

Feedbacks between human behavior, disease transmission, and vector ecology generate culture-linked dynamics in vector-borne diseases

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Abstract

Vector-borne diseases cause millions of deaths worldwide and are expanding into new regions and populations as climate change intensifies. Human behavior, shaped by social and cultural factors, is central to disease control, yet feedbacks among behavior, disease transmission, and vector ecology remain poorly understood. To address this gap, we develop a parsimonious malaria model coupling infection dynamics in human and mosquito populations with evolving protective behavior, perceived intervention effectiveness, and competition between insecticide-resistant and non-resistant mosquitoes. We show that the strength of social norms fundamentally reshapes disease dynamics. Weak norms can generate endemic waves of infection and protective behavior, whereas moderate norms can enable disease elimination but also produce culture-dependent tipping dynamics, including abrupt reversion to highly endemic states following ecological or economic change. Strong norms can lock in either beneficial or harmful behavioral states, sustaining high adoption when protective behavior is already established or promoted but otherwise stabilizing poor epidemiological outcomes. These complex socio-biological feedbacks can lead to counterintuitive results: the model suggests that under weak norms, untreated nets can yield lower infection prevalence than insecticide-treated nets. In addition, we identify dynamically changing behavior and perceived effectiveness as a standalone mechanism capable of generating the coexistence of insecticide-resistant and non-resistant mosquito populations. We complement the model with an exploratory analysis of the adoption of mosquito nets, malaria prevalence, and the changes in mosquito resistance in Liberia to examine whether some of the qualitative patterns suggested by the model are also visible in the available data. While the empirical analysis is not causal, the estimated associations are largely consistent with the theoretical predictions. Overall, our framework bridges game-theoretic and epidemiological approaches, providing a unified perspective for understanding vector-borne diseases as coupled human–biological systems and designing behaviorally informed interventions.

1 Introduction

Vector-borne diseases are caused by parasites, bacteria, or viruses transmitted to humans through the bites of infected arthropods, such as mosquitoes, ticks, lice, and flies (Obradovic *et al.*, 2022). They have contributed to gene-culture coevolution in humans (Livingstone, 1958; Wiesenfeld, 1967; Richerson *et al.*, 2010), shaped major demographic, economic, and cultural processes throughout human history (Athni *et al.*, 2021), and remain an increasingly urgent public health challenge, especially in the context of accelerating climate change (De Souza and Weaver, 2024). Today, more than 6 billion people live in areas at risk of vector-borne diseases, which cause more than 300 million disease cases and roughly 1 million deaths annually (Obradovic *et al.*, 2022; De Souza and Weaver, 2024). Climate change is expected to alter the geographic and seasonal suitability of many vector-borne infections, potentially expanding transmission risk into regions where these diseases are currently rare or absent (Githeko *et al.*, 2000; Leighton *et al.*, 2012; Caminade *et al.*, 2014; Koch *et al.*, 2017; Messina *et al.*, 2019). These trends underscore the need for a deeper understanding of vector-borne disease dynamics, improved predictive frameworks, and more nuanced intervention strategies and public health policies.

The challenge in mitigating vector-borne diseases stems from the coupled human-biological systems in which epidemic dynamics emerge from interactions among pathogen biology, vector ecology, environmental conditions, and human behavior. Human behavior shapes vector-human contact, vector mortality, and disease transmission through the use of protective measures (Asin-gizwe *et al.*, 2019; Phok *et al.*, 2022), mobility (Stoddard *et al.*, 2013; Reiner Jr *et al.*, 2014), and changing patterns of exposure (Berry *et al.*, 2018a). Crucially, protective actions change over time in response to perceived risk (Lopes-Rafegas *et al.*, 2023; Casella *et al.*, 2024), the costs and benefits of defensive behaviors (Agbo *et al.*, 2026), social norms (Perkins *et al.*, 2019; Olapeju *et al.*, 2022; Lopes-Rafegas *et al.*, 2023; Agbo *et al.*, 2026), information spread, authority messaging (Agbo *et al.*, 2026), and trust in public health institutions (Agbo *et al.*, 2026). These behavioral drivers are themselves shaped by disease prevalence, effectiveness of interventions, and changes in vector populations, creating feedbacks between socio-cultural context, pathogen transmission, and vector ecology. How do these feedbacks shape epidemic dynamics, and how can this knowledge inform interventions that reduce disease burden?

Mathematical models are widely used to disentangle the complexity of vector-borne disease systems (Mandal *et al.*, 2011; Smith *et al.*, 2012; Reiner Jr *et al.*, 2013; Aguiar *et al.*, 2022; Ogunlade *et al.*, 2023; Anwar *et al.*, 2024), and many incorporate human behavior. In most, however, behavior enters as an externally specified level of protection, intervention coverage, or compliance (see, e.g., Agosto *et al.*, 2013; Buonomo, 2015; Wairimu *et al.*, 2018; Ngonghala *et al.*, 2020; Nwankwo, 2021), rather than as an evolving decision-making process. The smaller class of models that treats behavior dynamically typically assumes that protective decisions respond to perceived infection risk and the associated costs and benefits of protection (Dumont and Thuilliez, 2016; Broom *et al.*, 2016; Roosa and Fefferman, 2022; Ngonghala *et al.*, 2022; Dayap and Rabajante, 2025; De Reggi *et al.*, 2025; Rahman *et al.*, 2026; Duve *et al.*, 2026; Shetgaonkar and Sharma, 2026). Cognitive, social, and normative determinants beyond perceived risk - including beliefs about intervention effectiveness, social influence, and conformity to local norms - have received less attention in mechanistic epidemiological models (for an exception, see Poterek *et al.*, 2025). Although some economic models of vector-borne diseases represent such behavioral determinants more explicitly, they are often not coupled with models of disease transmission and dynamics, and, thus, place less emphasis on pathogen-transmission dynamics and on feedbacks linking behavior, disease prevalence, and vector ecology (see, e.g., Brown and Kramer, 2018), or focus primarily on policymakers' decisions rather than individual decision-making (Brown *et al.*, 2013). This leaves open a broader question: how

do socially mediated behavioral feedbacks interact with ecological feedbacks, and can these interactions qualitatively reshape epidemic outcomes when protective measures alter selection pressures on vectors?

Malaria provides a particularly important setting for studying this question. Despite major progress in control (Cotter *et al.*, 2013), it remains one of the most consequential vector-borne diseases globally (Zhang *et al.*, 2024; Weiss *et al.*, 2025), and its prevalence depends heavily on the use of protective measures such as insecticide-treated nets, indoor residual spraying, and repellents (Wangdi *et al.*, 2018). The uptake and sustained use of these measures may depend not only on individual risk perception and material costs (Atkinson *et al.*, 2009; Pulford *et al.*, 2011), but also on religious and cultural beliefs (Suuron *et al.*, 2020), perceived effectiveness (Atkinson *et al.*, 2009; Suuron *et al.*, 2020; Babalola *et al.*, 2022), trust (De Hoop and van Kempen, 2010), authority messaging (Suuron *et al.*, 2020), and conformity to local norms. Studies from Cambodia (Phok *et al.*, 2022), rural Rwanda (Asingizwe *et al.*, 2019), Cameroon, and Côte d’Ivoire (Babalola *et al.*, 2022) suggest that social norms play an especially important role in the use of protective measures against malaria. At the same time, widespread use of insecticide-based measures can alter mosquito mortality and select for insecticide resistance, potentially undermining their long-term effectiveness (Ranson and Lissenden, 2016; Hemingway *et al.*, 2016; Alout *et al.*, 2017). Malaria therefore provides a natural setting for examining interactions among behavioral feedbacks, social norms, disease transmission, and vector ecology, yet existing models rarely integrate these components within a single dynamical framework.

Here, we address this gap by developing a mechanistic malaria model in which protective behavior and perceived intervention effectiveness evolve endogenously, according to a norm-utility approach (Gavrilets *et al.*, 2024). In this framework, individual decisions depend not only on material costs and perceived infection risk, but also on social norms and beliefs about the effectiveness of protective measures. Building on recent behavioral epidemiology models of COVID-19 (Yang *et al.*, 2022; Morsky *et al.*, 2023; Morsky, 2025; Wang *et al.*, 2026), we couple this behavioral framework with malaria transmission and mosquito population dynamics to examine feedbacks among social and cultural processes, disease transmission, and vector ecology within a unified dynamical system.

2 The model

Brief outline. Here, we develop a parsimonious model of malaria transmission that couples infection dynamics in human and mosquito populations with the use of protective measures, evolving perceived effectiveness of those measures, and the spread of insecticide resistance in mosquitoes. In our model, individuals decide whether to use protective measures, such as insecticide-treated nets or indoor spraying, based on perceived infection risk, adoption costs, and social norms. This creates a feedback loop in which human behavior and beliefs alter vector ecology, including competition between resistant and non-resistant mosquitoes, while vector and disease dynamics feed back into perceived risk and subsequent behavior. Our goal is to understand how dynamically changing human behavior shapes malaria transmission and the spread of insecticide resistance across different social and cultural contexts.

Disease dynamics in the human population. We model human infection dynamics using an SIS framework, $S_h \rightarrow I_h \rightarrow S_h$ (Torres-Sorando and Rodriguez, 1997; Mandal *et al.*, 2011; Agosto *et al.*, 2013; Buonomo, 2015; Wairimu *et al.*, 2018; Sequeira *et al.*, 2020; Corder *et al.*, 2020). For

analytical tractability, we neglect demographic processes and assume $S_h + I_h = N_h = \text{const}$ ¹:

$$\dot{S}_h = -\beta_h(p)S_h(I + I') + \gamma I_h, \quad (1)$$

$$\dot{I}_h = \beta_h(p)S_h(I + I') - \gamma I_h, \quad (2)$$

where $\beta_h(p)$ denotes the transmission rate from mosquitoes to humans, $I + I'$ is the total number of infected mosquitoes, and γ is the recovery rate. We assume that the transmission rate $\beta_h(p)$ depends on the mosquito biting rate m and the probability b_h that an infectious bite results in human infection (Mandal *et al.*, 2011; Agbata *et al.*, 2025). Use of protective measures, such as insecticide-treated nets (ITNs), reduces exposure to mosquito bites (Agbata *et al.*, 2025). We therefore define $\beta_h(p) = \frac{(1-\xi p)mb_h}{N_h}$, where $p \in [0, 1]$ is the fraction of individuals adopting protective measures and $\xi \in [0, 1]$ measures the effectiveness of protection in reducing exposure.

Disease dynamics in the mosquito population. We consider two mosquito populations: insecticide-resistant and non-resistant denoted by primed and unprimed variables, respectively (Nwankwo, 2021). Mosquito infection follows a susceptible-infected (SI) framework, since infected mosquitoes remain infectious throughout their lifespan (Mandal *et al.*, 2011; Agosto *et al.*, 2013; Buonomo, 2015; Wairimu *et al.*, 2018; Agbata *et al.*, 2025; Rajnarayanan *et al.*, 2025):

$$\dot{S} = r(S + I)\left(1 - \frac{N_v}{K}\right) - \beta_v(p)SI_h - d(p)S, \quad (3)$$

$$\dot{I} = \beta_v(p)SI_h - d(p)I, \quad (4)$$

and

$$\dot{S}' = r'(S' + I')\left(1 - \frac{N_v}{K}\right) - \beta_v(p)S'I_h - d'(p)S', \quad (5)$$

$$\dot{I}' = \beta_v(p)S'I_h - d'(p)I', \quad (6)$$

where r and r' are intrinsic growth rates, K is the baseline environmental carrying capacity associated with finite breeding habitat and resource limitation, $d(p)$ and $d'(p)$ are mortality rates, $\beta_v(p) = \frac{(1-\xi p)mb_v}{N_h}$ is the transmission rate from humans to mosquitoes, and $N_v = S + I + S' + I'$ is the total mosquito population size. We posit that both mosquito types are initially present, and that selection operates on their relative abundances over time.

Mosquito mortality includes natural mortality d_0 and additional mortality induced by protective measures (Agosto *et al.*, 2013; Buonomo, 2015; Wairimu *et al.*, 2018; Ngonghala *et al.*, 2020; Nwankwo, 2021): $d(p) = d_0 + d_1p$ for non-resistant mosquitoes and $d'(p) = d_0 + d'_1p$ for resistant mosquitoes. Here, d_1 and d'_1 are the maximal intervention-induced mortality rates under full adoption. This assumes average exposure to interventions proportional to population-level adoption p , consistent with a well-mixed approximation and community-wide effects of protection (Lindsay *et al.*, 1991; Pleass *et al.*, 1993; Service, 1997; Binka *et al.*, 1998; Killeen *et al.*, 2003; Hawley *et al.*, 2003; Zhou *et al.*, 2010; Brown and Kramer, 2018). We also postulate that resistance reduces intervention-induced mortality, so that $d'_1 < d_1$ (Nwankwo, 2021)².

We posit that resistance carries fitness costs (Alout *et al.*, 2017), represented by a lower intrinsic growth rate for resistant mosquitoes than for non-resistant mosquitoes, $r' < r$ ³. This assumption is supported by empirical evidence that pyrethroid-resistant malaria vectors, including *Anopheles*

¹The average global mortality from *P. falciparum* is about 0.28 per cent: Source: [https://www.thelancet.com/journals/lancet/article/PIIS0140-6736\(25\)00038-8/fulltext](https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(25)00038-8/fulltext).

²In Section 3.3 we also compare insecticide-treated nets with untreated nets for which $d'_1 = d_1 = 0$.

³We assume that $d_0 + d_1 < r$ and $d_0 + d'_1 < r'$.

gambiae and *An. funestus*, can exhibit reduced fecundity and lower net reproductive rates than their non-resistant counterparts (Tchouakui *et al.*, 2021; Osoro *et al.*, 2022).

Dynamics of human behavior. We assume that individuals decide whether to use protective measures ($q = 1$) or not ($q = 0$) based on a utility function that incorporates perceived infection risk, the costs of adoption, and social norms (Morsky *et al.*, 2023):

$$u(I_h, p, q) = \underbrace{-R(I_h, q)}_{\text{perceived risk}} - \underbrace{cq}_{\text{adoption costs}} - \underbrace{\theta(q - p)^2}_{\text{social norms}}, \quad (7)$$

where c denotes the cost of adoption, which may include material costs, discomfort associated with using protective measures, or perceived health risks from insecticide exposure. The parameter θ measures the strength of descriptive social norms, that is, individuals' tendency to conform to others' behavior (Cialdini *et al.*, 1990).

The true infection risk depends on the prevalence of infected mosquitoes, which is generally unobservable to individuals. We therefore assume that individuals use human infection prevalence, I_h/N_h , obtained through public information or social observation, as a proxy for disease risk (Broom *et al.*, 2016; Gutiérrez-Jara *et al.*, 2022; De Reggi *et al.*, 2025)⁴:

$$R(I_h, q) = (1 - \psi(t)q) \cdot \frac{I_h}{N_h}, \quad (8)$$

where $\psi(t) \in [0, 1]$ is the perceived effectiveness of protective measures. For tractability, we assume homogeneous perceived effectiveness: all individuals evaluate protective measures using the same belief $\psi(t)$, although they may differ in their adoption decisions $q \in \{0, 1\}$. Below, we consider several specifications for $\psi(t)$, ranging from a static form to a dynamic formulation in which this common belief about effectiveness evolves over time.

The fraction of adopters, p , is modeled using a Granovetter-Schelling threshold framework (Schelling, 1969, 1971; Granovetter, 1978; Granovetter and Soong, 1983; Brock and Durlauf, 2001; Morsky *et al.*, 2023):

$$\dot{p} = \varepsilon(BR(I_h, p) - p), \quad (9)$$

where $\varepsilon > 0$ is the rate of behavioral updating. The best-response function is given by $BR(I_h, p) = \frac{1}{1 + e^{k(u(I_h, p, 0) - u(I_h, p, 1))}}$, which represents a smooth threshold rule equivalent to a discrete-choice model with logistic errors. The parameter $k \gg 0$ controls the steepness of the decision rule.

3 Results

3.1 Epidemic dynamics with fixed behavior and no resistance

We begin with a baseline version of the model in which adoption p is fixed and insecticide resistance is absent. This setting isolates the epidemiological consequences of a static level of protection. Our analysis (Section S2 in the SI) reveals a threshold structure governed by the balance between transmission and removal. Specifically, if transmission is sufficiently weak even in the absence of adoption, so that

$$\beta_h(0)\beta_v(0)N_hN^*(0) < \gamma d(0),$$

⁴In the SI, we also consider alternative formulations in which risk depends on the mosquito population size (Ngonghala *et al.*, 2022; Roosa and Fefferman, 2022; Oraby *et al.*, 2025) or is determined by the incidence of human infection.

then the disease-free equilibrium is globally stable for all $p \in [0, 1]$. Here, $N^*(p) = K(1 - \frac{d(p)}{r})$ is the equilibrium size of the mosquito population at adoption level p . Conversely, if transmission remains sufficiently strong even under full adoption,

$$\beta_h(1)\beta_v(1)N_hN^*(1) > \gamma d(1),$$

then the system converges to an endemic equilibrium for all $p \in [0, 1]$. In the intermediate regime, the long-term outcome depends on adoption: there exists a critical threshold $p^\# \in (0, 1)$ such that the disease-free equilibrium is stable if $p > p^\#$, whereas the endemic equilibrium persists if $p < p^\#$.

3.2 Joint dynamics of disease and human behavior with no resistance

We next allow human behavior to evolve in response to disease prevalence, while excluding insecticide resistance and holding the perceived effectiveness of protective measures ψ fixed. This setting allows us to examine how epidemic outcomes depend on the strength of social norms θ and ψ .

Across values of ψ , absent or very weak norms lead to an endemic equilibrium characterized by low-to-moderate adoption p and moderate-to-high disease prevalence I_h/N_h (Figure 1a-b). In contrast, strong norms produce bistability between an endemic non-adoption equilibrium and a disease-free full-adoption equilibrium (Figure 1a). Outcomes then depend strongly on initial adoption: below a critical threshold, social inertia prevents behavioral change and drives the system toward the highly endemic non-adoption state. When norms are neither very weak nor very strong, qualitatively different dynamical patterns emerge depending on the perceived effectiveness of protective measures ψ . When ψ is low, adoption remains unattractive, and the system converges to a non-adoption endemic equilibrium (Figure 1a).

For intermediate or high ψ , weak norms generate sustained oscillations in infection prevalence and protective behavior (Figure 1a-b). The intuition is as follows. When adoption is low, infection spreads, increasing perceived risk and encouraging individuals to adopt protective measures. Because social norms are relatively weak, individuals can respond flexibly to this increased risk. Rising adoption then suppresses transmission, which reduces perceived risk and leads to a subsequent decline in adoption. As the strength of social norms θ increases, the inertia generated by norms increases both the amplitude and the period of these oscillations (Figure 1a-b). Thus, although the disease prevalence decreases only slightly with θ within the oscillatory regime, stronger norms produce higher infection peaks and therefore greater pressure on the healthcare system.

With moderate norms, the system converges either: (1) to the unique endemic non-adoption equilibrium, when ψ is intermediate (Figure 1a); or (2) to the unique disease-free full-adoption equilibrium, when ψ is high (Figure 1a-b). An approximation in the limit $k \rightarrow \infty$ (Section S3.1 in the SI) indicates that the latter occurs when

$$\theta \in (c, \eta\psi - c), \quad \psi \in \left(\frac{2c}{\eta}, 1\right], \quad \text{and } c < \eta/2, \quad (10)$$

where $\eta \in [0, 1]$ denotes the endemic human infection prevalence at zero adoption in the fixed-behavior, no-resistance model (see Section S3.1 of the SI). This implies that populations with moderate norms can transition to disease-free full-adoption even from low initial adoption, provided that adoption costs are not too large, the perceived effectiveness is sufficiently high, and the transmission rate is sufficiently high, but not so high as to preclude the existence of a disease-free equilibrium. This occurs because the benefits of adoption are sufficiently salient, and moderate social norms create the right balance: they allow enough flexibility to initiate behavioral change, but provide enough social inertia to stabilize full adoption once it becomes widespread. Note that all

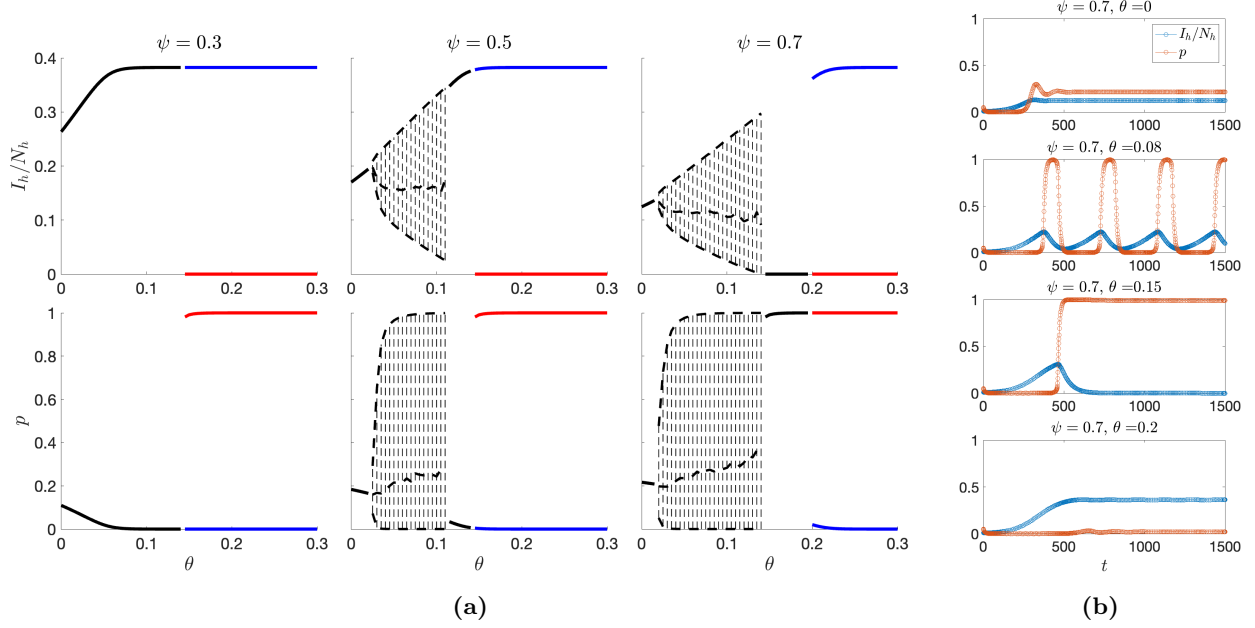


Figure 1: (a) Effects of the strength of social norms θ and the perceived effectiveness of protective measures ψ on long-term model behavior. Shown are: infection prevalence I_h/N_h (top row) and the fraction of adopters p (bottom row). Solid lines indicate equilibrium values. In the oscillatory regime, dashed lines show the long-term minima, maxima, and time-averaged values. Under strong norms, the system is bistable between an endemic non-adoption equilibrium (blue) and a disease-free full-adoption equilibrium (red). For each parameter combination, results are based on 1000 simulations with randomly sampled initial conditions. (b) Illustration of model dynamics for different values of θ . From top to bottom, increasing θ leads to a mixed-adoption endemic equilibrium, oscillatory adoption and infection dynamics, a disease-free full-adoption equilibrium, and a no-adoption endemic equilibrium. We set $c = 0.1$, with all other parameters fixed at their default values (see Section S1 in the SI).

the above results are robust to alternative formulations of perceived infection risk (Figures S1- S2 in the SI).

3.3 Joint dynamics of disease, insecticide resistance, and human behavior

We now consider the full model, which couples ecological competition between resistant and non-resistant mosquitoes with dynamic protective behavior and evolving perceived effectiveness. We model perceived effectiveness ψ as a shared belief updated through individual and social experience with protective measures. Specifically, we assume two baseline levels, ψ_h and ψ_l , with $\psi_l < \psi_h$, corresponding to high and low perceived effectiveness, respectively. We posit that ψ_l depends on ξ and d'_1 , whereas ψ_h depends on ξ and d_1 (see Section S1 in the SI). When non-resistant mosquitoes dominate, protective measures are more likely to have visible effects, such as reducing mosquito abundance or increasing mosquito mortality after spraying or ITN use, leading to higher perceived effectiveness (Suuron *et al.*, 2020). In contrast, when resistant mosquitoes are prevalent, these effects are weaker, shifting perceived effectiveness toward lower values. We therefore define the inferred level of perceived effectiveness as

$$w = \frac{N}{N_v} \psi_h + \frac{N'}{N_v} \psi_l, \quad (11)$$

where $N = S + I$ and $N' = S' + I'$ are the abundances of non-resistant and resistant mosquitoes, respectively, and $N_v = N + N'$ is the total mosquito population.

Because information about effectiveness is generated mainly through use of protective measures, and is more visible when mosquito abundance is higher, we assume that belief updating is proportional to adoption and mosquito abundance per individual:

$$\dot{\psi} = \mu p \frac{N_v}{N_h} (w - \psi), \quad (12)$$

where $\mu > 0$ is the updating rate, and the initial value ψ_0 captures prior experience, public information, or institutional guidance.

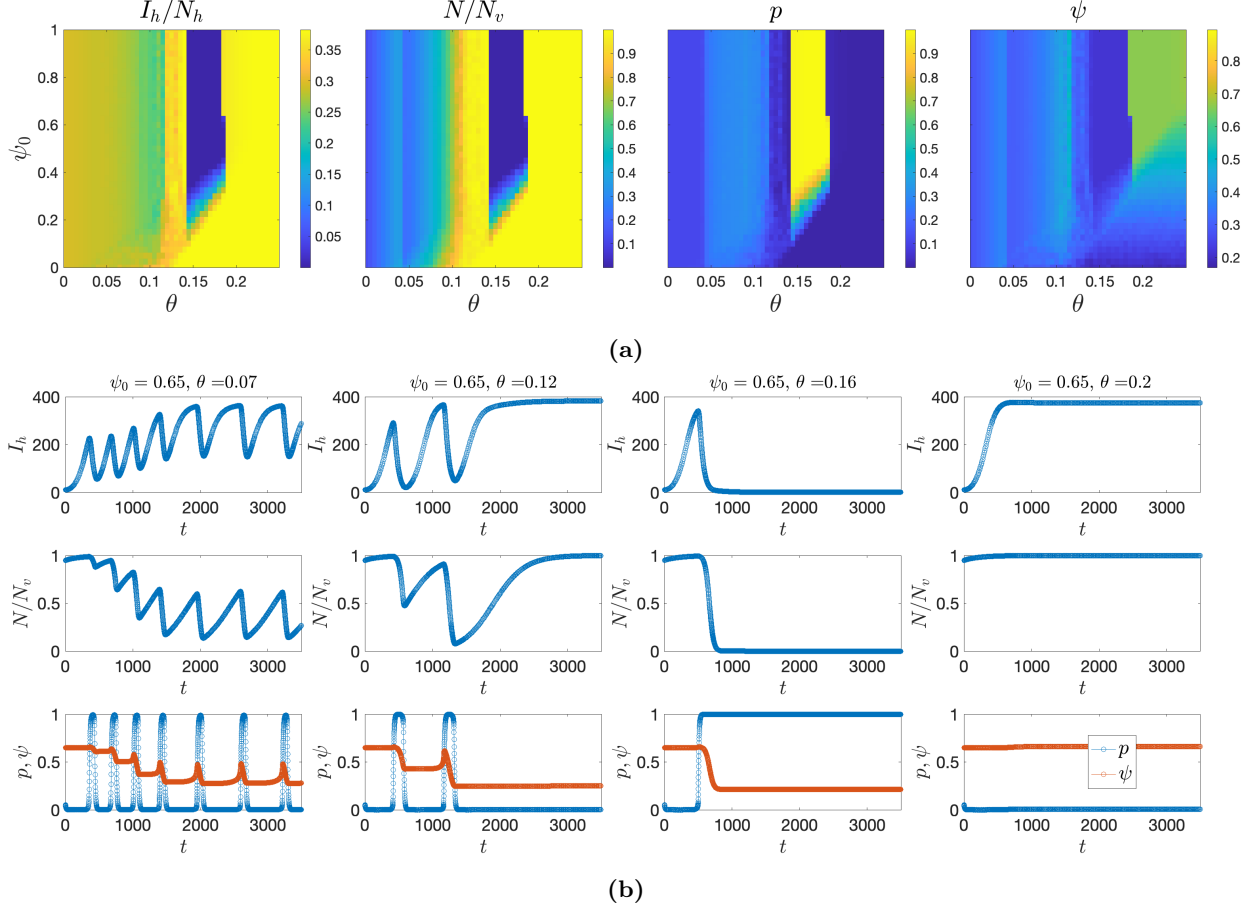


Figure 2: (a) Effects of the strength of norms θ and initial perceived effectiveness ψ_0 on long-term model behavior, assuming a small initial fraction of adopters $p(0) = 0.05$. Shown are: infection prevalence I_h/N_h , the fraction of non-resistant mosquitoes N/N_v , adoption p , and perceived effectiveness ψ . For each parameter combination, outcomes are averaged over 100 simulations with randomly sampled initial conditions. (b) Examples of model dynamics at increasing values of θ . From left to right, the system exhibits oscillatory adoption and infection with coexistence of resistant and non-resistant mosquitoes; a no-adoption endemic equilibrium with extinction of resistant mosquitoes; a disease-free full-adoption equilibrium with extinction of non-resistant mosquitoes; and a return to the no-adoption endemic equilibrium. $c = 0.1$ and all other parameters are fixed at their default values (see Section S1 of the SI).

