

Deconstructing the Impact of Malaria Vector Diversity on Disease Risk

Amber Gigi Hoi,* Benjamin Gilbert, and Nicole Mideo

Department of Ecology and Evolutionary Biology, University of Toronto, 25 Willcocks Street, Toronto, Ontario M5S 3B2, Canada

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ABSTRACT: Recent years have seen significant progress in understanding the impact of host community assemblage on disease risk, yet diversity in disease vectors has rarely been investigated. Using published malaria and mosquito surveys from Kenya, we analyzed the relationship between malaria prevalence and multiple axes of mosquito diversity: abundance, species richness, and composition. We found a net amplification of malaria prevalence by vector species richness, a result of a strong direct positive association between richness and prevalence alongside a weak indirect negative association between the two, mediated through mosquito community composition. One plausible explanation of these patterns is species niche complementarity, whereby less competent vector species contribute to disease transmission by filling spatial or temporal gaps in transmission left by dominant vectors. A greater understanding of vector community assemblage and function, as well as any interactions between host and vector biodiversity, could offer insights to both fundamental and applied ecology.

Keywords: malaria, vector management, community ecology, biodiversity-ecosystem function, dilution effect, amplification effect.

Introduction

One of the most publicized areas of research in ecology in recent years sits at the intersection of conservation and public health: the study of whether preserving biodiversity lowers infectious disease risk to humans, livestock, or managed wildlife (Ostfeld and Keesing 2017). Biodiversity can be measured along several axes, including species abundances, richness (number of species), and composition (species identity and relative abundance). The so-called dilution effect—where disease risk to a focal host species

decreases as the richness of potential host species increases (Ostfeld and Keesing 2000)—has been documented in a number of disease systems, including trees and water mold (e.g., Haas et al. 2011), freshwater fish and trematodes (e.g., Lagrue and Poulin 2015), toads and fungal pathogens (e.g., Searle et al. 2011), and arthropod-vector zoonoses, such as Lyme disease and West Nile fever (e.g., Ostfeld and Keesing 2000; Levine et al. 2017). Yet the generality of the dilution effect is strongly debated (Randolph and Dobson 2012), and attempts to broadly characterize the relationship between host biodiversity and disease risk via meta-analyses have generated conflicting results. Indeed, studies have reported overall dilution (Civitello et al. 2015), amplification (i.e., increased risk with increasing diversity; Wood et al. 2017), and no effect (Salkeld et al. 2013).

Given the lack of simple generalities, studies of the influence of host biodiversity on disease risk are moving toward elucidating mechanisms that give rise to a particular outcome. One of the best-studied examples of a dilution effect occurs with Lyme disease, caused by a tick-borne bacteria that infects a range of mammalian species, including humans. Mediating this effect is the fact that the white-footed mouse is both ubiquitous across communities and highly susceptible to the parasite (competent; Ostfeld and Keesing 2000). Species-rich communities therefore contain more low-quality hosts that act as competitors or predators regulating mouse population density and/or serve as alternative blood hosts for the ticks, reducing the prevalence of infected ticks in the area (Ostfeld and Keesing 2000). This case study emphasizes that the influence of host diversity on disease risk depends on both the variation in host competence and its covariation with other traits that determine species' distributions and abundances (see also Joseph et al. 2013).

Like many parasites, this archetypal case study for host diversity generating a dilution effect (Lyme disease) has multiple vector species, yet the influence of vector diversity on disease risk is underappreciated (Blaustein et al. 2010; Godfray 2013). Variation in the distribution, life

* Corresponding author; email: amb.g.hoi@gmail.com.

ORCID: Hoi, <https://orcid.org/0000-0002-7477-5269>; Gilbert, <https://orcid.org/0000-0002-4947-6822>; Mideo, <https://orcid.org/0000-0002-8719-3620>.

