Contact networks of small mammals highlight potential transmission foci of *Mammarenavirus lassaense*.

Authors

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Abstract

Lassa fever, from *Mammarenavirus lassaense* (LASV), is an endemic zoonosis in West Africa. Human infections arise from rodent-to-human transmission, mainly from the synanthropic reservoir *Mastomys natalensis*. In Sierra Leone, small-mammal

communities vary across land use gradients, shaping LASV transmission risk. How anthropogenic environments facilitate these interactions remains poorly understood.

We sampled small mammals over 43,266 trap nights in Sierra Leone's LASV-endemic Eastern Province, detecting 684 rodents and shrews. To assess potential transmission, we constructed space-sharing networks from co-trapping events within species-specific home range radii. These networks approximated shared space use, allowing comparison of encounter patterns across habitats.

Agricultural communities supported more distinct species (higher richness) and had denser networks (higher degree) than villages and forests, though space-sharing rates did not differ substantially. Network topology varied by land use; village networks were more modular (i.e., fragmented into distinct interaction clusters). Notably, intraspecific space sharing among *M. natalensis* was more frequent in agriculture than villages, suggesting land use modulates intraspecific transmission.

LASV seroprevalence was 5.7% across the community, with antibodies in nine species. We found no significant association between seroprevalence and land use or overall network structure. These findings show simple ecological drivers do not fully explain LASV exposure patterns, highlighting that species-specific space use and habitat-dependent interactions are crucial for identifying transmission pathways and high-risk settings.

Introduction

Lassa fever caused by *Mammarenavirus lassaense* (LASV) is a rodent-associated zoonosis endemic to West Africa, with an estimated 100,000-900,000 infections annually[1,2]. While outbreaks in Nigeria are routinely reported, cases in other endemic countries - Guinea, Liberia and Sierra Leone - are sporadically documented[3–5]. In Sierra Leone disease outbreaks frequently go undetected, consistent with findings of up to 80% LASV seropositivity among human communities in regions previously not considered endemic[6]. Human infections typically result from transmission from rodent hosts, with limited subsequent human-to-human transmission[7]. Understanding LASV transmission in endemic settings requires a detailed characterization of small-mammal community interactions, through which pathogen transmission occurs and is maintained.

The primary reservoir of LASV, *Mastomys natalensis* is a native synanthropic rodent species, widespread across sub-Saharan Africa. Pathogen challenge studies on *M. natalensis* colonies suggest that acute infection does not significantly alter rodent behaviour or cause clinical pathology[8–10]. LASV is transmitted through both direct contact (e.g., superficial wounds caused by infected conspecifics) and indirect contact (e.g., exposure to contaminated environments), at low infectious doses[9]. Vertical transmission (mother-to-pup) may also contribute to transmission dynamics[10,11]. Infected adult rodents exhibit detectable viral RNA as early as 3 days post-infection, with viral loads peaking within 1-2 weeks and resolving by 40 days[9]. Among individuals infected within the first 2 weeks of life viral RNA is detectable up to 16 months post infection[10]. The transient nature of acute infection - outside of neonatal infections - has

led many studies to focus on LASV-specific antibody detection, rather than circulating virus[12–14].

The antibody response dynamics to LASV in rodents are not yet well understood. Based on data from a similar arenavirus, Morogoro virus, seroconversion is expected to occur by 7 days post-infection, with antibodies (e.g., IgG) persisting beyond the decline of detectable RNA[11]. LASV-infected rodents are presumed to develop lifelong immunity to reinfection upon seroconversion, however, the efficacy of neutralizing antibodies is unclear and the role of immune or partially immune individuals in transmission networks is not known[9,10,15]. Although antibody-based studies have limitations, the higher prevalence of LASV seropositivity compared to acute infections provides valuable insights into viral dynamics within endemic regions. For example, a recent study in Bo district, Sierra Leone, reported a 2.8% LASV IgG seroprevalence among rodents and shrews, compared to a 0.3% prevalence of acute infection detected via PCR, underscoring the challenges of identifying acute infections in small-mammal populations[16].

While *M. natalensis* is considered the primary reservoir species of LASV, 10 further small-mammal species have been identified as acutely or previously infected with LASV in endemic regions[14,17–19]. The contribution of these additional species to pathogen transmission into human populations - and their role in viral transmission or maintenance within their species communities - remains unclear. In species-rich environments, both direct and indirect contact among small-mammals may result in incidental infections of non-reservoir species, which are subsequently detected through surveillance. Incidental infections of non-primary reservoir species may have little impact on viral transmission or maintenance[20]. Alternatively, these species could facilitate the transfer of LASV across landscapes, linking geographically isolated *M. natalensis* populations and causing reintroduction of the virus into reservoir species populations[21,22]. To account for this uncertainty we refer to *M. natalensis* as the primary reservoir species and other small-mammal species previously found to be infected as hosts. Increasing recognition of multi-species host systems in zoonoses underscores the importance of expanding surveillance efforts to the wider community in which the host resides to better understand pathogen prevalence and dynamics[23,24].

Understanding the structure of small-mammal contact networks in LASV-endemic regions may offer valuable insight into transmission potential, even in the absence of direct observations of infection. While prior studies have described rodent diversity and abundance, these do not capture interaction patterns relevant for pathogen spread[16,25,26]. Spatial proximity can be used as a proxy for potential contact and, when analysed as networks, can help identify structural features, such as connectivity, inter- and intra-specific mixing, and network fragmentation that influence pathogen transmission. In contexts where direct behavioural observation or tagging of individuals is infeasible, such as post-Ebola Sierra Leone, proximity-based approaches provide a pragmatic alternative to infer likely transmission pathways[27].

Small-mammal communities in LASV-endemic regions are structured along anthropogenic land use gradients[14,28]. As such, the risk of Lassa fever outbreaks in human populations is expected to correlate with these gradients[6,29,30]. Within human-

dominated land use types, the prevalence of typically synanthropic rodent hosts of LASV is anticipated to be higher due to increased food availability, shelter, and reduced predation pressure[31–33]. These factors influence rodent abundance and population dynamics which in turn may promote greater pathogen persistence, as observed in several other rodent-associated zoonosis systems[34–36]. Understanding how rodent contact networks, like rodent occurrence and abundance, vary along these anthropogenic gradients could reveal potentially distinct pathogen transmission networks in different habitats. We hypothesize that rodent contact rates — and the associated pathogen transmission networks — are greater in human-dominated habitats where resources are concentrated.

In this study, we define “contact” as the inferred co-occurrence within a spatial and temporal window (i.e., proximate capture locations within four trap-nights), consistent with prior spatial proximity models[37,38]. While this does not imply physical contact or indirect contact suitable for transmission, it serves as a proxy for shared habitat use and potential indirect or environmental contact relevant to LASV transmission. Interaction networks derived from wildlife data have previously been employed to study pathogen transmission. These networks are particularly valuable for investigating the role of community structure and the impact of contact rate heterogeneity between species in multi-host pathogen systems[39–41].

We leverage rodent and shrew trapping data collected over three years in the Lassa fever-endemic region of the Eastern Province, Sierra Leone. We characterize potential interactions — both direct and indirect — within these communities as a network, where the nodes represent rodents or shrews, and the connections (or edges) between them represent potential interactions. We hypothesize that the spatial clustering of conspecifics and the increased abundance of commensal species in anthropogenically dominated environments will lead to higher intra-specific contact rates compared to inter-specific contact rates within these communities. We use contact rates within and between species to explore their variation along an anthropogenic land use gradient with a particular focus on *M. natalensis*. Finally, we report the prevalence of antibodies against LASV among individual small mammals in the study region and investigate the association between contact rates with seropositivity.

Methods

Study area

Rodent trapping surveys were conducted between October 2020-April 2023 within and around four village study sites (Baiama, Lalehun, Lambayama, and Seilama) in the Lassa fever endemic zone of the Eastern Province of Sierra Leone (Figure 1A). Surveys were conducted within trapping grids along a land use gradient of anthropogenic disturbance comprising forest, agriculture (including fallow and currently in-use areas), and villages (within and outside of permanent structures) (see Supplementary Information for study site and trap grid locations). One study site (Lambayama) was more developed than the other villages due to its proximity to the province capital (Kenema), we refer to this site as peri-urban with the others termed rural. Trapping

survey sessions occurred four times annually with two trapping surveys in each of the rainy and dry seasons (May to November and December to April, respectively), producing a total of 10 trapping sessions over the study period.

