

1 **Abstract:** Host manipulation is a common strategy of parasites with complex
2 lifecycles. It directly affects predator-prey dynamics in trophically transmitted
3 parasites. Theoretical studies suggest that predation-enhancing manipulation
4 often decimates the prey population, making parasites prone to extinction. Host
5 manipulation, however, can also suppress predation due to conflicting interests
6 when multiple parasites infect a host, often neglected in theoretical studies.
7 Misaligned interests of coinfecting parasites can occur due to limited carrying
8 capacity or parasitoid developmental stage. Including this realistic complexity
9 in a mathematical model, the results depart from previous studies substantially.
10 We show that coinfecting multi-trophic parasites can preserve the predator-prey
11 system and themselves through a combination of manipulation and reproduction
12 parameters. Our study highlights the necessity and provides the means of incor-
13 porating the reality of multiple parasites and their multi-trophic life cycles in the
14 theory of parasite ecology.

15 **Introduction**

16 Parasites infect life on earth ubiquitously, and many of these parasites have complex life
17 cycles (Zimmer, 2001). While a complex lifecycle can be defined as abrupt ontogenetic changes
18 in morphology and ecology (Benesh, 2016), a complex parasitic lifecycle typically involves
19 numerous hosts that a parasite needs to traverse to complete its life cycle. This complex
20 lifecycle results in the evolution of various strategies that enable the success of parasite
21 transmission from one host to another. One famous strategy that inspires many science
22 fiction movies and novels is host manipulation, where a parasite can alter the morphology
23 and/or behaviour of its host to enhance its transmission to the next host (Hughes et al.,
24 2012). Host manipulation has been shown in many host-parasite systems, from parasites with
25 simple life-cycle to those with complex life-cycle that involves more than one host (Hughes
26 et al., 2012; Molyneux and Jefferies, 1986). For instance, sand flies infected by *Leishmania*
27 parasites bite more and take more time for a blood meal from mammals (the definitive host of
28 *Leishmania*) compared to their uninfected counterparts (Rogers and Bates, 2007). Copepods
29 infected by cestode parasites are more active and accessible to sticklebacks (the definitive
30 hosts of the cestodes) compared to uninfected copepods (Wedekind and Milinski, 1996).

31 Theoretical studies have long attempted to understand the ecological and evolutionary
32 consequences of host manipulation. Roosien et al. (2013) and Hosack et al. (2008) showed
33 that manipulative parasites could increase the disease prevalence in an epidemic. Gandon
34 (2018) studied the evolution of the manipulative ability of infectious disease parasites, show-
35 ing different evolutionary outcomes depending on whether the pathogen can control its vector
36 or host. Hadeler and Freedman (1989); Fenton and Rands (2006) and Rogawa et al. (2018)
37 showed that host manipulation could stabilise or destabilise the predator-prey dynamics de-
38 pending on how manipulation affects the predation response function and the assumption
39 on the fertility of the definitive infected host. Seppälä and Jokela (2008) showed that host
40 manipulation could evolve even when it increases the risk of the intermediate host being
41 eaten by a non-host predator, given that the initial predation risk is sufficiently low. These

42 models, however, lack a crucial aspect of parasite dynamics, multiple infections (Kalbe et al.,
43 2002)

44 Typical studies do not consider multiple infections, a phenomenon that is the norm rather
45 than an exception in parasitism. Multiple infections result in the coinfection of more than one
46 parasite inside a host, which may alter the manipulative outcomes (figure 1). An alignment of
47 interest between coinfecting parasites may enhance manipulation, while a conflict of interest
48 may reduce the manipulative effect. Indeed, Hafer and Milinski (2015) showed that copepods
49 infected by two cestode parasites reduce the activity of copepods when both parasites are
50 at the same noninfectious stage, i.e. both parasites are not ready to transmit. Thus the
51 reduction in mobility is suggested to reduce the predation rate by the definitive hosts. When
52 two infectious parasites infect the copepods, the copepods' activity increases, and so does the
53 predation risk for the copepod. However, when the copepods are infected by one infectious
54 and one noninfectious parasite, their interests clash, and one parasite wins over the other.

55 Theoretical work that considers multiple infections often focuses on the evolution of vir-
56 ulence (van Baalen and Sabelis, 1995; Alizon et al., 2013; Alizon and van Baalen, 2008;
57 Choisy and de Roode, 2010; Alizon, 2012). They show multiple infections can increase vir-
58 ulence (van Baalen and Sabelis, 1995; Choisy and de Roode, 2010). Evolutionary branching
59 of a less virulent and a hypervirulent parasite can occur when considering within-host dy-
60 namics (Alizon and van Baalen, 2008), and a reduction in virulence is possible if parasites
61 are co-transmitted (Alizon, 2012). These studies also involve host manipulation to a certain
62 extent, as it can affect transmission rates, even though they do not explicitly consider the
63 trait. Host manipulation in trophically transmitted parasites receives less attention. Although
64 manipulation correlates with the transmission rate in trophically transmitted parasites and
65 infectious diseases, there are differences. Host manipulation influences the predation rate in
66 trophically transmitted parasites, predominantly affecting predator-prey dynamics. Theoreti-
67 cal studies on host manipulation in trophically transmitted parasites with multiple infections
68 are rare (Parker et al., 2003; Vickery and Poulin, 2009). Moreover, they do not consider the



Figure 1: Who is in control?. Schistocephalus eggs, which overwinter at the bottom of bodies of water, hatch into microscopically small swimming larvae. These larvae are eaten by copepods (also known as Cyclops due to its single eye), where they develop to the second larval stage. However, the copepod is only the first intermediate host. The larvae are then eaten by sticklebacks, where they reach the third larval stage and grow significantly in size and weight. For the parasite to successfully reach its final host, a warm-blooded animal like a bird, it manipulates its intermediate hosts. The timing is crucial as the chances of success are greatest if the larvae develop in the copepod for 13 to 15 days before entering the stickleback. The presence of multiple parasites in the same host can lead to competition and strategic decision pertaining to investment in manipulation and growth. And indeed a stickleback can be infected by numerous tapeworms as shown above by Martin Kalbe.

69 prey-predator dynamics, which will likely have important feedback on the evolution of host
70 manipulation. A few studies considering the prey-predator dynamics do not incorporate mul-
71 tiple infections (Rogawa et al., 2018; Iritani and Sato, 2018; Hadeler and Freedman, 1989;
72 Fenton and Rands, 2006). More importantly, they assume that transmission from definitive
73 hosts to intermediate hosts is due to direct contact between the two types of hosts. This
74 is often not the case, as parasites are released from the definitive hosts into the environ-
75 ment. Transmission happens only when intermediate hosts have contact with this free-living
76 parasite pool.

77 Our study addresses the gap in the theoretical work on host manipulation in trophically
78 transmitted parasites. We include multiple infections and consider the dynamics of the free-
79 living parasite pool. Our compartment model helps illustrate a parasite's complex lifecycle
80 with two hosts: an intermediate host preyed upon by a definitive host. Transmission from the
81 intermediate host to the definitive host occurs when predation on infected intermediate hosts
82 happens. Reproduction only happens in the definitive hosts. New parasites then enter the
83 environment, where the cycle continues. We focus on the intermediate host manipulation,
84 such that the parasite increases the uptake of the intermediate host by the definitive host
85 to increase its transmission rate. We then analyse the effect of host manipulation on the
86 ecological dynamics in the prey-predator-parasite system. In contrast to the abovementioned
87 examples, our model consists of a single intermediate host as it already provides enough
88 complexity to discuss between transmission and manipulation. We found that sabotage in
89 host manipulation almost always pushes the dynamical system toward bistability, provided the
90 reproduction in a single infection is sufficiently small. The bistable nature suggests that the
91 predator-prey parasite system is finely balanced and susceptible to extinction via ecological
92 disturbances. Initially surprising, we showed that cooperation in host manipulation and
93 enhanced reproduction in co-infecting parasites is not always beneficial and might expose
94 the parasite population to the risk of extinction.

