Host switching vs. host sharing in overlapping sylvatic 
Trypanosoma cruzi transmission cycles

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ABSTRACT
The principle of competitive exclusion is well established for multiple populations competing for the same resource, and simple models for multistrain infection exhibit it as well when cross-immunity precludes coinfections. However, multiple hosts provide niches for different pathogens to occupy simultaneously. This is the case for the vector-borne parasite Trypanosoma cruzi in overlapping sylvatic transmission cycles in the Americas, where it is enzootic. This study uses cycles in the USA involving two different hosts but the same vector species as a context for the study of the mechanisms behind the communication between the two cycles. Vectors dispersing in search of new hosts may be considered to move between the two cycles (host switching) or, more simply, to divide their time between the two host types (host sharing). Analysis considers host switching as an intermediate case between isolated cycles and intermingled cycles (host sharing) in order to examine the role played by the host-switching rate in permitting coexistence of multiple strains in a single-host population. Results show that although the population dynamics (demographic equilibria) in host-switching models align well with those in the limiting models (host sharing or isolated cycles), infection dynamics differ significantly, in ways that sometimes illuminate the underlying epidemiology (such as differing host susceptibilities to infection) and sometimes reveal model limitations (such as host switching dominating the infection dynamics). Numerical work suggests that the model explains the trace presence of TcI in raccoons but not the more significant co-persistence observed in woodrats.

1. Introduction
Chagas’ disease, caused by the protozoan parasite Trypanosoma cruzi, is a vector-borne disease of growing concern in the Americas [15]. T. cruzi is enzootic in host–vector cycles throughout the Americas as far north as the USA. These transmission cycles involve over 100 mammalian species and dozens of triatomine vector species. Though millions of people are infected throughout Latin America, it is the sylvatic cycles which maintain the parasite, with infected vectors moving to human habitations in search of new bloodmeal sources. Some vector species, such as Triatoma infestans, have adapted nearly exclusively...
to domestic settings (where there is some evidence for independent transmission cycles), while others such as *Triatoma dimidiata* disperse continually in waves towards domestic sites [16]. In the USA, the disease is little known, with few documented autochthonous human cases, but the parasite remains highly endemic (and little studied) in sylvatic cycles, prevalent at over 50% in some sylvatic US hosts.

Two primary hosts are found in the southeastern USA, raccoons (*Procyon lotor*) and Virginia opossums (*Didelphis virginiana*). These hosts share a common vector, *Triatoma sanguisuga*. In Texas and northern Mexico, the dominant species of vectors is *Triatoma gerstaeckeri*, associated with the southern plains woodrat (*Neotoma micropus*) [19]. There are six different recognized strains of the parasite, I–VI (formerly I and IIa–e), classified by phylogenetic lineage. While all six types are found in South America, only types I and IV (formerly IIa) are known to circulate in the USA [42]. Type I, associated with Chagas' disease and human infections, is the only type found in Virginia opossums, which have proven immune to type IV [44,52]. Raccoons, as well as other US hosts, are instead associated with *T. cruzi* IV [18,42]. Most recently, both strains have been found in Texas woodrats [5]. Infection with a given strain of *T. cruzi* has been observed [27,33] to confer immunity against infection by other strains (precluding coinfection or superinfection).

The traditional means of infection with *T. cruzi* involves the vector feeding on a host. Vectors become infected via a bloodmeal on an infected host; then, the parasite reproduces in the vector's gut. Typically, hosts become infected through stercorarian transmission; the vector defecates near the feeding site shortly after feeding, and the host scratches the bite, rubbing the parasite into the wound. Vertical (congenital) transmission, which has been observed in humans as well as laboratory rats [32,45], may also be significant among other placental hosts (but not in marsupials such as opossums). Host consumption of infected vectors may be the dominant infection pathway in some cycles, more likely among raccoons than among opossums [4,25,36,39,41,43,51]. Oral transmission is a risky adaption in evolutionary terms (theoretically speaking, although a published estimate of *T. cruzi* IV's oral transmissibility exceeds those for *T. cruzi* VI by an order of magnitude, and some trypanosomes have been observed to affect vector behaviour in ways that increase host–vector contact [23, p. 4]) since the consumed vector can infect at most one host this way, as opposed to potentially many via stercorarian transmission, but as shown in [24] it can, under some conditions, maintain sylvatic cycles alone. Indeed, it may not be any riskier than stercorarian transmission, which by its nature is less efficient than the more classical vector-borne transmission seen in mosquito-transmitted diseases, where vector feeding transmits the parasite directly into the host from the vector's salivary glands (per-bite transmission probabilities to humans, for instance, have been estimated at around 0.058% for *T. cruzi* [34], compared to around 55% for malaria [48], a difference of three orders of magnitude).

The single-host model of Kribs-Zaleta and Mubayi [3,17,29] predicts competitive exclusion (consistent with other studies of populations at equilibrium [17,29]); in order to address reports of both strains being endemic in woodrats at roughly equal levels [5] and trace prevalence of *T. cruzi* I observed in raccoons [42], this study extends the modelling framework of Kribs-Zaleta and Mubayi [26] to a multihost scenario in which transmission cycles communicate via local vector dispersal. Triatomines such as *T. sanguisuga* typically remain in a single nest or den, feeding on a host when it returns to sleep, until either the host dies or fails to return, or else the nest is saturated with vectors, and the triatomine
is obliged to seek another host. The search for, and dispersal to, another host den [28] is called host switching, and for some host species may occur relatively infrequently, since the primary host returns regularly to a fixed number of sleeping places. In host switching, vectors seek the nearest available host, which may be of the same or a different species. Raccoons and opossums both typically sleep alone in tree hollows or in crevices or burrows (dug by other animals) in the ground, although when the need arises they can also sleep in dense underbrush, culverts, or other covered areas inaccessible to potential predators. Different animals sometimes use the same den, not necessarily at the same time, so that vectors in some locations may have the chance to feed on raccoons, opossums, armadillos, skunks, and more without dispersing [46]. There are also instances, however, when a given sleeping place may go unused for long periods of time, and studies of even occupied woodrat nests have shown a high proportion of adult vectors well outside the nest, for example, [37].

In describing a local population of triatomines collectively, therefore, the issue arises of whether to consider them a single population or several. A detailed model might use a nest (with a group of vectors and one or two hosts) as a population unit, but a simpler model considering each host species as a population could consider the local vector population either as a single group splitting their time (contacts) among host species (host sharing) in proportion to each species’ capacity to support vectors – including the scenario where different hosts use the same den on different nights – or as separate populations, each affiliated with a single host type, with individual vectors moving from one host-affiliated group to another at a rate that measures the frequency of host switching – as when a given den or nest is used by a single host which returns only periodically. Modeling studies for other multihost vector infections have typically used a host-sharing structure to capture dynamics where individual vectors (such as mosquitoes) frequently change host type at each feeding, generating reproductive numbers which are weighted averages of the reproductive numbers for each host, weighted by relative sizes of the host populations and the vectors’ preference for each host type (e.g. mosquitoes and West Nile Virus [11,47]). Structures corresponding to host switching are seen more commonly in metapopulation models, with vector dispersal analogous to host switching, for example, [10].

This study investigates both types of structure, where host sharing may serve as a limiting case in which host switching occurs ‘infinitely often.’ (Some studies have considered simple epidemiological models as limiting cases of more complex ones, with equilibria and threshold quantities reducing to those of the simpler model, for example, [30,31]. The limiting behaviour in the present study shall prove more complex.) We use this framework to address two main research questions, one primarily biological and the other primarily mathematical: First, to what extent can host switching by vectors explain the observed co-persistence of two *T. cruzi* strains in some North American transmission cycles? (In particular, we take into account the asymmetry created by opossums’ immunity to *T. cruzi* IV and to vertical transmission.) Second, does it matter which way vectors’ host-switching behaviour is modelled? Both questions hinge on the frequency of host switching as the means for communication between cycles where infection dynamics differ significantly between hosts. The following sections present both models in parallel and then compare the population dynamics followed by the infection dynamics. The article concludes with a discussion of the role of host-switching rates in the ongoing interstrain competition in *T. cruzi* transmission cycles.
2. Model development

The single-host model of Kribs-Zaleta and Mubayi [26] incorporates stercorarian, oral, and vertical transmission to hosts, and bloodmeal transmission to vectors. Both types of host–vector contacts (each species feeding on the other) were modeled with rates that saturated in the vector–host density ratio $Q$ via a Holling type I function (piecewise linear) [1,20–22]. The two parasite strains were assumed to differ in their various transmissibilities to hosts, but to infect vectors at the same rate (for a given host species). Analysis showed competitive exclusion between the two strains in a transmission cycle with one host and one vector population, the outcome determined by standard threshold quantities: the two strains’ basic reproductive numbers (BRNs) and invasion reproductive numbers (IRNs). Competition only occurs if both strains’ BRNs exceed 1 (which appears to be true from observed field data), in which case only one of the two IRNs can exceed 1. We now extend this model to incorporate a second transmission cycle communicating with the first through the vectors, in two different ways.

2.1. Host-switching model

To describe the host (type) switching process, one may use a metapopulation model involving two separate host–vector transmission cycles, coupled by dispersal-generated migration between the two vector populations. (Henceforth, we distinguish between dispersal – the local, small-scale movement of individuals – and migration – the resulting large-scale shift between two populations [9].) This connected two-cycle structure is similar to that for a two-patch model, but here the second ‘patch’ may involve a different host species in the same overall habitat as the first. Because vectors live in their hosts’ sleeping places, these cycles can be considered separate even if the overall geographical area (and even the set of dens) is the same.

A general compartmental model describing host switching of vectors between the two host populations, as well as the transmission dynamics of two strains, would involve three compartments (susceptible or infected with one of two strains) for each host as well as for its accompanying vector population, with the two vector populations connected by host-switching (dispersal) rates (here assumed independent of infection status). As an initial study, however, this article considers the special case of interest in which the hosts are raccoons and opossums, linked by the common vector species $T. sanguisuga$, so that the second cycle exhibits structural characteristics particular to opossums, notably their immunity to infection by $T. cruzi$ IV (henceforth, strain 2, with TcI as strain 1), as well as the lack of vertical transmission. (Vectors infected with strain 2 may switch hosts to opossums, but no new strain 2 infections of either species can occur there.) The rates at which vectors ‘migrate’ between host species reflect the host-switching rates (in fact, they are less than the rates at which vectors change individual hosts, since some host switches result in new hosts of the same species as the old) and are assumed constant (per capita). These rates take into account each host species's ability to withstand bites as well as real host loss rates and the probability of finding a new host.

The model resulting from these assumptions is depicted in Figure 1 and by the equations below. Subscripts of $o$ and $r$ for opossum and raccoon replace $h$ for host, with subscripts of $vo$ and $vr$ to denote vectors affiliated with each host type. Each of the two contact
processes (host predation and vector bloodmeals) is a function of the vector–host ratios $Q_o = N_{vo}/N_o$ and $Q_r = N_{vr}/N_r$. The ‘migration’ rates from each host type are denoted by $m_o$ and $m_r$. To facilitate analysis, equations are given for total densities, with $S_k$ defined as $N_k - I_k1 - I_k2$ ($k = o, r, vo, vr$). The vector populations $N_{vo}$ and $N_{vr}$ are state variables with birth rate(s) $b(N_{vo})$ and $b(N_{vr})$.

\[
\begin{align*}
I'_{o1} &= [c_{o1}(Q_o) + \rho_{o1}E_o(Q_o)]S_o \frac{I_{vo1}}{N_{vo}} - \mu_o I_{o1}, \\
I'_{vo1} &= c_{vo1}(Q_o)S_{vo} \frac{I_{o1}}{N_{vo}} - \mu_v I_{vo1} - E_o(Q_o)N_o \frac{I_{vo1}}{N_{vo}} - m_o I_{vo1} + m_r I_{vr1}, \\
I'_{r1} &= p_1 \frac{I_{r1}}{N_r}b_r(N_r) + [c_{r1}(Q_r) + \rho_{r1}E_r(Q_r)]S_r \frac{I_{vr1}}{N_{vt}} - \mu_v I_{r1}, \\
I'_{vr1} &= c_{vr1}(Q_r)S_{vr} \frac{I_{r1}}{N_r} - \mu_v I_{vr1} - E_r(Q_r)N_r \frac{I_{vr1}}{N_{vt}} - m_r I_{vr1} + m_o I_{vo1}, \\
I'_{vo2} &= -\mu_v I_{vo2} - E_o(Q_o)N_o \frac{I_{vo2}}{N_{vo}} - m_o I_{vo2} + m_r I_{vr2}, \\
I'_{r2} &= p_2 \frac{I_{r2}}{N_r}b_r(N_r) + [c_{r2}(Q_r) + \rho_{r2}E_r(Q_r)]S_r \frac{I_{vr2}}{N_{vt}} - \mu_v I_{r2}, \\
I'_{vr2} &= c_{vr2}(Q_r)S_{vr} \frac{I_{r2}}{N_r} - \mu_v I_{vr2} - E_r(Q_r)N_r \frac{I_{vr2}}{N_{vt}} - m_r I_{vr2} + m_o I_{vo2}, \\
N'_o &= b_o(N_o) - \mu_o N_o, \\
N'_r &= b_r(N_r) - \mu_v N_r, \\
N'_{vo} &= b_v(N_{vo}) - (\mu_v + m_o)N_{vo} - E_o(Q_o)N_o + m_r N_{vr}, \\
N'_{vr} &= b_v(N_{vr}) - (\mu_v + m_r)N_{vr} - E_r(Q_r)N_r + m_o N_{vo}.
\end{align*}
\]
As in [26], the host–vector contact rates (for stercorarian infection, infection of vectors, and host predation on vectors, respectively) are given by

\[ c_{ji}(Q_j) = \beta_{ji} \min(Q_j/Q_{vj}, 1), \]

\[ c_{vji}(Q_j) = \beta_{vji} \min\left(\frac{1/Q_j}{1/Q_{vj}}, 1\right) = \beta_{vji} \min\left(\frac{Q_{vj}}{Q_j}, 1\right), \]

\[ E_j(Q_j) = H_j \min\left(\frac{Q_j}{Q_{hij}}, 1\right), \]

where \( j \) is \( o \) or \( r \) for host type and \( i = 1, 2 \) for strain, and \( Q_{hij} \) and \( Q_{vj} \) are the saturation threshold levels for host predation and vector bloodmeals, respectively. Variables, notation, and parameters are summarized in Tables 1 and 2, with baseline parameter estimates taken from [23,26].