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Table 1: Summary of phenological and behavioral traits of four species of *Anopheles* mosquitoes present on Kenyan Coast

	<i>Anopheles arabiensis</i>	<i>Anopheles funestus</i>	<i>Anopheles gambiae</i>	<i>Anopheles merus</i>	References
Season	Dry	Dry	Rainy	Dry	Lindsay et al. 1998; Minakawa et al. 2002; Derua et al. 2012
Larval habitat preference	Well lit, clear, flowing water	Well lit, clear, fresh water	Well lit, clear, fresh, still water	Brackish and still water	Sinka et al. 2010
Endophilicity ^a	.49 (0–1)	.54 (0–1)	.57 (.15–1)	No data	Massey et al. 2016
Affinity for humans ^b	.42 (0–.80)	.87 (.64–1)	.81 (.63–.93)	.12	Killeen et al. 2017
Biting time	Dusk to night	Night to dawn	Night to dawn	Night	Sinka et al. 2010

^a Data are mean (range) of proportion of mosquitoes found biting indoors.

^b Data are mean (range) of proportion of blood meals obtained from humans. Only surveys that employ both indoor and outdoor sampling were included here.

history, and behavior of vectors will influence each species' capacity to transmit disease (i.e., vector competence; table 1). As a consequence, one might predict vector species richness to amplify disease risk if variation in traits across species includes variation in, for example, feeding time or seasonality, potentially increasing vector-host contacts in space and time. However, in reality, as with host diversity, the influence of vector diversity on disease risk will depend on the covariance between species richness, abundance, and community composition, and both negative (dilution) and positive (amplification) effects could arise in natural systems (table 2; Johnson et al. 2013; Mihaljevic et al. 2014; Rohr et al. 2020). Classic diversity-abundance scaling suggests that more diverse communities will be the ones with the highest total abundance (Fisher et al. 1943; Srivastava et al. 2008). In these cases, regardless of the distribution of community competence, increasing diversity is likely to increase disease risk, although potentially through different mechanisms (table 2,

top row). In contrast, some areas with high species diversity show little difference in total abundance (table 2, bottom row), such as when strong interspecies interactions regulate densities or when environmental differences favor more species but similar total abundances (MacArthur 1984). Regardless of the cause, the influence of vector diversity on disease risk in the absence of increasing total abundance will depend on the order in which vector species are added and the covariation between rarity in the regional pool and competence (table 2, bottom row; Roche et al. 2013).

A small body of theoretical and empirical work on vector diversity has provided support for some of these predictions borne out of general ecological theory. For example, when greater species diversity is associated with a large increase in total vector abundance, theory predicts that an increase in competent vectors can overwhelm any negative influence of including less competent vectors in the community, resulting in elevated disease risk (Roche et al. 2013). An amplification effect may also occur if competent vectors are

Table 2: Predicted effects of vector diversity on disease risk driven by covariance between abundance, composition, and species richness

	Competent species are rare		Competent species are common	
	Low variance in community competency	High variance in community competency	Low variance in community competency	High variance in community competency
Species richness and abundance covary	Amplification: total abundance increased	Amplification: competent species added	Amplification: total abundance increased	Amplification: abundance of competent species increased
Communities differ only in species richness	Little effect: species replaced by something similar	Amplification: low-competence species displaced by competent ones	Little effect: species replaced by something similar	Dilution: competent species displaced by less-competent ones

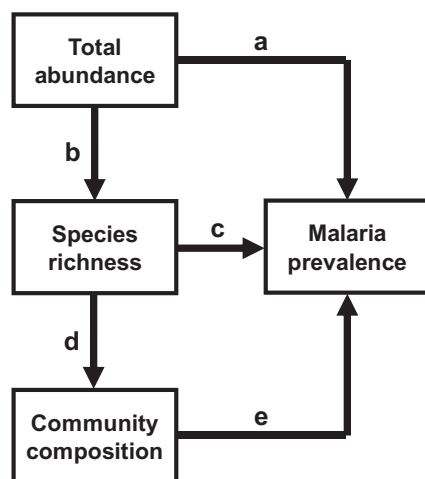
Note: Communities may differ in species richness because of having more or fewer individuals or via other processes, while abundance remains unchanged. When competent species are rare, community competence exhibits a left-skewed distribution, and these species tend to occur only in rich communities. The opposite holds when competent species are common. This—along with the variance in competence among species—determines the order in which species are added and thus how community competency and disease risk will change with increasing species richness. We note that these same predictions should apply to host communities as well.

