DYNAMICS OF A NETWORKED CONNECTIVITY MODEL OF EPIDEMICS

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(Communicated by Yuan Lou)

Abstract. A networked connectivity model of waterborne disease epidemics on a site of \( n \) communities is studied. Existence and local stability analysis for both the disease-free equilibrium and the endemic equilibrium are studied. Using an appropriate Lyapunov function and Lasalle invariance principle, global asymptotic stability of the disease-free equilibrium point is established. Existence of a transcritical bifurcation at the disease outbreak is also proved. This work extends previous research in networked connectivity models of epidemics.

1. Introduction. Mathematical models of epidemics can help understand the dynamics of the disease, in particular, the conditions under which epidemics can begin and spread in a spatially explicit area. This in turn is helpful in expanding emergency management of the disease, creating new health-care resources, and preventing the initiation and spread of the disease.

Waterborne diseases are a result of protozoa or bacteria in water causing contamination and infection as a result. Typically, disease models consider only one given community or location (e.g. \([3, 8, 11, 14]\)). In such cases, it has been shown that the so called basic reproduction number \( R_0 \), needs to be greater than one in order for an outbreak to take place. The model studied here, originally proposed by Gatto et al. \([5]\), considers a number \( n \) of communities connected by hydrological and human mobility networks, and the dynamics of the bacteria is explicitly included in the system. The condition that one or more of the corresponding basic reproduction numbers \( R_{0i}, i = 1, \ldots, n \) cross the value one, is in this case not sufficient nor necessary for an outbreak in the metacommunity to occur. Conversely, the
disease can start even if the basic reproduction number of each community is less than one [5]. This clearly illustrates the fact that adding spatial structure brings totally new phenomena and dynamics to the system. The idea in [5] is to introduce a kind of generalized reproduction number \( \Lambda_0 \), which will be related to a so-called general reproduction matrix \( G_0 \), and to study the spectral properties of \( G_0 \). In this work we extend the results of previous works on these connectivity models, among other things by introducing an appropriate Lyapunov function that allows global stability analysis of the disease-free equilibrium point and by proving existence on an endemic equilibrium, with some conditions for its local stability. We also prove existence of transcritical bifurcations and provide with some numerical calculations to illustrate these results.

2. Epidemic model. The model is a spatially explicit nonlinear differential model that uses both hydrological and human networks through which a disease can spread, because bacteria can spread from community to community carried by humans, or by traveling through river basins or man-made water distribution and sewage systems. For human mobility patterns, it is assumed that individuals leave their original community or node \( i \) with probability \( m_s \) for susceptibles and \( m_I \) for infectives, reach their target node \( j \) with a probability \( Q_{ij} \) and come back to node \( i \). We also assume that pathogens can move at a rate \( r \) from node \( i \) to node \( j \) along the hydrological network with probability \( P_{ij} \). A realistic assumption is that the graph \( \Gamma(P \cup Q) \) is strongly connected. This means that bacteria can move from any community to any other community in the network represented by hydrological connections or human mobility.

Let \( S_i, I_i, B_i \) represent the population densities of susceptibles, infected/infected, and bacteria concentration respectively at the \( i \)-th community of size \( H_i \), with \( i = 1, \ldots, n \). The corresponding system of \( 3n \) differential equations, where all parameters are assumed to be positive, is [5]:

\[
\begin{align*}
S_i' &= \mu(H_i - S_i) - \left( (1 - m_s)\beta_i f(B_i) + m_s \sum_{j=1}^{n} Q_{ij} \beta_j f(B_j) \right) S_i \\
I_i' &= \left[ (1 - m_s)\beta_i f(B_i) + m_s \sum_{j=1}^{n} Q_{ij} \beta_j f(B_j) \right] S_i - \phi I_i \\
B_i' &= -\mu_B B_i + r \left( \sum_{j=1}^{n} P_{ji} \frac{W_j}{W_i} B_j - B_i \right) + \frac{p_i}{W_i} \left( (1 - m_I) I_i + \sum_{j=1}^{n} m_I Q_{ji} I_j \right),
\end{align*}
\]

where \( f(B_i) = \frac{B_i}{K + B_i} \) (\( K \) being the half-saturation constant) is the probability of becoming infected due to exposure to bacteria \( B_i \) at node \( i \); \( \mu \) is the human birth and death rate, \( \beta_i \) is the rate of exposure to contaminated water, \( \phi \) represents the rate of recovery or death of infected, \( \mu_B \) is the rate of death of pathogens, and \( p_i \) is the rate at which bacteria are released by infected individuals in a water reservoir of volume \( W_i \). Clearly, \( P \) and \( Q \) are stochastic matrices, with \( Q_{ii} = P_{ii} = 0 \) for each \( i = 1, \ldots, n \).