**Table 1.** Variables and notation for sylvatic \emph{T. cruzi} transmission models \((j = o, r, i = 1, 2)\).

| Var.       | Meaning                                      | Units                |
|------------|----------------------------------------------|----------------------|
| \(S_j(t)\) | Density of uninfected hosts of type \(j\)     | hosts/area           |
| \(I_{ji}(t)\) | Density of host type \(j\) infected with strain \(i\) | hosts/area           |
| \(S_{vj}(t)\) | Density of uninf. vectors affil. with host type \(j\) | vectors/area         |
| \(I_{vji}(t)\) | Density of vectors affiliated with host type \(j\) and infected with strain \(i\) | vectors/area         |
| \(Q_j\) | Vector–host population density ratio \(\left(N_{vj}/N_j\right)\) | vectors/host         |
| \(c_{ji}(Q_j)\) | Strain \(i\) stercorarian infection rate of host \(j\) | 1/time               |
| \(c_{vji}(Q_j)\) | Strain \(i\) vector infection rate from host \(j\) | 1/time               |
| \(E_j(Q_j)\) | Per-host predation rate                      | vectors/host/time    |

**Table 2.** Parameter definitions and estimates (from [23,26]) for sylvatic \emph{T. cruzi} cycles: raccoons and \emph{T. sanguisuga} (R/S) \([j = r, v = s]\), opossums and \emph{T. sanguisuga} (O/S) \([j = o, v = s]\).

| Parm. | Definition                                      | Units | R/S | O/S |
|-------|------------------------------------------------|-------|-----|-----|
| \(\mu_j\) | Natural mortality rate of hosts                 | per year | 0.40 | 0.83 |
| \(\mu_v\) | Natural mortality rate of vectors               | per year | 0.271 | 0.271 |
| \(N_j^*\) | (Equilibrium) host population density          | hosts/acre | 0.080 | 0.041 |
| \(N_v^*\) | (Equilibrium) vector pop. density\(^a\)       | vectors/acre | 128 | 128 |
| \(Q_{hij}\) | Threshold vector–host density ratio for predation | vectors/host | 10 | 10 |
| \(Q_{vij}\) | Threshold vector–host density ratio for bloodmeals | vectors/host | 100 | 100 |
| \(\beta_{vij}\) | Vector infection rate                           | per year | 9.67 | 13.4 |
| \(\beta_{1j}\) | Stercorarian infection rate, strain 1          | per year | 0.394 | 0.394 |
| \(\beta_{2j}\) | Stercorarian infection rate, strain 2          | per year | 0.225 | 0 |
| \(p_j\) | Vertical xmsn proportion, strain 1             | dimensionless | 0.05 | 0 |
| \(p_{2j}\) | Vertical xmsn proportion, strain 2             | dimensionless | 0.1 | 0 |
| \(p_{ij}\) | Estimated proportion of hosts infected after consuming an infected vector | hosts per vector | 0.177 | 0.177 |
| \(H_j\) | (Maximum) per-host predation rate              | vectors/host/yr | 1 | 1 |
| \(\Lambda_{vij}\) | Vector birth/recruitment rate associated with host type \(j\) | vectors/year | 1 | 1 |
| \(m_j\) | Vector migration rate leaving host \(j\)       | per year | | |

\(^a\) The only published vector density estimate comes from a cycle involving woodrats, cf. [23].
2.2. Host-sharing model

An alternative formulation for describing such interacting transmission cycles is a host-sharing model, which instead considers all vectors in a given region as a single population, some of which feed on raccoons while others feed on opossums. This model, in which vectors contact each host type a certain proportion of the time, may be a better intuitive match for scenarios where vectors frequently have access to multiple host types in the same vicinity. We can derive such a model directly from this simple principle (the flow is illustrated by Figure 2), but in order to highlight the relationship between the host-sharing and host-switching models will instead derive it as a limiting case of the host-switching model in which host switching occurs ‘infinitely often’.

To consider the vectors as a single population, we define the state variables

\[ S_v(t) = S_{vo}(t) + S_{vr}(t), \]

\[ I_v^1(t) = I_{vo1}(t) + I_{vr1}(t), \]

\[ I_v^2(t) = I_{vo2}(t) + I_{vr2}(t) \]

for vector density by infection status, and

\[ N_v(t) = N_{vo}(t) + N_{vr}(t) \]

for total vector density. To formalize the notion of host sharing as a limiting case of host switching, one must establish a framework for scaling up the switching (migration) rates \( m_o \) and \( m_r \) in the host-switching model. To disentangle the differences in host biology and behaviour from the question of timescales, we can scale both switching rates by a factor of \( \phi \), preserving the relative magnitudes of \( m_o \) and \( m_r \); that is, to be explicit, \( m_o \) and \( m_r \) in the equations of system (1) are replaced, respectively, with \( \phi m_o \) and \( \phi m_r \), where \( \phi \) is a dimensionless scaling factor. As the timescale factor \( \phi \) increases, host switching occurs much faster (in both directions) than other changes in vector demographics, so that the switching rates drive the relative sizes of the two vector populations.

We consider first the vector density equations, which from system (1) become

\[
N_{vo}' = b_v(N_{vo}) - (\mu_v + \phi m_o)N_{vo} - E_o \left( \frac{N_{vo}}{N_o} \right) N_o + \phi m_r N_{vr},
\]

\[
N_{vr}' = b_v(N_{vr}) - (\mu_v + \phi m_r)N_{vr} - E_r \left( \frac{N_{vr}}{N_r} \right) N_r + \phi m_o N_{vo}.
\]

Figure 2. Flow chart for the host-sharing model, system (6). For space constraints, the following notation is used: \( \hat{\mu}_v = \mu_v + E_o(Q_o)/Q_o + E_r(Q_r)/Q_r \), \( P_i = p_i b_i(N_i)I_i/N_i \), \( C_{ji} = [c_{ji}(Q_j) + \rho_{ji}E_j(Q_j)]I_{ji}/N_v \), \( C_{vji} = c_{vji}(Q_j)k_{vji}/N_j \) and \( C_{vij} = C_{vio} + C_{vir} \) for \( j = o, r, i = 1, 2 \).
Since the equations for host densities $N_o(t)$ and $N_r(t)$ decouple from the rest of the system (and are unaffected by $\phi$), we consider them to have reached equilibrium (we address this more formally in the analysis in Appendix 1) and note that these Equations (3) also decouple from the infection dynamics. Since the $E_j$ predator functional responses defined in Equation (2) are either linear or constant, whenever the birth rate function $b_v$ is affine, say $b_v(N_{vj}) = \Lambda_{vj} + bN_{vj}$ (for appropriate nonnegative constants $\Lambda_{vj}$ and $b$), $j = o, r$, these equations are of the more general form

$$
X'_o = A_o - a_oX_o - \phi m_oX_o + \phi m_rX_r, \\
X'_r = A_r - a_rX_r - \phi m_rX_r + \phi m_oX_o,
$$

(4)

where $A_j$ and $a_j$ are appropriate nonnegative constants (which requires some reasonable assumptions on $\Lambda_{vj}$ and $b$ to ensure that, in the absence of host switching ($\phi = 0$), the vector population does not go extinct). This system has a unique equilibrium which is easily shown to be globally asymptotically stable (GAS) using standard methods,

$$
X^*_o = \frac{A_o a_r + \phi m_r(A_o + A_r)}{a_o a_r + \phi (m_r a_o + m_o a_r)}, \quad X^*_r = \frac{A_r a_o + \phi m_o(A_o + A_r)}{a_o a_r + \phi (m_r a_o + m_o a_r)}.
$$

We can now define constants $k_o = m_r/(m_o + m_r)$ and $k_r = m_o/(m_o + m_r)$ and rewrite this equilibrium, dividing through top and bottom of each fraction by $\phi(m_o + m_r)$:

$$
X^*_o = \frac{A_o a_r/\phi(m_o + m_r) + k_o(A_o + A_r)}{a_o a_r/\phi(m_o + m_r) + k_o a_o + k_r a_r}, \quad X^*_r = \frac{A_r a_o/\phi(m_o + m_r) + k_r(A_o + A_r)}{a_o a_r/\phi(m_o + m_r) + k_o a_o + k_r a_r}.
$$

Thus, if we define $X = X_o + X_r$ (corresponding here to $N_v = N_vo + N_vr$), we have

$$
\lim_{\phi \to \infty} X^*_o = \frac{A_o + A_r}{k_o a_o + k_r a_r}, \quad \lim_{\phi \to \infty} X^*_r = \frac{k_o X^*}{k_o a_o + k_r a_r}, \quad \lim_{\phi \to \infty} X^*_r = k_r X^*.
$$

and in the limiting-case model, $\lim_{t \to \infty} X(t) = X^*$, $\lim_{t \to \infty} X_o(t) = k_o X^*$, and $\lim_{t \to \infty} X_r(t) = k_r X^*$. This means that $k_o$ and $k_r$ represent, in the host-switching model, the respective proportions (at demographic equilibrium) of the total vector population associated with each host type (note $k_o + k_r = 1$), and in the limiting case – the host-sharing model – the respective proportions of host–vector contacts made with each host type.

Returning to the context of $N_v = N_vo + N_vr$, we now have that in the host-sharing model $N_vo \to k_oN_v$, $N_vr \to k_rN_v$; thus the overall vector density dynamics $N'_v = b_v(N_v) - \mu_vN_v - E_o(N_vo/N_o)N_o - E_r(N_vr/N_r)N_r$, eventually settles down to $N'_v = b_v(N_v) - \mu_vN_v - E_o(k_oN_vo/N_o)N_o - E_r(k_rN_vr/N_r)N_r$, and more generally if we assume the system to reach demographic equilibrium, we can take $N_{vj} = k_jN_v$, the ‘apparent’ vector density experienced by each host type based on the proportion of each vector’s contacts made with the given host type. For consistency in defining density ratios, for the host-sharing model, we therefore define $N_{vj} = k_jN_v$ and keep $Q_j = N_{vj}/N_j$ as de facto vector–host density ratios.
If we now consider the timescaled equations for vectors infected with strain 1,

\[ I'_{v1} = c_{v01}(Q_o)S_v N_o I_{v1} - \mu_v I_{v1} - E_o(Q_o)N_o I_{v1} + \phi m_o I_{v1} + \phi m_r I_{v1}, \]

\[ I'_{v1} = c_{v11}(Q_r)S_{v1} N_r I_{v1} - \mu_v I_{v1} - E_r(Q_r)N_r I_{v1} + \phi m_r I_{v1} + \phi m_o I_{v1}, \]

\[ I'_{v1} = c_{v01}(Q_o)S_v N_o I_{v1} + c_{v11}(Q_r)S_{v1} N_r I_{v1} - \mu_v I_{v1} - E_o(Q_o)N_o I_{v1} + \phi m_o I_{v1} + \phi m_r I_{v1}, \]

we can see that the first two fit the general form (4) as long as we hold the other compartments constant (incorporating them into the \( A_j \)) in order to examine the limiting effect of the timescale factor \( \phi \). The result is then that \( I'_{v01} \) and \( I'_{v1} \) approach \( k_o I_{v1} \) and \( k_r I_{v1} \), respectively. A similar result holds for \( I_{v02} \) and \( I_{v2} \) with regard to \( I_{v1} \), and finally (by subtraction and factoring) for \( S_v \) and \( S_{v1} \) with regard to \( S_{v2} \).