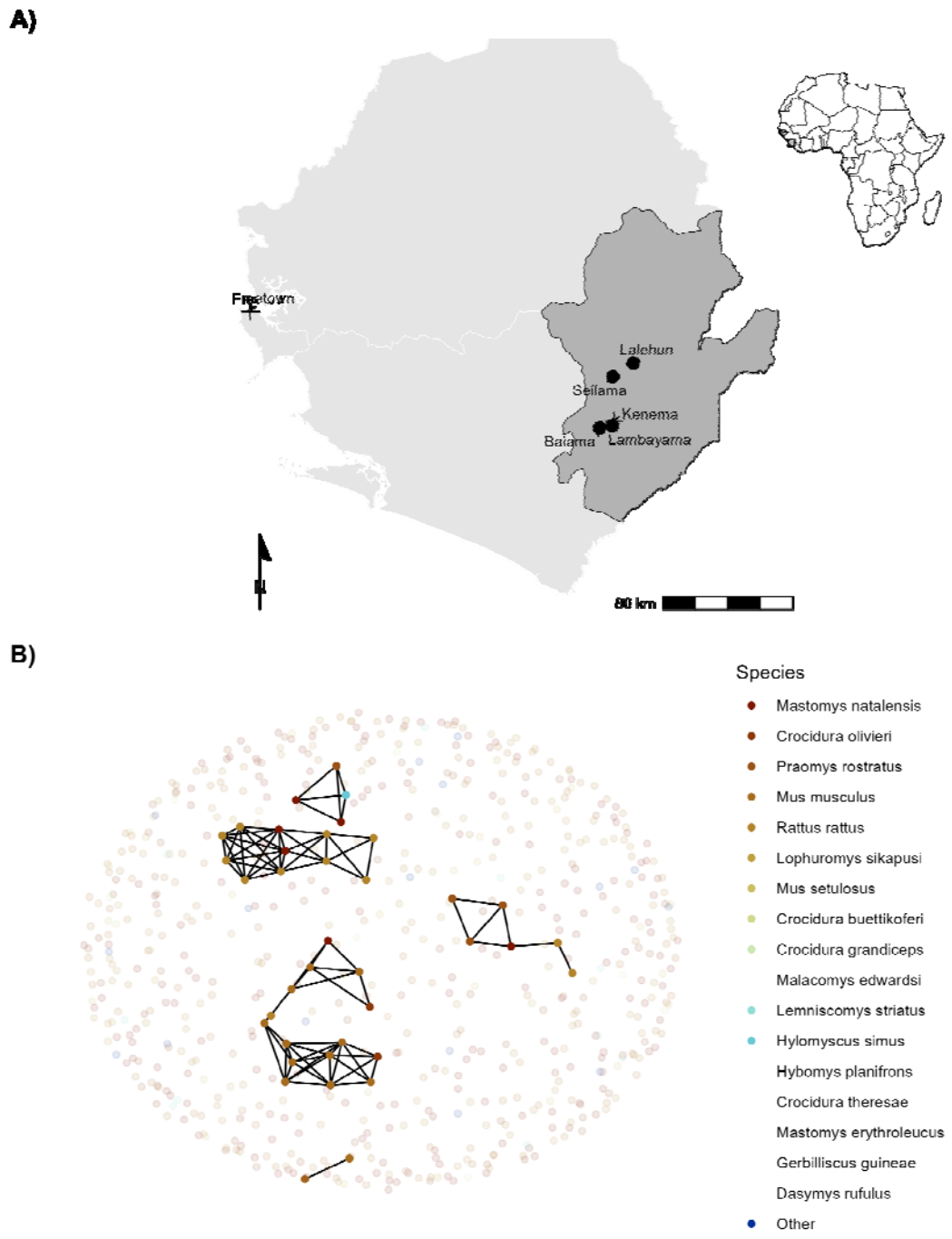

Figure 1: A) Location of village study sites (circles) in the Eastern Province of Sierra Leone. Kenema, the largest city in the province, and the national capital, Freetown, are also shown (+). The inset map highlights the location of Sierra Leone within West Africa. B) An example of a rodent contact network derived from trapping data during visit 5 in

village land use. Each coloured node represents an individual small mammal, with lines (edges) indicating inferred contacts between individuals. The number of edges connected to a node represents its degree. Betweenness reflects the importance of a node in connecting different parts of the network. Pale nodes indicate unobserved individuals, for whom contacts (edges) were not recorded. Species listed in the legend without colours were never detected in village land use types.

Study sites were selected to represent the range of land use in the Eastern Province of Sierra Leone, considering both accessibility throughout the year and acceptability of the study protocol to the village communities (Supplementary Information). At each trapping grid 49 Sherman traps (7.62cm x 8.89cm x 22.86cm) (H.B. Sherman Traps, Tallahassee, USA), were arranged in a 7 x 7 grid, with traps placed 7 metres apart in a grid conforming to the local landscape (median trapping grid area = 3,882 m^2). In permanent structures, trap placement deviated from the grid structure. At each visit, permanent structures were randomly selected from a grid projected over the village area, with four traps placed within each structure. The location of each trap within the grid was geolocated. Traps were baited with a locally produced mixture of oats, palm oil and dried fish. Each morning, traps were checked, closed for the day, and re-baited in the evening. Each trapping survey session consisted of four consecutive trap-nights (TN) at each trapping grid within the village study site. Rodents and shrews were associated with the coordinates of the trap they were detected. Geospatial processing was performed using the `sf` package R (version 4.1.2)[42,43].

All small mammals were handled by trained researchers using appropriate personal protective equipment. Animals were sedated using halothane and euthanized according to established protocols[44]. Morphological measurements and samples of blood and tissue (skin, liver and spleen) were collected. The study was approved by the Clinical Research Ethical Review Board and Animal Welfare Ethical Review Board of the Royal Veterinary College, UK (URN: 2019 1949-3), and the ethics committee of Njala University, Sierra Leone. The study adhered to national and institutional ethical guidelines for the humane treatment of animals and safe handling of zoonotic pathogens. Trapping and sampling protocols were designed to minimize stress to the animals and reduce the risk of pathogen exposure to researchers and communities. Community engagement sessions were conducted to ensure understanding and acceptance of the study objectives, and all work complied with the principles outlined in the ARRIVE guidelines (v2)[45]. All carcasses were incinerated to mitigate pathogen transmission risks.

Species identification

Species identification was performed in the field based on external morphological characteristics, including body length, tail length, ear length, and pelage colouration, following the taxonomic keys of Happold and Kingdon and Monadjem *et al.*[46,47]. Field identification was supplemented by molecular methods to confirm species identity for individuals identified as *Mastomys sp.*, *Mus sp.*, *Rattus sp.* and *Crocidura sp.* alongside a random subset of remaining individuals (50% of remaining samples).

All samples remained in Sierra Leone and were stored at -20°C until processing. Genomic DNA was extracted using QIAGEN DNAeasy kits as per the manufacturers instructions[48] (Supplementary Information). DNA extracts were amplified using platinum *Taq* polymerase (Invitrogen) and *cytochrome B* primers[16]. DNA amplification was assessed through gel electrophoresis, and successful amplification products were Sanger sequenced (performed by Eurofins Genomics). The sequences were attributed to rodent species using the BLAST program, comparing the obtained sequences to *cytochrome B* records in the NCBI database (accessed 2023-06-30)[49].

Serological Analysis

Serological status of trapped rodents and shrews was determined using the BLACKBOX® LASV IgG ELISA Kit (Diagnostics Development Laboratory, Bernhard Nocht Institute for Tropical Medicine), which has been validated for rodent samples[50,51]. The protocol is reproduced in Supplementary Information. Briefly, 1µL of whole blood was inactivated by mixing with the provided sample dilution buffer (1:50). Where whole blood was unavailable (21 samples, 3%), blood was extracted from dried blood spots stored on filter paper by incubating with phosphate-buffered saline containing 0.08% Sodium Azide and 0.05% Tween-20[52]. Samples, alongside negative and positive controls, were incubated on the ELISA kit plates for 24 hours at 4-8 °C in a wet chamber. Following incubation, the plates were washed and incubated for a further hour with 1:10,000 diluted HRP-labelled streptavidin. A final wash was performed prior to the addition of 100µL of 3,3',5,5'-Tetramethylbenzidine (TMB) substrate to wells, with incubation for 10 minutes. The colorimetric reaction was stopped by adding 100µL of a stop solution.

A deviation from the kit protocol occurred due to local ELISA plate reader limitations. We measured the optical density (OD) at 450nm and 630nm, as opposed to 450nm and 620nm but this was not expected to have an important effect on absorbance patterns, as advised by the manufacturer. The index value was calculated by subtracting OD_{630} from OD_{450} and dividing by the cut-off value (the mean values of the negative controls + 0.150). Samples were classified as positive if the index value was greater than or equal to 1.1, negative if the index value was less than or equal to 0.9, and inconclusive if the index value was between 0.9 and 1.1. Inconclusive results were retested.

The prevalence of seropositive individuals is reported aggregated by species. A Bayesian logistic regression model was constructed, using the `brms` package, to estimate the Odds Ratio (OR) of seropositivity for each species compared to *M. natalensis*, which served as the reference species[53]. Specifically, a Bernoulli regression with normal, uninformative priors for population-level effects was used, with a binary dependent variable for seropositivity and an independent variable of small mammal species. Only species with more than 10 individuals assayed for antibodies to LASV were included in this model. Posterior distributions are presented in graphical format, alongside the posterior mean and 95% Credible Interval (CrI). Unlike frequentist approaches, Bayesian inference does not rely on p-values; rather, statistical support for an effect is assessed based on the posterior distribution, with associations interpreted in terms of the central tendency (e.g., OR) and whether the CrI excludes the null value (OR = 1).

In addition to the species-level seroprevalence analysis, we conducted post-hoc exploratory analyses to assess differences in LASV seroprevalence by village and land use type. These analyses employed Bayesian logistic regression models analogous to those used in species-level comparisons, with results interpreted cautiously given their non-pre-specified nature.

Community Diversity Analysis

To compare small-mammal diversity across land use types, it was necessary to account for the different number of animals captured in each habitat. We therefore used individual-based rarefaction (IBR), a statistical technique that standardizes species richness estimates as if an equal number of individuals had been sampled in each habitat. This approach allows for a robust comparison of alpha diversity by controlling for unequal sampling effort.

Using the `mobr` package in R, we calculated the expected species richness for each habitat rarefied to a common sample size of 10 individuals, which was the minimum number of captures in any single habitat[54,55]. To further investigate the ecological processes shaping these communities, we compared these rarefied richness values to the expectations of a null model. This comparison allowed us to assess whether the observed species abundance patterns deviated from random community assembly, for instance, to detect potential dominance effects where one or a few species reduce overall community evenness and richness.

Contact Network Construction

Species space-sharing networks were reconstructed from the trapping data. Capture-mark-recapture (CMR) methods have previously been used to identify space-sharing by individuals[38,56,57]. In our study system, a CMR design was not feasible due to study communities concerns around the risk of releasing an infected animal. Therefore, we considered that spatial and temporal co-occurrence proxies direct or indirect contacts with other small-mammals[37]. We assumed these potential contacts were sufficient to transmit LASV if they were trapped within a species specific buffer zone centered on the location of the trap during the same 4 trap night session. A key assumption underlying this approach is that an individual was trapped at the center of their home range[57].

We obtained species specific buffer radii for the primary analysis using the `HomeRange` R package (version 1.0.2)[58]. Five rodent species from our study system had available home range data: *M. natalensis* (median home range radii = 10.6m), *Mus musculus* (9.6m), *Lemnisomys striatus* (8m), *Lophuromys sikapusi* (8.4m), and *Rattus rattus* (29.3m). For detected species with no species level home range data, *Praomys rostratus* (7.2m), *Mus setulosus* (9.6m), *Crocidura spp* (7.2m) and *Hylomyscus simus* (7.7m) we used the genus level median home range. For the sole species with no available home range data, *Malacomys edwardsi*, we used a conservative estimate of 17m. We further assessed the impact of this assumption on produced contact networks by performing pre-specified sensitivity analyses with buffer radii of 15, 30 and 50 meters.

Networks were constructed from observed animals (nodes) and the presence or absence of contact between them (edges). Data were aggregated by land use type and sampling visit, producing a potential 32 distinct networks from 201 trapping grid, village and visit combinations (see Figure 1B for an example network). However, as no rodents were detected in three networks derived from forest sites, only 29 networks were used in subsequent analysis.

Network Structure and Mixing Pattern Analysis

We analyzed the structure of the space-sharing networks at three distinct levels: 1) the overall network within each land use type, 2) the individual animals (nodes) within those networks, and 3) the specific roles of and interactions between species.