Effects of behavior and perceived effectiveness on mosquito ecology. Dynamic behavior and evolving perceived effectiveness can qualitatively alter mosquito ecology. With fixed behavior, coexistence between resistant and non-resistant mosquitoes is non-generic, occurring only under an exact balance between their effective carrying capacities⁵. In contrast, when behavior and

⁵Specifically, coexistence requires $K \left(1 - \frac{d(p)}{r}\right) = K \left(1 - \frac{d'(p)}{r'}\right)$, or equivalently $p = \frac{(r-r')d_0}{r'd_1 - rd'_1}$.

perceived effectiveness evolve, this balance can be maintained endogenously. This phenomenon typically occurs under relatively weak social norms, which allow flexible behavioral responses (Figures 2 and S3-S4 in the SI). Depending on the parameters, the system may converge to a stable coexistence equilibrium or exhibit endogenous oscillations in adoption, perceived effectiveness, disease prevalence, and mosquito composition. In the oscillatory case, coexistence is sustained by feedback between resistance and behavior: increasing resistance makes protective measures appear less effective, reducing perceived effectiveness and weakening adoption. Lower adoption then relaxes selection pressure favoring resistance, allowing non-resistant mosquitoes to recover. These results suggest that vector ecology is shaped not only by ecological conditions but also by material, social, and economic factors: coexistence emerges primarily under relatively weak norms, moderate adoption costs, and moderate-to-high vector carrying capacity (Figures S11-S14 in the SI).

Model dynamics. The qualitative regimes identified in the model without resistance persist in the full model but acquire an ecological dimension (Figures 2 and S3-S4 in the SI). In the oscillatory regime, mosquito composition can also oscillate. In the disease-free full-adoption regime non-resistant mosquitoes are driven to extinction, whereas in the no-adoption endemic regime resistant mosquitoes eventually go extinct. Resistance can also generate an additional outcome: a no-adoption endemic regime can emerge at the upper end of the weak-norm range, particularly when protection-induced mortality differs sufficiently in resistant and non-resistant mosquitoes (Figure 2). Here, the system initially oscillates, but during phases of resistant-mosquito dominance, protective measures appear substantially less effective, causing perceived effectiveness to decline and adoption to become very low. Because belief updating is proportional to adoption, low adoption then slows the recovery of perceived effectiveness. Together with norm-induced inertia, this feedback can trap the system in a no-adoption state, eliminating selection for resistance and ultimately driving resistant mosquitoes extinct.

Norms can therefore have highly nonlinear effects on disease dynamics and mosquito ecology. As θ increases, the system can shift from coexistence with malaria waves and oscillating adoption to a highly endemic no-adoption state with resistance extinction. Further increases in θ can induce an abrupt transition to full adoption, disease elimination, and dominance of resistant mosquitoes, followed at still higher θ by a return to the no-adoption endemic regime with resistance extinction. These results are robust to alternative formulations of perceived risk (Figures S5- S8 in the SI).

Nonlinear feedbacks promote counterintuitive outcomes. The above results show that feedbacks among human behavior, perceived effectiveness, disease transmission, and mosquito ecology can make epidemiological outcomes depend highly nonlinearly on behavioral and biological parameters. As a result, this can generate counterintuitive outcomes. We illustrate this by comparing insecticide-treated nets (ITNs) with untreated nets (UTNs). ITNs have generally been found to be more effective than UTNs even when mosquitoes are resistant to the insecticide (Strode *et al.*, 2014; Unwin *et al.*, 2023; Pryce *et al.*, 2018; D’alessandro *et al.*, 1995), although their effectiveness may decline to that of UTNs under resistance (Asidi *et al.*, 2012). Moreover, operational costs to produce and distribute ITNs and individual costs when not subsidized can be substantial barriers to their distribution and usage (Guyatt *et al.*, 2002; Webster *et al.*, 2005). UTNs may therefore retain an important role in vector-borne disease control (Okumu, 2020).

To compare ITN and UTN outcomes when uptake is governed by human behavior, we explore a model version in which nets do not kill mosquitoes, $d(p) = d'(p) \equiv d_0$ (Section S4.2 in the SI). Figure 3 presents the results under weak norms across different values of the cost parameter c . Strikingly, UTNs outperform ITNs over much of the parameter space in the weak-norm scenario. Since, ITNs generate oscillations over more of the parameter space explored, echoing previous research (Morsky *et al.*, 2023; Morsky, 2025). The underlying dynamics are discussed in Section S4.2 of the SI. This advantage of UTNs could be even more pronounced if ITNs carry higher perceived

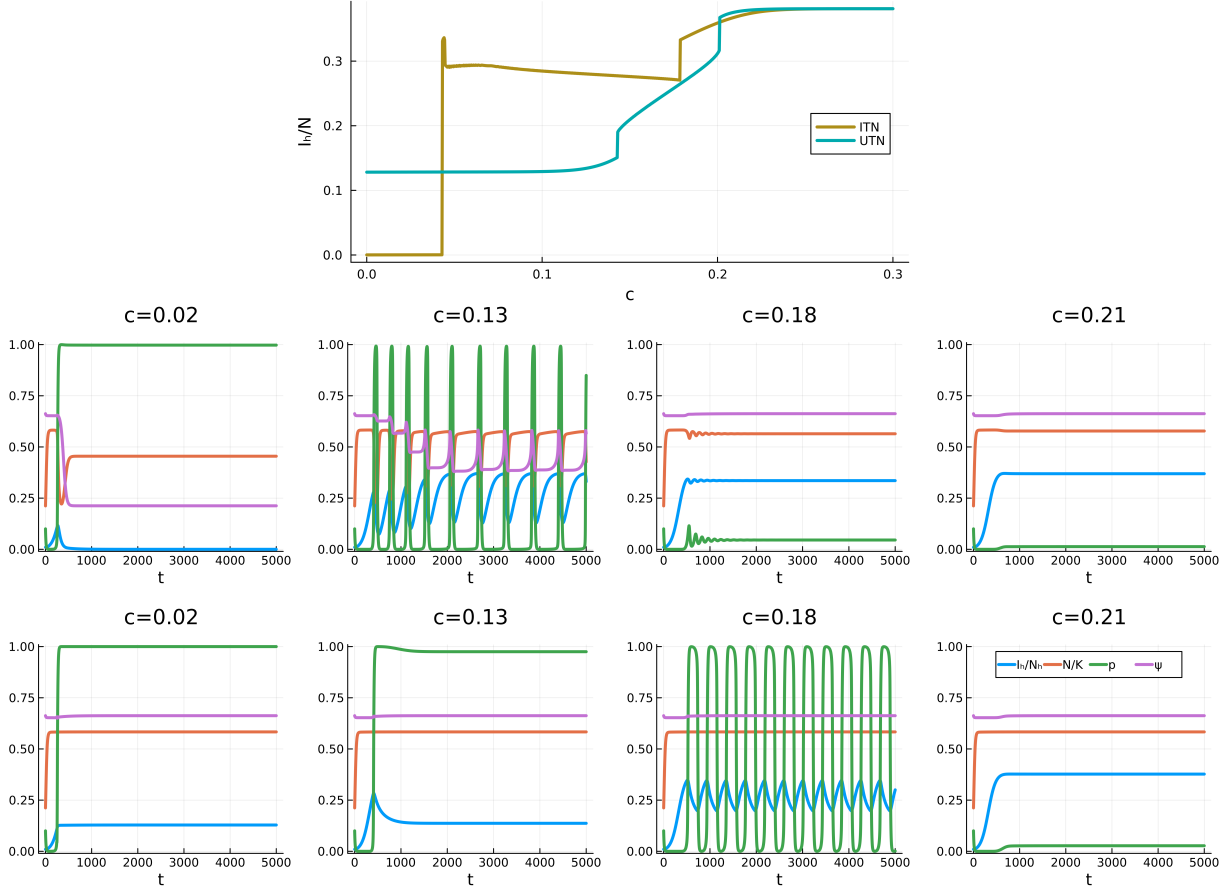


Figure 3: Effects of ITNs compared with UTNs. Shown are: the average infection prevalence I_h/N_h for $t \in [0, 5000]$ with outcomes saved and averaged over increments $\Delta t = 0.1$ (first row); representative time series for the ITN (second row); and UTN scenarios (third row). For much of the c -parameter space, UTNs outperforms ITNs in reducing I_h/N_h . $\theta = 0.08$ and all other parameters are fixed at their default values (see Section S1 of the SI).

costs due to concerns about the potential health effects of insecticides, even when their material costs are equal. Conversely, the disadvantage of ITNs can be somewhat mitigated if information about efficacy and adoption of the protective measure is localized (Morsky, 2025). However, previous models using the same behavioral mechanism show that populations that are weakly socially coupled can synchronize their behaviors over a wide range of parameter values (Wang *et al.*, 2026). And, this synchronization occurs even if disease cannot spread between the populations.

3.4 Responses to policy and environmental change

The above nonlinear and sometimes counterintuitive outcomes naturally motivate examining how the effectiveness of policy interventions varies across social and cultural contexts. First, our results indicate that transitions between weak, moderate, and strong norms can produce abrupt shifts in disease prevalence, protective behavior, and resistance dynamics. As a result, gradual social, cultural, or demographic changes - including migration, conflict, natural hazards, market integration, and globalization - may have disproportionate epidemiological consequences. Epidemiological forecasting and public-health planning should therefore account for processes reshaping social norms.

Second, interventions that increase the perceived effectiveness of protection (e.g., via increasing

the initial perceived effectiveness ψ_0), or that reduce adoption costs c , can be important tools for malaria control. Such interventions include mass bed-net distribution campaigns and educational campaigns highlighting the effectiveness of bed nets in protecting against mosquitoes and reducing malaria transmission, as well as addressing concerns about the safety of the insecticides used to treat them. The model suggests that their effects may remain limited until a critical threshold (in ψ_0 or c) is crossed, after which the system rapidly shifts to low or nearly zero infection. This implies that policymakers should sustain these efforts even when short-term improvements are limited. Specifically, the model suggests that increasing ψ_0 induces tipping-point dynamics mainly under moderate norms when initial adoption is low (Figure S3), whereas reducing c can induce such dynamics across a broader range of norm strengths, from weak to relatively strong (Figures S11-S12 in the SI). Interestingly, weak norms are most robust in maintaining nonzero adoption as c increases, regardless of initial adoption. For convergence to disease-free full adoption, however, moderate norms permit the largest increase in c when initial adoption is low, whereas strong norms do so when initial adoption is high. (Figures S11-S12 in the SI). Consequently, moderate norms produce the most favorable outcomes when initial adoption is low and costs are moderate, but can surprisingly produce the least favorable outcomes when both are high.

Third, public-health communication strategies should be tailored to the social and cultural context. To examine this idea, we introduce a model extension in which authority messaging promotes protective behavior and communicates an authority-promoted level of protective-measure effectiveness, while trust in authorities changes dynamically in response to the consistency between message content and perceived effectiveness and relaxes toward a baseline community- or culture-specific trust level (Section S5.2 of the SI). This extension shows that messaging can expand the conditions under which disease-free full adoption occurs in the context of baseline trust, but that the effective content of messaging depends on the strength of social norms. In populations with strong norms, highly optimistic messaging can trigger high adoption that is subsequently stabilized by social-norm inertia, even if trust later declines as resistant mosquitoes spread. In populations with weaker norms, more moderate messaging can be more effective because it remains more consistent with perceived effectiveness and therefore better preserves trust. By contrast, under cautious mistrust, public-health outcomes can depend nonlinearly on messaging intensity. For example, when messaging promotes moderate effectiveness, disease elimination occurs under moderate norms and only when messaging intensity is either very low or high, and disappears at intermediate intensity. Overall, the model suggests that appropriately chosen messaging content and intensity can lead to disease elimination in populations with moderate or strong norms.

Fourth, housing improvements such as screened windows, closed eaves, and better construction may reduce human-mosquito contact and the biting rate m . By lowering perceived infection risk, however, this may also weaken incentives to protection and trigger epidemic resurgence (Figures S15-S16 in the SI). The epidemiological benefits of urban development and broader economic growth should therefore be assessed alongside their behavioral feedbacks. Finally, the effects of environmental change are also mediated by social factors. An increase in mosquito carrying capacity K can produce an abrupt shift from a highly endemic state to a disease-free regime, but only under moderate or strong norms (Figure S13 in the SI). Conversely, lower K , for example due to drier conditions limiting breeding sites, directly reduces infection risk but may also weaken incentives to adopt protective measures. Under moderate or strong norms, this can trigger an abrupt reversion to a severe endemic state in populations where malaria had previously been under control (Figures S13-S14 in the SI). However, under strong norms once adoption has reached a sufficiently high level, the disease-free state persists even after ecological conditions change (Figure S14 in the SI).

3.5 Exploratory empirical illustration using data from Liberia

We complement our theoretical modeling with data from Liberia, where malaria accounts for 34% of outpatient visits, 47% of inpatient admissions, and 23% of hospital deaths (Tugbeh *et al.*, 2026). We compile a variety of data from the Demographic and Health Surveys (DHS) on individual behaviors and household characteristics, data on mosquito resistance to permethrin and deltamethrin from Hancock *et al.* 2022 (Hancock *et al.*, 2022), and data on the incidence of malaria from the Malaria Atlas (Malaria Atlas Project, 2025). As a proxy for the strength of social norms, we calculate a Herfindahl–Hirschman Index (HHI) for religious diversity in a population center (“cluster”) from the DHS surveys; the index is adjusted for the multi-stage sampling design and the different probabilities of respondents being in the sample. Higher values of the index indicate greater religious homogeneity, which we associate with stronger norms. We focus on the adoption of bed nets because very few respondents in the DHS samples reported using indoor residual spraying; most of the bed nets used were ITNs (Sections S6.1–S6.2 of the SI).

We examine associations between religious homogeneity, used as an indirect proxy for norm strength, and three outcomes: bed-net adoption, malaria incidence, and mosquito resistance to insecticides. Our modeling results suggest that these associations depend on the perceived costs and effectiveness of bed nets, ecological conditions, household characteristics that may impact perceived risk, and authority messaging. Because we use publicly available datasets, we lack fine-grained data on many of these factors. For this reason, we use religious diversity as an imperfect proxy for norm strength; we lack data on the degree of authority messaging and the costs of net adoption. None of the statistical models we use are causal; also we do not model household location choice and land use/land cover change. Instead, our analysis is exploratory: we examine whether the qualitative patterns suggested by the mathematical model are also visible in the available data.

Given evidence that trusted authorities play an important role in promoting protective measures against malaria in Liberia (Babalola *et al.*, 2016; Ministry of Health, Republic of Liberia, 2016), and drawing on insights from our model, we expect stronger norms to be more likely associated with a higher bed-net adoption and lower malaria burden, *ceteris paribus*. Resistance dynamics are more complex: different bed-net brands have used different insecticides, so resistance is shaped by the co-evolution of resistance to these insecticides, individual choices of net brands, procurement and distribution campaigns. Nevertheless, following the model’s logic that stronger norms promote social inertia, we expect resistance to a given insecticide to change more slowly over time in clusters with stronger norms.

To examine whether individual bed-net adoption is associated with norm strength in a manner consistent with the behavioral mechanism in Eq. 7, we estimate a logit model for net ownership, pooling the data from multiple DHS waves and clustering the standard errors at the population cluster level. The covariates included are HHI, the corresponding share of same-religion peers within a population cluster who adopt nets, their interaction, survey-year indicators, the percentage change in baseline local malaria incidence from 2000 to 2005, and demographic and socioeconomic proxies for the perceived risk (Section S6.3.1 of the SI). Since we do not have consistent data on the price of nets, the impact is captured by the constant estimated in the model. We find that HHI and its interaction with peers’ net ownership have positive, statistically significant coefficients (Section S6.3.1 of the SI). The estimated marginal effects between peers’ ownership and individual net ownership increases with HHI (Figure 4a), consistent with stronger social norms amplifying the influence of others’ behavior (Eq. 7). Interestingly, the predicted probability of net ownership increases with peer ownership but at a diminishing rate (Figure S20 in the SI). This suggests that peer effects are strongest when the baseline level of net adoption is low. A greater increase in local malaria incidence is also associated with a higher probability of net ownership.

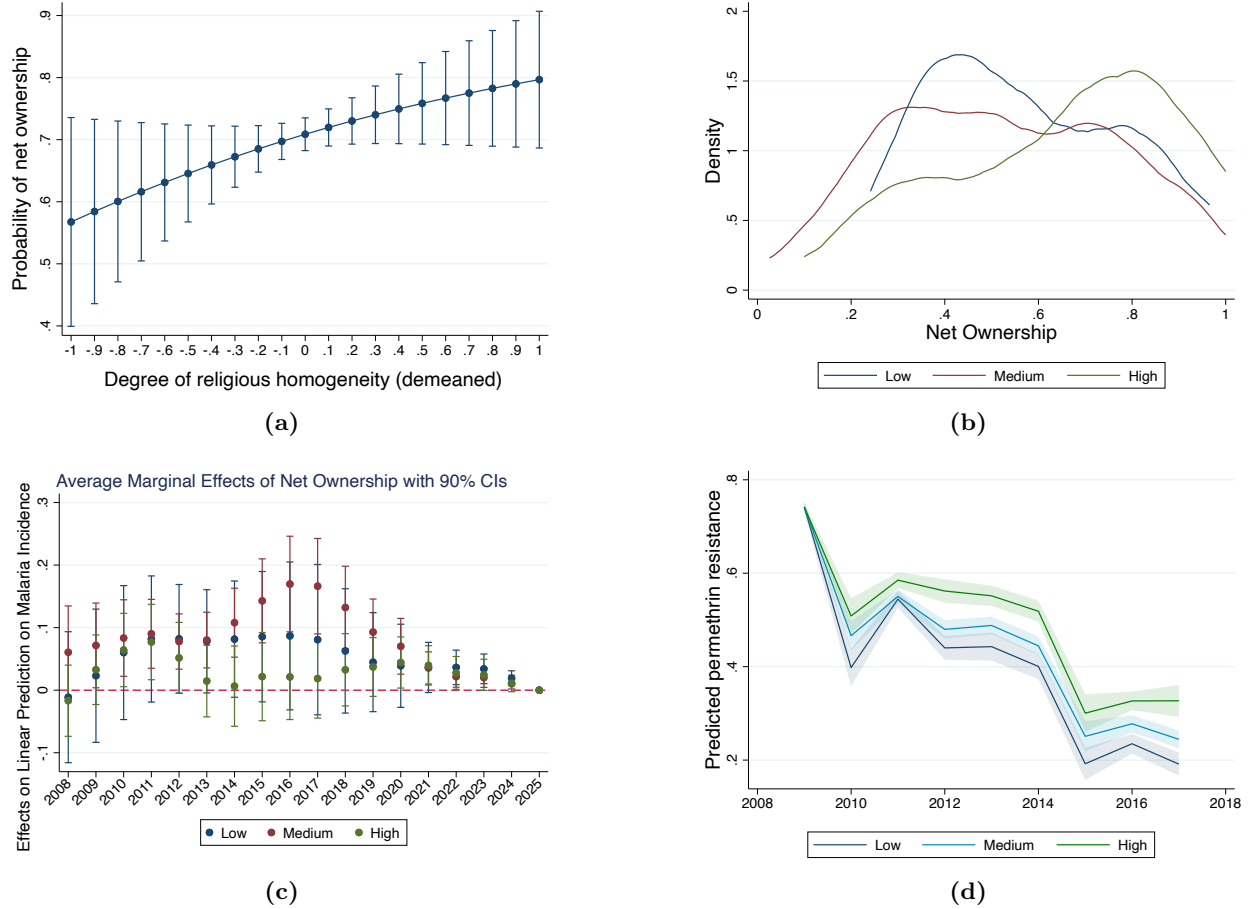


Figure 4: Exploratory associations between norm strength and malaria-related outcomes in Liberia. Norm strength is proxied by the degree of religious homogeneity, measured using the Herfindahl-Hirschman Index (HHI); higher HHI indicates greater religious homogeneity, which we use as a proxy for stronger norms. (a) Estimated marginal association between the share of same-religion peers who own a net and an individual’s probability of net ownership, plotted against demeaned HHI with 95% confidence intervals. (b) Distribution of cluster-level net ownership in 2009 by baseline HHI group. (c) Estimated marginal impact of baseline net ownership and HHI group on annual malaria incidence using 2025 as the reference year; bars indicate 90% confidence intervals. (d) Estimated permethrin resistance from 2009 to 2017 by baseline HHI group, based on a model with cluster and year fixed effects and HHI-group-by-year interactions. The trajectories share a fitted 2009 reference point and therefore show differences in subsequent changes. Shaded regions indicate 95% confidence intervals.

The surveyed DHS clusters are spatially explicit, which allowed us to link geospatial information to the surveyed locations. To examine the association between malaria incidence and HHI, we attached the annual malaria incidence estimates from the Malaria Atlas to DHS clusters’ 2009 HHI and net ownership. Descriptive statistics indicate that net ownership tended to be higher in the high-HHI group (Figure 4b), which is consistent with the mechanism suggested by the model.

To measure the association between malaria incidence and HHI, we estimated a model, with malaria incidence as the dependent variable and cluster and year fixed effects, and interactions among baseline HHI group, net ownership, and year (Section S6.3.2 in the SI); the standard errors are clustered at the population cluster level. The data span 2008 to 2025. We used only the 2009 clusters for this analysis to ensure a balanced panel. Figure 4c shows, for each HHI group, the estimated marginal association between demeaned baseline net ownership and malaria incidence in each year relative to 2025. For all three groups, the association is mostly positive initially, diverges

by group with the medium norms group increasing the most, and then declines toward zero.⁶ The association is smallest in clusters with strong norms and largest in clusters with medium norms, but the differences between groups are not statistically significant in any year. Overall, the pattern potentially reflects noisy transient dynamics.

Finally, to examine the association between norm strength and resistance, we estimate a panel data model with mosquito resistance as the outcome and HHI interacted with year and cluster and year fixed effects on 2009 DHS clusters for 2008-2017 due to data availability. We clustered the standard errors at the population cluster level. We find that over the study period, permethrin resistance declined overall, but the higher HHI communities exhibited slower decline relative to the low HHI communities (Figure 4d), consistent with the mechanism suggested by the model. This pattern is robust to multiple specifications and definitions of the HHI variable; results for deltamethrin are more nuanced (Section S6.3.3 in the SI).

Overall, the empirical results indicate that stronger norms are associated with increases in the probability of net ownership, but also with slower responses in terms of the malaria incidence and resistance. These are consistent with predictions from the theoretical model.

Discussion

Unlike the COVID-19 pandemic, which was global in scope but concentrated within a relatively limited historical period, vector-borne diseases have represented a persistent threat to humans throughout history and are now becoming an increasingly urgent concern in many parts of the world, including regions that have had little previous experience with them. What dynamics should we expect in these newly affected regions and in regions where vector-borne diseases are already endemic? Addressing this question requires accounting not only for healthcare-system capacity, governmental preventive actions, and information campaigns, but also for dynamically changing human behavior - including the adoption of protective measures, mobility, and patterns of exposure - and the material, social, cognitive, and cultural factors that shape it. Yet these behavioral dynamics and their feedbacks with disease transmission and vector ecology remain commonly overlooked in existing literature. Here, we developed a mathematical model to disentangle this complexity, focusing on malaria and the role of social norms in shaping the use of protective measures, such as bed nets and indoor residual spraying.

The main findings. First, our results demonstrate an important role of social norms in shaping malaria-related public-health outcomes. The model exhibits three main dynamical regimes: oscillations in protective behavior and disease prevalence; low adoption and high prevalence; and high adoption with low or absent disease. The oscillatory regime occurs primarily under weak norms, which allow behavior to respond flexibly to perceived risk. By contrast, moderate and strong norms can support sustained high adoption, but through different mechanisms and under different conditions. Strong norms can lock the system into the highly beneficial state when protective behavior is already well established in the population or strongly aligned with prevailing normative expectations, but can instead stabilize non-adoption when such behavior is initially uncommon. In the latter case moderate norms may be particularly beneficial, producing tipping-point dynamics towards high adoption and low disease. Indeed, moderate norms provide sufficient flexibility to initiate behavioral change and sufficient social inertia to reinforce and sustain the transition. Such transitions occur only when perceived disease risk and effectiveness of protection are sufficiently high and their perceived costs are sufficiently low. Interestingly, strong-norm populations can also

⁶As expected due to 2025 being the reference year. In other words, the results suggest the marginal associations decline across time, with the largest decline for the medium norms group.

undergo similar transitions when protective behavior is promoted by a highly trusted authority. Overall, our model suggests that social norms can sustain malaria protection or entrench non-adoption, depending on their strength, initial behavior, perceived risks and adoption costs, trust in authorities, and broader economic and ecological factors.

Second, dynamically changing human behavior can substantially alter vector ecology. We illustrate this by focusing on insecticide resistance, a major challenge in malaria control that can severely reduce the effectiveness of protective interventions. Indeed, the use of insecticide-based protective measures creates selective pressures affecting the ecological dynamics of resistant and non-resistant mosquito populations. These ecological changes, in turn, feed back on human behavior through their effects on perceived disease risk and perceived effectiveness of protective measures. We show that this coupling can generate non-trivial patterns of vector ecology. Specifically, whereas fixed levels of protective behavior generally allow only one mosquito type to persist, dynamically changing behavior can sustain coexistence between resistant and non-resistant mosquitoes. In our model, this coexistence occurs primarily under relatively weak norms, allowing protective behavior - and consequently selection pressure - to vary over time. The same feedbacks can also produce counterintuitive epidemiological outcomes: under weak norms, untreated nets can reduce average malaria prevalence more effectively than insecticide-treated nets. Together, these findings suggest that accounting for social and cultural context may improve predictions of insecticide-resistance dynamics and inform the planning and targeting of public-health interventions.

Policy implications. Our results highlight the importance of social and cultural context for designing effective interventions. Under conditions conducive to tipping-point dynamics - most commonly, though not exclusively, moderate or strong norm strength - interventions may produce little observable effect until a critical threshold is crossed. The system may then rapidly transition toward high adoption and low infection. Policymakers should therefore avoid abandoning interventions because of limited short-term effects, as sustained efforts may be required to move a population across a critical threshold. Among the interventions considered, reducing the costs of protection appears comparatively robust, as it can induce such transitions across a broad range of norm strengths, from weak to relatively strong. By contrast, authority messaging is more context dependent: under strong norms and baseline trust, it can facilitate a transition towards high adoption and low infection, whereas under cautious mistrust it may be ineffective or counterproductive, particularly under moderate norms. Policymakers should also anticipate that broader socioeconomic or ecological changes, such as migration, urban development, or shifts toward drier or wetter conditions may trigger abrupt behavioral and epidemiological transitions.

Empirical assessment and data needs. Our modeling results suggest that social factors, such as social norms, and their feedbacks with disease transmission and vector ecology can substantially reshape malaria dynamics. Our exploratory empirical analysis examined whether qualitative patterns suggested by the model are also visible in the available data. Our main takeaway is that, at a general level, the data do not contradict the model. Directly validating the model and predicting malaria dynamics while accounting for social and cultural contexts will require additional systematic data collection and further model development.

First, although empirical studies document an important role of norms in decisions to use malaria-protective measures (Asingizwe *et al.*, 2019; Perkins *et al.*, 2019; Babalola *et al.*, 2022; Olapeju *et al.*, 2022; Phok *et al.*, 2022; Lopes-Rafegas *et al.*, 2023; Agbo *et al.*, 2026), systematic and comparable community-level measures of norm strength remain scarce in malaria-endemic and at-risk settings. In our empirical analysis, we use religious homogeneity as a proxy for norm strength given data availability, although it is an indirect and imperfect measure. More detailed data are needed to account for social and cultural context in malaria control and prevention. One promising approach is to operationalize norm strength through cultural tightness (the degree of norm strength

and tolerance for deviant behaviors within a group) (Gelfand *et al.*, 2011, 2017). Validated tightness–looseness survey items (Gelfand *et al.*, 2011) could be incorporated into individual-level surveys and aggregated to construct community-level measures. Data on perceived effectiveness and costs of protective measures, exposure to authority messaging or behavioral nudges, and trust in their sources would also improve malaria prediction. In addition, the differing empirical associations between our proxy for norm strength and resistance to permethrin and deltamethrin underscore the importance of data on insecticide-specific exposure and history. Finally, our empirical results highlight the importance of longer time series for distinguishing long-term trends from transient dynamics. Together, such data could help explain differences in protective behavior and malaria burden across communities.

Additionally, our empirical results indicate that religious homogeneity can change substantially over time (Section S6.3.3 of the SI). These changes may reflect socioeconomic factors, as well as migration, and may signal shifts in the social context relevant to norms. This opens new directions for empirical and theoretical research on vector-borne diseases. On the theoretical side, models could represent spatially and culturally distinct communities coupled by human migration and mosquito dispersal (Ruktanonchai *et al.*, 2016). Human migration could introduce heterogeneity and change norm strength within communities (thereby linking social identity to epidemiological dynamics), while transferring infection between them. Mosquito dispersal could alter competition between resistant and non-resistant mosquitoes within communities, and local policy interventions could have effects across connected communities. Overall, such models can improve our understanding of epidemiological processes while capturing the dynamic nature of sociocultural contexts.