Now, finally, we can write the entire system for the host-sharing model, using \( k_j X \) in place of \( X_j \) (\( X = N_v, I_v, I_{v2}, S_v \)). The resulting model is described in Figure 2 and by the following system of equations:

\[ I'_{o1} = [c_{o1}(Q_o) + \rho_o E_o(Q_o)]S_o I_{o1} N_o - \mu_o I_{o1}, \]

\[ I'_{r1} = p_1 I_{r1} b_r(N_r) + [c_{r1}(Q_r) + \rho_r E_r(Q_r)]S_r I_{v1} N_r - \mu_r I_{r1}, \]

\[ I'_{v1} = c_{v01}(Q_o)k_o S_v I_{o1} N_o + c_{v11}(Q_r)k_r S_{v1} N_r I_{v1} - \mu_v I_{v1} - E_o(Q_o)N_o I_{v1} N_o - E_r(Q_r)N_r I_{v1} N_r, \]

\[ I'_{v2} = p_2 I_{r2} b_r(N_r) + [c_{r2}(Q_r) + \rho_r E_r(Q_r)]S_r I_{v2} N_r - \mu_r I_{v2}, \]

\[ N'_{o1} = b_o(N_o) - \mu_o N_o, \]

\[ N'_{r1} = b_r(N_r) - \mu_r N_r, \]

\[ N'_{v1} = b_v(N_v) - \mu_v N_v - E_o(Q_o)N_o - E_r(Q_r)N_r. \]

### 3. Comparative analysis

To address the research questions articulated in Section 1, it is helpful first to understand the behaviours exhibited by the two models, and in particular the extent to which the host-sharing model’s behaviour can be viewed as a limiting case of the host-switching model’s. In general, solutions of systems (1) and (6) approach GAS equilibria in accordance with standard threshold results using reproductive numbers, but the values of those reproductive numbers, and the possibility for co-persistence of the two strains, depends on model parameters in often complicated ways. The use of Holling Type I saturation to model host–vector contact rates (2) also makes computations lengthy, by subdividing into four cases depending on which (if either) of the two contact processes have saturated. We therefore relegate to the appendices the baseline computations for the two models, and
here focus on the comparison of the two sets of results, using at each stage the timescale approach of Section 2.1 via the dimensionless scale factor $\phi$.

### 3.1. Population dynamics

In both systems (1) and (6), the host and vector population density dynamics decouple from the infection dynamics and can be analysed separately. The details (see Appendix 1) are simple for host densities but more complicated for vector densities, due in large part to the form of the contact process saturation, but result in both densities approaching a single, GAS equilibrium level, completely independently of the infection dynamics (since these strains of *T. cruzi* are assumed to cause no additional mortality in vectors and primary hosts).

Study of both systems decomposes into four cases, based on the two vector–host ratios and their threshold values ($Q_{ho}$ and $Q_{hr}$) for the predation contact processes: (I) $Q_o < Q_{ho}$ and $Q_r < Q_{hr}$ (i.e. $N_{vo} < N_oQ_{ho}$, $N_{vt} < N_rQ_{hr}$); (II) $Q_o > Q_{ho}$ and $Q_r > Q_{hr}$ (i.e. $N_{vo} > N_oQ_{ho}$, $N_{vt} > N_rQ_{hr}$) (IIa) $Q_o > Q_{ho}$ but $Q_r < Q_{hr}$ (i.e. $N_{vo} > N_oQ_{ho}$, $N_{vt} < N_rQ_{hr}$); and (IIb) $Q_o < Q_{ho}$ but $Q_r > Q_{hr}$ (i.e. $N_{vo} < N_oQ_{ho}$, $N_{vt} > N_rQ_{hr}$). The corresponding equilibrium values, given in Appendix 1, can be used to gauge the limiting behaviour of the host-switching model (1) for both extremes of the timescale factor $\phi$ in Equations (3): as $\phi \to 0$, do the vector densities approach those of the isolated single-host cycles (depicted in [26])? and as $\phi \to \infty$, do they approach those of the host-sharing model (6)?

In order to compare the host-switching model with both the single-host and the host-sharing models, we use Equations (3) where the time-scaling factor $\phi$ is associated with both $m_o$ and $m_r$. We take the resulting equilibrium densities (cf. Table A1 in Appendix 1) and likewise multiply $m_o$ and $m_r$ by $\phi$ in each. For comparison to the single-host model of Kribs-Zaleta and Mubayi [26], we take the limit of each equilibrium as $\phi$ goes to 0. The results are given in Table 3, alongside the corresponding equilibria for the respective

| Case | Equilibrium host switching | Limiting values as $\phi \to 0$ | Equilibrium single host |
|------|---------------------------|----------------------------------|-------------------------|
| I    | $N_{vo}^*(\phi) = \frac{(\Lambda_{vo} + \Lambda_v)\mu_v + \Lambda_{vo}\mu_{vt}}{\mu_v + \frac{H_v}{Q_{vo}}} + \phi m_o \mu_v + \phi m_o \mu_{vt}$ | $N_{vo}^* = \frac{\Lambda_{vo}}{\mu_v + \frac{H_v}{Q_{vo}}}$ | $N_{vo}^* = \frac{\Lambda_v}{\mu_v}$ |
| II   | $N_{vo}^*(\phi) = \frac{(\Lambda_{vo} + \Lambda_v)\mu_v + \phi m_o \mu_v + \phi m_o \mu_{vt}}{\mu_v + \frac{H_v}{Q_{vo}}} + \phi m_o \mu_v + \phi m_o \mu_{vt}$ | $N_{vo}^* = \frac{\Lambda_{vo} - H_vN_o}{\mu_v}$ | $N_{vo}^* = \frac{\Lambda_v - HN_o}{\mu_v}$ |
| IIIa | $N_{vo}^*(\phi) = \frac{(\Lambda_{vo} + \Lambda_v - H_vN_o)\mu_v + (\Lambda_{vo} + \Lambda_v - H_vN_o)\mu_{vt}}{\mu_v + \phi m_o \mu_v + \phi m_o \mu_{vt}}$ | $N_{vo}^* = \frac{\Lambda_{vo} - H_vN_o}{\mu_v}$ | $N_{vo}^* = \frac{\Lambda_v - HN_o}{\mu_v}$ |
| IIb  | $N_{vo}^*(\phi) = \frac{(\Lambda_{vo} + \Lambda_v - H_vN_o)\mu_v + \phi m_o \mu_v + \phi m_o \mu_{vt}}{\mu_v + \frac{H_v}{Q_{vo}}} + \phi m_o \mu_v + \phi m_o \mu_{vt}$ | $N_{vo}^* = \frac{\Lambda_{vo} - H_vN_o}{\mu_v}$ | $N_{vo}^* = \frac{\Lambda_v - HN_o}{\mu_v}$ |

\[N_{vo}^* = \frac{\Lambda_{vo} - H_vN_o}{\mu_v} - \frac{\Lambda_v - HN_o}{\mu_v} = \frac{(\Lambda_{vo} - \Lambda_v)\mu_v}{\mu_v + \frac{H_v}{Q_{vo}}} + \phi m_o \mu_v + \phi m_o \mu_{vt}\]

\[N_{vt}^* = \frac{\Lambda_{vt} - H_vN_t}{\mu_v} - \frac{\Lambda_v - HN_t}{\mu_v} = \frac{(\Lambda_{vt} - \Lambda_v)\mu_v}{\mu_v + \frac{H_v}{Q_{vt}}} + \phi m_o \mu_v + \phi m_o \mu_{vt}\]
isolated cycles, using in the latter case \( \Lambda_{\nu \theta} \) or \( \Lambda_{\nu r} \) in place of \( \Lambda_{\nu} \), \( Q_{\nu \theta h} \) or \( Q_{\nu r h} \) in place of \( Q_{\nu h} \), and \( H_{\theta} \) or \( H_{r} \) in place of \( H \). The existence, uniqueness, and stability of the host-switching equilibria correspond perfectly to those of the isolated cycles, making the population dynamics of the latter a proper limiting case of the dynamics of the host-switching model.

For comparison to the host-sharing model, we instead take the limit of each equilibrium from Table A1 as \( \phi \) goes to \( \infty \). The results are shown in Table 4. Since, in the limit, the relative sizes of \( N_{\nu r} \) and \( N_{\nu \theta} \) are proportional to the migration rates into them (\( m_{\theta} \) and \( m_{r} \), respectively), for comparison purposes we identify \( k_{\theta} = m_{r} / (m_{\theta} + m_{r}) \) and likewise for \( k_{r} \). If we take \( \Lambda_{\nu} = \Lambda_{\nu \theta} + \Lambda_{\nu r} \), then (identifying \( N_{\nu} = N_{\nu \theta} + N_{\nu r} \)) in taking the limit we get precisely the same equilibrium as in the host-sharing model under the same cases. To determine the behaviour of the limiting case, we must compare the conditions under the same scenarios. If we assume that \( N_{\nu j} < N_{j}Q_{h j} \) for \( j = \theta, r \), then clearly \( N_{\nu j} / k_{j} = N_{\nu} < N_{j}Q_{h j} / k_{j} = N_{j} \tilde{Q}_{h j} \). So conditions for a single GAS equilibrium hold under the limiting case, and the population density dynamics of the host-switching model (1) are asymptotic to those of the host-sharing model (6) as \( \phi \to \infty \) (i.e. \( \lim_{\phi \to \infty} \lim_{t \to \infty} N_{\nu}(t; \phi) = \lim_{t \to \infty} \lim_{\phi \to \infty} N_{\nu}(t; \phi) \)).

### 3.2. Infection dynamics

With all population densities asymptotically constant, we can pass to simplified versions of systems (1) and (6) in which host and vector populations have reached their equilibrium densities. Note that this implies \( b_{j}(N_{r}) = \mu_{r}N_{r} \), so that \( b_{j}(N_{r}) / N_{r} = \mu_{r} \), allowing the vertical transmission term(s) to simplify as well. For simplicity of notation, we use \( Q \) in place of \( Q^{*} \) for the various demographic equilibrium vector–host ratios. Also, as done for the one-host model in [26], we define notation (see Appendix 2) to simplify the resulting (demographic equilibrium) infection and mortality rates, including labelling the disease-free equilibrium as \( E_{0} \), the equilibrium in which only strain 1 persists as \( E_{1} \), the strain 2-only equilibrium as \( E_{2} \), and any co-persistence equilibrium as \( E_{3} \).

Analysis of the infection dynamics of both models is given in Appendix 2. Each model is shown to exhibit classical threshold behaviour in terms of reproductive numbers: the BRN \( R_{j} \) for strain \( j \) (\( j = 1, 2 \)), defined as the average number of secondary infections produced by a single infective of type \( j \) introduced into a completely susceptible population [14,50]; and the IRN \( \tilde{R}_{j} \), defined as the average number of secondary infections produced by a single infective of type \( j \) introduced into a population where the other strain is already resident [8,38,53].

- If \( \tilde{R}_{0} = \max(R_{1}, R_{2}) < 1 \), then both strains die out (\( E_{0} \)).
- If \( R_{1} > 1 \) but \( R_{2} < 1 \), or if \( R_{1}, R_{2}, \tilde{R}_{1} > 1 \) but \( \tilde{R}_{2} < 1 \), then strain 1 persists and strain 2 dies out (\( E_{1} \)).
- If \( R_{2} > 1 \) but \( R_{1} < 1 \), or if \( R_{1}, R_{2}, \tilde{R}_{2} > 1 \) but \( \tilde{R}_{1} < 1 \), then strain 2 persists and strain 1 dies out (\( E_{2} \)).
- If \( \tilde{R}_{1}, \tilde{R}_{2} > 1 \) (which implies \( R_{1}, R_{2} > 1 \)), then both strains persist (\( E_{3} \)).

