Received: 31 July 2020 | Revised: 25 September 2020 | Accepted: 6 November 2020

DOI: 10.1111/tbed.13918

ORIGINAL ARTICLE

Transboundary and Emerging Diseases WILEY

Large herbivore loss has complex effects on mosquito ecology and vector-borne disease risk

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Funding information
This study was inspired by project results from an Ecology of Infectious Disease field course funded by a United States National Science Foundation (NSF) CAREER Award to VOE (IOS-1101836). Financial support for this project came from the University of Georgia and NSF Long-Term Research in Environmental Biology Grants 12-56004 and 19-31224. This study was also supported by a postdoctoral fellowship that was awarded to DPT through *icipe* from the UK's Foreign, Commonwealth & Development Office (FCDO) and the Swedish International Development Cooperation Agency (Sida). We also acknowledge financial support from the Swiss Agency for Development and Cooperation (SDC), Federal Democratic Republic of Ethiopia and the Government of the Republic of Kenya. The views expressed in this manuscript do not reflect the official opinion of the donors.

Abstract

Loss of biodiversity can affect transmission of infectious diseases in at least two ways: by altering host and vector abundance or by influencing host and vector behaviour. We used a large herbivore exclusion experiment to investigate the effects of wildlife loss on the abundance and feeding behaviour of mosquito vectors and to explore consequences for vector-borne disease transmission. Large herbivore loss affected both mosquito abundance and blood-feeding behaviour. For *Aedes mcintoshi*, the dominant mosquito species in our study and a primary vector of Rift Valley fever virus (RVFV), abundance decreased with large herbivore loss, while blood feeding on humans increased. Despite an elevated human biting rate in the absence of large herbivores, we estimated that the potential for RVFV transmission to humans doubles in the presence of large herbivores. These results demonstrate that multiple effects of biodiversity loss on vectors can lead to counterintuitive outcomes for human disease risk.

KEYWORDS

arbovirus, biodiversity, mosquito-borne disease, vector, vectorial capacity

1 | INTRODUCTION

The idea that the loss of biodiversity can increase the risk of infectious diseases in animals and humans is a topic of ongoing interest (Cardinale et al., 2012; Civitello et al., 2015; Keesing et al., 2010; Rohr et al., 2019; Salkeld et al., 2013; Wood et al., 2014). Most empirical studies on this issue focus on how pathogen prevalence or abundance in focal hosts or vectors varies in response to changes in biodiversity (reviewed in Ostfeld & Keesing, 2012). For example, an observational study in Louisiana, USA, found that the prevalence of West Nile virus (WNV) infection in mosquito vectors was strongly and negatively related to the diversity of non-passerine bird species occurring across sites (Ezenwa et al., 2006). Similarly, a manipulative study in Kenya, in which loss of diversity was simulated via the exclusion of large herbivores, showed that the densities of flea vectors and rodent hosts infected with *Bartonella* nearly doubled in plots missing large herbivores (Young et al., 2014). Given that both WNV and *Bartonella* are transmitted by generalist vectors that feed on both wildlife reservoirs and human hosts, these studies support the idea that biodiversity loss can modify human risks of acquiring vector-borne diseases.

There are several mechanisms that can account for why biodiversity is linked to vector-borne disease risk (Johnson & Thieltges, 2010; Keesing et al., 2006). For example, the presence of a diverse community of hosts for a vector might simply divert vector bites away from the most effective (i.e. competent) pathogen hosts, thereby disrupting transmission (Keesing et al., 2006). It is also possible that competitive interactions between species (including hosts and non-hosts) in a diverse community might suppress the population density of effective hosts, consequently dampening transmission (Keesing et al., 2006). These two types of mechanisms, whereby diversity alters aspects of vector or host behaviour and abundance, respectively, are the most frequently cited explanations for diversity-disease patterns (reviewed in Keesing et al., 2010). However, these mechanisms are rarely studied in tandem, yet their combined effects may be critical to fully understanding when biodiversity loss might substantially alter infectious disease risk.

A notable example of the synergism that can arise between behaviour and abundance-driven effects of biodiversity comes from studies of the tick-borne agent of Lyme disease, *Borrelia burgdorferi*. In the north-eastern United States, white-footed mice are the most competent reservoir host for *B. burgdorferi*; however, mouse densities tend to be lower in habitats with high abundance and richness of other vertebrate species (reviewed in Ostfeld & Keesing, 2012). Moreover, the presence of other species, particularly chipmunks, diverts ticks away from mice (Brunner & Ostfeld, 2008). Thus, differences in both host abundance and vector feeding behaviour in more diverse habitats appear to contribute to associations between host diversity and disease prevalence in the Lyme disease system.

For mosquito-borne diseases, vector abundance and vector behaviour are key drivers of disease transmission because transmission potential (i.e. vectorial capacity) depends, in part, on both the density of vectors and number of bites an infectious host receives (Garrett-Jones, 1964). Intriguingly, deforestation, which is often accompanied by diversity loss, has been linked to changes in both the abundance and biting behaviour of mosquito vectors of human malaria (Vittor et al., 2006; Yasuoka & Levins, 2007). In the Peruvian Amazon, for example, human biting rates of the malaria vector, *Anopheles darlingi*, were over 200 times higher in deforested compared to forested sites (Vittor et al., 2006). Although this change in vector behaviour was largely attributed to mosquito preferences for breeding sites, the potential direct or indirect contributions of diversity loss to such patterns are not well understood.

In this study, we investigated how biodiversity loss might impact vectors in ways that alter disease transmission to humans. Specifically, we examined the effects of simulated wildlife loss on the abundance and behaviour of mosquitoes. Mosquitoes are important vectors of a range of infectious diseases, many of which are zoonoses that affect both humans and animals, such as Rift Valley fever virus (RVFV), WNV and Yellow fever virus (Gubler, 1998; Sang & Dunster, 2001). We took advantage of a long-term replicated herbivore exclusion experiment, the Kenya Long-term Exclosure Experiment (KLEE), to examine how the loss of large native herbivorous mammals affected the abundance and blood-feeding behaviour of mosquitoes, including known vector species. KLEE was established in 1995 in an acacia savannah woodland, and the experiment has had profound effects on local plant and animal communities. Documented effects of the KLEE herbivore manipulations on animals include changes in the abundance and/or diversity of larger mammals (Kimuyu et al., 2017), small mammals (Keesing, 1998), birds (Ogada et al., 2008), reptiles (McCauley et al., 2006; Pringle et al., 2007) and invertebrates (Keesing et al., 2013; Pringle et al., 2007). The effects on plants are even more profound, ranging from changes in species diversity, plant biomass and canopy cover (reviewed in Goheen et al., 2018). Given these exclusion-associated changes in vegetation structure and blood meal host availability, we expected large herbivore exclusion to have potential direct and indirect effects on mosquito abundance and behaviour. We also quantified the potential repercussions of large herbivore loss for the transmission of mosquito-borne diseases by estimating the effect of herbivore exclusion on vectorial capacity for RVFV, a zoonotic pathogen of high relevance in African savannah ecosystems.

2 | MATERIALS AND METHODS

2.1 | Study site

Mosquitoes were sampled in the KLEE located in Laikipia County, Kenya (0°17.477′ N, 36°51.885′ E). KLEE consists of six adjacent 200 m × 200 m treatments, each replicated in three blocks (North, Central and South), that exclude different combinations of wild and domestic herbivore species (Young et al., 1998). Wild herbivore

exclusion is maintained with electric fencing. KLEE is located in a habitat with a diverse complement of large mammalian herbivores, including the mega-herbivores elephant (*Loxodonta africana*) and giraffe (*Giraffa camelopardalis*), and seven species of meso-herbivores including buffalo (*Syncerus caffer*), plains zebra (*Equus burchelli*) and Grant's gazelle (*Nanger granti*). The three KLEE wildlife treatments include (a) full exclusion of all herbivores over ~15 kg, (b) exclusion of mega-herbivores only and (c) full access to all herbivores. To examine the effects of complete large herbivore loss on mosquitoes, we focused our study on two of these three wildlife treatments: a versus c, representing full exclusion of all large herbivores versus full access to all larger herbivores. In total, we sampled one plot with large herbivores and one without in each of the three replicate blocks. The KLEE study site is routinely used by researchers for studies that involve comparisons in plots with and without large herbivores, with different treatment types used at the same relative frequency.