Social norms as mediators of societal responses to shocks. Our findings are consistent with recent theoretical work on how social norms mediate societal responses to epidemiological and environmental hazards. Models of mask wearing and vaccination during COVID-19 (Morsky *et al.*, 2023; Morsky, 2025; Wang *et al.*, 2026) and adaptation to climate-related natural hazards in non-industrial societies (Tverskoi *et al.*, 2024) identify similar patterns: weak norms enable flexible responses, strong norms generate path dependence, and moderate norms provide enough flexibility to initiate behavioral change when a new behavior is rare and enough inertia to reinforce the transition as it becomes more widespread. Together, these findings point to a broader principle governing how norm strength shapes societal adaptation to shocks. Here, we show how these patterns manifest in vector-borne diseases, highlighting that the effects of social norms on behavioral change depend on other material, social, psychological, ecological, and economic factors.

Generalization to other vector-borne diseases. The behavioral framework developed here could be adapted to other vector-borne diseases, although the epidemiological and ecological components would need to reflect the transmission cycle of each disease. One relevant example is Lyme disease, whose geographic distribution has expanded from historically high-incidence areas in the Northeast and upper Midwest into neighboring regions of the United States (Mead *et al.*, 2024). Human behavior is central to Lyme disease prevention through the use of repellents and protective clothing, tick checks and removal, residential yard management, and decisions about when and where to engage in outdoor activities (Aenishaenslin *et al.*, 2015; Berry *et al.*, 2018b; Richardson *et al.*, 2019; Keesing *et al.*, 2022; Bowser *et al.*, 2025). Residential acaricide application provides a partial analogue to indoor residual spraying for malaria: uptake of both may depend on perceived risk, costs, information, and social influence (Hornbostel *et al.*, 2021; Valois *et al.*, 2023). A Lyme disease model could therefore couple socially influenced protective behavior with tick and wildlife-host ecology, while also incorporating residential and recreational exposure patterns. More broadly, such extensions would help determine whether the effects of norm strength identified here also arise under substantially different vector-borne disease transmission cycles.

Conclusion. Overall, our results show that social norms can qualitatively reshape malaria

dynamics by governing behavioral adaptation and feedbacks between protective behavior, disease transmission, and vector ecology. More broadly, vector-borne diseases should be understood as coupled human–biological systems in which social and cultural processes can alter not only intervention uptake, but also epidemiological and ecological trajectories. Incorporating these feedbacks into forecasting and intervention design may be especially important as vector-borne diseases expand into populations with diverse social and cultural contexts.

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Supplementary Information

S1 Parameterization and simulation protocol

Default parameter values used throughout the paper are provided in Table S1. Parameters related to disease transmission, recovery, and mosquito mortality were drawn from the existing literature. The mosquito carrying capacity K , human population size N_h , and the intrinsic mosquito growth rate were chosen so that the mosquito-to-human ratio lies in the interval $[1, 10]$ for the specified mosquito mortality parameters.

The steepness parameter of the behavioral response, k , was selected to preserve a threshold-like mechanism while still allowing for some decision error. The behavior and belief updating rates, ϵ and μ , respectively, were chosen to reflect that beliefs are typically updated faster than behavior. Finally, we assume that the high and low levels of perceived effectiveness of protective measures denoted by ψ_h and ψ_l , respectively, are increasing functions of the actual effectiveness of these measures. We define

$$\psi_h = \frac{\xi}{\Xi} + \frac{d_1}{D} - \frac{\xi}{\Xi} \cdot \frac{d_1}{D}$$

and

$$\psi_l = \frac{\xi}{\Xi} + \frac{d'_1}{D} - \frac{\xi}{\Xi} \cdot \frac{d'_1}{D},$$

where Ξ and D are scaling coefficients. This functional form combines the effects of personal protection and mosquito mortality while keeping perceived effectiveness bounded between 0 and 1, provided that the scaled quantities lie between 0 and 1. In the numerical simulations, we set $\Xi = 2$ and $D = 0.08$.

For each parameter combination, we numerically integrate the ODE system 100 times using randomly sampled initial conditions. Unless otherwise stated, we draw

$$\begin{aligned} I_h(0) &\sim \text{Unif}\{1, \dots, 20\}, \\ I(0) &\sim \text{Unif}\{1, \dots, 100\}, \quad I'(0) \sim \text{Unif}\{1, \dots, 10\}, \\ S'(0) &\sim \text{Unif}\{1, \dots, 200\}, \quad S(0) \sim \text{Unif}\left\{1, \dots, \left\lfloor K \left(1 - \frac{d_0}{r}\right) \right\rfloor - I(0) - S'(0) - I'(0)\right\}, \\ p(0) &\sim U(0, 1), \quad \psi(0) \sim U(0, 1). \end{aligned}$$

Thus, the initial numbers of infected humans and infected mosquitoes are kept relatively small, while the initial mosquito population is sampled below the disease-free ecological carrying capacity. In contrast, the initial fraction of adopters and the initial perceived effectiveness of protective measures are sampled across the full interval from 0 to 1.

Each ODE system is integrated up to $t = 10000$, and outcomes are computed over the final 2000 time steps. In figures illustrating example dynamics, initial conditions are fixed at $I_h(0) = 10$, $S(0) = 2000$, and $I(0) = 10$. When resistance is included, we additionally set $S'(0) = 100$ and $I'(0) = 10$.

Par.	Meaning	Value	Source
r	Non-resistant mosquito growth rate	0.12	See text
r'	Resistant mosquito growth rate	0.11	See text
d_0	Baseline mosquito mortality rate	0.05	Agusto <i>et al.</i> (2013); Buonomo (2015); Wairimu <i>et al.</i> (2018); Mandal <i>et al.</i> (2011)
d_1	Mortality due to protection, non-resistant	0.05	Agusto <i>et al.</i> (2013); Buonomo (2015); Wairimu <i>et al.</i> (2018); Mandal <i>et al.</i> (2011)
d'_1	Mortality due to protection, resistant	0.01	Wairimu <i>et al.</i> (2018)
K	Mosquito carrying capacity	10000	See text
N_h	Human population size	1000	See text
m	Mosquito biting rate	0.2	Mandal <i>et al.</i> (2011)
b_h	Transmission probability (mosquito $\rightarrow$ human)	0.1	Mandal <i>et al.</i> (2011)
b_v	Transmission probability (human $\rightarrow$ mosquito)	0.1	Mandal <i>et al.</i> (2011)
ξ	Decrease in transmission due to protection	0.2	Wairimu <i>et al.</i> (2018)
γ	Human recovery rate	0.025	Mandal <i>et al.</i> (2011); Wairimu <i>et al.</i> (2018)
k	Steepness of behavioral response	100	See text
ϵ	Behavior updating rate	0.1	See text
μ	Perceived-effectiveness updating rate	0.2	See text
c	Cost of protection	Varied	-
ψ_0	Initial perceived effectiveness of protection	Varied	-
θ	Strength of social norms	Varied	-

Table S1: Model parameters, their descriptions, and default values.

S2 Additional results on epidemic dynamics with fixed behavior and no resistance

For fixed adoption p and in the absence of resistance, the total mosquito population $N(t) = S(t) + I(t)$ satisfies

$$\dot{N}(t) = rN(t)(1 - N(t)/K) - dN(t),$$

independently of infection. With $0 < d < r$ and assuming $0 < N(0) \leq K$, we have $N(t) \rightarrow N = K(1 - d/r)$. We first analyze the limiting infection system:

$$\dot{I}_h = \beta_h(N_h - I_h)I - \gamma I_h, \quad (\text{S1})$$

$$\dot{I} = \beta_v(N - I)I_h - dI. \quad (\text{S2})$$

Theorem S1. *Disease-free equilibrium $(I_h^*, I^*) = (0, 0)$ always exists. It is locally stable if*

$$C < \frac{\gamma d_0}{1 - \frac{d_0}{r}} \text{ or } C \in \left(\frac{\gamma d_0}{1 - \frac{d_0}{r}}, \frac{\gamma(d_0 + d_1)}{(1 - \xi)^2 \left(1 - \frac{d_0 + d_1}{r}\right)} \right) \text{ and } p > p^\#. \quad (\text{S3})$$

Endemic equilibrium

$$(I_h^*, I^*) = \left(\frac{\beta_v \beta_h N N_h - \gamma d}{\beta_v (\beta_h N + \gamma)}, \frac{\beta_v \beta_h N N_h - \gamma d}{\beta_h (\beta_v N_h + d)} \right) \quad (\text{S4})$$

exists and is locally stable if

$$C \in \left(\frac{\gamma d_0}{1 - \frac{d_0}{r}}, \frac{\gamma(d_0 + d_1)}{(1 - \xi)^2 \left(1 - \frac{d_0 + d_1}{r}\right)} \right) \text{ and } p < p^\# \text{ or } C > \frac{\gamma(d_0 + d_1)}{(1 - \xi)^2 \left(1 - \frac{d_0 + d_1}{r}\right)}, \quad (\text{S5})$$

where

$$C = \frac{m^2 b_h b_v K}{N_h} \quad (\text{S6})$$

and $p^\#$ is the unique solution to the cubic equation

$$C(1 - \xi p)^2 \left(1 - \frac{d_0 + d_1 p}{r}\right) = \gamma(d_0 + d_1 p) \text{ on } [0, 1]. \quad (\text{S7})$$

Proof. It follows directly from the equilibrium conditions that any equilibrium of the model must be either disease-free or endemic.

We first consider the disease-free equilibrium. It exists for all parameter values and its local stability is determined by evaluating the Jacobian matrix at the equilibrium point

$$J = \begin{pmatrix} -\beta_h I^* - \gamma & \beta_h (N_h - I_h^*) \\ \beta_v (N - I^*) & -\beta_v I_h^* - d \end{pmatrix}, \quad (\text{S8})$$

which transforms into

$$J = \begin{pmatrix} -\gamma & \beta_h N_h \\ \beta_v N & -d \end{pmatrix}. \quad (\text{S9})$$

The corresponding characteristic polynomial is

$$\lambda^2 + (\gamma + d)\lambda + \gamma d - \beta_h \beta_v N N_h = 0. \quad (\text{S10})$$

Since the discriminant $(\gamma - d)^2 + 4\beta_h \beta_v N N_h > 0$, the Jacobian has two distinct real eigenvalues

$$\lambda_{1,2} = \frac{-(\gamma + d) \pm \sqrt{(\gamma - d)^2 + 4\beta_h \beta_v N N_h}}{2}$$

with $\lambda_1 < 0$ and $\lambda_2 < 0$ if $\beta_h \beta_v N N_h < \gamma d$. This implies that the disease-free equilibrium is locally stable if $\beta_h \beta_v N N_h < \gamma d$.

Second, consider the endemic equilibrium given in (S4). It exists if and only if $I^* > 0$ and $I_h^* > 0$, which is equivalent to $\beta_h \beta_v N N_h > \gamma d$. To check the stability, the corresponding characteristic equation is

$$\lambda^2 + (A + B)\lambda + AB - \beta_h \beta_v (N - I^*)(N_h - I_h^*) = 0, \quad (\text{S11})$$

where $A = \beta_h I^* + \gamma$ and $B = \beta_v I_h^* + d$. Note that $AB = \beta_h \beta_v N N_h$ and $\beta_h \beta_v (N - I^*)(N_h - I_h^*) = \gamma d$, meaning that the characteristic equation transforms into

$$\lambda^2 + (A + B)\lambda + AB - d\gamma = 0,$$

with

$$\lambda_{1,2} = \frac{-(A + B) \pm \sqrt{(A - B)^2 + 4d\gamma}}{2}.$$

This implies $\lambda_1 < 0$ and $\lambda_2 < 0$ if $AB > d\gamma$. As a result, the endemic equilibrium exists and is locally stable if

$$\beta_h \beta_v N N_h > \gamma d. \quad (\text{S12})$$

Finally note that the condition $\beta_h \beta_v N N_h > \gamma d$ is equivalent to

$$F(p) = \gamma(d_0 + d_1 p) - C(1 - \xi p)^2 \left(1 - \frac{d_0 + d_1 p}{r}\right) < 0,$$

where C is defined by (S6). Note that

$$\forall p \in [0, 1] : \frac{dF}{dp} = \gamma d_1 + C(1 - \xi p) \left[2\xi \left(1 - \frac{d_0 + d_1 p}{r}\right) + \frac{d_1}{r}(1 - \xi p) \right] > 0 \quad (\text{S13})$$

so that F is a monotonically increasing function on $[0, 1]$. As a result, the endemic equilibrium exists and is locally stable if (1) $F(0) < 0$ and $F(1) < 0$ or (2) $F(0) < 0$, $F(1) \geq 0$ and $p < p^\#$, where $p^\#$ is the unique solution to the equation $F(p) = 0$ on $[0, 1]$. Otherwise, the disease-free equilibrium is locally stable, which proves the theorem. $\square$

Theorem S2. *Consider the reduced system (S1)-(S2) on the region*

$$\Omega = [0, N_h] \times [0, N].$$

Under the conditions in Theorem S1 for local stability of the disease-free equilibrium, the disease-free equilibrium is globally asymptotically stable in Ω . Under the conditions in Theorem S1 for existence and local stability of the endemic equilibrium, the endemic equilibrium is globally asymptotically stable in $\Omega \setminus \{(0, 0)\}$.

Proof. First, we show that Ω is positively invariant. On the boundary $I_h = 0$, we have

$$\dot{I}_h = \beta_h N_h I \geq 0,$$

whereas on the boundary $I_h = N_h$,

$$\dot{I}_h = -\gamma N_h < 0.$$

Similarly, on the boundary $I = 0$,

$$\dot{I} = \beta_v N I_h \geq 0,$$

whereas on the boundary $I = N$,

$$\dot{I} = -dN < 0.$$

Thus, the vector field points inward or is tangent on each boundary face of Ω . Since these inequalities also hold at the vertices, the tangent-cone condition is satisfied on the whole boundary, and therefore Ω is positively invariant (Girol, 2020).

Next, the divergence of the vector field is

$$\frac{\partial \dot{I}_h}{\partial I_h} + \frac{\partial \dot{I}}{\partial I} = -\beta_h I - \gamma - \beta_v I_h - d < 0$$

throughout Ω . Therefore, by the Bendixson-Dulac criterion, the system has no nontrivial periodic orbits in the interior of Ω .

Under the conditions in Theorem S1 for local stability of the disease-free equilibrium, the endemic equilibrium does not exist in Ω . Hence, the disease-free equilibrium is the only equilibrium

in Ω . Since Ω is compact and positively invariant, each trajectory has a nonempty compact ω -limit set. By the Poincare-Bendixson theorem, and because nontrivial periodic orbits are ruled out, any ω -limit set must either be an equilibrium or contain equilibria together with connecting orbits. Since the disease-free equilibrium is the only equilibrium in Ω and is locally asymptotically stable, each ω -limit set reduces to the disease-free equilibrium. Therefore, every trajectory in Ω converges to the disease-free equilibrium, meaning that it is globally asymptotically stable in Ω .

Under the conditions in Theorem S1 for existence and local stability of the endemic equilibrium, it exists in the interior of Ω , while the disease-free equilibrium is unstable. Moreover, any trajectory starting in $\Omega \setminus \{(0, 0)\}$ enters the interior of Ω . Indeed, if $I_h(0) > 0$ or $I(0) > 0$, then the coupling terms imply that both $I_h(t)$ and $I(t)$ are positive for all sufficiently small $t > 0$. It remains to rule out convergence to the disease-free equilibrium. Under the endemic-equilibrium conditions, the Jacobian at the disease-free equilibrium satisfies

$$\det J = \gamma d - \beta_h \beta_v N N_h < 0.$$

Thus, the disease-free equilibrium is a saddle. Let $\lambda_1 < 0$ denote the negative eigenvalue of J , and let $v = (v_1, v_2)$ be the corresponding eigenvector. From

$$(-\gamma - \lambda_1)v_1 + \beta_h N_h v_2 = 0,$$

we obtain

$$v_2 = \frac{\gamma + \lambda_1}{\beta_h N_h} v_1.$$

Since

$$\gamma + \lambda_1 = \frac{\gamma - d - \sqrt{(\gamma - d)^2 + 4\beta_h \beta_v N N_h}}{2} < 0,$$

the components of v have opposite signs. As a result by the stable manifold theorem, the local stable manifold of the disease-free equilibrium intersects Ω only at $(0, 0)$. Consequently, no trajectory starting in $\Omega \setminus \{(0, 0)\}$ can converge to the disease-free equilibrium. Then, by the Poincare-Bendixson theorem, each trajectory in $\Omega \setminus \{(0, 0)\}$ converges to the endemic equilibrium, meaning it is globally asymptotically stable in $\Omega \setminus \{(0, 0)\}$. $\square$

Remark. Note that since the total mosquito population converges independently to N , the infection subsystem approaches the reduced system analyzed above. Away from the threshold $\beta_h \beta_v N_h N = \gamma d$, standard asymptotically autonomous convergence theory (Thieme, 1992) extends these long-term outcomes to the original system, with convergence to the endemic equilibrium requiring nonzero initial infection.

S3 Additional results on joint dynamics of disease and human behavior with no resistance

S3.1 Additional analytical results

Here, we consider the case in which mosquito resistance is absent, individual behavior evolves dynamically, and the perceived effectiveness of protective measures ψ is fixed. In this case, the model reduces to the following four equations:

$$\dot{I}_h = \beta_h(p)(N_h - I_h)I - \gamma I_h, \tag{S14}$$

$$\dot{S} = r(S + I) \left(1 - \frac{S + I}{K}\right) - \beta_v(p)SI_h - d(p)S, \quad (\text{S15})$$

$$\dot{I} = \beta_v(p)SI_h - d(p)I, \quad (\text{S16})$$

$$\dot{p} = \varepsilon \left(\frac{1}{1 + e^{k(-\psi I_h/N_h + c + \theta(1-2p))}} - p \right). \quad (\text{S17})$$

Here, we restrict attention to biologically meaningful equilibria with $S^* + I^* > 0$. The mosquito-extinction equilibrium for which $S^* = I^* = 0$, necessarily has $I_h^* = 0$ and is unstable since $r > d_0 + d_1$.

Theorem S3. *Assume that $\theta > 0$. Then, for any $\delta \in (0, 0.5)$, there exists $k'(\delta)$ such that for all $k > k'(\delta)$, every equilibrium $(I_h^*(k), S^*(k), I^*(k), p^*(k))$ satisfying $p^*(k) \in [\delta, 1 - \delta]$ is locally unstable.*

Proof. Let $\delta \in (0, 0.5)$, $k > k'(\delta)$, and $(I_h^*(k), S^*(k), I^*(k), p^*(k))$ is an equilibrium with $p^*(k) \in [\delta, 1 - \delta]$, where

$$k'(\delta) = \frac{r + \gamma + \frac{b_h m}{N_h} K \left(1 - \frac{d_0}{r}\right) + b_v m + \epsilon}{2\theta\epsilon(1 - \delta)\delta}.$$

Then (S17) implies

$$-\psi I_h^*(k)/N_h + c + \theta(1 - 2p^*(k)) = \frac{1}{k} \ln \left(\frac{1 - p^*(k)}{p^*(k)} \right).$$

To determine the local stability of this equilibrium, we consider the Jacobian matrix of the system evaluated at the equilibrium point

$$J(k) = \begin{pmatrix} -\beta_h(p^*)I^* - \gamma & 0 & \beta_h(p^*)(N_h - I_h^*) & -\frac{b_h m \xi}{N_h}(N_h - I_h^*)I^* \\ -\beta_v(p^*)S^* & r \left(1 - 2\frac{S^* + I^*}{K}\right) - \beta_v(p^*)I_h^* - d(p^*) & r \left(1 - 2\frac{S^* + I^*}{K}\right) & \frac{b_v m \xi}{N_h}S^*I_h^* - d_1S^* \\ \beta_v(p^*)S^* & \beta_v(p^*)I_h^* & -d(p^*) & -\frac{b_v m \xi}{N_h}S^*I_h^* - d_1I^* \\ Z \frac{k\psi}{N_h} & 0 & 0 & 2kZ\theta - \epsilon \end{pmatrix}, \quad (\text{S18})$$

where

$$Z = \frac{\varepsilon e^{k(-\psi I_h^*/N_h + c + \theta(1-2p^*))}}{\left(1 + e^{k(-\psi I_h^*/N_h + c + \theta(1-2p^*))}\right)^2} = \epsilon(1 - p^*(k))p^*(k). \quad (\text{S19})$$

Then, the sum of all roots of the characteristic polynomial is

$$\begin{aligned} \lambda_1(k) + \lambda_2(k) + \lambda_3(k) + \lambda_4(k) &= \text{tr}(J(k)) = -r - \gamma - \beta_h I^* - \beta_v I_h^* - \epsilon + 2\theta\epsilon k(1 - p^*(k))p^*(k) > \\ &> -\beta_h I^* - \beta_v I_h^* + \frac{b_h m}{N_h} K \left(1 - \frac{d_0}{r}\right) + b_v m \geq 0. \end{aligned}$$

As a result, $\lambda_1(k) + \lambda_2(k) + \lambda_3(k) + \lambda_4(k) > 0$, which means that at least one eigenvalue of $J(k)$ has positive real part. Therefore, the equilibrium is locally unstable, which proves the theorem. $\square$

This theorem shows that, when k is sufficiently large, any locally stable equilibrium must lie near one of the two behavioral extremes: near-no adoption or near-full adoption. We next examine the existence and stability of equilibria in these two regimes.

By Theorem S1, every equilibrium of the model is either disease-free, satisfying $I_h^* = I^* = 0$, or endemic with (I_h^*, I^*) defined by (S4). We begin with the disease-free equilibria, examining their existence and local stability when k is large. Recall that $C = \frac{m^2 b_h b_v K}{N_h}$.

Theorem S4. *Assume that $\theta > 0$. Then, there exists $k' > 0$, such that for each $k > k'$ the following statements hold.*

- *A near-full-adoption disease-free equilibrium of the form $\left(0, K\left(1 - \frac{d_0 + d_1 p^*(k)}{r}\right), 0, p^*(k)\right)$ with $\lim_{k \rightarrow \infty} p^*(k) = 1$ exists if and only if $\theta > c$. When it exists, it is unique. Moreover, it is locally stable if $C < \frac{\gamma(d_0 + d_1)}{(1-\xi)^2\left(1 - \frac{d_0 + d_1}{r}\right)}$ and unstable if $C > \frac{\gamma(d_0 + d_1)}{(1-\xi)^2\left(1 - \frac{d_0 + d_1}{r}\right)}$.*
- *A near-no-adoption disease-free equilibrium of the form $\left(0, K\left(1 - \frac{d_0 + d_1 p^*(k)}{r}\right), 0, p^*(k)\right)$ with $\lim_{k \rightarrow \infty} p^*(k) = 0$ exists and is unique. It is locally stable if $C < \frac{\gamma d_0}{1 - \frac{d_0}{r}}$ and unstable if $C > \frac{\gamma d_0}{1 - \frac{d_0}{r}}$.*
- *These are the only locally stable disease-free equilibria.*

In both cases, $p^*(k)$ is the corresponding solution to

$$\frac{1}{1 + e^{k(c + \theta(1 - 2p^*(k)))}} = p^*(k). \quad (\text{S20})$$

Proof. Consider a disease-free equilibrium with the fraction of adopters $p^*(k)$. Then $p^*(k)$ satisfies (S20). The right-hand side of (S20) is a sigmoid function of p with threshold point

$$p = \frac{1}{2} \left(1 + \frac{c}{\theta}\right),$$

at which the exponent vanishes and the function equals $\frac{1}{2}$.

As $k \rightarrow \infty$, this sigmoid becomes increasingly steep, and the equation admits at most three solutions for sufficiently large k : one with $p(k) \rightarrow 0$, one with $p(k) \rightarrow 1$, and one satisfying $p(k) \rightarrow \frac{1}{2} \left(1 + \frac{c}{\theta}\right)$. By Theorem S3, the interior solution (if exists) is locally unstable for sufficiently large k . Therefore, any locally stable disease-free equilibrium must correspond to a solution satisfying either $p(k) \rightarrow 0$ or $p(k) \rightarrow 1$. With large k , the near-no-adoption solution to (S20) always exists as the threshold point is greater than zero. The near-full-adoption solution exists if and only if the threshold point satisfies

$$\frac{1}{2} \left(1 + \frac{c}{\theta}\right) < 1,$$

which is equivalent to $\theta > c$. Combining these observations with Theorem S1 gives the stated existence conditions for these equilibria in the large- k regime.

We now turn to the local stability of these equilibria. The Jacobian matrix evaluated at an equilibrium point is given by (S18). For disease-free equilibria with $p^*(k) \rightarrow 0$ or $p^*(k) \rightarrow 1$, we have

$$Z = \frac{\epsilon e^{k(c + \theta(1 - 2p^*(k)))}}{\left(1 + e^{k(c + \theta(1 - 2p^*(k)))}\right)^2}.$$

Since $p^*(k) \rightarrow 0$ or $p^*(k) \rightarrow 1$, the exponent in the above expression tends to $\pm\infty$, and therefore

$$\lim_{k \rightarrow \infty} kZ = 0.$$

As a result, the Jacobian becomes asymptotically block triangular, and

$$\lim_{k \rightarrow \infty} \det(J - \lambda I) = (-\epsilon - \lambda) \det(J_0 - \lambda I),$$

where J_0 is the Jacobian of the reduced system with fixed p . Hence, the eigenvalues of $J(k)$ converge to the eigenvalues of J_0 together with the additional eigenvalue $-\epsilon < 0$. By continuity of eigenvalues with respect to matrix entries, for all sufficiently large k , the sign of the real parts of the eigenvalues of $J(k)$ coincides with that of the limiting matrix, provided the reduced equilibrium is hyperbolic. Therefore, local stability of the full system for sufficiently large k is determined by local stability of the corresponding equilibrium of the reduced system. The stability conditions stated in this theorem then follow directly from Theorem S1, which proves the theorem. $\square$

We now turn to endemic equilibria and analyze their existence and local stability in the large- k regime.

Theorem S5. *Assume that $\theta > 0$. Then, there exists $k' > 0$, such that for each $k > k'$ the following statements hold.*

- *A near-full-adoption endemic equilibrium of the form $(I_h^*(p^*(k)), S^*(p^*(k)), I^*(p^*(k)), p^*(k))$ with $\lim_{k \rightarrow \infty} p^*(k) = 1$ exists if and only if $\theta > c - \frac{I_h^*(1)}{N_h} \psi$ and*

$$C > \frac{\gamma(d_0 + d_1)}{(1 - \xi)^2 \left(1 - \frac{d_0 + d_1}{r}\right)}. \quad (\text{S21})$$

When it exists, it is unique and is locally stable.

- *A near-no-adoption endemic equilibrium $(I_h^*(p^*(k)), S^*(p^*(k)), I^*(p^*(k)), p^*(k))$ with $\lim_{k \rightarrow \infty} p^*(k) = 0$ exists if and only if $\theta > \frac{I_h^*(0)}{N_h} \psi - c$ and*

$$C > \frac{\gamma d_0}{1 - \frac{d_0}{r}}. \quad (\text{S22})$$

When it exists, it is unique and is locally stable.

- *These are the only locally stable endemic equilibria.*

In both cases, $p^(k)$ is the corresponding solution to*

$$\frac{1}{1 + e^{k(-\psi I_h^*(p^*(k))/N_h + c + \theta(1 - 2p^*(k)))}} = p^*(k), \quad (\text{S23})$$

where $I_h^(p^*(k))$ and $I^*(p^*(k))$ are defined by (S4), and $S^*(p^*(k)) = K \left(1 - \frac{d_0 + d_1 p^*(k)}{r}\right) - I^*(p^*(k))$.*

Proof. Let

$$G(p) = -\psi \frac{I_h^*(p)}{N_h} + c + \theta(1 - 2p),$$

where $I_h^*(p)$ is defined by (S4). Then the equilibrium condition for p can be written as

$$\frac{1}{1 + e^{kG(p)}} = p.$$

As $k \rightarrow \infty$, any interior solution $p^* \in (0, 1)$ to this equation must satisfy

$$G(p^*) = 0.$$

Substituting the expression for $I_h^*(p)$ from (S4), this equation reduces to a polynomial equation of degree four. Therefore, it admits at most four real roots $p_1, \dots, p_4$ in $(0, 1)$.

Hence, for sufficiently large k , (S23) can admit at most six asymptotic solution branches: one with $p(k) \rightarrow 0$, one with $p(k) \rightarrow 1$, and at most four interior branches satisfying $p(k) \rightarrow p_j$, $j = 1, \dots, 4$. If these interior branches exist, they are locally unstable for sufficiently large k by Theorem S3. Therefore, any locally stable endemic equilibrium must correspond to a branch satisfying either $p(k) \rightarrow 0$ or $p(k) \rightarrow 1$.

