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The role of heterogeneity on the invasion probability of mosquito-borne diseases in multi-host models

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HIGHLIGHTS

- We investigate the invasion probability of vector-borne diseases with multiple hosts.
- We study the role of heterogeneous transmission among hosts using branching processes.
- We show that invasion probability via infected vector increases with heterogeneity.
- We show that invasion probability via infected host can decrease with heterogeneity.
- We find higher risk of outbreaks occurrence when pathogen is introduced by vectors.

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ABSTRACT

Heterogeneity in transmission and stochastic events can play a significant role in shaping the epidemic dynamics of vector-borne infections, especially in the initial phase of an outbreak. In this work, by using multi-type branching process methodologies, we assess how heterogeneities in transmission among a large number of host groups can affect the invasion probabilities of a mosquito-borne disease.

We show with both analytical and numerical methods that heterogeneities in transmission can shape the invasion probabilities differently from how they affect the basic reproduction number (R_0). In particular, we find that, while R_0 always increases with the heterogeneity, the invasion probability after the introduction of infected hosts can decrease with the increase of transmission heterogeneity, even approaching zero when the number of host groups is very large. In addition, we show that the invasion probability via infected vectors is always larger than via infected hosts when heterogeneous transmission is sufficiently high.

Our findings suggest that, for multi-species infections (e.g. West Nile fever and Rift Valley fever) or for single-species infections with patchy host distribution, the introduction of primary infected vectors may represent a higher risk for major outbreaks occurrence than introductions of infected hosts.

1. Introduction

Vector-borne diseases are infections transmitted by the bite of infected arthropods, such as mosquitoes, ticks and fleas. Among them, mosquito-borne diseases (such as malaria, West Nile virus, chikungunya, dengue fever, Rift Valley fever and yellow fever) represent major threats to human and animal health.

Mathematical models have been widely developed aiming at describing the complexity of host-mosquito-pathogen interactions. The first mathematical description of mosquito-borne infections is due to Sir Ronald Ross, who provided a synthetic theoretical framework for the transmission of human malaria (Ross, 1911). His pioneering work, later extended by Macdonald (1952), provided several insight on vector-borne disease control and prevention. The main achievement of the Ross–Macdonald model was the identification of a threshold condition for disease invasion – the basic reproduction number – which still is, to this day, the most important metric in mathematical epidemiology (see Section 2.1 for more details).

In more recent years the basic theory of the Ross–Macdonald model has been expanded to include eco-epidemiological complexities inherent in malaria and other mosquito-borne infections, such as waning immunity (Aron, 1988), multiple strain co-circulation

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(Ferguson et al., 1999), cross-immunity (Adams et al., 2006), seasonal variations (Dietz, 1971), and spatial, behavioural or genetic heterogeneity in transmission (Woolhouse et al., 1997). Although this large amount of work significantly improved the understanding of vector-borne diseases epidemiology, two recent review articles on mathematical models of mosquito-borne diseases published by Reiner et al. (2013) and Smith et al. (2014) pointed out that there exist still a small number of studies looking at the consequences of poorly mixed vector-host encounter and spatial heterogeneity on disease dynamics.

Several empirical surveys provided evidence for the existence of heterogeneities in the frequency of mosquito bites among humans. Differences in mosquito biting rate have been observed among people with different blood group type (Shirai et al., 2004), carbon dioxide emission – hence size – (Zainulabeuddin and Leal, 2007), health status of both vector and host (Ferguson and Read, 2004), skin bacteria composition (Verhulst et al., 2011), etc. Similarly, heterogeneous biting rates have been observed among the avian community of West Nile virus host species both in USA (Kilpatrick et al., 2006a) and Europe (Roiz et al., 2012).

From the epidemiological point of view, seminal theoretical papers by Barbour (1978), Dye and Hasibeder (1986) and Hasibeder and Dye (1988) based on deterministic models showed that heterogeneities in the transmission of vector-borne infections play a major role in defining the threshold condition for disease invasion. In particular, they show that, when vector biting rate differs among host groups, the basic reproduction number is always larger than under the hypothesis of completely homogeneous mixing.

However, deterministic models ignore the contribution of demographic stochasticity, which is especially relevant when the prevalence in hosts and/or vectors is low (for instance at the beginning of an outbreak). Historically, stochastic epidemic models have been developed within the framework of continuous-time Markov processes or branching processes at the population level (Bartlett, 1964; Bailey, 1975), although more complex models taking into account many details of mosquito-borne infections have been recently proposed (Magori et al., 2009; Perkins et al., 2013).

