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Does deforestation increase malaria prevalence? Evidence from satellite data and health surveys

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ABSTRACT

Deforestation can increase malaria risk factors such as mosquito growth rates and biting rates in some settings. But deforestation affects more than mosquitoes—it is associated with socio-economic changes that affect malaria rates in humans. Most previous studies have found that deforestation is associated with increased malaria prevalence, suggesting that in some cases forest conservation might belong in a portfolio of anti-malarial interventions. However, previous peer-reviewed studies of deforestation and malaria were based on a small number of geographically aggregated observations, mostly from the Brazilian Amazon. Here we combine 14 years of high-resolution satellite data on forest loss with individual-level and nationally representative malaria tests for more than 60,000 rural children in 17 countries in Sub-Saharan Africa, where 88% of malaria cases occur. Adhering to methods that we pre-specified in a pre-analysis plan, we used multiple regression analysis to test ex-ante hypotheses derived from previous literature. Aggregated across countries, we did not find either deforestation or intermediate levels of forest cover to be associated with higher malaria prevalence. In nearly all (n = 78/84) country-year-specific regressions, we also did not find deforestation or intermediate levels of forest cover to be associated with higher malaria prevalence. However, we can not rule out associations at the local scale or beyond the geographic scope of our study region. We speculate that our findings may differ from those of previous studies because deforestation in Sub-Saharan Africa is largely driven by the steady expansion of smallholder agriculture for domestic use by long-time residents in stable socio-economic settings where malaria is already endemic and previous exposure is high, while in much of Latin America and Asia deforestation is driven by rapid clearing for market-driven agricultural exports by new frontier migrants without previous exposure. These differences across regions suggest useful hypotheses to test in future research.

1. Introduction

Malaria incidence fell by 41% globally between 2000 and 2015, yet malaria remains a substantial health burden in many low- and middle-income countries with 212 million cases and 429,000 deaths worldwide in 2015 (WHO, 2016). Proven anti-malarial interventions include insecticide-treated bed nets, indoor residual spraying, and prompt clinical treatment (Bhatt, 2015), as well as environmental management (e.g. drainage and canal lining) and modified human habitation (e.g. house siting and design) (Keiser, Singer, & Utzinger, 2005).

The loss of natural forest cover ("deforestation") has been found to heighten malaria risk factors in some settings, suggesting that in some circumstances forest conservation could belong in a portfolio of anti-malaria interventions. However, more research is needed to establish whether and when deforestation increases malaria prevalence in humans.

Deforestation has been found to increase malaria risk factors in some settings through multiple ecological mechanisms. Relative to forests, deforested lands have higher temperatures (e.g. Lindblade, Walker, Onapa, Katungu, & Wilson, 2000), more sunlight, and more standing water (Patz, Graczyk, Geller, & Vittor, 2000), which can result in accelerated life cycles (Afrane, Lawson, Githeko, & Yan, 2005) faster pupation and growth rates (Munga, 2006), longer survival time (Zhong, 2016), and higher biting rates (Petney, 2001; Vittor et al., 2006) of malaria-transmitting mosquitoes. Relative to forests, cleared lands also have fewer insectivores, more species

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competing for ecological niche, and arguably fewer "dead-end hosts" to dilute malaria (Laporta, 2013; Wood et al., 2014).

There is heterogeneity in the strength and direction of these ecological mechanisms (Tucker Lima, Vittor, Rifai, & Valle, 2017). For example, different regions have different dominant malaria parasite species (Kar, Kumar, Singh, Carlton, & Nanda, 2014) and *Anopheles* malaria vectors (Kiszewski, 2004); deforested areas may be favored by some mosquito species but not others (e.g. Yasuoka & Levins, 2007; Kar et al., 2014; Burkett-Cadena & Vittor, 2017). Deforestation is canonically considered to lead to increases in malaria-transmitting mosquitoes in Africa and Latin America but decreases in Asia (Guerra, Snow, & Hay, 2006). Small clearings can increase malaria by producing land cover more hospitable to larvae, while large clear cuts can decrease larvae (Singer & de Castro, 2006). While primary forest might suppress mosquitoes, secondary regrowth might encourage them (Vittor, 2009).

Deforestation not only has ecological effects on malarial mosquitoes; it is also associated with socio-economic changes that affect malaria rates in humans. As one example, deforestation is commonly associated with unstable conditions, including rapid in-migration, new human exposure (e.g. Friedrich, 2016) and low immunity, poor housing quality, and sparse availability of health services, all of which can result in "frontier malaria" (de Castro, Monte-Mor, Sawyer, & Singer, 2006). Early stages of frontier settlement can have larger effects (de Castro et al., 2006), with malaria declining in later stages (Baeza, Santos-Vega, Dobson, & Pascual, 2017). Singer and de Castro (2006) suggest that "frontier malaria" effects dissipate after 6–8 years. As another example, deforestation is correlated with higher incomes and wealth (e.g. Busch & Ferretti-Gallon, 2017) and thus greater ability to undertake avoiding behavior. Ijumba and Lindsay (2001) described a "paddies paradox" in which communities near irrigation projects had less rather than more malaria in spite of increases in *Anopheles* mosquitoes, perhaps because increased wealth from irrigation led to increased use of bed nets and better access to improved healthcare.

Furthermore, many other factors besides deforestation affect malaria prevalence in humans, including temperature and precipitation (Beck-Johnson, 2013; Mordecai, 2013; Parham & Michael, 2010), seasonality (Hay, Snow, & Rogers, 1998), age (WHO, 2016), access to health facilities, and avoidance behaviors such as installing bed nets and window screens, reducing standing water, and spraying to repel mosquitoes. All of which is to say, "the linkage between deforestation. . .and malaria transmission is a subtle process requiring analysis at several temporal and spatial scales" (Singer & de Castro, 2006). A recent systematic review of studies of deforestation and malaria risk in the Brazilian Amazon "failed to find overwhelming evidence supporting a consistent simple and straightforward relationship between forests, deforestation rate, and malaria" (Tucker Lima et al., 2017).

Thus, even where there are established ecological links between deforestation and malaria-transmitting mosquito density, lower rates of deforestation might not always lead to lower malaria prevalence in humans in practice. We present a conceptual model of the coupled human and natural system of forest cover, deforestation, and malaria in Fig. 1.