3. Stability analysis of three communities. The ultimate goal is to study the model for an arbitrary \( n > 1 \), but for clarity and for illustration purposes we first study the case \( n = 3 \). The general case, which easily follows from \( n = 3 \), is
considered in the next section. As mentioned before, we assume the total network to be represented by a strongly connected graph.

3.1. Local stability. Although the approach to study local stability is no different than what is typically done for general models in population dynamics (e.g., [6, 9]), the particular properties of the matrices involved here can be exploited, which is especially important given the dimension of the system. With \( n = 3 \), the nine differential equations in (1) are rewritten by rescaling the bacteria population as \( B_i = B_i/K \). With this rescaling, the Jacobian at the disease-free equilibrium point \((H_1, H_2, H_3, 0, 0, 0, 0, 0, 0)\) is

\[
J = \begin{bmatrix}
-\mu & 0 & 0 & 0 & 0 & -H_1(1-m_S)\beta_1 & -H_1m_SQ_{12}\beta_2 & -H_1m_SQ_{13}\beta_3 \\
0 & -\mu & 0 & 0 & 0 & -H_2m_SQ_{21}\beta_1 & -H_2(1-m_S)\beta_2 & -H_2m_SQ_{23}\beta_3 \\
0 & 0 & -\mu & 0 & 0 & -H_3m_SQ_{31}\beta_1 & -H_3m_SQ_{32}\beta_2 & -H_3(1-m_S)\beta_3 \\
0 & 0 & 0 & -\phi & 0 & H_1(1-m_S)\beta_1 & H_1m_SQ_{12}\beta_2 & H_1m_SQ_{13}\beta_3 \\
0 & 0 & 0 & 0 & -\phi & H_2m_SQ_{21}\beta_1 & H_2(1-m_S)\beta_2 & H_2m_SQ_{23}\beta_3 \\
0 & 0 & 0 & 0 & 0 & -\phi & H_3m_SQ_{31}\beta_1 & H_3m_SQ_{32}\beta_2 & H_3(1-m_S)\beta_3 \\
0 & 0 & 0 & 0 & 0 & 0 & (\mu B + r) & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & (\mu B + r) & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & (\mu B + r)
\end{bmatrix}
\]

We can write the Jacobian in the following form:

\[
J = \begin{bmatrix}
J_{11} & 0 & J_{13} \\
0 & J_{22} & J_{23} \\
0 & J_{32} & J_{33}
\end{bmatrix}
\]

Each block \( J_{ij} \) is a \( 3 \times 3 \) matrix, and as usual denoting the identity matrix of appropriate order as \( I \), we have

\[
J_{22} = -\phi I, \quad J_{23} = m_SHQ\beta + (1-m_S)H\beta, \quad J_{32} = \frac{m_S}{K}pW^{-1}Q^T + \frac{1-m_S}{K}pW^{-1}, \quad J_{33} = -(\mu B + r)I + rW^{-1}P^T W,
\]

where \( H, W, W^{-1} \) and \( \beta \) are diagonal matrices with diagonal entries \( H_i, W_i, 1/W_i \), and \( \beta_i \) respectively. Since the eigenvalues of \( J_{11} \) are negative, the possible loss of stability of the disease-free equilibrium is determined by the eigenvalues of the matrix \( J^* \).

Recall that a matrix \( A_{n \times n} \) is irreducible if and only if its graph \( \Gamma(A) \) is strongly connected. Clearly, \( J^* \) is irreducible. On the other hand, \( A_{n \times n} \) is said to be Metzler if all of the off-diagonal entries are nonnegative. Clearly again, \( J^* \) is Metzler. The main property of a Metzler matrix \( A \) is that its eigenvalue with maximum real part is real. That is,

\[
\alpha(A) = \max_i \Re \lambda_i(A), \quad i = 1, 2, \ldots, n
\]

is a real eigenvalue of \( A \). The following theorem is an extension of the Perron-Frobenius theorem, and it will later be applied to the matrix \( J^* \).

**Theorem 3.1.** [10] Let \( A_{n \times n} \) be an irreducible Metzler matrix. Then \( \lambda^* = \alpha(A) \) is a simple eigenvalue, and it has an associated positive eigenvector.
Consequently, when the eigenvalue \( \lambda^* = \alpha(J^*) \) reaches zero from below, the disease-free equilibrium will lose stability (and we might also expect a transcritical bifurcation to occur). Observe that in this case we have \( \det(J^*) = 0 \). A result in [12] says that for matrices \[
\begin{bmatrix}
A & B \\
C & D
\end{bmatrix}
\]
in which all blocks are square and matrix \( A \) commutes with matrix \( C \), the following equality holds: \[
\det\left(\begin{bmatrix}
A & B \\
C & D
\end{bmatrix}\right) = \det(AD - CB).
\]
This property is true for \( J^* \) because \( J_{22}^* \) is a multiple of the identity matrix.

