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ON THE DYNAMICS OF A CLASS OF MULTI-GROUP MODELS FOR VECTOR-BORNE DISEASES

ABERRAHMAN IGGIDR, GAUTHIER SALLET, AND MAX O. SOUZA

ABSTRACT. The resurgence of vector-borne diseases is an increasing public health concern, and there is a need for a better understanding of their dynamics. For a number of diseases, e.g. dengue and chikungunya, this resurgence occurs mostly in urban environments, which are naturally very heterogeneous, particularly due to population circulation. In this scenario, there is an increasing interest in both multi-patch and multi-group models for such diseases. In this work, we study the dynamics of a vector borne disease within a class of multi-group models that extends the classical Bailey-Dietz model. This class includes many of the proposed models in the literature, and it can accommodate various functional forms of the infection force. For such models, the vector-host/host-vector contact network topology gives rise to a bipartite graph which has different properties from the ones usually found in directly transmitted diseases. Under the assumption that the contact network is strongly connected, we can define the basic reproductive number \mathcal{R}_0 and show that this system has only two equilibria: the so called disease free equilibrium (DFE); and a unique interior equilibrium—usually termed the endemic equilibrium (EE)—that exists if, and only if, $\mathcal{R}_0 > 1$. We also show that, if $\mathcal{R}_0 \leq 1$, then the DFE equilibrium is globally asymptotically stable, while when $\mathcal{R}_0 > 1$, we have that the EE is globally asymptotically stable.

1. INTRODUCTION

1.1. Background. The global resurgence of vector-borne diseases is a growing concern for public health officers in many countries [26]. Diseases like dengue and chikungunya continue to spread all over the world, hand in hand with the spread of their associated vectors; cf. [66]. Thus, in the United States the *Aedes albopictus*, the tiger mosquito, is fixating very rapidly, while in Europe *Ae. albopictus* is also spreading at a fast rate—cf. [53]. The result of this fixation is already evident: Italy and the South of France have already had documented cases of chikungunya [9], and there is a growing number of dengue cases detected in the US [4]. Furthermore, dengue is now the leading cause in US of acute febrile state of travelers returning from Asian, South American and Caribbean countries [10]. In the particular case of dengue, the main vector, *Ae. Aegypti*, is anthropophilic, and it lives only on urban or semi-urban areas. It is also a very sedentary mosquito: it will usually fly no more than about five hundred meters from its birth place, unless in extreme adverse conditions. These observations suggest that one should not expect that dengue will spread through the diffusion of the vector.

Indeed a number of such resurgent diseases occur in highly urban areas and are transmitted by vectors that do not disperse very far compared to other species—cf. [37] and references therein. On the other hand, in the case of a urban area with an efficient transportation system, movements from one location to another are fast. Then, for a given individual, disease transmission will mostly likely happen either at its home region or at its usual destination location. In this scenario, susceptible individuals can become infected in areas that are geographically apart from their residence area, and infected individuals can travel quite long distances and be able to infect vectors in very distinct areas were they themselves infected. Since the disease dynamics is likely to be largely dependent on whether one has a homogeneous or a heterogeneous population, with heterogeneity favoring the establishment of epidemics—cf. [17, 32, 72]—this suggests that in areas with significant population movement, the epidemiological dynamics can be strongly influenced by the circulation of human hosts. The link between host circulation and the disease dynamics seems to be first pointed out

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by [1, 12, 77] in slightly different frameworks. In any case, circulation naturally segregates host and vector by their registered and current location, and it is then natural to consider the so-called meta-population models as candidates for modeling the disease dynamics. Such meta-population models can be either of multi-patch or of multi-group type. In some regimes, the latter can arise as a limit model of the former—eg. in the case of fast sojourn times; cf. [1].

The previous discussion suggests that the use of multi-group models might become a valuable modeling tool for understanding the disease dynamics in urban settings, and indeed there is a growing interest in the literature on these models. See [73] for a recent review on such models, and for a discussion on their importance in the epidemiological modeling, and [62] for a study in a star network. In addition, see also [79] for empirical studies on the impact of human movement on the disease dynamics and [2] for complementary views to [1, 12]. For a theoretical review on multi-group models, see [82].

The overall interest in these epidemic models have, in turn, raised a natural interest in understanding their qualitative dynamical properties. This has fostered a considerable literature addressing this problem, and which we now briefly review.

1.2. Disease dynamics. From the point of view of epidemiological mathematical modeling, the first natural question about any disease-dynamics model is what are its stability features as a function of the basic reproduction number, \mathcal{R}_0 . Following [70], we say that an epidemic model has the *sharp \mathcal{R}_0 property* if the following holds: when $\mathcal{R}_0 \leq 1$, the only feasible equilibrium is the so-called disease free equilibrium (DFE), and it is globally asymptotically stable (GAS); when $\mathcal{R}_0 > 1$, there is a single interior equilibrium, the so-called endemic equilibrium (EE), which is then GAS.

The literature on mathematical epidemiology and the study of Sharp \mathcal{R}_0 property is long and large, particularly for directly transmitted diseases, but it is considerably smaller for vector-borne diseases. The development of the models for indirectly transmitted diseases can be traced back to Ross malaria model as discussed in [67]—see also the recent review in [71] and the classical monographs [6, 15]. Nevertheless, the bulk of the theory in the literature is leaned towards directly transmitted diseases and uniform populations—see [3, 13] for instance. For vector-borne diseases, a very natural model is the coupling of a SIR model for the humans with a SI model. This model is reasonable for mosquito borne diseases, since they do not have a well developed immunological system, while most of the arboviruses confer lifetime stability. This model seems to be first suggested in [6, 15] and it is now known as the Bailey-Dietz model. The global dynamics of this model was first studied in [18] using a Lyapunov function argument for the stability of the DFE, while the Poincaré-Bendixson property for 3-D competitive systems is used to show the stability of the EE; see also [8, 90] for later similar studies. A global stability analysis using only Lyapunov functions has been obtained only recently—[75]. See also [55, 80] for various results on global stability of epidemiological models.

In the framework of multi-group epidemic models for directly transmitted diseases, the first paper was probably by Rushton and Mauser [68], but seminal results are in Lajmanovich and Yorke [52] and in the book of Hethcote and Yorke [34]; but see also [65]. Stability results can be found in Thieme [33, 80]; see also chapter 23 of [82]. Global stability of multi-group SIR model is due to [27] by using a combinatorial argument arising from graph theory; see also [28] for a more extensive presentation of their method. For indirectly transmitted diseases, the first global stability result seems to be due to [32], who observed that a monotone dynamics argument of [52] was also applicable to a SI-SI multi-group model. More recently, general global stability results were obtained by [70]; see also [29] for results on multi-stage models. None of these results, however, cover the case of vector-borne diseases, since vector and host populations might follow different dynamics. Additional references in meta-population models for vector-borne diseases, but without studying the sharp \mathcal{R}_0 property are [5, 36] for models with heterogeneous populations and [89] for a numerical study of a multi-patch model with spatial heterogeneities.

For higher dimensional systems, global stability of endemic equilibrium is usually done by finding an appropriate Lyapunov function—[32] being a notable exception. The use of Lyapunov functions to study the global dynamics of ecological and epidemiological models can be traced at least to the works in the late seventies of Goh [21–24], Harrison [30, 31] and Hsu [38]. Since then, it has been successfully used in many studies, and even rediscovered [19, 20, 45–47, 49]. Recent applications of Lyapunov functions in epidemic and ecological models with meta-populations include [29, 40, 41, 43, 48, 51, 56, 57, 59, 69, 76, 78, 86, 91]. See also the recent surveys on the construction and use of Lyapunov functions in models of population dynamics

by [19, 39]. Additionally, there is also recent work aiming to obtain similar results for multi-group models, but without recurring to graph theoretic arguments [54, 63]. Shuai and van den Driessche [70] discuss two systematic approaches (graph-theoretic and matrix-theoretic) to guide the construction of Lyapunov functions. For results towards infinite dimensional problems, see [83].

In this work, we show that the sharp \mathcal{R}_0 property holds for a very natural multi-group extension of the Bailey-Dietz model—that has been used to model, *inter alia*, the dynamics of dengue [64]. This extension also accommodates a large number of choices for the modeling of the infection-force, including the most popular ones—see §2 for an additional discussion on this issue. A special case within the class of models discussed here was studied in [16] which, however, presents an incorrect proof of the global stability of the endemic equilibrium¹. This work can also be seen as an extension of the multi-group framework for direct-transmitted diseases in [27, 28].

1.3. Outline. In Section 2 we introduce the relevant class of multi-group models and identify the relevant network structure, which is a bipartite graph, that we term the host-vector network. This bipartite graph can be reducible, even when the group network is strongly connected. This is markedly different from directly transmitted diseases. On the assumption that the host-vector network is strongly connected, we can meaningfully define an \mathcal{R}_0 . For the models discussed here, the existence and uniqueness of the Endemic Equilibrium (EE), when, $\mathcal{R}_0 > 1$ is not obvious from the governing equations, and these issues are tackled in Section 3, where the local stability is also established. We then study the global dynamics in section 4: when $\mathcal{R}_0 \leq 1$, we show that the disease free equilibrium (DFE) is globally asymptotically stable. We then address the global stability of the EE and, we then show that it is globally asymptotically stable when $\mathcal{R}_0 > 1$ using a "vectorial" extension of the Lyapunov function used in [75] together with an extension of the graph-theoretical approach developed in [27, 28]. A discussion of the results is given in Section 5.

