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Density thresholds for Mopeia virus invasion and persistence in its host *Mastomys natalensis*

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HIGHLIGHTS

- We built a spatially explicit and individual-based SEIR model of Mopeia virus.
- Sharp density thresholds are observed for persistence, not invasion.
- Host dispersal is important for the spread and persistence of the infection.
- In the year following invasion, herd immunity can hinder persistence.
- The model is most sensitive to transmission rate and infectious period.

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ABSTRACT

Well-established theoretical models predict host density thresholds for invasion and persistence of parasites with a density-dependent transmission. Studying such thresholds in reality, however, is not obvious because it requires long-term data for several fluctuating populations of different size. We developed a spatially explicit and individual-based SEIR model of Mopeia virus in multimammate mice *Mastomys natalensis*. This is an interesting model system for studying abundance thresholds because the host is the most common African rodent, populations fluctuate considerably and the virus is closely related to Lassa virus but non-pathogenic to humans so can be studied safely in the field. The simulations show that, while host density clearly is important, sharp thresholds are only to be expected for persistence (and not for invasion), since at short time-spans (as during invasion), stochasticity is determining. Besides host density, also the spatial extent of the host population is important. We observe the repeated local occurrence of herd immunity, leading to a decrease in transmission of the virus, while even a limited amount of dispersal can have a strong influence in spreading and re-igniting the transmission. The model is most sensitive to the duration of the infectious stage, the size of the home range and the transmission coefficient, so these are important factors to determine experimentally in the future.

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1. Introduction

The driving force behind all infectious diseases is the transmission of parasites from an infected to a susceptible host (Begon et al., 2002). Sometimes direct contact between individuals is required for transmission, but also indirect contact (e.g. through contaminated excreta) can be sufficient. When the contact rate between hosts is constant, for example due to social rules, the transmission is called 'frequency-dependent'. When the contact rate between hosts increases at higher population densities, the transmission rate is 'density-dependent' (Begon et al., 2002; Lloyd-Smith et al., 2004, 2005; Keeling and Rohani, 2007). In the latter case, theoretical models predict that there exists a host abundance threshold, which is the minimum number of host individuals needed for an infection to spread in a population (Kermack and McKendrick, 1927).

The nature of the abundance threshold depends on the situation, and two different thresholds can be discerned (Deredec and Courchamp, 2003; Lloyd-Smith et al., 2005). When an infectious agent enters a naive (uninfected and wholly susceptible) population, the density of hosts should be high enough to ensure sufficient contacts between individuals during which transmission can take place. This is then called an invasion threshold (Deredec and

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Courchamp, 2003; Lloyd-Smith et al., 2005). When an infection is already present in a population, persistence will depend on the capacity of the population to produce a sufficient supply of new susceptible individuals, through birth or immigration. Thus, while invasion and persistence thresholds are part of the same concept, they are not completely equivalent (Deredec and Courchamp, 2003; Lloyd-Smith et al., 2005).

In general, human populations do not vary extensively in time, and consequently, these concepts have been hard to demonstrate for human infections (but see critical community size in measles, Bartlett, 1960; Nåsell, 2005; also mass vaccinations aiming at herd immunity are based on the same principle). However, wildlife populations often exhibit larger fluctuations, in which case such thresholds are more likely to be crossed. Periods during which a pathogen can invade, spread and (temporarily) persist, are then interrupted by periods below the threshold, when the pathogen can no longer invade or persist. One example of such an abundance threshold over time can be seen for plague in populations of great gerbils in Kazakhstan (Davis et al., 2004).

In this paper, we study the infection dynamics of Mopeia virus (an East African arenavirus) in its natural host the multimammate mouse (*Mastomys natalensis*). This is an interesting model system for studying epidemiological abundance thresholds, for several reasons. Mopeia virus transmission is thought to be density dependent because the home ranges of its host overlap more at higher densities (Monadjem and Perrin, 1998), so the existence of an abundance threshold is to be expected. Next, because the host populations exhibit large (seasonal as well as interannual) population fluctuations (ranging from <50 to 600 animals per hectare in the same year Leirs et al., 1997), this threshold is likely to be crossed regularly. Additionally, *M. natalensis* is one of the most common African small mammals and an agricultural pest, and as such, its ecology and demography have been studied extensively (e.g. Leirs, 1994). Last, because Mopeia virus is not known to cause disease in humans but is very closely related to the West African Lassa virus (which causes Lassa fever, a severe hemorrhagic fever in humans and thus difficult to study, but with the same rodent species as natural host), insights into Mopeia virus transmission can be used to better understand Lassa virus epidemiology.