Then, (see [5] for details)
\[
\det(J^*) = \det\left[\left(\begin{array}{c,c}
J_{22}^* & J_{23}^* \\
J_{32}^* & J_{33}^*
\end{array}\right) - \left(\begin{array}{c,c}
J_{22} & J_{23} \\
J_{32} & J_{33}
\end{array}\right)\right]\]
and the condition \( \det(J^*) = 0 \) is given by
\[
\det\left[\left(\begin{array}{c,c}
I & \frac{\mu_B + r}{\mu_B} P^T - \frac{\mu_B}{\mu_B + r} \left[\left(1 - m_l\right)\left(1 - m_S\right)R_0 + \frac{m_m l m_i}{K\mu_B}\phi pQ^T H\beta W^{-1} + \frac{m_l\left(1 - m_S\right)}{K\mu_B}\phi pQ^T H\beta W^{-1} + \left(1 - m_l\right)\frac{m_S}{K\mu_B}\phi pH\beta W^{-1}\right]\right]
\right) = 0.
\]
By letting
\[
R_0^R = \frac{pQ^T H\beta W^{-1}}{K\mu_B\phi}, \quad R_0^Q = \frac{pH\beta W^{-1}}{K\mu_B\phi}, \quad R_0^{1s} = \frac{pQ^T H\beta W^{-1}}{K\mu_B\phi},
\]
we can define the matrix
\[
T_0 = (1 - m_l)(1 - m_S)R_0 + m_m l m_i R_0^{1s} + m_l(1 - m_S)R_0^{1s} + (1 - m_l)m_S R_0^Q,
\]
where the diagonal matrix \( R_0 = \frac{pQ^T H\beta W^{-1}}{K\mu_B\phi} \) represents the reproduction numbers for the communities, that is, its diagonal entries are \( R_{0i} = \frac{p_i H_i\beta_i}{W_i K\mu_B\phi} \). Thus, the disease-free equilibrium loses stability when
\[
\det\left(I - \frac{r}{\mu_B + r} P^T - \frac{\mu_B}{\mu_B + r} T_0\right) = 0.
\]
The matrix
\[
G_0 = \frac{r}{\mu_B + l} P^T + \frac{\mu_B}{\mu_B + r} T_0
\]
is called the generalized reproduction matrix. Therefore, the condition \( \det(J^*) = 0 \) is equivalent to
\[
\det(I - G_0) = 0.
\]
Observe that \( G_0 \) is the convex sum of two matrices, the first depending on the hydrological matrix \( P \) and the second one on the human mobility matrix \( Q \), indicating how a possible outbreak directly depends on the networks through which the pathogen spreads.

The analysis above indicates that the disease-free equilibrium loses stability when the eigenvalue \( \lambda^* = \alpha(J^*) \) of \( J^* \) with maximal real part is zero, or equivalently
when \( \lambda = 1 \) is an eigenvalue of \( G_0 \). In fact, \( \lambda = 1 \) is the dominant eigenvalue of \( G_0 \). To show this, consider the following result:

**Theorem 3.2.** [2] Let \( A = sI - B \), where \( B \) is an \( n \times n \) nonnegative matrix. If there exists a vector \( x > 0 \), such that \( Ax \) has positive entries, then \( \rho(B) \leq s \).

First, since \( \det(I - G_0) = 0 \), \( \lambda = 1 \) is an eigenvalue of \( G_0 \). It is simple to see that \( G_0 \) is positive. Then, using the Perron-Frobenius Theorem, we know that \( \rho(G_0) \) is a simple eigenvalue, and \( \rho(G_0) \geq 1 \). On the other hand, it is straightforward to verify that the conditions of Theorem 3.2 are satisfied with \( B = G_0 \), and therefore \( \rho(G_0) \leq 1 \). Hence, \( \rho(G_0) = 1 \), and \( \lambda = 1 \) is the dominant eigenvalue of \( G_0 \).

Thus, the disease free equilibrium point loses stability (and we expect an outbreak to happen) exactly when the dominant eigenvalue of \( G_0 \) equals one.

### 3.2. Global stability

The model (1) for \( n = 3 \) can be rewritten as a compartmental model [11]

\[
x' = F^*(x,y) - V^*(x,y), \quad y' = g(x,y),
\]

where \( x = [x_1 \cdots x_6]^T \) is the disease compartment and \( y = [y_1 y_2 y_3]^T \) is the non-disease compartment, respectively. Here we have \( x = [I_1 I_2 I_3 B_1 B_2 B_3]^T \), and \( y = [S_1 S_2 S_3]^T \). Also, \( F^* = [F_1^* \cdots F_6^*]^T \) and each entry represents the rate of new infections in the \( i \)-th disease compartment, and \( V^* = [V_1^* \cdots V_6^*]^T \), where each entry represents the transition terms such as recover or death in the \( i \)-th disease compartment. Thus, we have (again, with \( B_i = B_i/K \))