2.2 | Mosquito trapping

Mosquitoes were sampled in KLEE during three trapping sessions held in July 2013, July 2014 and May 2015. Rainfall in Laikipia County is weakly trimodal with a primary peak in April–May and secondary peaks in June–July and October–November, although rainfall patterns can be highly variable across years (Keesing & Young, 2014). Based on actual rainfall patterns in the month preceding each trapping session, the two July trapping sessions were classified as dry periods (total rainfall: June 2013 = 0 mm and June 2014 = 0 mm) and our May 2015 session as a wet period (total rainfall: April 2015 = 131 mm).

Mosquitoes were sampled for five consecutive nights within each KLEE block using CO_2-baited CDC miniature light traps (John W Hock, model 512). CO_2 (dry ice) bait has been shown to increase the capture rate of many mosquito species (Tchouassi, Quakyi, et al., 2012; Tchouassi, Sang, et al., 2012; Tchouassi et al., 2019), and most of the species in our study region have previously been trapped in high numbers using CO_2-baited light traps (Arum et al., 2015; Sang et al., 2010). Within each sampling block, trapping occurred simultaneously in plots with and without large herbivore. Two mosquito traps were set at the centre of each 200 m × 200 m treatment plot, one at a height of 1 m and one at a height of 3 m. Traps were set between 1,630 and 1,730 hr and retrieved approximately 13–14 hr later. Given a flight distance of key mosquito species in our study region of typically less than 200 m (e.g. reported flight range of adult female *Aedes mcintoshi* = 150 m; Linthicum, Bailey, et al., 1985; Linthicum, Kaburia, et al., 1985), the positioning of our traps should have been sufficient to sample locally available mosquitoes. Trapped mosquitoes were immobilized using triethylamine, sorted, and placed into 1.5 ml tubes, and stored in liquid nitrogen for transport to the laboratory in Nairobi. Once in the laboratory, specimens were stored at −80°C until morphological identification. All mosquitoes were identified to species level using published taxonomic keys (Edwards, 1941; Gillett, 1972; Jupp, 1996).

2.3 | Blood meal analyses

To evaluate mosquito feeding behaviour, engorged mosquitoes were separated from other specimens for blood meal analysis. We focused the blood meal analyses on the dominant mosquito species from the May 2015 trapping session, *Ae. mcintoshi*, which accounted for the vast majority (75%–90%) of blood-fed specimens. Forty-three individual blood-fed *Ae. mcintoshi* out of a total of 48 blood-fed mosquitoes captured were processed, comprising 19 out of 2,962 (0.64%) specimens from plots without large herbivores and 24 of 4,247 (0.57%) specimens from plots with large herbivores. We extracted genomic DNA from all blood-fed specimens using the Qiagen DNeasy Blood and Tissue Kit (Qiagen, GmbH Hilden, Germany) per the manufacturer's instructions. DNA was amplified targeting a 500 bp fragment of the 12S mitochondrial rRNA gene using the primers 12S3F [5′-GGGATTAGATACCCCACTATGC-3′] and 12S5R [5′-TGCTTACCATGTTACGACTT-3′] (Roca et al., 2004). PCRs were performed using the MyTaq HS Mix kit (Bioline, Germany), in a final volume of 10 μl containing 10 M of each primer, 5 μl of 2xmytaq HS mix and 2 μl of DNA. The cycling parameters were 95°C for 3 min, followed by 35 cycles of 20 s at 95°C, 30 s at 56°C and 30 s at 72°C, and 72°C for 4 min. Amplicons were sized by 1.5% agarose gel electrophoresis against a 100 b DNA ladder (O'Gene Ruler, Fermentas, Fisher Scientific, UK). PCR products were purified using ExoSAP-IT (USB, Cleveland, OH, USA) prior to sequencing. DNA sequences were compared using the BLAST algorithm and the GenBank database (http://blast.ncbi.nlm.nih.gov/Blast.cgi). Species-level identification was determined when sequences exhibited ≥98% identity spanning at least 300 bp as previously described (Valinsky et al., 2014). Overall, blood meals were successfully identified from 16 of 19 (84%) and 18 of 24 (75%) blood-fed specimens from plots without and with large herbivores, respectively.

2.4 | Vectorial capacity estimation

Since *Ae. mcintoshi* plays a key role in the transmission of RVFV in Kenya (Sang et al., 2010; Tchouassi et al., 2014), we quantified the potential for large herbivore loss to affect disease risk by focusing on the ability of this mosquito to transmit RVFV. Specifically, we estimated the daily rate at which new infections with RVFV might arise from a single infected mosquito (i.e. vectorial capacity, VC) in the presence versus absence of large herbivores as:

$$VC = \frac{ma^2 e^{-\mu / EIP}}{\mu}$$

where m is the mosquito density, *a* is the human biting rate, μ is the daily probability of adult mosquito mortality, and EIP is the extrinsic incubation period (in days) of RVFV in *Ae. mcintoshi* (Garrett-Jones, 1964; Massad & Coutinho, 2012). A parameter for vector competence (i.e. the probability that a mosquito becomes infected and transmits virus) is often included in the expression for VC given by the equation:

VC = $ma^2bp^n/-\log e\, p$ (Kramer & Ciota, 2015), where m and a are as defined previously, p = the probability of daily survival, n = the extrinsic incubation period (EIP) and b = the vector competence. However, we did not include a term for competence in our estimate since we did not evaluate possible differences in vector competence across herbivore treatments in our study.

We estimated the mortality term (μ) for the VC equation as 1- the daily survival rate. Survival was calculated based on parous rates (i.e. proportion of females that have laid eggs) as described (Davidson, 1954), using the formula $p^n = M$ where p is the daily survival rate, M is the proportion of the population which is parous, and n is the number of days between emergence of adult females and first oviposition. Parity is widely used as proxy for estimating mosquito survival (Churcher et al., 2015; Reeves, 1965; Tchouassi, Quakyi, et al., 2012; Tchouassi, Sang, et al., 2012). We assumed an n value of 3 for *Ae. mcintoshi* following (Arum et al., 2016), and dissected the ovaries of randomly selected *Ae. mcintoshi* females collected during the wet sampling period for parity by scoring specimens as nulliparous or parous based on the degree of dilatation of the tracheolar skein (Detinova, 1962). Between 5% and 9% of the total number of *Ae. mcintoshi* specimens collected per treatment type were dissected, including 199 of 2,223 (8.9%) specimens from plots without large herbivores and 187 of 3,821 (4.9%) specimens from plots with large herbivores. This sample size exceeds those used for estimating survival for *Ae. mcintoshi* during RVFV outbreak situations in Kenya (Sang et al., 2010), and for *Anopheles* species in relation to VC for *Plasmodium* transmission (Ndoen et al., 2012; Pan et al., 2012), so should provide a good estimate of survival for RVFV VC estimation.

