land in Indonesia is owned by the government, and as such, all legal and illegal deforestation in Indonesia requires the implicit or explicit consent of the district head (*bupati*). Since each district is comprised of over 250 villages on average, it is unlikely that decisions to grant licenses or to acquiesce to illegal deforestation by the district head were contingent on malarial outbreaks in specific villages. As such, district-level deforestation in the Indonesian context is plausibly exogenous to village-level disease incidence.

I test this assumption in a few ways. First, I perform placebo tests and show that the effect is specific to malaria. If there were unobservables correlated with health and deforestation, then deforestation would also have an impact on other diseases whose disease ecology differs from that of malaria. Second, I use exogenous variation generated from the introduction of a timber certification program aimed at curtailing illegal exports of raw logs. Third, I provide falsification tests for this national level policy and show the effect is specific to the year in which it was implemented. Fourth, I demonstrate that the results are robust to the inclusion of a variety of controls including health care access, poverty measures,

[6]Indonesia has many levels of governance. The country is divided into 32 provinces (*propinsi*) which are further divided into districts (*kabupaten*). Districts are subdivided into villages (*desa*). There is also a sub-district (*kecamantan*) level of administration that is responsible for implementing some welfare programs.

distance from cities, sanitation facilities, population density, government sponsored health interventions, geographic terrain, precipitation and primary sector of employment. The identifying assumption, therefore, is that there are no time-variant omitted variables that are correlated with malarial outbreaks and deforestation but not correlated with health (other diseases)[7].

This paper also contributes to the economics literature on the health and mortality effects of environmental degradation. This literature has made advances in the causal identification of the effect of air pollution on infant mortality (Currie and Walker, 2011; Jayachandran, 2009; Currie and Neidell, 2005; Neidell, 2004; Chay and Greenstone, 2003), adult mortality (Ziebarth, Schmitt and Karlsson, 2014), productivity (Graff Zivin and Neidell, 2012; Hanna and Oliva, 2011) and educational outcomes (Currie et al., 2009).[8] However, the focus has been almost exclusively on air pollution with much less attention paid to understanding the health effects of other forms of environmental degradation such as deforestation. This paper fills that important void; to the best of my knowledge the economics literature has not previously addressed the causal impact of deforestation on health or disease outcomes.

This research has strong policy implications for payments for ecosystem services (PES) (Jayachandran, 2013; Alix-Garcia et al., 2013; Golden et al., 2011; Alix-Garcia et al., 2009;

[7]There are possible scenarios where this assumption would be violated. For instance, village heads in poor favor with the district head could withhold both anti-malarial programs and allow conglomerates to deforest in those villages. However, such political economy stories would have to be specific to malaria and not health in general. Furthermore, such a concern would be invariant to the type of forest and the specific effect on conservation/protection forests suggests that this scenario is unlikely.

[8]Several authors have made use of quasi-experimental methods. For instance, Chay and Greenstone (2003) used the geographic variation in air particulate matter induced by the 1981-82 recession to show that reduced air pollution led to decreases in infant mortality to the tune of 2500 fewer deaths per year. Currie and Walker (2011) study the introduction of electronic toll collection to show that reduced pollution through decreased traffic congestion resulted in lower incidence of prematurity and low birth weight amongst mothers within two kilometers of a toll plaza in NJ and PA. Jayachandran (2009) exploits exogenous variation in air pollution from forest fires in Indonesia to demonstrate a large effect of smoke on infant mortality.

Jack, Kousky and Sims, 2008). First, health benefits exceed carbon benefits, particularly in light of recent evidence that the carbon footprint of deforestation may be as low as 25% of previous estimates (Harris et al., 2012). In fact, recent research suggests that sustained forest cover may have negligible or even negative carbon benefits (Unger, 2014); this research shows that sustained forests have large economic benefits independent of any carbon effects. Second, the health benefits are local in nature, in sharp contrast to the carbon benefits that are global. Since the local health benefits exceed the global carbon benefits, PES programs, such as Indonesia's recently launched Rainforest Standard (RFS), may not require large external funding to avoid deforestation and instead motivate local institutions to promote conservation (JakartaPost, 2014).

This paper is organized as follows. Section 2 provides context for deforestation and the implications for the disease ecology of malaria, and section 3 describes the data. Section 4 discusses the identification strategy. Estimation results are presented in section 5. Section 6 considers alternative explanations and mechanisms and section 7 concludes and draws out policy-relevant findings.

2 Background

While its tropical conditions are ideal for forest growth, Indonesia has been experiencing alarmingly high rates of deforestation. Burgess et al. (2012) provide a conservative estimate that between 2001 and 2008, Indonesia lost about 4.8 million hectares of forest cover, which is roughly the size of the US states of Vermont and New Hampshire combined. This coincides with a period of high rates of contracting infectious diseases. In this section, I provide

background on the nature of deforestation as well as its implications for the spread of malaria.

2.1 The Political Economy of Deforestation in Indonesia

During the Suharto era, the national government maintained total control of the forest estate and concessions were awarded to a small group of forestry conglomerates in the production and conversion zones for the purposes of logging and clearing of forested land for industrial and palm oil operations respectively. Since the conglomerates had connections to the Suharto government and because the permits lasted up to 30 years, they had strong incentives to conduct logging selectively and sustainably. Logging was prohibited in all other zones that were designated for watershed protection (protection forest) and biodiversity protection (conservation forest) (Barber, 1990). However, after the fall of Suharto regime, the national government passed the decentralization laws that amongst other things, made district forest departments part of the district government, answerable to the head of the district (Burgess et al., 2012).[9]

60%-80% of all deforestation in Indonesia is illegal (CIFOR, 2004). To a large extent, the onus of authorizing, monitoring and enforcing[10] limits on logging (legal and illegal) falls on the district office. District office employees are supposed to stationed at the gate of every concession to monitor all logs leaving the concession, and at the entrance of all saw mills to check the legality of logs entering the mill. Furthermore, all logs require transport

[9]The district governments also became incredibly powerful. They were granted considerable autonomy over their jurisdiction and 25% of revenues from natural resources in a district were returned to the district governments as block grants.