\[
F^* = \begin{bmatrix}
((1 - m_s)\beta_1 \frac{B_1}{B_1 + \phi} + m_s(Q_{12}\beta_2 \frac{B_2}{B_2 + \phi} + Q_{13}\beta_3 \frac{B_3}{B_3 + \phi}))S_1 \\
((1 - m_s)\beta_2 \frac{B_2}{B_2 + \phi} + m_s(Q_{21}\beta_1 \frac{B_1}{B_1 + \phi} + Q_{23}\beta_3 \frac{B_3}{B_3 + \phi}))S_2 \\
((1 - m_s)\beta_3 \frac{B_3}{B_3 + \phi} + m_s(Q_{31}\beta_1 \frac{B_1}{B_1 + \phi} + Q_{32}\beta_2 \frac{B_2}{B_2 + \phi}))S_3 \\
(1 - m_s)I_1 + m_s(I_2 Q_{21} + Q_{23} I_3) \\
(1 - m_s)I_2 + m_s(I_1 Q_{12} + Q_{31} I_3) \\
(1 - m_s)I_3 + m_s(I_1 Q_{12} + Q_{32} I_2)
\end{bmatrix},
\]

\[
V^* = \begin{bmatrix}
\phi I_1 \\
\phi I_2 \\
(\mu_B + \tau)B_1 \\
(\mu_B + \tau)B_2 \\
(\mu_B + \tau)B_3
\end{bmatrix},
\]

By letting \( y_0 = [H_1 H_2 H_3]^T \), \( R_i = \frac{P_i}{K W_i} \), \( i = 1, 2, 3 \), and

\[
F = \left[ \frac{\partial F_i}{\partial x_j}(0, y_0) \right] \quad \text{and} \quad V = \left[ \frac{\partial V_i}{\partial x_j}(0, y_0) \right], \quad 1 \leq i, j \leq 6,
\]

we get

\[
F = \begin{bmatrix}
0 & 0 & 0 & (1 - m_s)\beta_1 I_1 + m_s Q_{12} \beta_2 H_2 & m_s Q_{13} \beta_3 H_3 \\
0 & 0 & 0 & m_s Q_{21} \beta_1 H_1 & (1 - m_s)\beta_2 I_2 + m_s Q_{23} \beta_3 H_3 \\
0 & 0 & 0 & m_s Q_{31} \beta_1 H_1 & m_s Q_{32} \beta_2 H_2 & (1 - m_s)\beta_3 I_3
\end{bmatrix},
\]

\[
V = \text{diag}(\phi, \phi, \phi, \mu_B + \tau, \mu_B + \tau, \mu_B + \tau).
\]

Thus, \( F \geq 0 \) and also \( V^{-1} \geq 0 \).
Proposition 1.
\[ \lambda^*(J^*) \leq 0 \iff \lambda^*_F(V^{-1}F) \leq 1, \]
where \( \lambda^* \) and \( \lambda^*_F \) denote the (simple) eigenvalues of \( J^* \) and \( V^{-1}F \) respectively, with maximal real part.

Proof. Observe that \( \lambda^*(J^*) \leq 0 \) if and only if \( \tilde{\lambda}^*(V^{-1}J^*) \leq 0 \), where \( \tilde{\lambda}^* \) denotes the (simple) eigenvalue of \( V^{-1}J^* \) with maximal real part. These eigenvalues are well-defined because both matrices are Metzler. On the other hand, since \( J^* = F - V \), we also have \( V^{-1}J^* = V^{-1}F - I \). Thus, \( \mu - 1 \) is an eigenvalue of \( V^{-1}J^* \) if and only if \( \mu \) is an eigenvalue of \( V^{-1}F \). In particular, \( \tilde{\lambda}^*(V^{-1}J^*) \leq 0 \) if and only if \( \lambda^*_F(V^{-1}F) \leq 1 \). (Note that also \( \lambda^*_F \) is well-defined)

Remark 1. Observe that the inequality \( \lambda^*(J^*) \leq 0 \) in (8) - and by Proposition 1 also the inequality \( \lambda^*_F(V^{-1}F) \leq 1 \) - is directly related to the stability of the disease-free equilibrium point in the networked connectivity model, just as the condition \( R_0 \leq 1 \) is for the case \( n = 1 \) of one single community.

To establish global stability, we first construct a Lyapunov function.

Proposition 2. Let \( \lambda^*_F(V^{-1}F) \leq 1 \), and let \( w \) be a left eigenvector of \( V^{-1}F \) corresponding to \( \lambda^*_F(V^{-1}F) \). Then the function \( Q(x) = w^T V^{-1}x \) is a Lyapunov function satisfying \( Q \leq 0 \).

Proof. First let \( f(x, y) = (F - V)x - F^*(x, y) + V^*(x, y) \).