At least in principle, econometric studies using multiple regressions can be used to disentangle the multiple channels through which deforestation could be associated with malaria rates while controlling for confounding factors. A recent strand of peer-reviewed literature has used multiple regressions to test the hypothesis that deforestation increases malaria rates in humans (Table 1). Most (n = 7/9) of these studies found that deforestation was associated with increased malaria prevalence (Austin, Bellinger, & Rana, 2017; Santos & Almeida, 2018; Wayant, Maldonado, de Arias, Cousino, & Goodin, 2010) or incidence (Fornace et al., 2016; Moreira Chaves, Conn, Mendoza Lopez, & Mureb Sallum, 2018; Olson, Gangnon, Silveira, & Patz, 2010; Terrazas et al., 2015) while two studies found that it was not (Hahn, Gangnon, Barcellos, Asner, & Patz, 2014; Valle & Clark, 2013). Studies in the grey literature (Berazneva & Byker, 2017;

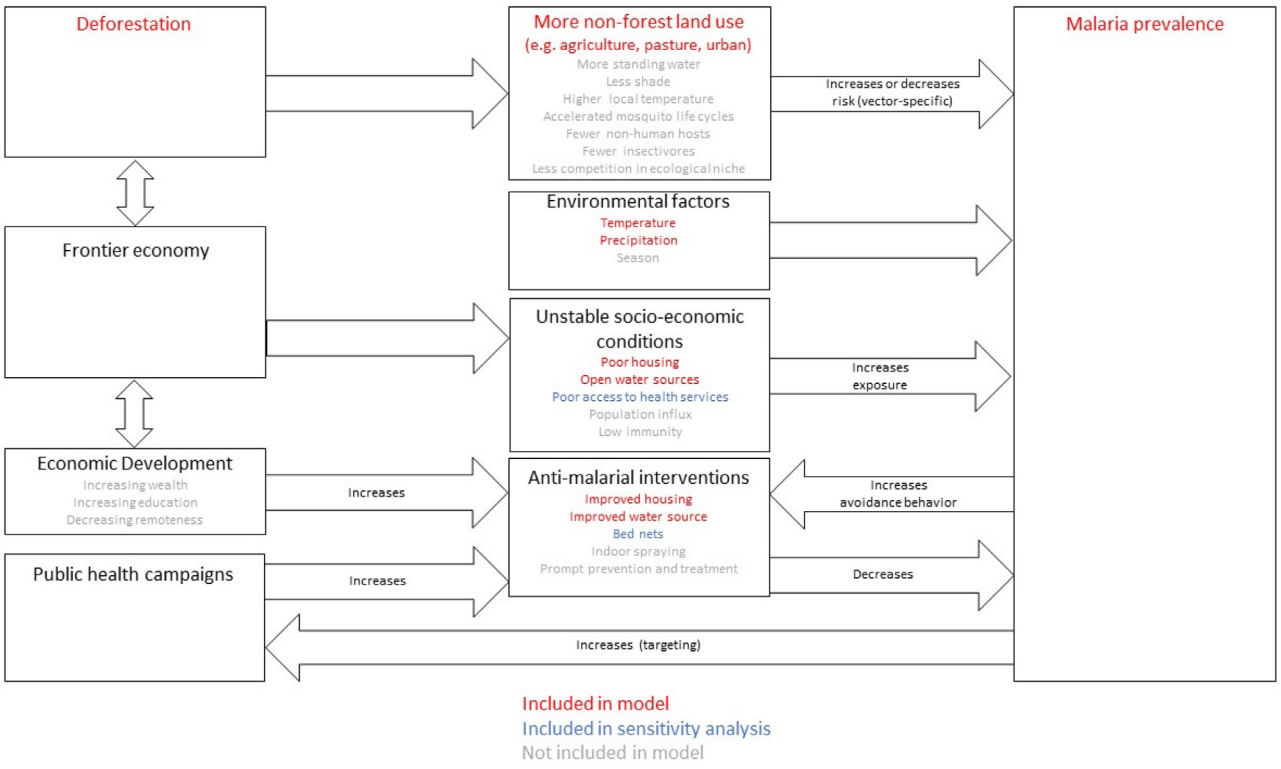

Fig. 1. Coupled human-natural system model of deforestation and malaria.

Table 1
Findings of previous peer-reviewed studies.

Study	Methods	Explanatory variables	Positive association between deforestation or forest cover reduction and malaria?
Wayant et al. (2010)	Univariate correlation between NDVI-forest cover change interaction and malaria case rates over 260 months in two departments in Paraguay	Forest cover change	YES
Olson et al. (2010)	Conditional correlation in cross-sectional regressions of deforestation and malaria incidence across 54 health districts in Mancio Lima County in Acre, Brazil	Deforested land area, deforestation, access to care, area	YES
Valle and Clark (2013)	Association between forest cover and malaria incidence across 401 20 km radii around towns in the Brazilian Amazon	Forest cover, deforestation, population, lagged precipitation, lagged drought index	NO
Hahn et al. (2014)	Cross-sectional regression of deforestation and incidence in 602 municipalities of the Brazilian Amazon	Deforested land area, deforestation, Paved road density, unpaved road density, area affected by fire,	NO
Terrazas et al. (2015)	Correlation between incidence of malaria and average annual deforestation rate across 62 municipalities of the state of Amazonas, Brazil	Forest cover, deforestation, human development, education, income, poverty, unemployment, health surveillance, watercourses	YES
Fornace et al. (2016)	Association between incidence of *P. knowlesi* and historical forest loss within a 1–5 km radius of 405 villages in Sabah, Malaysia	Forest cover, deforestation, elevation	YES
Austin et al. (2017)	Structural equation model of malaria prevalence rate in 2013 vs self reported changes in forest cover (FAO FRA) 2012–2013 across 67 countries	Forest cover change, latitude, GDP per capita, Sub-Saharan Africa, agriculture as % of GDP, rural population growth, public health conditions	YES
Moreira Chaves et al. (2018)	Pooled correlations between deforestation plus degradation and malaria incidence across municipality-months (DETER) or years (PRODES) in the Brazilian Amazon	Rainfall, timber and charcoal production, number of forest patches	YES
Santos and Almeida (2018)	Panel regression of deforestation and malaria prevalence in 757 municipalities in the Brazilian Amazon over 10 years	Deforestation, deforestation-squared, population density, forest stock, GDP, sanitary conditions, spending on health and sanitation, area planted in soybeans, area planted in other crops, head of livestock, temperature, precipitation	YES
This study	Conditional correlation in cross-sectional regression of deforestation and malaria prevalence in 60,305 children in 17 African countries; fever in 469,539 children in 41 countries	Forest cover, deforestation, temperature, precipitation, child age, floor type, water source	NO

Chakrabarti, 2018; Garg, 2014; Pattanayak, Corey, Lau, & Kramer, 2010) have found the same association, with the exception of Assaf, Gomez, Juan, & Fish, 2018. We do not refer to grey literature further in this paper.

However, the geographic evidence base of peer-reviewed econometric studies is narrow. Six of the nine studies were from the Brazilian Amazon, with one from Paraguay, one from Malaysia, and one study that compared national-level data across 67 countries. No previous peer-reviewed study was from Africa, where 88% of malaria cases occur (WHO, 2016). Furthermore, all of the peer-reviewed studies used data aggregated to the level of a few dozen or a few hundred jurisdictions, rather than localized grid-cell-level data on deforestation and individual-level data on malaria. Analyses of data aggregated to the level of heterogenous jurisdictions are potentially prone to the ecological fallacy (Piantadosi, Byar, & Green, 1988) or biases arising from unmeasured confounding factors.