95 **Model and Results**

96 Our model concerns the complex lifecycle of a trophically transmitted parasite that requires
97 two hosts: an intermediate host and a definitive host. Reproduction only happens inside the
98 definitive hosts, releasing new parasitic progeny in the environment. An intermediate host
99 can be infected if it encounters this free-living parasite pool. Finally, when a definitive host
100 consumes an infected intermediate host, the definitive host gets infected, and the parasite
101 completes its lifecycle.

102 For simplicity, we assume that hosts can be infected by one (single infection) or, at most,

103 two parasites (double infections). Our model is, therefore, more relevant to the macroparasitic system. Given that infection occurs, the probability that two parasites from the parasite pool co-transmit to an intermediate host is denoted by p . Thus $1 - p$ is the probability that a single parasite enters an intermediate host. When a definitive host consumes an intermediate host infected by two parasites, there is a probability q that the parasites co-transmit to the definitive host. With probability $1 - q$, only one parasite successfully transmits. This formulation assumes that infection always happens when hosts encounter parasites. The dynamics of a complex lifecycle parasite that requires two hosts is described by the following system of equations, firstly for the intermediate host as,

$$\begin{aligned}
 \frac{dI_s}{dt} &= R(I_s, I_w, I_{ww}) - dI_s - P_s(D_s, D_w, D_{ww})I_s - \eta I_s \\
 \frac{dI_w}{dt} &= (1 - p)\eta I_s - (d + \alpha_w)I_w - P_w(D_s, D_w, D_{ww}, \beta_w)I_w \\
 \frac{dI_{ww}}{dt} &= p\eta I_s - (d + \alpha_{ww})I_{ww} - P_{ww}(D_s, D_w, D_{ww}, \beta_{ww})I_{ww}
 \end{aligned} \tag{1}$$

112 where $R(I_s, I_w, I_{ww})$ represents the birth rate of the intermediate hosts, a function of both
 113 infected and uninfected individuals. P_s , P_w , P_{ww} are the predation functions of definitive
 114 hosts on susceptible, singly infected and doubly infected intermediate hosts. The predation
 115 function depends on the density of the definitive hosts and the manipulative strategies of
 116 parasites in the intermediate hosts. In particular, if a single parasite infects an intermediate
 117 host, the manipulation strategy is β_w . However, if the intermediate host is co-infected, the
 118 manipulation strategy is β_{ww} . In the scope of this model, we assume no specific relationship
 119 between β_w and β_{ww} to explore all possible ecological outcomes of the system. The force
 120 of infection by parasites in the environment is denoted by $\eta = \gamma W$. Since parasites can ma-
 121 nipulate intermediate and definitive hosts, here, whenever we mention host manipulation, it
 122 specifically refers to the manipulation in intermediate hosts, which correlates to the predation
 123 rate.

124 For the definitive hosts we have,

$$\begin{aligned}\frac{dD_s}{dt} &= B(D_s, D_w, D_{ww}, I_s, I_w, I_{ww}) - \mu D_s - (\lambda_{ww} + \lambda_w)D_s \\ \frac{dD_w}{dt} &= (\lambda_w + (1-q)\lambda_{ww})D_s - (\mu + \sigma_w)D_w - ((1-q)\lambda_{ww} + \lambda_w)D_w \\ \frac{dD_{ww}}{dt} &= q\lambda_{ww}D_s + ((1-q)\lambda_{ww} + \lambda_w)D_w - (\mu + \sigma_{ww})D_{ww}\end{aligned}\quad (2)$$

125 where $B(D_s, D_w, D_{ww}, I_s, I_w, I_{ww})$ represents the birth rate of definitive hosts. The birth
126 rates depend on the density of both intermediate and definitive hosts, infected or uninfected.
127 The force of infection that corresponds respectively to singly infected intermediate host (I_w)
128 and doubly infected intermediate hosts (I_{ww}) is denoted respectively by $\lambda_w = h(\rho + \beta_w)I_w$
129 and $\lambda_{ww} = h(\rho + \beta_{ww})I_{ww}$, where ρ is the baseline predation rate and h is the probability
130 that the parasite successfully establishes inside the host. If there is no manipulation, that is,
131 $\beta_w = \beta_{ww} = 0$, the parasite is still transmitted via the base line predation. The dynamics
132 of the free-living parasites in the environment are then given by,

$$\frac{dW}{dt} = f_w D_w + f_{ww} D_{ww} - \delta W - \eta I_s. \quad (3)$$

133 Definitions of different parameters can be found in Table SI.1.

134 Here, we focus on manipulation that enhances transmission from intermediate hosts to
135 definitive hosts; we thus simplify the transmission from the parasite pool to intermediate
136 hosts such that no sequential infection. This assumption is motivated given that the prey'
137 lifecycle is often shorter than that of the predator. A prey likely encounters the free-living
138 parasite pool once and then dies due to predation, making sequential transmission less likely
139 at this state. Sequential infection can happen when parasites transmit from intermediate
140 hosts to definitive hosts. Therefore, a singly infected definitive host can be further infected
141 by another parasite if it consumes infected intermediate hosts. Figure (2) illustrates the
142 system's dynamics.

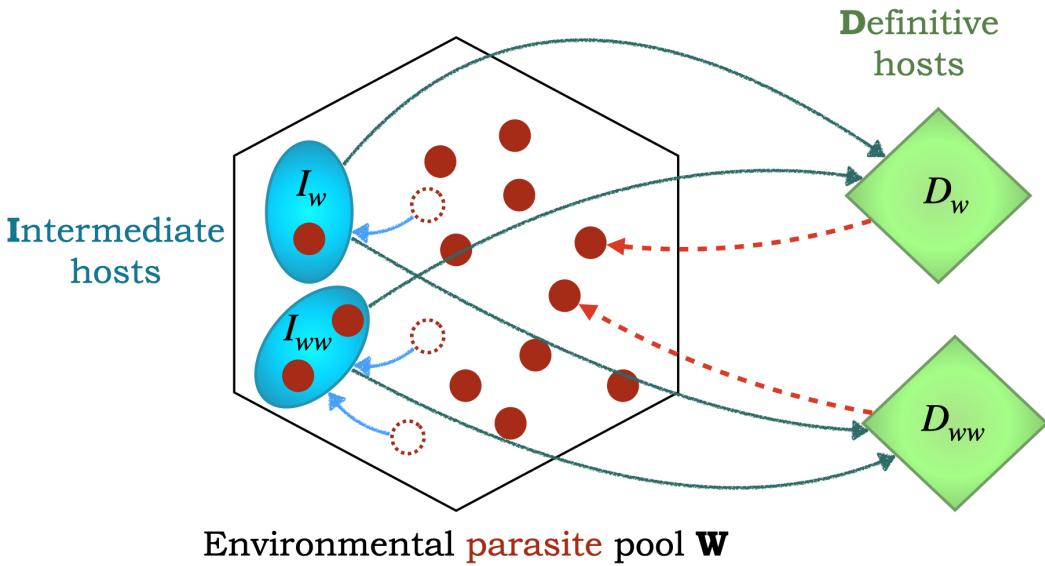


Figure 2: Schematic of the model. Blue ovals represent intermediate host compartment, green diamonds represent definitive host compartment, and the transparent hexagon represents the parasite pool compartment with red circles illustrating individual parasites.