[10]Burgess et al. (2012) demonstrate that illegal deforestation can not be explained by lack of enforcement capacity since the impact of new districts on deforestation rates increases over time. If lack of enforcement capacity was driving deforestation, the effect of new districts would decrease over time since newer districts would be able to build up enforcement capacity.

permits from the district office that are verified at regular checkpoints. Thus the extraction, transportation or processing of illegal logs would be impossible without the district office acquiescing to such activity (Burgess et al., 2012). In a production forest, the district head may issue permits for logging up to the point of the nationally sanctioned limit, and has the responsibility to ensure that only legal deforestation occurs. In the case of conversion forests, the district government petitions the central government to convert forest land to other uses, such as palm oil plantations, and is responsible for monitoring conversion to ensure that it occurs only in approved areas. In protection and conservation forests, all deforestation activity is illegal. These forests are biodiversity reserves and come under the control of the central government. But the district office is responsible for patrolling these forests to ensure no illegal logging takes place. The ecology of production/conversion forests is distinct from that of protection/conservation forests in part due to historical logging, but the political economy of deforestation is largely similar across the different forest zones.

2.2 Implications for Malaria

Malaria is spread only through the *Anopheles* mosquito and transmission occurs only if the mosquito was previously infected through a blood meal taken from an infected person[11]. Subsequently the malarial parasites from the first blood meal are mixed in with the saliva of the mosquito and then transmitted into the person being bitten. The typical lifespan of the female *Anopheles* is 4-6 weeks, but they can travel distances as far as 75 miles, making the the range of transmission particularly problematic. In Indonesia, malaria is widespread with 44% of the population, or over 130 million people, at risk of contraction (Elyazar, Hay

[11]See: `http://www.cdc.gov/malaria/about/faqs.html`

and Baird, 2011). In any given year, 19% of Indonesian villages report having an outbreak of malaria.

Pattanayak and Pfaff (2009) review potential mechanisms through which deforestation can impact the growth of anopheline larvae and consequently the incidence of malaria. First, post-deforestation land use change in the form of urbanization, construction and agricultural production has been associated with increased incidence of malaria (De Silva and Marshall, 2012; Petney, 2001). Second, deforestation commonly results in migration, which often increases the incidence of malaria for two reasons - migrants act as latent hosts for infectious diseases, acting as transport from an infected area to a non-infected area (Texier et al., 2013) and migrants tend to have limited access to medical facilities. These are socio-economic factors behind the link between deforestation and malaria but there is also an ecological link. Deforestation also alters the disease ecology of malaria. This occurs in two ways. First, cleared lands receive more sunlight and are more susceptible to the formation of puddles with a more neutral pH that favors anopheline larvae development (Pattanayak and Pfaff, 2009; Patz and Olson, 2006; Patz et al., 2000; Walsh, Molyneux and Birley, 1993). Second, deforestation adversely affects biodiversity of the region and increases malaria incidence by favoring the proliferation of malaria related species by reducing or eliminating species that prey on anopheline larvae. The evidence is bi-directional; increased biodiversity also reduces malaria incidence through dilution of disease carrying vectors in the overall species pool in forest ecosystems (Laporta et al., 2013; Yasuoka and Levins, 2007).

3 Data

3.1 Disease and Public Health

The Indonesian statistical agency, *Badan Pusat Statistik* (BPS) conducts a village census *Podes* every 2-3 years that documents a range of village characteristics and events including the incidence and extent of disease outbreaks for malaria, measles, respiratory infections, dengue fever and diarrheal diseases. The survey includes all of Indonesia's over 68,000 villages. During the period of study, *Podes* was conducted in 2000, 2003, 2006, and 2008. Village heads report on whether or not there had been an outbreak of each of these diseases in their village, and if there was an outbreak how many people died. For 2000, 2003 and 2006, no information was collected on the number of people who were infected with malaria. Starting in 2008, the survey also requested information on the number of people infected with malaria distinct from the mortality associated with malaria. In 2008, on average, 14-15 people were infected with malaria in a village that reported an outbreak of malaria. [12] The standard deviation was 20 people. This translated to just over 15 infected persons per thousand persons with a standard deviation of 26 infected persons per 1000 persons. A natural concern with recall health data, particularly at the group level, is measurement error. Since the error in reporting outbreaks or the thresholds used by village heads in declaring an outbreak would be error in the dependent variable, the OLS estimator is likely to be less efficient and generate larger standard errors[13]. As a result, the statistical significance is at

[12]The data is collected through oral recall from village heads and is then verified at the sub-district office. The village head makes a subjective decision on whether or not there was an outbreak in a given year. A kernel density graph of the number of infected individuals conditional on an outbreak is available in the appendix.

[13]This is assuming that measurement error is not correlated with deforestation.

best understated making inference more conservative.

Podes also contains information on the availability of medical facilities including hospitals, integrated health centers, village medicine posts and drug stores in a given village. When such a medical facility is not present in the village, the survey documents the distance to the nearest facility (in kilometers) and how easy it is to access such a facility on a scale of 1-4. This rich data set also includes information on the source of drinking water, sanitation facilities, transportation facilities, land utilization, geography (proximity to the shore, forests etc.) and topography (flat, hilly or valley). Tables 1 - 4 show the summary statistics of the key dependent and independent variables.

3.2 Deforestation Data

Since a large portion of the deforestation in Indonesia results from illegal logging, official records are likely inaccurate. Therefore, I use deforestation data from 2001-2008 based on satellite imagery data directly from Burgess et al. (2012). Deforestation is measured as the number of pixels (250m X 250m) in each district estimated from MODIS satellite images that have at least a 90% probability of tree cover loss compared to the previous year. The variable is converted into 1000s of hectares. While the Indonesian archipelago is comprised of a large number of islands, we focus on the four islands that have substantial forest cover: Sumatra, Kalimantan, Sulawesi and Papua. The remaining islands are excluded since they had negligible forest cover in the baseline year of the study (2000). The key data improvement from previous economic research on deforestation using satellite data (Foster and Rosenzweig, 2003) is building on the Normalized Difference Vegetation Index (NDVI)

(Tucker, 1979) by also making use of differences in spectral signatures between trees and other crops (Jensen, 1996). The deforestation data are then classified by the zone and district of the forest estate into production, conversion, protection or conservation forest (categories defined below). The resulting variable is $TotalDeforestation_{zdt}$ which measures thousands of hectares deforested in forest zone z, in district d at time t.

4 Estimation and Identification Strategy

There are two key parameters of interest. First, what is the effect of deforestation on the probability of malarial outbreak (i.e effects at the extensive margin)? Second, what is the effect of deforestation on the number of malaria deaths conditional on the village having an outbreak of malaria (i.e effects at the intensive margin)?[14] This section explains my strategy for generating plausible estimates of these causal effects.

4.1 Extensive Margin

Since the primary dependent variable is binary (whether or not there was a disease outbreak in the past year), I use a linear probability model[15] in the baseline specification. The primary

[14]Ideally, the intensive margin of study would be the number of individuals infected. However, those data are unavailable for all years prior to 2008, and so the village-level intensive margin being studied is the number of individuals dying from malaria. These characterizations would be different at the individual level, where a person getting infected would mark the extensive margin, and how much they suffer as result of malaria, including mortality, would mark the intensive margin.