The primary goal of our study was to test whether the deforestation-malaria relationship found by previous studies generalizes to a large and diverse set of countries across Sub-Saharan Africa. We tested two ex ante hypotheses derived from previous literature: first, that malaria prevalence in humans is higher where deforestation is higher (e.g. Vittor, 2009); and second, that malaria prevalence is higher where forest cover is intermediate (de Castro et al., 2006), i.e., that malaria prevalence has an inverted-U-shaped relationship with respect to forest cover. We used the most accurate currently available data set for measuring forest cover and deforestation over large scales—30-meter resolution Landsat. We used nationally representative data on malaria in individual children and data on deforestation at the local grid-cell level.

Because our sample size (n ~ 60,000 individuals) was considerably larger than most previous studies, we were able to explore heterogeneity. Most importantly, we used 23 disaggregated country-year survey waves conducted 84 tests of the relationship between deforestation, forest cover, and malaria, and 90 disaggregated country-year survey waves to conduct 176 tests of the relationship between deforestation, forest cover, and fever.

We sought to enhance the credibility of our analysis in two ways. First, prior to conducting any analyses, we pre-selected model specification and included variables based on explicit ex ante hypotheses grounded in previous literature and our understanding of the coupled human-natural system rather than based on an exploration of our data. Second, we wrote and adhered to a pre-analysis plan, in which all analyses to be undertaken in the paper were specified in writing in advance.[1]

2. Methods

2.1. Data

We obtained two indicators of malaria in children from the United States Agency for International Development's Demographic and Health Survey Program, which includes both Demographic and Health Surveys and Malaria Indicator Surveys, which followed the same protocols. The first dependent variable was the binary presence or absence of malaria in individual children under age five. This measure was obtained from rapid diagnostic tests ("rapid tests"), which detect parasite antigens rather than actual parasites (Florey, 2014). They are relatively simple, as they require only a single drop of blood and do not require skilled technicians or access to laboratory equipment (Florey, 2014). However, rapid

[1] Our time-stamped pre-analysis plans are logged with the Registry for International Development Impact Evaluations (RIDIE) at ridie.3ieimpact.org with study IDs 5b7a6687cb876 and 5b6ab5379f030.

tests are prone to false positives (Kiemde, 2017) and false negatives (Kozycki, 2017).

As an alternative dependent variable we used binary presence or absence of malaria as measured by lab microscopy tests ("lab tests") from the same suite of surveys. In this process blood smears are taken to laboratories to test for the presence or absence of malaria parasites. This process is often complicated under field conditions and can be more expensive than rapid tests (Florey, 2014). Rapid tests were more frequently positive than lab microscopy tests in our data as they were in Florey (2014). Furthermore the two malaria tests were relatively well correlated in our data ($r = 0.58$; Table SI2) as they were in Florey (2014).

As a sensitivity analysis we used the alternative dependent variable of binary self-reported fever ("fever") in the last two weeks. Results from malaria tests and fever recall surveys may differ because there is a lag of up to two weeks between the fever onset and the recall survey, because many cases of fever are caused by something other than malaria (Kiemde, 2017; Mayxay, 2013), especially outside of Sub-Saharan Africa, and because recall may be flawed. There is probably a positive and not-by-chance-alone correlation between reported fever and rapid test results (Okiro & Snow, 2010), but this correlation is noisy and varies by country (eg Mayxay, 2013). Indeed, in our data fever is only weakly correlated with rapid tests ($r = 0.15$) and lab tests ($r = 0.09$). While fever is likely not a reliable proxy for malaria, it is still weakly correlated with malaria prevalence and was surveyed much more broadly ($n \sim 470{,}000$ individuals), so we report results on this dependent variable as well.

Our binary outcome variables of malaria prevalence among children potentially underestimate the influence of deforestation on malaria because they do not include malaria cases that resulted in death. We sought to gain insight into whether this occurred through a sensitivity analysis which used the dependent variable of binary mortality in children under one year of age. This variable did not distinguish the cause of death, but some deaths were likely from malaria, especially in Sub-Saharan Africa. Young children in particular are at high risk of malaria and more than two-thirds of malaria deaths occur in this group (WHO, 2016).

Our candidate pool was all national surveys conducted under the auspices the Demographic and Health Surveys Program of the United States Agency for International Development ($n \sim 400$; DHS, 2017a). These included both Demographic and Health Surveys, which asked about many development indicators, and Malaria Indicator Surveys, which were specific to malaria. These surveys were executed by an implementing agency within a host country, typically a national statistical agency. The countries and years for which surveys were conducted on malaria were not randomly selected. Rather, our sample of surveys was probably biased toward places with higher levels of malaria (since Malaria Indicator Surveys were targeted to malaria-infected countries by design) and times with higher levels of malaria (since "the [Malaria Indicator Surveys are] usually timed to correspond with the high malaria transmission season"; DHS, 2017b).

We restricted the scope of our study to surveys from countries in Latin America/Caribbean, Africa, and South and Southeast Asia. We then limited the study to surveys that gathered observations between 2001 and 2014—the period for which temperature and precipitation data were available. We dropped those 2014 surveys that gathered some observations in 2015 for which we did not have temperature data. We further restricted the scope to rural areas, following the rural/urban binary coding used in the surveys. These scope restrictions resulted in 60,305 respondents from 23 surveys in 17 countries in Sub-Saharan Africa for rapid tests and 56,883 respondents from 22 surveys in 17 countries in Sub-Saharan Africa for lab tests, as well as 469,539 respondents from 90 surveys in 41 countries across Latin America/Caribbean, Africa, and South and Southeast Asia for fever (Table SI1). Of these 90 surveys, 79 were Demographic and Health Surveys and 11 were Malaria Indicator Surveys. We restricted the scope of our analysis to those observations for which data was available for all variables; that is we dropped observations lacking data for one or more variables. In a sensitivity analysis, we further restricted the scope to only those observations for which data on all three dependent variables were collected.

Respondents' locations were geo-located in the original surveys. In order to preserve respondent confidentiality, the Demographic and Health Surveys administrators added random positional errors of 0–5 km to 99% of rural clusters and random positional errors of 0–10 km to 1% of rural clusters, within the country and survey region (DHS, 2017c). Surveys were designed to be representative of the national population or particular subsets including rural populations, after appropriate sample weights are applied; see more below on weighting.

Our independent variables of interest were forest cover and deforestation in the geographic neighborhood of the interview location in the year of the interview; i.e. approximately contemporaneous with the malaria outcome variable. Both variables were obtained from a recent data set that was derived from satellite measurements (Hansen, 2013) and extended through 2015. The data was spatially explicit at 30-meter resolution, applied spatially consistent methods across the entire globe, and applied temporally consistent methods from 2001 to 2014. Forest cover included all vegetation taller than five meters, including natural forests as well as plantations and gardens, and primary as well as secondary forests. Non-forest cover included many categories of land use (e.g. agriculture, pasture, urban areas), each of which may have different malaria risk. Deforestation included both anthropogenic clearing and forest loss from natural causes such as fires. The measure of deforestation was gross rather than net; that is, it did not account for concurrent forest gain. The Hansen (2013) study also produced a measure of forest gain, but that data set was unreliable for our purposes—as cautioned by Tyukavina (2015), forest gain in the Hansen (2013) data set included only those lands that experienced a transition from non-forest to forest between 2001 and 2012; it did not capture regrowing forests that had not yet reached five meters in height by 2012, nor growth within forests that were established before 2000. Because forest cover was collected only for the year 2000, we approximated forest cover in later years by subtracting deforestation since 2000 from forest cover in the year 2000; this is an underestimate of actual forest cover because it does not account for forest gain.

