

# Discussion draft

## Conclusions and future research directions.

This thesis provides new insights into the role of rodent ecology on LASV transmission and subsequent spillover into human populations in Sierra Leone through the association of anthropogenic land use change with rodent community structure. The methods used within this thesis to investigate the research questions included a literature review and synthesis of publicly available data on rodent sampling across West Africa and primary data collection through systematic rodent trapping in a Lassa fever endemic region of Eastern Sierra Leone. This final thesis chapter summarises the key findings, including a reflection on the general strengths and limitations of the work, as more detailed issues pertaining to each study are covered at the end of each chapter. The final sections of this chapter focus on the implications for the understanding of Lassa fever epidemiology and provides suggestions for future research.

## Principle findings of the thesis

Within the Lassa fever disease system, primary data from rodent trapping studies in Sierra Leone remains limited (Monath et al. 1974; McCormick et al. 1987; Mahy 1992; Barnett et al. 2000; Leski et al. 2015; Bangura et al. 2021). Few rodent ecological studies in Sierra Leone or across the endemic region have been designed to investigate the role of the wider rodent community in LASV transmission (Demby et al. 2001; Fichet-Calvet et al. 2005; Mariën et al. 2018; Mariën et al. 2020). Much of the data that is available within the endemic region is derived from opportunistic sampling of rodents following Lassa fever outbreaks in human communities (Monath et al. 1974; Wulff, Fabiyi, and Monath 1975; Safronetz et al. 2010; Yadouleton et al. 2019; Happi et al. 2022). Therefore, Chapter 2 was a quantitative review of available studies to understand the current state of rodent and rodent-associated pathogen sampling across West Africa. Data from included studies were synthesised and compiled to produce a database that has been made available for re-use by the scientific and public health community (Simons 2022b). This study found that primary rodent trapping could usefully complement available consolidated datasets (i.e., IUCN and GBIF) by expanding the areas sampled. I identified regions and host-pathogen associations that have been comparatively under-sampled and therefore locations and disease systems in which inference may be limited when based on previously available datasets. Finally, the produced measure of relative sampling bias can be used to adjust for these spatial and taxonomic biases in future models to predict the risk of zoonotic infectious disease emergence.

From the findings of Chapter 2, in addition to that reported in the literature previously, multiple rodent host species are potentially involved in LASV transmission and maintenance among rodent populations (Wulff, Fabiyi, and Monath 1975; Demby et al. 2001; Fichet-Calvet et al. 2014; Olayemi et al. 2016; Yadouleton et al. 2019). Despite the increasing evidence of multi-species involvement, data remain limited to investigate the structure of these rodent populations in multi-species communities, particularly when incorporating how these communities may change along anthropogenic land use gradients. I therefore designed and implemented a three-year rodent trapping study in a Lassa fever endemic region of Eastern Sierra Leone. I designed this strategy to sample rodent communities along a land use gradient from natural forest settings, through agriculture and into areas of human habitation to characterise the rodent communities that exist within these habitats. Sampling was conducted at high temporal frequency to incorporate expected seasonal dynamics in rodent occupancy and abundance. I found that the primary host of LASV (*M. natalensis*) had a high probability of occurrence in areas of human habitation and agricultural settings and was effectively absent from less anthropogenically disturbed forest habitats. However, this was not the case in locations of high human population density where this species was found to occur at much lower rates than previously expected. If, as suggested by current evidence, *M. natalensis* conveys the greatest risk of subsequent human infection with LASV we would expect a general trend of increasing risk of LASV spillover from rodent communities

along a gradient from forest to villages along an anthropogenic land use gradient (Bonwitt et al. 2017). Although this risk may be dramatically reduced in the most urbanised settings by the low prevalence of the primary rodent host species. In concordance with a recent study conducted in Sierra Leone I identified seropositivity against LASV in a rodent species more commonly found in forest environments (*M. edwardsi*), it may be that this species is important for transmitting LASV among spatially discontinuous population of *M. natalensis* (Bangura et al. 2021).