The competitive exclusion exhibited by the single-host model [26] (where no \( E_{3} \) exists) prevents both IRNs from exceeding 1 simultaneously, forcing a single winner. In contrast, the host-switching and host-sharing models permit both strains to co-persist under certain circumstances. The ecological explanation for this behaviour is that the two host populations constitute different resources for vectors, and different landscapes on which for interstrain transmission.
Table 4. Limiting case of host-switching vs. host-sharing vector density equilibria as $\phi \to \infty$, by case (see main text).

| Case | Equilibrium host switching | Limiting values as $\phi \to \infty$ | Equilibrium host sharing |
|------|-----------------------------|---------------------------------|--------------------------|
| I    | $N^*_{vo}(\phi) = k_0(\Lambda_{vo} + \Lambda_{vr}) + \frac{\Lambda_{vo} \mu_{vr}}{\phi(m_v + m_r)} - \mu_{vr}$ | $N^*_{vo} = \frac{N^*_{vr}}{\mu_{vo}}$ | $N^*_{vo} = \frac{N^*_{vr}}{\mu_{vo}}$ |
|      | $N^*_{vr}(\phi) = k_0(\Lambda_{vr} + \Lambda_{vo}) + \frac{\Lambda_{vr} \mu_{vo}}{\phi(m_v + m_r)} - \mu_{vo}$ | $N^*_{vr} = \frac{N^*_{vo}}{\mu_{vr}}$ | $N^*_{vr} = \frac{N^*_{vo}}{\mu_{vr}}$ |
| II   | $N^*_{vo}(\phi) = \frac{k_0(\Lambda_{vo} + \Lambda_{vr} - H_v N_v - H_r N_r)}{\phi(m_v + m_r)} + \frac{\mu_v}{\phi(m_v + m_r)}$ | $N^*_{vo} = \frac{N^*_{vr}}{\mu_{vo}}$ | $N^*_{vo} = \frac{N^*_{vr}}{\mu_{vo}}$ |
|      | $N^*_{vr}(\phi) = \frac{k_0(\Lambda_{vr} + \Lambda_{vo} - H_r N_r - H_v N_v)}{\phi(m_v + m_r)} + \frac{\mu_v}{\phi(m_v + m_r)}$ | $N^*_{vr} = \frac{N^*_{vo}}{\mu_{vr}}$ | $N^*_{vr} = \frac{N^*_{vo}}{\mu_{vr}}$ |
| IIIa | $N^*_{vo}(\phi) = \frac{k_0(\Lambda_{vo} + \Lambda_{vr} - H_v N_v)}{\phi(m_v + m_r)} + \frac{\mu_v}{\phi(m_v + m_r)}$ | $N^*_{vo} = \frac{N^*_{vr}}{\mu_{vo}}$ | $N^*_{vo} = \frac{N^*_{vr}}{\mu_{vo}}$ |
|      | $N^*_{vr}(\phi) = \frac{k_0(\Lambda_{vr} + \Lambda_{vo} - H_r N_r)}{\phi(m_v + m_r)} + \frac{\mu_v}{\phi(m_v + m_r)}$ | $N^*_{vr} = \frac{N^*_{vo}}{\mu_{vr}}$ | $N^*_{vr} = \frac{N^*_{vo}}{\mu_{vr}}$ |
| IIIb | $N^*_{vo}(\phi) = \frac{k_0(\Lambda_{vo} + \Lambda_{vr} - H_v N_v)}{\phi(m_v + m_r)} + \frac{\mu_v}{\phi(m_v + m_r)}$ | $N^*_{vo} = \frac{N^*_{vr}}{\mu_{vo}}$ | $N^*_{vo} = \frac{N^*_{vr}}{\mu_{vo}}$ |
|      | $N^*_{vr}(\phi) = \frac{k_0(\Lambda_{vr} + \Lambda_{vo} - H_r N_r)}{\phi(m_v + m_r)} + \frac{\mu_v}{\phi(m_v + m_r)}$ | $N^*_{vr} = \frac{N^*_{vo}}{\mu_{vr}}$ | $N^*_{vr} = \frac{N^*_{vo}}{\mu_{vr}}$ |
competition to play out. The simplest scenario under which this occurs is that which has been observed in the field, namely strain 1 wins in opossums since they are immune to strain 2, while strain 2 wins the competition in raccoons.

However, the range of parameter values which permit co-persistence is different for each model – and indeed for each value of the dispersal scaling factor $\phi$, if we introduce it as in Equation (5). In order to compare the models, we again use a time-scaling factor $\phi$ for both $m_o$ and $m_r$, replacing $m_j$ with $\phi m_j$ ($j = o, r$) in the simplified infection dynamics system (A2) to obtain system (A4) (given in Appendix 2).

To compare the host-switching model to the single-host model of Kribs-Zaleta and Mubayi [26], we again take the limit of the key infection expressions – endemic equilibria and reproductive numbers – as $\phi \to 0$. The disease-free equilibrium $E_0$ is the same as the demographic equilibrium discussed in the previous section. The simpler single-strain endemic equilibrium is $E_2$, derived in Appendix 2 in a similar way to that of the single-host model in [26]. For $i_r = I_r/N_r$, the limiting case is as follows:

$$\lim_{\phi \to 0} i_{r2}^* = \lim_{\phi \to 0} \frac{\tilde{\beta}_r \tilde{\beta}_{vr2} - (1 - p_2)\mu_r(\tilde{\mu}_{vr} + \phi \mu_r / (\phi \mu_o + \phi m_o))}{\tilde{\beta}_{vr2}(\tilde{\beta}_r + (1 - p_2)\mu_r)} = \frac{\tilde{\beta}_r \tilde{\beta}_{vr2} - (1 - p_2)\mu_r \tilde{\mu}_{vr}}{\tilde{\beta}_{vr2}(\tilde{\beta}_r + (1 - p_2)\mu_r)},$$

(7)

where the new parameters, defined in Appendix 2, are consistent with the notation in [26]. If we take $\mu_r = \mu_h$, $\tilde{\mu}_{vr} = \mu_v$, $Q_{hr} = Q_h$, and $H_r = H$, then the expression becomes identical to that for $E_2$ in [26] (the other state variables follow similarly). As $\phi \to 0$, the constant coefficient in the cubic equation for $E_1$ in the host-switching model goes to zero, yielding a zero equilibrium and a quadratic with one root of each sign; the positive root is identical to the strain 1 equilibrium in [26]. (The co-persistence equilibrium $E_3$ in the host-switching model was found only numerically, so no such limiting argument can be used for it, but in any case the single-host model has no such equilibrium.)

Meanwhile, if we take $Q_r = Q_h$, $Q_{vt} = Q_v$, and $\mu_r = \mu_h$, then the limiting expressions in system (A4) for each strain's BRN take the form $R_i = \max_j R_{ij}$, $j = o, r$, with the host-specific $R_{ij}$ following exactly the form of $R_i$ for isolated cycles (see Table 5 for details.

Table 5. Limiting case of host-switching vs. single-host [26] reproductive numbers as $\phi \to 0$.

| Next-generation matrix | Reproductive number |
|------------------------|---------------------|
| host switching, limit as $\phi \to 0$ | host switching, limit as $\phi \to 0$ |
| $\lim_{\phi \to 0} A_1 = \begin{bmatrix} \frac{\tilde{\beta}_o Q_o}{\mu_o} & 0 & 0 & 0 \\ \frac{\tilde{\beta}_o Q_o}{\mu_o} & 0 & 0 & 0 \\ 0 & 0 & p_1 & 0 \\ 0 & 0 & \frac{\tilde{\beta}_o Q_o}{\mu_o} & 0 \end{bmatrix}$ | $R_{io} = \sqrt{\tilde{\beta}_o \tilde{\beta}_{oo} / \mu_o \tilde{\mu}_o} (p_{1o} = 0)$ |
| $\lim_{\phi \to 0} A_2 = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & p_2 & 0 & 0 \\ 0 & \frac{\tilde{\beta}_o Q_o}{\mu_o} & 0 & 0 \end{bmatrix}$ | $R_{ir} = \frac{1}{2} \left( p_1 + \sqrt{p_1^2 + 4 \tilde{\beta}_o \tilde{\beta}_{oo} \mu_o / \tilde{\mu}_o} \right)$ |

$R_1 = \frac{1}{2} \left( p_2 + \sqrt{p_2^2 + 4 \tilde{\beta}_o \tilde{\beta}_{oo} \mu_o / \tilde{\mu}_o} \right)$

$R_2 = \frac{1}{2} \left( p_2 + \sqrt{p_2^2 + 4 \tilde{\beta}_o \tilde{\beta}_{oo} \mu_o / \tilde{\mu}_o} \right)$

$R_3 = \frac{1}{2} \left( p_2 + \sqrt{p_2^2 + 4 \tilde{\beta}_o \tilde{\beta}_{oo} \mu_o / \tilde{\mu}_o} \right)$
beginning with the next-generation matrix used to derive \( R_0 \). The immunity of opossums to strain 2 and to vertical infection reduce \( R_{20} = 0 \) and simplify \( R_{10} \). The IRNs of the host-switching model (A4) simplify similarly to the maxima of the respective single-host IRNs for the given strain. The infection dynamics of the host-switching model, therefore, appear to approach those of two isolated cycles as \( \phi \to 0 \), although the graphical analysis that follows will illustrate how opossums’ immunity to strain 2 creates an asymmetry between strains.

Comparison of the host-switching and host-sharing models is more complicated, and begins to show a divergence (with the exception of \( E_0 \), which as before reduces to the demographie equilibrium previously discussed). Considering first the strain 2 endemic equilibrium \( E_2 \), we take the limit of the expression given above in Equation (7) (and for \( \phi = 1 \) in (A3) in Appendix 2) as \( \phi \to \infty \), making use of the fact that \( m_r/m_o = k_o/k_r \) to simplify:

\[
\lim_{\phi \to \infty} i^*_r = \frac{\tilde{\beta}_{r2} \tilde{\beta}_{vr2} - (1 - p_2) \mu \tilde{\mu}_{vr} + \phi m_r \tilde{\mu}_{vo} / (\tilde{\mu}_{vo} + \phi m_o)}{\tilde{\beta}_{vr2} (\tilde{\beta}_{r2} + (1 - p_2) \mu_r)} = \frac{\tilde{\beta}_{r2} \tilde{\beta}_{vr2} - (1 - p_2) \mu \tilde{\mu}_{vr} + (k_o/k_r) \tilde{\mu}_{vo}}{\hat{\beta}_{vr2} (\tilde{\beta}_{r2} + (1 - p_2) \mu_r)},
\]

an expression little changed by the limiting process. This form parallels that for \( i^*_r \) in the host-sharing model (A5) (given in (A6) in Appendix 2) but does not in general yield the same value. Similar results hold for the other single-strain endemic prevalences: in general, the infection-dynamic equilibria of the host-switching model do not approach those of the host-sharing model, unlike the population-dynamic equilibrium.

The contrast is even more marked when considering the infection persistence thresholds. Here, because of the complexity of the calculations, we use alternate dimensionless fitness measures \( M_i \) and \( \tilde{M}_i \) (i = 1, 2) in place of the reproductive numbers \( R_i \) and \( \tilde{R}_i \), respectively, as described in Appendix 2; although \( M_i \) and \( \tilde{M}_i \) cannot quite be interpreted biologically the same as \( R_i \) and \( \tilde{R}_i \), they have the same persistence threshold for strain \( i \), namely \( R_i > 1 \iff M_i > 1 \) and \( \tilde{R}_i > 1 \iff \tilde{M}_i > 1 \). Expressions for \( M_i \) and \( \tilde{M}_i \) are derived (using a standard next-generation approach) in Appendix 2, and in order to facilitate interpretation are written in terms of component measures \( M_{ji} \) which measure the transmission of strain \( i \) between vectors and host \( j \), and \( M_{or} \) which gives the proportion of infected vectors in the switching model which live long enough to complete a dispersal ‘round trip’, switching host types twice. As before, the comparison is made between the expressions for the host-sharing model (A5) and the limiting expressions as \( \phi \to \infty \) for the host-switching model (A4).