rare and thus more likely found in species-rich sites (i.e., a sampling effect), independent of any effects of abundance, as was found for the mosquito communities that vector West Nile virus (Martínez-De La Puente et al. 2018). Given the potential for vector diversity to impact disease risk in ways that are complex, indirect, and not always intuitive, there remains a need for dissecting these relationships in natural systems, especially since they could provide crucial insights for vector management programs (Allan et al. 2009; Ostfeld 2009; Tran et al. 2017).

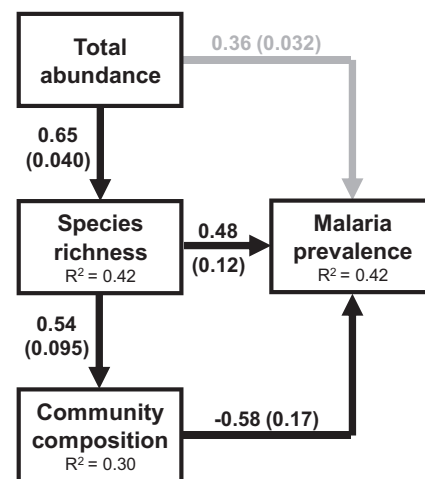
In this paper, we use previously published data on malaria prevalence in humans and sampling of a diverse mosquito vector community (Mbogo et al. 2003) to understand how disease prevalence changes with vector diversity and to disentangle the effects of vector abundance, species richness, and community composition on this relationship. We first test how these factors independently influence the prevalence of malaria and quantify the overall effect of vector species richness in this system. We then use path analysis (fig. 1A) to distinguish whether the effect of species richness we observe is driven by changes in (1) abundance, (2) community composition, or (3) spe-

cies richness per se (i.e., the relative strengths of paths $d \times b$, $e \times c$, and b , respectively). The four species found in this region (*Anopheles arabiensis*, *Anopheles funestus*, *Anopheles gambiae* sensu stricto [hereafter *An. gambiae*], and *Anopheles merus*; fig. A1, available online) are all considered competent vector species of malaria (i.e., capable of transmitting the disease; Hay et al. 2010; Sinka et al. 2010), although they differ in behavior, life history, and seasonal phenology (table 1; Mbogo et al. 2003). Notably, *An. arabiensis* and *An. funestus* are both dry season species thought to play important roles in sustaining transmission between rains (Minakawa et al. 2002). We therefore predict an amplification effect driven by the prolonged transmission period at villages with high vector species richness. In this region, however, the species often considered to be the most important vector for malaria because of its high affinity to humans (*An. gambiae*; table 1; Killeen et al. 2017) is also the most common (fig. A1). This obscures the effect of increasing species richness, as we could predict either amplification or dilution to occur depending on how richness changes with abundance (table 2). Our analysis allows us to distinguish between these contradictory

A) Hypothesized relationships



B) Model fit results



DF = 2, Fisher's C = 2.07, $p = 0.36$

Figure 1: Path diagram representing hypothesized relationships between characteristics of vector communities and malaria prevalence (A) and results from fitting this model (B). Paths (arrows) represent the direction of the hypothesized causal relationship between variables, with the arrow pointing from the predictor to the response. See table 3 for a detailed explanation of each path. In A, paths are labeled with letters to facilitate discussion in the text. In B, path labels represent standardized coefficient estimates (i.e., expressed in standard deviation units), with standard errors from our path analysis reported in parentheses. Note that increasing community composition in path c represents a high abundance of *Anopheles arabiensis* relative to *Anopheles gambiae*. Black paths were statistically significant at the $\alpha = 0.05$ level, while gray paths were not. Fisher's C statistic and associated P value from goodness-of-fit test (below path diagram) confirmed that the hypothesized model is consistent with data.

predictions by explicitly modeling the relationships between predictors and to calculate their relative contributions to disease transmission.

