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Host-parasite Red Queen dynamics archived in pond sediment

Ellen Decaestecker[1, 3], Sabrina Gaba[4, 5], Joost A.M. Raeymaekers[1, 2], Robby Stoks[1], Liesbeth Van Kerckhoven[1], Dieter Ebert[4*] and Luc De Meester[1*]

[1]*Laboratory of Aquatic Ecology and Evolutionary Biology,*

[2]*Laboratory for Animal Biodiversity and Systematics, KULeuven, Ch. de Bériotstraat 32, 3000 Leuven, Belgium*

[3]*Interdisciplinary Research Center (IRC), KULeuven Campus Kortrijk, E. Sabbelaan 53, 8500 Kortrijk, Belgium*

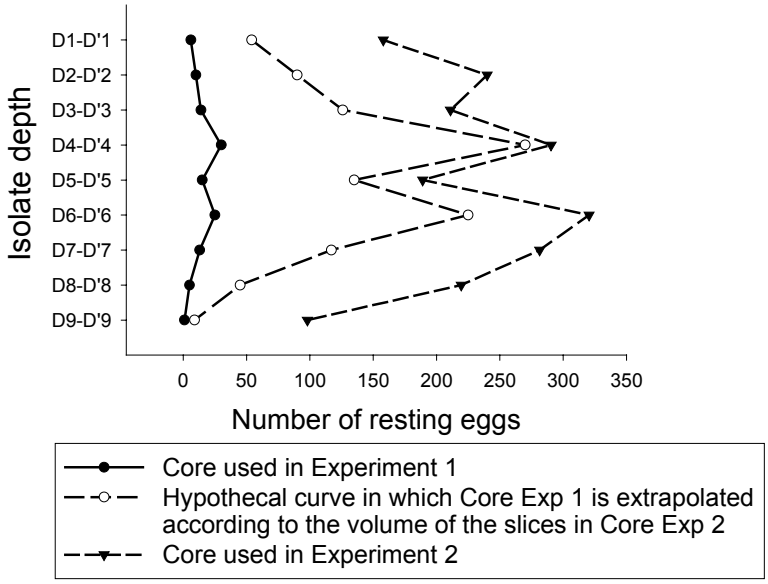
[4]*Universität Basel, Zoologisches Institut, Evolutionsbiologie, Vesalgasse 1, 4053 Basel, Switzerland*

[5]*INRA, UR1282, Infectiologie Animale et Santé Publique, Nouzilly, F- 37380, France*

** These authors contributed equally*

SI1. Dormant egg profile in the sediment cores.

There was a consistent profile in the number of dormant eggs over the different sediment depths of both cores, reflecting a low level of bioturbation in the sediment of our pond.



SI Figure 1. Number of *D. magna* dormant eggs in different sediment layers of cores. D1-D8, being the depths used in Experiment 1 and D'1-D'7 in Experiment 2 (D1-D'1 = 8-10 cm; D2-D'2 = 10-12 cm, …).

SI2. Clone depth x time shift interaction

The pattern of higher infectivity in contemporary associations was consistent across depths in Experiment 1, in which three parasite isolates per depth were used (clone depth x time shift interaction, $P = 0.92$). The use of a single parasite isolate per depth induced considerable stochasticity across depths in Experiment 2 (clone depth x time shift interaction, $P < 0.0001$). The significance of this interaction in a general analysis including all depths could be attributed to two depths (D5, D7 in Exp. 2), confounding our ability to estimate the odds ratios for the time shift main effect. Consequently, we performed an additional analysis including all but these two. Excluding the non-significant interaction term, the results on the time shift effect in the Logistic Regression on infectivity were: goodness of fit (Dev./d.f.) = 1.3, $P = 0.0003$ with the

contrast analyses on the log odds ratios for C – P: odds ratio = 1.76, $P = 0.0054$; for C – F: odds ratio = 2.01, $P = 0.0006$; where C, P and F correspond to contemporary, past and future, respectively.

SI3. Genetic continuity of the host population

We analysed ten *D. magna* genotypes from four depth layers for genetic variation at six polymorphic DNA microsatellite loci using standard protocols[34]. We quantified F_{ST} values (GENETIX 4.02[35]) as a measure of genetic differentiation among sediment layers. The results of this analysis yielded a low level of genetic differentiation through time for these neutral markers ($F_{ST} = 0.047$), which is consistent with a similar study on a different *D. magna* population[34]. In comparison, typical F_{ST} values among populations from similar habitats range from 0.2 to 0.8[36].