[15]In the appendix, I demonstrate the robustness of the baseline results to the use of logit or probit models in place of a linear probability model. I further demonstrate that only 3% of all predicted values lie outside the feasible [0,1] bounds under the LPM. My results are robust to the exclusion of these data points.

specification is,

$$Pr[disease_{vdt} = 1] = \beta_0 + \beta_1 TotalDeforestation_{dt} + \beta_2 X_{vdt} + \mu_d + \eta_{it} + \epsilon_{vdt} \quad (1)$$

The dependent variable $disease_{vdt}$ denotes whether or not there was a disease outbreak in village v in district d at time t and $TotalDeforestation_{dt}$ is the total deforestation in district d at time t, a continuous variable measured in thousands of hectares. X_{vdt} is a vector of controls including access to medical facilities, terrain and geography, altitude, sanitation and trash disposal facilities, dominant fuel source, rainfall average and rainfall variation, dominant occupation in the village as well as the interaction effects of occupation-year, occupation-province fixed effects, forest zone, forest zone-year and forest zone-province fixed effects. These controls are not included in the primary specification (1) but are included in extensions to the primary specification (Table 5, Columns 3-4). μ_d is the district-level fixed effect, η_{it} is the island - year fixed effect and ϵ_{vdt} is the regression residual. Standard errors are clustered at the district level to allow for arbitrary correlation within a district and over time accounting for spatial autocorrelation and serial correlation. I also include population density (population/total area) and number of other villages in the district that had an outbreak of malaria in that year to control for size of district effects and possible spill-over effects.

β_1 (extensive margin) is the parameter of interest, and there are two problems in its consistent identification: a) omission of key within-district time-varying variables that are correlated with both deforestation and malaria may generate omitted variable bias, and b)

malaria may be a factor in the decision to cut down trees resulting in reverse causality or simultaneity bias. The challenge is finding a plausibly exogenous source of variation in deforestation that minimizes these prospective sources of bias, without removing crucial context that is necessary to test for a biophysical mechanism and provide policy-relevant results. Identification is achieved using panel data methods using district fixed effects and island-year fixed effects, relying on the plausible assumption that the timing of deforestation is exogenous to local disease outcomes conditional on observables.

This assumption is justified for a few reasons. First, and as discussed in the previous section, all legal and illegal deforestation in Indonesia requires the implicit or explicit consent of the district government. Each district comprises over 250 villages, on average, making it unlikely that decisions by the district head to grant licenses or acquiesce to illegal deforestation were contingent on malarial outbreaks in specific villages. Second, the use of satellite deforestation data at the 250mX250m resolution means that any smallholder deforestation would have to be both large enough to be noticed and agreed upon by village heads and district governments. That too seems very unlikely. Third, the process of obtaining permits or getting authorization on plans to convert forest land from the national government is a cumbersome bureaucratic process which can last several years between when a permit is requested and when it is granted. Fourth, there were two major national level policies that altered the political economy of deforestation: a) the introduction of a timber certification program aimed at curtailing illegal exports of raw logs and b) the national moratorium on district splitting that temporarily halted the creation of new districts (Burgess et al., 2012). It is highly unlikely that these policies were introduced as a result of local level disease outbreaks.

Furthermore, I test these assumptions in a few ways, and the results of these tests are discussed in section 5. First, I use placebo tests by estimating the effect of deforestation on other diseases whose disease ecology differs from that of malaria. If there were unobservables correlated with health and deforestation, then deforestation would also have an impact on other diseases whose disease ecology differs from that of malaria. Second, I show that the interaction of deforestation with the timing of introduction of the certification (enforcement of the logging ban) is driving the core result. Third, I provide falsification tests for this national level policy and show the effect is specific to the year in which the certification was introduced. Fourth, I show that the results are robust to the inclusion of a range of control variables that are known to impact malaria including health care access, poverty measures, distance from cities, sanitation facilities, population density, government sponsored health interventions, geographic terrain, precipitation and primary sector of employment (Pattanayak and Pfaff, 2009).

4.1.1 Logging Ban Enforcement

In the post-Soeharto era, between 1998 and 2001, Indonesia reduced export taxes on logs from 200 percent to 10 percent, resulting in massive exports of logs. At the end of 2001, Indonesia banned all log exports and in late 2003 introduced the SVLK (Timber Legality Verification System) certification to ensure the legality of traded timber (JakartaPost, 2013). Since the policy and enforcement was nationwide, I interact the enforcement of the log export ban with deforestation to introduce exogenous variation in deforestation driven by a national

policy[16]. The resulting specification is,

$$Pr[disease_{vdt} = 1] = \beta_0 + \beta_1 TotalDeforestation_{dt} * PostLogExportBan_t + \beta_2 TotalDeforestation_{dt} + \beta_3 X_{vdt} + \mu_d + \eta_{it} + \epsilon_{vdt} \quad (2)$$

Since the policy was implemented nationwide, identification of β_1 in this specification rests on the assumption that the timing of the implementation of the national policy was uncorrelated with differential levels of malarial prevalence among districts within an island in Indonesia. Specifically, when we include both the interaction term, and deforestation in the regression, finding a statistically significant β_1 estimate and a statistically insignificant β_2 estimate is consistent with the assumption that the identification of the effect of deforestation on malaria is being driven by exogenous variation in deforestation following the enforcement of a national policy impacting the forest sector.

4.2 Intensive Margin

The second variable of interest is the number of malaria deaths in a given year conditional on there being an outbreak of malaria in that village. I estimate an equation similar to (1),

$$MalariaDeaths_{vdt} = \gamma_0 + \gamma_1 TotalDeforestation_{dt} + \gamma_2 X_{vdt} + \mu_d + \eta_{it} + \epsilon_{vdt} \quad (3)$$

[16]There were no measurable changes to the political economy of deforestation in Indonesia between 2004 and 2007 when there was a national moratorium on district splitting. Burgess et al. (2012) show that as the number of districts increased, the rate of deforestation increased in part due to a cornot competition between district heads to provide illegal wood

where γ_1 is the parameter of interest, $MalariaDeaths$ is the number of malaria deaths in village v of district d at time t, and as in the case of (1) I estimate the fixed effects model.[17] In addition, since the number of malaria deaths is a count variable, I check the robustness of the fixed effects estimates of (3) to re-estimation using a fixed effects poisson model estimated via maximum likelihood and to a fixed effects negative binomial model.