We aggregated forest cover and forest-cover loss to cells that are 0.05 degrees on a side (~5.5 km at the equator) by updating the 2001–2012 data prepared by Busch and Engelmann (2017) through 2014 using the same methods. Both forest measures were fractional: deforestation was measured as the area of forest loss in the year as a fraction of cell area; forest cover was measured as the area of forest cover in the year as a fraction of cell area. While aggregating forest cover and forest-cover loss to the grid-cell scale has the drawback of losing hyper-local information on deforestation associated with 30-meter satellite data, it has several advantages too. The aggregated scale has advantages as an indicator of malaria risk relative to just the immediate point: the maximum flight distance of *Anopheles* mosquitoes is 3–10 km (Kaufmann & Briegel, 2004) while the flight range of a mosquito is typically 2–5 km. We did not test for spatial autocorrelation between nearby observations, but the extent to which this may be occurring due to mosquitoes transmitting malaria between children included in surveys, these effects would be subsumed within the cell. The aggregated scale is also appropriate given the random positional errors of 0–5 km that were deliberately added to survey locations.

Both deforestation and forest cover were negatively correlated with the malaria indicator variables (malaria in rapid tests; malaria in lab tests) and with fever, though weakly so ($-0.10 < r < 0$) (Table SI2). Heat maps of the relationship between forest cover, deforestation, and illness-related dependent variables did not show any clear pattern (Fig. SI1). However, these simple first-order analyses could be biased because they did not control for the influence of other confounding variables that are correlated with deforestation and influence malaria prevalence. Thus we proceeded to multiple regression analysis, as explained below.

We included both deforestation and the combination of forest cover and forest cover squared as independent variables in our model, as we sought to differentiate between two distinct, though related, hypotheses derived from previous literature: that malaria prevalence is higher at higher rates of deforestation (e.g. Vittor, 2009); and that malaria prevalence is higher at intermediate levels of forest cover (e.g. de Castro et al., 2006). In sensitivity analyses we examined models that included only deforestation; only deforestation and forest cover; and only forest cover and forest-cover squared. Four previous studies included both forest cover and deforestation as explanatory variables (Fornace et al., 2016; Hahn et al., 2014; Terrazas et al., 2015; Valle & Clark, 2013), though none also included forest-cover squared.

We sought to control for the influence of other variables known to have a direct effect on the likelihood a subject would have malaria (Fig. 1). Because malaria risk varies with weather, we included temperature and precipitation during the month of the interview. We considered one month to be a reasonable time interval as this corresponds roughly to the 2–4 week life cycle of an *Anopheles* mosquito (CDC, 2015). We included squared terms for both temperature and precipitation, as previous literature suggests malaria risk is highest at intermediate values of these variables (Beck-Johnson, 2013; Mordecai, 2013; Parham & Michael, 2010). Malaria varies based on season, as does the timing of surveys: DHS surveys that collect biomarkers are generally fielded shortly after the rainy season when malaria risk is highest, whereas non-biomarker surveys are fielded in the dry season for logistical reasons (Measure Evaluation, 2013). We did not attempt to code season directly across many countries, though temperature and precipitation variables capture some aspects of seasonality.

Housing quality affects malaria exposure (Tusting, 2015). As a proxy for housing quality we included floor type as a control variable. We constructed a binary code of whether the floor type of the house was unfinished (e.g. clay, mud, or sand) or finished (e.g. brick, cement, or tile). We selected floor type as the proxy for housing quality because all surveys asked about floor type whereas more than 20 surveys did not ask about wall or roof type. As a sensitivity analysis we replaced floor type as a control variable with index of housing quality constructed by summing three binary measures of whether the floor, walls, and roof of the house were unfinished or finished. For example, we coded bamboo, thatch, and wood walls as unfinished and concrete, metal, and stone walls as finished; we coded canvas, palm, and straw roofs as unfinished and asbestos, shingles, and tin roofs as finished. For a list of how we coded all constructed variables see Table 2 of our pre-analysis plan.

Proximity to standing water affects exposure to malarial mosquitoes (Patz et al., 2000). Thus we constructed and included a binary variable for whether the household's water source was open (e.g. well, spring, pond) or piped or delivered (e.g. tap, bottled, tanker truck).

A child's age makes a difference in their exposure and risk of malaria. For example, malaria prevalence was higher in older children in Malawi, attributed to older children's greater independence and time spent outdoors during evening hours (Zgambo, 2017). We included as a control variable the child's age as a binary variable for each year between zero and four to allow for potential non-linear effects. Malaria biomarkers can linger in the bloodstream and malaria test results could potentially reflect exposure to malaria in months or years prior to the survey. Thus, to look only at malaria prevalence during the period of deforestation, we conducted a sensitivity analysis by including only children of age zero.

Households may adapt to increased environmental health risks by engaging in avoidance behavior (e.g. Moretti & Neidell, 2011). In the case of malaria, households may reduce their exposure to malarial mosquitoes by installing insecticide-treated bed nets, which are considered an effective malaria prevention measure (Bhatt, 2015; Lengeler, 2014). We constructed a binary variable for whether the survey respondents stated that "some or all children slept under a bed net last night." Because a considerable number of surveys (13%) did not ask about bed net usage, we included bed net usage only in a sensitivity analysis. Households at higher risk of malaria may be more likely to undertake avoidance behavior by installing bed nets; thus without considering avoidance behavior the coefficient on the effect of forest loss on malaria may be underestimated.

Access to local health services affects malaria. We constructed a binary indicator with the value of 1 if the child was delivered in a health facility (e.g. private, government, or NGO) and 0 if delivered at home or with a traditional birth attendant or midwife. We

Table 2
Results of multiple regression, in which contemporary forest cover, deforestation, and other factors are regressed on indicators of malaria prevalence and fever (primary specification; odds ratios with standard errors in parentheses).