2. A CLASS OF MULTI-GROUP MODELS FOR VECTOR-BORNE DISEASES

In the following, we provide the basic set up for a class of multi-group models for indirectly transmitted diseases. These models are built upon the classical single-patch/group model by [6, 15], and include some of the models studied in [1, 12] and the models studied in [2].

2.1. The basic model. We consider the classical Bailey-Dietz model:

$$(1) \quad \begin{cases} \dot{S}_h = \Lambda_h - \beta_1 \frac{S_h I_v}{N_h} - \mu_h S_h \\ \dot{I}_h = \beta_1 \frac{S_h I_v}{N_h} - \gamma_h I_h - \mu_h I_h \\ \dot{R}_h = \gamma_h I_h - \mu_h R_h \\ \dot{S}_v = \Lambda_v - \beta_2 \frac{S_v I_h}{N_h} - \mu_v S_v \\ \dot{I}_v = \beta_2 \frac{S_v I_h}{N_h} - \mu_v I_v, \end{cases}$$

where S_h, I, R denote, as usual, the class of susceptible, infections and removed, respectively. The superscripts h and v indicate that the quantity refers to the host or to the vector. Also, $N_h = S_h + I_h + R_h$ and $N_v = S_v + I_v$ are the total host and vector, respectively, populations. Although they are not necessarily constant, they are taken as so in many applications.

The constant β_1 is a composite biological constant that embodies all the biological processes relating to transmission from mosquito to man, from the biting rate of the mosquitoes through the probability to develop and infection after a bite. Analogously β_2 captures the effect of transmission from man to mosquito. The constant μ_h is the per capita human mortality, γ_h denotes the per capita rates at which infectious individual recover and are permanently immune. The parameter Λ_v is the constant recruitment of mosquitoes and μ_v is the per capita vector mortality.

Let

$$\mathbf{N} = \frac{\Lambda_h}{\mu_h} \text{ and } \mathbf{V} = \frac{\Lambda_v}{\mu_v}.$$

¹The matrix whose kernel should yield the coefficients for Lyapunov function is actually not singular for $n > 2$. For $n = 2$, a careful checking shows that the claimed cancellation properties do not hold.

Using the techniques in [84], it is straightforward to see that the reproduction number of (1) is

$$\mathcal{R}_0^2 = \frac{\beta_1 \beta_2 \mathbf{V}}{\mu_v (\mu_h + \gamma_h) \mathbf{N}} = \frac{\beta_1 \beta_2 \mathbf{m}}{\mu_v (\mu_h + \gamma_h)}$$

with $\mathbf{m} = \frac{\mathbf{V}}{\mathbf{N}}$, the classical vectorial density. The basic reproduction ratio \mathcal{R}_0 is the same than for a classical Ross's model [3, 5, 6, 67].

As for Ross's model we will use the prevalences, i.e., defining $x_1 = \frac{S_h}{N}$, $x_2 = \frac{I_h}{N}$, $x_3 = \frac{R_h}{N}$ and $y_1 = \frac{S_v}{V}$, $y_2 = \frac{I_v}{V}$. Then, two equilibria are possible : the disease free equilibrium $(\mathbf{1}, \mathbf{0}, \mathbf{0}, \mathbf{1}, \mathbf{0})$ and, when $\mathcal{R}_0 > 1$, a positive endemic equilibrium $(\bar{x}_1, \bar{x}_2, \bar{x}_3, \bar{y}_1, \bar{y}_2)$.

The global stability of (1) was originally studied by [18], who showed that the endemic equilibrium is globally asymptotically stable when $\mathcal{R}_0 > 1$, and that the disease-free is the global attractor when $\mathcal{R}_0 \leq 1$. using the so-called Poincaré-Bendixson theorem for competitive systems—cf. [74]. More recently, [75] has obtained a proof using only Lyapunov functions

2.2. A class of multi-group models for vector-borne diseases. We consider that both host and vector populations are divided in n groups, where each group i has a host population of $N_{h,i}$ and a vector population of $N_{v,i}$. At each node i , we assume a generalized form of (1) by allowing that the susceptible of group i to have contact of mosquitoes of group $j = 1, \dots, n$. This is specified by an infection term for the host \mathcal{T}_h , of the form

$$\mathcal{T}_{h,i} = S_{h,i} \sum_{j=1}^n L_{i,j}(N_h, N_v) I_{v,j}.$$

Analogously, we allow susceptible mosquitoes of each group i to have contact with infected hosts group $j = 1, \dots, n$, with an infection force for the vectors, \mathcal{T}_v , of the form:

$$\mathcal{T}_{v,i} = S_{v,i} \sum_{j=1}^n M_{i,j}(N_h, N_v) I_{h,j}.$$

These assumptions then lead to the following multi-group epidemic model:

$$(2) \quad \begin{cases} \dot{S}_{h,i} = \Lambda_{h,i} - S_{h,i} \sum_{j=1}^n L_{i,j}(N_h, N_v) I_{v,j} - \mu_{h,i} S_{h,i} \\ \dot{I}_{h,i} = S_{h,i} \sum_{j=1}^n L_{i,j}(N_h, N_v) I_{v,j} - \gamma_{h,i} I_{h,i} - \mu_{h,i} I_{h,i} \\ \dot{R}_{h,i} = \gamma_{h,i} I_{h,i} - \mu_{h,i} R_{h,i} \\ \dot{S}_{v,i} = \Lambda_{v,i} - S_{v,i} \sum_{j=1}^n M_{i,j}(N_h, N_v) I_{h,j} - \mu_{v,i} S_{v,i} \\ \dot{I}_{v,i} = S_{v,i} \sum_{j=1}^n M_{i,j}(N_h, N_v) I_{h,j} - \mu_{v,i} I_{v,i}, \end{cases}$$

where

$$N_h = (N_{h,i}), \text{ with } N_{h,i} = S_{h,i} + I_{h,i} + R_{h,i} \text{ and } N_v = (N_{v,i}), \text{ with } N_{v,i} = S_{v,i} + I_{v,i}.$$

The functions $L_{i,j}, M_{i,j} : \mathbb{R}^n \oplus \mathbb{R}^n \rightarrow \mathbb{R}$ are assumed to be smooth and positive when N_h, N_v have positive entries. These are mild assumptions, and they can accommodate a variety of functional forms for the infections force—see [88] for a discussion on the different conclusions implied by different assumptions on the infection force; see also [2] for a discussion on the different transmission force related to dengue. These functions also encode the cross-infection information among all the groups, which will depend on the modeling assumptions that led to the multi-group structure.

Remark 2.1. *Similar models have been considered in the literature. See [12] for a multi-group SIS-SI model and [1] for a multi-group SEIR-SEI model, obtained as the fast sojourn limit of a more general model.*

Remark 2.2. *While model (2) can be easily modified to include disease induced death, the analysis carried out in the sequel cannot be extended to such models, except in the case of constant population. However, for diseases as dengue or chikungunya, this is not a very restricting assumption, as their morbidity is, generally, not high. Dengue can be an exception to that, if there are two epidemics in a row with an intermediate*

time spacing. In this case, enhanced immunological reaction can cause the so-called severe dengue fever, previously known as haemorrhagic dengue, which can be highly fatal if not treated appropriately [25, 87].

We can rewrite (2) as

$$(3) \quad \begin{cases} \dot{N}_{h,i} = \Lambda_{h,i} - \mu_{h,i} N_{h,i} \\ \dot{N}_{v,i} = \Lambda_{v,i} - \mu_{v,i} N_{v,i} \\ \dot{S}_{h,i} = \Lambda_{h,i} - S_{h,i} \sum_{j=1}^n L_{i,j}(N_h, N_v) I_{v,j} - \mu_{h,i} S_{h,i} \\ \dot{I}_{h,i} = S_{h,i} \sum_{j=1}^n L_{i,j}(N_h, N_v) I_{v,j} - \gamma_h I_{h,i} - \mu_{h,i} I_{h,i} \\ \dot{I}_{v,i} = (N_{v,i} - I_{v,i}) \sum_{j=1}^n M_{i,j}(N_h, N_v) I_{h,j} - \mu_{v,i} I_{v,i}. \end{cases}$$

In what follows, we write $S_h = (S_{h,i})$, $i = 1, \dots, n$ and similarly for I_h and I_v . Also, let

$$\bar{N}_h = \left(\frac{\Lambda_{h,i}}{\mu_{h,i}} \right) \text{ and } \bar{N}_v = \left(\frac{\Lambda_{v,i}}{\mu_{v,i}} \right).$$

Then, it is clear that, for (3), the set

$$\Omega = \{(S_h, I_h, I_v, N_h, N_v) \in \mathbb{R}_+^{5n} \mid 0 \leq S_h + I_h \leq \bar{N}_h, 0 \leq I_v \leq \bar{N}_v, 0 \leq N_h \leq \bar{N}_h, 0 \leq N_v \leq \bar{N}_v\}$$

is a compact absorbing and positively invariant set.