The near-no-adoption branch exists if and only if

$$G(0) = -\psi \frac{I_h^*(0)}{N_h} + c + \theta > 0,$$

while the near-full-adoption branch exists if and only if

$$G(1) = -\psi \frac{I_h^*(1)}{N_h} + c - \theta < 0.$$

The uniqueness of these endpoint branches as well as their local stability properties follow by the same arguments used in the disease-free case. This proves the theorem. $\square$

These results provide intuition about the long-term behavior of the system in the limit $k \rightarrow \infty$. Specifically, consider the parameter regime

$$\frac{\gamma d_0}{(1 - \frac{d_0}{r})} < C < \frac{\gamma(d_0 + d_1)}{(1 - \xi)^2(1 - \frac{d_0 + d_1}{r})}. \quad (\text{S24})$$

The following corollary, which stems directly from Theorems S4 and S5, characterizes the long-term dynamics of the system as a function of the strength of social norms θ , the perceived effectiveness of protective measures ψ , and the cost of adoption c .

Corollary 1. *Within the parameter regime described by (S24) and in the limit $k \rightarrow \infty$, the following statements hold.*

- Let $\psi < \frac{cN_h}{I_h^*(0)}$. If $\theta \in (0, c)$, then the near-no-adoption endemic equilibrium is the unique locally stable equilibrium. If $\theta > c$, then the near-no-adoption endemic equilibrium coexists with the near-full-adoption disease-free equilibrium, and both are locally stable equilibria.
- Let $\frac{cN_h}{I_h^*(0)} < \psi < \frac{2cN_h}{I_h^*(0)}$. If $\theta < \frac{I_h^*(0)}{N_h}\psi - c$, then the model has no locally stable equilibrium. If

$$\theta \in \left(\frac{I_h^*(0)}{N_h}\psi - c, c \right),$$

then the near-no-adoption endemic equilibrium is the unique locally stable equilibrium. If $\theta > c$, then the near-no-adoption endemic equilibrium coexists with the near-full-adoption disease-free equilibrium, and both are locally stable equilibria.

- Let $\psi > \frac{2cN_h}{I_h^*(0)}$. If $\theta < c$, then the model has no locally stable equilibrium. If

$$\theta \in \left(c, \frac{I_h^*(0)}{N_h} \psi - c \right), \quad (\text{S25})$$

then the near-full-adoption disease-free equilibrium is the unique locally stable equilibrium. If $\theta > \frac{I_h^*(0)}{N_h} \psi - c$, the near-no-adoption endemic equilibrium coexists with the near-full-adoption disease-free equilibrium, and both are locally stable equilibria.

It follows that for large k and within the parameter regime described by (S24), the system may exhibit one of three possibilities: no locally stable equilibrium, a unique locally stable equilibrium (either the near-no-adoption endemic equilibrium or the near-full-adoption disease-free equilibrium), or coexistence of both equilibria as locally stable states. In the bistable regime, numerical simulations indicate that, when the initial fraction of adopters is low, the system converges to the near-no-adoption endemic equilibrium (see Figure 1 in the main text). As a result starting from a low level of adoption, convergence to the near-full-adoption disease-free equilibrium occurs only when the perceived effectiveness of protective measures ψ is sufficiently high, adoption costs c are not too large, and the strength of social norms θ is intermediate. Finally, endogenous oscillations in disease prevalence and adoption are observed when ψ is not small and θ is relatively weak.

Finally, we show that the near-full-adoption disease-free equilibrium is the unique locally stable equilibrium if the transmission rate is sufficiently high, but not so high that it precludes the existence of a disease-free equilibrium. Let $N^* = K \left(1 - \frac{d_0}{r} \right)$.

Corollary 2. Assume that $b_h = b_v = b$. Within the parameter regime described by (S24) and in the limit $k \rightarrow \infty$, the near-full-adoption disease-free equilibrium is the unique locally stable equilibrium if

$$b \in \left(\frac{1}{m} \sqrt{\frac{N_h}{K} \frac{\gamma d_0}{1 - \frac{d_0}{r}}}, \frac{1}{m} \sqrt{\frac{N_h}{K} \frac{\gamma(d_0 + d_1)}{(1 - \xi)^2 \left(1 - \frac{d_0 + d_1}{r} \right)}} \right), \quad (\text{S26})$$

$$b > \frac{\frac{(c+\theta)m\gamma}{\psi} + \sqrt{\left(\frac{(c+\theta)m\gamma}{\psi} \right)^2 + \frac{4\gamma d_0 m^2 N^*}{N_h} \left(1 - \frac{c+\theta}{\psi} \right)}}{\frac{2m^2 N^*}{N_h} \left(1 - \frac{c+\theta}{\psi} \right)}, \quad (\text{S27})$$

and

$$\psi > c + \theta > 2c. \quad (\text{S28})$$

Proof. Note that (S24) is equivalent to (S26), while the inequalities $\psi > \frac{2cN_h}{I_h^*(0)}$ and (S25) are equivalent to $\theta > c$ and

$$I_h^*(0) > (c + \theta) \frac{N_h}{\psi}.$$

The latter condition can be rewritten as $G(b) > 0$, where

$$G(b) = \frac{m^2 N^*}{N_h} \left(1 - \frac{c + \theta}{\psi} \right) b^2 - \frac{c + \theta}{\psi} m \gamma b - \gamma d_0. \quad (\text{S29})$$

Since $G(0) < 0$, and if $\psi \leq c + \theta$ then $G(b) < 0$ for all $b > 0$, it follows that the condition $G(b) > 0$ is equivalent to requiring $\psi > c + \theta$ and $b > b^\#$, where $b^\#$ is the largest root of the equation $G(b) = 0$. This yields (S27)-(S28). $\square$

S3.2 Supplementary figures

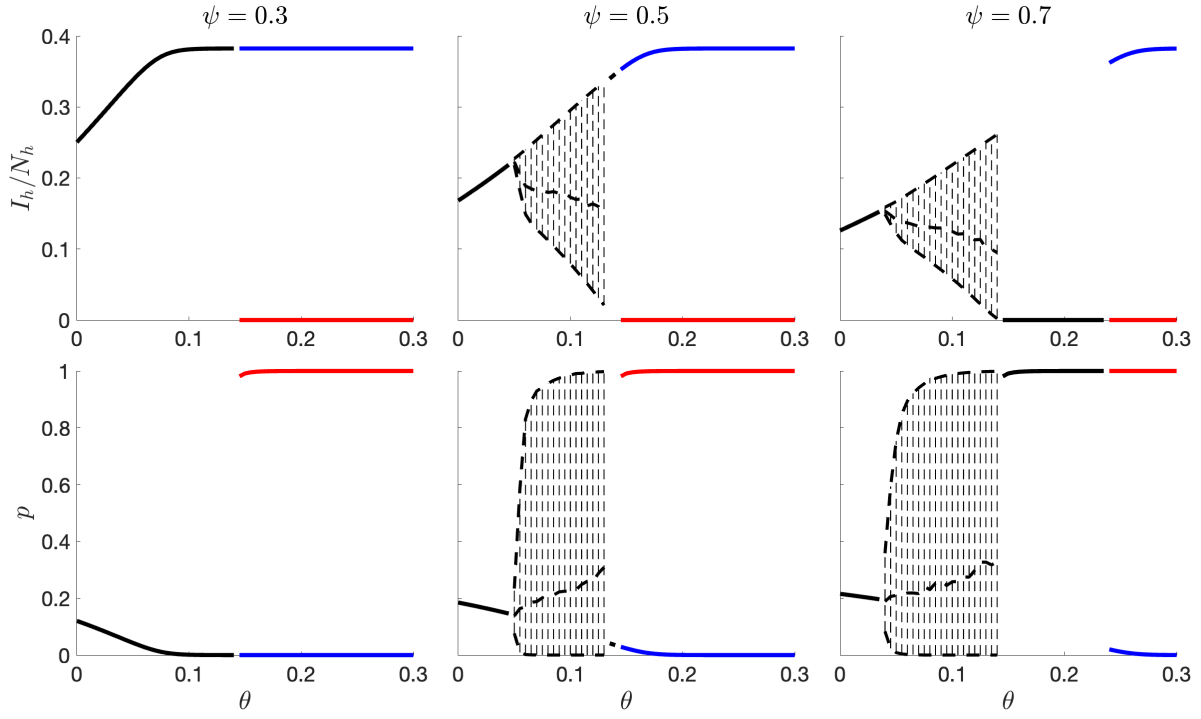


Figure S1: Same as Figure 1, but with perceived infection risk depending on mosquito abundance per human individual N_v/N_h : $R(I_h, N_v, q) = h(1 - \psi q) \cdot \frac{I_h}{N_h} \cdot \frac{N_v}{N_h}$, where $h > 0$ is a scaling parameter. In the simulations, we set $h = 0.2$.

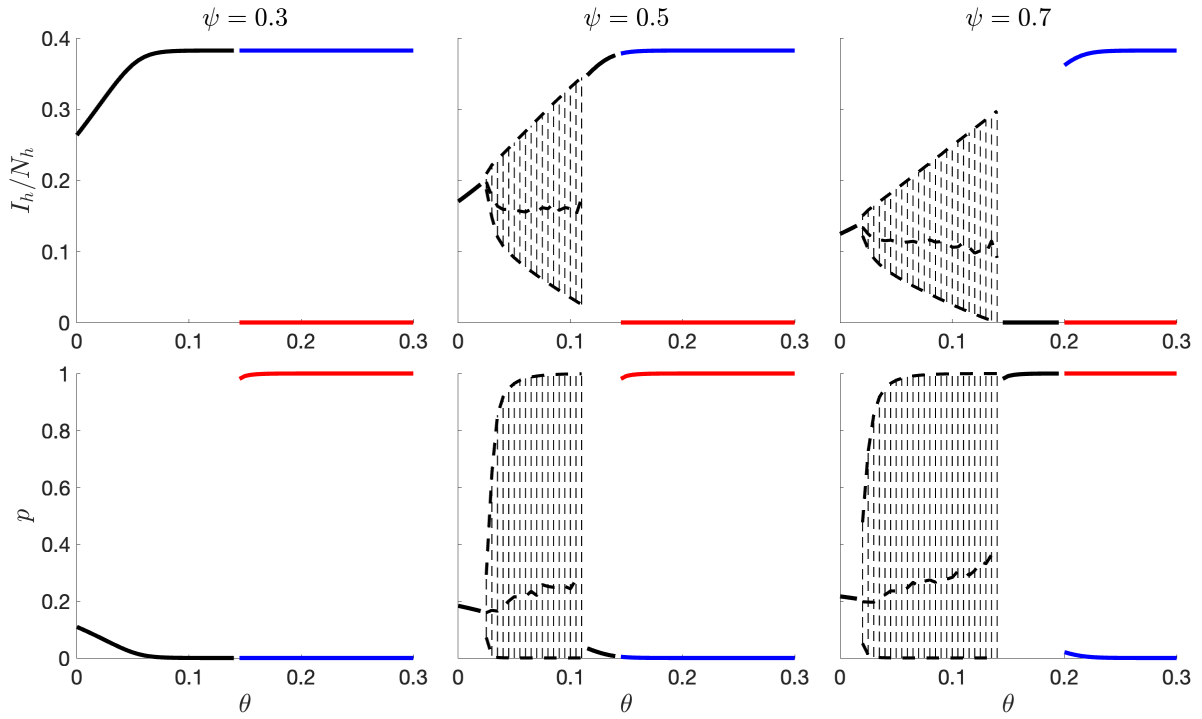


Figure S2: Same as Figure 1, but with perceived infection risk depending on human infection incidence $-\dot{S}_h + \gamma I_h$: $R(-\dot{S}_h + \gamma I_h, q) = h(1 - \psi q) \cdot \frac{-\dot{S}_h + \gamma I_h}{N_h}$, where $h > 0$ is a scaling parameter. In the simulations, we set $h = 45$.

S4 Additional results on joint dynamics of disease, malaria resistance, and human behavior

S4.1 Supplementary figures

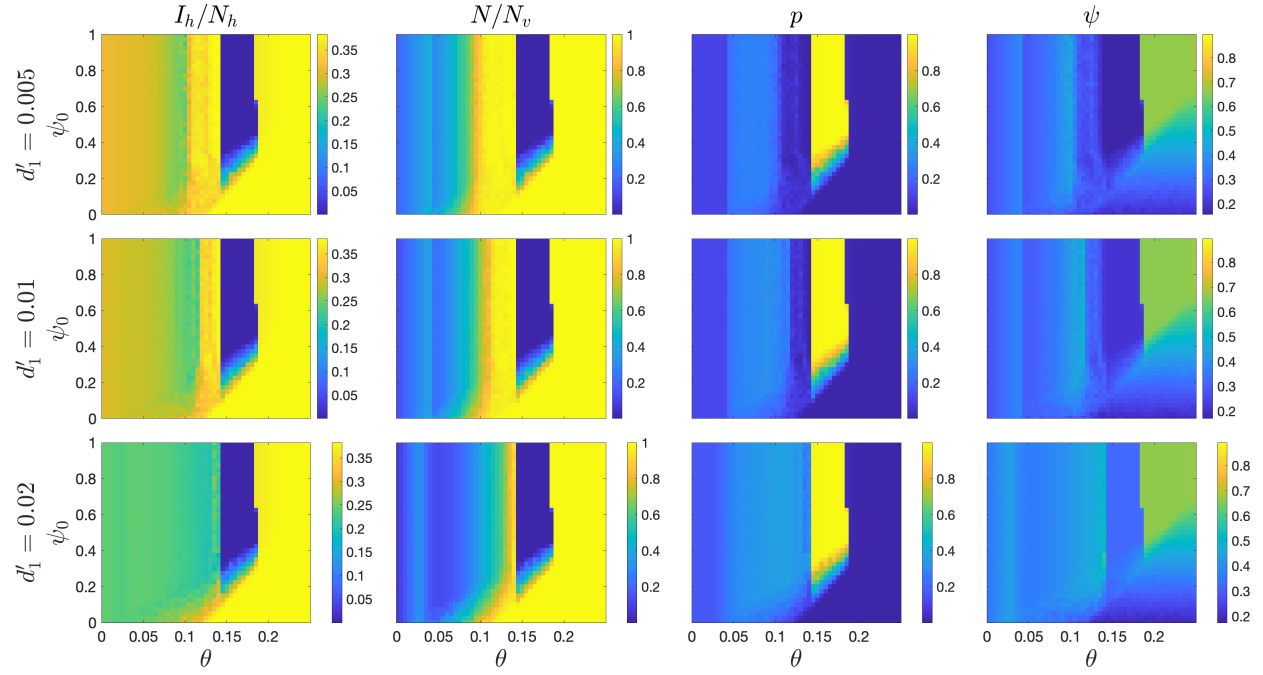


Figure S3: Same as Figure 2 in the main text, but for different values of the intervention-induced mortality of resistant mosquitoes d'_1 .

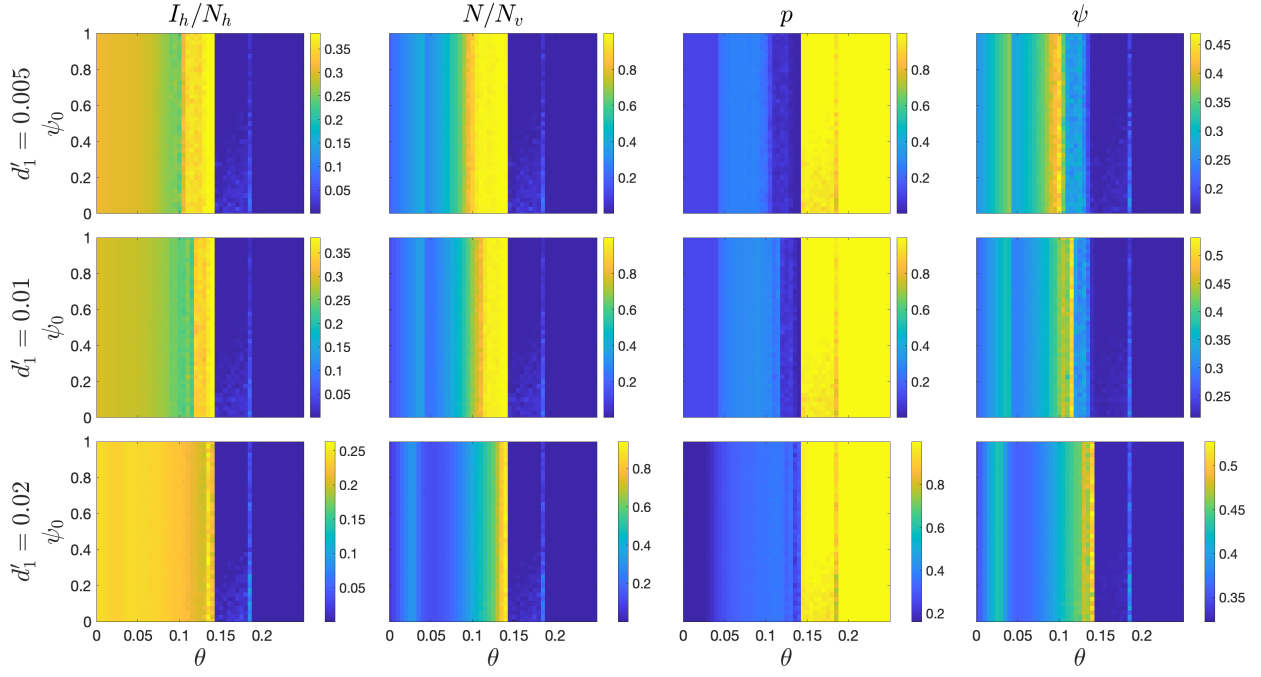


Figure S4: Same as Figure S3, but with a high initial fraction of adopters $p(0) = 0.8$.

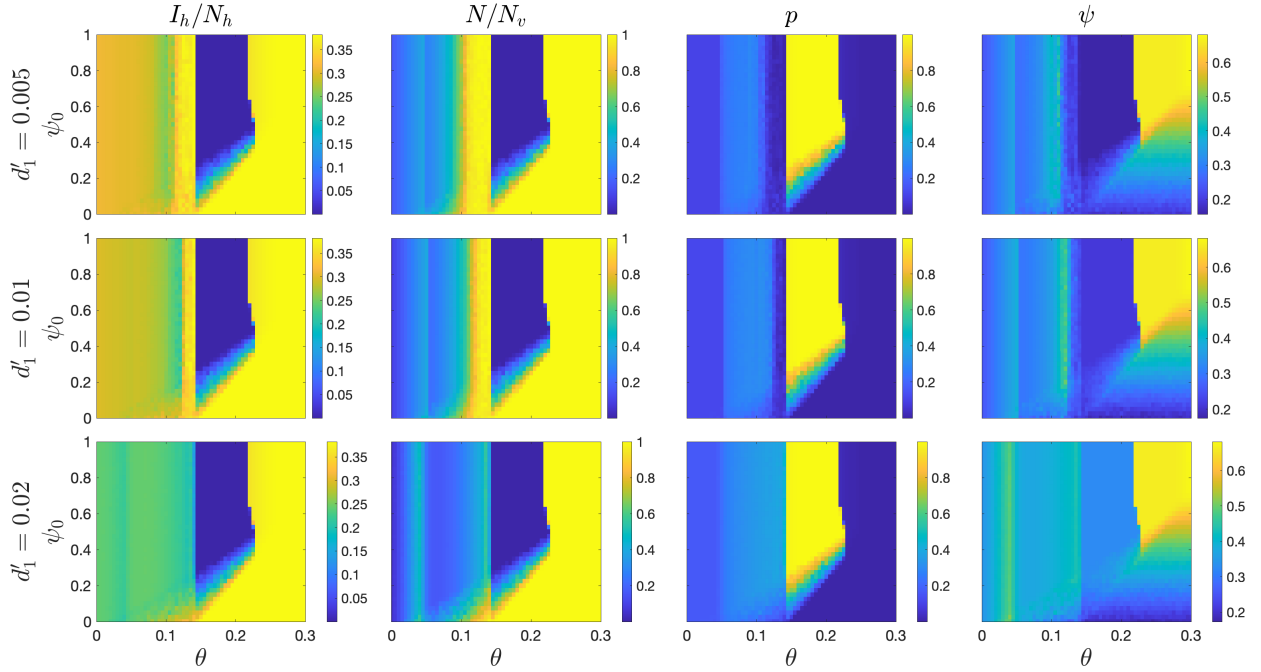


Figure S5: Same as Figure S3, but with perceived infection risk depending on mosquito abundance per human individual N_v/N_h : $R(I_h, N_v, q) = h(1 - \psi q) \cdot \frac{I_h}{N_h} \cdot \frac{N_v}{N_h}$, where $h > 0$ is a scaling parameter. In the simulations, we set $h = 0.2$.

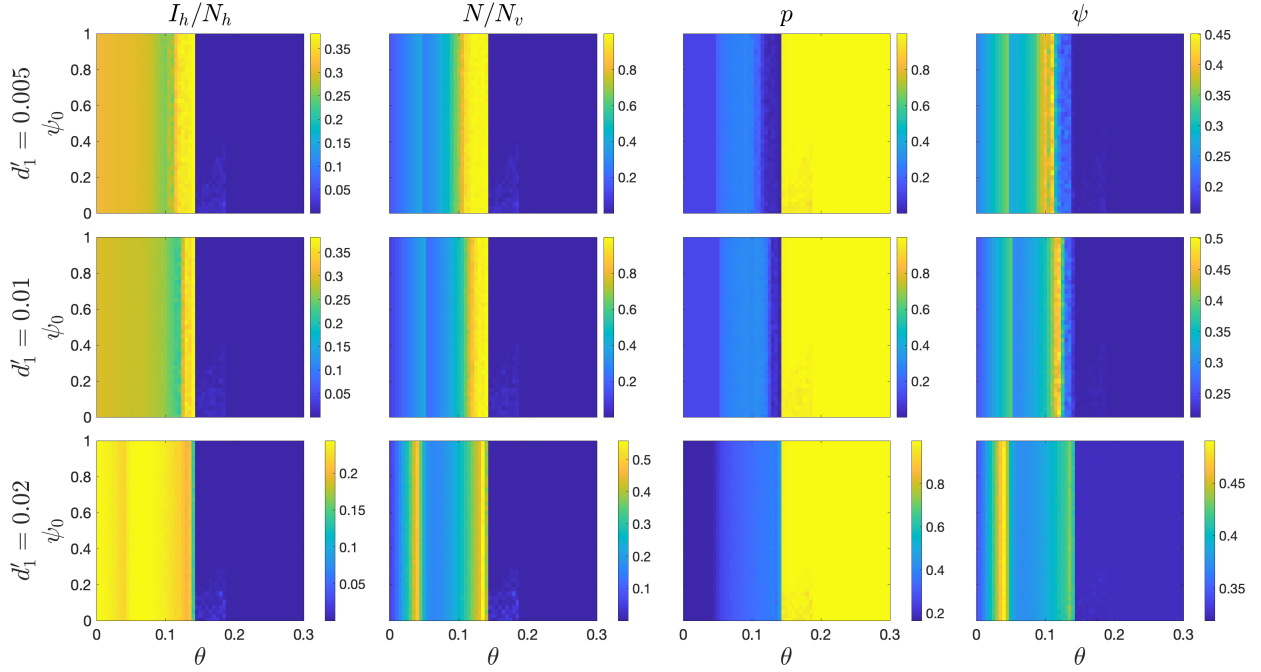


Figure S6: Same as Figure S3, but with a high initial fraction of adopters $p(0) = 0.8$ and with perceived infection risk depending on mosquito abundance per human individual N_v/N_h : $R(I_h, N_v, q) = h(1 - \psi q) \cdot \frac{I_h}{N_h} \cdot \frac{N_v}{N_h}$, where $h > 0$ is a scaling parameter. In the simulations, we set $h = 0.2$.

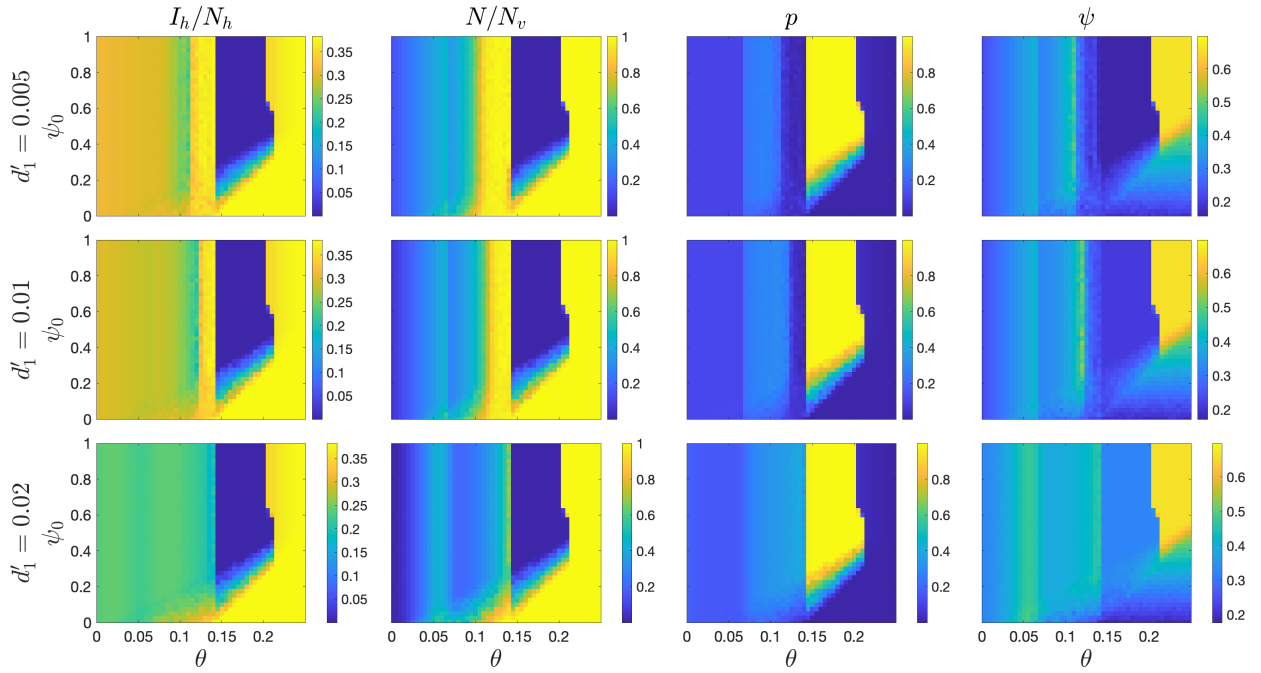


Figure S7: Same as Figure S3, but with perceived infection risk depending on human infection incidence $-\dot{S}_h + \gamma I_h$: $R(-\dot{S}_h + \gamma I_h, q) = h(1 - \psi q) \cdot \frac{-\dot{S}_h + \gamma I_h}{N_h}$, where $h > 0$ is a scaling parameter. In the simulations, we set $h = 45$.

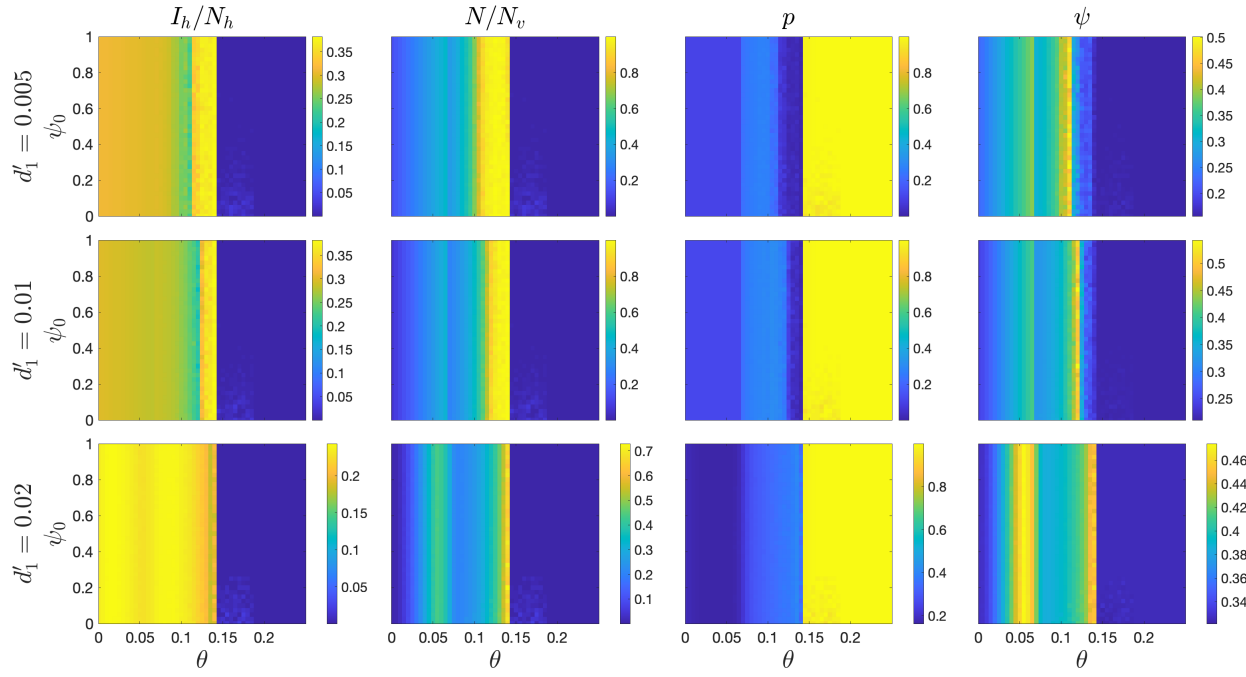


Figure S8: Same as Figure S3, but with a high initial fraction of adopters $p(0) = 0.8$ and with perceived infection risk depending on human infection incidence $-\dot{S}_h + \gamma I_h$: $R(-\dot{S}_h + \gamma I_h, q) = h(1 - \psi q) \cdot \frac{-\dot{S}_h + \gamma I_h}{N_h}$, where $h > 0$ is a scaling parameter. In the simulations, we set $h = 45$.