	(1) Rapid	(2) Lab	(3) Fever
Forest cover (share of cell)	1.23	0.24***	1.13
	(0.71)	(0.12)	(0.15)
Forest cover squared	0.72	3.71**	0.85
	(0.44)	(2.00)	(0.12)
Deforestation (share of cell × 100)	0.94 (0.04)	1.01 (0.05)	0.98 (0.02)
Mean monthly air temperature (°C)	1.78***	2.54***	1.03**
	(0.39)	(0.59)	(0.01)
Temperature squared	0.99**	0.98***	1.00
	(0.00)	(0.00)	(0.00)
Total monthly precipitation (cm)	1.01	1.01	1.00
	(0.01)	(0.01)	(0.00)
Precipitation squared	1.00*	1.00*	1.00
	(0.00)	(0.00)	(0.00)
Age 1	1.47***	1.44***	1.25***
Age 2	(0.13)	(0.10)	(0.03)
	1.82***	1.94***	0.96**
Age 3	(0.15)	(0.14)	(0.02)
	2.13***	2.35***	0.76***
Age 4	(0.17)	(0.17)	(0.02)
	2.35***	2.59***	0.66***
	(0.20)	(0.20)	(0.02)
Finished Floor	0.76***	0.84*	0.98
	(0.07)	(0.09)	(0.02)
Pumped or piped water source	0.52***	0.57***	0.97
Constant	(0.05)	(0.07)	(0.02)
	0.00***	0.00***	0.20***
	(0.00)	(0.00)	(0.04)
P-values from joint tests			
Forest cover & forest cover squared	0.759	0.012	0.502
Temperature & temperature squared	0.000	0.000	0.000
Precipitation & precipitation squared	0.004	0.010	0.033
Mean(y)	0.38	0.34	0.26
N	60,305	56,883	469,527
Pseudo-R2	0.14	0.11	0.05

*p < 0.10, **p < 0.05, ***p < 0.01 Logit odds ratios, s.e. clustered at the level of forest cells. All models include survey-wave fixed-effects.

considered this a reasonable proxy indicator for the availability of local health services because birth was universally experienced by children under age five and we assumed that birth in a health facility universally indicated better access to health services. Alternative proxy indicators such as 'child has received other vaccinations' were potentially less useful because recommended vaccines vary by country; some children may be too young to have received vaccines; and there could be a selection effect to vaccination campaigns, meaning that having been vaccinated might indicate either having better access to health services or living in an area with higher health risk. Because not all surveys asked about place of delivery, we included access to health services only in a sensitivity analysis.

We considered including socio-economic variables (e.g. wealth, education) and remoteness as potential control variables. However, while these variables may potentially be correlated with malaria prevalence (e.g. Austin et al., 2017), we decided not to include them as they should influence malaria transmission only through one of the direct channels above rather than directly (see Fig. 1). We considered but did not include variables related to deforestation-prevention (e.g. protected areas and payments for ecosystem services; Bauch, Birkenbach, Pattanayak, & Sills, 2017) for the same reason. There were also several variables hypothesized to directly affect malaria that we would have liked to include but for which data was not available, such as the intensity of anti-malarial initiatives in general, the intensity of indoor spraying (e.g. Over, Bakote'e, Velayudhan, Wilikai, & Graves, 2004) and artemisinin combination therapy (e.g. Okell, Drakeley, Bousema, Whitty, & Ghani, 2008) in particular, and population influx (see Fig. 1). If these explanatory variables were positively correlated with deforestation, e.g. because they were associated with economic growth, then excluding these variables from the model would have biased our coefficient on deforestation toward being less correlated with increased malaria rates.

The countries in our geographic scope varied widely in dominant mosquito vectors, dominant malaria parasite species, forest cover, and potentially malaria test result reliability, among other ecological, social, and institutional differences. We attempted to account for these variations across countries and time periods in two ways. First, in our aggregate-level analysis we included survey wave-specific dummy variables, e.g., for "Liberia 2009" and "Liberia 2011". Second, we conducted 90 independent analyses of individual country-year-specific survey waves. We applied a uniform model specification to each survey wave using universally available and applicable variables suggested by theory and previous literature rather than undertake a detailed variable selection process for each survey wave, e.g. as in Weiss (2015). We did not attempt to spatially disaggregate our data within countries, e.g. by regions defined by dominant mosquito vector. This is a potential avenue for future work.

3. Econometric model

We used spatially explicit cross-sectional logit regressions to estimate the association between deforestation and forest cover on malaria prevalence, controlling for the influence of other variables. That is:

$$\Pr(y_{ict} = 1|\boldsymbol{X}) = e^{\beta \boldsymbol{X}_{ict}}/(1 + e^{\beta \boldsymbol{X}_{ict}})$$

where

$$\beta \boldsymbol{X}_{ict} = \alpha + \beta_1 Forest_{ct} + \beta_2 Forest^2{}_{ct} + \beta_3 Deforestation_{ct} + \boldsymbol{Covariates_{ict}}'\beta_4 + Survey_{it} + \varepsilon_c$$

Here, y_{ict} is the binary health outcome for child i in grid cell c at interview time t. *Forest* is the forest area in the year of time t and *Deforestation* is forest loss between the year of time t-1 and the year of time t. *Covariates* include temperature, temperature squared, precipitation, and precipitation squared in the month of time t; and the floor type, water source, and age of child i. $Survey_{it}$ is a country-year specific dummy variable, used only in the aggregate analysis and not in the survey wave-specific analyses.ε_c is an error term, clustered at the level of the forest grid cell because the exposures (forest cover and forest-cover change) were common to all children within a grid cell. In a sensitivity analysis we clustered standard errors at the level of the DHS primary sampling unit instead.

We weighted the observations in our sample to be representative of the rural population across the set of countries and years for which surveys were conducted. In two sensitivity analyses, we weighted each observation equally and weighted each survey equally.

In a sensitivity analyses, we applied a cross-sectional OLS regression in place of a cross-sectional logit regression. Because our data contained some locations with repeat surveys in the same communities, we were able to conduct a sensitivity analysis using a panel regression with grid-cell level fixed effects and survey-specific fixed effects in an OLS model. The panel regression is theoretically preferable to the pooled cross-sectional regression because it can control for both observable and unobservable differences in malaria risk across sites. However, it comes at the cost of a smaller sample size. Only 11 of 23 surveys with rapid tests collected at least some repeat data from the same grid cells; nine of 22 surveys with lab tests collected at least some repeat data; and 78 of 90 surveys of fever collected at least some repeat data. Changing from a cross-sectional regression to a panel regression reduced the number of available observations by 86% for rapid tests; 90% for lab tests; and 79% for fever.

We also tested secondary ex ante hypotheses related to several other types of disaggregation, for example: that the effect of deforestation on malaria is greater in Latin America and Africa than Asia (Guerra et al., 2006); greater for smaller than larger cut sizes (Singer & de Castro, 2006); greater at higher levels of initial forest cover (de Castro et al., 2006); and diminishing after about seven years (Singer & de Castro, 2006). We tested for potential lagged effects by adding as explanatory variables deforestation 1–3 years prior; 4–6 years prior; and 7–9 years prior, to a regression including survey waves from 2010 onward.

We sought to enhance the credibility of our analysis in two ways. First, prior to conducting any analyses, we pre-selected model specification and included variables based on explicit ex ante hypotheses grounded in previous literature and our understanding of the coupled human-natural system rather than based on an exploration of our data. Second, we wrote and adhered to a pre-analysis plan in which all analyses to be undertaken in the study were specified in advance. Pre-analyses plans are common and even required in some clinical research, but are uncommon and new in social science research including economics (see Miguel, 2014; Coffman & Niederle, 2015; Olken, 2015). Pre-analysis plans impose methodological discipline and prevent mid-stream revisions to methods and variables, and can help avoid the perception of inadvertently or deliberately placing a thumb on the scale to achieve desired results. On the downside, full specification in advance is "close to impossible," while a pre-specified analysis "may miss the nuance that categorizes social science research" (Olken, 2015).