Methods

The data used in our analyses were collected by Mbogo et al. (2003). Briefly, they conducted entomological surveys at 30 villages along the eastern Kenyan coast from June 1997 to May 1998. Overnight human-landing catches were performed in 10 huts per village every 2 months over the course of the year-long study. Mosquito species were identified via molecular methods, and their abundances were recorded. At the end of the vector survey period (May 1998), 76–100 school children (ages 6–12) were sampled for malaria at each village. Endemic parasite prevalence (i.e., chronic and asymptomatic malaria) was estimated as the proportion of samples that showed blood-stage parasites in stained blood smears. *Plasmodium falciparum* was found to be responsible for more than 95% of the malaria cases in this region and thus is the only parasite species considered here. The longitudinal mosquito diversity data was aggregated (representing cumulative exposure to vectors over the entire study period) to match the cross-sectional nature of the malaria prevalence data. The data set we analyzed thus consisted of malaria prevalence, mosquito species richness, and mosquito species abundance from each village, with a sample size of 30 villages. We note that our

measure of species richness is unlikely to capture every mosquito species that occurs in a locality but instead provides an estimate of the species that people are likely to encounter in huts over the course of a year.

We performed a principle component analysis (PCA) on the relative abundance of mosquito species in each community. We retained only those axes that explained at least 25% of the variation in species relative abundance as our measures of mosquito community composition. Next, we constructed a series of linear models to assess the relationship between malaria prevalence and mosquito abundance, species richness, or community composition (equivalent to assessing paths *a*, *b*, and *c* in fig. 1A independently; see app. B; apps. A–C are available online). We first modeled each predictor on its own and then simultaneously in a multiple regression (app. B). We checked for spatial autocorrelation (geographic coordinates of the villages obtained from the Malaria Atlas Project database and provided in app. C; Hay and Snow 2006) and confirmed that malaria prevalence and mosquito diversity measures were not correlated between spatially proximate villages (Moran's *I*: [−0.05, 0.05]; $P > .1$ for all).

In order to account for potential relationships between predictors (fig. 1A, paths *d* and *e*) and to test all hypotheses under one unifying framework, we conducted a path analysis on the hypothesized full model, as depicted in figure 1A, using the R package *piecewiseSEM* (Lefcheck 2016). The rationale for the model structure, including directionality of relationships, is detailed in table 3. All statistical

Table 3: Rationale for each modeled path, their standardized effect sizes (and standard errors) from path analysis, and interpretation of observed relationships

Path	Rationale	Magnitude	Interpretation
a. Abundance → malaria prevalence	High vector abundance increases vector–host contact rates, which in turn increases disease transmission.	.36 (.032)	There is no direct effect of vector abundance on malaria prevalence.
b. Species richness → malaria	Species richness may dilute or amplify disease risk.	.48 (.12)*	High vector species richness results in high malaria prevalence.
c. Composition → malaria prevalence	Certain vector species combinations achieve high disease transmission, for example, via efficient niche partitioning.	−.58 (.17)*	A high <i>Anopheles arabiensis</i> to <i>Anopheles gambiae</i> ratio is associated with low malaria prevalence.
d. Abundance → species richness ^a	When local communities result from sampling a larger regional pool, high-abundance samples will include more species.	.65 (.04)*	High vector abundance sites consist of more species.
e. Species richness → composition	If certain species are ubiquitous, composition will converge in depauperate communities.	.54 (.095)*	Species-depauperate communities tend to be dominated by <i>An. gambiae</i> , in particular, relative to <i>An. arabiensis</i> . This ratio decreases as species richness increases.

^a The directionality of this path can, in theory, be reversed (e.g., high species richness leads to high abundance via niche complementarity). Here, we specified our model after Grace et al. (2007), who found abundance → species richness to dominate in natural systems. Competing alternative versions of our model (fig. 1A, with path *d* in the opposite direction; fitted with R package *lavaan* [Rosseel 2012]) confirmed that the model with abundance → species richness has a much better fit according to the corrected Akaike information criterion (−10.75 vs. 65.33).