S4.2 Additional results on the comparison of treated and untreated nets

Here, we provide more details on comparison between treated and untreated nets. To capture untreated nets, we consider a version of the model in which nets protect users from mosquito bites but do not kill mosquitoes, so that $d(p) = d'(p) \equiv d_0$. We assume that the high and low perceived effectiveness of UTNs are the same as those of ITNs, ψ_h and ψ_l , as they are described in Section S1. We make this assumption for two reasons. For one, these extreme values are perceived, derived from indirect observations. As such, ψ_h and ψ_l could be learned from ITNs even though UTNs are employed. The second reason is that setting these parameters the same for both the ITN and UTN models allows for clearer comparisons of their outcomes. Below, we explore the case where ψ_h and ψ_l are derived from the UTNs.

We plot the consequences of the UTN model with numerical solutions in Figure 3 of the main text, which includes time series and the average proportion of infected humans I_h/N_h for varying costs of the nets c . Overall, UTNs outperform ITNs over much of parameter space. The qualitative results can be divided into five intervals. In the first, costs are low and adoption of nets becomes universal. Resistance is not present under UTNs (as it is across parameter space). ITNs reduce the population of mosquitoes relative to UTNs and drives down effectiveness to $\psi \approx 0.2$. In comparison, perceived effectiveness of nets is $\psi \approx 0.65$ in the UTN scenario. Nonetheless, because of the low cost, ITNs are widely adopted and produce a superior outcome relative to UTNs even though they lead to insecticide-resistant mosquitoes. Time series for $c = 0.02$ demonstrate these effects.

As costs increase, however, oscillations are induced in the ITN model. In this second interval, these oscillations lead to worse outcomes for ITNs. Under UTNs, I_h/N_h and ψ slightly increase and decrease, respectively. However, the steady usage of the nets results in substantially fewer infected humans. Under ITNs, ψ varies from approximately 0.3 to 0.55 and infected human and the mosquito populations oscillate. The time series depict results for $c = 0.13$ (Figure 3 of the main text). In the third interval, oscillation begin for UTNs. However, the period is shorter and the duration of net usage longer. As such, though I_h/N_h increases, it is still less than I_h/N_h for the ITN case. As costs increase, I_h/N_h for both models nearly converge. At this point, oscillations for the ITN case end, which sharply increases I_h/N_h and reduces p . This is the fourth qualitative interval. Representative time series are shown for $c = 0.18$ (Figure 3 of the main text; time series for the third interval are not shown). Note that $p \approx 0.05$ for the ITN scenario. In the final interval, net usage is low and there are no oscillations in either case. Over a small interval of c , ITNs result in a marginally lower proportion of infected humans. However, as costs further increase, I_h/N_h converges for both models as fewer and fewer nets are used.

To assess the robustness of these results, we consider a version of the model in which ψ_h and ψ_l are parameterized for UTNs (see Section S1). In this case, $\psi_l^{UTN} = \psi_h^{UTN} < \psi_l^{ITN} < \psi_h^{ITN}$ because UTNs are equally effective against resistant and non-resistant mosquitoes and are less effective than ITNs even against resistant mosquitoes, as ITNs also increase mosquito mortality. The results shown in Figure S9a are consistent with our previous findings: UTNs can outperform ITNs, although over a narrower range of cost parameter values. However, this advantage is not observed under moderate or strong social norms (Figure S9b-c). We also consider the possibility that ITNs impose an additional perceived psychological cost arising from concerns about the potential adverse health effects of insecticides. Specifically, we set the costs of ITNs and UTNs to $c^{ITN} = c + c_a$ and $c^{UTN} = c$, respectively, where c represents the material cost and c_a represents the psychological cost associated with insecticide-related health concerns. Introducing this additional cost widens the range of c values over which UTNs outperform ITNs (Figure S10). Thus, concerns about insecticide exposure may strengthen the relative advantage of UTNs despite their lower biological effectiveness.

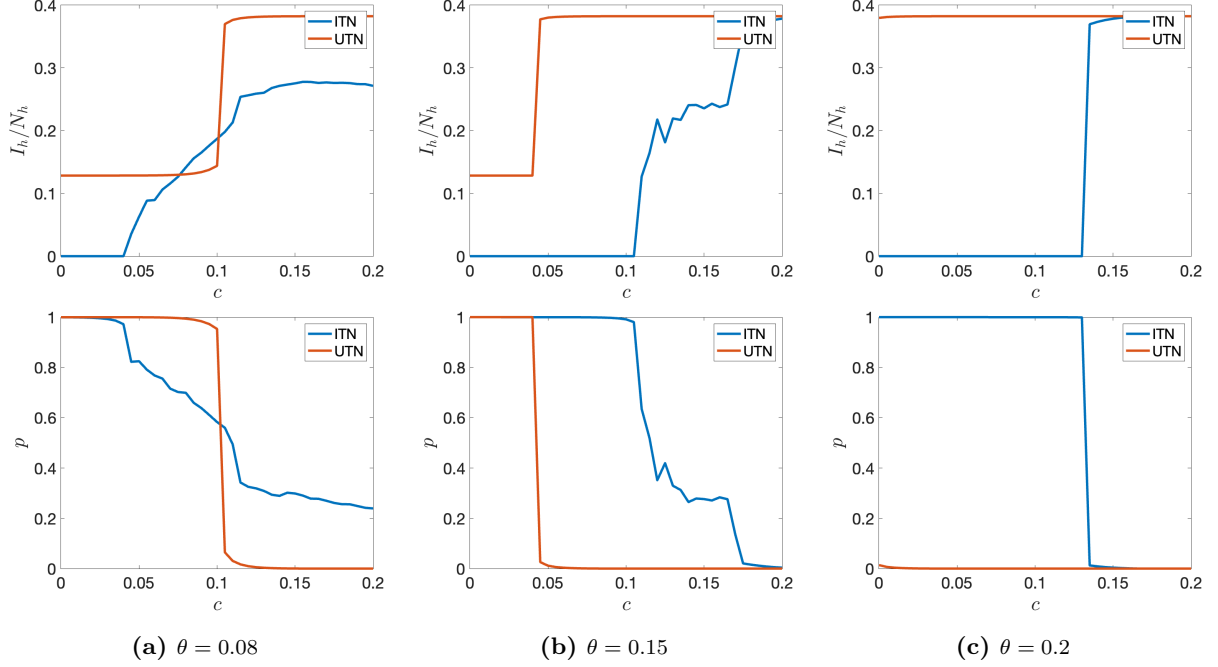


Figure S9: Effects of ITNs compared with UTNs for different values of the strength of norms θ , when ψ_h and ψ_l are parameterized for UTNs (see Section S1). Shown are: the average infection prevalence I_h/N_h (top) and the average fraction of adopters p (bottom). For each parameter combination, results are based on 100 simulations with randomly sampled initial conditions. $\Xi = 0.5$, $D = 0.08$, and all other parameters are fixed at their default values (see Section S1 of the SI).

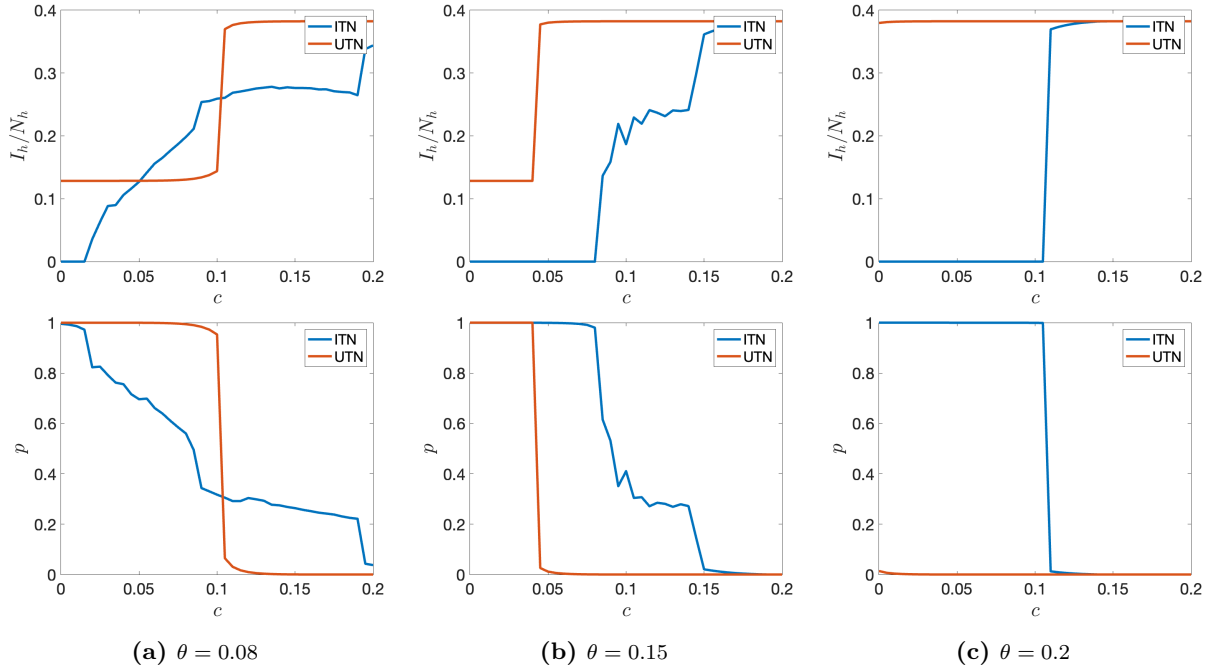


Figure S10: The same as Figure S9, but with additional perceived psychological cost $c_a = 0.25$ arising from concerns about the potential adverse health effects of insecticides.

S5 Additional results on responses to policy and environmental change

S5.1 Supplementary figures

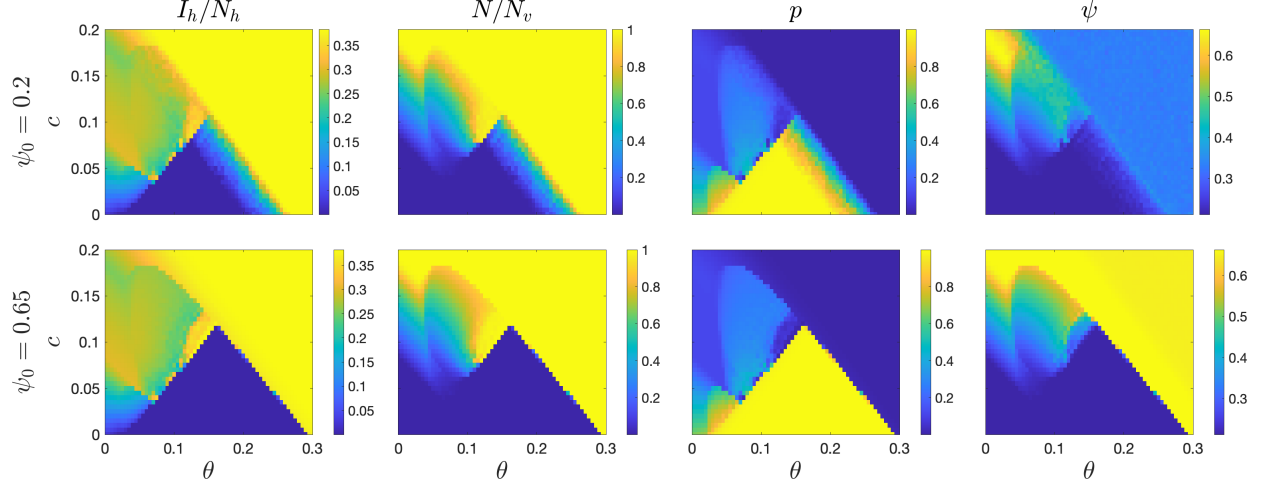


Figure S11: Effects of the strength of social norms θ , the initial perceived effectiveness of protective measures ψ_0 , and adoption costs c , on the long-term behavior of the model, assuming a small initial fraction of adopters $p(0) = 0.05$. Columns show infection prevalence I_h/N_h , the fraction of non-resistant mosquitoes N/N_v , the fraction of individuals using protective measures p , and the perceived effectiveness of protective measures ψ . Rows correspond to different values of ψ_0 . For each parameter combination, outcomes are averaged over 100 simulations with randomly sampled initial conditions. All parameters are fixed at their default values (see Section S1 of the SI for full simulation details).

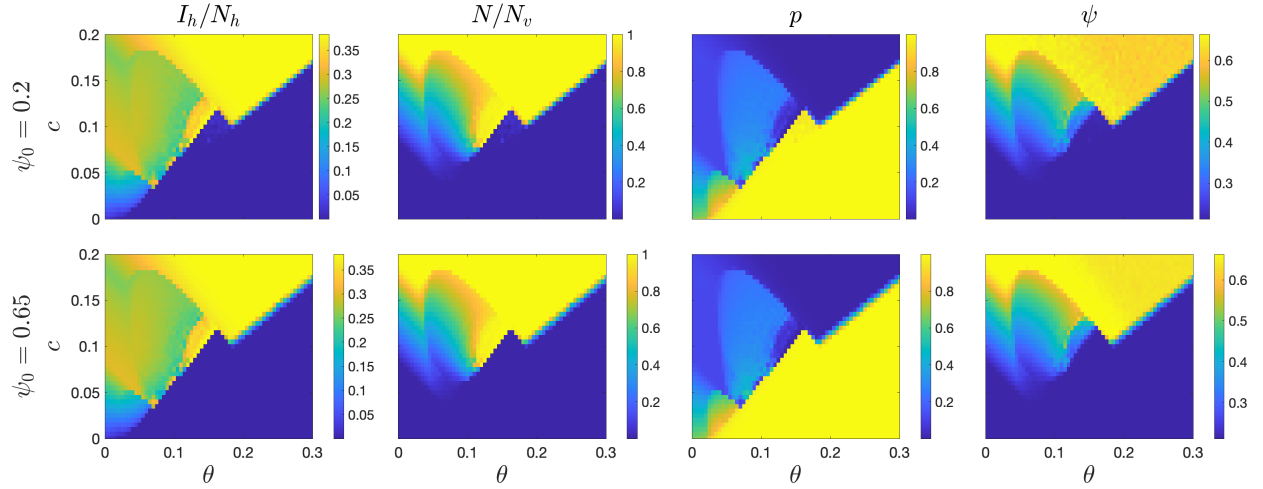


Figure S12: Same as Figure S11, but with a high initial fraction of adopters $p(0) = 0.8$.

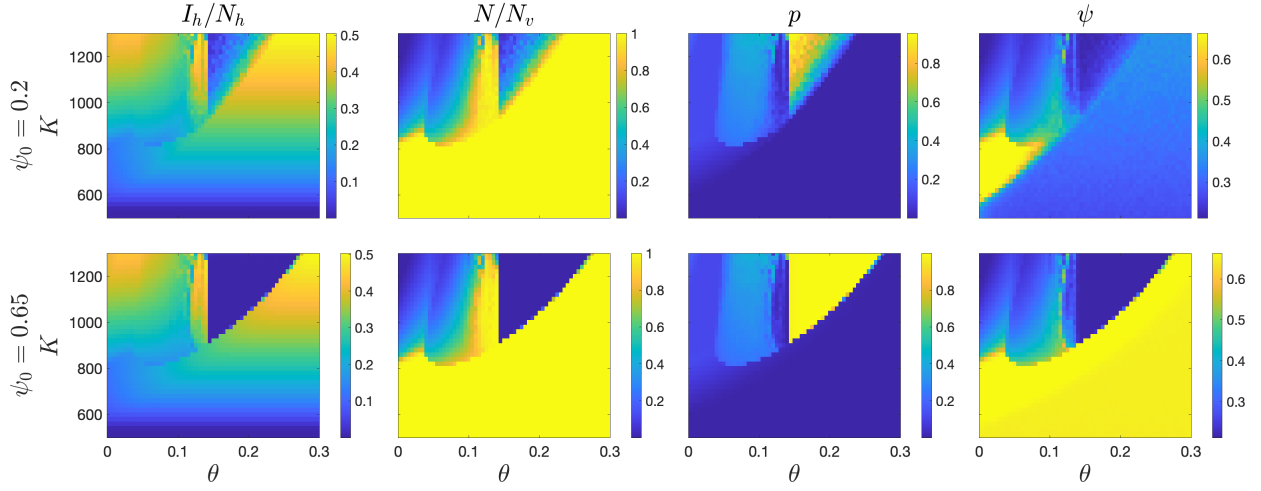


Figure S13: Effects of the strength of social norms θ , the initial perceived effectiveness of protective measures ψ_0 , and carrying capacity K , on the long-term behavior of the model, assuming a small initial fraction of adopters $p(0) = 0.05$. Columns show infection prevalence I_h/N_h , the fraction of non-resistant mosquitoes N/N_v , the fraction of individuals using protective measures p , and the perceived effectiveness of protective measures ψ . Rows correspond to different values of ψ_0 . For each parameter combination, outcomes are averaged over 100 simulations with randomly sampled initial conditions. $c = 0.1$ and other parameters are fixed at their default values (see Section S1 of the SI for full simulation details).

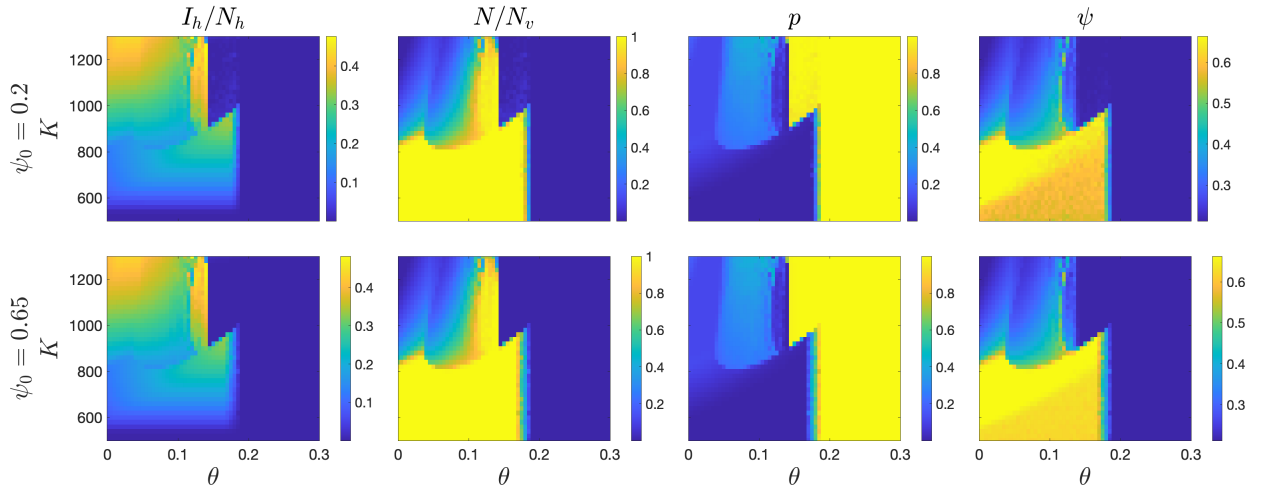


Figure S14: Same as Figure S13, but with a high initial fraction of adopters $p(0) = 0.8$.

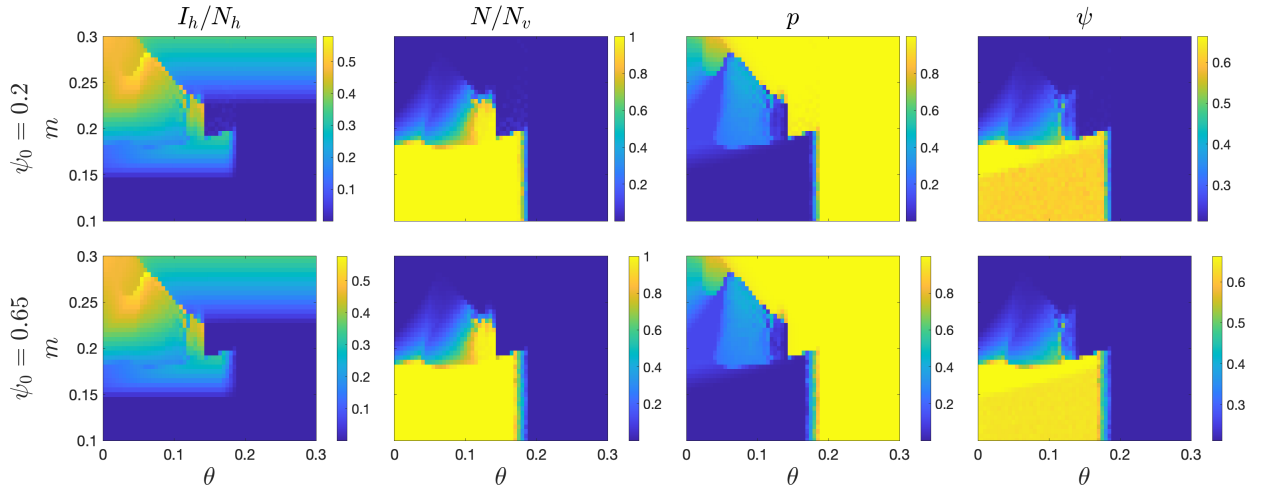
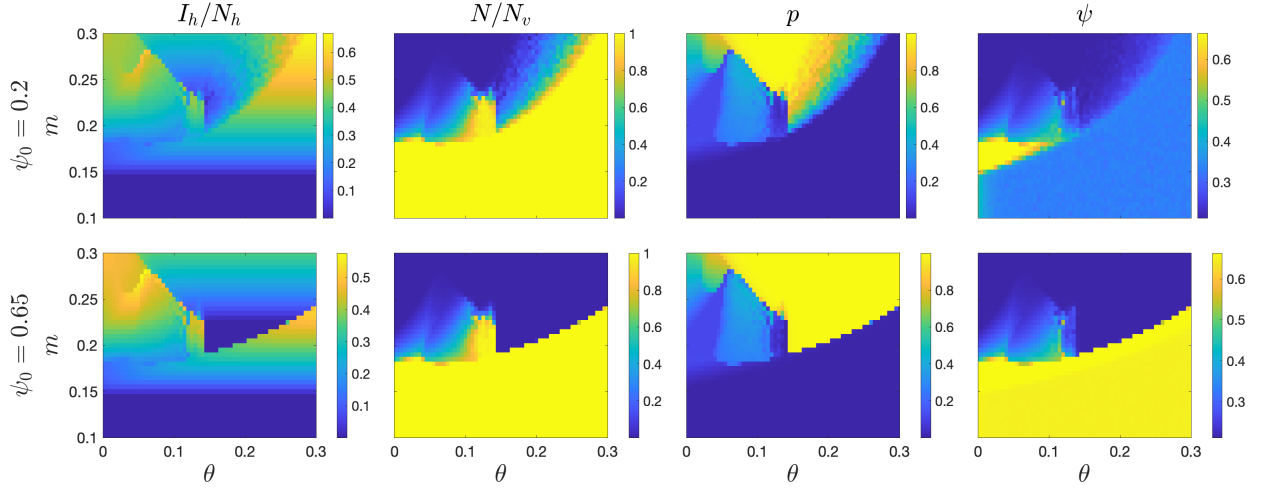


Figure S16: Same as Figure S15, but with a high initial fraction of adopters $p(0) = 0.8$.

S5.2 The effects of authority messaging

Public-health authorities, health-care providers, NGOs, political figures, and community organizations often promote the use of protective measures such as insecticide-treated nets or indoor spraying through media campaigns, outreach, education, and interpersonal communication. The effectiveness of such messaging likely depends on trust: trusted sources may increase adoption, whereas low trust or distrust may weaken official messages or even produce behavioral backfire. Trust may also evolve as individuals compare public-health recommendations with their own beliefs, observations, and experiences.

For simplicity, the main model does not explicitly represent authority messaging. Instead, it captures prior information and public-health campaigns indirectly through the initial perceived effectiveness of protective measures, ψ_0 . Here, we extend the model by incorporating messaging directly into individual decision-making and perceived-effectiveness dynamics, while allowing trust in authorities to evolve over time. This extension allows us to examine how messaging may have different effects across social and cultural contexts.

We assume that messaging can target both individual adoption decisions q and the perceived effectiveness of protective measures ψ . Specifically, messaging promotes adoption and communicates an authority-promoted level of effectiveness denoted by $\psi_g \in [0, 1]$. This value need not coincide with the realized effectiveness of protective measures. Namely, we assume that ψ_g does not necessarily track community-specific information about the dynamically changing composition of resistant and non-resistant mosquitoes, but instead reflects more generic information, for example at the regional or national level. For simplicity, we take ψ_g to be constant over time. Messaging intensity is denoted by $g \in [0, 1]$, with $g = 0$ corresponding to no messaging. Accordingly, each individual's utility function includes an additional term representing the effect of messaging on adoption decisions:

$$u(I_h, p, q) = \underbrace{-(1 - \psi q) \cdot \frac{I_h}{N_h}}_{\text{perceived risk}} - \underbrace{cq}_{\text{adoption costs}} - \underbrace{\theta(q - p)^2}_{\text{social norms}} - \underbrace{\tau g(1 - q)^2}_{\text{authority influence}}.$$

Similarly, the dynamics of perceived effectiveness of protective measures incorporates credibility-weighted authority messaging:

$$\dot{\psi} = \mu \left[\underbrace{p \frac{N_v}{N_h} (w - \psi)}_{\text{outcome-based learning}} + \underbrace{zg \frac{1 + \tau}{2} (\psi_g - \psi)}_{\text{authority messaging}} \right],$$

where $z > 0$ is a scaling parameter and $\tau \in [-1, 1]$ represents trust in the authority. Specifically, $\tau > 0$ corresponds to trust, $\tau \approx 0$ to neutrality or cautious mistrust, and $\tau < 0$ to distrust or reactance (Jennings *et al.*, 2021). Because trust is often associated with compliance (Mandal *et al.*, 2011; Jennings *et al.*, 2021), we assume that trusted messaging encourages individuals to align with the promoted behavior and shifts perceived effectiveness toward the authority-promoted value. Cautious mistrust implies a weak direct behavioral influence of messaging, while still allowing messaging to affect perceived effectiveness through credibility-weighted updating (Lenard, 2008; Jennings *et al.*, 2021). Distrust, by contrast, reflects the belief that the authority is untrustworthy (Jennings *et al.*, 2021) and may generate resentment, protest, or behavioral backfire. In the model, distrust therefore weakens the effect of authority messaging on perceived effectiveness and can reverse its effect on adoption decisions.

Finally, we assume that trust changes dynamically under two forces. First, trust returns at

rate α_2 toward a baseline community-, regional-, or country-specific level τ_0 , reflecting relatively stable institutional, cultural, or historical factors. Second, trust changes as individuals compare the authority-promoted effectiveness level ψ_g with their current perceived effectiveness ψ : trust increases when the two are consistent and decreases when they diverge:

$$\dot{\tau} = \alpha_1 g(\rho(\psi, \psi_g) - \tau) + \alpha_2(\tau_0 - \tau),$$

where $\alpha_1, \alpha_2 \geq 0$ are trust-updating rates. The function

$$\rho(\psi, \psi_g) = 2e^{-a(\psi - \psi_g)^2} - 1$$

maps the discrepancy between perceived and authority-promoted effectiveness to $[-1, 1]$: when $\psi \approx \psi_g$, trust is pulled toward 1, whereas large discrepancies pull trust toward -1 . The parameter $a \gg 1$ controls sensitivity to discrepancies: for large a , even small mismatches can reduce trust, while for small a , larger discrepancies are tolerated.