Finally, because RVFV is a zoonotic pathogen for which animal hosts contribute to transmission (Hoogstraal et al., 1979), we accounted for zoonotic transmission in the VC expression by modifying the a^2 term. In the classical VC equation, the squared human biting rate reflects both the contact rate between mosquitos and humans that leads to acquisition of infection by mosquitos and the contact rate that leads to transmission back to humans. Thus, for a pathogen with a human reservoir, the squared biting rate accounts for the fact that a mosquito must bite a human host twice for transmission to occur. This parameter, a, can be separately represented by feeding parameters in systems where transmission to humans involve a zoonotic blood meal (LaDeau et al., 2015). For the zoonosis case as is RVFV, we decomposed a^2 into $a_x \times a_y$, where a_x represents the contact rate between mosquitoes and all hosts that contribute to mosquito infection (hereafter called 'reservoir host biting rate'), and a_y represents the contact rate between mosquitoes and humans that leads to pathogen transmission (human biting rate). We used blood meal analysis data to calculate biting rates by first estimating frequencies of *Ae. mcintoshi* blood feeding (by herbivore treatment) as the proportion of blood-fed *Ae. mcintoshi* times the total number *Ae. mcintoshi* collected per trap night. Then, to estimate reservoir host (a_x) and human (a_y) biting rates, this value was adjusted to account for the combined *Ae. mcintoshi* feeding rate on all RVFV reservoir hosts (humans and African buffalo) or the feeding rate on humans only. We considered African buffalo to be a viable reservoir host for RVFV given evidence that this species plays a role in the persistence of the virus between outbreaks (Beechler et al., 2015; LaBeaud et al., 2011). Finally, since the KLEE study site is routinely used by human researchers (with all treatment types used at the same relative frequency), we expected to be able to calculate a biting rate for humans using our blood meal data.

2.5 | Statistical analyses

Mosquito counts over each 5-day collection period for each study block were aggregated by treatment plot and trapping period to test for effect of large herbivore presence in a plot on total and species-specific mosquito abundance. To deal with different seasonal mosquito abundance patterns, data for the dry periods (July 2013 and July 2014) and wet period (May 2015) were analysed separately. For the dry season analyses, total mosquito abundance and the abundance of two dominant individual species (*Culex univittatus* and *Culex pipiens*, Table S1) served as response variables in separate generalized linear models (GLM) with a Poisson error structure. The main predictor variable was large herbivore presence/absence. Replicate block (North/Central/South) and sampling session (July 2013/July 2014) were also included as covariates in the *Cx. univittatus* model. The *Cx. pipiens* model included only block as a covariate since this species was not encountered in the 2014 sampling session. For the wet season analyses, total mosquito abundance and the abundance of five species accounting for >98% of collections (*Ae. mcintoshi*, *Aedes hirsutus*, *Cx. univittatus*, *Cx. pipiens* and *Aedes tricholabis*, Table S1) served as response variables in separate GLMs. A Poisson error structure was used for *Ae. hirsutus*, *Ae. tricholabis* and *Cx. pipiens*, and a negative binomial error structure was used for the other species. Large herbivore presence was included as the predictor variable in these models, and block was included as a covariate. All GLMs were implemented in R version 3.3.1 (R Core Team, 2013) with the MASS package. Model validity was assessed by inspection of residuals. Lastly, we used Pearson chi-squared tests to evaluate the effects of large herbivore presence on blood-feeding behaviour and parity in *Ae. mcintoshi*.

2.6 | Ethical Statement

Approval of the study was obtained from the Scientific Ethics Review Unit (SERU) of the Kenya Medical Research Institute (SSC No. 2346).

3 | RESULTS

3.1 | Abundance

A total of 7,287 mosquitoes from 17 species were collected across three sampling sessions, but most of the specimens (7,209, 98.9%)

were collected during the wet sampling period (Table S1). *Ae. mcintoshi* was the dominant species collected in May 2015 during the wet period, accounting for 83.8% of all collections. During the dry periods, *Cx. univittatus* dominated the 78 collections accounting for 60.5% of mosquitoes collected in July 2013 and both of the individual mosquitoes collected in July 2014.

TABLE 1 Effect of large herbivore exclusion on mosquito abundance during the dry season; models represent GLMs with Poisson distribution

	Total abundance ($df = 6, 6$)			*Culex univittatus* abundance ($df = 6, 6$)			*Culex pipiens* abundance ($df = 4, 8$)		
	β estimate ± *SE*	*Z* value	*p*	β estimate ± *SE*	*Z* value	*p*	β estimate ± *SE*	*Z* value	*p*
Large Herbivores Present: Yes	−0.77 ± 0.25	−3.13	<.002**	−0.83 ± 0.32	−2.58	.01**	−0.69 ± 0.43	−1.60	.11
Session: July 14	−3.23 ± 0.72	−4.52	<.0001***	−2.77 ± 0.73	−3.80	.0001***	-	-	-
Block: North	3.04 ± 0.59	5.15	<.0001***	3.63 ± 1.01	3.59	.0003***	19.90 ± 2,769.57	0.01	.99
Block: South	1.39 ± 0.65	2.15	.03*	2.20 ± 1.05	2.08	.04*	17.97 ± 2,769.57	0.01	1.00

Note: Central is used as reference category for analysis of block effects, and July 13 is used as reference for analysis of the effect of trapping session.
*$p < .05$,
**$p < .01$,
***$p < .001$.

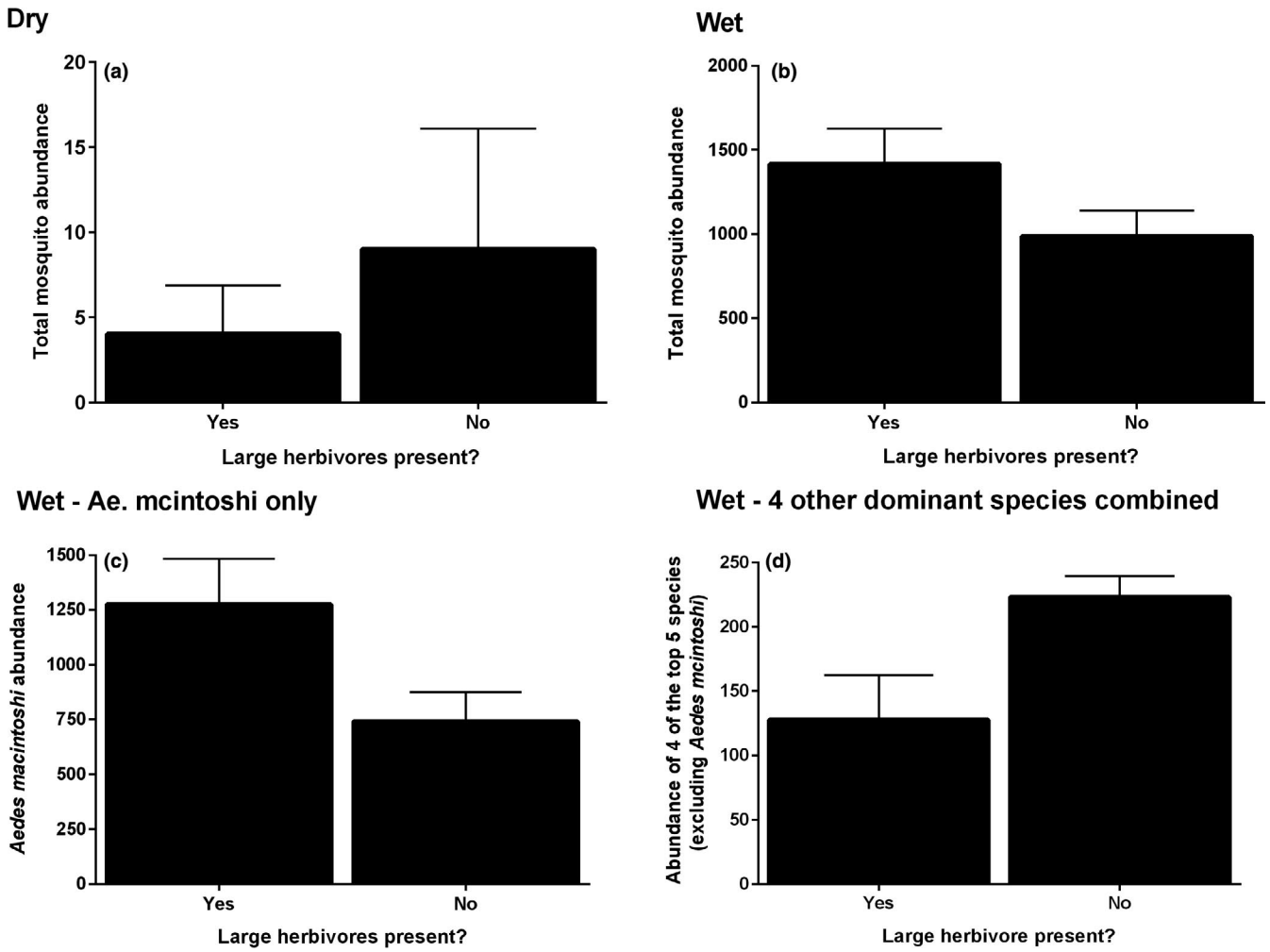

FIGURE 1 Mosquito abundance in the presence versus absence of large herbivores in the (a) dry compared to (b) wet sampling periods and for (c) *Aedes mcintoshi* in the wet season compared to (d) the other four dominant species combined (*Culex univittatus*, *Culex pipiens*, *Aedes hirsutus*, *Aedes tricholabis*) during this same period