* $P < .05$.

analyses were conducted with R statistical software version 3.5.0 (R Development Core Team 2018).

Results

A PCA of the relative abundance of mosquito species across 30 villages in Kenya (from Mbogo et al. 2003) showed that PC1 was occupied primarily by *Anopheles funestus*, while *Anopheles gambiae* and *Anopheles arabiensis* loaded onto PC2 in opposite directions (fig. 2). Together, these two axes explained >90% of the variance in the vector data. Results of univariate regression models are presented in appendix B. In brief, we confirmed that both mosquito abundance and community composition (PC2 only) were correlated with species richness (Pearson correlation coefficient = 0.45 and 0.53, respectively; $P < .01$ for both). Abundance and species richness were positively associated with malaria prevalence (fig. 3; table B1; tables B1, C1 are available online). Only PC2 (and not PC1) predicted malaria prevalence (table B1); therefore, we used this axis to represent community composition for the remainder of the analyses.

The path analysis showed that higher species richness (fig. 1B, path *b*) and changes in community composition that were more likely at high richness (fig. 1B, paths *c*

and *e*) had the largest impacts on malaria prevalence. Consistent with our prediction of an amplification effect due to variation in vector phenology, an increase in species richness caused an increase in malaria prevalence (fig. 1B; path *b* was positive). A high *An. arabiensis* to *An. gambiae* ratio was associated with decreased malaria prevalence (path *c* was negative), and this particular combination occurs at high vector species richness (path *e* was positive). This indirect dilution effect was relatively weak (obtained by multiplying the path coefficients of *c* and *e*: $0.54 \times -0.58 = -0.31$), causing the overall effect of species richness to be positive (obtained by summing the direct and indirect effects, paths *b* + *c* × *e*: $0.48 + (-0.31) = 0.17$). Mosquito abundance had only an indirect influence on malaria prevalence through its impact on species richness (fig. 1B; path *a* in gray was not significant, but paths *b* and *d* were). In sum, we found that villages with high malaria prevalence were characterized by high mosquito species richness and had a high *An. gambiae* to *An. arabiensis* ratio.

Discussion

In this study, we investigated several axes of vector diversity as drivers of disease risk and found that increasing mosquito species richness leads to a net increase in prevalence of

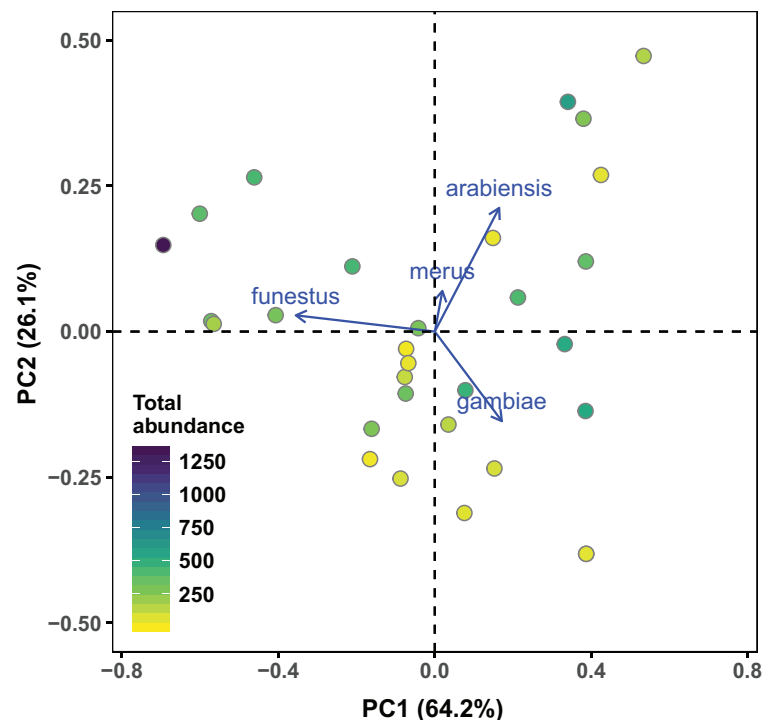


Figure 2: Biplot depicting similarities in relative abundances of mosquito species at each site. Principle component (PC) 1 explained 64.2% of the variation in the data, while PC2 explained 26.1%. Blue arrows = species loadings; circles = sites. Color scheme corresponds to total vector abundance at each site.