¹⁴³ **Basic reproduction ratio R_0 of the parasites**

¹⁴⁴ The basic reproduction ratio R_0 (or basic reproduction number as often used in epidemiology)
¹⁴⁵ indicates parasite fitness. It can be understood as the expected number of offspring a parasite
¹⁴⁶ produces during its lifetime when introduced to a susceptible host population. We calculate
¹⁴⁷ the basic reproduction ratio R_0 using the next-generation method (Diekmann et al., 1990,
¹⁴⁸ 2009; Hurford et al., 2010) (See SI1 for details).

$$R_0 = \underbrace{\gamma I_s^* \frac{pqh(\rho + \beta_{ww})}{\alpha_{ww} + d + P_{ww}} \frac{D_s^*}{\mu + \sigma_{ww}} \frac{f_{ww}}{\delta + \gamma I_s^*}}_{\text{Double infections}} + \underbrace{\gamma I_s^* \left(\frac{(1-p)h(\rho + \beta_w)}{\alpha_w + d + P_w} + \frac{p(1-q)h(\rho + \beta_{ww})}{\alpha_{ww} + d + P_{ww}} \right) \frac{D_s^*}{\mu + \sigma_w} \frac{f_w}{\delta + \gamma I_s^*}}_{\text{Single infection}} \quad (4)$$

¹⁴⁹ where I_s^* and D_s^* are the densities of susceptible intermediate and definitive hosts at the
¹⁵⁰ disease-free equilibrium. Here, the expression of R_0 contains the possible reproduction routes

151 of a parasite, which can be via double or single infections. The first component corresponds
 152 to the double infections route, in which the focal parasite co-transmits with another par-
 153 asite into a susceptible intermediate host, then co-transmits into a susceptible definitive
 154 host and reproduces. Here, parasites are so rare that only co-transmission matters and the
 155 compartments with sequential infections are therefore neglected. The second component
 156 corresponds to the single infection route, wherein the focal parasite infects a susceptible
 157 intermediate host via single or double infections. The parasite then transmits alone into the
 158 susceptible definitive host and eventually reproduces.

159 If $R_0 > 1$, a parasite spreads when introduced into the disease-free equilibrium of prey and
 160 predator. Intuitively, the higher the density of susceptible intermediate and definitive hosts,
 161 the larger the value of R_0 as the infection reservoir is more extensive. In contrast, regardless
 162 of the explicit form of the predation function, the higher the predation rate P_w and P_{ww} , the
 163 lower the value of R_0 given the smaller reservoir of intermediate hosts. The effect of host
 164 manipulation on the value of R_0 is not so straightforward; as host manipulation becomes
 165 efficient, the transmission rate from the intermediate host to the definitive host increases,
 166 but so does the predation rate. A higher predation rate results in a smaller intermediate
 167 host reservoir available for the parasites to infect. To understand the effect of manipulation
 168 on parasites' fitness and the system's ecological dynamics, we next specify the predation
 169 functions. We consider linear functions for predation to begin with,

$$P_s(D_s, D_w, D_{ww}) = \rho D_{total}$$

$$P_w(D_s, D_w, D_{ww}, \beta_w) = (\rho + \beta_w) D_{total}$$

$$P_{ww}(D_s, D_w, D_{ww}, \beta_{ww}) = (\rho + \beta_{ww}) D_{total}$$

170 where $D_{total} = D_s + D_w + D_{ww}$ is the total density of the definitive hosts, and ρ is the
 171 baseline capture rate of the predator on the prey. If an intermediate host is infected, it is
 172 captured by the definitive hosts with rate $\rho + \beta_w$ if it is singly infected and with rate $\rho + \beta_{ww}$

173 if it is doubly infected. Zero values for β_w and β_{ww} suggest no manipulation, and predation
174 is at the baseline value ρ .

175 For simplicity, we also consider a linear function of the birth of definitive hosts

$$B(D_s, D_w, D_{ww}, I_s, I_w, I_{ww}) = \rho c D_{total} I_{total}$$

176 where c is the efficiency of converting prey into predator's offspring, and $I_{total} = I_s + I_w + I_{ww}$
177 is the total density of the intermediate hosts. It is important to note that host manipulation
178 affects the population dynamics via its influence on predation rate but not the physiological
179 aspect of the definitive host, i.e. the predator. The birth rate of the predators thus depends
180 on the capture rate, but it is not affected by host manipulation; as to our best knowledge,
181 there is no supporting evidence to consider otherwise.

182 The explicit form of I_s^* and D_s^* , capturing the predator-prey dynamics, depends on the
183 precise form of all birth and predation functions B, R, P_s, P_w and P_{ww} . But, it does not
184 depend on the manipulation ability or any other parameter of the parasite. Given that the
185 birth rate of the predator and the predation rate are linear functions in prey and predator
186 density, the form of the birth rate R of the prey has a significant effect on the susceptible
187 intermediate and definitive host dynamics.

188 Birth function of intermediate hosts

189 The simplest form of the prey's birth rate is a linear function, in which case the disease
190 free equilibrium is always unstable. In particular, it has a cyclic behaviour because, at this
191 equilibrium, the jacobian matrix of the system (1, 2, 3) always has two pure imaginary
192 eigenvalues (see SI2). This follows from the Lotka-Volterra system using linear functions for
193 prey birth and predation (Lotka, 1920). Since the disease-free dynamics is cyclic, it is difficult
194 to analyse the spread of a parasite using the basic reproduction ratio, which is evaluated when
195 the disease-free state is stable. Here, $R_0 > 1$ happens when γ , the transmission rate from

196 the environment to intermediate hosts, and the reproduction rates f_w, f_{ww} are significantly
197 large (the specific mathematical conditions can be found in SI3). However, even when this
198 condition is satisfied, the parasite may not be able to spread and persist in cyclic susceptible
199 host dynamics (Figure SI1). This result agrees with the conclusion in (Ripa and Dieckmann,
200 2013), which suggests that it is difficult for a mutant to invade a cyclic resident population.
201 In our case, it is not the invasion of a mutant in a resident population but the invasion of
202 a parasite in a cyclic disease-free host population; the argument, however, remains valid in
203 both cases. This issue deserves a more thorough investigation, which is out of the scope of
204 this article. Here, we choose a non-linear birth function of the intermediate hosts to obtain a
205 stable disease circulation state and focus on the effect of host manipulation on the ecological
206 dynamics.

207 The logistic growth for the non-linear birth function follows by

$$R(I_w, I_s, I_{ww}) = rI_{total}(1 - kI_{total})$$

208 where k is the intraspecific competition coefficient. The disease-free equilibrium is as follows

$$I_s^* = \frac{\mu}{c\rho} ; D_s^* = \frac{c\rho(r - d) - k\mu r}{c\rho^2}$$

209 This equilibrium is positive and stable if components of the parasite, such as reproduction
210 and transmission are sufficiently small, details of the condition can be found in section SI 4.
211 (Figure 3B).

212 When a parasite appears in the disease-free equilibrium, it spreads if its reproduction ratio
213 $R_0 > 1$. Since the expression is complicated, we could not obtain analytical solutions for
214 this inequality without assumptions. We assume the same parasite virulence, $\alpha_w = \alpha_{ww}$,
215 $\sigma_w = \sigma_{ww}$, and reproduction in double infection as a linear function concerning reproduction
216 in single infections, $f_{ww} = \epsilon f_w$. When $\epsilon > 1$, reproduction in double infections is enhanced
217 as compared to in single infections, whereas $\epsilon \leq 1$, reproduction in double infections is