SI4. Supplementary information: model

The primary goal of this study was to test the patterns we found in our experiments. We present a simple simulation model derived from general coevolution models[37, 38]. It was designed to verify if a simple model of antagonistic coevolution could recreate the pattern observed from the analysis of the sediment core samples. Therefore, the model includes the specific aspects of the experimental design of the core study.

We assume that both the host and the parasite have constant and sufficiently large population sizes to avoid stochastic effects (e.g. genetic drift) on gene frequencies. Both interacting species evolve in a well-mixed population and have discrete, non-overlapping generations. We assume further that the parasite is haploid and the host is

diploid, and reproduce asexually. Mutations occur at both host alleles independent of each other. The infection success of the parasites depends on both host and parasite genotypes. The genetic determinism of parasite infectivity (host resistance) is governed by one locus with n alleles.

Following Gandon and Michalakis[37], the infection rate is proportional to the frequency of parasite genotypes and depends on the type of interaction between host and parasite. Different interactions are possible, but we focus here on the matching allele model (MAM, SI Table 1), which may underlie the genetic interaction in the *Daphnia magna-Pasteuria ramosa* system[39].

We further assume that the host and the parasite have identical generation times. Parasite virulence V measures the deleterious effect of the parasite on the infected host. *P. ramosa* is highly virulent, leading to strong reduction of *Daphnia* fecundity, hence in our simulations presented here, the virulence is close to its maximum (V = 0.95), as estimated by ref [40].

For each generation, both species are exposed to selection and mutation. Selection for infectivity in the bacteria and resistance in the *Daphnia* are described by the following set of difference equations describing the average change in p_i and h_j, the frequency of the i^{th} parasite genotype and the j^{th} host genotype, respectively (the superscript t and t+1 refer to successive generations):

$$p_i^{t+1} = p_i^t \frac{W_{i,t}^p}{\overline{W}_t^p} + \frac{1}{\overline{W}_t^p} \sum_{l=1}^{I} \left(p_l^t W_{l,t}^p \mu_{li} - p_i^t W_{i,t}^p \mu_{il} \right) \qquad \text{eqn1}$$

$$h_j^{t+1} = h_j^t \frac{W_{j,t}^H}{\overline{W}_t^H} + \frac{1}{\overline{W}_t^H} \sum_{j=1}^{J} (h_j^t W_{k,t}^H \mu_{kj} - h_j^t W_{j,t}^H \mu_{jk}) \qquad \text{eqn2}$$

where $W_{i,t}^{P}$ and $W_{j,t}^{H}$ are the relative fitness of the i^{th} parasite genotype and the j^{th} host genotype, respectively, at the t^{th} generation as defined by Gandon and Michalakis[37] with *M(i,j)* the interaction matrix:

$$W_{i,t}^{P} = \sum_{j=1}^{I} M(i,j) h_j^t \qquad \text{eqn3}$$

$$W_{j,t}^{H} = 1 - V \sum_{i=1}^{J} M(i,j) p_i^t \qquad \text{eqn4}$$

and $\overline{W}_t^{P}$ and $\overline{W}_t^{H}$ are the mean fitnesses in the parasite population and in the host population, respectively, at the t^{th} generation:

$$\overline{W}_t^{P} = \sum_{i=1}^{I} W_{i,t}^{P} p_i^t \qquad \text{eqn5}$$

$$\overline{W}_t^{H} = \sum_{j=1}^{J} W_{j,t}^{H} h_j^t \qquad \text{eqn6}$$

The second part of equations eqn1 and eqn2 describes mutation events. Mutation may occur independently on the locus with probability μ_h (in our simulation $\mu_h = \mu_{jk} = \mu_{kj} = 10^{-6}$) in the host and in the parasite (in our simulation $\mu_p = \mu_{il} = \mu_{li} = 10^{-6}$).