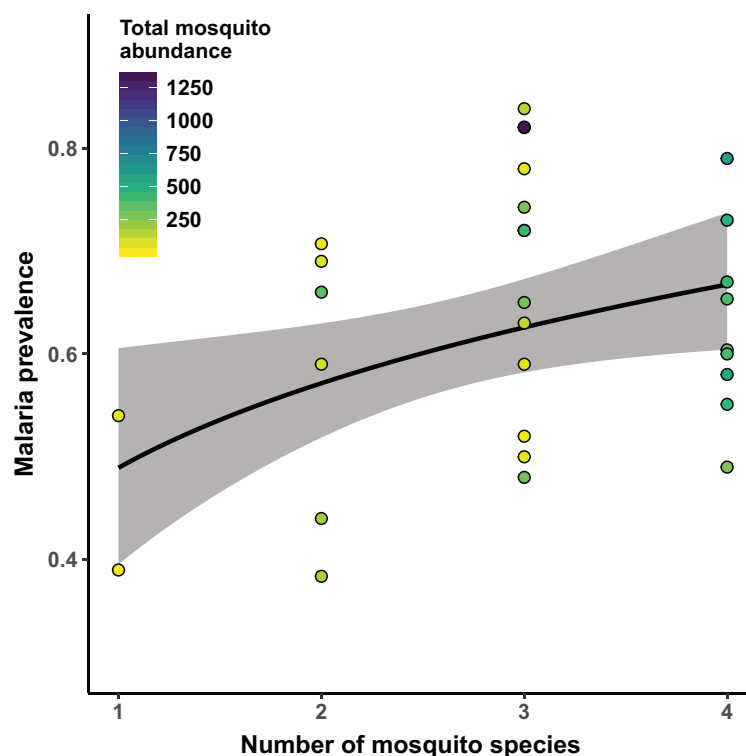


Figure 3: Relationships between mosquito species richness, abundance, and malaria prevalence. Malaria prevalence is positively related to mosquito species richness. Black line = predicted malaria prevalence from linear model with number of mosquito species as predictor ($\beta = 0.22$, $SE = 0.10$, $P = .03$; see table B1 [available online], model 1). Shaded area = 95% confidence interval of predicted values. Each point represents a single village in Kenya, as reported in Mbogo et al. (2003), and the colors show a positive relationship between mosquito abundance and malaria prevalence ($\beta = 0.06$, $SE = 0.03$, $P = .03$; see table B1, model 2).

malaria (figs. 1B, 3). The two components that made up this amplification effect were (1) a strong direct positive effect of species richness on malaria prevalence and (2) a weak indirect negative effect of richness mediated through community composition, which occurred only when the relative abundance of *Anopheles gambiae*—known to be a key vector of malaria parasites (Sinka et al. 2010)—decreased with increasing species richness. These results point to the complex nature of vector diversity and disease relationships, where any effect of species richness can be modified by the specific species composition present. Our path analysis was also able to distinguish between the influence of species richness and abundance on malaria prevalence, and counterintuitively, we found no significant direct effect of vector abundance on malaria prevalence (fig. 1B, path *a*). Although these results are broadly similar to those obtained from multiple regression (app. B) and other published reports (Park et al. 2015; Martínez-De La Puente et al. 2018), the path analysis was able to disentangle the direct and indirect pathways through which diversity can impact disease prevalence, and it revealed simultaneous dilution (indirect via community composition) and amplification

(direct) effects of species richness in this system (Luis et al. 2018).