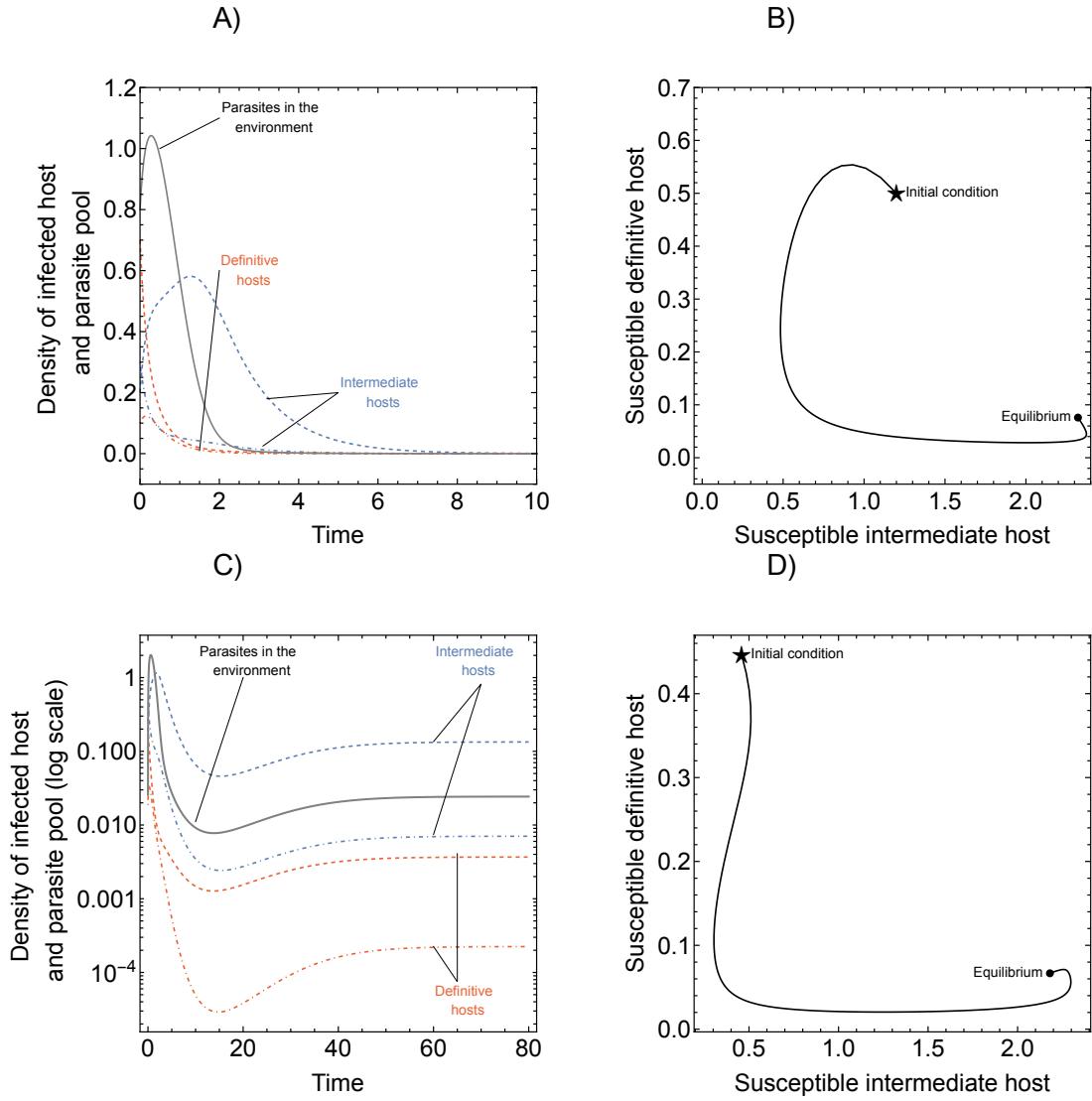


Figure 3: A, B) Disease free equilibrium where parasite cannot persist. C, D) Disease stable equilibrium. Solid gray line indicate the density of free-living parasites, blue lines indicate infected intermediate hosts while red lines indicate infected definitive hosts. Dashed lines indicate singly infected hosts while dot-dashed lines indicate doubly infected hosts. Parameters for disease free equilibrium $\rho = 1.2$, $d = 0.9$, $r = 2.5$, $\gamma = 2.9$, $\alpha_w = \alpha_{ww} = 0$, $\beta_w = \beta_{ww} = 1.5$, $p = 0.05$, $c = 1.4$, $\mu = 3.9$, $\sigma_w = \sigma_{ww} = 0$, $q = 0.05$, $f_w = f_{ww} = 7.5$, $\delta = 0.9$, $k = 0.26$, $h = 0.6$. Disease stable equilibrium have the same parameter values except for higher host manipulation $\beta_w = \beta_{ww} = 4.5$ and parasite reproduction $f_w = f_{ww} = 45$

218 depressed or equal to reproduction in single infections. We found that the parasite can
219 establish if its reproduction value in a single infection f_w is more significant than a threshold
220 (Figure 4, see section SI 5 and Eq. (SI.19)).

221 Our numerical results show that the parasite reproduction is substantial compared to other
222 parameters (its value is nearly 40 times greater than other parameters). This observation
223 suggests that trophically transmitted parasites must release many offspring into the environ-
224 ment to persist. Interestingly, bistability occurs if the reproduction rate of the parasite in
225 double infections is enhanced (Figure 4A). In the bistable region, the parasite population can
226 reach a stable equilibrium if the initial density is large enough. In contrast, with sufficient
227 disturbance, the parasite population could go extinct.

228 **The effect of host manipulation on ecological dynamics**

229 Host manipulation can be cooperative; two parasites increase the predation rate on inter-
230 mediate hosts, or $\beta_{ww} > \beta_w$. However, it can also be uncooperative; the predation rate on
231 doubly-infected intermediate hosts is lower than that on singly-infected ones or $\beta_{ww} < \beta_w$.
232 Cooperation in parasite manipulation increases the parasite's basic reproduction ratio R_0 ,
233 but the manipulation in a single infection substantially affects the value of R_0 (Figure 5
234 Left). Intuitively, if the manipulation in a single infection is minor, there is not enough
235 transmission, and the parasite goes extinct. However, suppose the ability to manipulate the
236 host in a single infection is merely enough for the parasite population to escape extinction.
237 In that case, cooperation in host manipulation leads to a bistable system state. Within the
238 bistable region, the basic reproduction ratio can be less than one, suggesting that the parasite
239 cannot spread when its manipulative values are within this area of weak manipulation when
240 coinfecte

241 Co-infecting parasites can influence each other in different life history traits besides ma-
242 nipulation. Parasites can have an enhanced reproduction rate in coinfections, i.e. $f_{ww} > f_w$.
243 Likewise, they can compete for resources, so reproduction in double infection is depressed