Within these rodent communities I also observed important biotic interactions between species. For example, the presence of an invasive rodent species (*M. musculus*), which was solely detected in areas of high human population density, was negatively associated with the occurrence of *M. natalensis* and other native rodent species. The same was not true for the association between another invasive rodent (*R. rattus*) and native rodent species where co-occurrence was common. These findings indicate that the ongoing expansion of the range of these species in West Africa will result in biotic interactions that could govern the distribution of native rodent species across the region (Lippens et al. 2017). While there is some evidence that both *M. musculus* and *R. rattus* can be infected with LASV their contribution to onward transmission is not known but is expected to be currently minimal compared to *M. natalensis* (Demby et al. 2001). Therefore, the potential changes in rodent community structure in response to land use change and invasive species expansion will likely alter LASV pathogen dynamics within the endemic region and could have important implications for Lassa fever outbreak risk.

The findings from Chapter 3 indicate that rodent communities within Sierra Leone have heterogenous contact rates which will play an integral role in pathogen transmission. I found that across an anthropogenic land use gradient rodent contact networks share properties of sparse networks, with a similar number of contacts for each individual rodent. However, there remains a substantial amount of heterogeneity in the network structure within and between land use types. For example, I found that *M. natalensis* was more likely to form intra-specific contacts than inter-specific contacts. Differential contact rates would promote pathogen transmission if this species were a more competent host than other potential hosts (Luis, Kuenzi, and Mills 2018). Intra-specific contacts among *M. natalensis* were more likely to occur within agricultural settings than areas of human habitation and might suggest that while human infection occurs primarily within settings of human habitation transmission among rodent communities predominates in agricultural settings. This could have important implications for public health interventions to control the transmission of LASV among rodent populations if rodent control is focussed solely in villages.

Finally, we found low prevalence of antibodies to LASV among our sampled rodent communities within an area of Sierra Leone considered highly endemic for Lassa fever. We tested 684 rodents trapped over 43,266 trap nights, detecting a prevalence of 3.3%. This is substantially lower than has been detected elsewhere in the region (e.g., 2% in Guinea and 76% in Nigeria) although the design of these studies varied (Adesina et al. 2023). For example, previous work in Sierra Leone and Nigeria has typically relied on opportunistic trapping of rodents in locations during a known outbreak (Monath et al. 1974; Wulff, Fabiyi, and Monath 1975; Happi et al. 2022). In contrast, longer term studies in Guinea have found high rodent seroprevalence in locations that do not typically report human Lassa fever cases []. This finding highlights the challenges of understanding this complex dynamic interaction between rodent ecology and pathogen ecology. I anticipate that the low levels of LASV in our study location are reflective of wider multi-year cycles of pathogen transmission and that if this work were to continue over a substantially longer timescale we would observe greatly elevated LASV transmission among the rodent community.

## General strengths and limitations

This thesis has several strengths. Principally among these is the adoption of Open Science practices (Foster and Deardorff 2017; Powers and Hampton 2019). Throughout this thesis I have produced and archived study protocols, data collection tools, raw and processed data and pre-printed manuscripts on Open Science platforms. Taking this approach, alongside making all code to reproduce data cleaning, processing and visualisation on my GitHub account will allow researchers to obtain the information that matters to them from my research outputs (Simons 2023). For example, to support re-use of the dataset in Chapter 2 I produced an accompanying web-based application to allow researchers to visualise the processed data and associated meta-data with the aim of improving uptake of this novel datasource (Simons 2022a). In addition,

the rodent trapping data has been deposited on the Pathogen Harmonised Surveillance (PHAROS) database, an open-access repository produced by the Verena Institute to allow researchers to re-use the data while the manuscripts produced from Chapter 3 and 4 proceed through the stages of peer-reviewed scientific publication (The Verena Institute 2023).