First, to characterize the overall networks, we calculated global metrics for each land use type (village, agriculture, and forest). These included the total number of nodes (animals) and edges (inferred contacts), as well as the network-wide mean degree and betweenness centrality.

Second, to evaluate the roles of individual animals, we calculated two key node-level centrality measures. We used degree centrality, the number of connections for each animal, as a direct measure of its contact frequency. We used betweenness centrality to identify individuals acting as critical bridges within the network by measuring how often they fell on the shortest path between other nodes. Finally, we investigated species-specific interaction patterns. We analyzed the degree distribution for each species to identify which were the most connected in each habitat.

To quantify mixing patterns, we also calculated the proportion of each species' contacts that were intraspecific (with its own species) versus interspecific (with other species), providing a measure of network homophily across the land use gradient.

Modeling Contact Probability in *Mastomys natalensis*

To examine the association between land use and species with the probability of a contact between two individuals, we modelled these contacts using Exponential-Family Random Graph Models (ERGMs)[59]. The analysis was limited to *M. natalensis*, the primary rodent host of LASV. Estimation of ERGM parameters provide an OR for the probability of an edge in a network – conditional on the rest of the network - based on network properties included in the model and nodal attributes. Within our trapping grids, only a subset of all individuals are detected in traps. Including unobserved individuals — and thus, unobserved contacts — enhances the interpretability and generalizability of the network models. This approach allows for a more accurate estimation of the total population size by accounting for missing data, thereby making the network models more representative of the entire population from which the analytic sample was derived.

Previous analysis of our study system suggests that the probability of detecting a rodent at each trap is less than 10% for 4 trap nights, provided that the species is present in the trapping grid[28]. To estimate the abundance of individuals of each species within a trapping grid, we modelled abundance (i.e., total population size) from repeated count

data using an N-mixture model implemented in the `unmarked` R package (version 1.2.5)[60,61]. The latent abundance distribution was modelled using Poisson, negative binomial or zero-inflated Poisson random variables. The abundance model included the number of trap nights and season as replicate-dependent detection covariates, as well as location (rural vs. peri-urban setting) and land use type (forest, agriculture or village) as occurrence covariates.

To select the most appropriate model for each species, we compared the Akaike Information Criterion (AIC) of the Poisson, negative binomial, and zero-inflated Poisson models. The best-fitting model was then used to derive the estimated abundance. The median estimated abundance from the distribution produced for each trapping grid was used to estimate the number of unobserved individuals in each network, aggregated by land use type (Supplementary Figures 2.1-2.12). The number of observed individuals was subtracted from the predicted abundance to derive the number of unobserved individuals for each species. These unobserved individuals were explicitly set to have missing (i.e., unobserved) edge values.

Finally, the constructed adjacency matrices were converted to networks using the `network` R package (version 1.13.0.1) for subsequent ERGM modelling[62] (Figure 1B) and Supplementary Figures 3.1-3.3).

ERGMs were specified for each of our inferred contact networks to compare the probabilities of edges forming based on rodent characteristics (i.e., species). The general model is shown in Equation 1:

$$\mathrm{P}(\mathrm{Y} = \mathrm{y}) \propto \exp\left(\theta_1 g_1(\mathrm{y}) + \theta_2 g_2(\mathrm{y}) + \ldots + \theta_p g_p(\mathrm{y})\right) \quad (1)$$

Where p is the number of terms in the model, and the values of the coefficients θ represent the size and direction of the effects of the covariates $g(\mathrm{y})$ on the overall probability of an edge being present in the network. At the edge level the expression for the probability of the entire graph can be re-expressed as the conditional log-odds of a single edge between two nodes (a contact between two rodents) as shown in Equation 2.

$$\mathrm{logit}\left(\mathrm{P}\left(\mathrm{Y}_{ij} = 1 | \mathrm{y}_{ij}^{c}\right)\right) = \theta' \delta\left(\mathrm{y}_{ij}\right) \quad (2)$$

Where Y_{ij} is the random variable for the state of the node pair ij and $y_{i,j}^{c}$ signifies all dyads in the network other than $y_{i,j}$. θ' is the coefficient for the change production of an edge between the two nodes conditional on all other dyads remaining the same ($\delta\left(\mathrm{y}_{ij}\right)$).

ERGMs are implemented using the `ergm` package (version 4.3.2) in R[63]. Three terms were included in the final ERGM to model the probability of the formation of ties (Equation 3). The first term (edges), describes the density of the network, representing the probability of a tie being observed in the network. The second term (species) represents the conditional probability of a tie forming, conditional on the species of the nodes. The third term (species homophily) accounts for intraspecific tie formation

among rodent individuals (i.e., the conditional probability of two individuals of the same species forming a tie). To reduce linear dependency of the nodal terms and due to data sparsity within our inferred networks all non-*M. natalensis* are grouped as "Other species" through the `levels` term of the nodal covariates for the analysis of the effect of land use on the probability of inter- or intra-specific contacts for *M. natalensis*.

$$\mathrm{P}(\mathrm{Y} = \mathrm{y}) \propto \exp\left(\theta_{\mathrm{edges}} g_{\mathrm{edges}}(\mathrm{y}) + \theta_{\mathrm{species}} g_{\mathrm{species}}(\mathrm{y}) + \theta_{\mathrm{homophily}} g_{\mathrm{homophily}}(\mathrm{y})\right) \quad (3)$$

ERGMs were implemented on the individual networks for each land use type at each visit. The effect sizes from each model were pooled through random-effects meta-analysis, stratified by land use, to produce a land use-specific summary effect size for each coefficient[64]. Inclusion in meta-analysis was restricted to ERGMs that produced stable estimates for each of the model terms (i.e., sufficient detections of *M. natalensis* within the network). Random-effects models were conducted using the `metafor` package (version 4.0.0) in R[65]. Heterogeneity across the models was assessed using the Q-test and the restricted maximum-likelihood estimator (τ^2) with a prediction interval for the true outcomes produced[64,66]. The Q-test assesses whether there is greater variability among effect sizes than expected by chance, with a significant result indicating substantial heterogeneity. The τ^2 statistic estimates the between-study variance, quantifying the degree of heterogeneity rather than just testing for its presence. Weights for each network included in meta-analysis were assigned using inverse-variance weights[67].

The presence of influential networks was assessed using Cook's distance, for models including influential networks leave-one-out sensitivity analysis were performed[68]. Forest plots were generated to visualize the summary OR of the probability of a tie for each model term, stratified by land use type.

Models with unstable estimates for the species homophily term were not included in the random-effects meta-analysis. No contact networks from forest land use contributed to meta-analysis as no *M. natalensis* were detected in these settings. Five models from agricultural settings and eight from village settings were included in meta-analysis.

Analysis of Network Centrality and Serostatus

To investigate pathogen transmission within our networks, using seropositivity as a proxy for prior exposure to LASV, we first report the small-mammal species found to contain individuals that were seropositive for LASV. We then compared the nodal degree of seropositive and seronegative individuals using a Wilcoxon rank-sum test with continuity correction[69]. This analysis was repeated stratified by species to assess whether contact rates were associated with an individual being seropositive. Finally, we compared the node-level betweenness of seropositive and seronegative individuals to determine whether an individual's position within a structured contact network was associated with prior exposure to LASV.

Modelling Seropositivity Risk Factors in *M. natalensis*

We next tested whether an individual *M. natalensis*'s risk of being LASV-seropositive was influenced by its position in the contact network. Specifically, we investigated the effects of two key properties: its total number of contacts (degree) and its tendency to connect with conspecifics (homophily). Because our trapping data provides only one realization of a dynamic social system, we used a simulation-based approach to account for this structural uncertainty. Based on the network formation rules derived from the ERGMs described previously, we generated 50 plausible network simulations for each trapping grid that contained at least one seropositive *M. natalensis*.

Using these simulated networks, we fitted a Bayesian generalized linear mixed-effects model (GLMM) to assess the association between network position and serostatus. For each *M. natalensis* individual in each simulation, we calculated its degree and a node-level homophily score (defined as the proportion of its direct contacts that were with other *M. natalensis*). The GLMM then predicted LASV serostatus as a function of these two metrics and their interaction. To account for the nested structure of the data, we included random intercepts for each original trapping grid and for each simulation replicate. Evidence for an association was assessed using the posterior distributions of the model coefficients generated by the `brms` package.

Results

A total of 684 small mammals were captured over 43,266 trap-nights, representing 17 species (13 rodent and 4 shrew species). *M. natalensis* was the most commonly detected species (N = 113, 16.5%), followed by *Crocidura olivieri* (N = 105, 15.3%) and *Praomys rostratus* (N = 102, 15%) (Table 1). Rarefied species richness, standardised to 10 individuals using individual-based rarefaction (IBR), was highest in agricultural habitats (S = 5.86), intermediate in forest (S = 5.13), and lowest in village settings (S = 4.36).

Observed richness showed a similar pattern, ranging from 6 species in forest, to 9 in villages, and 12 in agricultural sites. In village habitats, rarefied richness values were consistently lower than expected under null models of species abundance distributions, suggesting reduced evenness and potential dominance effects of synanthropic small mammal species in these settings.

LASV Seroprevalence Across Species and Habitats

Antibodies to LASV were identified in 39 rodents and shrews (39/684, 5.7%) from 9 species, including *M. natalensis* (11/39, 28%), *C. olivieri* (8/39, 21%), *Lophuromys sikapusi* (8/39, 21%) and *Rattus rattus* (4/39, 10%) (Table 1).

Compared to the primary reservoir of LASV (*M. natalensis*), the likelihood of LASV seropositivity was higher in *L. sikapusi* (OR = 2.09, 95% Credible Interval (CrI) = 0.86-4.78) but with wide credible intervals suggesting uncertainty in this result (Figure 2). There was weaker evidence for increased seropositivity in the sole positive shrew

species *C. olivieri* (OR = 1.15, 95% CrI = 0.47-2.66). In contrast *M. musculus* (OR = -0.31, 95% CrI = 0.22-0.057) and *P. rostratus* (OR = 0.39, 95% CrI = 0.13-1.11) were less likely to be positive for antibodies against LASV than *M. natalensis*. Wide posterior distributions in this analysis reflect the overall low antibody prevalence, relatively small sample sizes, and uninformative priors used in the model.

Table 1 provides the number of individuals tested and seroprevalence of LASV antibodies by species, while Figure 2 illustrates the odds ratios of seropositivty for species with sufficient sample sizes compared to *M. natalensis*.

Table 1: The number of individuals detected and antibodies to LASV among those individuals.