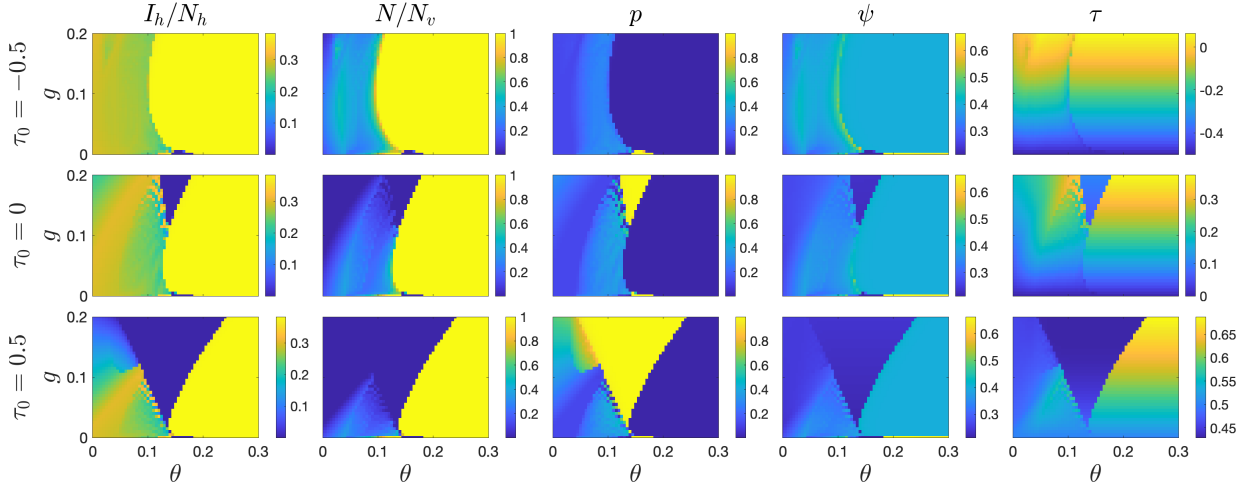


Figure S17: Effects of the strength of social norms θ , the baseline trust τ_0 , and the strength of messaging g on the long-term behavior of the model assuming a small initial fraction of adopters $p(0) = 0.05$. Columns show infection prevalence I_h/N_h , the fraction of non-resistant mosquitoes N/N_v , the fraction of individuals using protective measures p , the perceived effectiveness of protective measures ψ , and trust in authorities τ . Rows correspond to different values of τ_0 . For each parameter combination, outcomes are averaged over 100 simulations with randomly sampled initial conditions. $c = 0.1$, $\psi_g = 0.4$, $\alpha_1 = 0.3$, $\alpha_2 = 0.1$, $z = 5$, $a = 16$, and other parameters are fixed at their default values (see Section S1 of the SI for full simulation details).

Results. We examine how messaging content ψ_g and intensity g shape disease transmission, human behavior, and mosquito ecology across socio-political contexts, represented by the strength of social norms θ and baseline trust τ_0 .

In the context of baseline trust ($\tau_0 > 0$), increasing messaging intensity g generally expands the range of social-norm strengths θ for which the system converges to a disease-free full-adoption equilibrium, extending this regime beyond moderate norm strength. Specifically, messaging can trigger tipping-point dynamics toward disease elimination in populations with strong norms when it communicates high effectiveness of protective measures ψ_g (Figure S18), and in populations with relatively weak norms when it communicates moderate effectiveness (Figure S18). In the former case, messaging triggers an initial increase in the fraction of adopters p , which is then reinforced and stabilized by strong norms. In the latter case, a more conservative assessment of protective-measure effectiveness can remain more consistent with realized effectiveness under

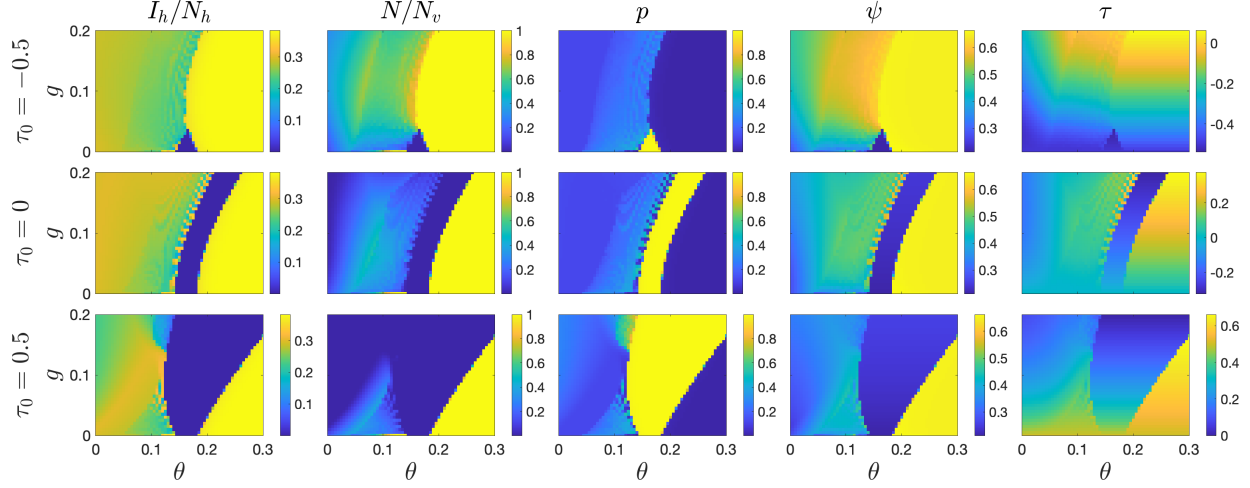


Figure S18: Same as Figure S17, but with a higher authority-promoted level of effectiveness $\psi_g = 0.65$.

resistance, thereby sustaining trust and motivating more behaviorally flexible individuals to adopt protective measures.

In the context of cautious mistrust ($\tau_0 \approx 0$), disease-free outcomes can still occur, but only under more restricted combinations of messaging content ψ_g , messaging intensity g , and social-norm strength θ . When messaging promotes moderate effectiveness, disease elimination is observed under moderate norms and only when messaging intensity is either very low or high, and disappears at intermediate values of g , suggesting a non-monotonic effect of messaging intensity (Figure S17). When messaging instead promotes high effectiveness, disease elimination occurs only within a narrow region of the (θ, g) parameter space, with higher values of g shifting the elimination-supporting range of θ toward stronger norms (Figure S18). Thus, appropriately chosen messaging intensity can lead to disease elimination in populations with moderate or strong norms. However, higher messaging intensity can also reduce trust, because highly optimistic messages may become inconsistent with perceived effectiveness as resistance spreads.

In the context of baseline distrust ($\tau_0 < 0$), increasing messaging intensity g eliminates the disease-free regime, suggesting that the absence of authority messaging is more effective under these conditions (Figures S17-S18).

Finally, coexistence between resistant and non-resistant mosquitoes is a common ecological outcome under authority messaging typically observed under relatively weak norms. It disappears only in the context of baseline trust and high messaging intensity, when resistant mosquitoes take over completely.

S6 Details on the empirical analysis

S6.1 Data sources

Socio-economic variables. The main data source for the socio-economic variables are the Demographic and Health Surveys (DHS) rolled in 2009, 2011, 2013, 2016, 2019, and 2022; we exclude earlier waves of the DHS survey due to the different sampling frames⁷. We obtained the location of each populated place (“cluster”) from which households were selected, although the exact location is randomly displaced within 5 km for the rural areas and 2 km for the urban⁸. The 2009 and 2011 waves use the same populated places (“clusters”), but different households sampled each year. The surveys were collected via a non-self-weighting multi-stage design, with the probabilities of each household and member of the household included in the DHS dataset reported (DHS: https://dhsprogram.com/data/Guide-to-DHS-Statistics/Analyzing_DHS_Data.htm. Accessed September 19, 2026). The DHS data provide extensive information on the household composition and demographics, knowledge of malaria causes, and the defensive measures against mosquitoes.

To model individual behaviors, we use the women’s responses in the DHS datasets. The reason is that these are the only datasets that report information in all survey waves on the respondents’ religion; none of the other DHS surveys for Liberia provide that information in all waves. We supplement the women’s information with data from the household-level surveys. In the main analysis, we proxy the strength of social norms by the degree of religious fractionalization in a populated place⁹. We use the share of different religions in a populated place, adjusted by the probability of selection into the sample, to construct a Herfindahl-Hirschman Index (HHI), which is given by

$$HHI = share_1^2 + share_2^2 + share_3^2 \dots$$

Thus, for more diverse communities (lots of small shares), the HHI will be small. For more homogeneous communities, the HHI will be larger. To ease interpretation, we discretize the variable into 3 categories: low HHI (HHI between 0.4 and 0.7), medium HHI (HHI between 0.7 and 1), and high HHI (HHI=1). We choose these values to create bins with the same width for the low and medium HHI groups, based on the distribution of the HHI variable in the sample¹⁰. This approach results in bins of comparable sizes (38, 65, and 47, respectively, for the DHS 2009 wave).

Malarial burden. The datasets also provide information on the malarial burden within a household; those data are based on self-reports in the 2013 and 2019 waves and self-reports, rapid diagnostic tests, and blood work for select households in the 2009, 2011, 2016, and 2022 waves. However, we did not use these in the analysis as they were available for only a few years and did not allow us to construct a balanced panel; instead, we constructed a cluster-level panel dataset using the modeled malaria incidence data available at 5 km resolution from the Malaria Atlas (https://data.malariaatlas.org/maps?layers=Malaria:202608_Global_Pf_Parasite_Rate&extent=-3654018.955087038,-751600.3885688547,1355358.1306102718,1725483.2107509763. Accessed September 15, 2026). The data capture the annual malaria incidence due to *Plasmodium falciparum* at 5 km resolution.

Mosquito resistance. The resistance data come from [Hancock *et al.* \(2022\)](#). The data come from annual predictive layers based on Bayesian analysis and data from gene mutations in

⁷The surveys collected after 2008 use the same sampling frame (World Bank: <https://microdata.worldbank.org/catalog/2165/study-description>. Accessed Oct. 1, 2026).

⁸About 1% of the rural clusters are randomly displaced within 10 km of the surveyed location.

⁹We use “populated place” to refer to a geographic entity and “community” to indicate a group of people or households with shared norms. Several communities may be nested in the same populated place.

¹⁰Note that the minimum HHI value is 0.4.

Anopheles gambiae, An. coluzzi, and An. arabiensis in 2418 specimen.¹¹ The data are available at approximately 4.5 km resolution annually for 2005-2017 for select countries in Africa, including Liberia. We focus on the resistance to deltamethrin and permethrin as these are the insecticides that have been well-documented to have fostered resistance in Liberia (Temu *et al.*, 2012). We consider the resistance to the two insecticides separately.

Processing geospatial information. As the DHS surveyed clusters are spatially explicit for all years since 2009, we supplement the individual- and household-level data with geospatial data from the Malaria Atlas as well as data on mosquito resistance. The rest of the geospatial variables at the cluster level come from the DHS geospatial data for a given year. To obtain the values per DHS cluster, we use value of the resistance data tile that overlaps the DHS cluster.

S6.2 Country context

Malaria burden in Liberia. Liberia is one of the countries with very high burdens of malaria due to *P. falciparum* (World Health Organization (WHO) 2023: <https://www.afro.who.int/sites/default/files/2023-08/Liberia.pdf>. Accessed Oct. 1, 2026). For example, in 2021 there were 1.9 million cases, corresponding to an incidence rate of 358.5 cases and 3,548 deaths. About 70% of the population in 2021 possessed Insecticide Treated Nets (ITNs), with about 50% of the population using them (WHO 2023). As of 2021, the country was not on track to meet malaria reduction targets (WHO 2023). The DHS data indicate that 98% of the individuals across the 2009 and 2011 waves (for which the data are available) have heard of malaria. About 84% name mosquitoes as the main cause; 18% also list dirty water as a reason, 35%-dirty surroundings, 3% certain foods, and 7% reported they did not know.

Methods for mosquito control in Liberia. The most common method for malaria control among households in the use of insecticide treated nets. For example, 78% of the sampled households in 2009 and 2011 report sleeping under a net as a way to avoid malaria; about 44% of respondents report seeing or hearing messages for the importance of sleeping under a net. Deltamethrin, permethrin, and alphacypermethrin have been the most common insecticides used in long-lasting insecticide treated nets in Liberia (Stebbins *et al.*, 2018). However, their use has also caused widespread resistance (Hancock *et al.*, 2022; Oxborough, 2016). In the analysis, we focus on permethrin and deltamethrin as they represent two different pyrethroid classes; alphacypermethrin belongs to the same group as deltamethrin (Breckenridge *et al.*, 2009; Hodosan *et al.*, 2023).

Most of the nets reported in the data are treated: For example, in the 2009 DHS wave only 46 out of the 1,534 individuals in the sample who reported sleeping under a net used an untreated one; the individuals using untreated nets are scattered in multiple populated places. Thirty-three of those are in urban areas. Using the 2009 DHS wave, we present descriptive statistics for the mean cluster-level net use by the degree of religious homogeneity (Table S2). The results suggest that the medium and low religious homogeneity is associated with lower mean levels of net use, relative to the highest religious homogeneity. However, the net use is not perfect: there is almost a bimodal distribution for the highest HHI (Fig. S19).

Indoor Residual Spraying (IRS) was available to households up to 2016, the program was discontinued by 2016 due to the very high resistance to the pyrethroids and DDT (Oxborough, 2016). Further, the DHS data indicate very few households relying on IRS: While at least 80% of households need to spray their homes, in order for IRS to get rid of mosquitoes in a community (Source: <https://www.cdc.gov/malaria/php/public-health-strategy/irs-strategies.html>. Accessed July 28, 2026), up to 11% of the surveyed households in the DHS between 2011 and 2016 used IRS.

¹¹Note, that the spatial resistance data are based on modeling including ITNs as a covariate. We control for this in the estimation.

Variable	N	Mean (sd)	Min	Max
HHI Group 1 (lowest religious homogeneity)	38	.340633 (.1737995)	.1304348	.7586207
HHI group 2 (medium religious homogeneity)	65	.3430823 (.2219605)	0	0.9142857
HHI group 3 (highest religious homogeneity)	47	.4155073 (.2119985)	.047619	.8518519

Table S2: Breakdown of the net use at the 2009 DHS cluster level by social norms, proxied by the HHI index, with group 1 indicating the lowest norms and group 3-the highest. Net use is based on whether the respondent slept under a net the night before the DHS survey.

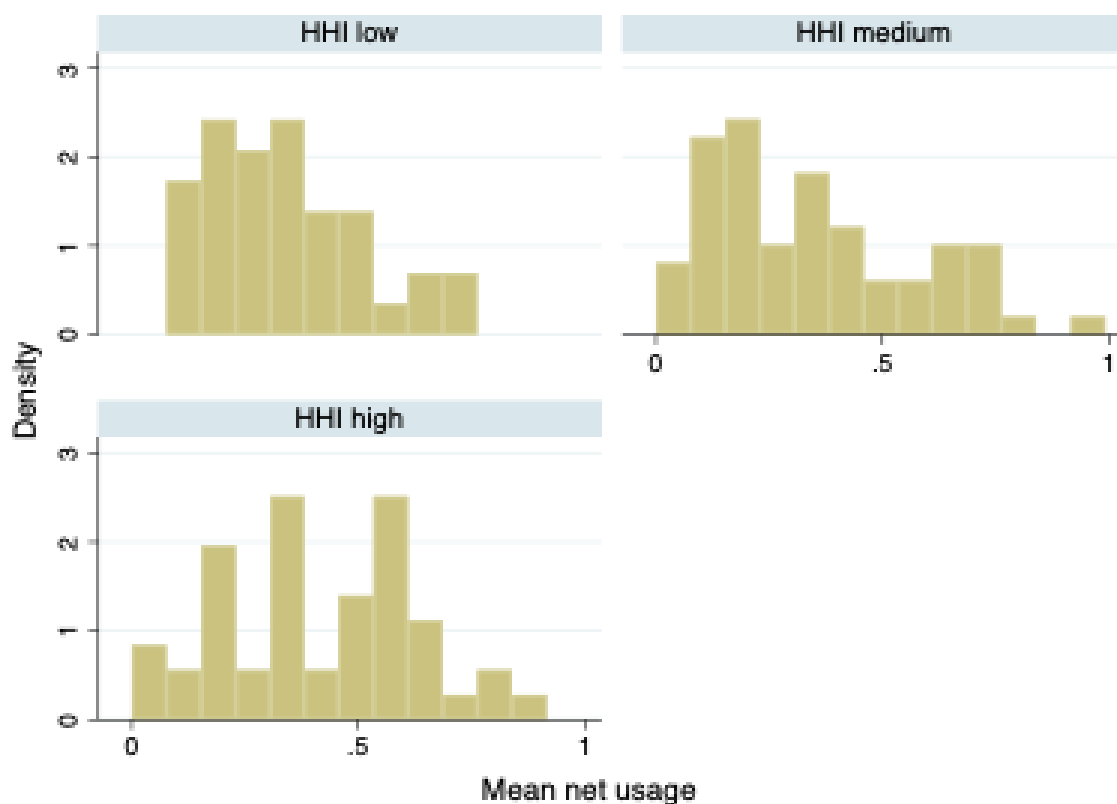


Figure S19: Histogram of the mean cluster-level net use by the degree of religious homogeneity based on the 2009 DHS clusters

The proportion of households using it dropped to less than 1% in 2016 (Table S3). Because of the very low uptake, we exclude IRS from the data analysis. Other defensive behaviors households in the 2009 and 2011 DHS samples reported were using mosquito coils (10% of respondents), keeping windows closed (7% of respondents), using repellents (1% of respondents), cutting the grass (6% of respondents), and keeping surroundings clean (50% of respondents).

Survey Year	N (households)	% Yes (sprayed)	% No	% Don't know
2011	4,162	10.55%	88.40%	1.06%
2013	9,333	10.10%	89.52%	0.36%*
2016	4,216	0.83%	98.79%	0.38%

*Note: 0.01% households had missing values/not stated; these are excluded from the table

Table S3: Share of households reporting IRS use, by survey year for the years in which the question was included in the DHS questionnaires for Liberia. The numbers below are sample-level summaries for the DHS sample (not adjusted to the population).

S6.3 Analytical approach

We test empirically three relationships: (1) social norms and the adoption of nets; (2) social norms and the malarial burden; and (3) social norms and mosquito resistance. Because of data limitations, none of the results reported below are causal; instead, they represent correlations, conditional on controlling for other factors.

S6.3.1 Social norms and bed-net adoption

Estimation. Based on Eq. 7 in the main text, the utility of an individual is given by $U = -R(I_h, q) - cq - \theta(q - p)^2$, where $R(I_h, q) = (1 - \psi(t)q) \frac{I_h}{N_h}$. An individual will choose to adopt a net if $U(I_h, p, \theta, q = 1) > U(I_h, p, \theta, q = 0)$. That is, $q^* = \mathbf{1} \left(\psi(t) \frac{I_h}{N_h} > \theta(1 - 2p) + c \right)$, so that adopt if the cost of doing so (social pressure and cost of obtaining net) is less than the perceived benefits (i.e., reduction in risk). Thus, the probability of adoption assuming logistic distribution for the errors is

$$P(q = 1) = \Lambda \left(\underbrace{\psi(t) \frac{I_h}{N_h}}_{\text{perceived risk}} - \underbrace{c}_{\text{cost}} - \underbrace{\theta}_{\text{norm strength}} + \underbrace{2\theta p}_{\text{social norm}} \right) \quad (\text{S30})$$

Let $\pi = I_h/N_h$ denote malaria prevalence. As we do not have good proxies for $\psi(t)$, we exclude it from the model, thereby implicitly assuming its value is 1. Setting $\psi = 1$, the utility of choice $q \in \{0, 1\}$ is

$$u(q) = -(1 - q) \pi - cq - \theta(q - p)^2,$$

so that the utility of adopting a net is $u(1) = -c - \theta(1 - p)^2$ and the utility of not adopting is $u(0) = -\pi - \theta p^2$. An individual adopts a net if the utility from adopting exceeds the utility from not adopting, that is, if

$$\Delta \equiv u(1) - u(0) = \pi - c - \theta + 2\theta p > 0.$$

With logistic errors, the probability of adoption is $\Lambda(k\Delta)$ in Eq. 30, where k is the scale parameter of the best-response function in the main text, and the logit index is

$$k\Delta = k\pi - kc - k\theta + 2k\theta p.$$

Because religious homogeneity is an imperfect proxy for norm strength, we assume $\theta = \theta_0 + \lambda \text{HHI}$, where $\lambda > 0$ (norm strength increases with religious homogeneity) and θ_0 is a constant that captures

baseline conformity unrelated to religious composition. Substituting yields

$$k\Delta = [k\pi - kc - k\theta_0] + (-k\lambda) \text{HHI} + (2k\theta_0)p + (2k\lambda) \text{HHI} \times p. \quad (\text{S31})$$

Eq. (S31) yields testable hypotheses for the logit coefficients. We expect (i) a negative coefficient on HHI, $-k\lambda < 0$, since k and $\lambda > 0$ by assumption;; (ii) a non-negative coefficient on peer adoption p , $2k\theta_0$, which is strictly positive if $\theta_0 > 0$; and (iii) a positive coefficient on their interaction, $2k\lambda > 0$. The marginal effect of HHI on the logit index, $k\lambda(2p - 1)$, is negative when $p < 0.5$ and positive when $p > 0.5$, so that the effect of norm strength changes sign at $p = 0.5$, irrespective of the values for θ_0 and λ .

We rewrite Eq. (S31) above as

$$V_{ni} = x_i\beta + \gamma Z_n + a + \varepsilon_{ni}, \quad (\text{S32})$$

where n indicates a person and i -a choice ($q = 1$ or $q = 0$). V is the indirect utility stemming from a choice. We assume that a person will choose $q = 1$ if the indirect utility from doing so exceeds the indirect utility from choosing $q = 0$ ¹². The constant a captures the unobserved factors for $q = 1$ relative to $q = 0$. The vector x includes the choice-specific factors; Z includes person-specific factors. β and γ are the estimated sets of coefficients. ε_{ni} is an error term, assumed to have a logistic distribution.

We estimate a logit model based on Eq. S32, pooling observations across all DHS waves, with the probability of net adoption being a function of the DHS survey year, the baseline malaria prevalence, strength of norms, and the share of a community using nets. We also include individual- and household-level characteristics like pregnancy, the number of children, level of education, and wealth as these may significantly impact the decision to adopt a net. For example, since pregnant women and young children are more adversely malaria, they may be more likely to use a net. Wealth may impact the household's ability to afford a net, find care if someone gets sick, or substitute for another protective measure if nets are too inconvenient. Because the DHS data on the price of nets contain a lot of missing values, we exclude that variable from the model. In some specifications, we proxy the cost of using a net with the ambient temperature as some respondents indicated high indoor temperature as a reason for not sleeping under a net.

We calculate the strength of social norms, θ , as the share of individuals from the same religion inside a community excluding a person's own religion. Similarly, we calculate the share of nets p adopted in a cluster excluding an individual's own response. Because of the high correlations between the original variables and their interaction, we use their demeaned values in the estimation.¹³ We use the change in malaria prevalence between 2000 and 2005 as the proxy for the malarial burden at the baseline. This variable is calculated at the cluster level. We prefer this as opposed to the different measures of malaria collected from individual households as only some waves contain blood tests.

The DHS datasets contain two proxies for the adoption of nets: net ownership and net use. However, we do not have data on covariates that would impact the decision to own a net and not the decision to use it. For example, while some individuals report a price for the nets in the 2009 DHS wave, there are a large number of missing values. For this reason, we ignore the price variable in the estimation of the logit of having a net. Thus, we estimate two separate probability models to

¹²The probability of choice i over choice j is given by $V_{ni} + \varepsilon_{ni} > V_{nj} + \varepsilon_{nj}$, where the utility is decomposed into an observed part (the indirect utility V) and an idiosyncratic error ε . Thus, *Prob of choice i* = $\int_{\varepsilon} I(\varepsilon_{nj} - \varepsilon_{ni} > V_{nj} - V_{ni}) f(\varepsilon) d\varepsilon_n$ where $f(\varepsilon)$ is an Extreme Value Type I probability density function.

¹³the demeaning changes the expected sign on the coefficient on HHI to be positive and equal to $k\lambda(2p_{bar} - 1)$, when $p_{bar} > 0.5$. The predictions on the coefficients for p and their interaction remain unchanged

model these decisions, instead of a hurdle model, where the first stage is the probability of having a net and the second - the probability of using it, conditional on ownership. The standard errors are clustered the standard errors at the community (cluster) level. Because the data are repeated cross-sections, we did not use cluster fixed effects (FEs) but include a robustness check with cluster-level fixed effects using the 2009 and 2011 DHS waves only. All specifications use survey weights¹⁴.

Based on Eq. S30 and our mathematical model, we hypothesize that higher malaria prevalence would translate into higher net adoption rates. Higher costs, proxied in the net use model, by higher temperature and wealth as these correlate with the inconvenience of sleeping under a net, should lower net use. As described earlier, we expect the coefficient on social norms to be positive when the mean net adoption of nets exceeds 0.5 (due to using the demeaned version of both the HHI and p variables) and the coefficient on the share of net ownership in the community to be negative otherwise. The interaction term between impact of social norms and the prevalence of nets in the community will have an ambiguous impact: If there are strong norms in one's community and high adoption of nets by other members in the religion-based community within a surveyed cluster, then we would expect that probability of an individual's adoption increases. Similarly, if the norms are strong, but most members of the community do not use nets, we expect to have lower adoption of nets. These are the hypotheses we test empirically.

The demographic variables in the models (pregnancy, wealth index, age of the respondent, number of children in the household, location (urban vs. rural), education level, current pregnancy) control for important characteristics the previous literature (Babalola *et al.*, 2016; Chacha *et al.*, 2026) has identified as being significant drivers of adoption. For example, since malaria is more dangerous to pregnant women, we expect a positive coefficient on that variable. Since young children are more vulnerable to malaria, we expect more children will translate into a higher probability of having a net. However, the variable's impact on use is ambiguous: More children in the household may decrease the probability a woman sleeps under a net if there are too few nets or, if children co-sleep with their parents, can increase the probability of a women sleeping under a net. We expect that wealth has a non-constant impact on the probability of having and using a net: The poorest households may not have the means to acquire nets and richer households may have other means for controlling mosquitoes. Thus, we expect the variable to have a positive coefficient for the middle-income households, relative to the poorest households. Older women may be more aware of the drivers of malaria and, thus, be more likely to use a net.

Two important caveats are in order, however: First, the current analysis does not attempt to address Manski's reflection problem or self-sorting related to household choosing where to live (Goldsmith-Pinkham and Imbens, 2013; Manski, 1993). Specifically, we cannot separate 3 potential effects: (1) own responses are dependent on others in the community (this is what we want to test); (2) contextual effects: there may be shared unobservable characteristics that impact both one's own decision as well as the decision of others inside the group; and (3) correlated effects - there may be an unobserved shock that affects individuals and their groups. Second, we cannot separate the effects of a religion itself from the degree of religious homogeneity. For example, the HHI variable captures the degrees of religious homogeneity, with many of the homogeneous communities in our datasets being Christian.

Results. We consider Models 1 to be the main specifications for both owning a net (Table S4) and using it (Table S5). The models are based on Eq. S31 that includes the share of net adoption in a community p and the interaction of the variable with the degree of religious homogeneity,

¹⁴We use a version of the Hausman test to assess whether survey weights are needed. However, the estimates with and without survey weights are very consistent.

θ . The inclusion of both p and $p\theta$ is suggested by Eq. S31 and necessitated due to omitted variable concerns¹⁵. We find that relative to the baseline year (2009), net ownership has increased (Table S4). However, the impact on net usage is not monotonic, with the last wave of the DHS data having lower probabilities of net use (Table S5).

The coefficients on the covariates have the expected signs: Consistent with our expectations, we find that higher malaria incidence results in a positive relationship with the adoption of nets. However, the impact is statistically significant only for the model on net ownership. We find that the respondent's age has a consistent and positive impact on both the probability of having a net and using it. The number of children in the household increase the probability of net ownership but decrease the probability of using it. Pregnancy increases the probability of using a net and has a positive but insignificant coefficient for having a net. Wealth has a non-monotonic relationship with net usage, with the coefficient being positive for the lower wealth levels (relative to the poorest households). However, higher wealth levels indicate lower net use. Wealth has a positive and monotonic impact on the probability of having a net. Rural households have a lower probability of using a net, but a higher probability of having one. One possible explanation for the differences in the observed patterns is that the net usage variable captures the use of the net the night before the survey and may not be representative of longer-term usage.

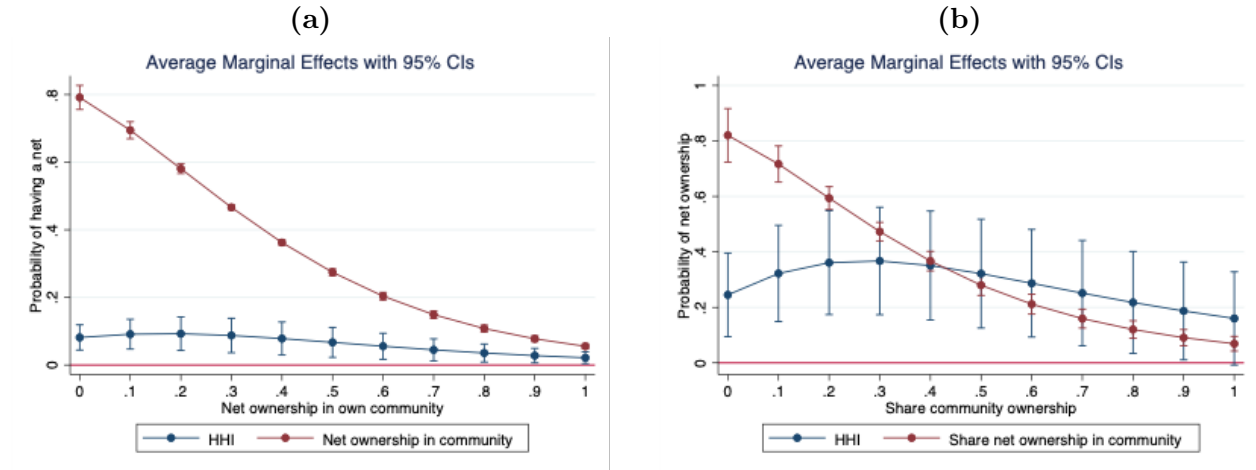


Figure S20: Marginal effects for the probability of having a net: (a) the full sample; (b) the 2009 sample only. The two covariates of interest have been demeaned to reduce the multicollinearity issue. These plots show the interaction between the two.