A methodological strength of this thesis is the systematic approach taken to sampling rodents across a land use gradient. The design of the rodent trapping study was selected to limit the impact of sampling biases on inference of habitat occupancy by rodent species within diverse communities. The approach taken within this thesis will be of use to other rodent ecology researchers, particularly those conducting rodent-associated disease research in West Africa, the adoption of the study protocol and data collection tools will improve harmonisation of study designs supporting meta-analysis of rodent ecology studies across geographic regions. To raise awareness of the availability of these study protocols I have presented the study design and results at scientific workshops and conferences within the field of disease ecology and beyond.

The work in this thesis benefited from strong integration within the communities in which the research was conducted. Throughout the study design stage informal consultations were conducted with local researchers and village communities within Eastern Sierra Leone, these consultations guided the selection of research questions and study design, incorporating valuable local knowledge to better understand the structure of rodent communities and the role these play in Lassa fever risk to human populations. Study protocols were piloted within the local communities and revised following input from field workers and members of the village community to improve acceptability of these protocols and accuracy of data collection. The success of the rodent trapping study would not have been possible without the strong support we received from our local collaborators and highlighted the importance of integrating local knowledge early in the research process.

Finally, through the research process I was able to directly contribute to the training and development of local researchers in Sierra Leone. During the training sessions I held on rodent trapping, sampling and laboratory analysis I was impressed with the existing skill set of my local collaborators leading me to reconsider the role of training and development within international collaborations. These collaborations do not always value the existing skills of local researchers and do not tend to promote sustainable career progression by helping researchers to advance beyond technical competence. It was evident that lack of local skills was not a cause of the limited prior research into rodent ecology in Sierra Leone and that local researchers would benefit from support to develop themselves as independent scientists. To help my collaborators develop the qualifications required to attract research funds I have assisted them in acquiring individual funding to enrol in higher education degrees at international universities.

Several general limitations of the thesis are important to highlight. First, the thesis had a narrow geographic scope and the applicability of my findings on the structure of rodent communities and the interactions between them may not apply to the wider endemic region of Lassa fever. More work is required to replicate this study in other regions of Sierra Leone and within West Africa to assess whether these are local effects or if the findings are replicable across geographic scales.

A second important limitation is the method of trapping used. In designing the rodent trapping study I balanced the need to obtain unbiased estimates of rodent occurrence with the requirement to be able to construct space sharing interactions between individual rodents. To achieve this I adopted a grid-based approach with trapping occurring in four-night sessions. Other researchers in the field have adopted line- and web-based trapping which are able to survey a greater area but would introduce difficulties in assessing space sharing []. The number of trap-nights required to adequately sample a habitat is also not known, with individual researchers adopting different numbers based on funding availability, timelines and the expected behaviour of local rodent populations. Whether my decision to adopt grid-based trapping or to trap for four nights may have biased the obtained data is difficult to assess without comparable studies in Sierra Leone.

Finally, I did not assess for acute infection with LASV in the samples obtained from the rodent trapping study. The primary reason for this was the low expected yield of positive results. Unpublished research from a rodent trapping study conducted in Eastern Sierra Leone suggested an incidence of infection of ~1% (Moses, L. personal communication), this matches with data from a recent publication showing an incidence of 0.3% in the same region. Financial resources were not available to perform viral PCR alongside serology, therefore serology only was performed to investigate how LASV transmission may be occurring within rodent contact

networks. Samples have been stored in conditions that would allow subsequent investigation for acute LASV infection and I am currently exploring collaborations that would allow this.

## Implications for Lassa fever epidemiology and future research directions

Current spatial risk models are limited by the spatial and temporal biases in sampling of rodent, pathogen and human populations. Figure 1 shows the outputs from four models of Lassa fever risk in human populations based on currently available data (Fichet-Calvet and Rogers 2009; Mylne et al. 2015; Redding et al. 2016; Basinski et al. 2021).