5 Results

The main result of this paper is that deforestation increases malarial incidence. The central result is that the average within sample deforestation of 1000 hectares in a district increases the probability of malarial outbreak in each village in that district by 0.36 percentage points (Table 5, column 4). I treat this as the baseline result. Using the sample average of the probability of malarial outbreak of 0.19, I find that 1000 hectares of deforestation in a district increases the probability of malarial outbreak in *each* village in that district by 2%. The effect is robust to the inclusion of a wide range of controls and to alternative estimation methods. This estimated effect in the robustness checks (table 7) range from 3.3% - 4.6% suggesting that 1000 hectares of deforestation in a district increase the probability of malaria outbreak in *each* village in that district by 2% - 4.6%.

This range of effects on malaria outbreak, using back of the envelope calculations and average infections per outbreak from the 2008 Podes data, translate to 45,000 - 162,000 additional infections each year. Over the 8-year course of the study that is 360,000 - 162,000 additional person-year infections. I then use estimates on average time loss due to malaria-

[17] I run the same specification with "malaria deaths per 1000 persons" as the dependent variable. The results are qualitatively similar, and reported in the appendix.

induced morbidity (2-4 weeks) and measures on per-capita income to estimate the morbidity associated health cost per hectare of deforestation in the range of $58 - $208. These estimates are an order of magnitude higher than the carbon benefit associated with avoided deforestation ($32 - $160 per hectare).

5.1 Placebo Tests

Different diseases have different disease ecologies and as such different mechanisms of transmission. For instance, measles is an airborne disease, while diarrhea is a water-borne disease. I use incidence data on other diseases to perform placebo tests of the core result and the underlying mechanism. In particular, I explore the relationship between deforestation and four other diseases and classes of diseases, namely dengue, measles, diarrhea and respiratory diseases using specification (4) from table 5. As shown in table 6, there is no statistically significant effect of deforestation on the incidence of the other diseases. The lack of any observable effect of deforestation on dengue, measles, diarrhea and respiratory diseases suggests that omitted variables, that could be correlated with both deforestation and health, are not a threat to the internal validity of the central result or the underlying ecological mechanism. The lack of a discernible effect of deforestation on dengue may superficially appear inconsistent with the core result since dengue is also spread via disease carrying vectors, particularly the *Aedes Aegypti* mosquito. However, the *Aedes Aegypti* mosquito, unlike the female *Anopheles* mosquito that carries malaria, has a lifespan of only 2-4 weeks and travels a maximum of 500m($<1/3$ of a mile; 1 hectare $= 10,000m^2$) (*Anopheles* can travel up to 75 miles) during its entire lifetime. This short lifespan and travel-span limit the mechanism of

exposure to dengue since the average within-sample deforestation in this study is just over 1000 hectares per district.

The placebo tests underscore the consistent identification of β_1 in equation (1), since any upward bias generated as a result of omission of variables correlated with human health and deforestation (eg. political economy variables such as corruption, favoritism, etc.) should also show up in the effect of deforestation on other diseases.

5.2 Falsification Tests

In table 7, I demonstrate the robustness of the central result presented in table 5. First, the interaction of deforestation with the enforcement of the log export ban has a 0.87 percentage point effect on the probability of malarial outbreak. Within the sample of this study, the average deforestation in a district (following the logging ban enforcement) increases the probability of malarial outbreak by an estimated 4.56% in every village in that district. The coefficient estimate on deforestation uninteracted is statistically insignificant. The credibility of this result is further strengthened when a "false" year for the enforcement of the log export ban is used instead of the actual year. Specifically, I create a dummy variable that assumes that the certification was introduced in 2006 instead of 2003 and interact that dummy variable with deforestation. The interaction term's coefficient becomes statistically indistinguishable from zero, and the deforestation coefficient has a remarkably similar point estimates to the fixed effects result from table 5 and is statistically significant at the 5% level (table 7, column 2). Finally, there are no lead or lagged effects (one lead, one lag) and I find no evidence for non-linearity (results for various non-linear specifications are shown in the

appendix).

5.3 Type of Forest and Proximity to Forest

In this sub-section, I provide evidence that the underlying mechanism linking deforestation to malarial outbreak appears ecological and is likely driven by reduced biodiversity. First, I disaggregate results by the type of forest in columns (1) and (2) of table 8. As mentioned in the section 2.1, production and conversion forests are where legal (and illegal) deforestation take place and are demarcated as such for the purposes of logging and land use change, respectively. Conversely, conservation and protection forests are designated as biodiversity reserves where all deforestation is illegal. Upon disaggregating the results by the type of forest, I find that the effect is largely concentrated in protection and conservation forests and is four times larger (0.0136 percentage points v. 0.00149 percentage points) than the effect when all forest types are considered. If the underlying mechanism was socio-economic then there would be no reason to expect different effects across different forest types since the major distinction between the forests is that one set is meant for (managed) clearing and the other set is demarcated for biodiversity protection. Because there is a substantial difference in the magnitude (and statistical significance) of the effect on malaria of deforestation across these different forest types, it is likely that the underlying mechanism is an induced ecological response.

Second, I disaggregate results by villages that are classified as "inside" and "on the border" of forests from villages classified as being "away" from forests [18]. These results are

[18]This characterization of the location of the village relative to the forest is provided by village heads and then verified at the district or sub-district office.

shown in column (3) and (4) of table 8. As is consistent with the maintained hypothesis, the effect is concentrated in villages inside or near forests. The effect in villages away from the forest is statistically indistinguishable from zero and less than half the magnitude of the estimated effect inside or near forests (0.00647 percentage points v. 0.00291 percentage points). This result supports the hypothesized mechanism since deforestation induced *Anopheline* growth is likely to have a larger impact on villages inside or near forests than those further away from them because of proximity to growth sites.

5.4 Intensive Margin

The nature of the secondary data used in this paper allows us to study the effects of deforestation on malaria at the both the extensive and intensive margins. Deforestation, if it alters the local disease ecology of malaria, should change the rates of exposure and hence the likelihood of contracting malaria. However, once a person or group of persons have contracted malaria, changes in vector concentration should not impact whether a person survives malaria since that would depend on availability and access to medical facilities. Therefore, if the underlying mechanism linking deforestation to malaria is an ecological one, then deforestation should have a direct impact on the contraction of infection (extensive margin) but conditional on medical facilities and other covariates, should not have a measurable impact on the number of malaria related deaths (intensive margin).

Consistent with this hypothesis, I find a robust result at the extensive margin (as reported in section 5.2) but no effect at the intensive margin (table 9). The absence of evidence of any effect is consistent across the range of fixed effects models and the poisson (table A.5,

column 1) and negative binomial models (table A.5, column 2).