Species	Order	Individuals (N)	LASV Antibody detected (%)	Percentage of all positive individuals	OR (95% CrI)
Mastomys natalensis	Rodentia	113	11 (9.7%)	28.2%	ref
Crocidura olivieri	Eulipotyphla	105	8 (7.6%)	20.5%	1.15 (0.47-2.66)
Praomys rostratus	Rodentia	102	2 (2%)	5.1%	0.39 (0.13-1.11)
Mus musculus	Rodentia	90	0 (0%)	0%	0.22 (0.06-0.73)
Rattus rattus	Rodentia	88	4 (4.5%)	10.3%	0.73 (0.27-1.85)
Lophuromys sikapusi	Rodentia	57	8 (14%)	20.5%	2.09 (0.86-4.78)
Mus setulosus	Rodentia	43	3 (7%)	7.7%	1.01 (0.31-2.96)
Crocidura buettikoferi	Eulipotyphla	23	0 (0%)	0%	0.44 (0.09-1.78)
Crocidura grandiceps	Eulipotyphla	15	0 (0%)	0%	0.51 (0.1-2.33)
Malacomys edwardsi	Rodentia	11	1 (9.1%)	2.6%	1.08 (0.22-4.55)
Lemniscomys striatus	Rodentia	11	0 (0%)	0%	0.59 (0.11-2.68)
Hylomyscus simus	Rodentia	9	0 (0%)	0%	
Hybomys planifrons	Rodentia	7	1 (14.3%)	2.6%	
Mastomys erythroleucus	Rodentia	4	1 (25%)	2.6%	
Crocidura theresae	Eulipotyphla	3	0 (0%)	0%	
Gerbilliscus guineae	Rodentia	2	0 (0%)	0%	
Dasymys rufulus	Rodentia	1	0 (0%)	0%	

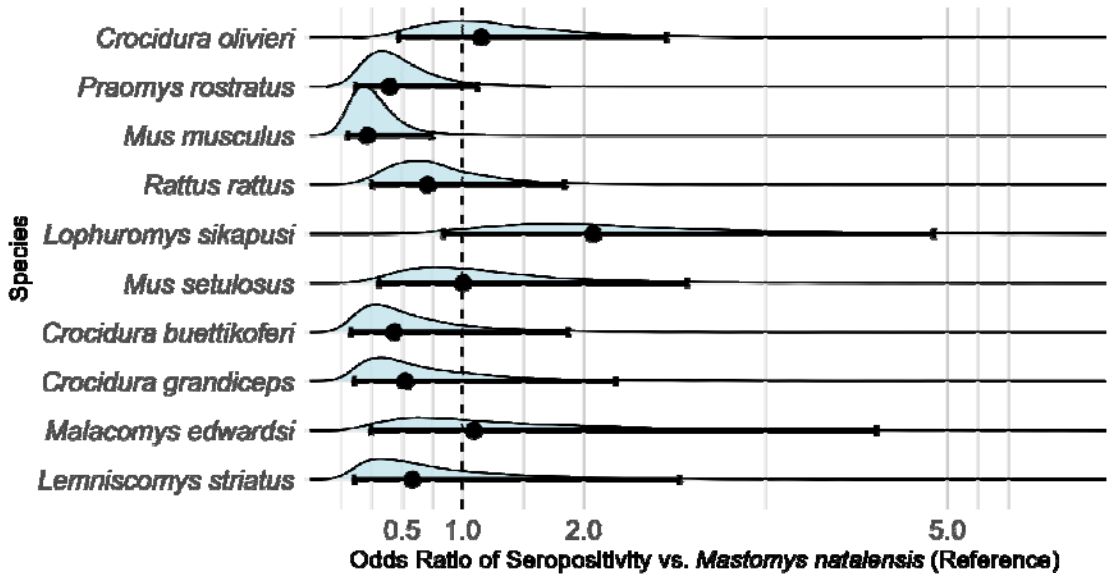

Figure 2: Odds Ratios of seropositivity to LASV among small-mammal species, compared to Mastomys natalensis. Only species with more than 10 individuals assayed for antibodies to LASV were included in this analysis.

Small mammals with antibodies to LASV were detected in three of the study villages, Lalehun (N = 18, 46%), Seilama (N = 12, 31%) and Baiama (N = 9, 23%). Lalehun had the highest percentage of antibody positive rodents (18/157, 12%), followed by Baiama (9/121, 7%) and Seilama (12/263, 5%). No positive individuals were detected in Lambayama, the most urbanised village study site, suggesting a potential association between lower seroprevalence and urban settings. Antibody positive individuals were detected during all study visits except visit 9 (2023-February).

Antibody positive small mammals were detected in all land use types. The proportion of antibody positive individuals among all small mammals trapped were similar across forest (2/44, 4.5%), agricultural (24/379, 6.3%) and village (13/261, 5%) land use types.

In exploratory analyses conducted post hoc, we observed potential differences in LASV seroprevalence by village and land use type accounting for differences in sampling effort. While these analyses were not pre-specified and should be interpreted with caution, seroprevalence appeared to be higher in Lalehun (OR = 2.56, 95% CrI = 1.27-5.27) and Baiama (OR = 1.57, 95% CrI = 0.67-3.52), than the more urbanised village Lambayama (OR = 0.21, 95% CrI = 0.06-0.68), with Seilama set as the reference village. Similarly, agricultural land use (OR = 1.27, 95% CrI = 0.66-2.5) showed the highest proportion of LASV antibody-positive small mammals, with lower seroprevalence observed in forest settings (OR = 0.85, 95% CrI = 0.24-2.71) compared to the reference land use type of village. Credible intervals for the OR of seropositivity with land use crossed an OR of 1 and were therefore not considered importantly different.

Structure of Small-Mammal Contact Networks

The number of individuals (nodes) was highest in agricultural settings (n = 379) compared to villages (n = 261) and forests (n = 44). Networks in village settings exhibited the highest overall connectivity, as indicated by the highest mean degree (mean = 3.4, standard deviation (SD) = 3.6), followed by agricultural (mean = 1.9, SD = 2.3) and forest settings (mean = 1.4, SD = 1.2). Additionally, maximum degree values—indicating individuals with the greatest number of connections—were observed in village (degree = 14) and agricultural (degree = 13) settings.

Mean betweenness centrality was highest in agricultural (mean = 1.4, SD = 8.1) and forest (mean = 1.4, SD = 4.7) settings, and lowest in villages (mean = 0.9, SD = 3.3). Network modularity, a measure of community fragmentation, was highest in agricultural settings (mean = 0.75, SD = 0.08), indicating a greater tendency for networks to separate into distinct clusters. Modularity was lower in villages (mean = 0.51, SD = 0.23) and lowest in forests (mean = 0.27, SD = 0.26).

Degree centrality varied across species, with the highest values typically observed in species more common in agricultural settings. Native rodent species *L. sikapusi*, *M. setulosus*, *P. rostratus*, and the shrew *C. olivieri* had individuals with degrees up to 13, though most were lower values (Figure 3). In villages, the invasive synanthropes *M. musculus* and *R. rattus* reached maximum degrees of 14 and 11 with high mean degrees (4.1 and 4.7 respectively). By contrast, M. natalensis, though abundant in both agricultural and village settings, had a lower maximum degree (8 in villages, 6 in agriculture) and similar mean values (2.3 and 1.8, respectively).

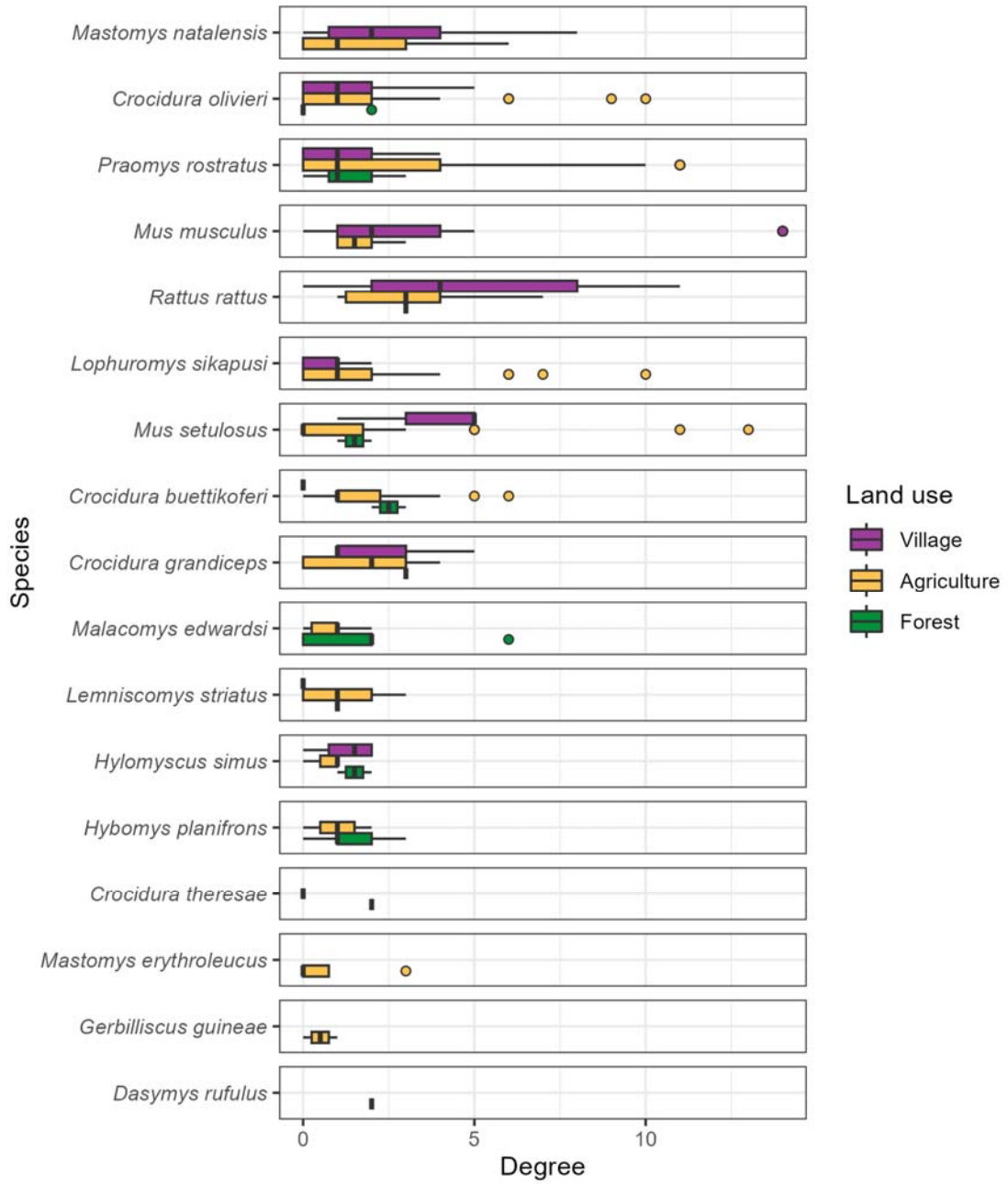

Figure 3: The degree (number of contacts) of individual small mammals stratified by species and land use type. Boxes contain the median and inter-quartile range of the degree distribution. Whiskers include the upper and lower quartile with outliers shown as points.