Also, notice that the system (3) is of triangular form, and hence its stability analysis can be considerably simplified. There are a number of results that allow for such a simplification in the study of global stability of systems of this kind [81, 85]. For the convenience of the reader, we recall the following result:

Theorem 2.1 (Vidyasagar [85], Theorem 3.1). :

Consider the following \mathcal{C}^1 system :

$$(4) \quad \begin{cases} \dot{x} = f(x) \\ \dot{y} = g(x, y) \\ \text{with an equilibrium point, } (x^*, y^*), \text{ i.e.,} \\ f(x^*) = 0 \text{ and } g(x^*, y^*) = 0. \end{cases} \quad x \in \mathbb{R}^n, y \in \mathbb{R}^m$$

If x^* is globally asymptotically stable (GAS) in \mathbb{R}^n for the system $\dot{x} = f(x)$, and if y^* is GAS in \mathbb{R}^m , for the system $\dot{y} = g(x^*, y)$, then (x^*, y^*) is (locally) asymptotically stable for (4). Moreover, if all the trajectories of (4) are forward bounded, then (x^*, y^*) is GAS for (4).

Since (\bar{N}_h, \bar{N}_v) is a globally asymptotically stable equilibrium for the first two equations of (3), we can use Theorem 2.1 to reduce the study of the stability properties of (3) to the study of the stability of

$$(5) \quad \begin{cases} \dot{S}_{h,i} = \Lambda_{h,i} - S_{h,i} \sum_{j=1}^n L_{i,j}(\bar{N}_h, \bar{N}_v) I_{v,j} - \mu_{h,i} S_{h,i} \\ \dot{I}_{h,i} = S_{h,i} \sum_{j=1}^n L_{i,j}(\bar{N}_h, \bar{N}_v) I_{v,j} - \gamma_{h,i} I_{h,i} - \mu_{h,i} I_{h,i} \\ \dot{I}_{v,i} = (N_{v,i} - I_{v,i}) \sum_{j=1}^n M_{i,j}(\bar{N}_h, \bar{N}_v) I_{h,j} - \mu_{v,i} I_{v,i}. \end{cases}$$

In what follows, we shall denote by Λ_h , μ_h and γ_h the vectors of \mathbb{R}_+^n whose components are respectively $\Lambda_{h,i}$, $\mu_{h,i}$ and $\gamma_{h,i}$. We shall also write $M = M(\bar{N}_h, \bar{N}_v)$ and $L = L(\bar{N}_h, \bar{N}_v)$. System (5) can then be written in

the following vectorial notation:

$$(6) \quad \begin{cases} \dot{S}_h = \Lambda_h - \text{diag}(S_h) L I_v - \text{diag}(\mu_h) S_h \\ \dot{I}_h = \text{diag}(S_h) L I_v - \text{diag}(\mu_h + \gamma_h) I_h \\ \dot{I}_v = \text{diag}(\bar{N}_v - I_v) M I_h - \text{diag}(\mu_v) I_v, \end{cases}$$

where for $\mathbf{v} \in \mathbb{R}^n$, $\text{diag}(\mathbf{v})$ denotes the $n \times n$ diagonal matrix whose main diagonal is \mathbf{v} .

2.3. The Host-Vector contact network. We shall need an assumption about the network topology in system (6). For a matrix A , we write $\Gamma(A)$ for the associated graph. We begin with a definition

Definition 2.1 (Host-Vector Contact Network). *Given nonnegative matrices L and M , we write*

$$\mathcal{M} = \begin{pmatrix} 0 & L \\ M & 0 \end{pmatrix}.$$

The graph associated to \mathcal{M} , $\Gamma(\mathcal{M})$, is denoted the host-vector contact network, or contact network for short.

Hypothesis 2.1. *The contact network is strongly connected, i.e., \mathcal{M} is nonnegative and irreducible.*

Remark 2.3. *Notice that irreducibility of L and M are neither necessary nor sufficient for the irreducibility of \mathcal{M} . As an example, consider*

$$C = \begin{pmatrix} 0 & 1 \\ 1 & 0 \end{pmatrix} \text{ and } D = \begin{pmatrix} 1 & 0 \\ 1 & 1 \end{pmatrix}; \quad \mathcal{M}_1 = \begin{pmatrix} 0 & C \\ C & 0 \end{pmatrix} \text{ and } \mathcal{M}_2 = \begin{pmatrix} 0 & D^t \\ D & 0 \end{pmatrix}.$$

Then C is irreducible and D is reducible. Nevertheless, \mathcal{M}_1 is reducible and \mathcal{M}_2 is irreducible.

The irreducibility of \mathcal{M} is associated to the strong connectivity of the corresponding directed bipartite graph. This is a consequence of the infection process, when considered between hosts (or vectors) themselves, is a two step process. Thus, even when the circulation structure (the non-zero patterns of L and M) is strongly connected, this is not necessarily the case for the host-vector contact structure of an indirectly transmitted disease, and this is a significant difference to directly transmitted ones.

In the following Proposition we shall give a useful characterization of the irreducibility of \mathcal{M} that will be used later on:

Proposition 2.1. *\mathcal{M} is irreducible if, and only if, the following conditions are satisfied:*

- (1) *Both LM and ML are irreducible;*
- (2) *We have that $Lv, Mv \gg 0$, for some $v \gg 0$ (and hence, for every $v \gg 0$).*

Moreover, in this case, we also have that

$$\rho(\mathcal{M})^2 = \rho(LM) = \rho(ML),$$

and that both LM and ML have right and left positive eigenvectors associated to $\rho(\mathcal{M})^2$.

Proof. Firstly, we compute

$$\mathcal{M}^{2k} = \begin{pmatrix} (LM)^k & 0 \\ 0 & (ML)^k \end{pmatrix} \quad \text{and} \quad \mathcal{M}^{2k+1} = \begin{pmatrix} 0 & L(ML)^k \\ M(LM)^k & 0 \end{pmatrix},$$

Assume \mathcal{M} is irreducible. Then there is some natural n such that

$$(I + \mathcal{M})^{2n} = \sum_{m=0}^{2n} \binom{2n}{m} \mathcal{M}^m = \begin{pmatrix} \sum_{k=0}^n \binom{2n}{2k} (LM)^k & L \sum_{k=0}^{n-1} \binom{2n}{2k+1} (ML)^k \\ M \sum_{k=0}^{n-1} \binom{2n}{2k+1} (LM)^k & \sum_{k=0}^n \binom{2n}{2k} (ML)^k \end{pmatrix} \gg 0.$$

Hence, we have that

$$(I + ML)^n, (I + LM)^n \gg 0,$$

and both LM and ML are irreducible as claimed. In addition, we have $L \sum_{k=0}^{n-1} \binom{2n}{2k+1} (ML)^k \gg 0$. Thus L applied to a column of $\sum_{k=0}^{n-1} \binom{2n}{2k+1} (ML)^k$ is positive. The argument for M is similar.

Conversely, if both LM and ML are irreducible, then we have that the main diagonals of $(I + \mathcal{M})^{2n}$ are positive. The remaining blocks are also positive, since L and M are acting on positive matrices.

Finally, let (u, v) be a positive eigenvector associated to $\rho(\mathcal{M}) = \rho_{\mathcal{M}}$. Then we necessarily have $Lu = \rho_{\mathcal{M}}v$ and $Mv = \rho_{\mathcal{M}}u$. Hence $LMv = \rho_{\mathcal{M}}^2v$, and similarly $MLu = \rho_{\mathcal{M}}^2u$. Furthermore u and v are positive right eigenvectors of ML and LM , respectively, associated to $\rho_{\mathcal{M}}^2$. The argument for left eigenvectors is analogous. \square

Remark 2.4. We observe that System (6) can be recast as a special case of the multigroup SIR model treated in [27], as follows: Replace $N_v - I_v$ by S_v in the last equation of (6). Include the redundant equation:

$$\dot{S}_v = \Lambda_v - \text{diag}(S_v) M I_h - \text{diag}(\mu_v) S_v.$$

Let $\mathbf{S} = (S_1, \dots, S_{2n})^t$, $\mathbf{I} = (I_1, \dots, I_{2n})^t$ and set $S_i = S_{h,i}$, $S_{n+i} = S_{v,i}$, $I_i = I_{h,i}$, $I_{n+i} = I_{v,i}$, for $i = 1, \dots, n$. Further, let \mathcal{M} be as given in definition 2.1 and let $\Lambda = (\Lambda_h \ \Lambda_v)^t$, $\mu = (\mu_h \ \mu_v)^t$, and $\gamma = (\gamma_h \ \mathbf{0})^t$. Then $(\mathbf{S} \ \mathbf{I})^t$ satisfies

$$\dot{\mathbf{S}} = \Lambda - \text{diag}(\mathbf{S}) \mathcal{M} \mathbf{I} - \text{diag}(\mu) \mathbf{S}$$

$$\dot{\mathbf{I}} = \text{diag}(\mathbf{S}) \mathcal{M} \mathbf{I} - \text{diag}(\mu + \gamma) \mathbf{I},$$

for which the sharp threshold property holds. Nevertheless, we shall obtain this result by considering equation (6) directly, and using a related but different approach. This can be seen as an extension to indirect transmitted diseases of the framework introduced in [27, 28].