The unusual results are shown in Table 6: the unlimited increase in host-switching rate completely dominates any infection or demographic processes, sending the new-infection measures \( M_{ji} \) to 0 and sending to 1 the proportion \( M_{or} \) that survive a ‘round trip’ switching host types and back, with the resulting basic reproductive measures \( M_i \) for each strain approaching 1 as well. As illustrated in Figure 3, the approach to 1 as \( \phi \to \infty \) need not be monotone, with the reproductive numbers sometimes crossing 1 (as \( \phi \) increases) before approaching it. The result holds even for invasion reproductive measures (and IRNs), although the behaviours (monotonicity or 1-crossing) of the BRN and IRN for a given strain (or of the same reproductive measures for different strains) may differ from each other.
### Table 6. Limiting case of host-switching vs. host-sharing reproductive numbers as $\phi \to \infty$.

| Basic reproductive measure host switching | Limiting values | Reproductive measure host sharing |
|-------------------------------------------|-----------------|----------------------------------|
| $M_{01}^2 = \frac{\tilde{\beta}_{01} \tilde{\beta}_{01}}{\mu_o (\phi m_o + \tilde{\mu}_o)}$ | $\lim_{\phi \to \infty} M_{01}^2 = 0$ | $M_{01}^2 = \frac{\tilde{\beta}_{01} \tilde{\beta}_{01}}{\mu_o \tilde{\mu}_o}$ |
| $M_{0r}^2 = \frac{\phi m_o \phi m_r}{(\phi m_o + \tilde{\mu}_o)(\phi m_r + \tilde{\mu}_r)}$ | $\lim_{\phi \to \infty} M_{0r}^2 = 1$ | |
| $M_{0i}^2 = \frac{\tilde{\beta}_{0i} \tilde{\beta}_{0i}}{(1 - p_i) \mu_r (\phi m_r + \tilde{\mu}_r)}$ | $\lim_{\phi \to \infty} M_{0i}^2 = 0$ | $M_{0i}^2 = \frac{\tilde{\beta}_{0i} \tilde{\beta}_{0i}}{(1 - p_i) \mu_r \tilde{\mu}_r}$ |
| $M = \sqrt{M_{01}^2 + M_{0r}^2 + M_{0i}^2 + \sqrt{(M_{01}^2 + M_{0r}^2 + M_{0i}^2)^2 - 4M_{01}^2 M_{0r}^2 M_{0i}^2}}$ | $\lim_{\phi \to \infty} M_{0i}^2 = 1$ | $M = \sqrt{M_{01}^2 + M_{0r}^2}$ |
| $M_r = \sqrt{M_{0r}^2 + M_{0r}^2}$ | $\lim_{\phi \to \infty} M_{r}^2 = 1$ | $M_r = M_r$ |

### Invasion reproductive measure host switching

| Invasion reproductive measure host sharing | Limiting values | Invasion reproductive measure host sharing |
|-------------------------------------------|-----------------|----------------------------------|
| $\tilde{M}_i = \frac{1}{2} \left\{ M_{01}^2 \phi_{x_2} + M_{0r}^2 \phi_{x_2} + M_{0i}^2 \phi_{x_2} \right\}$ | $\lim_{\phi \to \infty} \tilde{M}_1 = 1$ | $\tilde{M}_1 = \sqrt{M_{01}^2 s_{r2} + M_{0r}^2 s_{r2} + M_{0i}^2 s_{r2}}$ |
| $\tilde{M}_2 = \sqrt{M_{0r}^2 + M_{0r}^2}$ | $\lim_{\phi \to \infty} \tilde{M}_2 = 1$ | $\tilde{M}_2 = M_r \sqrt{s_{r1} s_{r1}}$ |

**Figure 3.** $R_2$ (left) and $\tilde{R}_1$ (right) for the host-switching model (A2) as a function of $\phi$, showing a non-monotone approach to 1 including a crossing. Parameters are as given in Table 2 except the $\beta_{ij}$ are reduced by an order of magnitude and $m_o = 0.1 \phi$, $m_r = \phi$, and for the second graph in addition $\beta_{r1} = 0.235/yr$ (removing most of strain 1’s advantage in raccoons).

This result appears to be at odds with the facts that the corresponding single-strain equilibrium prevalence levels do not approach zero and yet in general $t^*_{r2}, t^*_{vr2} \to 0$ as $M_2 \to 1$ (the persistence threshold). The apparent discrepancy can be resolved by observing that

$$t^*_{r2} = \frac{(1 - p_2) \mu_r (m_r \phi + \tilde{\mu}_r)}{\beta_{vr2} [\beta_{r2} + (1 - p_2) \mu_r]} (M_2^2 - 1),$$
which (since \((M^2 - 1) \to 0 \text{ as } \phi \to \infty\)) is an indeterminate form. That is, \(i^*_2\) is indeed a multiple of \((M^2 - 1)\), but the multiple becomes infinite as \(\phi \to \infty\), allowing the product to avoid going to 0.

Overall the impact of host-switching rates on infection dynamics may be seen most clearly graphically, especially where potentially nonuniform convergence complicates limiting behaviour at both extremes of \(\phi\). \(\tilde{R}_1\) and \(\tilde{R}_2\) can be considered implicit functions of \(R_1\) and \(R_2\), making \(\tilde{R}_1 = 1\) and \(\tilde{R}_2 = 1\) contours or level sets which appear as curves in the \(R_1\)–\(R_2\) plane. The persistence threshold for strain 1 is given by \(R_1 = 1\) when \(R_2 < 1\) and by \(\tilde{R}_1 = 1\) when \(R_2 > 1\); graphed in the \(R_1\)–\(R_2\) plane, these two connected curves form a (somewhat vertical) single boundary – forming the right side of the unit square and moving upward from there – which partitions the positive quadrant into two regions (corresponding, respectively, to persistence or eradication of strain 1). The curves \(R_2 = 1\) and \(\tilde{R}_2 = 1\) likewise form a (somewhat horizontal) single boundary – forming the top of the unit square and moving rightward from there – delimiting conditions where strain 2 persists or disappears. Figure 5 shows the four threshold conditions \((R_1 = 1, R_2 = 1, \tilde{R}_1 = 1, \tilde{R}_2 = 1)\) for several values of \(\phi\) (overall host-switching frequency) and \(m_o\) vs. \(m_r\) (relative rates of leaving each host), graphed in the \(R_1\)–\(R_2\) plane. For comparison, the corresponding graphs for the single-host model and the host-sharing model are given in Figure 4. In each graph, the unit square \(R_1, R_2 \leq 1\) (adjacent to the origin in the lower left corner) gives the region where both strains die out and the disease-free equilibrium is the unique attractor. To the right of the unit square is the region \(R_1 > 1, \tilde{R}_2 < 1\) where strain 1 defeats strain 2 and \(E_1\) is the unique attractor, and above the unit square is the region \(R_2 > 1, \tilde{R}_1 < 1\) where instead strain 2 defeats strain 1 and \(E_2\) is the unique attractor. Between the curves \(\tilde{R}_1 = 1\) and \(\tilde{R}_2 = 1\) in most graphs (the single-host model being the exception, where \(\tilde{R}_1 = 1 \Leftrightarrow \tilde{R}_2 = 1\)) there is a region where both strains persist and cocirculate: strain 1 alone in the opossums; strain 2 would normally win out in the raccoon cycle, but host

![Figure 4](image).

**Figure 4.** Asymptotic behaviour for the single-host model of Kribs-Zaleta and Mubayi [26] (left) and the host-sharing model (A5) (right) in terms of reproductive number threshold curves \((R_i = 1 \text{ and } \tilde{R}_i = 1, i = 1, 2)\). Note that for the single-host model the curves \(\tilde{R}_1 = 1, \tilde{R}_2 = 1\) coincide, signalling competitive exclusion.
switching brings a continual flow of strain 1 from the opossum cycle. (The graphs show the region $R_1, R_2 < 5$ except the last two which show wider areas in order to illustrate the narrower coexistence regions.) The reader should note that, since the various reproductive numbers are functions of many original model parameters, there is no single way to make them vary; for these graphs, the $\beta_{ij}$ and $\beta_{vij}$ were scaled by a dimensionless factor $\psi_i$ which was then varied to extend both the corresponding $R_i$ and $\tilde{R}_i$. It should also be noted that the reproductive numbers are functions of $\phi$, so leaving all other parameters fixed as

$$
\left(\frac{m_{ao}}{\phi}, \frac{m_{ro}}{\phi}\right)
$$

$\phi = 1/1000$  $\phi = 1$  $\phi = 1000$

1, 1000

1, 1

8, 4.1

1000, 1

Figure 5. Asymptotic behaviour for the host-switching model (A2) in terms of reproductive number threshold curves ($R_i = 1$ and $\tilde{R}_i = 1$, $i = 1, 2$), for varying $\phi$ and $m_o$ vs. $m_r$. 
\( \phi \) changes does not leave \((R_1, R_2)\) coordinates fixed. Illustrative values of \( \phi = \frac{1}{1000}, 1, 1000 \) were chosen to illustrate the effects of absolute host-switching rate, and the same values were chosen for \( m_o/m_r \) to illustrate the effects of asymmetries in departure rates from one host vs. the other; in addition, \( m_o/m_r = 8/4.1 \) was included as it reflects the ratio of the two hosts' estimated densities, and thus an assumption that an average raccoon accommodates as many vectors as an average opossum.

There are several patterns to observe in the graphs. First, in general the region of coexistence \((\bar{R}_1 > 1, \bar{R}_2 > 1)\) opens up as \( \phi \) increases (from left to right in each row of Figure 5), illustrating how connectivity between cycles produces, in ecological terms, two different host resources, allowing \textit{T. cruzi} strains to coexist by specializing in different host–vector cycles. Second, the region of coexistence also opens up as \( m_r \) increases relative to \( m_o \) (from bottom to top in each column of Figure 5): as vectors leave raccoons faster and stay longer with opossums (where they can only pick up strain 1 infections), it becomes easier for strain 1 to persist in the presence of strain 2 (note it is largely the \( \bar{R}_1 = 1 \) boundary that is moving to widen the coexistence region). However, the graphs at either extreme of \( \phi \) do not quite match up with those in Figure 4: At the low end, as \( \phi \to 0 \), the coexistence region does not close up entirely, but rather the \( \bar{R}_1 = 1 \) threshold hits a vertical asymptote beyond which the coexistence region continues to exist. This phenomenon occurs because, as \( R_2 \) increases, although the proportion \( x_{r2} \) of raccoons and their associated vectors available to be infected by strain 1 dwindles to 0, the corresponding proportion \( x_{o2} \) of opossums and their associated vectors remains bounded away from 0 (since the opossums are immune to strain 2), so to reach the threshold value \( \bar{R}_1 = 1 \) does not require an arbitrarily high \( R_1 \) (unlike the corresponding case with \( \bar{R}_2 \)). Thus, the asymmetry in host susceptibility creates this ‘opossum wall’ permitting strain 1 to persist in both cycles even when minimally connected; when \( \phi \) actually reaches 0 and the cycles disconnect, coexistence vanishes as the raccoon cycle reverts to the single-host competition scenario of Kribs-Zaleta and Mubayi [26] while the opossum cycle maintains only strain 1. Finally, at the high end of host switching, as \( \phi \to \infty \) the coexistence region opens up well beyond that for the host-sharing model. This occurs because in the limit the host-switching process dominates the infection process as well as the demographic renewal process, taking vectors infected with either strain (but particularly strain 1, since most of the coexistence region corresponds to values where strain 2 would normally push out strain 1 in the raccoon cycle) back and forth between host populations many times before they die. In the host-sharing model, there is no switching to detract from the other processes.

4. Results

The analysis and comparison of the behaviours of the host-switching and host-sharing models can now be applied to address the research questions posed in the introduction. We address the methodological question first. Although the two models exhibit the same set of possible outcomes in terms of infection persistence, the ranges of parameter values for which each outcome occurs may be quite different; in particular, we have seen that despite the demographics of the former converging to those of the latter model as the host-switching frequency increases, the infection dynamics of the host-switching model converge to a measurably distinct behaviour from those of the host-sharing model, because at high frequencies the host switching completely dominates the infection dynamics. On
the other hand, the host-sharing model is clearly unable to capture the limited nature of the communication between transmission cycles in situations where host switching is relatively infrequent. Therefore, in determining the extent to which co-persistence of parasite strains may occur (for a given set of contact and demographic rates), scenarios where different hosts use the same den at different times may be better described by a host-sharing model, while scenarios involving physically distinct groups of vectors which communicate via dispersal only occasionally may be better described by host switching.

To assess the models’ ability to explain the co-persistence of *T. cruzi* I and IV in raccoons [42] and woodrats [5] (at very different levels) in the southern USA, we consider opossums as a reservoir for *T. cruzi* I and take parameter estimates from [23,26] as context (see Appendix 3 for details). Of the 64 infected raccoons tested for parasite strain in [42], only 3 tested positive for type I, 1 of which tested as an indeterminate I/IV (the remaining 60 tested positive for type IV only). If strain 1’s ability to infect raccoons is set to replicate this frequency of strain 1 among infected raccoons in the host-switching model for low to moderate host-switching rates, two curious observations can be made: First, the host-sharing model using the same parameter values yields $\bar{R}_1 < 1$ instead, and thus strain 1 would die out in the presence of strain 2 if the vector populations are not distinct (although $\bar{R}_1 > 1$ in the host-switching model for all values of $\phi$). Second, with co-persistence at the levels observed in raccoons, strain 1 persists at a much higher level in the associated vectors (25%, compared to 66% strain 2, when $\phi = 1$) than in the raccoons, most likely due to the higher host-to-vector infection rate (compared to vector-to-host ). This phenomenon should be testable – that is, if the trace prevalence of strain 1 in raccoons is due to ‘leakage’ via vectors’ host-switching from opossums, then vectors living in raccoon dens should have a significant strain 1 prevalence.

The isolates from 23 woodrats for which parasites were genetically analysed in [5] yielded 10 TcI (43%) and 13 TcIV (57%) classifications, a more substantial co-persistence; however, a similar analysis performed substituting parameter values for woodrats in place of those for raccoons in the model fails to predict co-persistence.