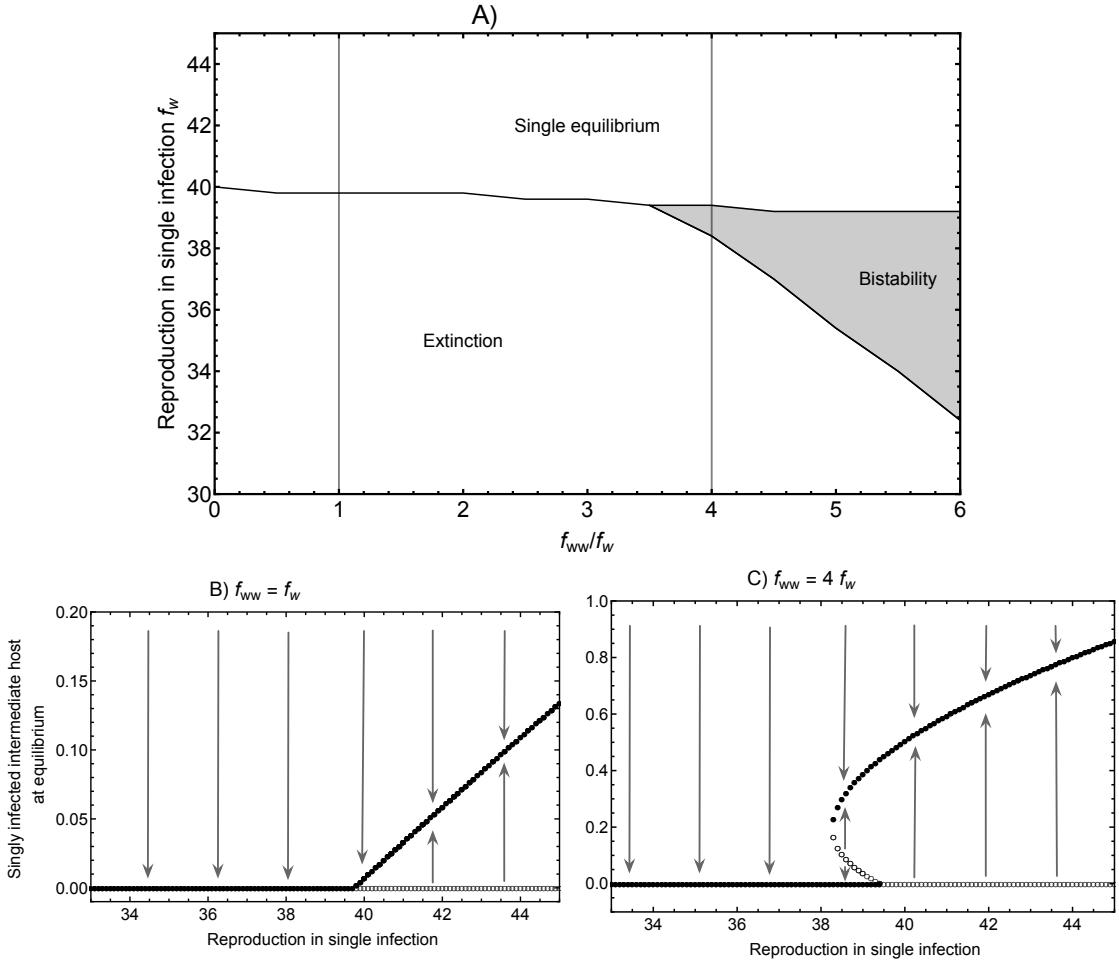


Figure 4: Effect of parasite reproduction on the ecological dynamics. A) Enhanced reproduction in double infection leads to bistability, B, C) Density of singly infected host at equilibrium when reproduction of parasites are the same in singly and doubly infected hosts $f_{ww} = f_w$, and when reproduction of parasites in doubly infected hosts is enhanced four times than those in singly infected hosts $f_{ww} = 4f_w$. Filled circles indicate stable equilibrium and open circles indicate unstable equilibrium. Parameter $\rho = 1.2$, $d = 0.9$, $r = 2.5$, $\gamma = 2.9$, $\alpha_w = 0$, $\alpha_{ww} = 0$, $\beta_w = 1.5$, $\beta_{ww} = 1.5$, $p = 0.05$, $c = 1.4$, $\mu = 3.9$, $\sigma_w = 0$, $\sigma_{ww} = 0$, $q = 0.05$, $\delta = 0.9$, $k = 0.26$, $h = 0.6$.

as compared to in single infection. Without any assumption on the relationship between manipulative ability and reproduction, we explore all possible combinations of cooperation-sabotage range in manipulation and depressed-enhanced range in reproduction. If parasites are uncooperative in manipulations and shows depressed reproduction, they cannot persist

248 (Figure 5). In contrast, if they are highly cooperative in manipulation and show enhanced re-
249 production (i.e. $\beta_{ww}/\beta_w \rightarrow \infty$ and $f_{ww}/f_w \rightarrow \infty$), there is a guaranteed single equilibrium
250 for parasite existence.

251 For intermediate levels of coordination in reproduction and manipulation, a bistable area
252 could occur. However, the size of this area is sensitive to the value of reproduction and
253 manipulation in a single infection. In particular, higher values of these two parameters
254 reduce the bistability area, whereas larger values increase the bistability area (Figure 5,
255 Figure SI.1). If the parasites sabotage each other, the system is highly prone to bistability
256 and only has a single equilibrium when reproduction is especially enhanced. Interestingly,
257 sufficiently high reproduction enhancement leads to bistability (i.e. f_{ww} is at least four times
258 f_w), and depressed reproduction always leads to a single equilibrium of the system (Figure
259 5). While a single equilibrium guarantees the existence of a parasite population, bistability
260 indicates that a disturbance of the system may likely lead to the extinction of the parasite
261 population. This suggests that the benefits of coordination in reproduction and manipulation
262 are context-dependent. Coordinating holds an advantage if there are no significant tradeoffs
263 and if reproduction or manipulation in single infections are large enough.

264 Co-transmission probability from the parasite pool to intermediate hosts p has the opposite
265 effect on the bistable area compared to co-transmission probability q from intermediate hosts
266 to intermediate hosts (Figure 6). In particular, when the parasite sabotages the manipula-
267 tion, increasing p enlarges the bistable area, whereas increasing q reduces it. In contrast,
268 when parasites cooperate in manipulation, reducing p decreases the bistable area while re-
269 ducing q widens it. If cooperation in manipulation is exceptionally high, the population will
270 always exist with one stable equilibrium regardless of the co-transmission value. However,
271 as there are always limitations and trade-offs, high values may not be possible. Bistability
272 indicates vulnerability to disturbance, suggesting that cooperation in manipulation may be
273 beneficial when the co-transmission from the pool to the intermediate host increases. How-
274 ever, cooperation in manipulation may harm the population when the co-transmission from

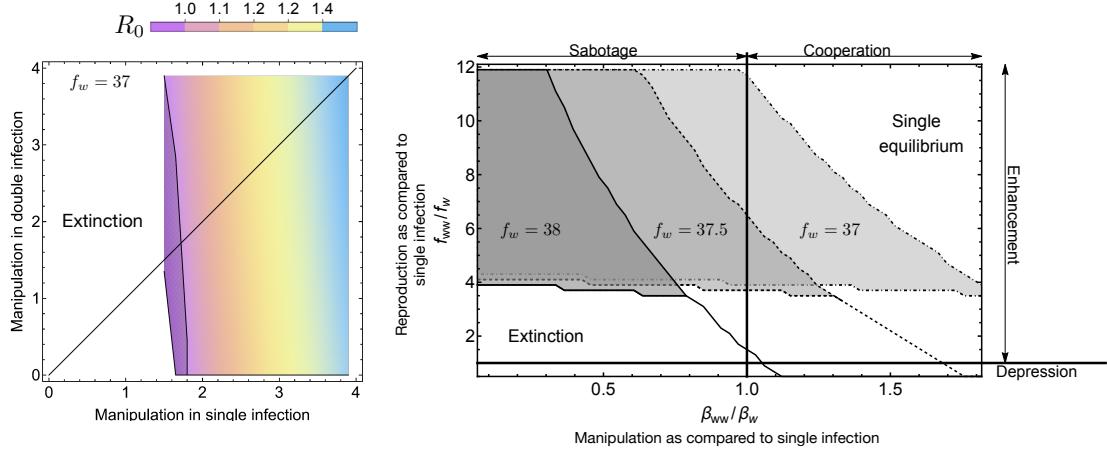


Figure 5: Left: R_0 values increase with more efficient manipulation in both single and double infection. Hatched area indicates bistable region. As manipulation in single infection increases, the system only has one stable equilibrium. On the black line, manipulation is indifference between single infection and double infection ($\beta_w = \beta_{ww}$). Right: Changes of the bistability area (shaded areas) with respect to different reproduction rates in single infection (different boundary styles). Manipulation and reproduction is indifference between single infection and double infection on the vertical and horizontal lines respectively. Common parameter: $\rho = 1.2$, $d = 0.9$, $r = 2.5$, $\gamma = 2.9$, $\alpha_w = 0$, $\alpha_{ww} = 0$, $p = 0.05$, $c = 1.4$, $\mu = 3.9$, $\sigma_w = 0$, $\sigma_{ww} = 0$, $q = 0.05$, $\delta = 0.9$, $k = 0.26$, $\beta_w = 1.65$, $h = 0.6$.

275 the intermediate host to the definitive host increases.

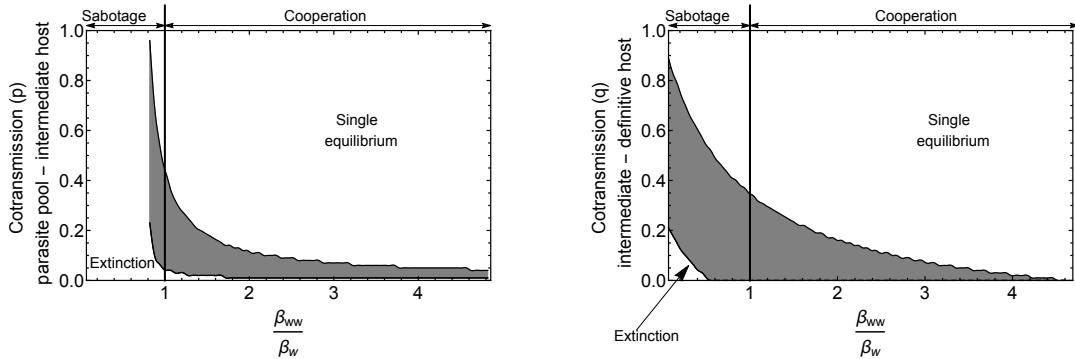


Figure 6: Left: Effect of cotransmission from parasite pool to intermediate host. Right: Effect of cotransmission from intermediate to definitive host. Common parameters: $\rho = 1.2$, $d = 0.9$, $r = 2.5$, $\gamma = 2.9$, $\alpha_w = 0$, $\alpha_{ww} = 0$, $p = 0.05$, $c = 1.4$, $\mu = 3.9$, $\sigma_w = 0$, $\sigma_{ww} = 0$, $q = 0.05$, $\delta = 0.9$, $k = 0.26$, $\epsilon = 4.5$, $\beta_w = 1.45$, $f_w = 38$, $h = 0.6$.