In a first-stage pre-analysis plan we constructed and tested the software code on a subset of just two surveys: Liberia 2009 and Liberia 2011. These two surveys were chosen at random from the set of countries that had data for both malaria and fever; had more than one year of data; and were in Africa. Then we produced a second-stage pre-analysis plan for the full sample. We made changes to the original pre-analysis plan only to ensure that our

code was correct, not based on the results of the small sample. Our pre-analysis plan describes in detail methods related to both those analyses that we undertook (e.g., correlations; heatmaps; cross-sectional regressions; panel regressions) as well as analyses that we had originally planned but were ultimately unable to undertake (e.g., mediation analyses; cost-effectiveness analysis).

We interpreted our results through the frame of whether or not they were consistent with the ex ante hypotheses registered in the pre-analysis plan. That is, results that were statistically significant and in the expected direction were considered to be consistent with ex ante hypotheses. Results that were not statistically significant, or were statistically significant but in the opposite direction as expected, were considered not to be consistent with our ex ante hypothesis.

During the course of review, we added four additional analyses that were not included in our initial pre-analysis plan. These analyses are exploratory and can potentially generate hypotheses to be tested in future research. The results of these analyses do not represent tests of pre-specified ex ante hypotheses. First, we explored potential lag by running three regressions in which lags of 1–3 years; 4–6 years; and 7–9 years were included individually. Second, excluded sites with zero forest cover in the year of the survey. Third, we allowed for flexibility in the relationship between forest cover and indicators of malaria and fever by applying a non-parametric specification with binary variables for each forest cover decile. Fourth, we tested for multicollinearity between forest cover and deforestation.

4. Results

The results of our primary specification were not consistent with our ex ante hypotheses that malaria prevalence is higher at greater levels of deforestation, controlling for other factors (Table 2). That is, in a pooled, weighted cross-sectional multiple logit regression, the odds-ratio for the effect of deforestation on malaria was not significantly greater than one. This was the case for the both dependent variables related to malaria (rapid test results and lab test results), as well as for fever.

The results of our primary specification were also not consistent with our ex ante hypothesis that malaria prevalence is higher at intermediate levels of forest cover, controlling for other factors (Table 2). That is, in a pooled, weighted cross-sectional multiple logit regression, there was not an odds-ratio above one for forest cover, an odds-ratio below one for forest-cover squared, and joint significance. In the case of malaria rapid test results and fever, these odds ratios were in the expected direction and not jointly significant. In the case of malaria lab test results, the odds ratios were jointly significant but in the opposite direction than expected.

The relationships between covariates and malaria were mostly as expected. Malaria prevalence was significantly higher at intermediate temperature, lower with a finished floor, and higher with an open water source. Malaria prevalence was also higher in older children. The signs of the coefficients on precipitation and precipitation squared were consistent with malaria prevalence being higher at intermediate precipitation, but the coefficients were not jointly significant.

When disaggregated by survey waves, results were consistent with our ex ante hypothesis in only 3/44 cases for malaria rapid tests (Fig. 2; Table 3; Table SI4); 3/40 cases for malaria lab tests (Fig. 3; Table 3; Table SI4); and 29/176 cases for fever (Fig. SI2; Table 3; Table SI4). These results are similar to the results of placebo hypotheses that deforestation and intermediate forest cover were associated with lower malaria, which was the case for 6/44 for malaria rapid tests; 3/40 for malaria lab tests; and 15/176 for fever.

The results of multiple pre-specified sensitivity analyses were also not consistent with our ex ante hypotheses related to forest cover and deforestation. These sensitivity analyses included alter-

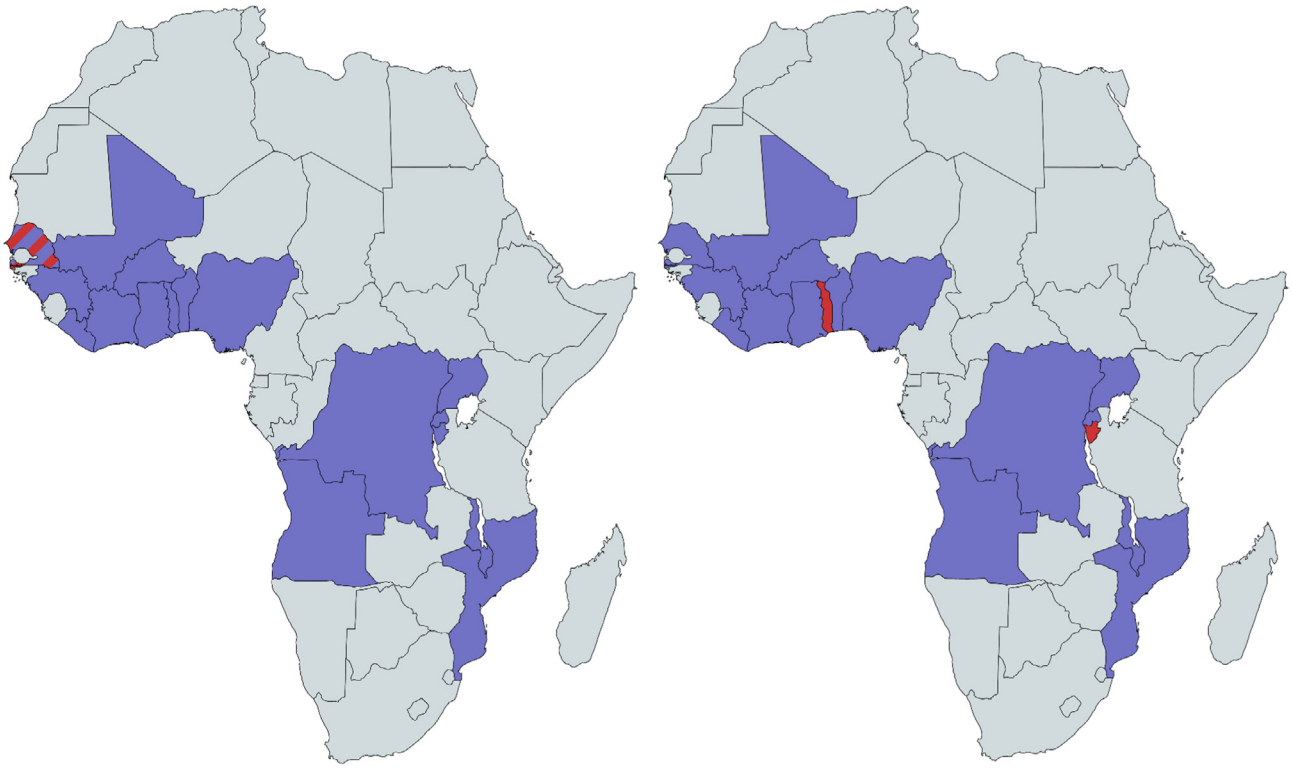

Fig. 2. Country-by-country effect on malaria as measured by rapid tests of a) greater deforestation and b) intermediate forest cover. Red: positive and significant effect in all survey waves; blue: positive and significant effect in no survey waves; red and blue stripes: positive and significant effect in some but not all survey waves. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Table 3
Survey wave-by-survey wave results. "+/sig," "no sig.," and "−/sig" denote that the coefficient on the variable is positive and significant, not significant, or negative and significant, respectively. "joint +/sig," "joint no sig.," and "joint −/sig" denote that coefficients on the variable and its squared terms are jointly positive and significant, not significant, or negative and significant, respectively.