5. Discussion

In investigating possible explanations for the observed persistence of both *T. cruzi* strains native to the USA in some sylvatic cycles, this study examined two distinct ways of modelling inter-cycle connectivity driven by vectors switching hosts, the difference being whether the host switching is modelled explicitly or implicitly. Modelling host switching explicitly allows one to distinguish the frequency with which such switching occurs from the relative proportions of vector contacts made with each host type; however, an extreme level of switching causes the switching behaviour to dominate infection and demographic dynamics in ways that may not reflect reality. The host-switching model illustrates some aspects of *T. cruzi* transmission dynamics, such as the ‘opossum wall’ which allows both strains to persist (in a single population) even for minimally connected transmission cycles, in ways the host-sharing model cannot, but may overestimate the extent to which vector movement fosters co-circulation when host switching is more common, as may be expected when two host species live in proximity to each other but individual hosts often abandon their sleeping places for long periods of time. In the latter case, the host-sharing model provides connectivity between transmission cycles without allowing it to
interfere in transmission dynamics (and demographics). The failure of the host-switching model’s infection dynamics to converge smoothly to the limiting models at either extreme of the switching rate (isolated cycles or host sharing) is especially striking given how perfectly the demographics do approach those in the limiting models. This comparison of models provides a baseline for understanding the broader relationship between other existing multihost [sharing] models (such as for West Nile virus) and metapopulation (vector migration) models for vector-borne infections.

The scientific literature on modelling the population dynamics and infection dynamics of *T. cruzi* hosts and vectors has focused largely on Chagas’ disease proper, that is, on infection of humans, with especial attention to vector control in domestic settings (see [35] for a review). However, even some of the earliest work noted the importance of vector migration to and from domestic settings [40], and a more recent study of such vector dispersal (in a context of *Triatoma dimidiata* infesting rural villages in Mexico) [2] estimated that roughly half (55%) of the vectors entering the domestic setting arrived from the peridomestic environment, and roughly half migrated from sylvatic habitat. One widely cited computational study pointed out the impact of dogs in the domestic setting as highly infected alternative hosts to humans, exacerbating infection risk to humans (although it should be noted all dogs in the model were assumed infected) [7]. The only other study we found which considered transmission of two *T. cruzi* strains to multiple hosts (humans and reservoir hosts in a domestic setting, but without the cross-immunity that produces interstrain competition) found that the presence of the reservoir hosts in the infection network was critical for reproducing the observed prevalence levels [13]. It is not surprising that any form of interconnecting transmission cycles predicts that a given strain will either persist in all cycles or die out in all cycles, but the nature of the connection impacts the form of the reproductive numbers that provide persistence thresholds. Host-sharing models are common in multihost models of mosquito-borne diseases (e.g. West Nile virus [11]) since mosquitoes may change hosts at every feeding, and the reproductive numbers for such infection networks take the form of weighted sums of the reproductive numbers for each cycle (*R*₂ₐ *R*₂ₑ = ∑ᵢ *R*₂ᵢ), as in [12] and the present study. Host-switching models with distinct vector populations for each host are effectively metapopulation models, and have the more complex reproductive number structure seen in such networks (e.g. [10]), involving a term representing the bidirectional vector migration. As seen in this study, the latter term’s importance varies directly with the migration rates, and can actually dominate the reproductive measures at high enough frequencies. The present study relates these two classes of model via a single framework which illustrates how highly connected vector populations actually overestimate the possibility of co-persistence in a unified vector population, in cases of cross-immunity.

As a caveat, it should be noted that neither model addresses the proportion of time that vectors spend switching from one host to another, assuming implicitly instead that vectors find new hosts in each case quickly enough to maintain their maximum available feeding (contact) rate. Work in progress uses agent-based models (ABMs) to estimate some basic data such as these about host–vector contacts.

It is clear from both models that vectors’ host-switching behaviour does foster persistent co-circulation of two strains of vector-borne infections with cross-immunity. One may also consider, however, the perspective of a single pathogen strain: is this connectivity between transmission cycles advantageous for both competing strains? Strain 1 is, of course, advantaged, as its dominance in the opossum-*T. sanguisuga* cycle is unshakable for high enough
R1, and communication between the raccoon and opossum cycles therefore strengthens its ability to persist in the raccoon cycle. Strain 2, on the other hand, gets a mixed bag: when vectors leave raccoon hosts much more often than they leave opossums (m_r >> m_o, top row of Figure 5) high connectivity fosters strain 2’s persistence – in particular, among the vectors feeding on opossums, via exportation from the raccoon cycle. When instead vectors leave opossums much more frequently than they leave raccoons (m_o >> m_r, bottom row of Figure 5), strain 2’s ability to persist is largely unaffected (the average vector abandons an opossum host before becoming infected, so does not import strain 1 infection to the raccoon cycle) compared to isolated cycles (where the threshold is the diagonal line R_2 = R_1). However, when host switching in each direction is on the same order of magnitude (middle rows of Figure 5), moderate to high connectivity actually reduces strain 2’s ability to persist. This result suggests that parasites like T. cruzi I which have a monopoly on one host resource may flourish when the associated vector is more likely to switch hosts (perhaps implying an evolutionary benefit to the parasite if it can make vectors get hungry faster), whereas parasites such as T. cruzi IV only benefit when vectors are entering, rather than leaving, transmission cycles they dominate (so that hungry vectors only help the parasite by spreading it when neighbouring transmission cycles are receptive to invasion).

The co-circulation of T. cruzi I and IV seen at some level in the host-switching model even for minimally connected cycles offers one possible explanation for the trace prevalence of T. cruzi I observed in raccoons (cf. [42]), where T. cruzi IV normally dominates. Applying the two models to a hypothetical connection between opossum and woodrat cycles for T. sanguisuga does not, however, account for the broader co-persistence observed in woodrats. Future work will consider the more general two-strain model to study the observed presence of both strains in multiple woodrat populations [5].

Another possible explanation for persistence of two strains in a transmission cycle involves local stochasticity, which can extend transient dynamics to a timescale of many years. Further research in progress uses two types of stochastic models – stochastic differential equations and ABMs – to describe the transient dynamics caused by small-scale variations in infection and demographic processes which allow both strains to coexist in a population for a time.

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Disclosure statement

No potential conflict of interest was reported by the authors.

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Appendices. Computations

Appendix 1. Population dynamics

The host densities $N_o$ and $N_r$ from the host-switching and host-sharing models, unaffected by infection dynamics and (by assumption) by vector feeding more generally, attain equilibrium levels dictated by their inherent reproduction and mortality rates. For example, $N_o' = b_o(N_o) - \mu_o N_o$ has an asymptotically constant solution under any nonnegative birth function $b_o$ which is bounded for large $N_o$. As in the analysis of the single-host model [26], a theorem of Thieme [49] then allows us to pass to the simpler system in which $N_o$ is that constant. The same is true for $N_r$. (These constants may not be unique in general, but are unique for most common birth rates, such as constant...
Table A1. Expressions for the equilibrium in each of the four submodels of system (A1).

| Case | \( N^{\infty}_{vo} = \frac{\lambda v_0}{\mu v_0} \cdot \frac{\mu v_0 + m_r}{\mu v_0 + m_r + m_o(\mu v_0/\mu v)} \) + \( \frac{\lambda v_0}{\mu v_0} \cdot \frac{m_r}{\mu v_0 + m_o(\mu v_0/\mu v)} \) | \( N^{\infty}_{vr} = \frac{\lambda v_0}{\mu v_0} \cdot \frac{\mu v_0 + m_r}{\mu v_0 + m_r + m_o(\mu v_0/\mu v)} \) + \( \frac{\lambda v_0}{\mu v_0} \cdot \frac{m_r}{\mu v_0 + m_o(\mu v_0/\mu v)} \) |
|------|-------------------------------------------------|-------------------------------------------------|
| II   | \( N^{\infty}_{vo} = \frac{\lambda v_0 - H_0 N_0}{\mu v_0} \cdot \frac{\mu v_0 + m_r}{\mu v_0 + m_r + m_o(\mu v_0/\mu v)} \) + \( \frac{\lambda v_0 - H_0 N_0}{\mu v_0} \cdot \frac{m_r}{\mu v_0 + m_o(\mu v_0/\mu v)} \) | \( N^{\infty}_{vr} = \frac{\lambda v_0 - H_0 N_0}{\mu v_0} \cdot \frac{\mu v_0 + m_r}{\mu v_0 + m_r + m_o(\mu v_0/\mu v)} \) + \( \frac{\lambda v_0 - H_0 N_0}{\mu v_0} \cdot \frac{m_r}{\mu v_0 + m_o(\mu v_0/\mu v)} \) |
| IIIa | \( N^{\infty}_{vo} = \frac{\lambda v_0 - H_0 N_0}{\mu v_0} \cdot \frac{\mu v_0 + m_r}{\mu v_0 + m_r + m_o(\mu v_0/\mu v)} \) + \( \frac{\lambda v_0 - H_0 N_0}{\mu v_0} \cdot \frac{m_r}{\mu v_0 + m_o(\mu v_0/\mu v)} \) | \( N^{\infty}_{vr} = \frac{\lambda v_0 - H_0 N_0}{\mu v_0} \cdot \frac{\mu v_0 + m_r}{\mu v_0 + m_r + m_o(\mu v_0/\mu v)} \) + \( \frac{\lambda v_0 - H_0 N_0}{\mu v_0} \cdot \frac{m_r}{\mu v_0 + m_o(\mu v_0/\mu v)} \) |
| IIIb | \( N^{\infty}_{vo} = \frac{\lambda v_0}{\mu v} \cdot \frac{\mu v_0 + m_r}{\mu v_0 + m_r + m_o(\mu v_0/\mu v)} \) + \( \frac{\lambda v_0}{\mu v} \cdot \frac{m_r}{\mu v_0 + m_o(\mu v_0/\mu v)} \) | \( N^{\infty}_{vr} = \frac{\lambda v_0}{\mu v} \cdot \frac{\mu v_0 + m_r}{\mu v_0 + m_r + m_o(\mu v_0/\mu v)} \) + \( \frac{\lambda v_0}{\mu v} \cdot \frac{m_r}{\mu v_0 + m_o(\mu v_0/\mu v)} \) |

and logistic. More complicated dynamics such as Allee effects can be incorporated without disturbing infection dynamics, but since the latter are the focus of this study, further discussion of host population dynamics is omitted here.)

Analysis of the vector densities is more complicated and differs for each model, although the result is the same: for constant vector birth, a single globally attracting equilibrium.

A.1.1 Host switching

For the host-switching model, we consider the subsystem of Equation (1) given by

\[ N^{\infty}_{vo} = b_v(N_{vo}) - (\mu_v + m_o)N_{vo} - E_v(Q_o)N_o + m_rN_{vr}, \]

\[ N^{\infty}_{vr} = b_v(N_{vr}) - (\mu_v + m_r)N_{vr} - E_r(Q_r)N_r + m_oN_{vo}, \]

with Holling type I (sharp) saturation in the predation functional response \( E_r(Q_r) = H_j \min(Q_j/Q_{hj}), 1 \) \( (j = o, r) \). The form of the saturation causes study of this system to decompose into four cases:

(1) \( Q_o < Q_{ho} \) and \( Q_r < Q_{hr} \) (i.e. \( N_{vo} < N_o Q_{ho} \), \( N_{vr} < N_r Q_{hr} \));

(II) \( Q_o > Q_{ho} \) and \( Q_r > Q_{hr} \) (i.e. \( N_{vo} > N_o Q_{ho} \), \( N_{vr} > N_r Q_{hr} \));

(IIIa) \( Q_o > Q_{ho} \) but \( Q_r < Q_{hr} \) (i.e. \( N_{vo} > N_o Q_{ho} \), \( N_{vr} < N_r Q_{hr} \));

(IIIb) \( Q_o < Q_{ho} \) but \( Q_r > Q_{hr} \) (i.e. \( N_{vo} < N_o Q_{ho} \), \( N_{vr} > N_r Q_{hr} \)). The result when vector recruitment is constant (some detail is given below) is a single, GAS equilibrium.

For \( b_v(N_{ij}) = \lambda v_j \) \( (j = o, r) \), the system has a single equilibrium for each of the four cases described above, given in Table A1 using the notation \( \mu v_j = \mu v_0 + H_j/Q_{hj} \) \( (j = o, r) \). Some algebra is then necessary to verify that each equilibrium appears in the composite model (A1) precisely when the others do not, for example, \( N^{I}_{vo} < Q_{ho} N_o \iff N^{II}_{vo} < Q_{ho} N_o \) (the Case I equilibrium falls below the \( N_{vo} \) saturation threshold, and thus appears in the model if \( N_{vr} \) is low enough, precisely when the Case II equilibrium also falls below that threshold, and thus does not appear in the model). This verifies the uniqueness of the composite model’s equilibrium. Stability follows from standard methods and the Poincaré–Bendixson Theorem.

A.1.2 Host sharing

For the host-sharing model (6), the population dynamics again decouple from the infection dynamics and can be studied separately. Again the host population dynamics can be taken as asymptotically constant, allowing vector density dynamics to be studied with host densities at equilibrium.