Mosquito abundance was affected by the presence of large herbivores in both dry and wet periods. In the dry periods, plots with large herbivores had significantly fewer mosquitoes (Table 1; Table S2), with total mosquito abundance declining by half in plots with large herbivore (Figure 1a; Table S2). This pattern was driven by the dominant mosquito species captured during this period, *Cx. univittatus* (Table 1). While large herbivore presence also reduced the abundance of *Cx. pipiens*, there was no significant difference between plot types for this species. In contrast, in the wet sampling period, there were >50% more mosquitoes captured in plots with large herbivores (Figure 1b). This pattern was driven by *Ae. mcintoshi*, the dominant mosquito collected during this period (Figure 1c; Table 2). For the other four most common species, including two other *Aedes* species, *Ae. tricholabis* and *Ae. hirsutus*, and two *Culex* species, *Cx. univittatus* and *Cx. pipiens*, abundance was significantly lower in plots with large herbivores (Figure 1d; Table 2), similar to the pattern observed during the dry periods.

3.2 | Blood-feeding patterns

Out of 7,209 mosquitoes collected in May 2015 (4,247 from plots with large herbivores and 2,962 from plots without large herbivores), 48 were blood-fed. This included 43 *Ae. mcintoshi*, 2 *Cx. univittatus*, 1 *Cx. pipiens*, 1 *Ae. tricholabis* and 1 *Cx. theileri*. Twenty-six blood-fed specimens came from plots with large herbivores (~0.6% of mosquitoes collected) and 22 specimens from plots without large herbivores (~0.7% of mosquitoes collected). Of the 43 blood-fed *Ae. mcintoshi*, 24 (55.8%) were from plots with large herbivores and 19 (44.2%) were from plots without large herbivores. Thirty-four of these specimens were successfully analysed for blood meals (n = 18/24 from plots with large herbivores and n = 16/19 from plots without large herbivores) revealing six host species: humans (70.6% of blood meals), elephants (11.8%), buffalo (8.8%), plus nightjars (birds in the family Caprimulgidae), hartebeest (*Alcelaphus bucephalus*) and steenbok (*Raphicerus campestris*) (2.9% each). A greater number of host species served as blood meal sources in plots with large herbivores (n = 5), compared to plots without large herbivores, where only two host species, humans and nightjars, accounted for all blood meals (Figure 2). In fact, 94% (15 of 16) of blood meals taken in plots without large herbivores came from humans compared to only 50% (9 of 18) in plots with large herbivores; thus, mosquitoes from plots without large herbivores were significantly more likely to have fed on human blood (Pearson chi-squared test: $\chi^2 = 5.84$, $p = .02$).

3.3 | Survival

We estimated the parity (proxy for survival) of *Ae. mcintoshi* collected during the wet period in plots with and without large herbivores. The parous rate for plots with large herbivores was 76.5% (143/187) compared to 57.3% (114/199) in plots without large herbivores, and this difference was significant (Pearson chi-squared test:

TABLE 2 Effect of large herbivore exclusion on mosquito abundance during the wet season; models represent GLMs with negative binomial distribution (total mosquito abundance, *Aedes mcintoshi* and *Culex univittatus*) or Poisson distribution (*Aedes hirsutus, Aedes tricholabis* and *Culex pipiens*)

	Total abundance (*df* = 5, 2)			***Aedes mcintoshi* abundance (*df* = 5, 2)**			***Aedes hirsutus* abundance (*df* = 4, 2)**		
	β estimate ± *SE*	***Z* value**	***p***	**β estimate ± *SE***	***Z* value**	***p***	**β estimate ± *SE***	***Z* value**	***p***
Large Herbivores Present: Yes	0.36 ± 0.05	6.64	<.0001***	0.55 ± 0.07	7.30	<.0001***	−0.25 ± 0.09	−2.66	.01*
Block: North	−0.04 ± 0.07	−0.58	.56	−0.01 ± 0.09	−0.15	.88	−0.73 ± 0.14	−5.22	<.0001***
Block: South	−0.48 ± 0.07	−7.23	<.0001***	−0.53 ± 0.09	−5.73	<.0001***	0.33 ± 0.10	3.17	.002**
	***Aedes tricholabis* abundance (*df* = 4, 2)**			***Culex univittatus* (*df* = 5, 2)**			***Culex pipiens* (*df* = 4, 2)**		
	β estimate ± *SE*	***Z* value**	***p***	**β estimate ± *SE***	***Z* value**	***p***	**β estimate ± *SE***	***Z* value**	***p***
Large Herbivores Present: Yes	−0.48 ± 0.19	−2.55	.01*	−1.44 ± 0.33	−4.33	<.0001***	−0.49 ± 0.14	−3.48	.001**
Block: North	−1.11 ± 0.24	−4.73	<.0001***	−0.01 ± 0.38	−0.03	.97	−0.60 ± 0.16	−3.88	.0001***
Block: South	−1.25 ± 0.25	−5.03	<.0001***	−1.56 ± 0.42	−3.69	<.0001***	−1.36 ± 0.20	−6.65	<.0001***

Note: Central is used as reference category for analysis of block effects.

*$p < .05$,
**$p < .01$,
***$p < .001$.

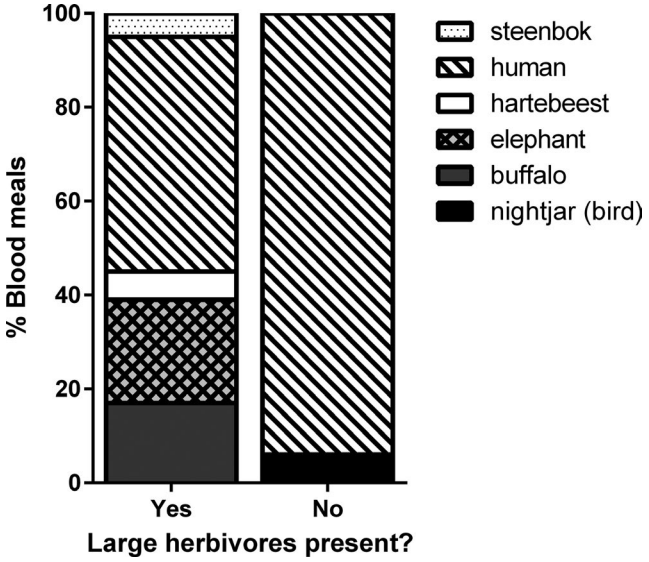

FIGURE 2 Proportion of *Aedes mcintoshi* blood meals coming from different host species in the presence versus absence of large herbivores

$\chi^2 = 15.10$, $p = .0001$). Daily survival as estimated from the parous rate was 0.91 for plots with large herbivores and 0.83 for plots without large herbivores indicating that *Ae. mcintoshi* survival was higher in plots with large herbivores. This difference reflects a 0.09 versus 0.17 daily mortality rate in plots with large herbivores compared to those without large herbivores.

3.4 | Vectorial capacity

We quantified the VC of *Ae. mcintoshi* for RVFV in the presence and absence of large herbivores by combining our wet season data on mosquito abundance, blood-feeding behaviour and mortality (Table 3). We used a fixed value of 7 days for the EIP based on experimental studies performed at a temperature of 26–27°C (Turell et al., 2008), which approximates the ambient temperature at our field site during the mosquito collections. Based on these parameters, we estimated VC as 300 in plots with large herbivores and 148 in plots without large herbivores (Table 3). Thus, the potential for *Ae. mcintoshi* to transmit RVFV effectively doubles in the presence of large herbivores.