3. EQUILIBRIA AND LOCAL STABILITY

We will show that for our vectorial disease with sub-populations structure, System (2), the results of [33, 82] are conserved. Namely we obtain that the DFE is locally asymptotically stable, iff $\mathcal{R}_0 \leq 1$, and the existence and uniqueness of a strongly endemic equilibrium when $\mathcal{R}_0 > 1$. This equilibrium is always locally asymptotically stable. For global results, see Section 4.

Using the now standard techniques [14, 84], we define the basic reproduction ratio as

$$\mathcal{R}_0 = \rho(\mathcal{N}), \quad \mathcal{N} = \begin{pmatrix} 0 & \text{diag}(\mu_h + \gamma_h)^{-1} \text{diag}(\bar{N}_h) L \\ \text{diag}(\mu_v)^{-1} \text{diag}(\bar{N}_v) M & 0 \end{pmatrix}.$$

Remark 3.1. Since $\mu_v, \mu_h \gg 0$, we have from Proposition 2.1 that \mathcal{N} is irreducible if, and only if, \mathcal{M} is irreducible. In particular, if Hypothesis 2.1 holds then \mathcal{N} is irreducible and we have that

$$\mathcal{R}_0^2 = \rho \left(\text{diag}(\mu_h + \gamma_h)^{-1} \text{diag}(\mu_v)^{-1} \text{diag}(\bar{N}_h) L \text{diag}(\bar{N}_v) M \right).$$

Theorem 3.1. Assume that hypothesis 2.1 holds. Then system (6) (and hence system (2)) has a unique endemic equilibrium if, and only if, $\mathcal{R}_0 > 1$. Moreover this equilibrium is locally asymptotically stable with respect to System (6).

Proof. We denote by S_h^* , I_h^* and I_v^* the expression of an endemic equilibrium. Recall that the notation $\mathbf{1}$ refers to the vector of \mathbb{R}_+^n whose components are all equal to 1. We have the following relation, defining an endemic equilibrium:

$$(7a) \quad \Lambda_h = \text{diag}(\mu_h + L I_v^*) S_h^*$$

$$(7b) \quad \text{diag}(\mu_h + \gamma_h) I_h^* = \text{diag}(S_h^*) L I_v^*$$

$$(7c) \quad \text{diag}(\mu_v) I_v^* = \text{diag}(\bar{N}_v - I_v^*) M I_h^*$$

From (7a) we obtain

$$S_h^* = \text{diag}(\mu_h + L I_v^*)^{-1} \Lambda_h$$

Rewriting (7c) as

$$\text{diag}(\mu_v) I_v^* = \text{diag}(M I_h^*) (\bar{N}_v - I_v^*)$$

Substituting for S_h^* in (7b) we obtain

$$(8a) \quad I_h^* = \text{diag}(\mu_h + \gamma_h)^{-1} \text{diag}(\mu_h + L I_v^*)^{-1} \text{diag}(L I_v^*) \Lambda_h$$

$$(8b) \quad I_v^* = \text{diag}(\mu_v + M I_h^*)^{-1} \text{diag}(M I_h^*) \bar{N}_v$$

Hence (I_h^*, I_v^*) is a fixed point of the following application

$$F(x, y) = \begin{bmatrix} \text{diag}(\mu_h + \gamma_h)^{-1} \text{diag}(\mu_h + L y)^{-1} \text{diag}(L y) \Lambda_h \\ \text{diag}(\mu_v + M x)^{-1} \text{diag}(M x) \bar{N}_v \end{bmatrix}$$

We will use a result of Hethcote and Thieme [33], which we recall for the convenience of the reader:

Lemma 3.1 (Theorem 2.1 in [33]). *Let $F(w)$ be a continuous, monotone non-decreasing, strictly sublinear, bounded function which maps the nonnegative orthant $\mathbb{R}_+^n = [0, \infty)^n$ into itself. Let $F(0) = 0$ and $F'(0)$ exist and be irreducible. Then $F(w)$ does not have a nontrivial fixed point on the boundary of \mathbb{R}_+^n . Moreover, $F(x)$ has a positive fixed point iff $\rho(F'(0)) > 1$. If there is a fixed point, then it is unique.*

We have to check, for our function F defined on $\mathbb{R}_+^n \times \mathbb{R}_+^n$, the conditions of Theorem 3.1.

It is immediate that F is continuous, bounded and maps the nonnegative orthant $\mathbb{R}_+^n \times \mathbb{R}_+^n$ into itself.

The function F is monotone since the Jacobian of F is

$$JF(x, y) = \begin{bmatrix} 0 & A_1 \\ A_2 & 0 \end{bmatrix}$$

With

$$A_1 = \text{diag}(\mu_h + \gamma_h)^{-1} \text{diag}(\mu_h + L y)^{-1} \text{diag}(\Lambda_h) [I_n - \text{diag}(\mu_h + L y)^{-1} \text{diag}(L y)] L.$$

and

$$A_2 = \text{diag}(\bar{N}_v) \text{diag}(\mu_v + M x)^{-1} [I_n - \text{diag}(\mu_v + M x)^{-1} \text{diag}(M x)] M.$$

Then $JF(x, y)$ is a Metzler matrix, i.e. a matrix whose off diagonal terms are nonnegative [42, 58]. These matrices are also known as quasi-positive matrix [74, 82]. This proves that F is monotone [35, 74]. Now, we have to check the strict sublinearity. We use the equivalent definition of [35], using the standard ordering of \mathbb{R}^n and the classical notations $x \leq y$ if, for any index i , $x_i \leq y_i$; $x < y$ if $x \leq y$ and $x \neq y$; $x \ll y$ if $x_i < y_i$ for any index i ;

F is strongly sublinear if

$$0 < \lambda < 1, \quad w \gg 0 \implies \lambda F(w) \gg F(\lambda w).$$

With $x \gg 0$ and $y \gg 0$, since \mathcal{M} is irreducible, we must have $\mathcal{M} \begin{pmatrix} x \\ y \end{pmatrix} \gg 0$, and hence we have $L y \gg 0$ and $M x \gg 0$. Thus, $\mu_h + \lambda L y \ll \mu_h + L y$ and a similar inequality $\mu_v + \lambda M x \ll \mu_v + M x$. This proves the strict sublinearity. Using the formula for the Jacobian of F , we have

$$JF(0, 0) = \begin{bmatrix} 0 & \text{diag}(\mu_h + \gamma_h)^{-1} \text{diag}(\bar{N}_h) L \\ \text{diag}(\mu_v)^{-1} \text{diag}(\bar{N}_v) M & 0 \end{bmatrix}$$

This matrix is irreducible, since \mathcal{M} is irreducible, and $\rho(JF(0, 0)) = \mathcal{R}_0$. All the requirements of Theorem 3.1 are satisfied. This proves that there exists a unique positive endemic equilibrium in \mathbb{R}_+^n when $\mathcal{R}_0 > 1$. Moreover, looking at the expression of F , it is clear that this equilibrium is in the compact Ω .

We will prove the asymptotic stability of this positive equilibrium. The proof is adapted from [33], using Krasnosel'skiĭ's trick [50]. The difference is that we have to vectorize this proof for the infective of human host and vectors. We will show that the linearized equation has no solution of the form $X(t) = \exp(z t) X_0$ with $X_0 \in \mathbb{C}^{3n}$, $z \in \mathbb{C}$, $\Re z \geq 0$ for X_0 eigenvector and z corresponding eigenvalue of the Jacobian computed at the endemic equilibrium. Let $X_0 = (U, V, W) \in \mathbb{C}^{3n}$ be such an eigenvector for the eigenvalue z . Then

$$(9a) \quad z U = -\text{diag}(\mu_h) U - \text{diag}(L I_v^*) U - \text{diag}(S_h^*) L W$$

$$(9b) \quad z V = \text{diag}(L I_v^*) U - (\mu_h + \gamma_h) V + \text{diag}(S_h^*) L W$$

$$(9c) \quad z W = \text{diag}(\bar{N}_v - I_v^*) M V - \mu_V W - \text{diag}(M I_h^*) W$$

Adding the sub-equations (9a) and (9b) we obtain the relation

$$\text{diag}(\mu_h + z \mathbf{1}) U = -\text{diag}(\mu_h + \gamma_h + z \mathbf{1}) V$$

Replacing U in (9b) and (9c) yields after some rearrangements

$$(10) \quad \begin{bmatrix} \text{diag}(\mathbf{1} + z \text{diag}(\mu_h + \gamma_h)^{-1} \mathbf{1} + \text{diag}(z \mathbf{1} + \mu_h + \gamma_h) \text{diag}(z \mathbf{1} + \mu_h)^{-1} \text{diag}(\mu_h + \gamma_h)^{-1} L I_v^*) V \\ \text{diag}(\mathbf{1} + z \text{diag}(\mu_v)^{-1} \mathbf{1} + \text{diag}(\mu_v)^{-1} M I_h^*) W \end{bmatrix} = \begin{bmatrix} 0 & \text{diag}(\mu_h + \gamma_h)^{-1} \text{diag}(S_h^*) L \\ \text{diag}(\mu_v)^{-1} \text{diag}(\bar{N}_v - I_v^*) M & 0 \end{bmatrix} \begin{bmatrix} V \\ W \end{bmatrix}$$

The matrix

$$H = \begin{bmatrix} 0 & \text{diag}(\mu_h + \gamma_h)^{-1} \text{diag}(S_h^*) L \\ \text{diag}(\mu_v)^{-1} \text{diag}(\bar{N}_v - I_v^*) M & 0 \end{bmatrix}$$

is a nonnegative irreducible matrix, since its associated graph is isomorphic to $\Gamma(\mathcal{M})$. From equations (7b) and (7c), we have that

$$H \begin{bmatrix} I_h^* \\ I_v^* \end{bmatrix} = \begin{bmatrix} I_h^* \\ I_v^* \end{bmatrix}.$$

Note that $\begin{bmatrix} I_h^* \\ I_v^* \end{bmatrix}$ is the positive Perron-Frobenius vector of H .