The coefficients on HHI, share of net ownership, and their interaction have signs consistent with our hypothesis.¹⁶ For example, the coefficient on the demeaned HHI variable (`hhi_demeaned`) and share of nets in an individual's community (`loo_mean_own_ownrel`) are both positive and statistically significant and so is their interaction (Table S4). What these results suggest is that social norms reinforces net possession. The peer effects are strongest for low levels of net ownership and decline monotonically with net ownership in a community (Fig S20). The impact of the social norms first increases and then decreases along the share of net ownership in a community¹⁷ For net use (Table S5), the coefficient on the share of net users in a community is positive and statistically significant. However, the degree of religious homogeneity has a negative but insignificant coefficient.

¹⁵ As a robustness check we provide the estimation results without that p variable. The omission flips the sign on the HHI to positive and the interaction term becomes statistically significant and negative.

¹⁶ note that, since HHI is demeaned in the estimation, the coefficient is $k\lambda(2p_{bar} - 1)$, which positive for $p_{bar} > 0.5$.

¹⁷ Note, the $\beta = \partial \logit(p) / \partial X_i$, with the marginal effects being $\beta \cdot \Lambda(X_i\beta)(1 - \Lambda(X_i\beta))$

Variable	Main model	Rural only	Urban only	m2	m3	m4	m5
dhsyear 2011	0.10** (0.05)	0.01 (0.06)	0.20*** (0.06)	0.10** (0.05)	0.25* (0.14)	0.27* (0.14)	0.11** (0.05)
dhsyear 2013	0.19*** (0.04)	0.22*** (0.07)	0.17*** (0.05)	0.19*** (0.04)	0.49*** (0.12)	0.51*** (0.12)	0.20*** (0.04)
dhsyear 2016	0.22*** (0.05)	0.19** (0.08)	0.24*** (0.06)	0.23*** (0.05)	0.68*** (0.14)	0.69*** (0.13)	0.24*** (0.05)
dhsyear 2019	0.18*** (0.04)	0.17** (0.08)	0.17*** (0.05)	0.17*** (0.04)	0.47*** (0.11)	0.46*** (0.11)	0.20*** (0.04)
dhsyear 2022	0.57*** (0.06)	0.79*** (0.14)	0.51*** (0.07)	—	1.43*** (0.15)	1.41*** (0.15)	0.55*** (0.07)
hhi_demeaned	0.36*** (0.08)	0.30** (0.14)	0.37*** (0.12)	0.38*** (0.09)	0.42** (0.19)	0.65*** (0.21)	—
share net owners in own community	3.55*** (0.09)	3.63*** (0.13)	3.36*** (0.12)	3.47*** (0.09)	—	—	3.50*** (0.09)
hhi_demeaned × share net owners in own community	0.99** (0.47)	1.74** (0.79)	0.28 (0.63)	0.81 (0.52)	-2.95*** (0.98)	—	—
age	0.00** (0.00)	0.00** (0.00)	0.00 (0.00)	0.00** (0.00)	0.00** (0.00)	0.00*** (0.00)	0.00** (0.00)
educ_level = 1	0.13*** (0.05)	0.09* (0.05)	0.16** (0.08)	0.14*** (0.05)	0.19*** (0.05)	0.18*** (0.05)	0.13*** (0.05)
educ_level = 2	0.03 (0.05)	0.04 (0.07)	0.04 (0.07)	0.06 (0.05)	0.09* (0.05)	0.08 (0.05)	0.04 (0.05)
educ_level = 3	0.01 (0.12)	0.21 (0.46)	0.03 (0.13)	0.07 (0.13)	0.10 (0.12)	0.11 (0.12)	0.01 (0.12)
v190 = poorer	0.31*** (0.06)	0.36*** (0.06)	0.04 (0.14)	0.31*** (0.06)	0.43*** (0.07)	0.43*** (0.07)	0.30*** (0.06)
v190 = middle	0.39*** (0.06)	0.44*** (0.08)	0.18 (0.12)	0.41*** (0.07)	0.41*** (0.09)	0.40*** (0.09)	0.39*** (0.06)
v190 = richer	0.48*** (0.07)	0.42*** (0.15)	0.30** (0.12)	0.60*** (0.08)	0.30*** (0.11)	0.28*** (0.10)	0.49*** (0.07)
v190 = richest	0.41*** (0.08)	0.38** (0.15)	0.22* (0.12)	0.56*** (0.09)	0.19 (0.12)	0.17 (0.12)	0.43*** (0.08)
rural	0.25*** (0.04)	—	—	0.23*** (0.04)	0.17* (0.09)	0.17* (0.09)	0.22*** (0.04)
num_kids	0.11*** (0.02)	0.09*** (0.02)	0.13*** (0.03)	0.09*** (0.02)	0.10*** (0.02)	0.10*** (0.02)	0.11*** (0.02)
pregnant	0.01 (0.06)	0.03 (0.07)	0.00 (0.10)	0.05 (0.07)	-0.00 (0.06)	-0.00 (0.06)	0.01 (0.06)
change_malar_incidencep	0.91*** (0.16)	0.37 (0.28)	1.07*** (0.22)	0.05 (0.16)	2.22*** (0.41)	2.24*** (0.41)	0.85*** (0.16)
annual_precipitation_2005	—	—	—	-2.72e-03 (6.28e-04)	—	—	—
maximum_temperature_2005	—	—	—	2.78e-03 (6.39e-04)	2.64e-05 (8.61e-06)	2.77e-05 (8.73e-06)	8.67e-06 (3.53e-06)
Constant	-0.21** (0.10)	-0.00 (0.12)	-0.03 (0.16)	0.19 (0.14)	-0.40** (0.16)	-0.40** (0.16)	-0.19* (0.10)
N	33,241	17,891	15,350	28,891	33,241	33,662	33,251
Pseudo R ²	0.131	0.149	0.113	0.116	0.050	0.048	0.130

Significance levels: ***1%, **5%, *10%.

Table S4: Results from the logit of net ownership as a function of religious homogeneity (standard errors in parentheses). The standard errors are clustered at the cluster level. The two key variables (hhi and net ownership within own group excluding own values) have been demeaned to reduce correlations between them. Note these results for net ownership are missing a price variable.

Since this coefficient is based on the demeaned values for HHI and p , one possible explanation is that the mean p is low. However, the post-estimation margins plots have very consistent patterns (Fig S21)

In addition, we test if thresholds exist for the impact of norms as suggested by Eq. S30. That is, the model predicts that for $p < 0.5$, social norms should lower the probability of owning a net; $p > 0.5$ should increase it¹⁸. The results based on the marginal effects from Model 1 on net

¹⁸Because we estimate the model with demeaned HHI and p , the corresponding predicted switching points and

Variable	[Main Model]	Rural	Urban	[2]	[3]	[4]	[5]
dhsyear 2011	0.37*** (0.09)	0.37*** (0.12)	0.37*** (0.13)	0.37*** (0.09)	0.48*** (0.11)	0.49*** (0.11)	0.37*** (0.09)
dhsyear 2013	-0.00 (0.07)	0.03 (0.09)	-0.02 (0.12)	-0.00 (0.07)	0.09 (0.09)	0.11 (0.09)	-0.00 (0.07)
dhsyear 2016	0.11 (0.09)	0.25** (0.12)	0.02 (0.13)	0.10 (0.09)	0.28** (0.11)	0.28** (0.11)	0.10 (0.09)
dhsyear 2019	0.42*** (0.08)	0.45*** (0.09)	0.40*** (0.12)	0.42*** (0.08)	0.59*** (0.10)	0.59*** (0.10)	0.41*** (0.08)
dhsyear 2022	-0.19** (0.08)	-0.21** (0.10)	-0.17 (0.12)	—	0.08 (0.11)	0.09 (0.11)	-0.17** (0.08)
hhi_demeaned	-0.10 (0.14)	-0.29 (0.19)	-0.01 (0.20)	0.03 (0.16)	0.02 (0.18)	0.03 (0.18)	—
loo_mean_net_c	1.74*** (0.12)	1.76*** (0.16)	1.73*** (0.18)	1.71*** (0.13)	—	—	1.74*** (0.12)
hhi_demeaned loo_mean_net_c × age	-0.12 (0.66)	0.43 (0.91)	-0.12 (0.96)	-0.08 (0.71)	-1.59* (0.84)	—	—
educ_level = 1	0.03*** (0.00)	0.04*** (0.00)	0.03*** (0.00)	0.03*** (0.00)	0.03*** (0.00)	0.03*** (0.00)	0.03*** (0.00)
educ_level = 2	-0.13** (0.06)	-0.14** (0.06)	-0.07 (0.11)	-0.19*** (0.06)	-0.12** (0.06)	-0.12** (0.06)	-0.13** (0.06)
educ_level = 3	0.02 (0.06)	-0.09 (0.09)	0.09 (0.09)	0.02 (0.07)	0.03 (0.06)	0.03 (0.06)	0.02 (0.06)
v190 = poorer	-0.12 (0.13)	-0.65** (0.29)	-0.03 (0.14)	-0.22* (0.13)	-0.14 (0.13)	-0.13 (0.13)	-0.13 (0.13)
v190 = middle	0.12* (0.07)	0.16** (0.07)	-0.02 (0.17)	0.15** (0.07)	0.16** (0.07)	0.16** (0.07)	0.12* (0.07)
v190 = richer	0.01 (0.07)	-0.04 (0.08)	0.09 (0.17)	0.09 (0.09)	-0.01 (0.08)	-0.02 (0.08)	0.01 (0.07)
v190 = richest	-0.19** (0.10)	-0.31* (0.17)	-0.13 (0.18)	-0.20* (0.11)	-0.32*** (0.10)	-0.33*** (0.10)	-0.20** (0.10)
rural	-0.60*** (0.10)	-0.44** (0.19)	-0.58*** (0.18)	-0.63*** (0.12)	-0.74*** (0.11)	-0.76*** (0.11)	-0.62*** (0.10)
num_kids	-0.21*** (0.06)	—	—	-0.15** (0.07)	-0.18** (0.07)	-0.18** (0.07)	-0.19*** (0.06)
pregnant	-0.06*** (0.02)	-0.07*** (0.02)	-0.04 (0.03)	-0.05** (0.02)	-0.06*** (0.02)	-0.06*** (0.02)	-0.06*** (0.02)
Percent change_malaria_incidence annual_precipitation_2005	0.18** (0.08)	0.21** (0.09)	0.17 (0.13)	0.15* (0.09)	0.18** (0.08)	0.18** (0.08)	0.18** (0.08)
maximum_temperature_2005	0.03 (0.27)	0.33 (0.35)	0.02 (0.41)	0.07 (0.40)	0.82** (0.33)	0.85*** (0.32)	0.08 (0.27)
Constant	—	—	—	-8.72e-04 (1.30e-03)	—	—	—
N	—	—	—	8.75e-04 (1.32e-03)	2.47e-06 (7.86e-06)	1.95e-06 (7.83e-06)	-6.59e-06 (6.40e-06)
Pseudo R ²	0.05 (0.13)	-0.28** (0.14)	0.06 (0.23)	0.16 (0.25)	0.10 (0.15)	0.10 (0.15)	0.03 (0.13)
	21,339	11,873	9,466	17,878	21,339	21,605	21,344
	0.061	0.064	0.057	0.063	0.044	0.044	0.061

Significance levels: ***1%, **5%, *10%

Table S5: Results from the logit of net use as a function of religious homogeneity (standard errors in parentheses). The standard errors are clustered at the cluster level. The two key variables (hhi & low_mean_net) have been demeaned to reduce correlations between them.

ownership indicate that for this particular dataset the threshold occurs around $p = 0.23$ (Fig. S22). This threshold value may indicate that net use becomes sufficiently visible even when fewer than half of the population adopt nets, or that adopters are more active in encouraging net use than non-adopters are in discouraging it. However, we do not have the data to test these.

Robustness checks. In addition to assessing the robustness of the results to different model specifications and outcome variables used, we also run tests to address three potential concerns: the omission of cluster fixed effects that control for unobserved location-specific characteristics, the omission of the price of nets, and the mass distribution of nets in 2018 (<https://lcm.org.lr/liberia->

coefficient predictions are unchanged. That is $k\lambda(2\bar{p} - 1)$ is positive when the mean peer adoption $\bar{p}$ exceeds 0.5, $2k(\theta_0 + \lambda \overline{\text{HHI}}) > 0$ on p , and $2k\lambda > 0$ on the interaction.

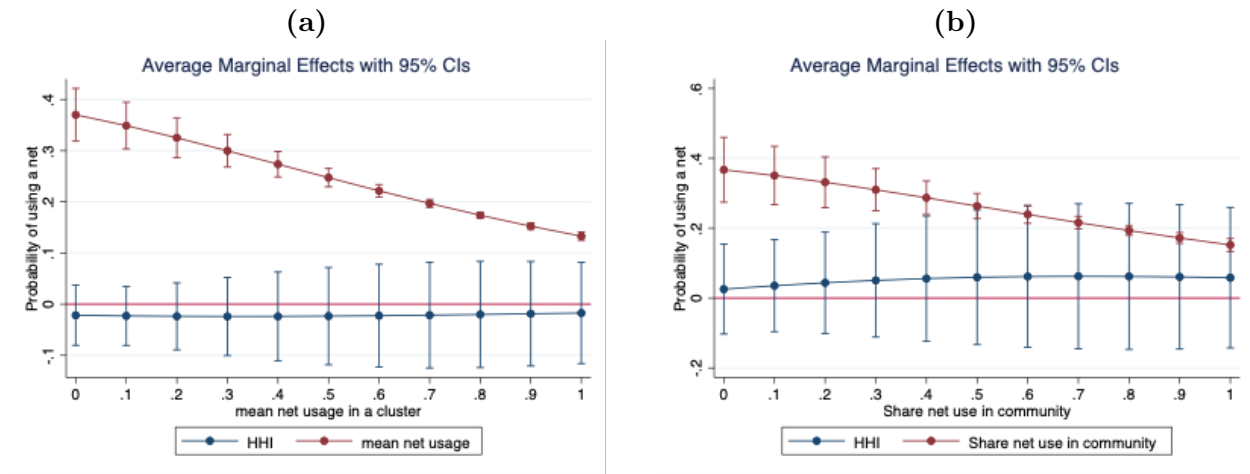


Figure S21: Marginal effects for the impact of HHI and net usage in a cluster on an individual's probability of using a net based on the main model: (a) the full sample; (b) the 2009 sample only. The results indicate the probability decreases with the share of other net users in a community, holding everything else constant. These plots show the interaction between the two.

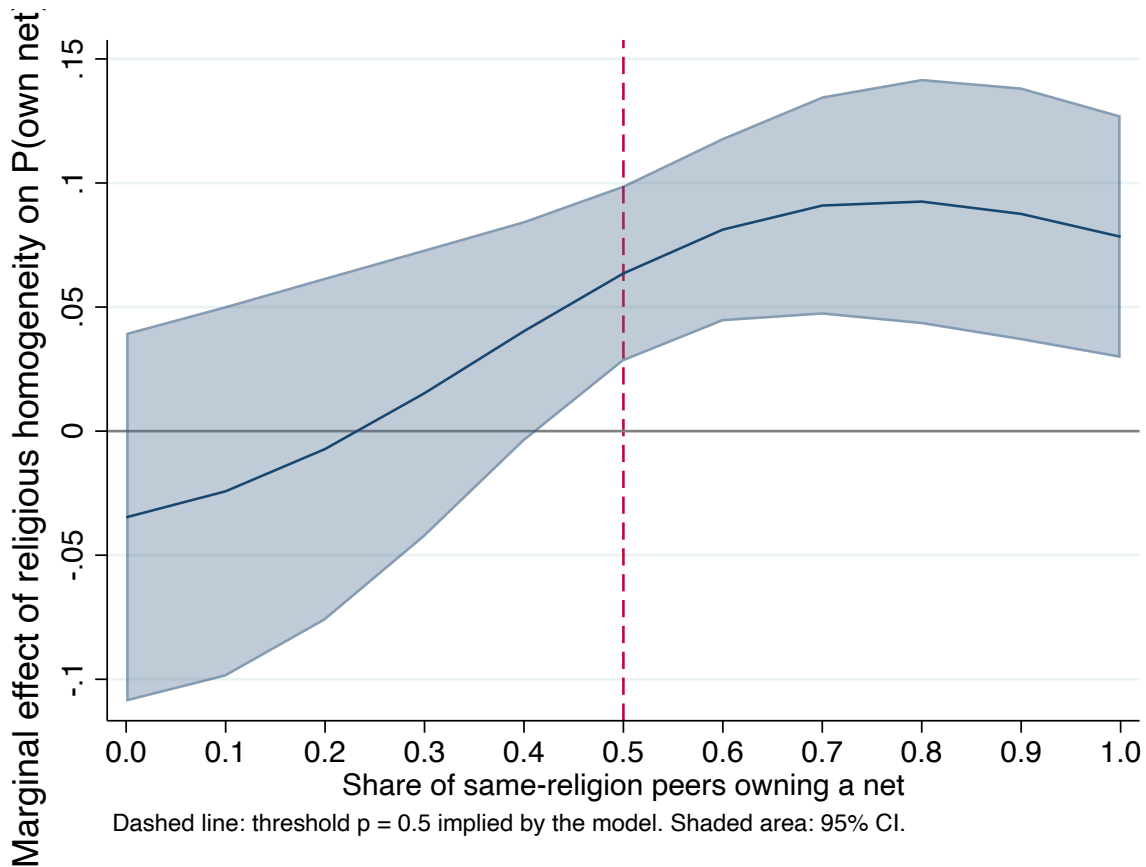


Figure S22: Estimated critical value of p that determines the sign of the marginal effect of HHI on the probability of net adoption. The figure suggests that HHI have a negative impact when $p < 0.23$ and positive when $p > 0.23$.

commences-nationwide-mosquito-net-distribution/). The omission of the price of nets in the probability of net adoption in a logit model raises concerns of an omitted variable bias, even when the

omitted variable is uncorrelated with the other covariates. This may lead to a downward bias in a logit model. For this reason, we also estimate a linear probability model using the same covariates as above. Lastly, the mass distribution of nets is likely to increase the probability that a household has a net. For this reason, we conduct a robustness check using just the data for 2009. The results from the three robustness checks are consistent with the main models¹⁹.

S6.3.2 Social norms and community-level malaria burden

Estimation. The main theoretical models focus on malaria prevalence, whereas we have only data on the malaria incidence. However, the theoretical results for malaria incidence are very consistent (Figures S7-S8). The theoretical model predicts that stronger norms reinforce the prevailing behavior: They should push net adoption toward full adoption in communities where initial adoption exceeds 0.5, and toward non-adoption where it is below 0.5. Because malaria transmission depends on net use, stronger norms should reduce incidence over time in communities with high baseline net adoption and raise it in communities with low net baseline adoption, relative to communities with weak norms. Under the assumption of no migration, communities should converge gradually to their long-run state. For this reason, the differences between communities with weak and strong norms should grow in magnitude over time. These predictions are relative to 2009; with 2025 as the reference year, the signs are reversed.

In terms of oscillations, we expect the highest variability in communities with weak norms relative to strong norms. This is because under weak norms adoption responds flexibly to perceived risk: Low adoption raises prevalence, which induces adoption, which lowers prevalence and, in turn, reduces adoption. Under strong norms, we expect social inertia would dampen these responses, with communities converge to a stable state with either high or low adoption.

To estimate the association between social norms and malaria incidence, we estimate the following equation:

$$y_{it} = \alpha + \alpha_i + \alpha_t + v * nets_i * HHI_i * t + \delta * nets_i * t + \mu * HHI_i * t + \varepsilon_{it} \quad (S33)$$

where y is malarial incidence for cluster i and time t , α , α_i and α_t are a constant, cluster and time fixed effects, respectively. For example, α_t absorbs any common shocks like the universal distribution of nets. α_i controls for any time-invariant, linear, and unobserved cluster-level characteristics like the baseline local ecology, baseline mosquito carrying capacity, baseline transmission intensity. The variable $nets_i$ indicates the adoption of nets in cluster i at the baseline, and HHI_i is the social norms strength at the baseline in a cluster. δ , μ and v are coefficients to be estimated and ε_{it} is an error term. The coefficients capture changes in the incidence, rather than absolute levels. As before, we cluster the standard errors at the population cluster level. To reduce multicollinearity, we use the demeaned version of $nets_i$. This means that the interaction terms captures the impact of social norms along the mean net baseline adoption per cluster. The interaction terms use 2025 as the base year; but the $HHI*t$ interaction uses 2009 as the base year. To ease interpretation, we discretize HHI into 3 groups: low norms (HHI between 0.4 and 0.7), medium norms (HHI between 0.7 and 1) and high norms (HHI=1).

Given 2025 as the reference year, the model predicts that v should be > 0 ; the values should be larger for the stronger norms relative to the weaker norms. The expected value of μ depend on the baseline raw values of the share of net adoption p : If $p > 0.5$, $\mu > 0$ given the reference 2009 year; if $p < 0.5$, then $\mu < 0$. We expect $\delta > 0$, but for weaker norms it may be less consistent than for higher norms. The magnitudes of the estimated parameters should shrink as t approaches 2025.

¹⁹The results are available upon request.

We use the 2009 DHS clusters and construct a balanced panel for 2008 to 2025, so that we can test the predictions on the same clusters over time. We estimate the model using both net ownership and net use as baseline covariates. The results are qualitatively consistent. Because of concerns stemming from a potential Nickell bias (Nickell, 1981), we estimate the model with and without a lagged outcome. The two models have different interpretations, with the lagged model giving us short-run conditional effects, and model without a lag-cumulative changes over time.

Results. We consider Model 5 to be our main model due to Nickell bias concerns (Table S6). The positive coefficients for δ match our predictions. μ is positive and mostly significant for the medium norms and mostly positive but insignificant for the high norms. The statistically significant coefficient for HHI in 2008 for the strongest norms group suggests different trajectories in 2008 between the strong and weak norms groups. The signs of ν are consistent for the medium norms but mostly contradict the expectations for the high norms.

There are three possible explanations for the observed inconsistency for the strong norms group. First, the main model uses ownership and not use. However, even though net usage, proxied by the use of the net the night before the survey is not a perfect indicator, the model with net use and no lags is consistent with the main model ²⁰. Second, the model excludes important contextual factors like resistance and campaigns to distribute nets as the one in 2018 that may have been rolled out in a staggered manner and not captured by the year fixed effects (<https://lcm.org.lr/liberia-commences-nationwide-mosquito-net-distribution/>). Third, the theoretical models assume away migration, which is likely in a real-life setting. For these reasons, our estimation may be capturing transitional dynamics, which combine all of these potential explanations.

Table S6: Results for the correlation between malaria incidence and social norms (standard errors in parentheses).

	(1) Ownership (v459)	(2) Ownership (v459), no lag	(3) Use (v461)	(4) Ownership, centered (v459_c)	(5) Ownership, centered (v459_c), no lag
Lagged malaria incidence	0.9120***(0.0120)	—	0.9132***(0.0117)	0.9114***(0.0125)	—
Year fixed effects					
2008	—	(base)	—	—	0.0124***(0.0024)
2009	(base)	-0.0213*** (0.0043)	(base)	(base)	(base)
2010	-0.0024(0.0017)	-0.0431*** (0.0086)	-0.0022(0.0014)	-0.0030*(0.0018)	-0.0142*** (0.0030)
2011	-0.0038(0.0029)	-0.0644*** (0.0110)	-0.0051** (0.0025)	-0.0090*** (0.0028)	-0.0332*** (0.0058)
2012	-0.0033(0.0053)	-0.0833*** (0.0122)	-0.0086* (0.0044)	-0.0205*** (0.0035)	-0.0621*** (0.0080)
2013	0.0175*** (0.0060)	-0.0797*** (0.0120)	0.0113** (0.0049)	0.0004(0.0033)	-0.0674*** (0.0086)
2014	0.0348*** (0.0062)	-0.0592*** (0.0112)	0.0325*** (0.0048)	0.0275*** (0.0035)	-0.0452*** (0.0082)
2015	0.0332*** (0.0063)	-0.0421*** (0.0109)	0.0324*** (0.0047)	0.0317*** (0.0041)	-0.0207** (0.0082)
2016	0.0404*** (0.0065)	-0.0194* (0.0105)	0.0374*** (0.0052)	0.0427*** (0.0041)	0.0126(0.0086)
2017	0.0281*** (0.0049)	-0.0109(0.0096)	0.0226*** (0.0042)	0.0231*** (0.0029)	0.0233*** (0.0084)
2018	0.0027(0.0047)	-0.0285*** (0.0083)	-0.0055(0.0042)	-0.0157*** (0.0035)	-0.0058(0.0077)
2019	-0.0007(0.0051)	-0.0480*** (0.0083)	-0.0107** (0.0046)	-0.0181*** (0.0033)	-0.0346*** (0.0075)
2020	0.0012(0.0043)	-0.0639*** (0.0096)	-0.0060(0.0039)	-0.0130*** (0.0030)	-0.0559*** (0.0080)
2021	-0.0278*** (0.0064)	-0.1074*** (0.0138)	-0.0364*** (0.0054)	-0.0470*** (0.0046)	-0.1092*** (0.0107)
2022	-0.0117** (0.0055)	-0.1309*** (0.0165)	-0.0189*** (0.0047)	-0.0261*** (0.0040)	-0.1369*** (0.0127)
2023	0.0090** (0.0045)	-0.1316*** (0.0169)	0.0030(0.0040)	-0.0011(0.0034)	-0.1371*** (0.0132)
2024	0.0073(0.0055)	-0.1340*** (0.0174)	-0.0018(0.0051)	-0.0046(0.0040)	-0.1408*** (0.0137)
2025	0.0107* (0.0058)	-0.1329*** (0.0178)	0.0009(0.0053)	-0.0005(0.0041)	-0.1400*** (0.0139)
Year $\times$ net adoption					
2008	—	0.0284(0.0362)	—	—	-0.0112(0.0633)
2009	0.0226*(0.0119)	0.0460(0.0356)	0.0155(0.0199)	0.0507** (0.0198)	0.0231(0.0644)

²⁰The results are available upon request

Table S6 (continued)

	(1) Ownership (v459)	(2) Ownership (v459), no lag	(3) Use (v461)	(4) Ownership, centered (v459_c)	(5) Ownership, centered (v459_c), no lag
2010	0.0225*(0.0114)	0.0618*(0.0347)	0.0122(0.0188)	0.0564*** (0.0177)	0.0601(0.0647)
2011	0.0140(0.0088)	0.0678** (0.0325)	0.0007(0.0157)	0.0445*** (0.0129)	0.0819(0.0609)
2012	-0.0066(0.0086)	0.0527* (0.0281)	-0.0226(0.0155)	0.0250** (0.0111)	0.0822(0.0524)
2013	-0.0068(0.0081)	0.0387(0.0273)	-0.0196* (0.0118)	0.0205** (0.0082)	0.0781(0.0499)
2014	0.0090(0.0107)	0.0417(0.0316)	-0.0025(0.0147)	0.0278* (0.0162)	0.0816(0.0561)
2015	0.0179(0.0111)	0.0534(0.0363)	0.0085(0.0150)	0.0285* (0.0172)	0.0856(0.0629)
2016	0.0234** (0.0113)	0.0695* (0.0407)	0.0206(0.0148)	0.0262(0.0195)	0.0868(0.0714)
2017	0.0114* (0.0068)	0.0722* (0.0408)	0.0087(0.0092)	0.0191(0.0120)	0.0808(0.0726)
2018	-0.0105(0.0076)	0.0528(0.0348)	-0.0178(0.0121)	0.0065(0.0129)	0.0628(0.0600)
2019	-0.0092(0.0068)	0.0364(0.0279)	-0.0120(0.0100)	0.0049(0.0111)	0.0448(0.0478)
2020	-0.0030(0.0062)	0.0276(0.0235)	-0.0098(0.0095)	0.0155(0.0096)	0.0390(0.0402)
2021	-0.0103(0.0106)	0.0123(0.0145)	-0.0196(0.0157)	0.0182(0.0177)	0.0364(0.0242)
2022	-0.0027(0.0068)	0.0060(0.0103)	-0.0110(0.0101)	0.0207** (0.0097)	0.0365** (0.0167)
2023	0.0037(0.0047)	0.0065(0.0091)	-0.0030(0.0070)	0.0182*** (0.0064)	0.0341*** (0.0143)
2024	-0.0006(0.0015)	0.0028(0.0048)	-0.0027(0.0022)	0.0053*** (0.0019)	0.0191*** (0.0072)
Year × HHI group 2					
2008	—	—	—	—	-0.0029(0.0039)
2010	—	—	—	0.0006(0.0022)	0.0032(0.0045)
2011	—	—	—	0.0033(0.0037)	0.0088(0.0075)
2012	—	—	—	0.0032(0.0057)	0.0139(0.0098)
2013	—	—	—	0.0041(0.0056)	0.0194* (0.0107)
2014	—	—	—	0.0068(0.0054)	0.0272** (0.0106)
2015	—	—	—	0.0080(0.0058)	0.0355*** (0.0107)
2016	—	—	—	0.0001(0.0055)	0.0350*** (0.0110)
2017	—	—	—	-0.0038(0.0042)	0.0308*** (0.0106)
2018	—	—	—	0.0009(0.0048)	0.0316*** (0.0093)
2019	—	—	—	-0.0039(0.0045)	0.0276*** (0.0088)
2020	—	—	—	-0.0013(0.0039)	0.0265*** (0.0094)
2021	—	—	—	-0.0020(0.0059)	0.0248* (0.0126)
2022	—	—	—	-0.0020(0.0050)	0.0232(0.0150)
2023	—	—	—	-0.0024(0.0039)	0.0214(0.0156)
2024	—	—	—	-0.0059(0.0049)	0.0163(0.0163)
2025	—	—	—	-0.0075(0.0051)	0.0100(0.0166)
Year × HHI group 3					
2008	—	—	—	—	-0.0173*** (0.0046)
2010	—	—	—	-0.0024(0.0022)	0.0133*** (0.0046)
2011	—	—	—	-0.0097** (0.0040)	0.0183** (0.0077)
2012	—	—	—	-0.0158** (0.0065)	0.0166(0.0102)
2013	—	—	—	-0.0127** (0.0063)	0.0182(0.0113)
2014	—	—	—	-0.0070(0.0054)	0.0253** (0.0113)
2015	—	—	—	-0.0066(0.0058)	0.0322*** (0.0114)
2016	—	—	—	-0.0200*** (0.0065)	0.0251** (0.0113)
2017	—	—	—	-0.0216*** (0.0052)	0.0171(0.0107)
2018	—	—	—	-0.0123** (0.0054)	0.0191** (0.0094)
2019	—	—	—	-0.0215*** (0.0058)	0.0116(0.0087)
2020	—	—	—	-0.0173*** (0.0049)	0.0091(0.0093)
2021	—	—	—	-0.0186*** (0.0068)	0.0055(0.0126)
2022	—	—	—	-0.0196*** (0.0060)	0.0012(0.0151)
2023	—	—	—	-0.0189*** (0.0048)	-0.0021(0.0156)
2024	—	—	—	-0.0266*** (0.0064)	-0.0128(0.0165)
2025	—	—	—	-0.0294*** (0.0066)	-0.0252(0.0173)
Year × HHI group 2 × net adoption					
2008	—	-0.0126(0.0285)	—	—	0.0719(0.0775)
2009	0.0087(0.0094)	-0.0107(0.0275)	0.0082(0.0161)	-0.0250(0.0235)	0.0485(0.0763)