Note again that \( F - V = J^* \). Then, \( f(x, y) \) is the vector
\[
\begin{pmatrix}
H_1((1 - m_S)\beta_1B_1 + m_S(Q_{12}\beta_2B_2 + Q_{13}\beta_3B_3)) - S_1((1 - m_S)\beta_1L_1 + m_S(Q_{12}\beta_2L_2 + Q_{13}\beta_3L_3)) \\
H_2((1 - m_S)\beta_2B_2 + m_S(Q_{23}\beta_3B_3)) - S_2((1 - m_S)\beta_2L_2 + m_S(Q_{21}\beta_1L_1 + Q_{23}\beta_3L_3)) \\
H_3((1 - m_S)\beta_3B_3 + m_S(Q_{31}\beta_1B_1 + Q_{32}\beta_2B_2)) - S_3((1 - m_S)\beta_3L_3 + m_S(Q_{31}\beta_1L_1 + Q_{32}\beta_2L_2)) \\
0 \\
0 \\
0
\end{pmatrix},
\]
where \( L_i = B_i / (1 + B_i) \), for \( i = 1, 2, 3 \).

Observe that \( f(x, y) \geq 0 \) because \( H_i \geq S_i \) for \( i = 1, 2, 3 \). Clearly, we also have \( V^{-1}F \geq 0 \).

The Lyapunov function \( Q = w^TV^{-1}x \), is given by
\[
Q = \frac{1}{K\phi(\mu B + r)} \left[ I_1 \left( \frac{p_1(1 - m_1)}{W_1} + \frac{p_2m_1Q_{12}}{W_2} + \frac{p_3m_1Q_{13}}{W_3} \right) + 
I_2 \left( \frac{p_1m_1Q_{21}}{W_1} + \frac{p_2(1 - m_1)}{W_2} + \frac{p_3m_1Q_{23}}{W_3} \right) + 
I_3 \left( \frac{p_1m_1Q_{31}}{W_1} + \frac{p_2m_1Q_{32}}{W_2} + \frac{p_3(1 - m_1)}{W_3} \right) \right] + \frac{1}{\mu B + r} \left( B_1 + B_2 + B_3 \right).
\]

We immediately observe that \( Q(x_0) = Q(0, 0, S) = 0 \) and \( Q(x) > 0 \) when \( x \neq x_0 \). Since \( V^{-1}F \) is a Metzler matrix, we can assume its left eigenvector \( w \) is positive, and since \( \lambda^*_F \leq 1 \), we get that...
By Proposition 1, the condition $0 < \lambda^* \leq 0$ implies that the disease-free equilibrium point of system (1) is unstable. Here we rely on Proposition 1.

Theorem 3.3. Let $\Omega$ be a compact invariant set in $\mathbb{R}^3_+$ containing the disease-free equilibrium point of (1). Let $F$, $V$, and $f(x,y)$ be defined as above, and assume $0 < \lambda^*_p(V^{-1}F) < 1$. If the disease-free system $y' = g(0, y)$ has a unique equilibrium $y = y_0 > 0$ that is globally asymptotically stable then, the disease-free equilibrium of the nine-dimensional system (1) is globally asymptotically stable in $\Omega$.

Proof. From the above calculations, it is clear that $F$, $V^{-1}$, $f(x,y) \geq 0$. The disease-free system $y' = g(0, y)$ is equivalent to $S_i' = \mu H_i - \mu S_i$ where $i = 1, 2, 3$. Therefore, $S = [H_i + C_1e^{-\mu \tau} H_2 + C_2e^{-\mu \tau} H_3 + C_3e^{-\mu \tau}]^T$, and as $t \to \infty$, all solutions move toward the disease-free equilibrium point $y_0 = (H_1 H_2 H_3)^T$, hence, it is globally asymptotically stable in the disease-free system. Now for the Lyapunov function $Q$, assume that $Q' = 0$. Since $Q' = (\lambda^*_p - 1)w^T x - w^T V^{-1} f(x,y)$, this implies $(\lambda^*_p - 1)w^T x = w^T V^{-1} f(x,y)$. But since $\lambda^*_p < 1$, we can see that $(\lambda^*_p - 1)w^T x \leq 0$, and by our assumptions we have $w^T V^{-1} f(x,y) \geq 0$. Then, we conclude that $x = 0$. Therefore, the set of all points where $Q' = 0$ is $E = \{ \{I_i, B_i, S_i\} : I_i = B_i = 0\}$, and as shown above, the largest (and only) invariant set in $E$ is the equilibrium $S_i = H_i$, which we know is globally asymptotically stable in the disease-free system. Applying LaSalle’s invariance principle, then we can state that the disease-free equilibrium is globally asymptotically stable in $\Omega$.

Now we establish existence of an endemic equilibrium of the model.