We assume that $\Re z \geq 0$. Let $\eta(z)$ be the minimum of the real part of the components of the two vectors

$$z \text{diag}(\mu_h + \gamma_h)^{-1} \mathbf{1} + \text{diag}(z \mathbf{1} + \mu_h + \gamma_h) \text{diag}(z \mathbf{1} + \mu_h)^{-1} \text{diag}(\mu_h + \gamma_h)^{-1} L I_v^*$$

and

$$z \text{diag}(\mu_v)^{-1} \mathbf{1} + \text{diag}(\mu_v)^{-1} M I_h^*$$

Since $\Re z \geq 0$, $I_v^* \gg 0$, $I_h^* \gg 0$, the irreducibility of \mathcal{M} implies that we have $\eta(z) > 0$. Taking the absolute values in (10) gives

$$[1 + \eta(z)] \begin{bmatrix} |V| \\ |W| \end{bmatrix} \leq H \begin{bmatrix} |V| \\ |W| \end{bmatrix}$$

Let r the minimum number such that

$$\begin{bmatrix} |V| \\ |W| \end{bmatrix} \leq r \begin{bmatrix} I_h^* \\ I_v^* \end{bmatrix}.$$

We now have

$$[1 + \eta(z)] \begin{bmatrix} |V| \\ |W| \end{bmatrix} \leq H \begin{bmatrix} |V| \\ |W| \end{bmatrix} \leq r H \begin{bmatrix} I_h^* \\ I_v^* \end{bmatrix} = r \begin{bmatrix} I_h^* \\ I_v^* \end{bmatrix}.$$

Since $\eta(z) > 0$ if $\Re z \geq 0$, we obtain a contradiction to the minimality of r . Thus $\Re z < 0$, which proves the asymptotic stability at the endemic equilibrium. \square

4. GLOBAL DYNAMICS

In this section, we discuss a number of results concerning the global dynamics of system (6). We begin by introducing some notation to allow an easier handling of the vector calculations.

Definition 4.1. *The entry-wise product for vectors, the Hadamard product, will be denoted by \circ . Namely, if $(X_1, \dots, X_n), (Y_1, \dots, Y_n) \in \mathbb{R}^n$, then*

$$(X_1, \dots, X_n) \circ (Y_1, \dots, Y_n) = (X_1 Y_1, \dots, X_n Y_n).$$

For a vector $\mathbf{X} = (X_1, \dots, X_n) \in \mathbb{R}^n$ and for $f : I \subset \mathbb{R} \rightarrow \mathbb{R}$, we shall write

$$f(\mathbf{X}) = (f(X_1), \dots, f(X_n)).$$

In particular, if $X = (X_1, \dots, X_n) \gg 0$, then $X^{-1} = (X_1^{-1}, \dots, X_n^{-1})$.

We collect some useful facts about the manipulation of expression involving Hadamard products in the following Lemma:

Lemma 4.1. *If $\mathbf{X}_1, \dots, \mathbf{X}_m \in \mathbb{R}^n$ and $M \in M_n(\mathbb{R})$ then we have*

- (1) $\mathbf{X}_1 + \dots + \mathbf{X}_m \geq m \sqrt[m]{\mathbf{X}_1 \circ \dots \circ \mathbf{X}_m}$;
- (2) $\mathbf{X}_1 \circ (M\mathbf{X}_2) = \text{diag}(\mathbf{X}_1)M\mathbf{X}_2 = \text{diag}(M\mathbf{X}_2)\mathbf{X}_1$;
- (3) *if $\mathbf{X}_1 = \mathbf{X}_1(t)$, and if f is differentiable then $\frac{d}{dt}f(\mathbf{X}_1) = \dot{\mathbf{X}}_1 \circ f'(\mathbf{X}_1)$.*

It turns out that it is more convenient to work with system (6) in prevalence form, so that the susceptible population at the disease-free equilibrium (DFE), for both host and vector populations in each group, is unity. Let

$$D_h = \text{diag}(\bar{N}_h), \quad D_v = \text{diag}(\bar{N}_v),$$

$$(X, Y) = D_h^{-1}(S_h, I_h), \quad Z = D_v^{-1}I_v$$

$$A = LD_v \text{ and } B = MD_h.$$

introduce

In this case system (6) reads

$$(11) \quad \begin{cases} \dot{X} &= \mu_h \circ (\mathbf{1} - X) - \text{diag}(X)AZ \\ \dot{Y} &= \text{diag}(X)AZ - (\mu_h + \gamma_h) \circ Y \\ \dot{Z} &= \text{diag}(\mathbf{1} - Z)BY - \mu_v \circ Z \end{cases}$$

I suggest to use $X^*, Y^*, \mathcal{N}^*, \dots$ instead of $\bar{X}, \bar{Y}, \bar{\mathcal{N}}, \dots$ for all what is related to the endemic equilibrium since bar has already been used and we used stars for the EE in the previous section

With this notation, the DFE is $(\mathbf{1}, 0, 0)$ and we shall write the EE as $(\bar{X}, \bar{Y}, \bar{Z})$, with

$$\bar{X}_i = \left(\frac{X_i^*}{\bar{N}_{h,i}} \right), \quad \bar{Y}_i = \left(\frac{Y_i^*}{\bar{N}_{h,i}} \right) \quad \text{and} \quad \bar{Z}_i = \left(\frac{Z_i^*}{\bar{N}_{v,i}} \right).$$

Notice that, since in the new coordinates we have $\text{diag}(\bar{N}_h) = \text{diag}(\bar{N}_v) = \text{diag}(\mathbf{1})$, the next generation operator is now given by

$$\mathcal{N} = \begin{pmatrix} 0 & \text{diag}(\mu_h + \gamma_h)^{-1}A \\ \text{diag}(\mu_v)^{-1}B & 0 \end{pmatrix}.$$

Also, the absorbing set can now be written as

$$K = \{(X, Y, Z) \in \mathbb{R}^{3n} \text{ s.t. } 0 \leq X + Y \leq \mathbf{1}, \quad 0 \leq Z \leq \mathbf{1}\}.$$

We begin with the stability of the DFE when $\mathcal{R}_0 \leq 1$:

Theorem 4.1. *Assume that hypothesis 2.1 holds and that $\mathcal{R}_0 \leq 1$. Then the DFE is globally asymptotically stable. If $\mathcal{R}_0 > 1$, then the DFE is unstable.*

Proof. Since \mathcal{N} is irreducible, let (α, β) be a left, positive eigenvector of \mathcal{N} , associated to the eigenvalue \mathcal{R}_0 . Let

$$V = \langle \alpha, Y \rangle + \langle \beta, (\mu_h + \gamma_h) \circ \mu_v^{-1} \circ Z \rangle \quad \text{and} \quad R = \langle \alpha, \text{diag}(\mathbf{1} - X)AZ \rangle + \langle \beta, (\mu_h + \gamma_h) \circ \mu_v^{-1} \circ \text{diag}(Z)BY \rangle.$$

Notice that $R \geq 0$, and that R vanishes in the set

$$S_0 = \{(X, Y, Z) \in K : \text{diag}(\mathbf{1} - X)AZ = \text{diag}(Z)BY = 0, Y, Z \neq 0\}.$$

Computing the derivative of V along the flow, we have:

$$\begin{aligned} \dot{V} &= \langle \alpha, \dot{Y} \rangle + \langle \beta, (\mu_h + \gamma_h) \circ \mu_v^{-1} \circ \dot{Z} \rangle \\ &= \langle \alpha, \text{diag}(X)AZ - (\mu_h + \gamma_h) \circ Y \rangle + \langle \beta, (\mu_h + \gamma_h) \circ \mu_v^{-1} \circ (\text{diag}(\mathbf{1} - Z)BY - \mu_v \circ Z) \rangle \\ &= \langle \alpha, AZ - (\mu_h + \gamma_h) \circ Y \rangle + \langle \beta, (\mu_h + \gamma_h) \circ \mu_v^{-1} \circ (BY - \mu_v Z) \rangle - R \\ &= [\mathcal{R}_0 \langle (\mu_h + \gamma_h) \circ \beta, Z \rangle - \langle (\mu_h + \gamma_h) \circ \alpha, Y \rangle + \mathcal{R}_0 \langle (\mu_h + \gamma_h) \circ \alpha, Y \rangle - \langle (\mu_h + \gamma_h) \circ \beta, Z \rangle] - R \\ &= (\mathcal{R}_0 - 1) [\langle (\mu_h + \gamma_h) \circ \alpha, Y \rangle + \langle (\mu_h + \gamma_h) \circ \beta, Z \rangle] - R \\ &\leq 0, \end{aligned}$$

provided that $\mathcal{R}_0 \leq 1$.