4 | DISCUSSION

Large mammal biodiversity is being lost at an unprecedented rate across the globe, and the repercussions of these losses for ecosystem function and human well-being have garnered increasing attention (Cardinale et al., 2012; Díaz et al., 2006; Johnson et al., 2017; Johnson & Thieltges, 2010). We investigated the effects of the loss of large herbivorous mammals on mosquito ecology and evaluated the implications for vector-borne zoonotic disease transmission in an ecosystem where humans and wildlife coexist, but where large herbivore populations are threatened by the intensification of livestock production and other anthropogenic activities (Crego et al., 2020; Ogutu et al., 2016). We found that the exclusion of large wild mammals changed key aspects of mosquito ecology and behaviour. When large herbivores were absent from experimental plots, systematic differences in mosquito abundance, biting behaviour and survival emerged. First, the presence of large herbivores significantly decreased total mosquito abundance during dry periods, but this pattern was reversed during wet periods. However, the increase in wet season mosquito abundance in large herbivore plots was driven entirely by effects on a single dominant species, *Ae. mcintoshi*. Second, large herbivores deflected mosquito bites away from humans, and *Ae. mcintoshi* displayed a strong shift towards human blood feeding in response to large herbivore loss. Third, daily survival of this species was lower in plots without large herbivores. Given that *Ae. mcintoshi* serves as the major vector of RVFV, the causative agent of RVF, a vector-borne disease of concern in our study region (Sang et al., 2010; Tchouassi et al., 2014), we used our data on changes in *Ae. mcintoshi* abundance, biting behaviour and survival in the presence versus absence of large herbivores to estimate the impact of large mammal loss on the transmission efficiency of RVFV. RVFV causes periodic disease outbreaks in domestic ruminants, and humans become infected via mosquito bite (Linthicum et al., 2016). Accounting for the fact that both humans and wild ruminants potentially serve as maintenance hosts for the virus

TABLE 3 Parameters and data sources used for vectorial capacity (VC) estimation

Parameters	Definition	Large Herbivores present	Large Herbivores absent	Source
m	Mosquito density	127.4	74.1	This study
a_x	Reservoir host (human + buffalo) biting rate	0.535	0.590	This study
a_y	Human biting rate	0.401	0.590	This study
μ	Daily mortality	0.09	0.17	This study and Arum et al. (2016)
EIP	Extrinsic incubation period	7	7	Turell et al. (2008)
$VC = \frac{(ma_xa_y)e^{-\mu / EIP}}{\mu}$		300	148	

(Evans et al., 2008; Meegan, 1979; Swanepoel & Coetzer, 2004), we found that despite the shift in *Ae. mcintoshi* blood-feeding behaviour towards humans in plots without large herbivores, the potential for RVFV transmission was lower in these sites. These results underscore the importance of considering multiple mechanisms by which biodiversity loss can influence pathogen transmission when trying to predict how biodiversity might influence infectious disease risk.

The contrasting effect of wildlife exclusion on the abundance patterns of *Ae. mcintoshi* versus *Culex* and other *Aedes* mosquitoes was one of the striking findings of our study. This difference in response to large herbivore loss is most likely associated with variation in ecological and life-history attributes across mosquito species. For example, mosquito abundance patterns could be shaped by differences in host preferences. Many *Culex* species, including *Cx. pipiens* and *Cx. univittatus*, preferably feed on birds (Anderson et al., 2004). Previous work in the KLEE plots has shown that bird diversity increased in plots without large herbivores (Ogada et al., 2008), a pattern that may explain the positive effect of large herbivore loss on *Culex* abundance. Patterns of host availability might also contribute to the *Ae. mcintoshi* abundance pattern. A preference for larger mammals (e.g. cattle, goats, sheep) by *Ae. mcintoshi* has been reported throughout this species' range in East Africa (Linthicum, Bailey, et al., 1985; Linthicum, Kaburia, et al., 1985; Lutomiah et al., 2014; Tchouassi et al., 2016). Thus, the loss of similarly sized large mammal hosts from the plots without larger herbivores may help explain the negative effect of large herbivore loss on the abundance of this species. Indeed, our blood-feeding data suggest that in response to the loss of large wild mammals in the large herbivore exclusion plots, *Ae. mcintoshi* mosquitoes shifted their blood meal diets to the largest available mammal they could find (i.e. humans). The difference in the abundance patterns between *Ae. mcintoshi* and other *Aedes* species (*Ae. hirsutus* and *Ae. tricholabis*) is more difficult to explain. While data on blood-feeding pattern of *Ae. hirsutus* and *Ae. tricholabis* are scarce, a recent field study based on host choice showed that both species are attracted to and indeed feed on livestock hosts (Tchouassi et al., 2016). Given this, the difference in the effect of large herbivore exclusion on *Ae. mcintoshi* versus these other two *Aedes* species may be a consequence of processes other than host availability. Since all three of these *Aedes* species are floodwater mosquitoes that hatch in response to flooding (Arum et al., 2015; Tchouassi et al., 2016), one hypothesis is that *Ae. mcintoshi* might outcompete the other species in same habitats during larval development. Overall, our abundance findings indicate that large herbivore loss has differential effects on the abundance of *Ae. mcintoshi* and other key mosquito species at our study site. Coupled with the fact that at least three (*Ae. mcintoshi*, *Cx. pipiens*, *Cx. univittatus*) of the five dominant mosquitoes in our study serve as vectors for various arboviruses (e.g. RVFV, WNV), it seems likely that the implications of large herbivore loss for mosquito-borne disease transmission will vary depending on the specific vector species involved. Finally, it is important to note that our data on variation in *Aedes* abundance patterns in response to large herbivore loss come from a single wet season sampling period, so future longitudinal studies will be crucial for identifying specific drivers of the patterns we describe here, including the impacts of seasonality and the variable responses of different mosquito species.

For mosquitoes, blood feeding controls opportunities for pathogen infection; thus, identifying the factors that influence mosquito blood-feeding behaviour is crucial for understanding patterns of disease transmission (Kilpatrick et al., 2007; Reeves, 1965). We used blood-fed *Ae. mcintoshi* to investigate differences in blood-feeding behaviour in response to large herbivore loss. In plots with large herbivores, *Ae. mcintoshi* fed on a diverse group of hosts, including wild mammals and humans, with the majority of blood meals coming from humans (50%), elephants (22%) and African buffalo (17%). In plots without large herbivores, these mosquitoes shifted their blood meal sources almost exclusively to humans (94%), suggesting that large herbivores play a substantial role in deflecting *Ae. mcintoshi* bites away from humans. The very high human feeding rates we uncovered for *Ae. mcintoshi* in this study contrast sharply with past findings on human blood-feeding behaviour in this species where humans accounted for between 0.2% and 5.1% of all blood meals recorded. It is worth noting that the blood meals are most likely from researchers who access these plots and the area is not inhabited by humans. Previous studies report that *Ae. mcintoshi* prefers to feed on livestock, particularly cattle (Linthicum, Bailey, et al., 1985; Linthicum, Kaburia, et al., 1985; Tchouassi et al., 2016); therefore, the high feeding rates we observed for humans may be due, in part, to the absence of preferred livestock hosts. Unlike past studies of blood feeding in *Ae. mcintoshi*, which have largely been carried out in livestock-dominated systems, our study provides insight into the blood-feeding behaviour of this mosquito in a wildlife context. In the absence of livestock, *Ae. mcintoshi* appears to prefer buffalo, elephants and humans over other large wild mammals. Given the close phylogenetic relationship between buffalo and cattle, the preference for buffalo is not surprising. However, this behaviour has important implications for pathogen transmission, exemplified here by RVFV. First, African buffalo are known to become infected with RVFV, and recent evidence suggests that this species plays some role in the persistence of the virus in between large outbreaks (Beechler et al., 2015; LaBeaud et al., 2011). Second, the strong preference of *Ae. mcintoshi* for buffalo and humans highlights the potential for virus spillover to occur from buffalo maintenance hosts to humans, particularly during non-epidemic periods. Indeed, our results shed new light on possible pathways of RVFV inter-epidemic persistence and spillover to humans.

