



Original Research Article

Vector borne diseases on an urban environment: The effects of heterogeneity and human circulation[☆]A. Iggidr^a, J. Koiller^{b,c}, M.L.F. Penna^d, G. Sallet^a, M.A. Silva^e, M.O. Souza^{f,*}^a Inria, Université de Lorraine, and CNRS, IECL (UMR 7502), ISGMP Bat. A, Ile du Saulcy, 57045 Metz Cedex 01, France^b Instituto Nacional de Metrologia, Qualidade e Tecnologia (INMETRO), Av. Nossa Senhora das Graças 50, Duque de Caxias, RJ 25250-020, Brazil^c Instituto Politécnico, Universidade do Estado do Rio de Janeiro, Rua Bonfim 25, Friburgo, RJ 28625-570, Brazil^d Departamento de Epidemiologia e Bioestatística, Instituto de Saúde da Comunidade, Universidade Federal Fluminense, Rua Marquês de Paraná 303, Niterói, Rio de Janeiro 24030-210, Brazil^e Escola de Matemática Aplicada, Fundação Getúlio Vargas, Praia de Botafogo 190, Rio de Janeiro, RJ 22250-040, Brazil^f Departamento de Matemática Aplicada, Universidade Federal Fluminense, Rua Mário Santos Braga, s/n, Niterói, Rio de Janeiro 24020-140, Brazil

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ABSTRACT

We study the effect of human circulation and host/vector heterogeneities on the onset of epidemics of arboviruses. From a meta-population dynamics based on the classical Bailey–Dietz model, we derive a multi-group model under three assumptions: (i) fast host sojourn time-scale; (ii) mosquitoes do not move; (iii) time homogeneity and strong connectivity of human circulation. Within this modelling framework, three different kinds of R_0 appear: (i) the “true” or “global” R_0 —derived from the corresponding next generation matrix; (ii) the uniform R_0 —obtained if the patches are taken homogeneous; (iii) the local R_0 —obtained if the patches are disconnected. We show that there is relevant epidemiological information associated to all of them. In particular, they can be used to understand the effects of changing the circulation on the value of the global R_0 . We also present additional results on the effects on R_0 of different vector control policies, and a simulation with data from the city of Rio de Janeiro, Brazil.

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1. Introduction

Arboviruses are becoming increasingly widespread in a growing number of countries (Gubler, 2002; Liang et al., 2015). While such a fast growth is likely to have many causes, the increasingly expansion of the associated vectors habitat's is certainly an important one—e.g. Lambrechts et al. (2010). Indeed, two of the main and most effective vectors for such diseases, namely *Aedes albopictus* (the tiger mosquito) and *Aedes aegypti* (the yellow fever mosquito), are spreading well beyond tropical areas (Bhatt et al., 2013; Kraemer et al., 2015). The consequences of this expansion is already evident: The south of Europe (Italy and southern France) has already documented cases of chikungunya (CDC, 2016), in the US there is a growing number of dengue cases

reported (Añez and Rios, 2013), and Zika is becoming a widespread concern (Samarasekera and Triunfol, 2016).

In large urban areas, the main vector for these arboviruses, *A. aegypti*, has a number of particular features: it is anthropophilic, lives only on urban or semi-urban areas, and it typically disperses no more than 500 m from its birth place—cf. Honorio et al. (2003). On the other hand, the existence of large urban transportation networks allow for long host movements. Thus, hosts can infect or become infected in regions that are geographically apart from their residence area, and circulation patterns should play a major role in the disease dynamics. Such importance seems to be first pointed out by Adams and Kapan (2009), Cosner et al. (2009), and Stoddard et al. (2009) in somewhat different contexts. Nevertheless, it seems apparent that this circulation effect will increase the population heterogeneity, and this typically favours epidemics (Hasibeder and Dye, 1988; Dushoff and Levin, 1995; Smith et al., 2004).

The study of multi-group models of epidemics dynamics can be traced back at least to the work of Lajmanovich and Yorke (1976), and subsequently in Nold (1980) and Hethcote and Yorke (1984). Additional modelling work in subdivided populations

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can be found in Hasibeder and Dye (1988), Dushoff and Levin (1995), Adler (1992), Andreasen and Christiansen (1989) and also in the general analysis by Diekmann et al. (1990) formulated in terms of a continuous model. For meta-population models early analytical work can be traced back at least to the work of Arino and van den Driessche (2003).

The recognition of the importance of host movement and population heterogeneities led to an increasing interest in the modelling literature on multi-group and meta-population models, and this has driven much recent work aiming to assess the impact of spatial heterogeneities and circulation patterns in the disease dynamics (Bowong et al., 2013; Xiao and Zou, 2014; Bichara et al., 2015; Stoddard et al., 2013; Padmanabha et al., 2012; Teurlai et al., 2012; Arino et al., 2012; Gatto et al., 2012). Theoretical reviews on multi-group and meta-population models can be found in Thieme (2003) and in Arino (2009), respectively.

Most of earlier work seems to have focused on a specific framework, i.e. either on meta-population or multi-group models. The latter might be thought as a fast sojourn limit of the former—e.g. Adams and Kapan (2009). Nevertheless a systematic study of such asymptotic limit seems to be lacking. The aim of this work is three-fold: (i) to provide a systematic derivation of a multi-group model in the fast-sojourn limit such that the multi-group model parameters are obtained in terms of the parametrisation used in the meta-population model; (ii) understand how different degrees of heterogeneity impact on the disease dynamics; (iii) illustrate how such model can be parametrised with available data.