276 **Discussion & Conclusion**

277 Host manipulation is a ubiquitous phenomenon suggested to affect the prey-predator dynam-
278 ics in trophically transmitted parasites. In particular, manipulation of infected intermediate
279 hosts to increase the predation rate of definitive hosts may result in a heavy burden of preda-
280 tors on the intermediate host population. This pressure can make parasites more vulnerable
281 to extinction (Hadeler and Freedman, 1989; Fenton and Rands, 2006).

282 Our model shows that parasites cannot spread quickly in a cyclic predator-prey system.
283 This delay is an expected result since even though the parasite's basic reproduction ratio R_0
284 is larger than one, it is estimated at the predator and prey's unstable equilibrium (or cyclic
285 equilibrium). Thus, when the density of the prey and predator is at the minimum value of the
286 cycle, the "effective" R_0 of the parasite can be smaller than one. Another interesting result
287 is that the reproduction value is much larger than other parameter values. This result is
288 likely due to the introduction of a free-living parasitic pool. Our model shows that in making
289 the system more realistic, we also obtain a more realistic quantitative value for parasitic
290 reproduction.

291 In the study by Rogawa et al. (2018), a non-manipulative parasite can invade a susceptible
292 prey-predator population and cause the system to cycle. The system stops cycling and
293 approaches a fixed point when the parasite becomes manipulative, and this stability increases
294 with increased manipulation. In our model, non-manipulative parasites cannot persist in the
295 system, and the parasite never leads to cyclic dynamics. These results may contradict with
296 Rogawa et al. (2018), where non-manipulative parasites can still exists via cyclic behaviour.
297 We suggest that the different results may be due to our introduction of a parasite pool and
298 multiple infections, unlike the model of Rogawa et al. (2018). In their system, transmission
299 from the definitive host to the intermediate host was assumed to result from direct contact
300 between the two hosts. Such immediate transmission could directly accelerate the feedback
301 loop between prey and predator. Hence, faster predator-prey dynamics occur, which may
302 lead to cyclic dynamics when parasites are introduced.

303 In our study, population dynamics exhibit bistability under certain circumstances. This
304 is very likely due to the introduction of co-transmission, which has been shown to result in
305 bistable population dynamics in plant virus Allen et al. (2019) and infectious diseases Gao
306 et al. (2016). In this bistability region, if the system is disturbed (e.g. migration of the
307 intermediate or definitive hosts or predation of intermediate hosts by other predators), then
308 the density of the infected hosts may crash, leading to parasite extinction. The bistability
309 region widens as parasites show enhanced reproduction but sabotage manipulation. This
310 extension is because the density of the doubly infected hosts is always much smaller than
311 the singly infected hosts, limited by sequential transmission and a small probability of co-
312 transmission. If manipulation in a single infection is not sufficient then the transmission of
313 the parasites depends mainly on the doubly infected hosts, which is rare. So, extinction is
314 possible if manipulation in double infections is low.

315 Iritani and Sato (2018) show that manipulative parasites persist if they can alternate
316 manipulation between boosting and suppressing predation rate. In our model, the para-
317 site cannot switch its manipulative strategy. Sabotaging manipulation reduces the basic
318 reproduction ration R_0 and makes the system bistable, exposing the parasite to the risk of
319 extinction. This result contrasts with Iritani and Sato (2018) because in our model, sabotage
320 decreases transmissmion rate from intermediate to definitive host, and does not benefit the
321 parasite.

322 Finally, our study focuses on the ecological dynamics of the trophically transmitted para-
323 site. However, investigating the evolution of host manipulation is a natural extension beyond
324 the scope of a single manuscript, given the complexities that arise in the ecological dynamics
325 itself. Studying the evolution of host manipulation, considering the free-living parasite pool,
326 calls for thorough analyses, which could be a standalone study. For example, we would need
327 to include differences between the traits of the multiple parasites and hence the ecological
328 model becomes more complex than presented in this study. The combinatorics and orderings
329 of sequential infections wil lthen become important. In addition, the occurrence of bistabil-

330 ity in our model suggests that the evolution of host manipulation may drive the parasite to
331 extinction simply because of the rarity of the mutant and the Allee effect as per Adaptive
332 dynamics approaches. The coinfecting parasites can increase manipulation and enhance re-
333 production freely if there exist no tradeoffs. Nevertheless, our model shows that the benefits
334 of this strategy are context-dependent, making it suboptimal in certain cases. Evolutionary
335 dynamics would therefore depend on the tradeoff between host manipulation and other traits
336 of the parasites, such as reproduction, virulence, and survivorship in the parasite pool, to list
337 a few. This extension deserves thorough analysis, and we will treat it as a separate matter.

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Supplementary Information:

On multiple infections by parasites with complex life cycles

SI 1. Reproduction ratio R_0

The reproduction ratio of the parasite is derived from the dynamical system of the parasite which only include infected intermediate and definitive hosts and the free-living parasite pool. The dynamical system can be written in matrix form as followed:

$$\frac{d\mathbf{n}}{dt} = \mathbf{M}\mathbf{n}$$

where \mathbf{n} is the vector of singly and doubly infected intermediate hosts, singly and doubly infected definitive hosts and free-living parasites ($dI_w, I_{ww}, D_w, D_{ww}, W$) and \mathbf{M} is the matrix that describes the dynamics

$$\mathbf{M} = \begin{pmatrix} -d - \alpha_w - P_w & 0 & 0 & 0 & (1-p)\gamma I_s \\ 0 & -d - \alpha_{ww} - P_{ww} & 0 & 0 & p\gamma I_s \\ h(\beta_w + \rho)D_s & h(\beta_{ww} + \rho)(1-q)D_s & -\lambda_w - (1-q)\lambda_{ww} - \mu - \sigma_w & 0 & 0 \\ 0 & h(\beta_{ww} + \rho)qD_s & \lambda_w + (1-q)\lambda_{ww} & -\mu - \sigma_{ww} & 0 \\ 0 & 0 & f_w & f_{ww} & -\delta - \gamma I_s \end{pmatrix}$$

The matrix \mathbf{M} can be written as $\mathbf{M} = \mathbf{F} - \mathbf{V}$, where

$$\mathbf{F} = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & f_w & f_{ww} & 0 \end{pmatrix}$$

is the matrix in which its elements are the reproduction contribution of one compartment to the other compartments in the next generation, and

$$\mathbf{V} = \begin{pmatrix} \alpha_w + d + P_w & 0 & 0 & 0 & -(1-p)\gamma I_s \\ 0 & \alpha_{ww} + d + P_{ww} & 0 & 0 & -p\gamma I_s \\ -h(\rho + \beta_w)D_s & -h(\rho + \beta_{ww})(1-q)D_s & \lambda_w + \lambda_{ww}(1-q) + \mu + \sigma_w & 0 & 0 \\ 0 & -h(\rho + \beta_{ww})qD_s & -\lambda_w - \lambda_{ww}(1-q) & \mu + \sigma_{ww} & 0 \\ 0 & 0 & 0 & 0 & \delta + \gamma I_s \end{pmatrix}$$

is the matrix in which its elements include death rates or transition rates from one compartment to the others (Diekmann et al., 1990, 2009; Hurford et al., 2010).

The reproduction ratio R_0 is then the leading eigenvalue of the matrix $\mathbf{F}\mathbf{V}^{-1}$, evaluated at the disease-free equilibrium of the intermediate and definitive hosts I_s^* , D_s^* , and $I_w = I_{ww} = D_w = D_{ww} = 0$.