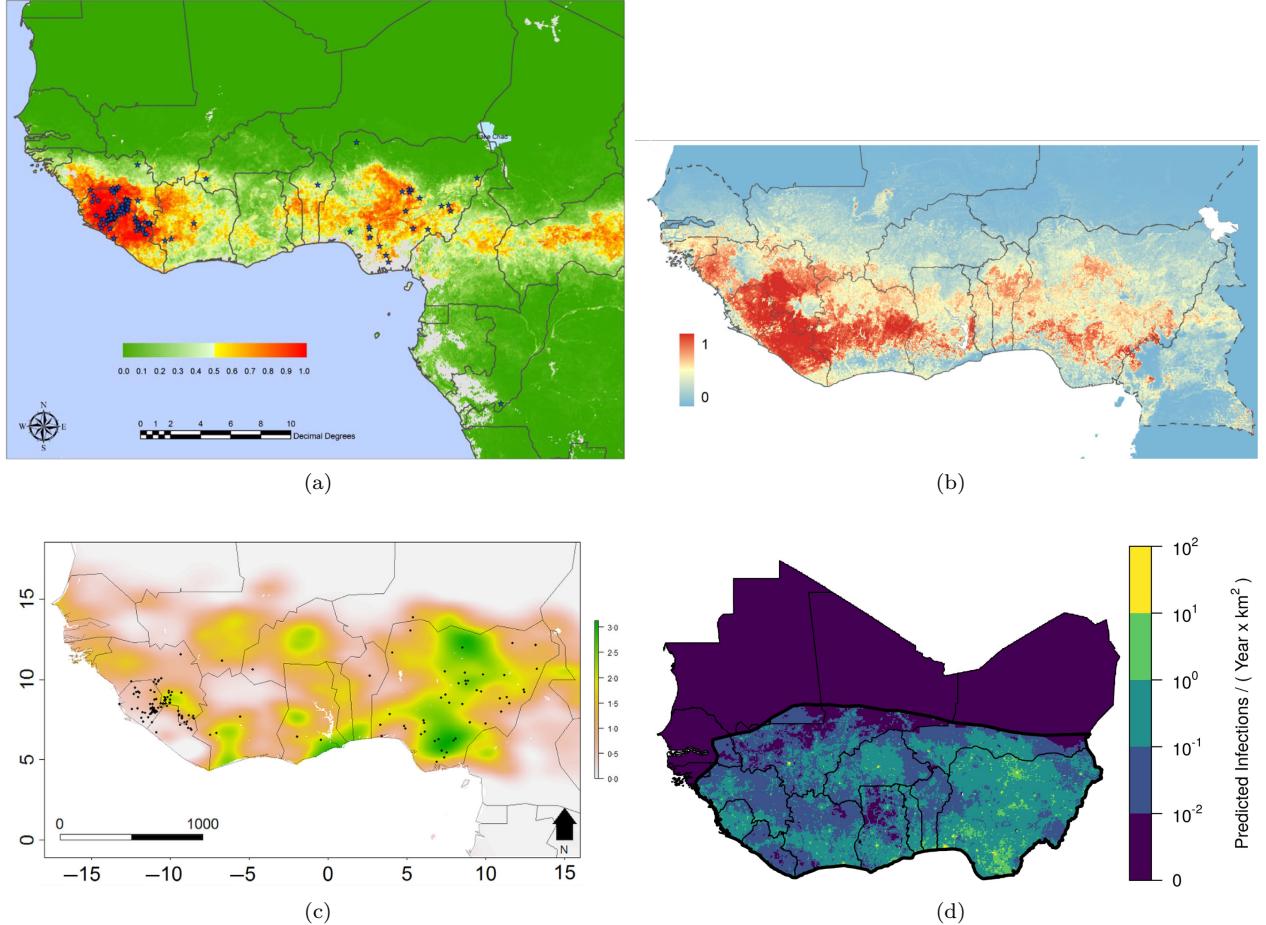


Figure 1: Risk maps of Lassa fever in West Africa, images reproduced from (a) Fichet-Calvet, 2009, (b) Mylne, 2015, (c) Redding, 2016, (d) Basinski, 2021

A recent illustrative example is an identified outbreak of Lassa fever in Accra, Ghana. Here, 12 acute cases were identified in February 2023 in a region not previously considered endemic and a region considered at low risk by previous spatial models of Lassa fever risk [1]. It is likely that a lack of host or pathogen sampling in Ghana led to inaccurate estimates of the true risk of disease emergence. For example, while *M. natalensis*, the primary host of LASV is expected to exist throughout Ghana sampling for this species has only been conducted at X locations, or Z% of the country [1]. Assessment for LASV within this species' population in Ghana has been conducted at even fewer sites with no acute LASV infections detected [1]. This led to reduced estimates of spillover risk in Ghana consistently from multiple models [1]. Which ultimately failed to predict that the pathogen was already prevalent or recently invaded the country leading to the observed outbreak. Models therefore specified on biased data may lead to under-appreciation of the current risk of disease outbreaks limiting a health services ability to plan for these events.