2. Methods

The aim of this session is to present a consistent derivation of a multi-group model for urban environments. We begin by taking a detailed Lagrangian view for describing the disease dynamics. The urban environment will be denoted the City, and we assume that it is naturally divided into n patches—which can be thought as districts and suburbs. The key assumptions in the model are as follows:

1. Each host individual is registered in some patch i , and we assume that individuals do not change their registered domiciles. Hence the total population that is registered in patch i , namely N_i^h , changes only as the result of birth and death. Further, there is no outward or inward flow—i.e. the outskirts are included in the city, and visitors are not accounted for.
2. The commuting time-scale within the city is fast when compared to either the demographic or epidemiological time-scales. The ratio of the latter time-scales to the former will be denoted by the positive parameter $\epsilon \ll 1$. The commuting dynamics is also assumed to be time homogeneous.
3. Mosquitoes do not move.
4. In each patch, the disease dynamics is given by a Bailey–Dietz model, namely consists of two sub-models: SIR for hosts and SI for vectors (Bailey, 1975; Dietz, 1975).

Remark 2.1. We now comment on the assumptions above:

1. While this seems to be a simplifying assumption, we are not aware of any available data that could be used to model domicile registration dynamics. Moreover, disease notifications usually take domiciles for registration.
2. In large cities, a significant fraction of the population commutes daily—sometime through long distances. The typical average time for commuting ranges from 25 min to 50 min (Pereira and

Schwanen, 2015), which is faster than the time-scales of both human, mosquito and disease dynamics. See Rocha et al. (2013) and Souza (2014) for a discussion of the different time-scales and possible fast-slow scalings in arboviruses models. The assumption of time homogeneity is a simplification.

3. Most of urban mosquitoes—as is the case of *A. aegypti* are lazy flyers, and usually do not fly more than 500 m away from the place they were born (Maciel-de Freitas and Lourenço-de Oliveira, 2009).
4. The Bailey–Dietz model has been extensively used to describe dengue dynamics (Esteva and Vargas, 1998; Nishiura, 2006), and it is the simplest model for such dynamics.

Let $N_{ij}^h(t)$ be the number of individuals registered in patch i that are in patch j at time t . These N_{ij} are further subdivided in susceptible, infected and recovered, $S_{ij}^h, I_{ij}^h, R_{ij}^h$. Thus, we have the following aggregation relations:

$$S_i^h = \sum_{j=1}^n S_{ij}^h, \quad I_i^h = \sum_{j=1}^n I_{ij}^h, \quad R_i^h = \sum_{j=1}^n R_{ij}^h \quad \text{and} \quad (1)$$

$$N_i^h = S_i^h + I_i^h + R_i^h.$$

The total population is $N^h(t) = \sum_{i=1}^n N_i^h(t)$. Recalling that mosquitoes do not move from the residence patch, let us denote by N^v the total mosquito population, which is also divided into patch populations $N_i^v, i = 1, \dots, n$. Analogously, this population of N_i^v mosquitoes is further subdivided in S_i^v and I_i^v . Under the assumptions discussed above, we are led to consider the following model with $3n^2 + 2n$ equations:

$$\begin{aligned} \dot{S}_{ij}^h &= \mu_i^h (F_{ij}^h - S_{ij}^h) - \beta_{ij}^h \frac{S_{ij}^h}{\sum_{k=1}^n N_{kj}^h + A_j} I_j^v + \epsilon^{-1} \sum_{k=1}^n M_{jk}^i S_{ik}^h \\ \dot{I}_{ij}^h &= \beta_{ij}^h \frac{S_{ij}^h}{\sum_{k=1}^n N_{kj}^h + A_j} I_j^v - (\mu_i^h + \gamma_i) I_{ij}^h + \epsilon^{-1} \sum_{k=1}^n M_{jk}^i I_{ik}^h \\ \dot{R}_{ij}^h &= \gamma I_{ij}^h - \mu_i^h R_{ij}^h + \epsilon^{-1} \sum_{k=1}^n M_{jk}^i R_{ik}^h \\ \dot{S}_i^v &= \mu_i^v (F_i^v - S_i^v) - S_i^v \sum_{k=1}^n \beta_{ki}^v \frac{I_{ki}^h}{\sum_{l=1}^n N_{li}^h + A_i} \\ \dot{I}_i^v &= S_i^v \sum_{k=1}^n \beta_{ki}^v \frac{I_{ki}^h}{\sum_{l=1}^n N_{li}^h + A_i} - \mu_i^v I_i^v. \end{aligned} \quad (2)$$

System (2) is a Bailey–Dietz model for the population registered in i , which is visiting patch j and with fast migration. The meaning of the remaining parameters is given in Table 1. Similar systems have been considered before for direct transmission diseases (Arino and van den Driessche, 2003; Adams and Kapan, 2009; Cosner et al., 2009).

The carrying capacity for the hosts in patch i is $F_i^h = \sum_{j=1}^n F_{ij}^h$.

Table 1

Parameters in System (2) and their respective meaning. All rates are per-day, while the remaining parameters are non-dimensional.

Parameters	Meaning
μ_i^h	Death rate for host in patch i
μ_i^v	Death rate for mosquitoes in patch i
F_{ij}^h	Carrying capacity associated to individuals registered in patch i which frequently visit patch j
F_i^v	Carrying capacity of mosquitoes in patch i
β_{ij}^h	Infectivity rates of host registered in i visiting j
A_i	Alternative blood sources in patch i
γ_i	Recovering rate of host in patch i
β_{ij}^v	Infectivity rates of vector in patch j interacting with host from vector i
M_{jk}^i	Migration rates matrix for host registered in patch i

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