SI 2. Equilibrium stability - linear birth function for intermediate hosts

The jacobian matrix of the system of equations (1), (2), and (3), as given in the main text is evaluated at the disease-free equilibrium, and $B(D_s, D_w, D_{ww}, I_s, I_w, I_{ww}) = \rho c D_{total} I_{total}$ is

$$\begin{pmatrix} 0 & r & r & -\frac{\mu}{c} & -\frac{\mu}{c} & -\frac{\mu}{c} & -\frac{\gamma\mu}{c\rho} \\ 0 & -\alpha_w + \frac{(\beta_w+\rho)(d-r)}{\rho} - d & 0 & 0 & 0 & 0 & \frac{\gamma\mu(1-p)}{c\rho} \\ 0 & 0 & -\alpha_{ww} + \frac{(\beta_{ww}+\rho)(d-r)}{\rho} - d & 0 & 0 & 0 & \frac{\gamma\mu p}{c\rho} \\ -c(d-r) & \frac{\beta_w(d-r)}{\rho} - c(d-r) & \frac{\beta_{ww}(d-r)}{\rho} - c(d-r) & 0 & \mu & \mu & 0 \\ 0 & -\frac{\beta_w(d-r)}{\rho} & -\frac{\beta_{ww}(1-q)(d-r)}{\rho} & 0 & -\mu - \sigma_w & 0 & 0 \\ 0 & 0 & -\frac{\beta_{ww}q(d-r)}{\rho} & 0 & 0 & -\mu - \sigma_{ww} & 0 \\ 0 & 0 & 0 & 0 & f_w & f_{ww} & -\frac{\gamma\mu}{c\rho} - \delta \end{pmatrix}$$

This jacobian has seven eigenvalues, two of which have explicit expressions as $\pm\sqrt{d-r}$. Here, we always have $r > d$ so that the equilibrium is positive, therefore these two eigenvalues are always pure imaginary. We cannot obtain the explicit expression of the other five eigenvalues but the dynamics remain unstable regardless of their values.

SI 3. Invasion of parasite - Linear birth function

$R_0 > 1$ when the transmission rate from the parasite pool to intermediate hosts satisfies

$$\gamma > \frac{c\delta\rho(\mu + \sigma_w)(\mu + \sigma_{ww})(\beta_w(r-d) + \rho(\alpha_w + r))(\beta_{ww}(r-d) + \rho(\alpha_{ww} + r))}{\mu} \times \frac{1}{\left(\begin{array}{l} d^2(f_w h(\mu + \sigma_{ww})(\beta_{ww}(1-p)\rho + \beta_w \beta_{ww}(1-pq) + \beta_w p(1-q)\rho) - \\ \beta_w(\mu + \sigma_w)(\beta_{ww}(\mu + \sigma_{ww}) - f_{ww}hpq(\beta_{ww} + \rho))) + \\ d(f_w h(\mu + \sigma_{ww})(-\alpha_{ww}(1-p)\rho(\beta_w + \rho) - \alpha_w p(1-q)\rho(\beta_{ww} + \rho) - 2\beta_w \beta_{ww}r(1-pq)) + \\ \beta_w \rho r(p(2q-1) - 1) + \rho r(\beta_{ww}(pq + p - 2) + \rho(pq - 1))) + \\ (\mu + \sigma_w)((\mu + \sigma_{ww})(\beta_{ww}\rho(\alpha_w + r) + \beta_w \rho(\alpha_{ww} + r)) + \\ 2\beta_w \beta_{ww}r) - f_{ww}hpq(\beta_{ww} + \rho)(\rho(\alpha_w + r) + 2\beta_w r)) + \\ f_w h r(\mu + \sigma_{ww})(\alpha_{ww}(1-p)\rho(\beta_w + \rho) + \alpha_w p(1-q)\rho(\beta_{ww} + \rho) + r(\beta_w + \rho)(\beta_{ww} + \rho)(1-pq)) - \\ (\mu + \sigma_w)(\alpha_w \rho + r(\beta_w + \rho))(\alpha_{ww}\rho(\mu + \sigma_{ww}) + r(\beta_{ww} + \rho)(-f_{ww}hpq + \mu + \sigma_{ww})) \end{array} \right)} \quad (\text{SI.1})$$

and the reproduction rates f_w and f_{ww} satisfies either of the following conditions

$$f_{ww} \geq \frac{(\mu + \sigma_{ww})(-\alpha_{ww}\rho + \beta_{ww}d - r(\beta_{ww} + \rho))}{hpq(\beta_{ww} + \rho)(d - r)} \quad (\text{SI.2})$$

or

$$f_{ww} < \frac{(\mu + \sigma_{ww})(-\alpha_{ww}\rho + \beta_{ww}d - r(\beta_{ww} + \rho))}{hpq(\beta_{ww} + \rho)(d - r)} \quad (\text{SI.3})$$

$$f_w > \frac{(\mu + \sigma_w)(-\alpha_w \rho + \beta_w d - r(\beta_w + \rho))}{h(d - r)(\mu + \sigma_{ww})} \times \frac{(r - d)(\beta_{ww}(\mu + \sigma_{ww}) - f_{ww}hpq(\beta_{ww} + \rho)) + \rho(\mu + \sigma_{ww})(\alpha_{ww} + r)}{\left(\begin{array}{l} d(-\beta_{ww}(1-p)\rho + \beta_w \beta_{ww}(-(1-pq)) - \beta_w p(1-q)\rho) + \\ \alpha_{ww}(1-p)\rho(\beta_w + \rho) + \alpha_w p(1-q)\rho(\beta_{ww} + \rho) + r(\beta_w + \rho)(\beta_{ww} + \rho)(1-pq) \end{array} \right)} \quad (\text{SI.4})$$

SI 4. Equilibrium stability - Non-linear birth function for intermediate hosts

The disease free equilibrium of the system of equations (1), (2), (3) given in the main text is

$$I_s^* = \frac{\mu}{c\rho} \quad (\text{SI.5})$$

$$D_s^* = \frac{c\rho(r-d) - k\mu r}{c\rho^2} \quad (\text{SI.6})$$

D_s^* is positive if $X = c\rho(r-d) - k\mu r$ is positive.

The eigenvalues of the Jacobian matrix established at the disease free equilibrium are roots of the following polynomial:

$$A_4\lambda^4 + A_3\lambda^3 + A_2\lambda^2 + A_1\lambda + A_0 \quad (\text{SI.7})$$

The disease free equilibrium is stable if the above polynomial has all negative real roots. Using the Descarte rule, the polynomial has all negative real root if all the coefficients are positive.

We know that $A_4 = 1$ is always positive.

$$A_3 = \rho^7 (X(\beta_w + \beta_{ww} + 2\rho) + c\rho^2(2\alpha + \delta + \mu + \sigma + 2d) + \gamma\mu\rho) \quad (\text{SI.8})$$

is always positive as all the elements of A_3 are positive.

$$\begin{aligned} A_2 = & \rho^{14} (c\rho^3(c\rho(\alpha^2 + 2\alpha(\delta + \mu + \sigma) + \delta(\mu + \sigma)) + (2\alpha + d + \mu + \sigma)(cd\rho + \gamma\mu) + cd\rho(2\delta + \mu + \sigma) + \gamma d\mu) \\ & + X(\beta_w + \beta_{ww} + 2\rho)(c\rho^2(\alpha + d + \delta + \mu + \sigma) + \gamma\mu\rho) + X^2(\beta_w + \rho)(\beta_{ww} + \rho)) \end{aligned} \quad (\text{SI.9})$$

is always positive because all elements of A_2 are positive.

$$A_1 = \rho^{22} (c^2\rho^2 A_{10} + A_{11} - c\gamma X A_{12} f_w h \mu \rho^2) \quad (\text{SI.10})$$

is positive if reproduction in single infection f_w , the probability to successfully established in the definitive host h , and cooperation in reproduction ϵ are small enough because

$$A_{10} = \alpha\rho^2(\alpha\gamma\mu + \alpha c\rho(\delta + \mu + \sigma) + 2(\mu + \sigma)(c\delta\rho + \gamma\mu)) \quad (\text{SI.11})$$

is always positive and

$$A_{11} = c\rho^2(2cd\rho\rho + X(\beta_w + \beta_{ww} + 2\rho))(\alpha\gamma\mu + \alpha c\rho(\delta + \mu + \sigma) + (\mu + \sigma)(c\delta\rho + \gamma\mu)) + \quad (\text{SI.12})$$

$$cd\rho^2(c\rho(\delta + \mu + \sigma) + \gamma\mu)(cd\rho^2 + X(\beta_w + \beta_{ww} + 2\rho)) + \quad (\text{SI.13})$$

$$X^2(\beta_w + \rho)(\beta_{ww} + \rho)(c\rho(\delta + \mu + \sigma) + \gamma\mu) \quad (\text{SI.14})$$

is always positive.

$$A_{12} = \beta_w(1 - p) + p(\beta_{ww} + q(\epsilon - 1)(\beta_{ww} + \rho)) + \rho \quad (\text{SI.15})$$

is always positive because $0 \leq p \leq 1$ and $0 \leq q \leq 1$. If $\epsilon > 1$, then A_{12} is always positive. The smaller the value of ϵ , the more likely A_{12} is negative. However, even when $\epsilon = 0$, $A_{12} = \beta_w(1 - p) + \beta_{ww}p(1 - q) + \rho(1 - pq)$ is always positive. Because A_{12} is positive, if f_w , h and ϵ are sufficiently large, A_1 can be negative and the polynomial will not have all negative eigenvalues.