Theorem 3.4. Let $\Omega$ be a compact invariant set containing the disease-free equilibrium point $P_0 = (0, 0, S)$ of the system (1). Let $F$, $V$, and $f(x,y)$ be defined as above. If $\lambda^*_p(V^{-1}F) > 1$, then the system has an endemic equilibrium.

Proof. By Proposition 1, the condition $\lambda^*_p(V^{-1}F) > 1$ is equivalent to having $\lambda^*(J^*) > 0$ which implies that the disease-free equilibrium point $P_0$ is unstable, so that solutions starting on the positive cone close to $P_0$ move away from $P_0$, which is equivalent to uniform persistence of (1) (see Theorem 4.3 in [4], where $P_0$ plays the role of the invariant set $N$ in that theorem). Therefore, D.3 in [13] tells us that the invariance of $\Omega$ and the uniform persistence imply the existence of the endemic equilibrium.

It is worth noting that the condition $f(x,y_0) = 0$ is not necessarily true for this model. Such condition can sometimes be used in related models (see [11]) to prove that $P_0$ is unstable. Here we rely on Proposition 1.

3.3. Local stability of endemic equilibrium. The existence of an endemic equilibrium of system (1) is established in Theorem 3.4 for the case $n = 3$, and as remarked in the next section, this result extends to the case of an arbitrary $n \geq 1$. Clearly, for arbitrary values of $n$, explicit expressions for the endemic equilibrium or for the eigenvalues of the Jacobian are not feasible anymore. Let us however focus on the case $n = 3$. In such a case, one can see a particular structure on the (rescaled) Jacobian $J$ at the endemic equilibrium $(S^*_i, I^*_i, B^*_i)$, $i = 1, 2, 3$. Indeed,
\[ J \] is given by

\[
\begin{pmatrix}
-(\mu + d_1) & 0 & 0 & 0 & 0 & -\frac{m_3 p_3 Q_{13} S_i^*}{(1 + B_3^* )^2} & 0 \\
0 & -(\mu + d_2) & 0 & 0 & 0 & -\frac{m_2 p_2 Q_{12} S_i^*}{(1 + B_2^* )^2} & 0 \\
0 & 0 & -(\mu + d_3) & 0 & 0 & -\frac{m_1 p_1 Q_{11} S_i^*}{(1 + B_1^* )^2} & 0 \\
0 & d_1 & 0 & 0 & -\phi & 0 & 0 \\
0 & d_2 & 0 & 0 & -\phi & 0 & 0 \\
0 & d_3 & 0 & 0 & -\phi & 0 & 0 \\
0 & 0 & 0 & \tilde{c}_1 & c_1 Q_{21} & c_1 Q_{31} & - (\mu B + r) \\
0 & 0 & 0 & c_2 Q_{12} & \tilde{c}_2 & c_2 Q_{32} & r P \frac{W_1}{W_2} \\
0 & 0 & 0 & c_3 Q_{13} & c_3 Q_{23} & \tilde{c}_3 & r P \frac{W_1}{W_3} \\
\end{pmatrix}
\]

where all diagonal entries are negative, and \((i = 1, 2, 3)\).

\[ c_i = \frac{p_i m_i}{W_i K}, \quad \tilde{c}_i = \frac{p_i (1 - m_i) }{w_i K}, \quad d_i = (1 - m_S) \tilde{b}_i \tilde{f}(B_i^*) + m_S \sum_{j=1}^{3} Q_{ij} \tilde{b}_j \tilde{f}(B_j^*), \]

with \( \tilde{f}(B) = B/(1 + B) \). Observe that we can write this Jacobian in block form similar to that in (2). Indeed, the blocks \( J_{22}, J_{32} \) and \( J_{33} \) are exactly the same. We also have

\[
J_{11} = -\mu U_3 - D, \quad J_{13} = -J_{23}, \\
J_{21} = D, \quad J_{23} = [m_S S^* Q \beta + (1 - m_S) S^* \beta] C^{-1},
\]

where \( C \) and \( D \) are diagonal matrices with the obvious entries. A sufficient condition for a matrix \( A_{n \times n} \) to be Hurwitz (all eigenvalues have negative real part) is that it is row diagonally dominant \([1]\). That is, we need

\[
J_{ii} + \sum_{j=1, j \neq i}^{n} |J_{ij}| < 0,
\]

for all \( i = 1, \ldots, n \). Let us now denote

\[
d_i^* = \left( (1 - m_S) \tilde{b}_i g(B_i^*) + m_S \sum_{j=1}^{3} Q_{ij} \tilde{b}_j g(B_j^*) \right) S_i^*,
\]

with \( g(B) = 1/(1 + B)^2 \). In the notation below we still consider the system (1) with the usual rescaling. Then, since the endemic equilibrium \((S_i^*, I_i^*, B_i^*)\) satisfies the three equations \( I_i' = 0 \), so that we must have

\[
\phi = \frac{d_1 S_1^*}{I_1^*} = \frac{d_2 S_2^*}{I_2^*} = \frac{d_3 S_3^*}{I_3^*}.
\]