6 Alternative Explanations

In theory, there are three other plausible mechanisms could drive the statistical relationship between deforestation and the incidence of malaria: 1) urbanization and land use post-deforestation drive the incidence of malaria, 2) less remote places with no deforestation are more likely to receive anti-malarial and insecticide treated nets (ITNs) than those with deforestation, and 3) increased migration correlated with deforestation could lead to higher prevalence of malaria since migrants can act as latent hosts of malaria. In this section, I examine these mechanisms as a test of the internal validity of the core result.

6.1 Post-deforestation land use change

A threat to the internal validity of the core result and the underlying mechanism could be that it is not deforestation that causes an increased likelihood of malarial outbreak, but rather the urbanization or land use change (e.g. agriculture, palm oil plantations, etc.) that follows the clearing of forested land (Keesing et al., 2010). This study takes advantage of the rich data in *Podes* and the nature of deforestation in Indonesia to rule out this possibility in three ways. First, I isolate results based on the type of forests being cleared. If post-deforestation land use change drives the relationship between deforestation and malaria, we would observe the effect in production and conversion forests (where forest land can be legally, with permits, cleared for logging and land use change) and not in protection and conservation forests (where forest clearing is illegal but still occurs; see Burgess et al. (2012)).

Table 8 shows the exact opposite when I break down the results by forest zoning.

Second, the inclusion of a large number of controls and dummy variables that are likely correlated with land use change does not alter the central result. In particular, I control for area under rice cultivation, total forest cover in a given year and dominant occupation (disaggregated within agriculture) fixed effects as well as occupation-province and occupation-year fixed effects.[19] This helps remove any spurious correlation that was driven by otherwise unobserved changes in occupational choices in villages, as well as any idiosyncratic changes to these occupations in specific provinces or years.

Finally, if urbanization drives the results, we would expect a direct effect on the outbreak of dengue as well since dengue disproportionately affects urban areas and risk of dengue increases with degree of urbanization (Bhatt et al., 2013). The lack of any effect on dengue prevalence suggests that urbanization can not explain the effect of deforestation on increased risk of malarial incidence.

6.2 Correlation between anti-malarial programs and deforestation

If there is correlation between anti-malarial programs and deforestation in a spatially systematic manner, in that presumably less remote places with no forests are more likely to receive anti-malarial devices, services and programs, then the fixed effects estimator will generate inconsistent results. I address this concern in two ways. First, I disaggregate results by proximity of villages to the forest (table 8, columns 3-4). When considering only the set of villages inside or near forests(table 8, column 4), the results are stronger (both

[19]The dominant occupation of a village was one of eleven different options - crops and plantations, livestock, inland fisheries, marine fisheries, forestry, other agriculture, mining, industry, wholesale/retail, services and other occupations. Data on percentage of population employed in each sector were unavailable.

in magnitude and statistical significance) suggesting that such a correlation, if it exists, at worst downward biases the central result. Second, the central result is robust to controlling for percentage (table 5, columns 3-4) of the population in each village that receives some form of welfare payment (poor cards) or subsidized medical care (health cards) making it unlikely that such a spatially systematic correlation exists that would upward bias the main finding of this paper.

6.3 Migration

A concern with the internal validity of the central result and the underlying ecological mechanism is that increased migration correlated with deforestation could lead to higher prevalence of malaria because migrants can act as latent hosts of malaria (Texier et al., 2013). This can be ruled out for a few reasons. First, the placebo tests demonstrate that the effect is specific to malaria; migrants working in deforestation would have to be carrying malaria and no other disease. The public health literature has documented several instances where migratory behavior has been associated with the outbreak of other diseases such as diarrhea and dengue in addition to malaria (Eisenberg et al., 2006). Second, I explicitly test for migration using population and population density as control variables and find that the result doesn't change.

However, migration could still bias the core result if conditional on migration rates, migrants to villages experiencing deforestation are more likely to carry malaria than those to villages not experiencing deforestation. This concern can be ruled out for a few reasons. First, the core result remains robust to the inclusion of the dominant occupation

in a village and occupation-year and occupation-province fixed effects. These allow me to control for changes in dominant occupation specific to a particular province or year that could be correlated with migration and deforestation. Second, if the migration hypothesis is true, districts with production and conversion forests should attract a larger migrant population than districts with protection and conversion forests since average deforestation in production and conversion forests is substantially (five times) higher than in protection and conversion forests. Thus, if migration were the underlying mechanism then production and conversion forests would see larger effects of deforestation on malaria since they attract more deforestation related migrants. Yet, when the results are disaggregated by forest zone, the effect is substantially stronger in protection and conservation forests, making it unlikely that migration is driving the effect of deforestation on increased malaria incidence.

Therefore, migration would be a threat to the validity of the ecological mechanism only if migrants near production and conservation forests differ systematically from those near conservation and protection forests and that these migrants are unlikely to carry any other disease except malaria. This seems unlikely.

7 Conclusion

Every year over 300 million people are affected by malaria and other infectious diseases resulting in losses in productivity, labor supply, and education as well as increased human suffering. This paper provides causal evidence on the effect of deforestation on the incidence of malaria. I find that using within-sample averages, the mean deforestation in a district in a given year resulted in a 2% - 7.2% increase in the incidence of malaria in every village

in that district. I rule out post-deforestation land use change, anti-malarial programs and migration as possible explanations for this effect, highlighting the likely ecological response that drives the effect.

Given the lack of consensus in the political institutions towards action on climate change, this paper provides further motivation for forest conservation and in particular strong evidence of the direct human health impacts of deforestation. First, the absolute numbers are large; deforestation in Indonesia over the 8 year period of this study can explain 360,000 - 1,296,000 additional infected individuals. This accounts for This represents a sizable human health cost of deforestation to the tune of $58 - $208 per hectare. This is substantially larger than the carbon benefit of avoided deforestation of $32 - $160 per hectare. Second, the effect seems more pronounced in villages closer to forests that those further away highlighting that there are indeed local benefits to forest conservation in addition to the more often discussed global carbon benefits. This is particularly important in light of Indonesia's adoption of the Rainforest Standard (RFS), a payment for ecosystem services program. Third, the concentration of the effect in protection and conservation forests means that preservation of forest reserves provides health benefits to local communities. However, these are short term results and provide motivation for research in to the long term relationship between natural resource degradation and local public health.