To understand how the changes in mosquito abundance and biting behaviour observed in response to wildlife exclusion might translate to disease dynamics, we estimated the vectorial capacity of *Ae. mcintoshi* for RVFV in the presence versus absence of large herbivores. Vectorial capacity summarizes the potential for virus transmission to humans by accounting for the density of mosquitoes, the daily rate at which they bite infectious and susceptible

hosts, the mosquito mortality rate, and the interval between virus acquisition and transmissibility in the mosquito (i.e. the EIP). Based on evidence from the literature that humans mount viremia levels sufficient to infect mosquitoes (Meegan, 1979; Swanepoel & Coetzer, 2004) and that buffalo are also likely permissive for viral replication (Davies & Karstad, 1981; Evans et al., 2008), we assumed that these two hosts would be most likely to contribute to RVFV infection of mosquitoes in our study system. As a result, we found that VC doubled in the presence of large herbivores. This is the opposite of what we would expect based on the observed shift in mosquito feeding behaviour alone. However, this result emerges because both higher *Ae. mcintoshi* abundance and improved survival in plots with large herbivores more than compensate for the higher human biting rate in plots without large herbivores. Interestingly, the effects of large herbivore loss on mosquito biting behaviour and survival may be linked. Previous studies have described survival costs of host switching in mosquitoes (Harrington et al., 2001; Phasomkusolsil et al., 2013); thus, the loss of preferred wildlife blood meals and concomitant dietary shift to human blood that occurred in large herbivore exclusion plots may have been associated with survival costs. If so, biodiversity loss-associated changes in mosquito feeding behaviour might commonly be accompanied by changes in survival rates. Since feeding behaviour and survival both affect pathogen transmission potential, the fact that they both may change simultaneously reinforces the need for considering a combination of factors when evaluating the effects of biodiversity loss on disease risk in vector-borne disease systems.

It is important to acknowledge several simplifying assumptions that we made in our estimate of VC which might have affected our conclusions. We did not consider the potential effect of herbivore exclusion on vector competence which includes two components: the probability of transmission from an infected host to the mosquito and the probability of transmission from an infected mosquito to a susceptible host. For the first component, we assumed that humans and buffalo are equally effective at infecting mosquitoes with RVFV. While RVFV viremia levels in humans have been quantified directly (Meegan, 1979; Swanepoel & Coetzer, 2004), inference about the efficiency of buffalo at infecting mosquitoes has largely been made from serological patterns (Evans et al., 2008). However, even if we assume that buffalo make no contribution to VC and that humans drive transmission (i.e. replace $a_x \times a_y$ with a_y^2), VC in the large herbivore plots still exceeds VC in plots without large herbivores by ~50% (225 versus 148), indicating that the observed shift in human feeding behaviour of *Ae. mcintoshi* in large herbivore exclusion plots is still insufficient to overcome the increased abundance and lower mortality of these mosquitoes in these plots. For the second component, it is possible that variation in habitat structure and host availability, both of which are associated with large herbivore exclusion in the KLEE plots (Augustine & Mcnaughton, 2004; Goheen et al., 2007; Ogada et al., 2008), might affect the ability of an infectious mosquito to infect a susceptible human host with RVFV. However, at present, the magnitude and direction of any such effects are hard to predict. The same caveat holds for our estimate of mosquito survival for which we assumed a fixed value for the number of days between female mosquito emergence and first oviposition, a trait that could also be affected by habitat and host availability differences between herbivore treatments.

5 | CONCLUSION

Overall, we found that biodiversity loss in the form of the loss of large wild mammalian herbivores altered patterns of mosquito abundance, blood-feeding behaviour and survival. In combination, these effects have implications for the transmission of RVFV, the causative agent of a zoonotic vector-borne disease. Surprisingly, despite the strong effect large herbivores had on deflecting mosquito bites away from humans, RVFV transmission potential was maximized in plots with large herbivores. This outcome is the result of simultaneous positive effects of large herbivore presence on mosquito abundance and survival. Our results demonstrate how multiple effects of biodiversity loss on vectors combine to shape infectious disease risk.

ACKNOWLEDGEMENTS

We thank John Ewoi, Danielle Spitzer, Robinson Okiro, Julia Wanjiru and Wyckliff Omondi for assistance in the field and laboratory; the Mpala Research Centre for logistical support; and Courtney Murdock and members of the Ezenwa laboratory for helpful comments on the manuscript.

CONFLICTS OF INTEREST

The authors declare that they have no competing interests.

AUTHORS' CONTRIBUTIONS

V.O.E. conceived the study. D.P.T., B.T., R.S. and V.O.E. designed the study and performed research. C.R. maintained and provided access to the experimental plots. D.P.T. and V.O.E. analysed the data and wrote the first draft of the manuscript. All authors contributed substantially to revisions.

DATA AVAILABILITY STATEMENT

Should the manuscript be accepted, the data supporting the results will be archived on Dryad and the data DOI will be included at the end of the article.

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REFERENCES

Anderson, J. F., Andreadis, T. G., Main, A. J., & Kline, D. L. (2004). Prevalence of West Nile virus in tree canopy–inhabiting *Culex pipiens* and associated mosquitoes. *The American Journal of Tropical Medicine and Hygiene*, *71*(1), 112–119.

Arum, S. O., Weldon, C. W., Orindi, B., Landmann, T., Tchouassi, D. P., Affognon, H. D., & Sang, R. (2015). Distribution and diversity of the vectors of Rift Valley fever along the livestock movement routes in the northeastern and coastal regions of Kenya. *Parasites and Vectors*, *8*(1), 294. https://doi.org/10.1186/s13071-015-0907-1

Arum, S. O., Weldon, C. W., Orindi, B., Tigoi, C., Musili, F., Landmann, T., Tchouassi, D. P., Affognon, H. D., & Sang, R. (2016). Plant resting site preferences and parity rates among the vectors of Rift Valley Fever in northeastern Kenya. *Parasites and Vectors*, *9*(1), 310. https://doi.org/10.1186/s13071-016-1601-7

Augustine, D. J., & Mcnaughton, S. J. (2004). Regulation of shrub dynamics by native browsing ungulates on East African rangeland. *Journal of Applied Ecology*, *41*(1), 45–58.

Beechler, B. R., Bengis, R., Swanepoel, R., Paweska, J. T., Kemp, A., Vuren, P. J., Joubert, J., Ezenwa, V. O., & Jolles, A. E. (2015). Rift Valley fever in Kruger National Park: Do buffalo play a role in the inter-epidemic circulation of virus? *Transboundary and Emerging Diseases*, *62*(1), 24–32.

Brunner, J. L., & Ostfeld, R. S. (2008). Multiple causes of variable tick burdens on small-mammal hosts. *Ecology*, *89*(8), 2259–2272.

Cardinale, B. J., Duffy, J. E., Gonzalez, A., Hooper, D. U., Perrings, C., Venail, P., Narwani, A., Mace, G. M., Tilman, D., & Wardle, D. A. (2012). Biodiversity loss and its impact on humanity. *Nature*, *486*(7401), 59–67.

Churcher, T. S., Trape, J.-F., & Cohuet, A. (2015). Human-to-mosquito transmission efficiency increases as malaria is controlled. *Nature Communications*, *6*, 6054.

Civitello, D. J., Cohen, J., Fatima, H., Halstead, N. T., Liriano, J., McMahon, T. A., Ortega, C. N., Sauer, E. L., Sehgal, T., & Young, S. (2015). Biodiversity inhibits parasites: Broad evidence for the dilution effect. *Proceedings of the National Academy of Sciences of the United States of America*, *112*(28), 8667–8671.

Crego, R. D., Ogutu, J. O., Wells, H. B. M., Ojwang, G. O., Martins, D. J., Leimgruber, P., & Stabach, J. A. (2020). Spatiotemporal dynamics of wild herbivore species richness and occupancy across a savannah rangeland: Implications for conservation. *Biological Conservation*, *242*, 108436.