Table S6 (continued)

	(1) Ownership (v459)	(2) Ownership (v459), no lag	(3) Use (v461)	(4) Ownership, centered (v459_c)	(5) Ownership, centered (v459_c), no lag
2010	0.0091(0.0096)	-0.0085(0.0259)	0.0119(0.0163)	-0.0288(0.0225)	0.0233(0.0745)
2011	0.0142*(0.0073)	-0.0015(0.0234)	0.0204(0.0135)	-0.0209(0.0161)	0.0083(0.0694)
2012	0.0141**(0.0070)	0.0048(0.0194)	0.0216*(0.0129)	-0.0199(0.0156)	-0.0044(0.0588)
2013	0.0172*** (0.0060)	0.0137(0.0189)	0.0245** (0.0094)	-0.0019(0.0137)	0.0021(0.0567)
2014	0.0234*** (0.0078)	0.0280(0.0223)	0.0298** (0.0121)	0.0164(0.0205)	0.0262(0.0653)
2015	0.0263*** (0.0085)	0.0439*(0.0262)	0.0330** (0.0130)	0.0254(0.0216)	0.0573(0.0749)
2016	0.0138*(0.0082)	0.0460(0.0291)	0.0178(0.0125)	0.0227(0.0232)	0.0829(0.0850)
2017	0.0058(0.0049)	0.0399(0.0290)	0.0053(0.0076)	0.0019(0.0137)	0.0855(0.0860)
2018	0.0112*(0.0062)	0.0397(0.0251)	0.0117(0.0104)	-0.0165(0.0153)	0.0694(0.0721)
2019	0.0032(0.0056)	0.0316(0.0205)	0.0009(0.0088)	-0.0231(0.0143)	0.0481(0.0575)
2020	0.0074(0.0053)	0.0283(0.0173)	0.0075(0.0085)	-0.0206(0.0125)	0.0312(0.0484)
2021	0.0044(0.0080)	0.0224** (0.0108)	0.0062(0.0134)	-0.0372*(0.0217)	-0.0008(0.0288)
2022	0.0060(0.0052)	0.0185** (0.0077)	0.0084(0.0087)	-0.0221*(0.0128)	-0.0149(0.0195)
2023	0.0069*(0.0037)	0.0159** (0.0068)	0.0083(0.0060)	-0.0086(0.0086)	-0.0141(0.0171)
2024	0.0021*(0.0012)	0.0087** (0.0036)	0.0034*(0.0019)	-0.0038(0.0027)	-0.0088(0.0088)
Year $\times$ HHI group 3 $\times$ net adoption					
2008	—	0.0045(0.0286)	—	—	-0.0056(0.0720)
2009	0.0442*** (0.0112)	0.0330(0.0289)	0.0574*** (0.0194)	0.0072(0.0289)	0.0096(0.0726)
2010	0.0373*** (0.0108)	0.0522*(0.0286)	0.0498*** (0.0188)	-0.0121(0.0277)	0.0041(0.0738)
2011	0.0264*** (0.0079)	0.0587** (0.0267)	0.0378** (0.0147)	-0.0161(0.0189)	-0.0049(0.0710)
2012	0.0143*(0.0080)	0.0526** (0.0231)	0.0219(0.0144)	-0.0336*(0.0175)	-0.0306(0.0626)
2013	0.0159** (0.0065)	0.0486** (0.0227)	0.0242** (0.0108)	-0.0431*** (0.0151)	-0.0635(0.0607)
2014	0.0265*** (0.0081)	0.0555** (0.0257)	0.0374*** (0.0132)	-0.0249(0.0206)	-0.0752(0.0682)
2015	0.0310*** (0.0089)	0.0663** (0.0287)	0.0434*** (0.0145)	-0.0028(0.0221)	-0.0639(0.0760)
2016	0.0095(0.0088)	0.0547*(0.0307)	0.0121(0.0139)	-0.0148(0.0236)	-0.0655(0.0824)
2017	0.0091*(0.0054)	0.0436(0.0305)	0.0106(0.0086)	-0.0099(0.0148)	-0.0622(0.0820)
2018	0.0291*** (0.0064)	0.0536** (0.0265)	0.0419*** (0.0110)	0.0190(0.0170)	-0.0302(0.0694)
2019	0.0149** (0.0057)	0.0485** (0.0216)	0.0213** (0.0094)	0.0123(0.0156)	-0.0077(0.0556)
2020	0.0195*** (0.0058)	0.0484*** (0.0185)	0.0279*** (0.0099)	0.0048(0.0151)	0.0053(0.0471)
2021	0.0158*(0.0084)	0.0446*** (0.0119)	0.0246*(0.0146)	-0.0090(0.0215)	0.0033(0.0308)
2022	0.0115** (0.0054)	0.0369*** (0.0086)	0.0182*(0.0093)	-0.0194(0.0135)	-0.0089(0.0230)
2023	0.0137*** (0.0043)	0.0321*** (0.0079)	0.0199*** (0.0074)	-0.0090(0.0108)	-0.0096(0.0208)
2024	0.0028** (0.0011)	0.0168*** (0.0041)	0.0044** (0.0019)	-0.0069** (0.0028)	-0.0082(0.0108)
Constant	-0.0020(0.0066)	0.2923*** (0.0165)	0.0075(0.0058)	0.0217*** (0.0045)	0.3031*** (0.0030)
Lagged dependent variable	Yes	No	Yes	Yes	No
Cluster fixed effects	Yes	Yes	Yes	Yes	Yes
Year fixed effects	Yes	Yes	Yes	Yes	Yes
Year $\times$ HHI group terms	No	No	No	Yes	Yes
Observations	2,550	2,700	2,550	2,550	2,700
Clusters	150	150	150	150	150
R ² (within)	0.9446	0.7299	0.9433	0.9460	0.7380

Significance levels *** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$.

S6.3.3 Social norms and mosquito resistance

Estimation. We hypothesize that stronger social norms will drive the adoption of nets to either low or very high net usage. This implies that we should see the highest mosquito resistance with the highest norms when the net adoption is high. Low social norms will have varying impacts on net adoption, and, hence, are likely to result in smaller resistance. To quantify the association

norms with the evolution of mosquito resistance, we estimate:

$$y_{it} = \alpha_i + \alpha_t + \sum_s \beta * hhi_{i0} * 1(t = s) + \varepsilon_{it},$$

where y_{it} is the reported insecticide resistance at cluster i at time t , α_i indicate cluster-level fixed effects, hhi is the religious diversity in a cluster at the baseline $t = 0$ (the year of the data collection), and ε is an error term. Because of the likely correlation between the norm strength and net usage, we include that net usage only in the robustness checks specifications. The net usage and the HHI variables are based on the baseline (2009) values. Because of concerns with Nickell bias in models with lagged dependent variable and a small number of time periods (Nickell, 1981), we include the lagged outcome only in robustness checks. In addition, correlations between HHI and the adoption of nets at the cluster level may result in multicollinearity. To address this issue, we conduct a sequentially drop each variable and estimate the model separately.

For the main model we use the cluster data for 2009, with a cluster being the unit of analysis, (n=150) and construct a balanced panel between 2009 and 2017. The reason we exclude data from later DHS waves are concerns with biases stemming from pooling data for later cohorts that are observed for only a fraction of the years. We use cluster-level fixed effects. We use a discrete and a continuous version of HHI at the cluster level. We consider mosquito resistance to permethrin and deltamethrin separately.

Results (Permethrin). We choose Model 8 (with no lags and no nets in Table S7) as the main model for the following reasons: First, models with a small number of time periods (T=8) and a lagged outcome may result in biased estimates. Second, the lagged outcome variable is statistically significantly correlated with the coefficients of interest. When it is included, only 4 of the coefficients are statistically significant (Model 7). When it is excluded, almost all coefficients become statistically significant (Model 8). As it is not possible to perform an F-test in this case, Model 8 yields a higher R^2 value ($R^2=0.68$ as opposed to 0.57 for Model 7). However, the differences in the R^2 values can also be due to the difference in the sample sizes. Since the inclusion of ITNs in the original models used to generate the spatial resistance data, we include a specification with the baseline use of nets as a robustness check. The results are very consistent with the main model.

We find that permethrin resistance declines over time, which could be due to different insecticides being used. We find a significant association between HHI and permethrin resistance, with higher HHI translating into a slower decline in resistance (Model 8). That is, relative to the lowest HHI group, higher levels increase permethrin resistance, with the highest HHI group having the highest resistance. The results are robust across specifications. This is consistent with the main hypothesis.

Table S7: Results from a fixed effects regression of permethrin resistance as a function of social norms (proxied by the HHI variable) across years (standard errors in parentheses). The hhi_group variable indicates a discrete group of the dstrength of norms, with 1 being the lowest and 3-the highest. Model 8 is our preferred specification. The sample size is 150 DHS clusters observed 2009-2017.

Variable	[1]	[2]	[3]	[4]	[5]	[6]	[7]	[8]
Lagged outcome (L1)	-0.10*** (0.03)	-0.09*** (0.03)			-0.21*** (0.03)		-0.22*** (0.03)	
Year*HHI (continuous version)								
year#c.hhi 10	0.21*** (0.02)	0.28*** (0.02)	0.17*** (0.02)	0.23*** (0.01)				
year#c.hhi 11	0.38*** (0.02)	0.35*** (0.01)	0.37*** (0.02)	0.33*** (0.01)				
year#c.hhi 12	0.34*** (0.02)	0.30*** (0.01)	0.31*** (0.02)	0.27*** (0.01)				

Table S7 (continued)

Variable	[1]	[2]	[3]	[4]	[5]	[6]	[7]	[8]
year#c.hhi 13	0.35*** (0.02)	0.29*** (0.01)	0.33*** (0.02)	0.27*** (0.01)				
year#c.hhi 14	0.29*** (0.02)	0.25*** (0.01)	0.26*** (0.02)	0.23*** (0.01)				
year#c.hhi 15	-0.03* (0.02)	0.01 (0.01)	-0.05*** (0.01)	-0.01 (0.01)				
year#c.hhi 16	0.07*** (0.02)	0.02** (0.01)	0.08*** (0.02)	0.03*** (0.01)				
year#c.hhi 17	(omitted)	(omitted)	(omitted)	(omitted)				
Year *net usage (v461)								
year#c.v461 10	0.16*** (0.04)		0.15*** (0.04)		(base)	0.25*** (0.05)		
year#c.v461 11	-0.05 (0.05)		-0.08 (0.05)		-0.26*** (0.05)	-0.06*** (0.02)		
year#c.v461 12	-0.10*** (0.03)		-0.10*** (0.03)		-0.29*** (0.06)	-0.04 (0.04)		
year#c.v461 13	-0.13*** (0.04)		-0.13*** (0.04)		-0.32*** (0.06)	-0.07* (0.04)		
year#c.v461 14	-0.08** (0.04)		-0.08** (0.03)		-0.26*** (0.06)	0.0004 (0.03)		
year#c.v461 15	0.09** (0.04)		0.08** (0.04)		-0.01 (0.02)	0.23*** (0.05)		
year#c.v461 16	-0.10*** (0.03)		-0.12*** (0.03)		-0.20*** (0.05)	-0.0025 (0.03)		
year#c.v461 17	(omitted)		(omitted)		-0.10** (0.04)	0.15*** (0.04)		
year#c.hhi 9			0.53*** (0.03)	0.54*** (0.02)				
year#c.v461 9			0.03 (0.06)			(base)		
Year fixed effects								
year 10					(base)	-0.43*** (0.03)	(base)	-0.34*** (0.02)
year 11					0.16*** (0.03)	-0.18*** (0.01)	0.07*** (0.02)	-0.20*** (0.01)
year 12					0.10*** (0.03)	-0.29*** (0.02)	-0.0006 (0.03)	-0.30*** (0.01)
year 13					0.10** (0.04)	-0.27*** (0.02)	-0.02 (0.03)	-0.30*** (0.02)
year 14					0.03 (0.04)	-0.34*** (0.02)	-0.06** (0.03)	-0.34*** (0.01)
year 15					-0.27*** (0.02)	-0.63*** (0.03)	-0.28*** (0.01)	-0.55*** (0.02)
year 16					-0.21*** (0.03)	-0.51*** (0.02)	-0.28*** (0.03)	-0.51*** (0.01)
year 17					-0.28*** (0.03)	-0.60*** (0.02)	-0.32*** (0.02)	-0.55*** (0.01)
Year*HHI (discrete)								
hhi_group#year 2 10					(base)	0.07** (0.03)	(base)	0.07** (0.03)
hhi_group#year 2 11					-0.05** (0.02)	0.01 (0.01)	-0.05** (0.02)	0.01 (0.01)
hhi_group#year 2 12					-0.03 (0.03)	0.04** (0.02)	-0.03 (0.03)	0.04** (0.02)
hhi_group#year 2 13					-0.01 (0.03)	0.05** (0.02)	-0.01 (0.04)	0.05** (0.02)
hhi_group#year 2 14					-0.01 (0.04)	0.04** (0.02)	-0.01 (0.04)	0.04** (0.02)
hhi_group#year 2 15					-0.0001 (0.01)	0.06** (0.03)	-0.0000054 (0.01)	0.06** (0.03)
hhi_group#year 2 16					-0.01 (0.02)	0.04*** (0.02)	-0.01 (0.03)	0.04*** (0.02)

Table S7 (continued)

Variable	[1]	[2]	[3]	[4]	[5]	[6]	[7]	[8]
hhi_group#year 2 17					-0.01 (0.02)	0.05*** (0.02)	-0.01 (0.02)	0.05*** (0.02)
hhi_group#year 3 10					(base)	0.09*** (0.03)	(base)	0.11*** (0.03)
hhi_group#year 3 11					-0.03 (0.02)	0.04*** (0.01)	-0.05* (0.03)	0.04*** (0.01)
hhi_group#year 3 12					0.04 (0.03)	0.12*** (0.02)	0.02 (0.03)	0.12*** (0.02)
hhi_group#year 3 13					0.04 (0.03)	0.11*** (0.02)	0.02 (0.04)	0.11*** (0.02)
hhi_group#year 3 14					0.05 (0.04)	0.12*** (0.02)	0.03 (0.04)	0.12*** (0.02)
hhi_group#year 3 15					0.02* (0.01)	0.09*** (0.03)	0.02* (0.01)	0.11*** (0.03)
hhi_group#year 3 16					0.02 (0.02)	0.09*** (0.02)	0.01 (0.03)	0.09*** (0.02)
hhi_group#year 3 17					0.05** (0.02)	0.13*** (0.02)	0.05* (0.02)	0.14*** (0.02)
Constant	0.30*** (0.01)	0.29*** (0.01)	0.27*** (0.01)	0.27*** (0.01)	0.62*** (0.02)	0.74*** (0.01)	0.62*** (0.02)	0.74*** (0.01)
N	1,120	1,120	1,260	1,260	1,120	1,260	1,120	1,260
R ²	0.5377	0.5201	0.6432	0.6315	0.5835	0.6924	0.5713	0.6832

Significance levels. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$.

Results (Deltamethrin). We consider Model 8 to be the main specification (Table S8). Similar to permethrin, we find that resistance declines over time (Figure S23a). We find that the highest HHI group only sometimes exhibits higher resistance relative to the lowest group - the statistically significant coefficients exhibit both positive and negative signs. However, the difference between the medium and high HHI groups is statistically significant for year=2017 (when the highest group has the highest resistance). There are five reasons why the discrepancy between deltamethrin and permethrin resistance as a function of social norms may exist. First, these are two different types of pyrethroids, affecting mosquitoes in different ways (Breckenridge *et al.*, 2009). Second, it could be a remnant of the history of insecticide usage, with one insecticide being introduced earlier in a given location. The different timing for the introduction means that we may be capturing transitional dynamics and not equilibria for deltamethrin. Second, while some studies suggest that deltamethrin was the insecticide of ITN choice for most of the country (Temu *et al.*, 2012), we do not know if the ITN insecticide heterogeneity is higher in the high HHI clusters. Third, the resistance data capture 3 mosquito species (*Anopheles gambiae*, *An. coluzzi*, and *An. arabiensis*), so it is unclear how these are distributed along the DHS clusters (e.g., correlated with the strength of social norms) and if these separate species evolve in the same way (at the same speed) when faced with the same insecticide. Lastly, 2011 data from the same DHS clusters indicate population changes (Figure S23a). The regression of changes in permethrin resistance between 2009 and 2011 on changes in the net usage calculated in the same way indicate a positive and statistically significant relationship ($coef = .0412678$, $se = .0175138$, $P < 5\%$, $n = 140$). The coefficient on deltamethrin is also positive but statistically insignificant ($coef = .0271813$, $se = .0235654$, $P > 10\%$). The net usage change is only positively correlated with the change in norms only for the lowest HHI group in 2009 (Table S9).

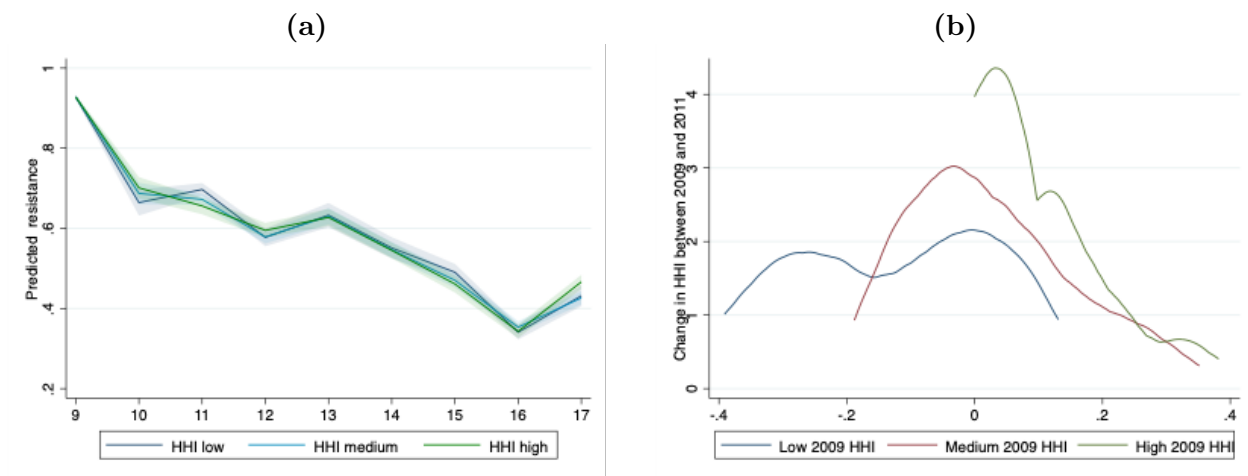


Figure S23: (a) Predicted resistance to deltamethrin as a function of the degree of religious homogeneity (captured by the HHI). (b) Kernel density plot of the changes in the norms between 2009 and 2011 as a function of the baseline norms. The change in norms is calculated as the HHI value in 2009-HHI value in 2011. The figure suggests that DHS clusters with high HHI in 2009 experienced only decline in the HHI, whereas for clusters with low HHI, the data suggest the reverse pattern.

Table S8: Impact of social norms and net usage on deltamethrin resistance (standard errors in parentheses). The hhi_group variable indicates a discrete group of the degree of religious homogeneity (our proxy for the norms strength), with 1 being the lowest and 3-the highest. Model 8 is our preferred specification. The sample size is 150 DHS clusters observed 2009-2017.

Variable	[1]	[2]	[3]	[4]	[5]	[6]	[7]	[8]
Lagged outcome	0.06 (0.05)	0.07 (0.04)			-0.03 (0.03)		-0.02 (0.03)	
Year*HHI (continuous)								
year#c.hhi 9			0.52*** (0.02)	0.55*** (0.01)				
year#c.hhi 10	0.18*** (0.03)	0.23*** (0.03)	0.21*** (0.02)	0.28*** (0.01)				
year#c.hhi 11	0.27*** (0.02)	0.23*** (0.02)	0.28*** (0.02)	0.26*** (0.01)				
year#c.hhi 12	0.19*** (0.02)	0.14*** (0.01)	0.21*** (0.02)	0.16*** (0.01)				
year#c.hhi 13	0.26*** (0.02)	0.19*** (0.01)	0.28*** (0.02)	0.21*** (0.01)				
year#c.hhi 14	0.14*** (0.02)	0.10*** (0.01)	0.16*** (0.02)	0.12*** (0.01)				
year#c.hhi 15	-0.004 (0.02)	0.01 (0.01)	0.01 (0.01)	0.03*** (0.01)				
year#c.hhi 16	-0.05*** (0.02)	-0.12*** (0.01)	-0.04** (0.02)	-0.11*** (0.01)				
year#c.hhi 17	(omitted)	(omitted)	(omitted)	(omitted)				
Year #continuous net usage (v461)								
year#c.v461 9			0.07* (0.04)					
year#c.v461 10	0.15*** (0.05)		0.16*** (0.05)			0.17*** (0.04)		
year#c.v461 11	-0.08* (0.04)		-0.06 (0.04)		-0.26*** (0.05)	-0.10*** (0.02)		
year#c.v461 12	-0.13*** (0.03)		-0.12*** (0.03)		-0.28*** (0.05)	-0.11*** (0.02)		
year#c.v461 13	-0.16*** (0.03)		-0.15*** (0.03)		-0.34*** (0.06)	-0.17*** (0.03)		

Table S8 (continued)

Variable	[1]	[2]	[3]	[4]	[5]	[6]	[7]	[8]
year#c.v461 14	-0.10*** (0.03)		-0.10*** (0.03)		-0.26*** (0.06)	-0.08*** (0.03)		
year#c.v461 15	0.05 (0.03)		0.05 (0.03)		-0.09** (0.04)	0.08*** (0.03)		
year#c.v461 16	-0.17*** (0.03)		-0.15*** (0.03)		-0.25*** (0.05)	-0.08*** (0.02)		
year#c.v461 17	(omitted)		(omitted)		-0.11** (0.05)	0.06** (0.02)		
Year Fixed Effects								
year 10					(base)	-0.32*** (0.02)	(base)	-0.26*** (0.02)
year 11					0.12*** (0.02)	-0.20*** (0.01)	0.03 (0.02)	-0.23*** (0.01)
year 12					0.01 (0.03)	-0.31*** (0.01)	-0.09*** (0.02)	-0.35*** (0.01)
year 13					0.08** (0.04)	-0.24*** (0.02)	-0.04 (0.03)	-0.29*** (0.01)
year 14					-0.03 (0.03)	-0.35*** (0.02)	-0.12*** (0.03)	-0.38*** (0.01)
year 15					-0.15*** (0.02)	-0.47*** (0.01)	-0.18*** (0.03)	-0.44*** (0.01)
year 16					-0.25*** (0.03)	-0.56*** (0.01)	-0.33*** (0.02)	-0.59*** (0.01)
year 17					-0.21*** (0.03)	-0.52*** (0.02)	-0.25*** (0.03)	-0.50*** (0.01)
Year *HHI (discrete)								
hhi_group#year 2 10					(base)	0.02 (0.02)	(base)	0.02 (0.02)
hhi_group#year 2 11					-0.05* (0.02)	-0.02** (0.01)	-0.05* (0.03)	-0.02** (0.01)
hhi_group#year 2 12					-0.02 (0.03)	0.002 (0.01)	-0.02 (0.03)	0.001 (0.02)
hhi_group#year 2 13					-0.02 (0.04)	-0.002 (0.02)	-0.03 (0.04)	-0.003 (0.02)
hhi_group#year 2 14					-0.03 (0.04)	-0.004 (0.02)	-0.03 (0.04)	-0.004 (0.02)
hhi_group#year 2 15					-0.04** (0.02)	-0.02 (0.01)	-0.04** (0.02)	-0.02 (0.01)
hhi_group#year 2 16					-0.01 (0.03)	0.01 (0.01)	-0.01 (0.03)	0.01 (0.01)
hhi_group#year 2 17					-0.03 (0.03)	-0.01 (0.02)	-0.03 (0.03)	-0.01 (0.02)
hhi_group#year 3 10					(base)	0.03 (0.02)	(base)	0.04* (0.02)
hhi_group#year 3 11					-0.06** (0.02)	-0.03*** (0.01)	-0.08*** (0.03)	-0.04*** (0.01)
hhi_group#year 3 12					-0.002 (0.03)	0.02* (0.01)	-0.02 (0.03)	0.02 (0.02)
hhi_group#year 3 13					-0.02 (0.03)	0.005 (0.02)	-0.04 (0.04)	-0.01 (0.02)
hhi_group#year 3 14					-0.03 (0.03)	-0.001 (0.02)	-0.04 (0.04)	-0.01 (0.02)
hhi_group#year 3 15					-0.06*** (0.02)	-0.03** (0.02)	-0.07*** (0.02)	-0.03* (0.02)
hhi_group#year 3 16					-0.02 (0.03)	0.01 (0.01)	-0.04 (0.03)	0.002 (0.01)
hhi_group#year 3 17					0.005 (0.03)	0.03* (0.02)	-0.002 (0.03)	0.03** (0.02)
Constant	0.43*** (0.02)	0.42*** (0.02)	0.45*** (0.01)	0.45*** (0.01)	0.71*** (0.03)	0.93*** (0.003)	0.71*** (0.03)	0.93*** (0.003)
N	1,120	1,120	1,260	1,260	1,120	1,260	1,120	1,260
R ²	0.5955	0.5760	0.7157	0.7001	0.6304	0.7985	0.6233	0.7808

Table S8 (continued)

Variable	[1]	[2]	[3]	[4]	[5]	[6]	[7]	[8]
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Significance levels: ***1%, **5%, *10%.

HHI in 2009	stats	Change in net usage	Change HHI	in Change permethrin resistance	in Change deltamethrin resistance	in
HHI 2009 Group 1	N	38	38	34	34	
	mean	-0.04	-0.13	0.1971818	0.2308686	
	sd	0.17	0.16	0.0507444	0.0491274	
	min	-0.44	-0.39	0.095915	0.137636	
	max	0.36	0.13	0.298247	0.313585	
HHI 2009 Group 2	N	65	65	60	60	
	mean	-0.06	0.03	0.1914505	0.2554638	
	sd	0.25	0.13	0.0486305	0.0583601	
	min	-0.52	-0.19	0.086894	0.160595	
	max	0.47	0.35	0.301764	0.409455	
HHI 2009 Group 3	N	47	47	46	46	
	mean	-0.03	0.09	0.1565419	0.2720433	
	sd	0.21	0.10	0.0540519	0.0749139	
	min	-0.52	0.00	0.065328	0.126339	
	max	0.54	0.38	0.267764	0.439168	

Table S9: Summary statistics for the changes in net usage and degree of religious homogeneity between 2009 and 2011 as a function of the baseline 2009 homogeneity. All differences are calculated as value in 2009-value in 2011, with negative values indicating an increase and positive-a decrease.