No consistent trend in degree centrality was observed across all species when stratified by land use type (Figure 3). For commensal species such as *M. natalensis*, *R. rattus* and *M. musculus* the median degree centrality was elevated in villages. However, for *M. natalensis* and *R. rattus*, no statistically significant differences were detected in degree distributions when stratified by land use.

Inter- and Intra-specific Mixing Patterns

Species with more detected individuals generally exhibited a greater number of inter-specific contacts ($r = 0.73$, $p < 0.001$, $n = 15$, Pearson correlation). For instance, the frequently detected species, *M. natalensis*, *P. rostratus* and *R. rattus* each had contacts with more than 11 other species. An exception to this trend was *M. musculus*, which, despite being the fourth most frequently detected species, only had observed contacts with four other species (Figure 4 and Supplementary Figures 4.1 and 4.2).

Intra-specific contacts were common for most species, but notable differences emerged across land use types. For example, *M. natalensis* interacted with 12 other species in agricultural land use settings, with 45% of all observed contacts involving other individuals of the same species (Figure 3). However, in village settings, where *M. natalensis* interacted with fewer species (9), the proportion of intra-specific contacts decreased to 31% (Supplementary Figure 4.2). Not all species showed a dominance of intra-specific contacts. For example, in agricultural areas, *L. sikapusi* interacted with 11 other species. However, a similar proportion of contacts for this species came from *P. rostratus* (27%) as from other individuals of *L. sikapusi* (26%).

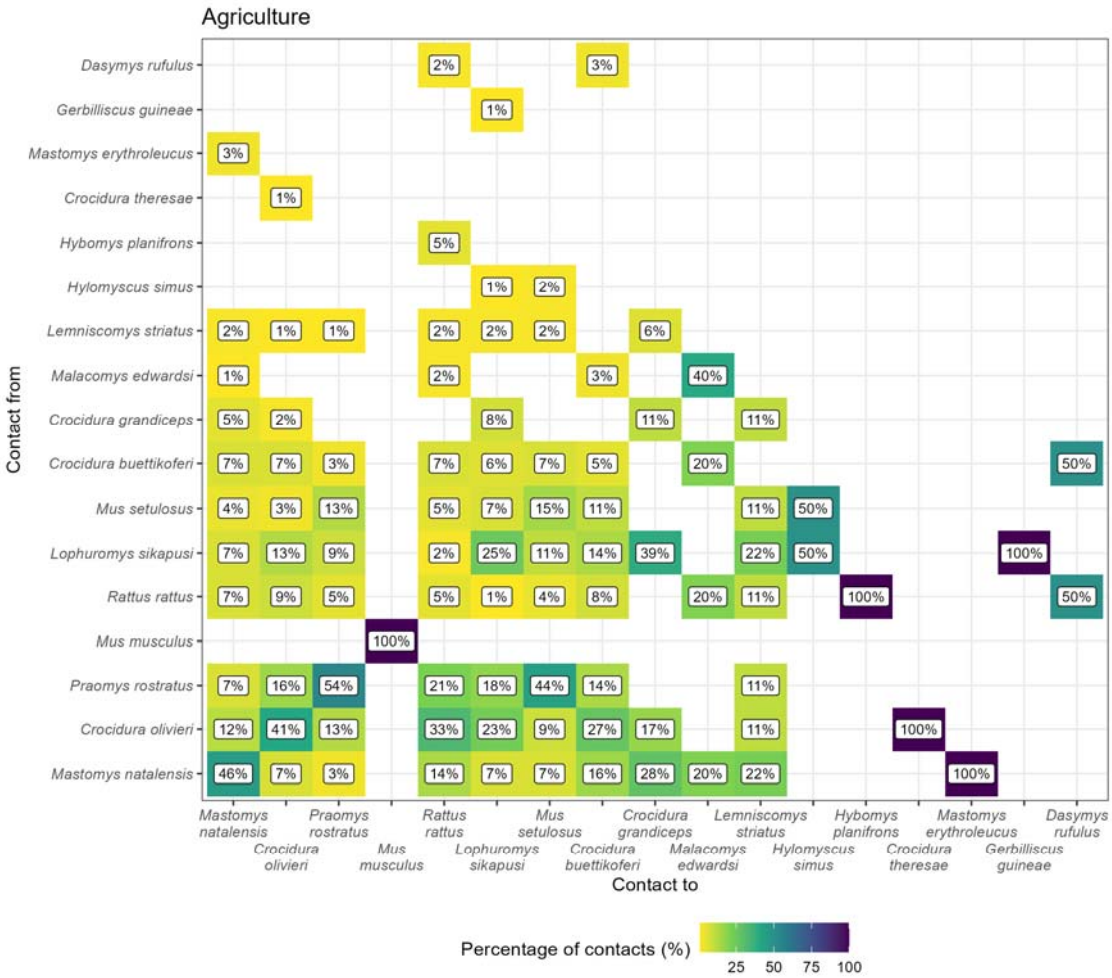

Figure 4: The proportion of contacts between individual small mammals in agricultural land use. Darker colours indicate increasing proportions of observed contacts to a species (Contact to) from named species (Contact from). Numbers in the cells correspond to the proportion of contacts to a species from a named species. For example, 45% of all contacts to Mastomys natalensis are from other M. natalensis while 9% of contacts are from Lophuromys sikapusi. Percentages sum to 100% in the `Contact to` *axis, while they may exceed 100% in Contact from. Species are ordered by the total number detected in this study with M. natalensis (N = 113) in the bottom left.*

Modeling Contact Probability in *Mastomys natalensis*

Focusing on the reservoir species of LASV, *M. natalensis*, we analysed the probability of contact using 11 ERGM models (6/10 village networks, 5/10 agricultural networks), which were deemed suitable for random-effects meta-analysis. The odds of an edge being observed for *M. natalensis* were low compared to all potential edges (observed and unobserved edges) and similar across both agricultural (Odds Ratio = 0.05, 95% Confidence Interval = 0.03-0.07, $p < 0.001$) and village land use (OR = 0.15, 95% C.I. = 0.09-0.23, $p < 0.001$) (Figure 5A). There was substantial heterogeneity in odds between networks across different visits for both land use types ($\hat{\tau}^2_{\text{agriculture}} = 0.21$, $Q = 38$, $p <$

0.001 and $\hat{\tau}^2_{\text{village}}$ = 0.29, Q = 35, p < 0.001). Compared to other rodent species *M. natalensis* was observed to form fewer contacts overall.

When examining inter-specific space-sharing, *M. natalensis* showed a reduced odds of interacting with other species in village settings (OR = 0.49, 95% C.I. = 0.33-0.73, p < 0.001) and a marginally reduced non-statistically significant odds in agricultural settings (OR = 0.6, 95% C.I. = 0.33-1.08, p = 0.089) compared to inter-specific contacts among other rodent species (Figure 5B). There was no substantial heterogeneity in inter-specific contact odds between networks ($\hat{\tau}^2_{\text{agriculture}}$ = 0.22, Q = 8.5, p = 0.07 and $\hat{\tau}^2_{\text{village}}$ = 0.09, Q = 8, p = 0.15).

Conversely, *M. natalensis* showed a statistically significantly increase in the odds of intra-specific space-sharing in agricultural settings (OR = 5.14, 95% C.I. = 2.17-12.2, p < 0.001), but not in village settings (OR = 1.96, 95% C.I. = 0.79-4.82, p = 0.15) (Figure 5C). Heterogeneity in intra-specific contact odds was low in both land use types ($\hat{\tau}^2_{\text{agriculture}}$ = 0, Q = 2.6, p = 0.63 and $\hat{\tau}^2_{\text{village}}$ = 0.14, Q = 6, p = 0.3). These findings suggest that *M. natalensis* was statistically significantly more likely to engage in intra-specific space-sharing in agricultural settings compared to other small mammals but not in villages.

Sensitivity analyses revealed no changes in the direction of effect sizes when altering the contact radius although the magnitude of the effect sizes varied (Supplementary Figures 5.1-3). Additionally, leave-one-out sensitivity analyses for influential networks did not indicate meaningful changes in the effect size magnitude or direction. These results support the robustness of the findings across space-sharing buffer area assumptions and community composition changes across visits.