Also, from the three equations \( B_i^* = 0 \) we have

\[
\mu B + r = \frac{1}{p_1}(I_1^* a_4 + I_1^* a_5 + I_1^* a_6 + r B_2^* a_2 + r B_3^* a_3) \\
= \frac{1}{p_2}(I_2^* b_4 + I_2^* b_5 + I_2^* b_6 + r B_1^* b_1 + r B_3^* b_3) \\
= \frac{1}{p_3}(I_3^* c_4 + I_3^* c_5 + I_3^* c_6 + r B_1^* c_1 + r B_2^* c_2),
\]

where \( I_i^*, B_i^* \) are the endemic equilibrium values.
where $a_i, b_i, c_i > 0$, for $i = 2, \ldots, 6$.

With the notation above we can establish the following theorem, where the assumptions indicate that at endemic equilibrium, the state variables must satisfy certain conditions. For instance, relatively large bacteria concentration as well as large enough population of susceptibles within each community, and pathogens moving at a high enough rate between nodes.

**Theorem 3.5.** Consider the assumptions of Theorem 3.4, and let $d_i > d_i^*$. Further, assume that $\min \left\{ \frac{S_i^*}{T_i^*} \right\} > 1$, and $\min \left\{ \left( \frac{S_i}{T_i^*} \right)_j, \left( \frac{B_i}{T_i^*} \right)_j \right\} > 1$, for $i, j = 1, 2, 3$. Then, the endemic equilibrium of (1) for $n = 3$ is asymptotically stable.

**Proof.** This first inequality in the assumptions immediately implies that (12) is satisfied for $i = 1, 2, 3$. The condition $\min \{ \frac{S_i^*}{T_i^*} \} > 1$ implies that $\phi > \max \limits_i d_i$. This along with first inequality in the assumptions imply that (12) is also satisfied for $i = 4, 5, 6$. Finally, the last inequality in the assumptions clearly implies that (12) is true for $i = 7, 8, 9$. Then, the Jacobian at the endemic equilibrium is row diagonally dominant, and therefore all eigenvalues have negative real part.

**3.4. Bifurcations.** As we have seen above, the disease-free equilibrium loses stability when $\det(J) = 0$. Indeed, the simple eigenvalue of $J^*$ with maximum real part equals zero at that moment, and if a bifurcation is to occur, it should be a transcritical bifurcation. To prove existence of such bifurcation, we have the following result.

**Theorem 3.6.** The system (1) with $n = 3$ undergoes a transcritical bifurcation at the disease-free equilibrium point, under the parameter $p_1$.

**Proof.** Denote with $f$ the vector field of (1), let $P_0$ be the disease-free equilibrium point, and $p_0^1$ a value of the parameter $p_1$ so that $f(P_0, p_0^1) = 0$. Let $v$ be the right eigenvector of $Df(P_0, p_0^1)$, and let $w$ be its left eigenvector. Then, it is straightforward to see that $w^T f_{p_1}(P_0, p_0^1) = 0$. One can also see that

$$w^T [Df_{p_1}(P_0, p_0^1)] w = \frac{H_1}{\phi} [(1 - m_s) \beta_1 + m_s Q_{12} \beta_2 + m_s Q_{13} \beta_3] (1 - m_i) W_i - 1 \quad + \frac{H_2}{\phi} [m_s Q_{21} \beta_1 + (1 - m_s) \beta_2 + m_s Q_{23} \beta_3] m_i Q_{21} W_1 - 1 \quad + \frac{H_3}{\phi} [m_s Q_{31} \beta_1 + m_s Q_{32} \beta_2 + (1 - m_s) \beta_3] m_i Q_{31} W_1 - 1 > 0.$$ 

One can verify that $v_1, v_2, v_3 < 0$ and also $w_4, w_5, w_6 < 0$. Then, we get that

$$w^T [D^2 f(P_0, p_0^1)](w, v) = w_4 (2(1 - m_s) \beta_1 v_1 + 2 m_s Q_{13} \beta_2 v_1 + 2 m_s Q_{13} \beta_3 v_1) \quad - 2 H_1 (1 - m_i) \beta_1 - 2 H_1 m_s Q_{12} \beta_2 - 2 H_1 m_s Q_{13} \beta_3 \quad + w_5 (2 m_s Q_{21} \beta_1 v_2 + 2 (1 - m_s) \beta_2 v_2 + 2 m_s Q_{23} \beta_3 v_2) \quad - 2 H_2 m_s Q_{21} \beta_1 - 2 H_2 (1 - m_i) \beta_2 - 2 H_2 m_s Q_{23} \beta_3 \quad + w_6 (2 m_s Q_{31} \beta_1 v_3 + 2 m_s Q_{32} \beta_2 v_3 + 2 (1 - m_s) \beta_3) \quad - 2 H_3 m_s Q_{31} \beta_1 - 2 H_3 m_s Q_{32} \beta_2 - 2 H_3 (1 - m_i) \beta_3 \quad > 0.$$ 

Thus, by Sotomayor’s Theorem, the existence of a transcritical bifurcation has been established.