Measures of parasite performance. Two measures of parasite performance are used to check whether parasites are temporally adapted. Contemporary performance measures the average infectivity of the parasite (*P. ramosa*) when exposed to a contemporary host (*Daphnia*), i.e., both are sampled in the same moment. Allotemporary performance measures the average infectivity of the host when exposed to parasites from previous or future generations (analogue to contemporary and allotemporary combinations in the sediment core experiment). In analogy to local adaptation experiments in a

metapopulation, contemporary represents sympatric combinations, while allotemporary represents novel or allopatric combinations[41].

Contemporary performance (C) is a measure of the average infectivity of the host when interacting with parasites from the same generation. We calculate the average over T generations.

$$C = 1/T \sum_{t=1}^{T} \sum_{i=1}^{I} \sum_{j=1}^{J} M(i,j) h_{j,t} p_{i,t} \qquad \text{eqn7}$$

Allotemporary performance ($A(\alpha)$) is a measure of average infectivity of a host population to parasite populations from a different generation. α indicates the time shift between the parasite generation and the host generation, the host generation being the reference here. For example, $A(+1)$ measures the performance of the host when it interacts with the parasite of the next future generation. In the model, we explore the adaptation for $\alpha \in \{-30 \text{ to } 30 \text{ by } 1\}$ but without $\alpha=0$ because $A(\alpha=0)=C$. The measure of allotemporary performance is given by the following equations:

- when hosts interact with parasites from future generations ($\alpha>0$)

$$A(\alpha) = 1/(T-\alpha) \sum_{t=1}^{T-\alpha} \sum_{j=1}^{J} \sum_{i=1}^{I} M(i,j) h_{j,t} p_{i,t+\alpha} \qquad \text{eqn8a}$$

- when hosts interact with parasites from previous generations ($\alpha<0$)

$$A(\alpha) = 1/(T-|\alpha|) \sum_{t>|\alpha|}^{T} \sum_{j=1}^{J} \sum_{i=1}^{I} M(i,j) h_{j,t} p_{i,t+\alpha} \qquad \text{eqn8b}$$

In sediment core experiments, each slice contains material from several generations. Thus, a sample from a slice represents an average across these generations. To take this into account, we averaged over 5, 11, 15 and 21 generations in order to check the

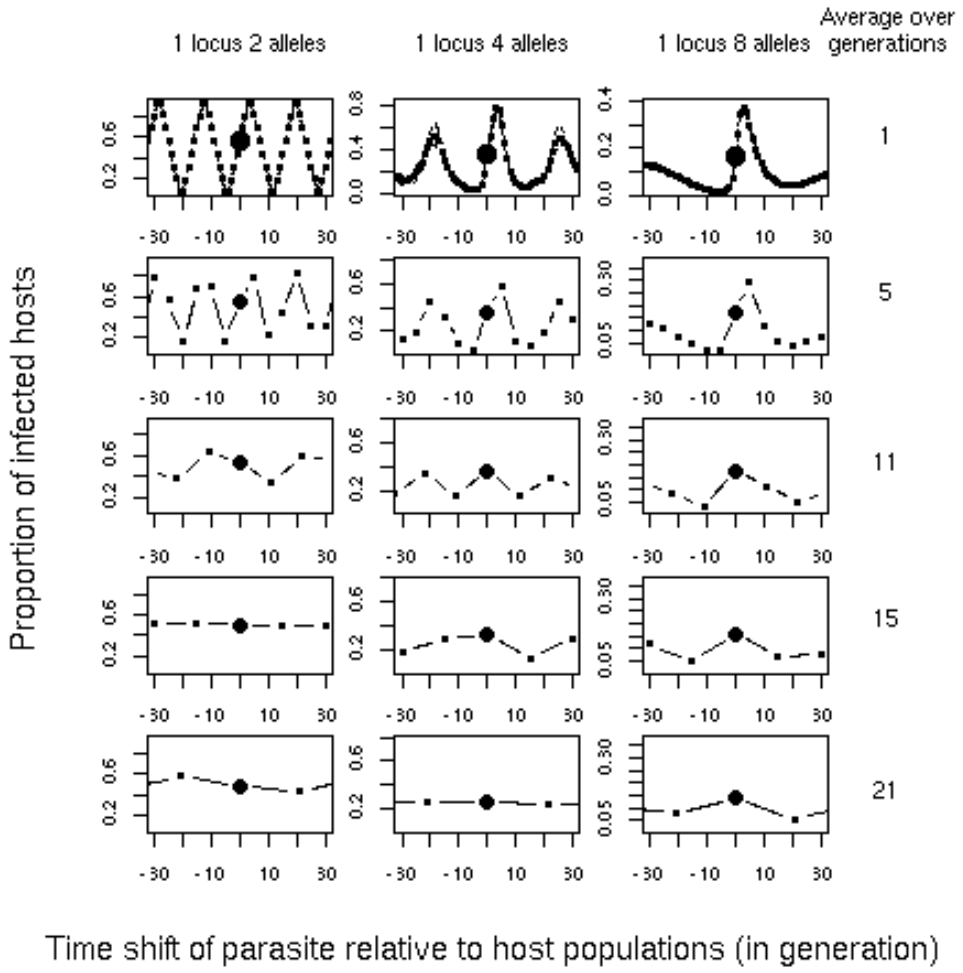
evolutionary dynamic of host-parasite interaction under experimental situations that approximate our study.