Also, notice that when $\mathcal{R}_0 < 1$, we have that $\dot{V} = 0$ if, and only if, $Y = Z = 0$. Since the DFE is the unique invariant compact set in this latter case, LaSalle principle implies that it is globally asymptotically stable. If $\mathcal{R}_0 = 1$ then we observe that $\dot{V} = 0$ holds in S_0 , which contains the set $\{(X, Y, Z) | Y = Z = 0\}$. Nevertheless, it can then be easily verified from system (11) that the DFE is the only invariant set contained in S_0 . Thus the result follows once again from LaSalle invariance principle.

If $\mathcal{R}_0 > 1$, then if both Y and Z are sufficient close to zero, we have $\dot{V}(\mathbf{1}, Y, Z) > 0$. By continuity, this is also true in a neighbourhood of $(\mathbf{1}, 0, 0)$, and hence the DFE is unstable. \square

Before we can tackle the global stability of the endemic equilibrium, when $\mathcal{R}_0 > 1$, we need some preliminary results.

Lemma 4.2. *Assume that Hypothesis 2.1 holds, and let*

$$\bar{\mathcal{N}} = \begin{pmatrix} 0 & \text{diag}(\mu_h + \gamma_h)^{-1} \text{diag}(\bar{X})A \\ \text{diag}(\mu_v)^{-1} \text{diag}(\mathbf{1} - \bar{Z})B & 0 \end{pmatrix}.$$

Then, $\bar{\mathcal{N}}$ is irreducible, $\rho(\bar{\mathcal{N}}) = 1$ and $\bar{\mathcal{N}}$ has a positive left eigenvector $(\xi, \eta)^t$ to $\rho(\bar{\mathcal{N}})$. In addition, let

$$T = \text{diag}(\mu_v)^{-1} \text{diag}(\mu_h + \gamma_h)^{-1} \text{diag}(\bar{X})A \text{diag}(\mathbf{1} - \bar{Z})B.$$

Then $\rho(T) = 1$, and $T^t \eta = \eta$.

Proof. Since Hypothesis 2.1 holds, we have that \mathcal{N} is irreducible, and hence $\bar{\mathcal{N}}$ is irreducible. From the equilibrium relationship we also have

$$\bar{\mathcal{N}} \begin{pmatrix} \bar{Y} \\ \bar{Z} \end{pmatrix} = \begin{pmatrix} \bar{Y} \\ \bar{Z} \end{pmatrix},$$

and hence we have

$$\rho(\bar{\mathcal{N}}) = 1.$$

The remaining claims follow from Proposition 2.1. \square

Before giving the next definition, we introduce some terminology. For a given digraph G , we will denote its set of vertices by $\mathcal{V}(G)$, and the set of edges of G by $\mathcal{E}(G) \subset \mathcal{V}(G) \times \mathcal{V}(G)$. A c -edge colored multidigraph (c -ECM for short) is a multi-digraph where the parallel edges must have different colors—and therefore a maximum of c parallel edges are allowed. If G is a c -ECM, we will write $\mathcal{C}(G)$ for its set of colors. Thus each edge of G can be uniquely described as an ordered triple $(v_1, v_2, c) \in \mathcal{E}(G) \subset \mathcal{V}(G) \times \mathcal{V}(G) \times \mathcal{C}(G)$.

Definition 4.2 (Transitive Contact Multigraph). *Given a contact network $\Gamma(\mathcal{M})$, we define the transitive contact multigraph (TCM for short) $\Gamma(\mathfrak{M})$ as the n -ECM of order n , obtained from $\Gamma(\mathcal{M})$ by taking $\mathcal{V}(\Gamma(\mathfrak{M})) = \{1, \dots, n\}$ and defining $(i, j, k) \in \mathcal{E}(\Gamma(\mathfrak{M}))$ if $L_{i,k}M_{k,j} \neq 0$.*

Remark 4.1. Notice that if we collapse all the parallel edges, then we obtain a graph isomorphic to $\Gamma(LM)$. In particular, Proposition 2.1 then says that $\Gamma(\mathfrak{M})$ is strongly connected.

Remark 4.2. If $(i, j, k) \in \mathfrak{M}$, then this means that an infected host in group j can be the origin of an infection of a host in group i by infecting a vector of group k , which then infects the host in group i . Within the fast travelling interpretation, this means that an infected host that is resident in region j can travel to region k , where it infects a vector there. This infected vector will subsequently infect a susceptible host of region i that travels to region k . See Figure 1 for an example of a host-vector contact network, and the corresponding transitive contact multigraph.

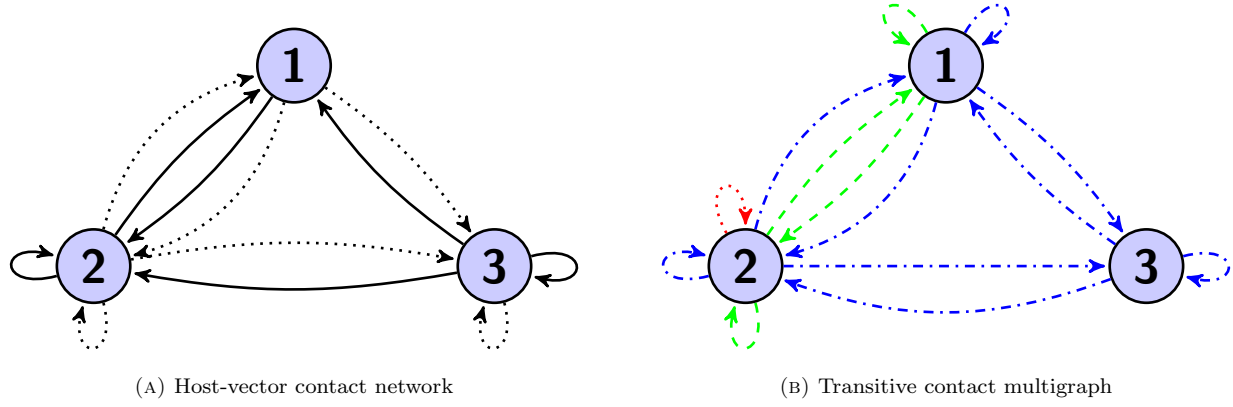


FIGURE 1. In (a) we display a host-vector contact network. Within the travelling interpretation of the model, the solid lines indicate the travelling patterns of susceptible hosts (specified by the nonzero entries of L), while the dotted lines indicate the travelling pattern of the infected hosts (specified by the nonzero entries of M). Notice that, in this example, neither L or M are irreducible, but \mathcal{M} is. In (b) we display the corresponding TCM: the red dotted edge indicates a connection through region 1, the green dashed edges indicate connections through region 2, and the blue dashed-dotted edges indicate connections through region 3.

We will now give a graph-theoretical interpretation of η .

Proposition 4.1. Let $\zeta = \text{diag}(\bar{Y})\eta$. Then ζ spans the kernel of the graph Laplacian of \mathfrak{M} . In particular, its entries are given by (a multiple of) the principal minors along the diagonal and, therefore, it is equal to the sum of the weight product of weights of a spanning tree of \mathfrak{M} , over all such spanning trees.

Proof. From the equilibrium relations, we have

$$T\bar{Y} = \bar{Y}$$

and hence

$$\tilde{T} \cdot \mathbf{1} = \mathbf{1}, \text{ where } \tilde{T} = \text{diag}(\bar{Y})T \text{diag}(\bar{Y}).$$

Thus, we also have

$$\tilde{T}^t \zeta = \zeta, \quad \zeta = \text{diag}(\bar{Y})\eta.$$

Notice now that

$$I - \tilde{T}^t = \begin{pmatrix} 1 - \tilde{T}_{11} & -\tilde{T}_{21} & \cdots & -\tilde{T}_{n1} \\ -\tilde{T}_{12} & 1 - \tilde{T}_{22} & \cdots & -\tilde{T}_{n2} \\ \vdots & \vdots & \ddots & \vdots \\ -\tilde{T}_{1n} & -\tilde{T}_{2n} & \cdots & 1 - \tilde{T}_{nn} \end{pmatrix} = \begin{pmatrix} \sum_{i \neq 1} \tilde{T}_{i1} & -\tilde{T}_{21} & \cdots & -\tilde{T}_{n1} \\ -\tilde{T}_{12} & \sum_{i \neq 2} \tilde{T}_{i2} & \cdots & -\tilde{T}_{n2} \\ \vdots & \vdots & \ddots & \vdots \\ -\tilde{T}_{1n} & -\tilde{T}_{2n} & \cdots & \sum_{i \neq n} \tilde{T}_{in} \end{pmatrix},$$

where we have used that

$$\sum_{i=1}^n \tilde{T}_{i,j} = 1, \quad j \in \{1, \dots, n\}.$$

Therefore ζ is in the kernel of the matrix Laplacian of \tilde{T}^t .