Using the apparent vector densities \( N_{vj} = k_j N_j \) for the saturation in the predator’s functional response, \( E_j(Q_j) = H_j \min(Q_j/Q_{hj}) \), the threshold vector densities are effectively rescaled by the contact proportions \( k_j \): \( E_j(Q_j) = H_j \min(N_v/N_j Q_{hj}) \) with \( Q_{hj} = Q_{hj}/k_j > Q_{hj} \). The analysis
requires consideration of three cases similar to those from the host-switching model: (I) \( N_v < \min(\tilde{Q}_{ho}N_o, \tilde{Q}_{hr}N_r) \); (II) \( N_v > \max(\tilde{Q}_{ho}N_o, \tilde{Q}_{hr}N_r) \); and (III) \( N_v \) between \( \tilde{Q}_{ho}N_o \) and \( \tilde{Q}_{hr}N_r \). For simplicity, we consider constant vector recruitment \( b_v(N_v) = \Lambda_v \).

In Case I, the vector density dynamics simplify to

\[
N_v' = \Lambda_v - \left( \mu_v + \frac{H_o}{\tilde{Q}_{ho}} + \frac{H_r}{\tilde{Q}_{hr}} \right) N_v,
\]

which has the single, GAS equilibrium

\[
N_v^I = \frac{\Lambda_v}{\mu_v + H_o/\tilde{Q}_{ho} + H_r/\tilde{Q}_{hr}}.
\]

This equilibrium appears in the saturation model if and only if its value is below the threshold \( \min(\tilde{Q}_{ho}N_o, \tilde{Q}_{hr}N_r) \).

In Case II, the dynamics are

\[
N_v' = (\Lambda_v - H_oN_o - H_rN_r) - \mu_v N_v,
\]

which has the single, GAS equilibrium

\[
N_v^II = \frac{\Lambda_v - H_oN_o - H_rN_r}{\mu_v}.
\]

This equilibrium appears in the saturation model if and only if its value is above the threshold \( \max(\tilde{Q}_{ho}N_o, \tilde{Q}_{hr}N_r) \).

In Case III, we again must consider two scenarios, depending on the relative order of \( \tilde{Q}_{ho}N_o \) and \( \tilde{Q}_{hr}N_r \). Suppose (a) that \( \tilde{Q}_{ho}N_o < N_v < \tilde{Q}_{hr}N_r \). Then we have

\[
N_v' = (\Lambda_v - H_oN_o) - (\mu_v + H_r/\tilde{Q}_{hr})N_v,
\]

with GAS equilibrium

\[
N_v^{IIIa} = \frac{\Lambda_v - H_oN_o}{\mu_v + H_r/\tilde{Q}_{hr}}.
\]

This equilibrium appears in the saturation model if and only if its value is in the interval \( [\tilde{Q}_{ho}N_o, \tilde{Q}_{hr}N_r] \). If instead (b) \( \tilde{Q}_{hr}N_r < N_v < \tilde{Q}_{ho}N_o \), then

\[
N_v' = (\Lambda_v - H_rN_r) - (\mu_v + H_o/\tilde{Q}_{ho})N_v,
\]

with GAS equilibrium

\[
N_v^{IIIb} = \frac{\Lambda_v - H_rN_r}{\mu_v + H_o/\tilde{Q}_{ho}}.
\]

which appears in the composite model if and only if it is in \( [\tilde{Q}_{hr}N_r, \tilde{Q}_{ho}N_o] \).

To determine the behaviour of the composite model, we compare conditions for each case’s equilibrium to be within its interval of validity. Under scenario (a) in which \( \tilde{Q}_{ho}N_o < \tilde{Q}_{hr}N_r \), \( N_v^I \) is an equilibrium of the saturation model when \( N_v^I < \tilde{Q}_{ho}N_o \). This condition can be simplified to \( \Lambda_v < H_oN_o + (\mu_v + H_r/\tilde{Q}_{hr})\tilde{Q}_{ho}N_o \). The condition that \( N_v^{IIIa} \) not be too low (\( N_v^{IIIa} > \tilde{Q}_{ho}N_o \)) simplifies to \( \Lambda_v > H_oN_o + (\mu_v + H_r/\tilde{Q}_{hr})\tilde{Q}_{ho}N_o \), the reverse of the previous condition, which shows that \( N_v^I \) is an equilibrium of the saturation model precisely when \( N_v^{IIIa} \) is not (because it is too low). Similarly, we can rewrite the condition \( N_v^{II} > \tilde{Q}_{hr}N_r \) that the Case II equilibrium appear in the saturation model as \( \Lambda_v > H_oN_o + H_rN_r + \mu_v \tilde{Q}_{ho}N_o \), which is the reverse of the condition that \( N_v^{IIIa} \) not be too high (\( N_v^{IIIa} < \tilde{Q}_{hr}N_r \)). Thus \( N_v^I \) appears in the saturation model precisely when \( N_v^{IIIa} \) is too high. Hence a predator functional response with Holling type I saturation and constant vector recruitment leads to a single GAS equilibrium. (Analogous results hold under the alternative scenario (b) where \( \tilde{Q}_{ho}N_o > \tilde{Q}_{hr}N_r \).)
Appendix 2. Infection dynamics

A.2.1 Host switching

Defining \( \tilde{\beta}_{ji} = c_{ji}(Q_j) + \rho_{ji}E_j(Q_j) \), \( \tilde{\beta}_{vji} = c_{vji}(Q_j) \), and \( \tilde{\mu}_{vji} = \mu_v + E_j(Q_j) \), for \( j = o, r, i = 1, 2 \), the simplified host-switching system is then

\[
I'_{o1} = \tilde{\beta}_{o1}S_{o1} I_{v01} - \mu_o I_{o1},
\]

\[
I'_{v01} = \tilde{\beta}_{v01}S_{v01} I_{o1} - \tilde{\mu}_o I_{v01} - m_o I_{v01} + m_r I_{vr1},
\]

\[
I'_1 = p_1 \mu_r I_{r1} + \tilde{\beta}_{r1}S_{r1} I_{vr1} - \mu_r I_{r1},
\]

\[
I'_{vr1} = \tilde{\beta}_{vr1}S_{vr1} I_{r1} - \mu_{vr} I_{vr1} - m_{vr} I_{vr1} + m_o I_{v01},
\]

\[
I'_{vo2} = -\tilde{\mu}_o I_{vo2} - m_o I_{vo2} + m_r I_{vr2},
\]

\[
I'_2 = p_2 \mu_r I_{r2} + \tilde{\beta}_{r2}S_{r2} I_{vr2} - \mu_r I_{r2},
\]

\[
I'_{vr2} = \tilde{\beta}_{vr2}S_{vr2} I_{r2} - \mu_{vr} I_{vr2} - m_{vr} I_{vr2} + m_o I_{v02}.
\]

(A2)

Computation of each strain’s BRN \( R_1, R_2 \) using standard next-generation operator methods [14,50] is complicated by the vertical transmission terms (which prevent the next-generation matrices from being sparse enough to write simple expressions for the dominant eigenvalue of each), but analysis can proceed using the same methods to develop alternative fitness measures for each strain. Next-generation operator methods require new infection terms, including from vertical transmission terms, to be considered separately from other terms; as a result, it is incorrect to coalesce the vertical transmission terms \( p_{ji} \mu_r I_{ri} \) in the \( I_{ri} \) equations (\( i = 1, 2 \)) with the mortality terms \( -\mu_r I_{ri} \), even though mathematically they can be simplified to \( -(1 - p_i)\mu_r I_{ri} \). However, a system in which those terms are coalesced behaves identically to system (A2), and therefore the BRNs of the alternative ‘simplified’ system have the same mathematical significance, in terms of equilibrium stability, as those of system (A2). The same holds for the systems’ invasion reproductive numbers. Therefore we calculate the various reproductive numbers for the simplified system but denote them by \( M \) (rather than \( R \)) and use them as dimensionless fitness measures.

Standard computations (see section A.2.3 for details) then lead to the following expressions:

\[
M_1 = \sqrt{\frac{1}{2} (M_{o1}^2 + M_{or}^2 + M_{r1}^2 + \sqrt{(M_{o1}^2 + M_{or}^2 + M_{r1}^2)^2 - 4M_{o1}^2M_{r1}^2})}, \quad M_2 = \sqrt{M_{r2}^2 + M_{or}^2},
\]

where

\[
M_{o1}^2 = \frac{\tilde{\beta}_{o1}\tilde{\beta}_{v01}}{\mu_o(m_o + \tilde{\mu}_o)}, \quad M_{or}^2 = \frac{m_o}{m_o + \tilde{\mu}_o}, \quad m_r + \tilde{\mu}_r, \quad M_{r1}^2 = \frac{\tilde{\beta}_{r1}\tilde{\beta}_{r2}}{(1 - p_i)\mu_r(m_r + \tilde{\mu}_r)} \quad (i = 1, 2).
\]

If \( M_i > 1 \) (\( i = 1, 2 \)) then strain \( i \) can persist in a naïve population. If both strains’ basic fitness measures exceed 1, then persistence is determined by each strain’s invasion fitness measure, given by

\[
\tilde{M}_1 = \sqrt{\frac{1}{2} (M_{o1}^2X_{o2} + M_{or}^2 + M_{r1}^2X_{r2} + \sqrt{(M_{o1}^2X_{o2} + M_{or}^2 + M_{r1}^2X_{r2})^2 - 4M_{o1}^2X_{o2}M_{r1}^2X_{r2})}}, \quad M_2 = \sqrt{M_{r2}^2X_{r1} + M_{or}^2},
\]

\[
\tilde{M}_2 = \sqrt{M_{r2}X_{r1} + M_{or}^2}, \quad \text{where} \quad X_{ji} = S_{ji}^*, S_{ji}^* \text{ is } S_j/N_j \text{ evaluated at the equilibrium where } \{\text{only}\} \text{ strain } i \text{ is endemic } (j = o, r), \text{ and likewise for } s_{vji}^*. \text{ Each strain can persist in a population where the other strain is [already] endemic precisely when its invasion fitness measure exceeds 1.}
Since the equilibrium condition for $I_{v02}$ simplifies to $I^*_{v02} = m_r I^*_{vrt2}/(\bar{\mu}_{v0} + m_o)$, the strain 2 endemic equilibrium $E_2$ can be derived similarly to that for the one-host model of Kribs-Zaleta and Mubayi [26]:

$$I^*_{r2} = \frac{I^*_{v2}}{N_r} = \frac{\beta_{r2}\bar{\beta}_{v2} - (1 - p_2)\mu_r\kappa}{\beta_{v2}(\bar{\beta}_{r2} + (1 - p_2)\mu_r)}, \quad I^*_{vrt2} = \frac{I^*_{v2}}{N_v} = \frac{\beta_{r2}\bar{\beta}_{vrt2} - (1 - p_2)\mu_r\kappa}{\bar{\beta}_{vrt2}(\bar{\beta}_{r2} + \kappa)},$$

(A3)

where $\kappa = \bar{\mu}_{vrt} + m_r \cdot \bar{\mu}_{v0}/(\mu_{v0} + m_o)$. Some algebra shows that $E_2$ is biologically relevant if and only if $M_2 > 1$ ($R_2 > 1$). The strain 1 endemic equilibrium conditions reduce to a cubic equation so $E_1$ is most easily found numerically.

A special case of host switching is documented in [6], in a context of biological invasion, featuring unidirectional migration of infected vectors $I_{o1}$ into the $r$ cycle. In that study, however, the scenario under study is not different hosts but different geographical areas, with only those vectors infected with strain 1 leaving patch 1 for patch 2. This is equivalent to not only setting $m_r = 0$ but also shutting off migration altogether for $S_o$. In the case where $R_{o1} > 1$ ($M_{o1} > 1$) and $R_2 > \tilde{R}_1$ ($M_2 > \tilde{M}_1$), both strains can persist, similarly to the present model.

To consider limiting cases, the dispersal rates $m_o$ and $m_r$ in system (A2) must be multiplied by the timescale factor $\phi_r$ resulting in the following system:

$$I'_{o1} = \tilde{\beta}_{o1} S_o \frac{I_{v01}}{N_v} - \mu_o I_{o1},$$

$$I'_{v01} = \tilde{\beta}_{v01} S_v \frac{I_{v01}}{N_o} - \mu_v I_{v01} - \phi m_o I_{v01} + \phi m_r I_{v1r},$$

$$I'_{r1} = p_1 \mu_r I_{r1} + \tilde{\beta}_{r1} S_r \frac{I_{v1r}}{N_v} - \mu_r I_{r1},$$

$$I'_{v1r} = \tilde{\beta}_{v1r} S_v \frac{I_{v1r}}{N_r} - \mu_v I_{v1r} - \phi m_r I_{v1r} + \phi m_o I_{v01},$$

$$I'_{v02} = -\tilde{\mu}_{v0} I_{v02} - \phi m_o I_{v02} + \phi m_r I_{v2r},$$

$$I'_{r2} = p_2 \mu_r I_{r2} + \tilde{\beta}_{r2} S_r \frac{I_{v2r}}{N_v} - \mu_r I_{r2},$$

$$I'_{v2r} = \tilde{\beta}_{v2r} S_v \frac{I_{v2r}}{N_r} - \mu_v I_{v2r} - \phi m_r I_{v2r} + \phi m_o I_{v02}.$$  

(A4)

Equilibria, reproductive numbers and fitness measures are analogous to those given above for system (A2).