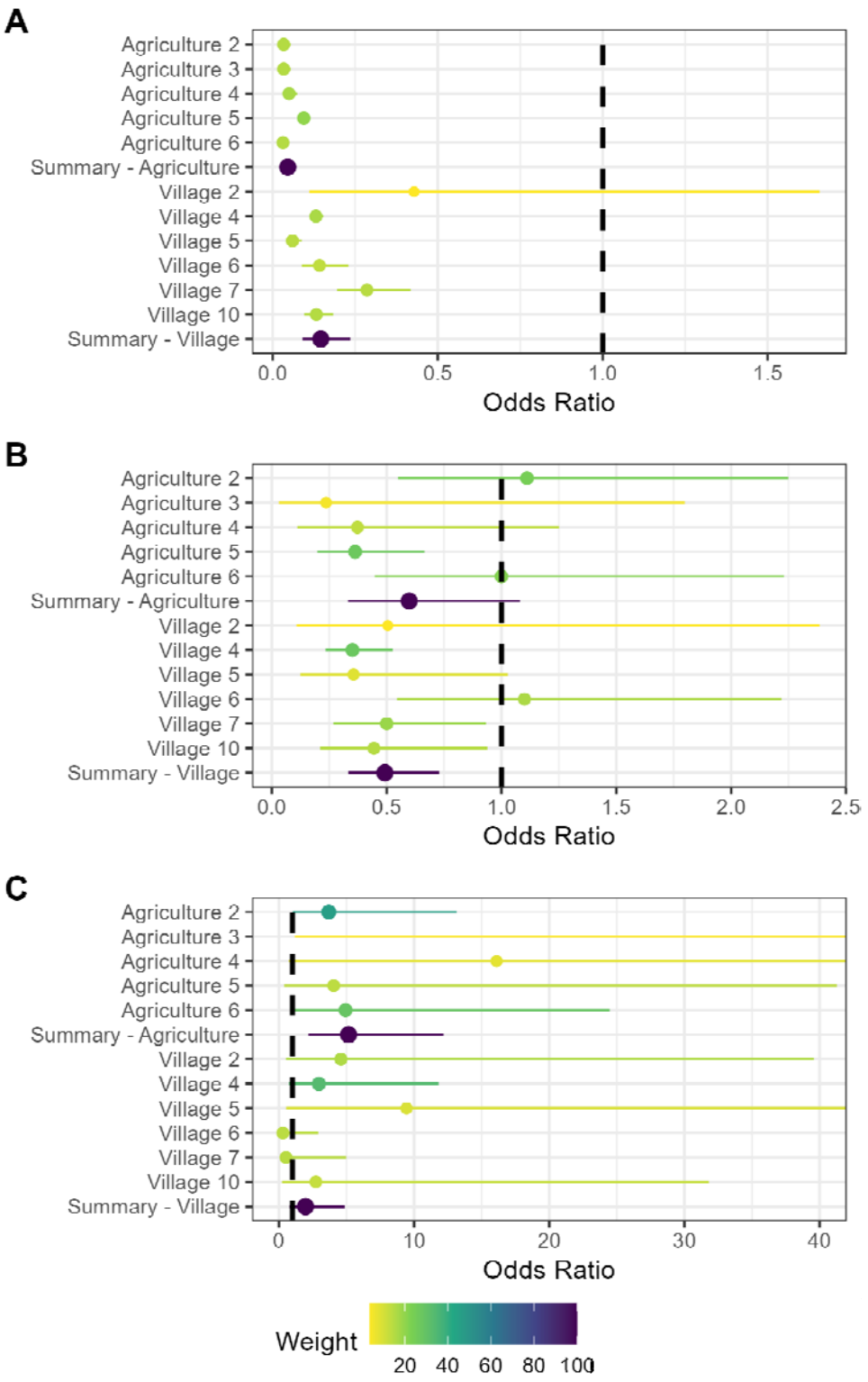

Figure 5: Random effects meta-analysis of ERGM network models reporting the odds of a contact being observed for M natalensis. A) The odds ratio of a contact being observed for M. natalensis in Agricultural or Village land use types. B) The odds ratio of a contact being observed between M. natalensis and an individual of a different rodent

species. C) The odds ratio of a contact being observed between M. natalensis and another M. natalensis.

Network Position and LASV Seropositivity

We found a significant association between an individual's number of contacts and its serostatus. Rodents and shrews that were LASV seropositive had a significantly lower mean degree centrality (mean = 3.7, SD = 2.9) compared to seronegative individuals (mean = 5.5, SD = 5.1) (W = 10018, p = 0.039). This pattern held for *M. natalensis* (W = 354, *p* = 0.048) and *L. sikapusi* (W = 99.5, *p* = 0.03), but not for *C. olivieri* (W = 429.5, *p* = 0.54).

In contrast, we found no significant difference in betweenness centrality between seropositive and seronegative individuals. This was true both for the overall community and within species-specific analyses (e.g., *M. natalensis*, W = 585.5, *p* = 0.63). This suggests that while infected animals tended to have fewer direct contacts, their role as bridges connecting different parts of the network was not distinct from that of uninfected animals.

Association between Contact Patterns and LASV Seropositivity

In our model for *M. natalensis*, an individual's odds of being LASV seropositive increased with its total number of contacts (degree; OR = 1.25, 95% CrI: 1.10–1.40). We also found some evidence that risk increased with a higher proportion of conspecific contacts (homophily; OR = 2.02, 95% CrI: 1.00–3.96).

Critically, we identified a strong negative interaction between degree and homophily (OR = 0.52, 95% CrI: 0.40–0.67). This indicates that while having more contacts increased risk overall, this effect was significantly weaker for individuals whose contacts were primarily with other *M. natalensis*. In other words, for highly connected animals, contacts with other species were more strongly associated with seropositivity than contacts with their own species.

Discussion

In the Eastern Province of Sierra Leone, we found that small-mammal contact networks varied significantly by habitat. Networks were most densely connected in village settings (mean degree = 3.4), followed by agricultural (1.9) and forest (1.4) habitats. While *M. natalensis* exhibited a high probability of intra-specific contacts in agricultural settings—suggesting these areas may serve as focal points for LASV transmission, consistent with findings linking anthropogenic environments to zoonotic disease risk[70,71]. LASV seropositivity was detected at low prevalence across nine rodent and shrew species, with *M. natalensis* comprising most seropositive individuals. However, a key finding was that seropositive individuals had lower degree centrality overall, though this pattern was not consistent across all species

A priori, we hypothesised that individuals with higher degree centrality (i.e., those with greater space-sharing interactions) would be at greater risk of LASV exposure, and that

this effect would vary by land use and species. While LASV seropositivity was detected at low prevalence across nine rodent and shrew species, seropositive individuals tended to have lower degree centrality, this pattern was inconsistent and potentially confounded by age or habitat.

Although we hypothesised that contact rates would be higher in anthropogenic habitats, global degree centrality was similar across land use types. However, the upper tail of the degree distribution was skewed towards individuals in villages and agriculture, underscoring the importance of individual-level variation not captured by aggregated metrics[72,73]. Agricultural areas also exhibited the highest rarefied species richness and a greater proportion of inter-specific space-sharing, likely reflecting edge effects that facilitate interactions between synanthropic and sylvatic species[74,75]. Notably, native species such as *P. rostratus* in agricultural habitats displayed high degree centrality, suggesting that a few highly interactive species may disproportionately shape network connectivity and transmission risk[31].

Network structure also varied across land use types. Agricultural and forest networks had higher mean betweenness centrality than village networks, indicating that individuals in those habitats more frequently acted as bridges connecting otherwise separate parts of the network. This suggests more cohesive contact structures in agricultural and forest settings, potentially supporting faster transmission cycles[76].

In contrast, we found no consistent difference in betweenness centrality between seropositive and seronegative individuals suggesting that, while seropositive rodents tended to have fewer direct contacts (lower degree), their role in linking network clusters was not markedly different.

The structure of *M. natalensis* interactions also varied by land use. In agricultural settings, *M. natalensis* exhibited significantly higher odds of intra-specific clustering consistent with the species' weak territoriality[10,77–79]. In villages, this strong tendency for intra-specific contact was not statistically significant, and the odds of forming ties with other species were reduced. This suggests that while *M. natalensis* may have a more diverse range of inter-specific encounters in villages proportionally, their overall social interactions are less frequent compared to other species in that habitat. This dynamic could dilute intra-specific transmission chains while potentially creating varied, if infrequent, spillover pathways. Movement between habitats by individuals driven by resource availability may further modulate these dynamics[80].

The significant negative interaction between degree and homophily reveals a potential ecological dynamic: for individuals with a limited number of social partners, transmission risk appears tightly linked to contact with conspecifics (*M. natalensis*). However, for highly-connected 'hub' individuals, this specificity may break down. Their increased risk appears to be driven by the sheer volume of contacts, irrespective of species, suggesting that inter-species transmission events may be particularly important at these high-traffic nodes in the network. This finding could challenge the paradigm of *M. natalensis* as the sole driver of transmission, pointing towards a more complex, community-level maintenance system, especially in settings that foster high

connectivity. However, as this analysis was exploratory and based on simulated networks, these findings must be interpreted with caution pending further validation.

The structure of agricultural networks (i.e., relatively cohesive with high intra-specific connectivity), may support rapid local spread and potential pathogen fadeout if host turnover cannot sustain transmission[76,81]. In contrast, the more fragmented village networks may allow slower transmission and longer-term persistence[82]. These contrasts suggest interventions should be tailored to land use contexts, particularly given agriculture's potential role in amplifying transmission.

The seroprevalence of LASV (5.7%), was consistent with prior estimates from Sierra Leone[16]. Our study included forest sites further from human habitation, yet the proportion of *M. natalensis* individuals testing positive was similar (~9%). However, *M. natalensis* represented a smaller fraction of total seropositive individuals (28% vs. 75%), and we detected LASV antibodies in additional species (*C. olivieri*, *P. rostratus*, *R. rattus*, *L. sikapusi*, *M. setulosus*, *Malacomys edwardsi*, *Hybomys planifrons* and *Mastomys erythroleucus*), suggesting broader host involvement than previously documented[16]. This finding supports a multi-host perspective on LASV ecology[12,18].

Several key assumptions of our network inference methods warrant consideration. First, contacts were inferred from spatio-temporal co-occurrence, which assumes that proximity is a reliable proxy for interactions capable of transmission. This assumption may not hold uniformly across species with variable movement behaviours and social structures[37]. Although our use of species-specific home range radii provides a more ecologically realistic model, our approach must still assume that a capture location represents a central point within an individual's short-term activity space[57]. While real-time tracking (e.g., radio-tagging) could refine these estimates, significant ethical and logistical challenges currently preclude their use in our study communities[83,84].

Second, our trapping methodology has inherent limitations. Like all trapping studies, we captured only a subset of the active individuals, and unobserved behavioural differences between captured and uncaptured animals could bias network inferences[85,86]. Furthermore, removal trapping may have created temporary spatial vacuums filled by transient individuals with potentially different contact behaviours[87]. Although trapping sessions were spaced by ≥3 months to allow for community re-equilibration, as recommended in regional protocols, we cannot exclude residual bias from altered community composition. Our leave-one-out meta-analysis helps account for spatial hotspots between trapping sessions, but it cannot fully address structural changes caused by the removal and replacement of key individuals.

Ultimately, replicating the study across a greater number of sites would be valuable for evaluating the consistency of these patterns and the potential impact of these methodological biases.

In conclusion, this study highlights the variability in contact rates and network structures among rodent and shrew species across land-use types in LASV-endemic settings. The complex contact networks involving multiple species may facilitate LASV transmission in unexpected ways, particularly in agricultural settings where intra- and inter-specific interactions are frequent. These findings underscore the importance of tailoring control

strategies to specific ecological contexts and host species behaviours to mitigate Lassa fever risks effectively.