The phase portraits in Figures 1 and 2 illustrate the cases $p_1 < p_0^1$ and $p_1 > p_0^1$ respectively, with $p_1 = 0.1834548$. Keep in mind that the figures shown are projections from $\mathbb{R}^n$ onto $(S_1, I_1, B_1)$, and that on Figure 1, the endemic equilibrium is not present. Instead, in Figure 2 both the disease-free and the endemic equilibrium
are present. In this case the disease free equilibrium is unstable, and the endemic equilibrium is stable.

4. **n number of communities.** Now we can easily generalize all these ideas and results on local and global stability analysis as well as the existence of a transcritical bifurcation to the case of an arbitrary \(n\) number of communities. That is, with the obvious modifications on the dimensions of the vectors, matrices and functions involved in the calculations, the results of Section 3 also apply to the general system (1) of \(3n\) differential equations, and the corresponding proofs are natural extensions of those in the case \(n = 3\) to an arbitrary integer \(n \geq 1\). In particular, the disease-free equilibrium \(x_0 = (H_1, H_2, ..., H_n, 0, ..., 0)\) is \(3n\)-dimensional, and the Jacobian at such point is

\[
J(x_0) = \begin{bmatrix}
J_{11} & 0 & J_{13} \\
0 & J_{22} & J_{23} \\
0 & J_{32} & J_{33}
\end{bmatrix},
\]

where each \(J_{ij}\) block is an \(n \times n\) matrix, and the corresponding blocks \(J_{ij}\) are defined as in Section 3 but now each of them is an \(n \times n\) matrix. Due to the same block structure of \(J\), it is evident that the stability again depends on the eigenvalues.
of the bottom right $2n \times 2n$ matrix,
\[
J^* = \begin{bmatrix}
J_{22} & J_{23} \\
J_{32} & J_{33}
\end{bmatrix},
\]
which is irreducible and Metzler. Thus, the same conclusions that were made for the case of $n = 3$ apply to the general $n$ scenario, including writing the general system in compartmental form as in (6), and defining the corresponding Lyapunov function as $Q = w^T V^{-1} x$. For the endemic equilibrium, a similar blocking of the Jacobian is done, and the definitions in (11) naturally extend for arbitrary $n$. More specifically, below we summarize the main results

1. The disease-free equilibrium loses stability when $\lambda^*(J^*)$ crosses zero.
2. The condition $\det(J^*) = 0$ is equivalent to $\det(I - G_0) = 0$.
3. $\lambda = 1$ is the dominant eigenvalue of $G_0$.
4. If $\lambda^*_F(V^{-1} F) \leq 1$, then $Q = w^T V^{-1} x$ is a Lyapunov function satisfying $Q' \leq 0$.
5. If $\lambda^*_F(V^{-1} F) < 1$ and the disease-free equilibrium is globally asymptotically stable in the disease-free system, then it is a globally asymptotically stable equilibrium of the general system (1).
6. If $\lambda^*_F(V^{-1} F) > 1$, then the system (1) has at least one endemic equilibrium.
7. Under some conditions, the endemic equilibrium is locally asymptotically stable.
8. The system (1) undergoes a transcritical bifurcation at the disease-free equilibrium.

5. **Final remarks.** A model of waterborne disease that considers a number $n \geq 1$ of communities connected via human mobility and hydrological networks has been considered in this work. The model was introduced by Gatto et al. in [5]. For clarity and illustration purposes, we gave details for the case $n = 3$ but the results presented here extend to an arbitrary $n \geq 1$. We in part extended the work in [5] (and related articles, including [15]), by rigorously establishing global stability of the disease-free equilibrium via the introduction of an appropriate Lyapunov function, by proving existence of an endemic equilibrium and its local stability properties, and by establishing existence of a transcritical bifurcation. The condition for $n = 1$ that the basic reproduction number satisfies $R_0 > 1$ has been extended to $\lambda^*_F(V^{-1} F) > 1$ for the general case of an arbitrary number $n$ of communities.

This work can lead to some generalizations and further avenues of research, including the ones suggested in [5] such as considering age-structured populations or competition between pathogen strains. One could also explicitly include water treatment or sanitation and vaccination within the model. In some cases diseases are seasonal and we expect that including this periodicity in the model may generate some periodic solutions. Establishing the possible existence or nonexistence of other bifurcations, as well as results on global stability of the endemic equilibrium are also worth studying.

**Acknowledgments.** We would like to thank the anonymous referees for their valuable suggestions and comments which led to the improvement of this article.

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Received April 2015; revised September 2016.

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