Simulations. Both species are subject to selection and mutation in every generation. Each simulation started with random initial allele frequencies. After the foundation event, simulations were deterministic; hence simulations were run for 1000 generations before we collected the statistics. We investigated the robustness of the pattern to the parameter values and to the number of alleles for parasite virulence and host resistance. Ten runs were performed for each analysis. Statistics were calculated for the last 1000 generations of each run (T = 1000).

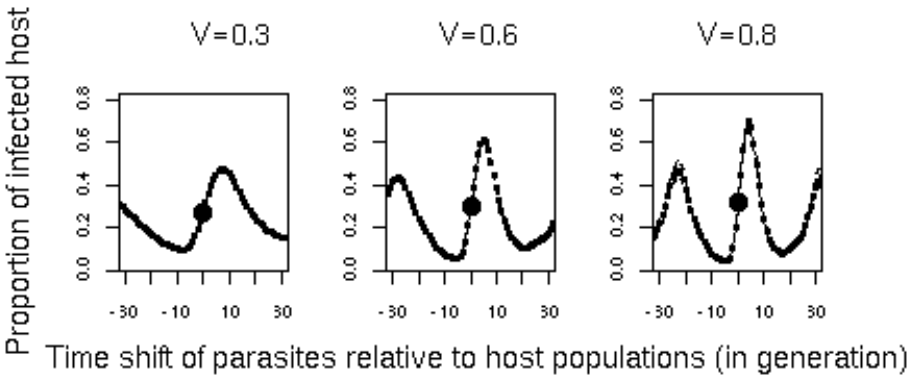
SI Table 1. Compatibility matrix of parasite and host genotypes defined by a matching allele model (MAM) with 1 locus and 4 alleles (as described in ref [42]). Resistance of the host is recessive.

	AA	*Aa*	*aa*
A	1	1	0
a	0	1	1

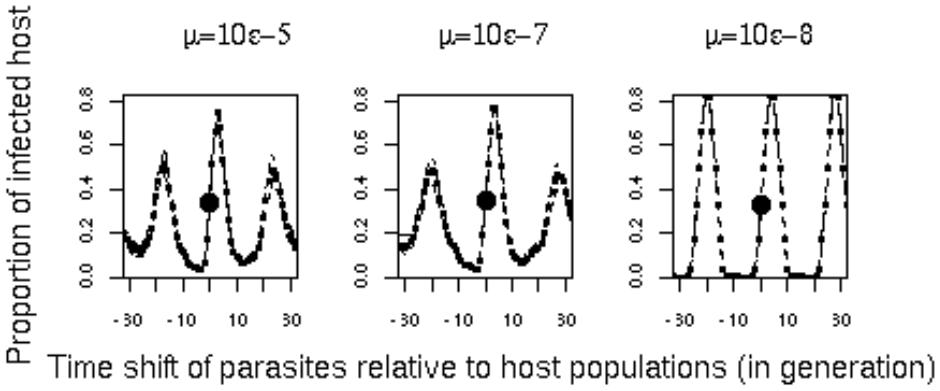
The first column shows the parasite alleles and the first row the host alleles. The other cells give the infectivity: 1 indicates that the parasite infects successfully, whereas 0 indicates that the host resists. Resistance of the host is recessive.