References

1. McCormick JB, Webb PA, Krebs JW, Johnson KM, Smith ES., 1987. A prospective study of the epidemiology and ecology of lassa fever. *J Infect Dis 155*: 437–44

2. Basinski AJ, Fichet-Calvet E, Sjodin AR, Varrelman TJ, Remien CH, Layman NC, Bird BH, Wolking DJ, Monagin C, Ghersi BM, Barry PA, Jarvis MA, Gessler PE, Nuismer SL., 2021. Bridging the gap: Using reservoir ecology and human serosurveys to estimate lassa virus spillover in west africa. *PLoS Comput Biol 17*: e1008811

3. Jetoh RW, Malik S, Shobayo B, Taweh F, Yeabah TO, George J, Gbelee B, Teahton J, Jaryan F, Tegli M, Umeokonkwo CD, MaCauley J., 2022. Epidemiological characteristics of lassa fever cases in liberia: A retrospective analysis of surveillance data, 2019-2020. *International Journal of Infectious Diseases*

4. Shaffer JG, Schieffelin JS, Momoh M, Goba A, Kanneh L, Alhasan F, Gbakie M, Engel EJ, Bond NG, Hartnett JN, Nelson DKS, Bush DJ, Boisen ML, Heinrich ML, Rowland MM, et al., 2021. Space-time trends in lassa fever in sierra leone by ELISA serostatus, 2012–2019. *Microorganisms 9*: 586

5. Bausch DG, Demby AH, Coulibaly M, Kanu J, Goba A, Bah A, Condé N, Wurtzel HL, Cavallaro KF, Lloyd E, Baldet FB, Cissé SD, Fofona D, Savané IK, Tolno RT, et al., 2001. Lassa fever in guinea: I. Epidemiology of human disease and clinical observations. *Vector Borne Zoonotic Dis 1*: 269–281

6. Grant DS, Engel EJ, Yerkes NR, Kanneh L, Koninga J, Gbakie MA, Alhasan F, Kanneh FB, Kanneh IM, Kamara FK, Momoh M, Yillah MS, Foday M, Okoli A, Zeoli A, et al., 2023. Seroprevalence of anti-lassa virus IgG antibodies in three districts of sierra leone: A cross-sectional, population-based study. *PLOS Neglected Tropical Diseases 17*: e0010938

7. Lo Iacono G, Cunningham AA, Fichet-Calvet E, Garry RF, Grant DS, Khan SH, Leach M, Moses LM, Schieffelin JS, Shaffer JG, Webb CT, Wood JLN., 2015. Using modelling to disentangle the relative contributions of zoonotic and anthroponotic transmission: The case of lassa fever. *PLOS NEGLECTED TROPICAL DISEASES 9*

8. Walker DH, Wulff H, Lange JV, Murphy FA., 1975. Comparative pathology of lassa virus infection in monkeys, guinea-pigs, and mastomys natalensis. *Bull World Health Organ 52*: 523–534

9. Safronetz D, Rosenke K, Meade-White K, Sloan A, Maiga O, Bane S, Martellaro C, Scott DP, Sogoba N, Feldmann H., 2022. Temporal analysis of lassa virus infection and transmission in experimentally infected mastomys natalensis. *PNAS Nexus 1*: pgac114

10. Hoffmann C, Krasemann S, Wurr S, Hartmann K, Adam E, Bockholt S, Müller J, Günther S, Oestereich L., 2024. Lassa virus persistence with high viral titers following experimental infection in its natural reservoir host, Mastomys natalensis. *Nature Communications 15*: 9319

11. Borremans B, Vossen R, Becker-Ziaja B, Gryseels S, Hughes N, Van Gestel M, Van Houtte N, Günther S, Leirs H., 2015. Shedding dynamics of morogoro virus, an african arenavirus closely related to lassa virus, in its natural reservoir host mastomys natalensis. *Sci Rep 5*: 10445

12. Demby AH, Inapogui A, Kargbo K, Koninga J, Kourouma K, Kanu J, Coulibaly M, Wagoner KD, Ksiazek TG, Peters CJ, Rollin PE, Bausch DG., 2001. Lassa fever in guinea: II. Distribution and prevalence of lassa virus infection in small mammals. *Vector-Borne and Zoonotic Diseases 1*: 283–297

13. Kerneis S, Koivogui L, Magassouba N, Koulemou K, Lewis R, Aplogan A, Grais RF, Guerin PJ, Fichet-Calvet E., 2009. Prevalence and risk factors of lassa seropositivity in inhabitants of the forest region of guinea: A cross-sectional study. *PLoS Neglected Tropical Diseases [electronic resource] 3*: e548

14. Fichet-Calvet E, Becker-Ziaja B, Koivogui L, Gunther S., 2014. Lassa serology in natural populations of rodents and horizontal transmission. *Vector Borne & Zoonotic Diseases 14*: 665–674

15. Mariën J, Borremans B, Gryseels S, Soropogui B, De Bruyn L, Bongo GN, Becker-Ziaja B, Bellocq JG de, Günther S, Magassouba N, Leirs H, Fichet-Calvet E., 2017. No measurable adverse effects of lassa, morogoro and gairo arenaviruses on their rodent reservoir host in natural conditions. *Parasites & Vectors 10*: 210

16. Bangura U, Buanie J, Lamin J, Davis C, Bongo GN, Dawson M, Ansumana R, Sondufu D, Thomson EC, Sahr F, Fichet-Calvet E., 2021. Lassa virus circulation in small mammal populations in bo district, sierra leone. *BIOLOGY-BASEL 10*

17. Monath TP, Newhouse VF, Kemp GE, Setzer HW, Cacciapuoti A., 1974. Lassa virus isolation from mastomys natalensis rodents during an epidemic in sierra leone. *Science 185*: 263–265

18. Olayemi A, Cadar D, Magassouba N, Obadare A, Kourouma F, Oyeyiola A, Fasogbon S, Igbokwe J, Rieger T, Bockholt S, Jérôme H, Schmidt-Chanasit J, Garigliany M, Lorenzen S, Igbahenah F, et al., 2016. New Hosts of The Lassa Virus. *Scientific Reports 6*: 25280

19. Simons D, Attfield LA, Jones KE, Watson-Jones D, Kock R., 2023. Rodent trapping studies as an overlooked information source for understanding endemic and novel zoonotic spillover. *PLOS Neglected Tropical Diseases 17*: e0010772

20. Gilbert AT, Fooks AR, Hayman DTS, Horton DL, Müller T, Plowright R, Peel AJ, Bowen R, Wood JLN, Mills J, Cunningham AA, Rupprecht CE., 2013. Deciphering

serology to understand the ecology of infectious diseases in wildlife. *EcoHealth 10*: 298–313

21. Caron A, Cappelle J, Cumming GS, Garine-Wichatitsky M de, Gaidet N., 2015. Bridge hosts, a missing link for disease ecology in multi-host systems. *Veterinary Research 46*: 83

22. Cardenas NC, Sykes AL, Lopes FPN, Machado G., 2022. Multiple species animal movements: Network properties, disease dynamics and the impact of targeted control actions. *Veterinary Research 53*: 14

23. Keesing F, Ostfeld RS., 2021. Impacts of biodiversity and biodiversity loss on zoonotic diseases. *Proceedings of the National Academy of Sciences 118*: e2023540118

24. Albery GF, Becker DJ, Brierley L, Brook CE, Christofferson RC, Cohen LE, Dallas TA, Eskew EA, Fagre A, Farrell MJ, Glennon E, Guth S, Joseph MB, Mollentze N, Neely BA, et al., 2021. The science of the host–virus network. *Nat Microbiol 6*: 1483–1492

25. Fichet□Calvet E, Audenaert L, Barrière P, Verheyen E., 2010. Diversity, dynamics and reproduction in a community of small mammals in upper guinea, with emphasis on pygmy mice ecology. *African Journal of Ecology 48*: 600–614

26. Happi AN, Olumade TJ, Ogunsanya OA, Sijuwola AE, Ogunleye SC, Oguzie JU, Nwofoke C, Ugwu CA, Okoro SJ, Otuh PI, Ngele LN, Ojo OO, Adelabu A, Adeleye RF, Oyejide NE, et al., 2022. Increased prevalence of lassa fever virus-positive rodents and diversity of infected species found during human lassa fever epidemics in nigeria. *Microbiol Spectr 10*: e0036622

27. Rohan H., 2022. Beyond Lassa Fever: Systemic and structural barriers to disease detection and response in Sierra Leone. *PLOS Neglected Tropical Diseases 16*: e0010423

28. Simons D, Gibb R, Bangura U, Sondufu D, Lamin J, Koninga J, Jimmy M, Dawson M, Lahai J, Ansumana R, Fichet-Calvet E, Watson-Jones D, Kock R, Jones K., 2025. Land use gradients drive spatial variation in Lassa fever host communities in the Eastern Province of Sierra Leone.

29. Klitting R, Kafetzopoulou LE, Thiery W, Dudas G, Gryseels S, Kotamarthi A, Vrancken B, Gangavarapu K, Momoh M, Sandi JD, Goba A, Alhasan F, Grant DS, Okogbenin S, Ogbaini-Emovo E, et al., 2022. Predicting the evolution of the lassa virus endemic area and population at risk over the next decades. *Nature communications 13*: 5596

30. Longet S, Leggio C, Bore JA, Key S, Tipton T, Hall Y, Koundouno FR, Bower H, Bhattacharyya T, Magassouba N, Günther S, Henao-Restrapo A-M, Rossman JS, Konde MK, Fornace K, et al., 2023. Influence of landscape patterns on exposure to lassa fever virus, guinea. *Emerg Infect Dis 29*: 304–313

31. Gibb R, Redding DW, Chin KQ, Donnelly CA, Blackburn TM, Newbold T, Jones KE., 2020. Zoonotic host diversity increases in human-dominated ecosystems. *Nature 584*: 398–402

32. Albery GF, Carlson CJ, Cohen LE, Eskew EA, Gibb R, Ryan SJ, Sweeny AR, Becker DJ., 2022. Urban-adapted mammal species have more known pathogens. *Nature Ecology & Evolution 6*: 794–801

33. Ecke F, Han BA, Hörnfeldt B, Khalil H, Magnusson M, Singh NJ, Ostfeld RS., 2022. Population fluctuations and synanthropy explain transmission risk in rodent-borne zoonoses. *Nat Commun 13*: 7532

34. Sauvage F, Langlais M, Yoccoz NG, Pontier D., 2003. Modelling hantavirus in fluctuating populations of bank voles: The role of indirect transmission on virus persistence. *Journal of Animal Ecology 72*: 1–13

35. Laverty SM, Adler FR., 2009. The role of age structure in the persistence of a chronic pathogen in a fluctuating population. *Journal of Biological Dynamics 3*: 224–234

36. Salkeld DJ, Salathé M, Stapp P, Jones JH., 2010. Plague outbreaks in prairie dog populations explained by percolation thresholds of alternate host abundance. *Proceedings of the National Academy of Sciences 107*: 14247–14250

37. Perkins SE, Cagnacci F, Stradiotto A, Arnoldi D, Hudson PJ., 2009. Comparison of social networks derived from ecological data: Implications for inferring infectious disease dynamics. *Journal of Animal Ecology 78*: 1015–1022