Davidson, G. (1954). Estimation of the survival-rate of anopheline mosquitoes in nature. *Nature*, *174*(4434), 792–793.

Davies, F. G., & Karstad, L. (1981). Experimental infection of the African buffalo with the virus of Rift Valley fever. *Tropical Animal Health and Production*, *13*(1), 185–188.

Detinova, T. S. (1962). Age grouping methods in Diptera of medical importance with special reference to some vectors of malaria. *Monograph Series World Health Organisation*, *47*, 13–191.

Díaz, S., Fargione, J., Chapin, F. S. III, & Tilman, D. (2006). Biodiversity loss threatens human well-being. *PLoS Biology*, *4*(8), e277.

Edwards, F. W. (1941). *Mosquitoes of the Ethiopian Region. III.-Culicine Adults and Pupae*, London, UK:British Museum (Nat. Hist.).

Evans, A., Gakuya, F., Paweska, J. T., Rostal, M., Akoolo, L., Van Vuren, P. J., Manyibe, T., Macharia, J. M., Ksiazek, T. G., & Feikin, D. R. (2008). Prevalence of antibodies against Rift Valley fever virus in Kenyan wildlife. *Epidemiology & Infection*, *136*(9), 1261–1269.

Ezenwa, V. O., Godsey, M. S., King, R. J., & Guptill, S. C. (2006). Avian diversity and West Nile virus: Testing associations between biodiversity and infectious disease risk. *Proceedings of the Royal Society of London B: Biological Sciences*, *273*(1582), 109–117.

Garrett-Jones, C. (1964). Prognosis for interruption of malaria transmission through assessment of the mosquito's vectorial capacity. *Nature*, *204*(4964), 1173–1175.

Gillett, J. D. (1972). *Common African mosquitoes and their medical importance*, London, UK: William Heinemann.

Goheen, J. R., Augustine, D. J., Veblen, K. E., Kimuyu, D. M., Palmer, T. M., Porensky, L. M., Pringle, R. M., Ratnam, J., Riginos, C., & Sankaran, M. (2018). Conservation lessons from large-mammal manipulations in East African savannas: The KLEE, UHURU, and GLADE experiments. *Annals of the New York Academy of Sciences*, *1429*(1), 31–49.

Goheen, J. R., Young, T. P., Keesing, F., & Palmer, T. M. (2007). Consequences of herbivory by native ungulates for the reproduction of a savanna tree. *Journal of Ecology*, *95*(1), 129–138.

Gubler, D. J. (1998). Resurgent vector-borne diseases as a global health problem. *Emerging Infectious Diseases*, *4*(3), 442.

Harrington, L. C., Edman, J. D., & Scott, T. W. (2001). Why do female Aedes aegypti (Diptera: Culicidae) feed preferentially and frequently on human blood? *Journal of Medical Entomology*, *38*(3), 411–422.

Hoogstraal, H., Meegan, J. M., Khalil, G. M., & Adham, F. K. (1979). The Rift Valley fever epizootic in Egypt 1977–1978 2. Ecological and entomological studies. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, *73*(6), 624–629.

Johnson, C. N., Balmford, A., Brook, B. W., Buettel, J. C., Galetti, M., Guangchun, L., & Wilmshurst, J. M. (2017). Biodiversity losses and conservation responses in the Anthropocene. *Science*, *356*(6335), 270–275.

Johnson, P. T. J., & Thieltges, D. W. (2010). Diversity, decoys and the dilution effect: How ecological communities affect disease risk. *Journal of Experimental Biology*, *213*(6), 961–970.

Jupp, P. G. (1996). *Mosquitoes of Southern Africa: Culicinae and toxorhynchitinae*, Hartebeespoort, South Africa: Ekogilde Publishers.

Keesing, F. (1998). Impacts of ungulates on the demography and diversity of small mammals in central Kenya. *Oecologia*, *116*(3), 381–389.

Keesing, F., Allan, B. F., Young, T. P., & Ostfeld, R. S. (2013). Effects of wildlife and cattle on tick abundance in central Kenya. *Ecological Applications*, *23*(6), 1410–1418.

Keesing, F., Belden, L. K., Daszak, P., Dobson, A., Harvell, C. D., Holt, R. D., Hudson, P., Jolles, A., Jones, K. E., & Mitchell, C. E. (2010). Impacts of biodiversity on the emergence and transmission of infectious diseases. *Nature*, *468*(7324), 647–652.

Keesing, F., Holt, R. D., & Ostfeld, R. S. (2006). Effects of species diversity on disease risk. *Ecology Letters*, *9*(4), 485–498.

Keesing, F., & Young, T. P. (2014). Cascading consequences of the loss of large mammals in an African savanna. *BioScience*, *64*(6), 487–495.

Kilpatrick, A. M., Kramer, L. D., Jones, M. J., Marra, P. P., Daszak, P., & Fonseca, D. M. (2007). Genetic influences on mosquito feeding behavior and the emergence of zoonotic pathogens. *The American Journal of Tropical Medicine and Hygiene*, *77*(4), 667–671.

Kimuyu, D. M., Veblen, K. E., Riginos, C., Chira, R. M., Githaiga, J. M., & Young, T. P. (2017). Influence of cattle on browsing and grazing wildlife varies with rainfall and presence of megaherbivores. *Ecological Applications*, *27*(3), 786–798.

Kramer, L. D., & Ciota, A. T. (2015). Dissecting vectorial capacity for mosquito-borne viruses. *Current Opinion in Virology*, *15*, 112–118.

LaBeaud, A. D., Cross, P. C., Getz, W. M., Glinka, A., & King, C. H. (2011). Rift Valley fever virus infection in African buffalo (Syncerus caffer) herds in rural South Africa: Evidence of interepidemic transmission. *The American Journal of Tropical Medicine and Hygiene*, *84*(4), 641–646.

LaDeau, S. L., Allan, B. F., Leisnham, P. T., & Levy, M. Z. (2015). The ecological foundations of transmission potential and vector-borne disease in urban landscapes. *Functional Ecology*, *29*(7), 889–901.

Linthicum, K. J., Bailey, C. L., Davies, F. G., & Kairo, A. (1985). Observations on the dispersal and survival of a population of Aedes lineatopennis (Ludlow) (Diptera: Culicidae) in Kenya. *Bulletin of Entomological Research*, *75*(4), 661–670.

Linthicum, K. J., Britch, S. C., & Anyamba, A. (2016). Rift Valley fever: An emerging mosquito-borne disease. *Annual Review of Entomology*, *61*, 395–415.

Linthicum, K. J., Kaburia, H. F. A., Davies, F. G., & Lindqvist, K. J. (1985). A blood meal analysis of engorged mosquitoes found in Rift Valley fever epizootics areas in Kenya. *Mosquito News*, *1*(1), 93–95.

Lutomiah, J., Omondi, D., Masiga, D., Mutai, C., Mireji, P. O., Ongus, J., Linthicum, K. J., & Sang, R. (2014). Blood meal analysis and virus

detection in blood-fed mosquitoes collected during the 2006–2007 Rift Valley fever outbreak in Kenya. *Vector-Borne and Zoonotic Diseases*, *14*(9), 656–664.

Massad, E., & Coutinho, F. A. B. (2012). Vectorial capacity, basic reproduction number, force of infection and all that: Formal notation to complete and adjust their classical concepts and equations. *Memórias do Instituto Oswaldo Cruz*, *107*(4), 564–567.

McCauley, D. J., Keesing, F., Young, T. P., Allan, B. F., & Pringle, R. M. (2006). Indirect effects of large herbivores on snakes in an African savanna. *Ecology*, *87*(10), 2657–2663.

Meegan, J. M. (1979). The Rift Valley fever epizootic in Egypt 1977–1978 1. Description of the epizootic and virological studies. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, *73*(6), 618–623.