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Tables

Table 1: Fraction of Villages with Disease Outbreaks (Village Level)

(Yes = 1, No = 0)	(1) Full Sample	(2) In/Near Forest	(3) Away from Forest
Was there an outbreak of malaria?	0.190	0.243	0.169
	(0.392)	(0.429)	(0.374)
Was there an outbreak of dengue?	0.0620	0.0421	0.0702
	(0.241)	(0.201)	(0.255)
Was there an outbreak of diarrhea?	0.176	0.204	0.164
	(0.380)	(0.403)	(0.370)
Was there an outbreak of respiratory?	0.104	0.122	0.0971
	(0.306)	(0.327)	(0.296)
Was there an outbreak of measles?	0.0806	0.0892	0.0771
	(0.272)	(0.285)	(0.267)
Observations	143,305	41,575	101,730

Standard deviations in parentheses. Column 2 shows mean and standard deviation for the sub-sample of villages inside or on the border of the forest area. Column 3 shows mean and standard deviation for the sub-sample of villages. These characterizations are made by the village head and verified at the sub-district office. All row-wise differences between columns (2) and (3) are statistically significant.

Table 2: Number of Deaths in a village in a given year as a Result of Disease Outbreaks (Village Level)

	(1) Full Sample	(2) In/Near Forest	(3) Away from Forest
Number of deaths from malaria	0.873	0.746	0.945
	(5.275)	(4.498)	(5.670)
Number of deaths from dengue	0.240	0.165	0.282
	(2.190)	(2.026)	(2.277)
Number of deaths from diarrhea	0.779	0.684	0.834
	(5.197)	(3.915)	(5.804)
Number of deaths from respiratory diseases	0.665	0.492	0.765
	(5.958)	(4.767)	(6.541)
Number of deaths from measles	0.341	0.260	0.387
	(3.036)	(2.639)	(3.240)
Observations	12,454	4,531	7,923

Standard deviations in parentheses. Column 2 shows mean and standard deviation for the sub-sample of villages inside or on the border of the forest area. Column 3 shows mean and standard deviation for the sub-sample of villages. All three columns only report averages from the sample of villages that reported an outbreak of that particular disease. All row-wise differences between columns (2) and (3) are statistically significant.

Table 3: Average Forest Cover and Deforestation by Forest Zone (District Level)

	(1) Full Sample	(2) Production/Conversion	(3) Protection/Conservation
Deforestation (1000 Ha) - All Districts	0.724	1.269	0.199
	(2.821)	(4.008)	(1.022)
N	9,352	3,280	3,672
Deforestation (1000 Ha) - Deforested Districts	0.986	1.671	0.310
	(3.253)	(4.526)	(1.262)
N	6,864	2,491	2,359
Forest Cover (1000 Ha)	113.6	160.7	90.36
	(206.6)	(279.6)	(155.5)
N	10,521	3,690	4,131

Sample means by district in 1000s of hectares. Standard deviation in parentheses followed by sample observations. For more details see Burgess et al. (2012). All row-wise differences between columns (2) and (3) are statistically significant.

Table 4: Key Independent Variables (Village Level)

	(1) Full Sample	(2) In/Near Forest	(3) Away from Forest
100s of Families Receiving Poor Status Papers	0.308	0.226	0.342
	(0.779)	(0.674)	(0.815)
100s of Families Receiving Health Cards	0.792	0.600	0.870
	(1.404)	(1.071)	(1.511)
Hospital in Village? (Yes = 1; No = 0)	0.485	0.419	0.512
	(0.853)	(0.813)	(0.868)
Ease of Access to Nearest Hospital[a]	2.610	3.002	2.453
	(0.888)	(0.840)	(0.857)
Population Density	0.00650	0.00142	0.00855
(1000s of person per hectare)	(0.0334)	(0.00788)	(0.0391)
Rice Field Area (1000 Ha)	0.242	0.279	0.228
	(1.133)	(1.476)	(0.960)
Is the village by a river? (Yes = 1, No = 0)	0.711	0.801	0.675
	(0.453)	(0.399)	(0.468)
Elevation Above Sea Level (100 meters)	1.866	2.455	1.629
	(4.589)	(5.815)	(3.964)
Mean Annual Rainfall (1000mm)[b]	0.0697	0.0728	0.0684
	(0.0172)	(0.0184)	(0.0165)
Standard Deviation in Rainfall (1000mm)[b]	0.0315	0.0305	0.0319
	(0.00956)	(0.00901)	(0.00974)
Observations	132,676	38,127	94,549

Standard deviations in parentheses. Column 2 shows mean and standard deviation for the sub-sample of villages inside or on the border of the forest area. Column 3 shows mean and standard deviation for the sub-sample of villages. These characterizations are made by the village head and verified at the sub-district office.

[a] Ease of access defined as follows. 0 = hospital in village, 1 = very easy, 2 = easy, 3 = difficult and 4 = very difficult. These delineations are made by the village head and verified at the district/sub-district office.

[b] Rainfall variables calculated over the sample range of 2001-2008.

Table 5: Impact of Deforestation on the Probability of Malarial Outbreak

Was there an outbreak of Malaria in the Previous Year?	(1)	(2)	(3)	(4)
Total Deforestation in the Previous Year (1000 Ha)	0.00203***	0.00319**	0.00287**	0.00364**
	(0.000607)	(0.00139)	(0.00122)	(0.00154)
One Lead, One Lag		X	X	X
District Fixed Effects	X	X	X	X
Island X Year Fixed Effects	X	X	X	X
Distance to Cities, Families on Welfare			X	X
Agricultural Production, Forest Cover			X	X
Medical Facilities Access, Terrain, Geography			X	X
Sanitation, Trash Disposal, Fuel Source and Rainfall				X
Forest Zone, Forest Zone x Province, Forest Zone x Year				X
Income Source, x Province FE, x Year FE				X
Observations	111,201	111,201	100,570	100,570
R-squared	0.125	0.125	0.128	0.140

Standard errors clustered at the district level in parentheses. Statistical significance is denoted as *** p <0.01, ** p <0.05, * p <0.1. Columns 1 and 2 include no controls other than district and island x year fixed effects. Column 3 includes limited controls and column 4 includes the full set of controls. Column 1 includes only the previous year deforestation variables. Columns 2-4 include 3 time periods of deforestation variables.

Table 6: Impact of Deforestation on the Probability of Outbreak of Different Diseases

Was there an outbreak of disease in the previous year?	(1) Malaria	(2) Dengue	(3) Diarrhea	(4) Respiratory	(5) Measles
Total Deforestation in the Previous Year (1000 Ha)	0.00364**	0.00102	0.00125	0.00127	0.00119
	(0.00154)	(0.00101)	(0.00146)	(0.00151)	(0.00156)
Observations	100,570	100,570	100,570	100,570	100,570
R-squared	0.140	0.141	0.136	0.136	0.136

Standard errors clustered at the district level in parentheses. Statistical significance is denoted as *** p <0.01, ** p <0.05, * p <0.1. All specifications include the full range of controls in columns (4) of table 5. The unit of observation is village-forestzone-year.