38. Clay CA, Lehmer EM, Previtali A, St. Jeor S, Dearing MD., 2009. Contact heterogeneity in deer mice: Implications for sin nombre virus transmission. *Proc Biol Sci 276*: 1305–1312

39. Böhm M, Hutchings MR, White PCL., 2009. Contact networks in a wildlife-livestock host community: Identifying high-risk individuals in the transmission of bovine TB among badgers and cattle. *PLOS ONE 4*: e5016

40. Drewe JA, Eames KTD, Madden JR, Pearce GP., 2011. Integrating contact network structure into tuberculosis epidemiology in meerkats in south africa: Implications for control. *Prev Vet Med 101*: 113–120

41. White LA, Forester JD, Craft ME., 2017. Using contact networks to explore mechanisms of parasite transmission in wildlife. *Biological Reviews 92*: 389–409

42. Pebesma E., 2018. Simple features for r: Standardized support for spatial vector data. *The R Journal 10*: 439–446

43. R Core Team., 2021. R: A language and environment for statistical computing. Vienna, Austria: R Foundation for Statistical Computing

44. Fichet-Calvet E., 2014. Chapter 5 - lassa fever: A rodent-human interaction. Johnson N, ed. *The role of animals in emerging viral diseases*. Boston: Academic Press, 89–123

45. Percie Du Sert N, Ahluwalia A, Alam S, Avey MT, Baker M, Browne WJ, Clark A, Cuthill IC, Dirnagl U, Emerson M, Garner P, Holgate ST, Howells DW, Hurst V, Karp NA, et al., 2020. Reporting animal research: Explanation and elaboration for the ARRIVE guidelines 2.0. *PLOS Biology 18*: e3000411

46. Happold DCD, Kingdon J eds., 2013. *Mammals of Africa. Vol. 3: Rodents, hares and rabbits*. London: Bloomsbury

47. Monadjem A, Taylor PJ, Denys C, Cotterill FPD., 2015. *Rodents of sub-saharan africa: A biogeographic and taxonomic synthesis*. Berlin, München, Boston: DE GRUYTER

48. QIAGEN., 2023. DNeasy blood & tissue kits. Available at: https://www.qiagen.com/us/products/discovery-and-translational-research/dna-rna-purification/dna-purification/genomic-dna/dneasy-blood-and-tissue-kit. Accessed. January 20, 2023

49. Altschul SF, Gish W, Miller W, Myers EW, Lipman DJ., 1990. Basic local alignment search tool. *J Mol Biol 215*: 403–410

50. Gabriel M, Adomeh DI, Ehimuan J, Oyakhilome J, Omomoh EO, Ighodalo Y, Olokor T, Bonney K, Pahlmann M, Emmerich P, Lelke M, Brunotte L, Ölschläger S, Thomé-Bolduan C, Becker-Ziaja B, et al., 2018. Development and evaluation of antibody-capture immunoassays for detection of lassa virus nucleoprotein-specific immunoglobulin m and g. *PLOS Neglected Tropical Diseases 12*: e0006361

51. Soubrier H, Bangura U, Hoffmann C, Olayemi A, Adesina AS, Günther S, Oestereich L, Fichet-Calvet E., 2022. Detection of lassa virus-reactive IgG antibodies in wild rodents: Validation of a capture enzyme-linked immunological assay. *Viruses 14*: 993

52. Grüner N, Stambouli O, Ross RS., 2015. Dried blood spots - preparing and processing for use in immunoassays and in molecular techniques. *J Vis Exp*: 52619

53. Bürkner P-C., 2017. brms: An R package for Bayesian multilevel models using Stan. *Journal of Statistical Software 80*: 1–28

54. McGlinn D, Xiao X, McGill B, May F, Engel T, Oliver C, Blowes S, Knight T, Purschke O, Gotelli N, Chase J., 2024. Mobr: Measurement of biodiversity

55. McGlinn DJ, Blowes SA, Dornelas M, Engel T, Martins IS, Shimadzu H, Gotelli NJ, Magurran A, McGill BJ, Chase JM., 2025. Disentangling nonrandom structure from random placement when estimating β□diversity through space or time. *Ecosphere 16*: e70061

56. Carslake D, Bennett M, Bown K, Hazel S, fer S, Begon M., 2005. Space–time clustering of cowpox virus infection in wild rodent populations. *Journal of Animal Ecology 74*: 647–655

57. Wanelik KM, Farine DR., 2022. A new method for characterising shared space use networks using animal trapping data. *Behav Ecol Sociobiol 76*: 127

58. Broekman MJE, Hoeks S, Freriks R, Langendoen MM, Runge KM, Savenco E, Harmsel R ter, Huijbregts MAJ, Tucker MA., 2023. HomeRange: A global database of mammalian home ranges. *Global Ecology and Biogeography 32*: 198–205

59. Hunter DR, Handcock MS, Butts CT, Goodreau SM, Morris M., 2008. Ergm: A package to fit, simulate and diagnose exponential-family models for networks. *Journal of Statistical Software 24*: 1–29

60. Royle JA., 2004. N-mixture models for estimating population size from spatially replicated counts. *Biometrics 60*: 108–115

61. Fiske I, Chandler R., 2011. Unmarked: An r package for fitting hierarchical models of wildlife occurrence and abundance. *Journal of Statistical Software 43*: 1–23

62. Butts CT., 2008. **Network** : A package for managing relational data in *r*. *J Stat Soft 24*

63. Handcock MS, Hunter DR, Butts CT, Goodreau SM, Krivitsky PN, Morris M., 2022. Ergm: Fit, simulate and diagnose exponential-family models for networks. The Statnet Project (https://statnet.org)

64. Riley RD, Higgins JPT, Deeks JJ., 2011. Interpretation of random effects meta-analyses. *BMJ 342*: d549

65. Viechtbauer W., 2010. Conducting meta-analyses in r with the metafor package. *Journal of Statistical Software 36*: 1–48

66. Cochran WG., 1954. The combination of estimates from different experiments. *Biometrics 10*: 101–129

67. Borenstein M, Hedges LV, Higgins JPT, Rothstein HR., 2010. A basic introduction to fixed-effect and random-effects models for meta-analysis. *Research Synthesis Methods 1*: 97–111

68. Cheung MW-L., 2019. A guide to conducting a meta-analysis with non-independent effect sizes. *Neuropsychol Rev 29*: 387–396

69. Bauer DF., 1972. Constructing confidence sets using rank statistics. *Journal of the American Statistical Association 67*: 687–690

70. Pei S, Yu P, Raghwani J, Wang Y, Liu Z, Li Y, Cheng Y, Lin Q, Song C, Dharmarajan G, Faust CL, Tian Y, Xu Y, Liang Y, Qu J, et al., 2024. Anthropogenic land

consolidation intensifies zoonotic host diversity loss and disease transmission in human habitats. *Nature Ecology & Evolution 9*: 99–110

71. Carlson CJ, Brookson CB, Becker DJ, Cummings CA, Gibb R, Halliday FW, Heckley AM, Huang ZYX, Lavelle T, Robertson H, Vicente-Santos A, Weets CM, Poisot T., 2025. Pathogens and planetary change. *Nature Reviews Biodiversity 1*: 32–49

72. Farine DR, Whitehead H., 2015. Constructing, conducting and interpreting animal social network analysis. *Journal of Animal Ecology 84*: 1144–1163

73. Silk MJ, Fisher DN., 2017. Understanding animal social structure: Exponential random graph models in animal behaviour research. *Animal Behaviour 132*: 137–146

74. Despommier D, Ellis BR, Wilcox BA., 2007. The Role of Ecotones in Emerging Infectious Diseases. *EcoHealth 3*: 281–289

75. Pruvot M, Chea S, Hul V, In S, Buor V, Ramassamy J-L, Fillieux C, Sek S, Sor R, Ros S, Nuon S, San S, Ty Y, Chao M, Sours S, et al., 2024. Small mammals at the edge of deforestation in Cambodia: Transient community dynamics and potential pathways to pathogen emergence. *One Earth 7*: 123–135

76. Keeling MJ, Eames KTD., 2005. Networks and epidemic models. *J R Soc Interface 2*: 295–307

77. Anderson PK., 1961. Density, social structure, and nonsocial environment in house-mouse populations and the implications for regulation of numbers. *Trans N Y Acad Sci 23*: 447–451

78. Whisson DA, Quinn JH, Collins KC., 2007. Home range and movements of roof rats (rattus rattus) in an old-growth riparian forest, california. *Journal of Mammalogy 88*: 589–594

79. Borremans B, Hughes NK, Reijniers J, Sluydts V, Katakweba AAS, Mulungu LS, Sabuni CA, Makundi RH, Leirs H., 2014. Happily together forever: Temporal variation in spatial patterns and complete lack of territoriality in a promiscuous rodent. *Population Ecology 56*: 109–118

80. Marien J, Kourouma F, Magassouba N, Leirs H, Fichet-Calvet E., 2018. Movement Patterns of Small Rodents in Lassa Fever-Endemic Villages in Guinea. *Ecohealth 15*: 348–359

81. Messinger SM, Ostling A., 2009. The consequences of spatial structure for the evolution of pathogen transmission rate and virulence. *The American Naturalist 174*: 441–454

82. Peel AJ, Pulliam JRC, Luis AD, Plowright RK, O'Shea TJ, Hayman DTS, Wood JLN, Webb CT, Restif O., 2014. The effect of seasonal birth pulses on pathogen persistence in wild mammal populations. *Proceedings of the Royal Society B: Biological Sciences 281*: 20132962

83. Mohr K, Leirs H, Katakweba A, Machang'u R., 2007. Monitoring rodents movements with a biomarker around introduction and feeding foci in an urban environment in tanzania. *afzo 42*: 294–298

84. Borremans B, Reijniers J, Hughes NK, Godfrey SS, Gryseels S, Makundi RH, Leirs H., 2017. Nonlinear scaling of foraging contacts with rodent population density. *Oikos 126*: 792–800

85. Parmenter RR, Yates TL, Anderson DR, Burnham KP, Dunnum JL, Franklin AB, Friggens MT, Lubow BC, Miller M, Olson GS, Parmenter CA, Pollard J, Rexstad E, Shenk TM, Stanley TR, et al., 2003. Small-mammal density estimation: A field comparison of grid-based vs. Web-based density estimators. *Ecological Monographs 73*: 1–26

86. Moore JE, Swihart RK., 2005. Modeling patch occupancy by forest rodents: Incorporating detectability and spatial autocorrelation with hierarchically structured data. *The Journal of Wildlife Management 69*: 933–949

87. Sullivan TP, Sullivan DS., 2013. Influence of Removal Sampling of Small Mammals on Abundance and Diversity Attributes: Scientific Implications