Finally, we have

$$A_0 = c^9\rho^{31}(-f_w h \gamma \mu A_{00} + A_{01}) \quad (\text{SI.16})$$

where

$$A_{00} = X(c\rho^2(\alpha + d)A_{12} + X(\beta_w + \rho)(\beta_{ww} + \rho)(pq(\epsilon - 1) + 1)) \quad (\text{SI.17})$$

is always positive, and

$$A_{01} = (\mu + \sigma)(c\delta\rho + \gamma\mu)(c^2\rho^4(\alpha + d)^2 + cX\rho^2(\beta_w + \rho)(\alpha + d) + cX\rho^2(\beta_{ww} + \rho)(\alpha + d) + X^2(\beta_w + \rho)(\beta_{ww} + \rho)) \quad (\text{SI.18})$$

is always positive. Therefore, if f_w , h , and ϵ are sufficiently large, A_0 could be negative, leading to non-negative eigenvalues of the polynomial.

For all the above argument, the disease free equilibrium is positive and stable if f_w , h and ϵ are sufficiently small, even though we cannot deduce the explicit expression for the condition.

SI 5. Non-linear birth function for intermediate hosts - invasion condition

The condition for parasite invasion is $R_0 > 1$, which is satisfied when

$$f_w > \frac{\left((\mu + \sigma)(c\delta\rho + \gamma\mu)(k\mu r(\beta_w + \rho) - c\rho(\beta_w(-d) + \rho(\alpha + r) + \beta_w r))(k\mu r(\beta_{ww} + \rho) - c\rho(\beta_{ww}(-d) + \rho(\alpha + r) + \beta_{ww}r)) \right)}{\left(\gamma h\mu(c\rho(d - r) + k\mu r)(k\mu r(\beta_w + \rho)(\beta_{ww} + \rho)(pq(\epsilon - 1) + 1) - c\rho(\beta_{ww}d(p - 1)\rho - \beta_w d(\beta_{ww} + \beta_{ww}pq(\epsilon - 1) + p\rho(q(\epsilon - 1) + 1)) + \alpha\rho(\beta_w + \beta_w(-p) + p(\beta_{ww} + \beta_{ww}q(\epsilon - 1) + q\rho(\epsilon - 1)) + \rho) + r(\beta_w + \rho)(\beta_{ww} + \rho)(pq(\epsilon - 1) + 1)) \right)} \quad (\text{SI.19})$$

Tables

Supplementary Figure

References

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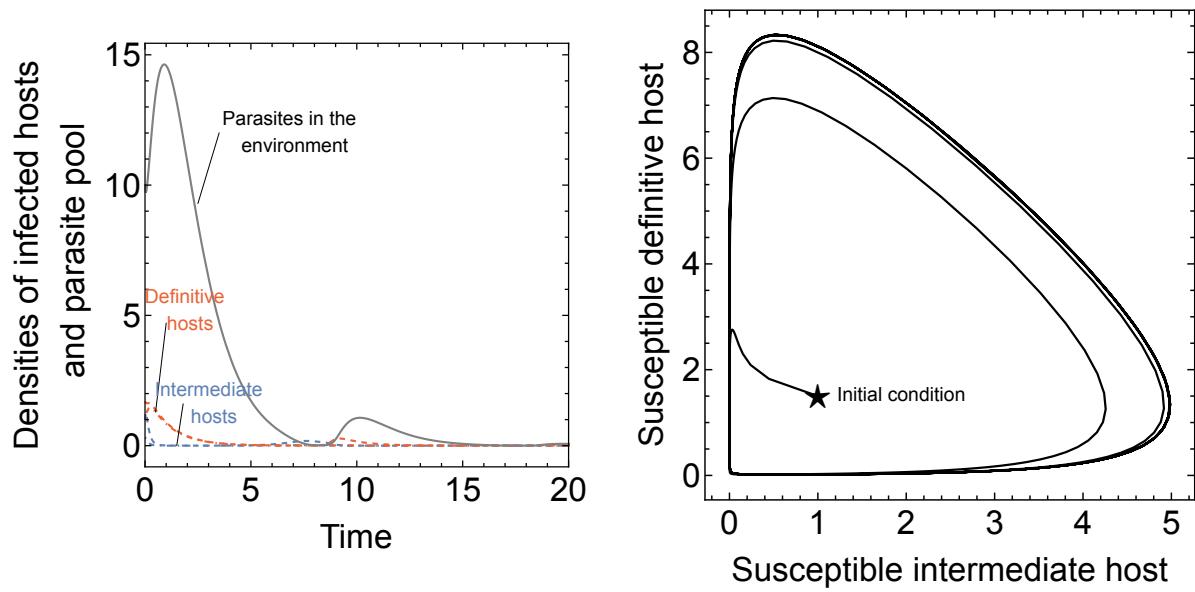


Figure SI.1: Disease-free equilibrium using linear birth function, where parasite goes extinct (left panel), and susceptible hosts demonstrate cyclic dynamics (right panel). Solid gray line indicate the density of free-living parasites, blue lines indicate infected intermediate hosts while red lines indicate infected definitive hosts. Dashed lines indicate singly infected hosts while dot-dashed lines indicate doubly infected hosts. Parameter values $\rho = 1.2$, $d = 0.9$, $r = 2.5$, $\gamma = 2.9$, $\alpha_w = \alpha_{ww} = 0$, $\beta_w = 1.5$, $\beta_{ww} = 1.5$, $p = 0.1$, $c = 1.4$, $\mu = 0.9$, $\sigma_w = \sigma_{ww} = 0$, $q = 0.01$, $f_w = 6.5$, $f_{ww} = 7.5$, $\delta = 0.9$, $h_1 = h_2 = 0.8$, $R_0 = 4.997$

Table SI.1: Description of variables and parameters

Parameters and Variables	Description
I_i	Density of intermediate hosts that are susceptible $i = s$, singly infected $i = w$, or doubly infected $i = ww$
D_i	Density of definitive hosts that are susceptible $i = s$, singly infected $i = w$, or doubly infected $i = ww$
W	Density of parasites released from definitive hosts into the environment
d	Natural death rate of intermediate hosts
α_i	Additional death rate of intermediate hosts due to infection by a single parasite ($i = w$) or two parasites ($i = ww$)
p	Probability that two parasites cotransmit from the environment to an intermediate host
γ	Transmission rate of parasites in the environment to intermediate hosts
μ	Natural death rate of definitive hosts
σ_i	Additional death rate of definitive hosts due to infection by a single parasite ($i = w$) or two parasites ($i = ww$)
σ_i	Additional death rate of the hosts due to being infected by a singly parasite ($i = w$) or two parasites ($i = ww$)
q	Probability that two parasites cotransmit from intermediate hosts to definitive hosts
β_i	Transmission rate of parasites from intermediate hosts to definitive hosts
f_i	Reproduction rate of parasites in singly infected definitive hosts ($i = w$) or doubly infected hosts ($i = ww$)
δ	Natural death rate of parasites in the environment
h	Probability that the parasites successfully established inside the definitive host