Table 7: Impact of Deforestation on the Probability of Malarial Outbreak: Robustness Checks

Was there an outbreak of Malaria in the Previous Year?	(1) Log Ban Enforcement	(2) Falsification
Total Deforestation in the Previous Year (1000 Ha)	-0.00139	0.00412**
	(0.00159)	(0.00190)
Deforestation in Previous Year X Log Ban Enforcement	0.00867***	
	(0.00297)	
Deforestation in Previous Year X False Log Ban		0.000305
		(0.00354)
Observations	74,691	74,691
R-squared	0.145	0.145

Standard errors clustered at the district level in parentheses. Statistical significance is denoted as *** p <0.01, ** p <0.05, * p <0.1. All columns include full set of controls in column (1) of table 5. In column (1), deforestation two years ago and deforestation two years ago interacted with logging ban enforcement were both statistically significant but in opposite direction and the sum of the two is statistically indistinguishable from zero. That result is available in the appendix.

Table 8: Impact of Deforestation on the Probability of Outbreak of Malaria: Disaggregated Results by Forest Zone Proximity to Forests

Was there an outbreak of Malaria in the Previous Year?	(1) Conservation/Protection	(2) Production/Conversion	(3) Outside Forest	(4) Inside/Near Forest
Total Deforestation in the Previous Year (1000 Ha)	0.0136***	0.00149	0.00291	0.00647***
	(0.00274)	(0.00183)	(0.00212)	(0.00193)
Observations	40,938	33,753	53,070	21,621
R-squared	0.142	0.170	0.146	0.180

Standard errors clustered at the district level in parentheses. Statistical significance is denoted as *** p <0.01, ** p <0.05, * p <0.1. All specifications include the full range of controls in columns (4) of table 5. The unit of observation is village-forest zone-year.

Table 9: Impact of Deforestation on the Malaria Related Deaths

Number of people who died from malaria	(1) FE	(2) FE	(3) FE	(4) FE
Total Deforestation in the Previous Year (1000 Ha)	0.0295	0.00443	0.0172	0.00882
	(0.0203)	(0.0328)	(0.0238)	(0.0258)
Observations	111,201	111,201	100,570	100,570
R-squared	0.143	0.143	0.153	0.184

Standard errors clustered at the district level in parentheses. Statistical significance is denoted as *** p <0.01, ** p <0.05, * p <0.1. Columns (1) - (4) use the same set of controls as Columns (1) - (4) respectively of table 5.

A Appendix

A.1 Descriptives of "Outbreak" of malaria

The following is a kernel density plot of the number of infected persons in a village that declared an outbreak in 2008. The range is 1 to 98.

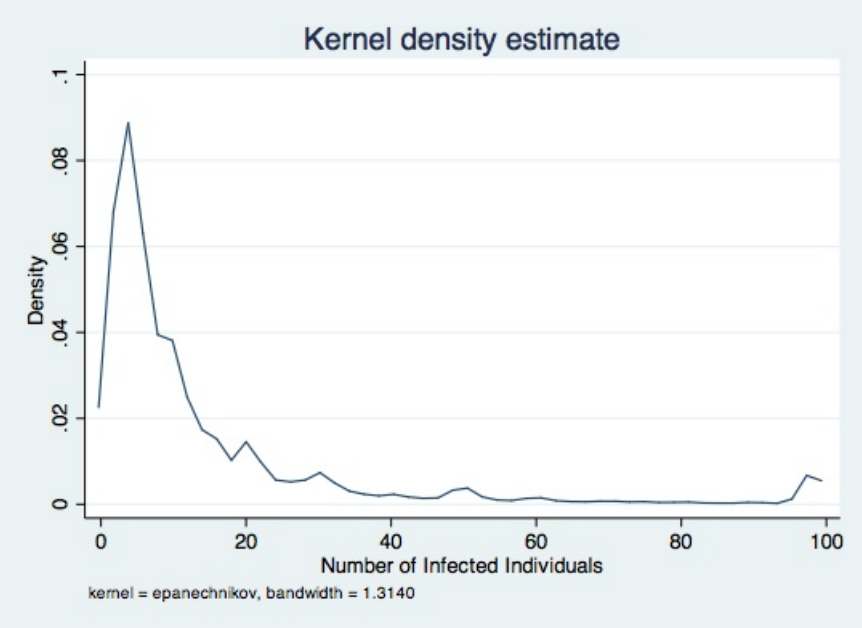

Figure 1: *Kernel Density Distribution of Infections conditional on reported outbreak in 2008*

A.2 Choice of Estimator - LPM v. Logit, Probit

The central results are robust to the choice of estimator. Typically, coefficients on logit and probit estimates are 4 and 2.5 times those from the linear probability models. Table A.1 shows that the magnitudes are to the order 8.5 and 4.75 for logit and probit respectively. That means that at best, the linear probability model provides an underestimate of the true effect of deforestation on disease incidence.

Second, I also check to see the percentage of predicted values that lie outside the (0,1) interval. Figure 1 shows the kernel density plot. Only 4% of all predicted observations fell outside the range. My results remain robust to the exclusion of those data points as shown in table A.2.

Table A.1: Comparison of Results from LPM, Logit and Probit

Was there an outbreak of malaria in the previous year?	(1) LPM	(2) Logit	(3) Probit
Total Deforestation in the Previous Year (1000 Ha)	0.00287**	0.0246***	0.0136**
	(0.00122)	(0.00951)	(0.00530)
Observations	100,570	100,262	100,262

Standard errors clustered at the district level in parentheses. Statistical significance is denoted as *** p <0.01, ** p <0.05, * p <0.1. All specifications include the limited range of controls in column (3) of table 5. I don't use the full set of controls in order to reduce computational time associated with the convergence of logit/probit estimators when using maximum likelihood. The unit of observation is village-forestzone-year.

Table A.2: Linear Probability Model: Full Sample v. Limited Sample

Was there an outbreak of malaria in the previous year?	(1) Full Sample	(2) Limited Sample
Total Deforestation in the Previous Year (1000 Ha)	0.00287**	0.00337***
	(0.00122)	(0.00122)
Observations	100,570	96,536
R-squared	0.128	0.122

Standard errors clustered at the district level in parentheses. Statistical significance is denoted as *** p <0.01, ** p <0.05, * p <0.1. All specifications include the limited range of controls in column (3) of table 5. Limited sample column (2) results are obtained by reestimating equation in column (1) after dropping observations whose predicted values lie outside the feasible [0,1] range. The unit of observation is village-forestzone-year.