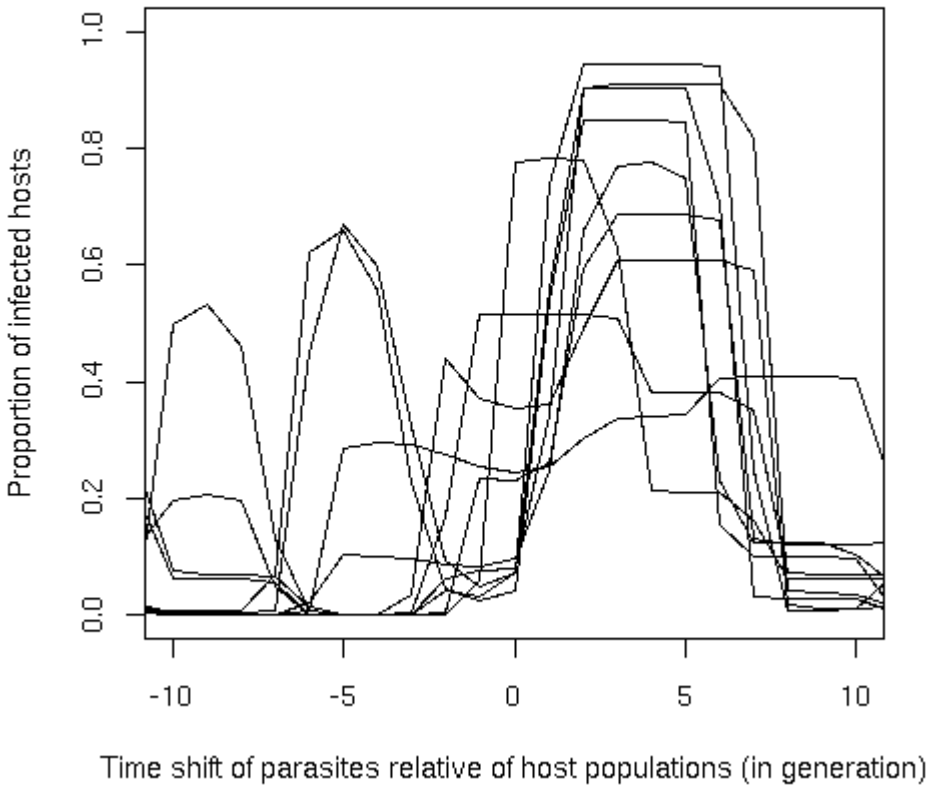
SI Figure 2. Relationship between the proportion of infected hosts and the time shift of parasites relative to the host population for different genetic determinism: 1 locus with 2, 4 or 8 alleles. The bigger dot indicates the infectivity of the parasite population interacting with its contemporary host population (time shift = 0), whereas the small dots show the infectivity of past and future parasites when exposed to a standard host population ($\alpha \neq 0$). The genetics underlying the *Daphnia-Pasteuria* system are currently unknown, but it has recently been suggested that they are based on a simple system with few, possible only one locus[43].



SI Figure 3. Relationship between the proportion of infected hosts and the time shift of parasites relative to the host population for different degrees of virulence, V. The dark line represents the infection rate averaged over the 500 last generations and the scatter around the line represents the 95% confidence interval. The dot indicates the infection rate of the parasite population interacting with its contemporary host population. Each point represents one generation (no averaging was done). Resistance (infectivity) is determined by 1 locus, 4 alleles and $\mu_h = \mu_p = 10^{-6}$. Virulence of *Pasteuria* is very high around V=0.95[44].



SI Figure 4. Relationship between the proportion of infected hosts and the time shift of parasites relative to the host population for different mutation rates, μ. Each point represents one generation (no averaging was done). Resistance (infectivity) is determined by 1 locus 4 alleles, V = 0.95 and $\mu = \mu_p = \mu_h$. Higher mutation rates promote faster dynamics.



SI Figure 5. Model simulation of parasite infectivity as a function of the time shift of the parasites relative to the host population, given for 10 moments from the 1000 last generations. Each line represents a specific host population and its calculated interactions with parasites from the contemporary (time shift = 0) and the 12 previous and future generations.

SI5. References to Supplementary Notes SI1-SI4

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