In addition, we have that

$$\Gamma(\tilde{T}^t) = \Gamma(\tilde{T}) = \Gamma(T),$$

and the latter is isomorphic to \mathfrak{M} when collapsing all the parallel edges, and hence the Laplacian of \mathfrak{M} with its edge directions reversed is $I - \tilde{T}^t$. Furthermore, since $\Gamma(M)$ is strongly connected we have, by Proposition 2.1, that \mathfrak{M} is also strongly connected and thus the kernel of the associated Laplacian is one-dimensional [11]. The other claims follow from Kirchhoff's theorem for multigraphs—cf. [7]. \square

Theorem 4.2. *Assume that Hypothesis 2.1 holds and that $\mathcal{R}_0 > 1$. Then the EE is globally asymptotically stable.*

Proof. Let

$$V = \langle X - \bar{X} \circ \log(X), \eta \rangle + \langle Y - \bar{Y} \circ \log(Y), \eta \rangle + \langle Z - \bar{Z} \log(Z), \bar{\xi} \rangle, \quad \bar{\xi} = (\mu_h + \gamma_h) \circ \mu_v^{-1} \circ \xi,$$

where $(\xi, \eta)^t$ is the positive left eigenvector of \bar{N} as discussed in Lemma 4.2. In particular, we have that

$$A^t \text{diag}(\bar{X})\eta = \mu_v \circ \bar{\xi}. \quad \text{and} \quad B^t \text{diag}(\mathbf{1} - \bar{Z})\bar{\xi} = (\mu_h + \gamma_h) \circ \eta.$$

Then

$$\begin{aligned} \dot{V} &= \langle \dot{X} \circ (\mathbf{1} - \bar{X} \circ X^{-1}), \eta \rangle + \langle \dot{Y} \circ (\mathbf{1} - \bar{Y} \circ Y^{-1}), \eta \rangle + \langle \dot{Z} \circ (\mathbf{1} - \bar{Z} \circ Z^{-1}), \bar{\xi} \rangle \\ &= \langle \mu_h \circ (\mathbf{1} - X) - \text{diag}(X)AZ - \mu_h \circ (\mathbf{1} - X) \circ \bar{X} \circ X^{-1} + (\text{diag}(X)AZ) \circ \bar{X} \circ X^{-1}, \eta \rangle \\ &\quad + \langle \text{diag}(X)AZ - (\mu_h + \gamma_h) \circ Y - (\text{diag}(X)AZ) \circ \bar{Y} \circ Y^{-1} + (\mu_h + \gamma_h) \circ \bar{Y}, \eta \rangle \\ &\quad + \langle \text{diag}(\mathbf{1} - Z)BY - \mu_v \circ Z - (\text{diag}(\mathbf{1} - Z)BY) \circ \bar{Z} \circ Z^{-1} + \mu_v \circ \bar{Z}, \bar{\xi} \rangle \\ &= \langle \mu_h \circ (\mathbf{1} + \bar{X} - X - \bar{X} \circ X^{-1}), \eta \rangle + \langle (AZ) \circ \bar{X}, \eta \rangle - \langle \mu_v \circ Z, \bar{\xi} \rangle - \langle (\mu_h + \gamma_h) \circ Y, \eta \rangle \\ &\quad + \langle (\mu_h + \gamma_h) \circ \bar{Y}, \eta \rangle - \langle (\text{diag}(X)AZ) \circ \bar{Y} \circ Y^{-1}, \eta \rangle + \langle \text{diag}(\mathbf{1} - Z)BY, \bar{\xi} \rangle \\ &\quad - \langle (\text{diag}(\mathbf{1} - Z)BY) \circ \bar{Z} \circ Z^{-1}, \bar{\xi} \rangle + \langle \mu_v \circ \bar{Z}, \bar{\xi} \rangle. \end{aligned}$$

Now observe that

$$\langle (AZ) \circ \bar{X}, \eta \rangle = \langle \text{diag}(\bar{X})AZ, \eta \rangle = \langle Z, A^t \text{diag}(\bar{X})\eta \rangle = \langle \mu_v \circ Z, \bar{\xi} \rangle.$$

Also, from the equilibrium equations:

$$(\mu_h + \gamma_h) \circ \bar{Y} = \mu_h \circ (\mathbf{1} - \bar{X}) \quad \text{and} \quad \text{diag}(\bar{X})A\bar{Z} = \mu_h \circ (\mathbf{1} - \bar{X}).$$

Thus,

$$\langle \mu_v \circ \bar{Z}, \bar{\xi} \rangle = \langle \bar{Z}, A^t \text{diag}(\bar{X})\eta \rangle = \langle \mu_h (\mathbf{1} - \bar{X}), \eta \rangle.$$

Combining all this information, we find that

$$\begin{aligned} \dot{V} &= \langle \mu_h \circ (3\mathbf{1} - \bar{X} - X - \bar{X} \circ X^{-1}), \eta \rangle - \langle (\mu_h + \gamma_h) \circ Y, \eta \rangle - \langle (\text{diag}(X)AZ) \circ \bar{Y} \circ Y^{-1}, \eta \rangle \\ &\quad + \langle \text{diag}(\mathbf{1} - Z)BY, \bar{\xi} \rangle - \langle (\text{diag}(\mathbf{1} - Z)BY) \circ \bar{Z} \circ Z^{-1}, \bar{\xi} \rangle \\ &= \langle \mu_h \circ (3\mathbf{1} - \bar{X} - X - \bar{X} \circ X^{-1}), \eta \rangle + \langle \text{diag}(\mathbf{1} - \bar{Z})BY, \bar{\xi} \rangle - \langle (\mu_h + \gamma_h) \circ Y, \eta \rangle \\ &\quad - \langle (\text{diag}(X)AZ) \circ \bar{Y} \circ Y^{-1}, \eta \rangle + \langle \text{diag}(\bar{Z} - Z)BY, \bar{\xi} \rangle - \langle (\text{diag}(\mathbf{1} - Z)BY) \circ \bar{Z} \circ Z^{-1}, \bar{\xi} \rangle. \end{aligned}$$

We also have

$$\begin{aligned} \langle \text{diag}(\mathbf{1} - \bar{Z})BY, \bar{\xi} \rangle &= \langle Y, B^t \text{diag}(\mathbf{1} - \bar{Z})\bar{\xi} \rangle \\ &= \langle (\mu_h + \gamma_h) \circ Y, \eta \rangle. \end{aligned}$$

and

$$\begin{aligned} & \langle \text{diag}(\bar{Z} - Z)BY, \bar{\xi} \rangle - \langle (\text{diag}(\mathbf{1} - Z)BY) \circ \bar{Z} \circ Z^{-1}, \bar{\xi} \rangle \\ &= \langle [2\bar{Z} - Z - \bar{Z} \circ Z^{-1}] \circ BY, \bar{\xi} \rangle. \end{aligned}$$

Hence, we are left with

$$\begin{aligned} \dot{V} &= \langle \mu_h \circ (3\mathbf{1} - \bar{X} - X - \bar{X} \circ X^{-1}), \eta \rangle + \langle [2\bar{Z} - Z - \bar{Z} \circ Z^{-1}] \circ BY, \bar{\xi} \rangle \\ &\quad - \langle (\text{diag}(X)AZ) \circ \bar{Y} \circ Y^{-1}, \eta \rangle. \end{aligned}$$

Now we write

$$\mathbf{1} = \bar{X} + \mathbf{1} - \bar{X} \quad \text{and} \quad \mathbf{1} = \bar{Z} + \mathbf{1} - \bar{Z}.$$

Then, we also have

$$-X - \bar{X}^2 \circ X^{-1} \leq -2\bar{X},$$

and analogously for $Z - \bar{Z}^2 \circ Z^{-1}$.

Therefore, we find

$$\begin{aligned} \dot{V} &\leq 3\langle \mu_h \circ (\mathbf{1} - \bar{X}), \eta \rangle - \langle \mu_h \circ \bar{X} \circ (\mathbf{1} - \bar{X}) \circ X^{-1}, \eta \rangle \\ &\quad - \langle \bar{Z} \circ (\mathbf{1} - \bar{Z}) \circ Z^{-1} \circ (BY), \bar{\xi} \rangle - \langle (\text{diag}(X)AZ) \circ \bar{Y} \circ Y^{-1}, \eta \rangle. \end{aligned}$$

Notice that the inequality above for \dot{V} is strict, except when $X = \bar{X}$ and $Z = \bar{Z}$.