Principles of community assembly have been instrumental in elucidating plausible effects of species richness on disease risk (tables 2, 3). In addition to the direct effect of species richness, associated shifts in abundance and composition as species richness increases can influence the distribution of vector competence in a community and therefore the number of effective bites a focal host population will receive. Species richness can amplify disease risk due to total (or individual species) abundance rising simultaneously, dilute disease risk when individuals of one species are replaced by those of a less competent species, or have little to no effect when replaced by individuals with similar competence. Our path analysis suggests that in our system, abundance only influenced malaria prevalence indirectly through its effect on richness (fig. 1B; path *a* was not significant). However, the strong correlation between abundance and species richness here and in the literature more broadly suggests that in the absence of other information, total vector abundance could act as a surrogate to capture some of the consequences of higher species richness.

The variation in community composition across sites (fig. 2) suggests that abundances of different species accumulated at different rates. More specifically, although *An. gambiae* was ubiquitous (fig. A1), it tended to dominate at low-abundance sites, while *Anopheles funestus* dominated higher-abundance communities (fig. 2). The rate of increase of *An. gambiae* abundance therefore slowed as species richness increased. This effect was captured in part by the positive association between community composition (PC2 as proxy) and species richness (fig. 1A, path *e*). Despite species-rich communities harboring a greater proportion of the more generalist vector, *Anopheles arabiensis*, relative to the human specialist vector, *An. gambiae* (fig. 1A, path *e*; table 1), we found that increasing species richness tended to increase malaria prevalence. We hypothesize that malaria transmission could be mostly driven by *An. gambiae*, but the other species, despite lower affinity for humans, fill transmission gaps in time and/or space. Since the most common species in our study area, *An. gambiae*, is also the only rainy season species, the presence of any other species is likely to prolong transmission into the dry season (table 1; Minakawa et al. 2002). This highlights the often neglected roles of opportunistic vector species, as they may possess other traits important to disease transmission (Kiware et al. 2012; Waite et al. 2017). Understanding the correlations between traits in vectors (e.g., competitiveness, host-seeking behavior, compatibility with parasite, phenology) and how they scale up to influence community assemblage and function will be an asset in making accurate predictions about diversity-disease relationships (Strauss et al. 2015).

The complex epidemiology of malaria involves seasonal transmission driven by climate-regulated mosquito population dynamics (Selvaraj et al. 2018) as well as chronic infections that may persist for many months. Thus, while the malaria prevalence data reported in Mbogo et al. (2003) was collected in May 1998, it is impossible to tell when those infections were acquired. The appropriate comparator vector data are also not intuitive (Gu et al. 2003; Selvaraj et al. 2018). We chose to correlate malaria prevalence with vector abundance and diversity measures aggregated over the year-long study (June 1997–May 1998). This aggregation provides an estimate of the cumulative exposure to vectors and is akin to annual entomological inoculation rate (number of infectious bites per person per year), which is a common metric of malaria endemicity and transmission intensity (Beier et al. 1999; Hay et al. 2000). Nonetheless, we do caution that the temporal aggregation of vector diversity data may lead to biased effect estimates if there is high turnover in the species that drive transmission (Sajjad et al. 2017). Future studies that explicitly track disease incidence and host/vector population dynamics over multiple transmission seasons will be crucial for pinpointing mechanisms (e.g., Park et al. 2015).