Quantifying the spatial biases of rodent host and pathogen sampling is an important first step in overcoming the limitations of sampling bias. Although in order to deliver potentially efficacious interventions such as rodent control or raising awareness within human communities, a better understanding of both temporally and spatially varying risk is required [1]. An example of the beneficial effect of long-term monitoring elsewhere in Africa are studies of rabies [2] in wild and domesticated dogs. In this disease system, mobility and contact networks of potential hosts of rabies have been developed that allow targeted interventions (i.e., dog vaccination programmes) to reduce the incidence of rabies infections in human communities [3]. Interventions such as these are only able to achieve cost-effective control through harmonised study protocols leading to unbiased estimates of animal population dynamics and pathogen transmission [4].

Limiting studies to investigating the pathogen either within hosts or humans will not allow us to understand the interplay between these dynamic systems which occur on different geographic and temporal scales. A recent study (Adesina 2023) conducted in Nigeria reported extraordinarily high prevalence of active infection with Lassa fever among rodent communities. In one town, rodent LASV prevalence was reported at 70%. Sequences of pathogens were sequenced and compared with human derived sequences from the same geographic region, to compare most recent common ancestors to estimate transmission dynamics among the rodents in these settings. Unfortunately, this work was solely conducted in rodent communities but only data from the two native Mastomys species were reported. Evidence of reverse zoonosis in urban settings with pathogen spillback from human populations to rodent populations based on most recent common ancestor, potential issues with bottlenecks, sampling etc.

Rodent borne transmission of LASV occurs at different spatial and temporal scales to human infection. Multiple studies have found asynchrony in human infection pressure and transmission among rodents. This is evident in locations where human seroprevalence is high while among rodents it is low and in the reverse where transmission among rodents is observed but human cases of infection are limited.

This may be occurring for several reasons. First, rodent life span is a small fraction of humans with most native rodent species having life expectancy of less than 1 year. This increased turnover in the rodent population compared to human population means that seroconversion and the reflection of this on prior exposure to LASV is difficult to reconcile. For example, a 30 year old human individual could have been infected at any point after they lost maternally derived immunity from which point they would develop antibodies that are detectable for up to X years. For rodents this is further complicated by difficulties in age estimation as it is unclear during which time period they may have developed immunity.

In settings with high pathogen prevalence among rodents it follows that humans are exposed to the pathogen during their younger years. It may be that this leads to a mild infection and therefore these infections aren't detected by the passive surveillance in place for Lassa in the endemic regions. Therefore while the risk of infection in these locations is high the risk for Lassa fever disease are low. Further work is needed to tease this out. One approach would be to quantify the force of infection among rodents and people by running paired prospective cohort studies of both rodents and humans. To achieve the aims of this better data collection on rodent ages are required. For example, rodents can be age classified by using eye lens weight. This is suitable for most rodent species but only a few have standardised charts to compare eye lens weight to in order to estimate age. These exist for *M. natalensis* but not for other species that may be involved in transmission. This approach is not suitable for shrews.

This study was looking at land use this is an important process ongoing in West Africa, likely to be important for Lassa epidemiology. Understanding that risk is dramatically governed by land use through the rodent communities is important... Changes in rodent communities occurs at a different timescale to population abundance fluctuations and pathogen invasion. To get at this high intensity long-term studies are needed to identify causal processes in land use change.