Since

$$\bar{\xi} = \text{diag}(\mu_v)^{-1} A^t \text{diag}(\bar{X})\eta,$$

we can then write

$$\begin{aligned} \dot{V} &\leq 3\langle \mu_h \circ (\mathbf{1} - \bar{X}), \eta \rangle - \langle \mu_h \circ \bar{X} \circ (\mathbf{1} - \bar{X}) \circ X^{-1}, \eta \rangle \\ &\quad - \langle \mu_v^{-1} \circ \bar{X} \circ A (\bar{Z} \circ (\mathbf{1} - \bar{Z}) \circ Z^{-1} \circ (BY)), \eta \rangle - \langle (\text{diag}(X)AZ) \circ \bar{Y} \circ Y^{-1}, \eta \rangle. \end{aligned}$$

Let

$$\bar{A} = \text{diag}(\bar{X})A \text{diag}(\bar{Z}) \quad \text{and} \quad \bar{B} = \text{diag}(\mu_v)^{-1} \text{diag}(\bar{Z})^{-1} \text{diag}(\mathbf{1} - \bar{Z})B \text{diag}(\bar{Y}).$$

Then $\bar{A}\mathbf{1} = \mu_h \circ (\mathbf{1} - \bar{X})$ and $\bar{B}\mathbf{1} = \mathbf{1}$. We can then write

$$\begin{aligned} \dot{V} &\leq 3\langle \bar{A}\mathbf{1}, \eta \rangle - \langle (\bar{A}\mathbf{1}) \circ \bar{X} \circ X^{-1}, \eta \rangle \\ &\quad - \langle \bar{A} (\bar{Z} \circ Z^{-1} \circ (\bar{B} (Y \circ \bar{Y}^{-1}))) , \eta \rangle - \langle X \circ \bar{X}^{-1} \circ (\bar{A} (\bar{Z} \circ \bar{Z}^{-1})) \circ \bar{Y} \circ Y^{-1}, \eta \rangle \\ &= \sum_{i=1}^n \eta_i \left[3(\bar{A}\mathbf{1})_i - \frac{\bar{X}_i}{X_i} (\bar{A}\mathbf{1})_i - (\bar{A} (\bar{Z} \circ Z^{-1} \circ (\bar{B} (Y \circ \bar{Y}^{-1}))))_i - \frac{X_i \bar{Y}_i}{\bar{X}_i Y_i} (\bar{A} (\bar{Z} \circ \bar{Z}^{-1}))_i \right] \\ &= \sum_{i,j=1}^n \eta_i \bar{A}_{i,j} \left[3 - \frac{\bar{X}_i}{X_i} - \frac{\bar{Z}_j}{Z_j} (\bar{B} (Y \circ \bar{Y}^{-1}))_j - \frac{X_i \bar{Y}_i Z_j}{\bar{X}_i Y_i \bar{Z}_j} \right] \\ &= \sum_{i,j,k=1}^n \eta_i \bar{A}_{i,j} \bar{B}_{j,k} \left[3 - \frac{\bar{X}_i}{X_i} - \frac{\bar{Z}_j Y_k}{Z_j \bar{Y}_k} - \frac{X_i \bar{Y}_i Z_j}{\bar{X}_i Y_i \bar{Z}_j} \right] \\ &= H_n. \end{aligned}$$

Before proceeding, we recall that a unicyclic graph is a connected digraph with a unique oriented cycle such that, if we remove the edges on the cycle, we are left with a family of directed rooted trees, whose roots are precisely the vertices in the cycle—see [28, 44, 61]. Given the graph \mathfrak{M} , we shall denote by $\mathcal{D}(n, l)$ the set of unicyclic subgraphs of \mathfrak{M} , that has order n , with cycle of length l . Recalling that \mathfrak{M} is a n -ECM, we

notice that, in a similar way as in Guo et al [27, 28] we have

$$\begin{aligned}
H_n &= \sum_{i,j,k}^n \eta_i \bar{A}_{i,j} \bar{B}_{j,k} \left[3 - \frac{\bar{X}_i}{X_i} - \frac{Y_k}{\bar{Y}_k} \frac{\bar{Z}_j}{Z_j} - \frac{X_i \bar{Y}_i}{\bar{X}_i Y_i} \frac{Z_j}{\bar{Z}_j} \right] \\
&= \sum_{l=1}^n \left\{ \sum_{Q \in \mathcal{D}(n,l)} \left(\prod_{(k,h,j) \in \mathcal{E}(CQ)} \bar{A}_{k,j} \bar{B}_{j,h} \right) \right. \\
&\quad \times \left. \sum_{(r,m,j) \in \mathcal{E}(CQ)} \left[3 - \frac{\bar{X}_r}{X_r} - \frac{Y_m}{\bar{Y}_m} \frac{\bar{Z}_j}{Z_j} - \frac{X_r \bar{Y}_r}{\bar{X}_r Y_r} \frac{Z_j}{\bar{Z}_j} \right] \right\},
\end{aligned}$$

where CQ denotes the unique cycle in the unicyclic graph Q . Along such a cycle, we have

$$\begin{aligned}
&\sum_{(r,m,j) \in \mathcal{E}(CQ)} \left[3 - \frac{\bar{X}_r}{X_r} - \frac{Y_m}{\bar{Y}_m} \frac{\bar{Z}_j}{Z_j} - \frac{X_r \bar{Y}_r}{\bar{X}_r Y_r} \frac{Z_j}{\bar{Z}_j} \right] \\
&= 3|\mathcal{E}(CQ)| - \sum_{(r,m,j) \in \mathcal{E}(CQ)} \left[\frac{\bar{X}_r}{X_r} + \frac{Y_m}{\bar{Y}_m} \frac{\bar{Z}_j}{Z_j} + \frac{X_r \bar{Y}_r}{\bar{X}_r Y_r} \frac{Z_j}{\bar{Z}_j} \right] \\
&\leq 3|\mathcal{E}(CQ)| - 3|\mathcal{E}(CQ)| \left[\prod_{(r,m,j) \in \mathcal{E}(CQ)} \frac{Y_m \bar{Y}_r}{\bar{Y}_m Y_r} \right]^{1/3|\mathcal{E}(CQ)|} \\
&= 0.
\end{aligned}$$

Hence, we have that $H_n \leq 0$, with equality being attained only when

$$\frac{\bar{X}_r}{X_r} = \frac{Y_m}{\bar{Y}_m} \frac{\bar{Z}_j}{Z_j} = \frac{X_r \bar{Y}_r}{\bar{X}_r Y_r} \frac{Z_j}{\bar{Z}_j}, \quad (r, m, j) \in \mathcal{E}(CQ).$$

But since, we have $\dot{V} \leq H_n$, with equality only when $X = \bar{X}$ and $Z = \bar{Z}$, we find that $\dot{V} \leq 0$, with equality attained only when, for each $Q \in \mathcal{D}(n, l)$, $l = 1, \dots, n$, we have

$$1 = \frac{Y_m}{\bar{Y}_m} = \frac{\bar{Y}_r}{Y_r}, \quad (r, m, \cdot) \in \mathcal{E}(CQ).$$

But since CQ is a cycle, we have that

$$Y_r = \bar{Y}_r, \quad r \in \mathcal{V}(CQ).$$

Since \mathcal{M} is irreducible, we have that $\bar{A}\bar{B}$ is also irreducible by Proposition 2.1. Thus, we have that any two vertices will be in some unicyclic graph, and hence we have equality only when

$$Y = \bar{Y}.$$

□

5. DISCUSSION

We have considered a class of multi-group models for vector-borne diseases. This class is a natural extension of the classical Bailey-Dietz model and it is a natural candidate for modeling the impact of fast urban movement in some vector transmitted diseases, as for instance, in the case of dengue fever—cf. [1, 2, 12]. The host-vector interaction along the network gives rise to what we call the host-vector contact network—denoted by $\Gamma(\mathcal{M})$ —and that has a number of distinguishing features from the networks that arise in directly transmitted diseases. The most striking one is, perhaps, that the irreducibility of the circulation topology is not sufficient to guarantee the irreducibility of the host-vector topology. In addition, we also characterize the irreducibility of $\Gamma(\mathcal{M})$ through the irreducibility of the product sub-networks. With this assumption, we are able to provide a complete analysis of the dynamics in the sense that this class of models possesses the so-called sharp \mathcal{R}_0 property, i.e., \mathcal{R}_0 is a threshold parameter with the disease free equilibrium being both locally and globally asymptotically stable when $\mathcal{R}_0 \leq 1$, and being unstable when $\mathcal{R}_0 > 1$. In addition,

an interior equilibrium (the endemic equilibrium) that is biologically feasible, i.e. has positive coordinates, exists if and only if $\mathcal{R}_0 > 1$. Furthermore, it is globally asymptotically stable, whenever it exists.

From a mathematical point of view, these results extend previous results of directly transmitted diseases to the class considered here. The global stability of the disease free equilibrium (which has been obtained by [16] for a special case, and more restricted conditions) is a very natural extension of the argument presented in [27]; see also [70] for a very general presentation of this argument. The existence, uniqueness and local stability of the endemic equilibrium shows that the corresponding results of [33] for sub-populations hold for this class of models. Finally, the global stability proof brings a new ingredient in the graph-theoretic framework introduced in [27, 28]: the identification of $\Gamma(\mathcal{M})$ with a multi-graph—that we have termed a transitive contact multi-graph—which is a c -edge colored multi-digraph, and which contains all the information of the host-vector contact network encoded on a different way. The product of the host and vector networks can then be interpreted as a contact matrix for such a graph, and that allows us to organize the calculation of the Lie-derivative of the Lyapunov function within a similar graph-theoretical framework of [27, 28].

The analysis presented here shows that, despite the complexity of the models in the class considered here, the long-term global dynamics is very simple. This, however, does not imply that the transient dynamics of the model is necessarily simple, and further studies are necessary. As an example of this complexity, we refer to [2] which provides examples of situations—including in the class analyzed here—that have a local group \mathcal{R}_0 less than unity, but a global \mathcal{R}_0 that is greater than unity—and hence bounded to evolve to an endemic state in the long term. While this duality of local versus global \mathcal{R}_0 has been observed in other contexts—see [60]—we believe that it should be further studied and understood in the realm of epidemic models.

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