		Rapid	Lab	Fever
Intermediate forest cover	joint +/sig.	2	2	17
	joint no sig.	18	17	66
	joint −/sig.	2	1	5
Deforestation	+/sig.	1	1	12
	no sig.	17	17	66
	−/sig.	4	2	10
Intermediate temperature	joint +/sig.	10	8	21
	joint no sig.	12	12	55
	joint −/sig.	1	2	14
Intermediate precipitation	joint +/sig.	3	3	10
	joint no sig.	15	13	66
	joint −/sig.	5	6	14
Age = 1	+/sig.	10	7	50
	no sig.	13	15	40
	−/sig.	0	0	0
Age = 2	+/sig.	17	16	22
	no sig.	6	6	54
	−/sig.	0	0	14
Age = 3	+/sig.	21	17	7
	no sig.	2	5	40
	−/sig.	0	0	43
Age = 4	+/sig.	21	18	1
	no sig.	2	4	32
	−/sig.	0	0	57
Finished floor	+/sig.	0	0	5
	no sig.	12	9	74
	−/sig.	11	13	11
Pumped or piped water source	+/sig.	0	0	2
	no sig.	13	10	81
	−/sig.	9	11	6

native weightings (Tables SI5-6); use of OLS (Table SI7); reduction to the subset of observations for which data on all three dependent variables were collected (Table SI8); clustering standard errors at the level of the DHS primary sampling unit (Table SI9); the inclusion of a bed-net variable and place of delivery variable (Tables SI10–SI11); and the inclusion of the housing quality index in place of floor type (Table SI12). In these analyses bed net usage and delivery in a facility were both associated with significantly less malaria, and higher housing quality was associated with significantly less malaria and fever, as expected.

Nor was the hypothesized relationships with deforestation and forest cover borne out after limiting the sample to children of age zero only (Table SI13). That these hypotheses were also not borne out when an alternative dependent variable of mortality in age-zero children (Table SI14) was applied suggests that our estimates of the effects of deforestation on malaria were not biased by our measures of disease burden having excluded mortality.

Alternative combinations of independent variables of interest also did not produce results consistent with our ex ante hypotheses. These included removing deforestation as an independent variable (Table SI15), removing forest-cover squared as a dependent variable (Table SI16), and removing both forest cover and forest-cover squared as dependent variables (Table SI17).

Our ex-ante hypotheses related to disaggregations were also not borne out. We did not find that the effect of deforestation on fever was significantly greater in Latin America and Africa than in Asia (Table SI18). We did not find that the effect of deforestation on malaria or fever was significantly higher at higher levels of initial forest cover (Table SI19). Nor did we find that the effect of deforestation on fever was disproportionately greater for smaller amounts of deforestation (Table SI20). In no lagged period prior to the survey year were our ex-ante hypotheses related to forest cover and deforestation borne out (Table SI21).

In our panel analysis we did find, in accordance with our ex ante hypothesis, that malaria as measured in lab tests was significantly higher at intermediate levels of forest cover (Table SI22). However, neither malaria as measured in rapid tests nor fever was significantly higher at intermediate levels of forest cover. None of the three dependent variables was significantly higher at higher levels of deforestation.

In addition to the results of tests of ex ante hypotheses above, we also undertook several exploratory analyses that could potentially generate ex ante hypotheses to be tested in future research. An exploration of lags in which periods prior to the survey year were included individually did not showed expected relationships between deforestation and malaria and fever indicators, nor between forest cover and malaria and fever indicators (Tables SI23–SI25). After excluding observations for which forest cover was zero in the year of the survey, one of the six combinations of dependent variables (malaria rapid tests; malaria lab tests; fever) and explanatory variables (deforestation; forest cover) showed the expected relationship: an inverted-U-shaped relationship between forest cover and fever (Table SI26). A non-parametric regression uncovered no clear pattern between forest cover decile and rapid test, lab test, or fever (Table SI27). Forest cover and deforestation were not collinear, as evidenced by a variance inflation factor (VIF) of 1.11 on each variable.

5. Discussion

The results of our geographically aggregated analysis were not consistent with either of our primary ex ante hypotheses—that malaria prevalence would be higher at intermediate levels of forest cover, and that malaria prevalence would be higher at higher levels of deforestation. In five out of six combinations of dependent variables (malaria rapid tests; malaria lab tests; fever) and explanatory variables (deforestation; forest cover), this was because the coefficients were not significant. In one of the six combinations (malaria lab tests and forest cover), this was because the coefficients were significant but in the opposite direction as the ex ante hypothesis.

Results from only a small number (n = 6/84) of individual survey wave-specific analyses were consistent with our primary ex ante hypotheses. Survey waves in which results were consistent with our primary ex ante hypotheses showed no clear pattern with respect to dominant malaria vector (Table SI4) nor geographic region (Fig. 2, Fig. 3, Fig. SI2). Because we did not test any ex ante hypothesis with respect to differences across individual countries, we consider the distribution of countries in which results were consistent with our ex ante hypothesis to be exploratory. It should be noted that even in the absence of any general or local relationship between deforestation and malaria, or between forest cover and malaria, it would still be possible for some survey wave-specific analyses to produce some positive and significant findings by chance alone.

Taken together, our aggregate and survey wave-specific results imply the absence of a geographically generalizable relationship between deforestation and malaria, and between forest cover and malaria, across the countries studied. This absence is not because the analysis would not be able to detect a relationship if one were present; the expected effects of temperature, precipitation, housing quality, and water source on malaria were found in both the aggregate analysis (Table 2) and in many survey wave-specific analyses (Table 3). Because we conducted our analyses at the