This study emphasizes the importance of considering multiple axes of vector diversity when investigating correlates of disease risk, and future work could extend this in a couple of ways. The data set we analyzed considered a single mosquito genus collected during in-hut catches (Mbogo et al. 2003). In-hut catches may result in underestimation of the abundances of outdoor-feeding species (although the species reported here exhibit similar propensities to feed indoors vs. outdoors; table 1), while employing mixed survey design can improve the accuracy in quantifying vector abundance and community composition (Killeen et al. 2017). Although the genus *Anopheles* is responsible for malaria transmission, if ecological interactions among mosquito genera are important (e.g., competition for breeding sites), then studies that capture this broader diversity will be important. Our analyses also cannot account for any variation in abundance or composition of species fed on by the vectors. Although humans are the only major host species for *Plasmodium falciparum*, diversity in available sources of blood meals could produce a dilution effect if mosquito feeding patterns track that availability (Roche et al. 2013). This interaction of biodiversity at different trophic levels has been studied in avian malaria and West Nile virus systems, which encompasses multiple bird, mosquito, and parasite species. In those systems, malaria prevalence in birds depends on mosquito species richness (Martínez-De La Puente et al. 2018), vector host preference (Martínez-De La Puente et al. 2018), contact rate between hosts and vectors (Medeiros et al. 2015), geographic range of vectors (Fecchio et al. 2017), and parasite-host compatibility when vectors are all generalists (Medeiros et al. 2013). While the diversity of *Anopheles* species captured in our study, with distinct feeding preferences (table 1), may capture some of the consequences of diversity at other trophic levels, these remain mostly unexplored for vector-borne diseases. Future work in this area will not only enrich our understanding of community assemblage and functioning but also have implications for applied ecology as well.

Vector management plays an integral role in the control of malaria and other diseases (Godfray 2013). Knowledge of how mosquito communities are structured and function can bring important insights on how to design effective management programs, for example, by suggesting particular species or sites to target and enabling predictions for how those communities will respond following intervention. Vector management for the control of malaria aims to decrease mosquito density (e.g., through the application of insecticides) and/or decrease host-vector contact rate (e.g., through the distribution of bed nets), but any strategy may have unintended consequences for the broader vector community. For instance, depending on the relative sensitivity and behavior of each species, insecticides could

lead to competitive release of competent vector species (Coetzee et al. 2000; Godfray 2013; Mwangangi et al. 2013). Further, if competent vectors have high abundance at broader spatial scales, then they may be able to recolonize cleared-out patches quickly, compromising the efficacy of a localized eradication strategy (Laporta et al. 2013). The efficacy of bed nets for reducing contact rates between hosts and vectors could similarly be compromised if the vector communities are dominated by outdoor, opportunistic feeders. In such areas, so-called zooprophylaxis may be a successful alternative (Asale et al. 2017). This strategy, dating back to the 1950s, introduces alternative sources of blood meals (such as domesticated animals) with the expectation of diverting mosquitoes away from humans (Macdonald 1956; Asale et al. 2017).

There is no doubt that managing vectors is key for controlling disease transmission. Taking a community ecology perspective can help shape vector management strategies by shedding light on the characteristics that make certain communities the most important ones to target. We as well as others (Roche et al. 2013; Park et al. 2015; Martínez-De La Puente et al. 2018) have demonstrated that the predicted beneficial effects of diversity for disease risk (i.e., the dilution effect) do not necessarily apply to vectors. Although we know of no one advocating for the conservation of, for example, mosquitoes, moving forward it will be important to determine if and how maintaining diversity in host communities influences diversity of any associated vector communities. Put another way, could attempts to reduce the risk of one disease inadvertently increase the risk of another? Preserving biodiversity has intrinsic value and many important benefits, but its impact on disease prevalence is unlikely to be straightforward and should be promoted with caution.

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Statement of Authorship

A.G.H. conceived of the study. All authors participated in data analysis. A.G.H. drafted the manuscript, and B.G.

and N.M. supervised in the preparation of the final version. All authors read and approved the final version of the manuscript.

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“The engraving illustrating this article, brings out clearly some of the characteristic features of the scenery of the coast of Labrador. In the foreground the rocky shore of the Horsechops, as the deep fiord is called, which is situation far up on the eastern coast of Labrador, has been ground down, smoothed and polished by the great mass of land ice which formerly filled Hamilton bay and moved slowly down from the tableland in the interior, and whose ice front must have presented to the sea a wall, perhaps 500 to 1000 feet high, at the end of which was probably a submarine bank or terminal moraine like those known to exist at the present day on the coast of Greenland and Spitzbergen.” From “Glacial Marks in Labrador” by A. S. Packard Jr. (*The American Naturalist*, 1882, 16:30–33).