The development of the theory of stochastic epidemic models has allowed, largely on the basis of branching process approximations, for the computation of disease invasion probabilities in several types of models (Andersson and Britton, 2000). In the case of vector-borne diseases, branching process approximations have been extensively analysed in the case of one host (Bartlett, 1964; Griffiths, 1972; Ball, 1983; Lloyd et al., 2007) and two host groups (Lloyd et al., 2007). Here, we extend the analysis to the case of n different host groups by studying the effect of stochastic processes on the invasion probability of a vector-borne infection under different assumptions of heterogeneous host-vector mixing. In particular, we analyse the role of the number of host groups (Section 3.3.1.) and of the heterogeneity in vector biting rates among them (Section 3.3.2.) in shaping the invasion probability of infections introduced in a new population by a single vector or host.

2. The deterministic multi-group model

The multi-group model (Dye and Hasibeder, 1986) is a generalization of the classical Ross–Macdonald host-vector model (Ross, 1911; Macdonald, 1952), which takes into account different host types. Each host type can indicate either a host species (Kilpatrick et al., 2007) or the patch (or group) to which an individual host belongs in populations characterized by spatial/behavioural heterogeneity (Woolhouse et al., 1997). The model assumes that the vector population (V) and the n host type populations (H_j, with $j=1,\ldots,n$) are constant in their sizes, and that they can be subdivided at any time t into two compartments with respect to the disease: infectious (I for vectors and Y_j for type-j host) or susceptibles (V–I and H_j–Y_j, respectively).

A susceptible vector [host] can acquire infection by biting [being bitten by] an infected host [vector]. The multi-group model assumes that the rate at which vectors bite hosts is a constant, say λ, independent of host density. This implies that host density does not represent a limiting factor for vectors to find a valuable meal. Then, the rate at which susceptible vectors become infected is equal to λ, times the probability that the bite is on an infected host, times the probability that the vector becomes infected (assumed to be a constant value q_V independent of host type).

Letting γ_j represent the proportion of vector bites allocated to type-j host (with $\sum_j \gamma_j = 1$) and δ the mortality rate of vectors, one arrives at the equation

$$\dot{I} = \beta(V-I)\left(\sum_{j=1}^{n} \gamma_j \frac{Y_j}{H_j}\right) - \delta I \tag{1}$$

where $\beta = \lambda q_V$.

Similarly, a susceptible host of type j gets infected when it is bitten by an infected vector (this occurs at rate $\lambda\gamma_j\, I\,[1-Y_j/H_j]$), times the probability of becoming infected in that case, say q_H. Then, if ζ represents the recovery rate of infected hosts (assumed also to be constant among types), the equations for infected type-j hosts are

$$\dot{Y}_j = \alpha\gamma_j I\left(1-\frac{Y_j}{H_j}\right) - \zeta Y_j, \quad j=1,\ldots,n \tag{2}$$

where $\alpha = \lambda q_H$.

Eqs. (1) and (2) constitute the system of differential equations for the multi-group model as in Dye and Hasibeder (1986).

Transmission is considered to be homogeneous if the fraction of bites γ_j allocated to type-j hosts is proportional to their relative abundance, i.e. if $\gamma_j = h_j$ (where $h_j = H_j/H$ and $H = \sum_j H_j$ is total host abundance). Correspondingly, the heterogeneity generated by variations among different host types in their exposure to mosquito feeding can be measured by the variance of the γ_j/h_j weighted by the frequency distribution of h_j, namely $\mathrm{var}(\gamma_j/h_j; h_j) = \sum_j[(\gamma_j - h_j)^2/h_j]$.

2.1. The basic reproduction number

The basic reproduction number, R_0, represents the average number of secondary infections that one primary infective individual produces during its infectious period into an entirely susceptible population (Diekmann et al., 1990). In the case of vector-borne diseases the infection cycle lies in a two-step process: from host-to-vector and from vector-to-host. Then, as shown by Dye and Hasibeder (1986), the basic reproduction number for multi-group model (1) and (2) can be broken up in two different terms: $R_0^{H_jV} = \beta\gamma_j V/(\zeta H h_j) = r_{HV}\gamma_j/h_j$, which represents the average number of secondary infections among vectors that arise from a single type-j host; and $R_0^{VH_j} = \alpha\gamma_j/\delta = r_{VH}\gamma_j$, which represents the average number of secondary infections among type-j hosts that arise from a single vector. Thus, over the entire transmission cycle, one infective host or vector gives rise to an average of

$$R_0 = \sum_{j=1}^{n} R_0^{H_jV} R_0^{VH_j} = \frac{\alpha\beta V}{\zeta\delta H}\sum_{j=1}^{n}\frac{\gamma_j^2}{h_j} = r_{HV}r_{VH}\sum_{j=1}^{n}\frac{\gamma_j^2}{h_j} \tag{3}$$

where $\sum_j \gamma_j^2/h_j$ is defined as the relative reproduction number, $R_{0,rel}$, which represents the proportion of R_0 due to the heterogeneous mixing among hosts and vectors (Woolhouse et al., 1997). In particular, Dye and Hasibeder (1986) showed that the relative