As a first step in studying Mopeia virus dynamics, we here develop an individual-based SEIR model to simulate the infection dynamics. Using the current best estimates of the model parameters, the model should be able to tell us if abundance thresholds are to be expected and in which situations. Moreover, as not all epidemiological parameters have been determined experimentally, this model can give important insights into the relative importance of the different transmission parameters in the infection dynamics. This should provide a more reliable foundation for future studies and a guide as to which parameters are more important to be determined experimentally and to what detail.

2. Model

To simulate the spread of Mopeia virus infection, we built an individual-based spatially explicit SEIR model, taking into account demography, spatial behaviour of the host, and the infection dynamics of Mopeia virus. Birth, death, dispersal and the transitions between infection stages are applied stochastically on individual hosts, because the host population can be small and/or the numbers of infected mice can be low. We did not alter the demographic and spatial components after infection, because no overt signs of disease were observed after Lassa virus infection (Walker et al., 1975; Günther and Lenz, 2004).

2.1. Demographic component

In the course of a year, *M. natalensis* shows strong population size fluctuations. These are due to seasonal reproduction, which is driven by seasonal rainfall (May until July, Leirs et al., 1997). Because it is to be expected that the population density greatly influences the infection transmission, it is important to include demography in the model. We include two maturation stages: juvenile and adult, which are governed by the demographic processes of birth, maturation and death, as outlined below.

Birth: Reproduction rates (v) between 0.044 and 0.3 births per day per adult have been observed (Leirs, 1994; Leirs et al., 1997). In the model, an average birth rate of 0.172 mice/day is assumed during the breeding season. Only female adults can reproduce, and the birth rate is adjusted accordingly ($v=0.344$ mice/day). Empirical data have shown that litter sizes are smaller at high densities (Leirs et al., 1997). This density dependence was found to differ between seasons (in reality, some litters are raised outside the breeding season), and is smallest in the reproductive season. For this reason, we do not include this in the model. The litter size was taken to be 11, i.e., the average litter size in Morogoro (11.31, in Leirs, 1994). At birth, the pups are randomly assigned to a sex.

Maturation: In years with a normal rain pattern, the juveniles undergo a long period of reduced growth in the dry season. Because of this, reproduction is postponed until the next breeding season (Leirs et al., 1993, 1997). This delay in the maturation is included in the model.

Death: The mortality of *M. natalensis* was found to be density dependent: at low population densities (below a critical density of 150 mice/ha), the mortality rate (μ) was measured to be 0.0104 mice/day; at higher population densities (well above 150 mice/ha), μ was found to be 0.0158 mice/day (Leirs et al., 1997). In the model, the density-dependent mortality was implemented in the same way, using the above mortality rates, separated by a critical density C. Due to the increased mortality above the critical density C, the population density will level off quickly above C and for this reason, in the following, we refer to C as the carrying capacity. This method, albeit unconventional, has the advantage that it mimics the empirical data and that it allows to change the population density using a single parameter C. Different habitats can sustain a different number of mice (depending on food availability, soil type, etc., Borremans et al., 2011), and therefore, in the analysis we will not restrict ourselves to the experimentally measured $C=150$ mice/ha and vary C to study the effect of the carrying capacity of the habitat on the infection dynamics. It proved necessary though, to lower the overall mortality by 30% in order to adjust it to the birth rate, so that the population would not fade out. This is due to the fact that the experimentally determined mortality rates were somewhat overestimated, because they were inferred from the mice's presence in an open grid of 1 ha, not taking into account emigration as a possible alternative to death when animals disappear from the grid.

2.2. Spatial component

The *M. natalensis* individuals in the simulations are bound to their home burrow. While foraging, an individual moves through the landscape around his burrow, covering only a small area of the total focus. This home range size was estimated by Leirs (590 m^2 for females and 598 m^2 for males, Leirs, 1994; Leirs et al., 1996) and Monadjem and Perrin (1998, 652 m^2 for females and 718 m^2 for males). Due to this limited home range, contacts between individuals cannot be described using a random mixing

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