In addition to understanding current risk of rodent associated zoonoses, there is significant interest need in to better understanding the effect of climate, land use and biodiversity change on the future risk of endemic and emerging zoonoses [5]. Unfortunately, extant biases in host and pathogen sampling will reduce the confidence in inferred risk models if underlying taxonomic and spatial biases are not incorporated [6]. For example, in the rodent associated Lyme disease system in North America long-term studies have been able to estimate the effect of multi-year cycles of habitat productivity (i.e., masting) and animal biodiversity with small mammal

abundance and pathogen prevalence. Similar multi-season or multi-year cycles are expected to exist with in rodent associated zoonoses in West Africa, but this has not been systematically assessed []. For example in Lassa fever, increased human cases are expected to be associated with a population boom in the rodent hosts of LASV, based on data collected from elsewhere in these rodents range []. The existence of similar dynamics in West Africa, and whether they occur homogenously throughout the region is not known. It is likely that populations dynamics among rodent hosts will play an important role in rodent associated pathogen dynamic and disease spillover risk into human populations in these settings but the magnitude of their effect cannot be estimated from currently available datasources.

Similarly, long-term studies in Oceania have characterised the link between land use and bat host occurrence and abundance with zoonoses spillover into domesticated animals []. In order to quantify the association of land use change on rodent host occurrence and abundance improved harmonisation of rodent sampling data is needed []. Inferring the effect of ongoing land use change on rodent communities from currently available data is challenging when studies have been conducted over short time periods []. Landscape level responses to changing habitats among rodent communities may currently be masked by inter-seasonal or inter-annual variations in rodent occurrence and abundance that are detected within shorter sampling periods. In order to identify long term trends in changing occupancy in response to land use comprehensive studies such as those applied to Nipah virus in Australia are required in West Africa.

Further, in chapter 3 I found that the interaction between *M. musculus*, an invasive rodent species, and the primary host of LASV may be a cause of the apparent lack of human infections within urban settings. This needs to be expanded on with studies of pathogen competence within non-*Mastomys* rodents and studies in other urban settings within the endemic region to investigate whether this is a consistent finding. Conducting systematic trapping across the anthropogenic land use gradient allowed the construction of potential rodent contact networks. Chapter 4 showed that ...

Harmonisation of rodent trapping methods and adoption of open science practices. Similar approaches for estimation of rodent abundance

Maintaining the dataset produced in Chapter 2 and incorporating this into actively curated biodiversity resources would be particularly helpful in increasing the availability of zoonoses host species' data in currently under-surveilled settings. Current geographic and temporal biases in this data will not solely be counteracted by increased data availability. Despite this, incorporation of primary data onto maintained data repositories such as GBIF or PHAROS will allow researchers, conservationists and public health specialists to interrogate up-to-date consolidated datasets to investigate the occurrence of hosts of rodent-associated zoonoses. Progress in the adoption of Open Science practices with comprehensive study protocols, detailed meta-data and appropriate licensing are needed to support data consolidation and reuse (ref?). Increased availability of high quality zoonoses host data will improve confidence in current estimates of zoonoses spillover risk.

Potential...

Spillover rates into human populations, age-stratified contact rates how does infection actually occur? Immunity and disease severity among humans, who gets sick, why?

Expansion of invasive species and changes in LASV epidemiology among humans.

## Concluding remarks

A greater understanding of rodent community ecology is required to design public health interventions against Lassa fever in the endemic region. This thesis aimed to gain a better understanding of how these communities are structured along anthropogenic land use gradients with a view to informing interpretation of human Lassa fever epidemiology. This was achieved through a systematic review and synthesis of published rodent trapping studies across West Africa and primary data collection through a three-year rodent trapping study. Current biases in sampling of rodent hosts and their pathogens across West Africa limit the inference able to be drawn from currently available data on the current and future risks of zoonotic disease emergence. Results from the rodent trapping survey characterised the structure of rodent communities within the Lassa fever endemic region of Eastern Sierra Leone and informed the contact networks pathogens such as LASV will transmit through within rodent populations. These findings can be used to inform the design of combined

rodent and human epidemiological studies of Lassa fever and to guide the development of public health interventions.

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