### A.2.2 Host sharing

Taking total host and vector densities as their (constant) equilibrium values, the vector–host ratios are thus constant as well, leading to a simplified system using the notation $\tilde{\beta}_{ji} = c_{ji}(Q_j) + \rho_{ji} E_j(Q_j)$, $\tilde{\mu}_v = k_v c_{vji}(Q_j)$ for $j = o, r, i = 1, 2$, and $\mu_r = \mu_v + k_o E_o(Q_o)/Q_o + k_r E_r(Q_r)/Q_r$:

$$I'_{o1} = \tilde{\beta}_{o1} S_o \frac{I_{v1}}{N_v} - \mu_o I_{o1},$$

$$I'_{r1} = p_1 \mu_r I_{r1} + \beta_{r1} S_r \frac{I_{v1}}{N_v} - \mu_r I_{r1},$$

$$I'_{v1} = \tilde{\beta}_{v1} S_v \frac{I_{v1}}{N_v} + \beta_{v1r} S_v \frac{I_{v1r}}{N_r} - \mu_v I_{v1},$$

$$I'_{r2} = p_2 \mu_r I_{r2} + \beta_{r2} S_r \frac{I_{v2}}{N_v} - \mu_r I_{r2},$$

$$I'_{v2} = \tilde{\beta}_{v2} S_v \frac{I_{v2}}{N_r} - \mu_v I_{v2}.$$  

(A5)
Although in this case the BRN for strain 2 is easily calculable (and identical to that for the one-host model with the tildes exchanged for hats), the corresponding computation for strain 1 remains unmanageable, so we shall once again use dimensionless fitness measures to describe outcomes, derived from the alternative but mathematically equivalent model in which the vertical transmission term is coalesced into the mortality term in the two raccoon equations. Then the basic reproductive measures are

\[ M_1 = \sqrt{M_{v1}^2 + M_{r1}^2}, \quad M_2 = M_{r2}, \quad \text{where } M_{v1}^2 = \frac{\hat{\beta}_{v1} \hat{\beta}_{v01}}{\mu_o \mu_v}, \quad M_{r1}^2 = \frac{\hat{\beta}_{r1} \hat{\beta}_{vri}}{(1-p_i) \mu_r \mu_v} \quad (i = 1, 2). \]

If \( M_i > 1 \) \( (i = 1, 2) \) then strain \( i \) can persist in a naïve population. If both strains’ basic fitness measures exceed 1, then persistence is determined by each strain’s invasion fitness measure, given by

\[ \hat{M}_1 = \sqrt{M_{v1}^2 \hat{s}_{v2}^* + M_{r1}^2 \hat{s}_{v2}^* \hat{s}_{v1}^*}, \quad \hat{M}_2 = M_{r2} \sqrt{\hat{s}_{v1}^* \hat{s}_{v1}^*}, \]

where \( \hat{s}_{v1}^* \) is \( S_i/N_i \) evaluated at the equilibrium where only strain \( i \) is endemic, and likewise for \( \hat{s}_{v1}^* \). Each strain can persist in a population where the other strain is [already] endemic precisely when its invasion fitness measure exceeds 1.

As with the single-host model, we can denote the disease-free (zero) equilibrium by \( E_0 \), the equilibrium in which only strain 1 is endemic by \( E_1(t_{v1}^*, N_o, t_{v1}^*, r_{v1}^*, t_{v1}^*, N_v, 0, 0) \), and the equilibrium in which only strain 2 is endemic by \( E_2(0, 0, 0, t_{v2}^*, r_{v2}^*, t_{v2}^*, N_v) \). The equilibrium conditions for \( E_2 \) are the same as for the single-host model, and thus yield

\[ t_{v2}^* = \frac{\hat{\beta}_{v2} \hat{\beta}_{v2} - (1-p_2) \mu_r \hat{\mu}_v}{\hat{\beta}_{v2} (1-p_2) \mu_r} \]

\[ (A.6) \]

clearly biologically relevant if and only if \( M_2 > 1 \) \( (R_2 > 1) \). The equilibrium conditions for \( E_1 \) yield the quadratic equation \( f(t_{v1}^*) = 0 \), where

\[ f(x) = \frac{\hat{\beta}_{v1} \hat{\beta}_{v1} + \hat{\beta}_{v1} + \hat{\mu}_v}{\mu_o (1-p_1) \mu_r \hat{\mu}_v} x^2 + \left[ \frac{\hat{\beta}_{v1} (1-p_1) \mu_r \hat{\mu}_v}{\mu_o \hat{\mu}_v} + \frac{\hat{\beta}_{v1} (1-p_1) \mu_r \hat{\mu}_v}{\mu_o (1-p_1) \mu_r \hat{\mu}_v} - \frac{\hat{\beta}_{v1} (1-p_1) \mu_r \hat{\mu}_v}{\mu_o (1-p_1) \mu_r \hat{\mu}_v} \right] x + (1-M_1^2). \]

When \( M_1 > 1 \), the constant term is negative, so there are one positive and one negative solution to the equation; the positive solution is guaranteed to be in the meaningful interval \( (0,1) \) since \( f(1) > 0 \). When \( M_1 < 1 \), the constant term is positive, but then one can show that the linear coefficient is negative, so there are no positive solutions. Thus a unique \( E_1 \) exists iff \( M_1 > 1 \).

### A.2.3 Fitness measure computations

For the host-switching model (1), the next-generation matrix takes the block-diagonal form

\[
A_1 = \begin{bmatrix}
0 & \frac{\hat{\beta}_{v1} Q_o}{m_o + \hat{\mu}_v} & 0 & 0 \\
\frac{\hat{\beta}_{v01} Q_o}{m_o} & 0 & 0 & \frac{m_r}{m_r + \hat{\mu}_v} \\
0 & 0 & p_1 & \frac{\hat{\beta}_{r1} Q_v}{m_r + \hat{\mu}_v} \\
0 & \frac{m_o}{m_o + \hat{\mu}_v} & \frac{\hat{\beta}_{v1} Q_r}{\mu_r} & 0
\end{bmatrix},
\]

where
\[
A_2 = \begin{bmatrix}
0 & 0 & \frac{m_r}{m_r + \mu_{vt}} \\
0 & p_2 & \frac{\tilde{\beta}_{2v} Q_r}{m_r + \mu_{vt}} \\
\frac{m_o}{m_o + \mu_{vo}} & \frac{\tilde{\beta}_{vt2} Q_r}{\mu_r} & 0
\end{bmatrix}.
\]

Expressions for the eigenvalues of these submatrices are complicated. However, for the mathematically equivalent system in which the vertical transmission terms in the raccoon equations are coalesced into the mortality terms, the submatrices are instead

\[
A_1 = \begin{bmatrix}
0 & \frac{\tilde{\beta}_{vo} Q_o}{m_o + \mu_{vo}} & 0 & 0 \\
\frac{\tilde{\beta}_{vo} Q_o}{\mu_o} & 0 & 0 & \frac{m_r}{m_r + \mu_{vt}} \\
0 & 0 & 0 & \frac{\mu_r}{m_r + \mu_{vt}} \\
0 & \frac{m_o}{m_o + \mu_{vo}} & \frac{\tilde{\beta}_{vt1} Q_r}{(1 - p_1) \mu_r} & 0
\end{bmatrix},
\]

\[
A_2 = \begin{bmatrix}
0 & 0 & \frac{\tilde{\beta}_{vo} k_o}{\mu_v Q_o} & \tilde{\beta}_{vo} k_o \\
0 & p_1 & \frac{\tilde{\beta}_{vt1} k_r}{\mu_{vt} Q_r} & \tilde{\beta}_{vt1} k_r \\
\frac{\tilde{\beta}_{vo} Q_o}{\mu_v k_o} & \frac{\tilde{\beta}_{vt1} Q_r}{\mu_{vt} k_r} & 0 & 0
\end{bmatrix},
\]

for which the eigenvalues can be written in a relatively straightforward way. The eigenvalues are identical in form save for signs (\(\pm\)) of the various radicals involved, so taking the + each time yields the dominant eigenvalue, as shown in the main text for the various fitness measures.

The invasion fitness measures for this model use nearly identical matrices, the only difference being that terms with a \(1/Q_j\) (\(j = o, r\)) are multiplied by \(s_{ji}^*\) and terms with a \(Q_j\) are multiplied by \(s_{vji}^*\) (where \(i\) is the resident, not the invading, strain).

Similarly, for the host-sharing system (6) the submatrices of the next-generation matrix are

\[
A_1 = \begin{bmatrix}
0 & 0 & \frac{\tilde{\beta}_{vo} k_o}{\mu_v Q_o} \\
0 & p_1 & \frac{\tilde{\beta}_{vt1} k_r}{\mu_{vt} Q_r} \\
\frac{\tilde{\beta}_{vo} Q_o}{\mu_v k_o} & \frac{\tilde{\beta}_{vt1} Q_r}{\mu_{vt} k_r} & 0
\end{bmatrix},
\]

\[
A_2 = \begin{bmatrix}
p_2 & \frac{\tilde{\beta}_{vt2} k_r}{\mu_{vt} Q_r} \\
\frac{\tilde{\beta}_{vt2} Q_r}{\mu_r k_r} & 0
\end{bmatrix}.
\]

The dominant eigenvalue of \(A_2\) can be calculated as

\[
R_2 = \frac{1}{2} \left( p_2 + \sqrt{p_2^2 + 4 \mu_{vt}^2} \right),
\]

but since the computation is more difficult for \(A_1\) we instead pass to the mathematically equivalent system in which the vertical transmission terms are coalesced into the mortality terms, which has
next-generation submatrices

\[
A_1 = \begin{bmatrix}
0 & 0 & \hat{\beta}_{o1}k_o & \hat{\beta}_{v1}k_o \\
0 & 0 & \hat{\beta}_{r1}k_r & \hat{\beta}_{vr}k_r \\
\hat{\beta}_{so}Q_o & \hat{\beta}_{sv}Q_v & 0 \\
\mu_o k_o & \mu_v Q_o & (1-p_1)\mu_r k_r \\
\end{bmatrix},

A_2 = \begin{bmatrix}
0 & \hat{\beta}_{r2}k_r \\
\hat{\beta}_{vr}Q_v & \mu_v Q_r \\
(1-p_2)\mu_r k_r & 0 \\
\end{bmatrix},
\]

which can be used to derive the basic and invasion fitness measures (again adjusting terms in the latter case to incorporate \(s^*_j\) and \(s^*_v\)).

**Appendix 3. Parameter estimates**

Parameter values used in the numerical analysis described in the results were obtained as follows.

From [23], \(\mu_o = 0.83/yr, N_o = 0.0409\text{opo/acre}, \mu_r = 0.4/yr, N_r = 0.08\text{rac/acre}, \mu_v = 0.271/yr, N_v^* = 128\text{vec/acre} \) (used only to generate \(A_v = \tilde{\mu}_v N_v^*\)), \(Q_h = 10\text{vec/host}, Q_v = 100\text{vec/host}, H = 1\text{vec/host/yr}; \) and single-cycle prevalence levels \(i^*_{o1} = 0.28, i^*_{v1} = 0.565, i^*_{r2} = 0.387, i^*_{vr} = 0.565\).

From [26], \(p_{r1} = 0.05, p_{r2} = 0.1, \beta_{o1} = 0.394/yr, \tilde{\beta}_{vo} = \beta_{vo} = 13.4/yr, \beta_{r2} = 0.225/yr, \beta_{r1} = 0.225/yr \times 0.755/0.431, \beta_{vr} = \beta_{vr} = 9.67/yr\). Also, equations (A1) in [26] were used to back-calculate \(\tilde{\beta}_{o1} = 0.571/yr\) and \(\tilde{\beta}_{r2} = 0.402/yr\).

To reflect a rough equivalence in raccoons’ and opossums’ abilities to withstand vector bites, \(m_o\) was taken to be twice \(m_r\) (since \(N_r \approx 2N_o\)), with \(m_r\) set arbitrarily to 1/yr, to be scaled by \(\phi\).

Finally, in order to replicate the observed frequency of strain 1 infection relative to strain 2 infection in raccoons in the host-switching model for \(\phi\) on the order of 1, \(\tilde{\beta}_{r1}\) was set to 0.05/yr. The result in the host-sharing model was \(M_1 = 0.83 < 1, M_2 = 1.34 > 1\), while in the host-switching model both IRNs exceed 1 for all values of \(\phi\).

Analogous parameter values to those above were taken for woodrats from [23,26].