Ndoen, E., Wild, C., Dale, P., Sipe, N., & Dale, M. (2012). Mosquito longevity, vector capacity, and malaria incidence in West Timor and Central Java, Indonesia. *International Scholarly Research Network*, *2012*,5. Article ID 143863.

Ogada, D. L., Gadd, M. E., Ostfeld, R. S., Young, T. P., & Keesing, F. (2008). Impacts of large herbivorous mammals on bird diversity and abundance in an African savanna. *Oecologia*, *156*(2), 387.

Ogutu, J. O., Piepho, H.-P., Said, M. Y., Ojwang, G. O., Njino, L. W., Kifugo, S. C., & Wargute, P. W. (2016). Extreme wildlife declines and concurrent increase in livestock numbers in Kenya: What are the causes? *PLoS One*, *11*(9), e0163249.

Ostfeld, R. S., & Keesing, F. (2012). Effects of host diversity on infectious disease. *Annual Review of Ecology, Evolution, and Systematics*, *43*, 157–182.

Pan, J.-Y., Zhou, S.-S., Zheng, X., Huang, F., Wang, D.-Q., Shen, Y.-Z., Su, Y.-P., Zhou, G.-C., Liu, F., & Jiang, J.-J. (2012). Vector capacity of *Anopheles sinensis* in malaria outbreak areas of central China. *Parasites and Vectors*, *5*, 136.

Phasomkusolsil, S., Tawong, J., Monkanna, N., Pantuwatana, K., Damdangdee, N., Khongtak, W., Kertmanee, Y., Evans, B. P., & Schuster, A. L. (2013). Maintenance of mosquito vectors: Effects of blood source on feeding, survival, fecundity, and egg hatching rates. *Journal of Vector Ecology*, *38*(1), 38–45.

Pringle, R. M., Young, T. P., Rubenstein, D. I., & McCauley, D. J. (2007). Herbivore-initiated interaction cascades and their modulation by productivity in an African savanna. *Proceedings of the National Academy of Sciences of the United States of America*, *104*(1), 193–197.

Reeves, W. C. (1965). Ecology of mosquitoes in relation to arboviruses. *Annual Review of Entomology*, *10*(1), 25–46.

Roca, A. L., Bar-Gal, G. K., Eizirik, E., Helgen, K. M., Maria, R., Springer, M. S., O'Brien, S. J., & Murphy, W. J. (2004). Mesozoic origin for West Indian insectivores. *Nature*, *429*(6992), 649–651.

Rohr, J. R., Civitello, D. J., Halliday, F. W., Hudson, P. J., Lafferty, K. D., Wood, C. L., & Mordecai, E. A. (2019). Towards common ground in the biodiversity–disease debate. *Nature Ecology & Evolution*, *4*, 24–33.

Salkeld, D. J., Padgett, K. A., & Jones, J. H. (2013). A meta-analysis suggesting that the relationship between biodiversity and risk of zoonotic pathogen transmission is idiosyncratic. *Ecology Letters*, *16*(5), 679–686.

Sang, R. C., & Dunster, L. M. (2001). The growing threat of arbovirus transmission and outbreaks in Kenya: A review. *East African Medical Journal*, *78*(12), 655–661.

Sang, R., Kioko, E., Lutomiah, J., Warigia, M., Ochieng, C., O'Guinn, M., Lee, J. S., Koka, H., Godsey, M., & Hoel, D. (2010). Rift Valley fever virus epidemic in Kenya, 2006/2007: The entomologic investigations. *The American Journal of Tropical Medicine and Hygiene*, *83*(2 Suppl), 28–37.

Swanepoel, R., & Coetzer, J. A. W. (2004). Rift valley fever. In J. A. W. Coetzer & R. C. Tustin (Ed.), *Infectious diseases of livestock* (pp. 1037–1070). Oxford University Press.

Tchouassi, D. P., Bastos, A. D. S., Sole, C. L., Diallo, M., Lutomiah, J., Mutisya, J., Mulwa, F., Borgemeister, C., Sang, R., & Torto, B. (2014). Population genetics of two key mosquito vectors of Rift Valley fever virus reveals new insights into the changing disease outbreak patterns in Kenya. *PLoS Neglected Tropical Diseases*, *8*(12), e3364. https://doi.org/10.1371/journal.pntd.0003364

Tchouassi, D. P., Jacob, J. W., Ogola, E. O., Sang, R., & Torto, B. (2019). *Aedes* vector-host olfactory interactions in sylvatic and domestic dengue transmission environments. *Proceedings of the Royal Society B*, *286*, 20192136.

Tchouassi, D. P., Okiro, R. O. K., Sang, R., Cohnstaedt, L. W., McVey, D. S., & Torto, B. (2016). Mosquito host choices on livestock amplifiers of Rift Valley fever virus in Kenya. *Parasites and Vectors*, *9*(1), 184. https://doi.org/10.1186/s13071-016-1473-x

Tchouassi, D. P., Quakyi, I. A., Addison, E. A., Bosompem, K. M., Wilson, M. D., Appawu, M. A., Brown, C. A., & Boakye, D. A. (2012). Characterization of malaria transmission by vector populations for improved interventions during the dry season in the Kpone-on-Sea area of coastal Ghana. *Parasites and Vectors*, *5*(1), 212. https://doi.org/10.1186/1756-3305-5-212

Tchouassi, D. P., Sang, R., Sole, C. L., Bastos, A. D. S., Mithoefer, K., & Torto, B. (2012). Sheep skin odor improves trap captures of mosquito vectors of Rift Valley fever. *PLoS Neglected Tropical Diseases*, *6*(11), e1879. https://doi.org/10.1371/journal.pntd.0001879

R Core Team. (2013). R: A language and environment for statistical computing [Computer software manual]. *RA Lang Environ Stat Comput*, *55*, 275–286.

Turell, M. J., Linthicum, K. J., Patrican, L. A., Davies, F. G., Kairo, A., & Bailey, C. L. (2008). Vector competence of selected African mosquito (Diptera: Culicidae) species for Rift Valley fever virus. *Journal of Medical Entomology*, *45*(1), 102–108.

Valinsky, L., Ettinger, C., Bar-Gal, G. K., & Orshan, L. (2014). Molecular identification of bloodmeals from sand flies and mosquitoes collected in Israel. *Journal of Medical Entomology*, *51*(3), 678–685.

Vittor, A. Y., Gilman, R. H., Tielsch, J., Glass, G., Shields, T. I. M., Lozano, W. S., Pinedo-Cancino, V., & Patz, J. A. (2006). The effect of deforestation on the human-biting rate of *Anopheles darlingi*, the primary vector of falciparum malaria in the Peruvian Amazon. *The American Journal of Tropical Medicine and Hygiene*, *74*(1), 3–11.

Wood, C. L., Lafferty, K. D., DeLeo, G., Young, H. S., Hudson, P. J., & Kuris, A. M. (2014). Does biodiversity protect humans against infectious disease? *Ecology*, *95*(4), 817–832.

Yasuoka, J., & Levins, R. (2007). Impact of deforestation and agricultural development on anopheline ecology and malaria epidemiology. *The American Journal of Tropical Medicine and Hygiene*, *76*(3), 450–460.

Young, H. S., Dirzo, R., Helgen, K. M., McCauley, D. J., Billeter, S. A., Kosoy, M. Y., Osikowicz, L. M., Salkeld, D. J., Young, T. P., & Dittmar, K. (2014). Declines in large wildlife increase landscape-level prevalence of rodent-borne disease in Africa. *Proceedings of the National Academy of Sciences of the United States of America*, *111*(19), 7036–7041.

Young, T. P., Okello, B., Kinyua, D., & Palmer, T. M. (1998). KLEE: A long-term multi-species herbivore exclusion experiment in Laikipia, Kenya. *African Journal of Range and Forage Science*, *14*, 92–104.

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

How to cite this article: Tchouassi DP, Torto B, Sang R, Riginos C, Ezenwa VO. Large herbivore loss has complex effects on mosquito ecology and vector-borne disease risk. *Transbound Emerg Dis*. 2021;68:2503–2513. https://doi.org/10.1111/tbed.13918