A.3 Linear v. Non-Linear Specification

In this section, I check the robustness of my linear estimation results to non-linear specifications. There is no evidence to support the presence of non-linearities. This could be on account of the relatively short time frame over which we estimate our results.

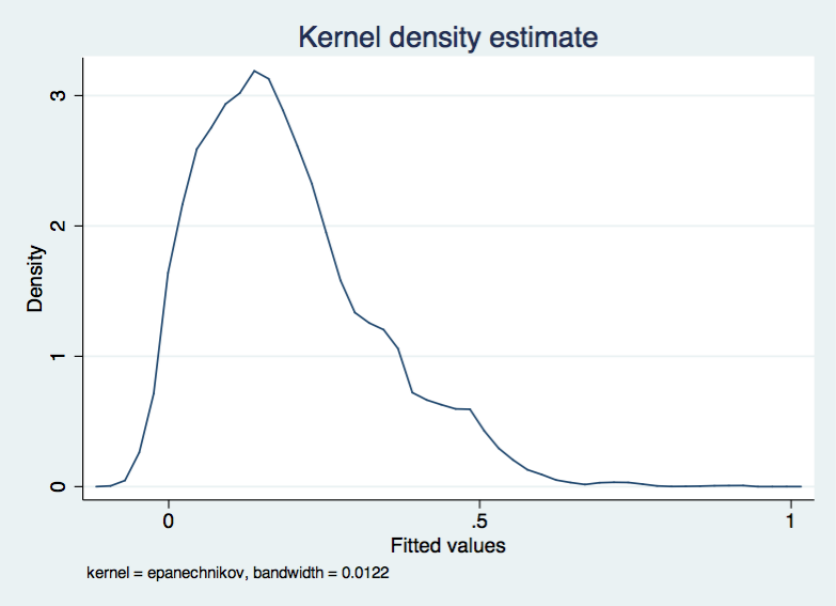

Figure 2: *Kernel Density Distribution of Predicted Outbreaks: Linear Probability Model*

Table A.3: Testing for presence of non-linearities

Was there an outbreak of malaria in the previous year?	(1) Linear	(2) Linear Approximation	(3) Quadratic
Total Deforestation in the Previous Year (1000 Ha)	0.00364**	0.00365**	0.00384
	(0.00154)	(0.00153)	(0.00479)
Any Deforestation in Previous Year?		0.00294	
		(0.0124)	
Deforestation in Previous Year - Squared			-0.0000076
			(0.000155)
Constant	0.547***	0.548***	0.547***
	(0.142)	(0.142)	(0.142)
Observations	100,570	100,570	100,570
R-squared	0.140	0.140	0.140

Standard errors clustered at the district level in parentheses. Statistical significance is denoted as *** p <0.01, ** p <0.05, * p <0.1. All specifications include the limited range of controls in column (4) of table 5. Column (1) is the same as Column (4) of 5. Column (2) includes a dummy variable = 1 if there was any deforestation in the previous year. Column (3) includes a quadratic term. The unit of observation is village-forestzone-year.

A.4 Malaria Related Deaths

In this section, I discuss the robustness of the results in equation (3). In particular, I provide 2 forms of robustness checks: 1) Using deaths per thousands of persons as the dependent variable 2) estimating (3) as a poisson regression and a negative binomial regression.

A.4.1 Cases per thousands of Persons

As shown below the results are qualitatively similar to table 9, in that conditional on a malarial outbreak, there is no effect of deforestation on malaria related deaths.

Table A.4: Using "Number of Deaths per thousand people" as the dependent variable

	(1)	(2)	(3)	(4)
Number of Malaria Deaths Per Thousand People	FE	FE	FE	FE
Total Deforestation in the Previous Year (1000 Ha)	8.66e-08	6.53e-08	1.63e-07	1.82e-07
	(1.62e-07)	(9.59e-08)	(1.11e-07)	(1.24e-07)
Total Deforestation Two Years Ago (1000 Ha)	-7.52e-08		-2.19e-07	1.62e-08
	(2.51e-07)		(1.68e-07)	(1.63e-07)
Observations	21,482	21,482	19,088	19,088
R-squared	0.238	0.237	0.235	0.262

Standard errors clustered at the district level in parentheses. Statistical significance is denoted as *** p <0.01, ** p <0.05, * p <0.1. Columns (1) - (4) use the same set of controls as Columns (1) - (4) respectively of table 5.

A.4.2 OLS v. Poisson, Negative Binomial

I demonstrate below the robustness of equation (3) to poisson and negative binomial regressions. The poisson and negative binomial results are interpreted as semi-elasticities. The key result doesn't change with the specification - that conditional on a village having an outbreak of malaria, deforestation has no impact on malaria related deaths.

Table A.5: OLS v. Poisson, Negative Binomial

Number of people who died from malaria	(1) Poisson	(2) Negative Binomial
Total Deforestation in the Previous Year (1000 Ha)	0.00891	0.00417
	(0.0208)	(0.0104)
Observations	99,297	100,570

Standard errors clustered at the district level in parentheses. Statistical significance is denoted as *** p <0.01, ** p <0.05, * p <0.1. All columns use limited controls as in column (3) of table 5. The poisson and negative binomial coefficients are interpreted as semi-elasticities.

A.5 Back of the envelope calculations

In this section, I describe the assumptions used in making back of the envelope calculations.

A.5.1 Probability of Outbreak to Infected Individuals

The baseline result is that 1000 hectares of deforestation increases malarial outbreak by 2% in every village in that district. An outbreak, on average, means that 15 individuals are infected. There are 250 villages, on average, in each district. That means 1000 hectares of deforestation results in $0.02x15x250 = 75$ additional infected individuals in each district. Between 2001 and 2008, Indonesia lost 600,000 hectares of forest cover each year. That translates to 45,000 additional infected individuals each year. The same calculations can be repeated at the upper bound estimate of 7.2%, which translates to 162,000 additional infected individuals each year.

A.5.2 Morbidity Cost of Deforestation based on Per Capita Income

A single bout of malaria costs 10-20 working days [20]. Based on Indonesia's per capita income (USD 9,260 PPP), that means 1000 hectares translates to USD 57,900 - USD 208,400. On a per-hectare basis, that translates to $58 - $208.5.

[20] See: `http://malaria.jhsph.edu/about_malaria/`