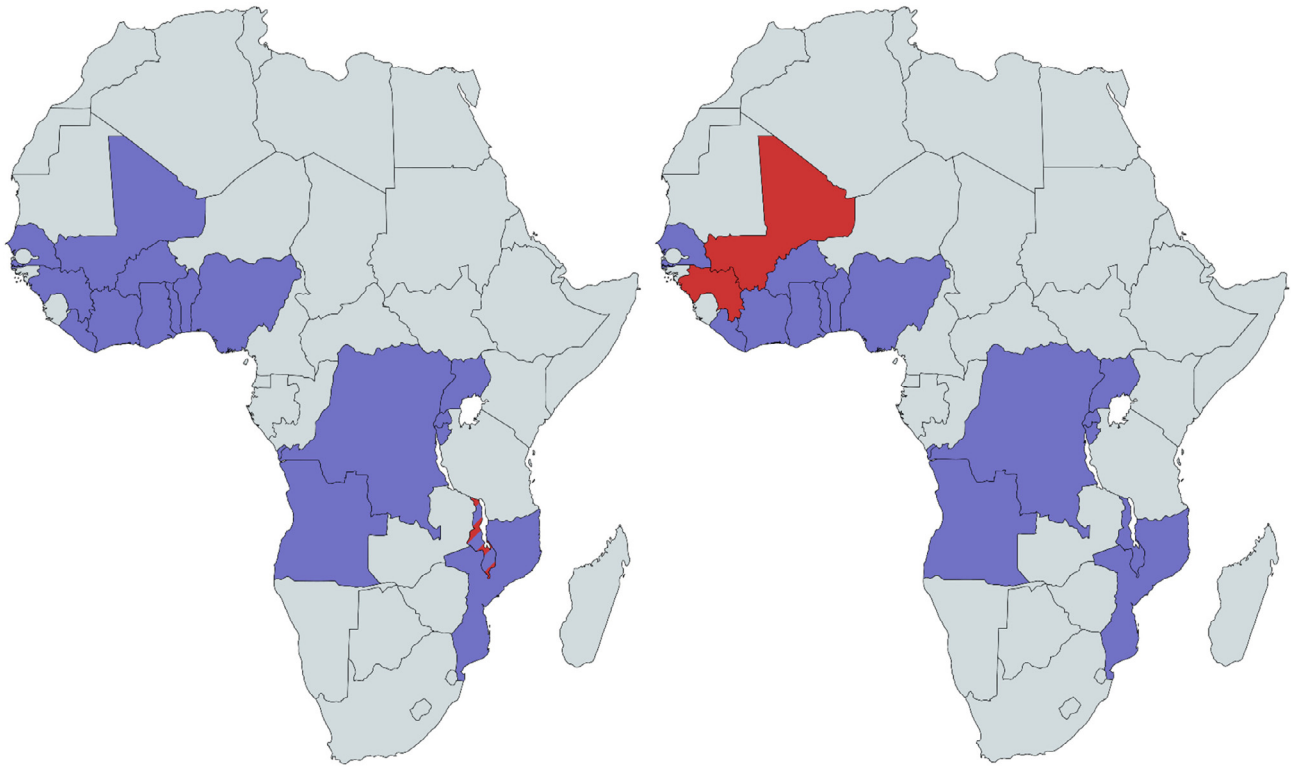

Fig. 3. Country-by-country effect on malaria as measured by lab tests of a) greater deforestation and b) intermediate forest cover. Red: positive and significant effect in all survey waves; blue: positive and significant effect in no survey waves; red and blue stripes: positive and significant effect in some but not all survey waves. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

international and national scales, we are unable to rule out associations at the local scale, nor beyond the geographic scope of our study region.

Our findings come with caveats related to the geographic distribution and selection of interviews. Our malaria analysis was limited to 17 African countries, which together comprised 51% of the population and 65% of the deforestation of Sub-Saharan Africa circa 2010. Our fever analysis was geographically broader, sampling from 41 tropical countries, including 30 African countries which together comprised 93% of the population and 87% of the deforestation of Sub-Saharan Africa circa 2010, but still was not representative of the entire Tropics. As discussed above, fever is not a reliable proxy for malaria. Furthermore, the countries and years for which surveys were conducted on malaria were not randomly selected. Rather, our sample of surveys was probably biased toward both places and times with higher levels of malaria. The limited geographic scope of surveyed countries and years means that our results are difficult to extrapolate beyond the set of countries included in the sample. Even within the countries and years of analysis, our pooled sample represented an artificial super-national aggregation of countries and years rather than a cohesive and intuitive bloc. It is important to note however that survey observations were designed to be, once properly weighted, representative of the countries and years in which they took place. Since the results of so few survey-specific analyses were consistent with our ex ante hypothesis, and results were robust to alternative weighting schemes, we are confident that our main findings are not an artifact of the artificial super-national aggregate of countries nor the weighting scheme employed.

How do we reconcile our findings from Sub-Saharan African countries with previous studies that found a positive relationship between deforestation and malaria prevalence in the Brazilian Amazon, Malaysia, and Paraguay? We do not think that the difference in findings across studies can be explained by variation in malaria-transmitting mosquito types across continents. While the effect of deforestation and forest cover on mosquito density and malaria transmission (e.g. Yasuoka & Levins, 2007; Kar et al., 2014; Burkett-Cadena & Vittor, 2017) certainly depend on mosquito vector ecology, the malarial mosquito species that are most prevalent across Sub-Saharan Africa (e.g. *A. gambiae; A. funestus; A. arabiensis;* Kiszewski, 2004) do favor deforested areas (Burkett-Cadena & Vittor, 2017; Kar et al., 2014; Yasuoka & Levins, 2007). And literature suggests that it is Asia rather than Africa where deforestation has a lower effect on malaria risk (Guerra et al., 2006).

We speculate that at least some of the difference in findings across studies relates to differences in the phenomenon of deforestation between Africa and other tropical continents, and thus the channels through which deforestation leads to changes in malaria. Fisher (2010) describes an "African exception to drivers of deforestation," in which deforestation in Africa is largely driven by the slow expansion of subsistence or smallholder agriculture for domestic use rather than market-driven agricultural exports as in Latin America and Asia. It could be that in Latin America and Asia relatively more deforestation is undertaken by new frontier migrants with associated unstable socio-economic conditions of poor housing stock, unimproved water sources, poor access to health services, previously low exposure to malaria, and less familiarity with malaria-avoidance practices. Meanwhile in Africa relatively more deforestation is by long-time residents of malaria-endemic communities with higher previous exposure for whom these conditions are less likely to be changed by deforestation. These differences across regions suggest hypotheses to test in future research.

Differences in data sets and methodology could potentially also have contributed to the difference in findings across studies.

Applying our methodology to Brazil, Malaysia, or Paraguay would have let us hold methods constant across regions. However, we were not able to do so because Demographic and Health Surveys did not collect data on malaria in these countries.

Our findings can help prioritize interventions in both the health sector and the forest sector. For anti-malarial efforts, our findings do not support the inclusion of forest conservation within a portfolio of anti-malarial interventions, at least in Sub-Saharan Africa where 88% of malaria cases occur (WHO, 2016). It would be more effective to prioritize proven anti-malarial interventions such as bed nets, spraying, and housing improvements. For forest conservation efforts in Africa, it makes sense to focus management interventions on securing the many other values of standing forests, including carbon storage, biodiversity habitat, clean water provision, food provision, and other aspects of human health (Seymour & Busch, 2016). The total social value of standing forests, and the case for conserving them, is considerable even without an effect on malaria.

6. Availability of data and code

Data on malaria, fever, and individual co-variates are available upon request from the United States Agency for International Development Demographic and Health Surveys Program (https://dhsprogram.com/). Data on air temperature and precipitation are available for download from the University of Delaware Department of Geography (http://climate.geog.udel.edu/~climate/html_pages/download.html). Data on forest cover and deforestation are available for download from the University of Maryland Department of Geographical Sciences (https://earthenginepartners.appspot.com/science-2013-global-forest/download_v1.5.html). Code used for analysis is available from the authors upon request.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.worlddev.2019.104734.

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