Learning-induced switching costs in a parasitoid can maintain diversity of host aphid phenotypes although biocontrol is destabilised under abiotic stress

Katharine F. Preedy*[a], Mark A.J. Chaplain[b], Daniel J. Leybourne[c], Glenn Marion[d] and Alison J. Karley[c]

[a]Biomathematics and Statistics Scotland, Invergowrie, DD2 5DA
[b]Department of Mathematics and Statistics, University of St Andrews
[c]The James Hutton Institute, Invergowrie, DD2 5DA
[d] Biomathematics and Statistics Scotland, James Clerk Maxwell Building, Edinburgh, EH9 3FD

*Corresponding author: katharine.preedy@bioss.ac.uk

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[*]katharine.preedy@bioss.ac.uk

Summary

1. Aphid populations frequently include phenotypes that are resistant to parasitism by hymenopterous parasitoid wasps, which is often attributed to the presence of ‘protective’ facultative endosymbionts residing in aphid tissues, particularly *Hamiltonella defensa*. In field conditions, under parasitoid pressure, the observed coexistence of aphids with and without protective symbionts cannot be explained by their difference in fitness alone.

2. Using the cereal aphid *Rhopalosiphum padi* as a model, we propose an alternative mechanism whereby parasitoids are more efficient at finding common phenotypes of aphid and experience a fitness cost when switching to the less common phenotype.

3. We construct a model based on delay differential equations and parameterise and validate the model with values within the ranges obtained from experimental studies. We then use it to explore possible effects on system dynamics under conditions of environmental stress, using our existing data on the effects of drought stress in crops as an example.

4. We show the ‘switching penalty’ incurred by parasitoids leads to stable coexistence of aphids with and without *H. defensa* and provides a potential mechanism for maintaining phenotypic diversity amongst host organisms. We show that drought-induced reduction in aphid development time has little impact. However, greater reduction in fecundity on droughted plants of symbiont-protected aphids can cause insect population cycles when the system would be stable in the absence of drought stress.

5. The stabilising effect of the increased efficiency in dealing with more commonly encountered host phenotypes is applicable to a broad range of consumer-resource systems and could explain stable coexistence in competitive environments. The loss of stable coexistence when drought has different effects on the competing aphid phenotypes highlights the importance of scenario testing when considering biocontrol for pest management.

Keywords

climate change, drought, *H. defensa*, mathematical model, parasitoid, symbiont

Introduction

Understanding the biotic and abiotic factors regulating insect pest dynamics is critical for reducing agriculture's reliance on pesticides and developing non-chemical alternatives. Progress in this area is often constrained, however, by limited knowledge and supporting data about factors regulating pest populations. Aphids are successful herbivores and crop pests (Dixon, 1985), feeding on the phloem sap of plants and inflicting damage by removing plant resources and transmitting economically damaging plant viruses (Stevens and Lancomme, 2017). Like many plant-feeding insects, they can be infected by facultative bacterial endosymbionts that alter insect fitness traits (Zytynska et al., 2019, preprint), but evidence is scarce regarding the overall contribution of these symbionts to aphid population regulation. As empirical data are scarce, mathematical modelling provides a tool for examining the effects of symbiont infection on aphid population dynamics under different environmental scenarios.

Aphid-natural enemy dynamics

The summer asexual morph is the most damaging part of the aphid life cycle: it exhibits rapid development to adulthood, reproduces parthenogenetically, and gives birth to live nymphs, which means that aphid numbers can build up quickly on summer vegetation (Dixon, 1985). Summer aphid population dynamics are influenced by host plant availability and quality, and by natural enemy abundance and activity (e.g. Karley et al., 2004). Modelling and experimental studies have shown that the combined effects of these natural enemies have the potential to regulate aphid populations (Kindlmann and Dixon, 2010; Karley et al., 2003, 2004), but aphid suppression is not always achieved. Variable aphid suppression by natural enemies might be explained by symbiont-encoded fitness traits that lead to coexistence of natural enemy-resistant and -susceptible aphids (Oliver and Higashi, 2019). Aphids are attacked by several hymenopterous endoparasitoid species (Boivin et al., 2012). Female parasitoid wasps insert an egg into the aphid abdomen; the egg hatches and the emerging larva consumes and eventually kills the aphid, which becomes a dried husk or 'mummy' case for the pupating wasp. In many aphid species, a proportion of individuals fail to succumb to parasitism after wasp oviposition, which has been attributed to aphid-encoded resistance factors (Martinez et al., 2014; Clarke et al., 2017) and to the presence of 'protective' facultative endosymbionts residing in aphid tissues, particularly *Hamiltonella defensa* (previously known as T type or PABS endosymbiont: Oliver et al. (2003); Moran et al. (2005); see Guo et al. (2017); Vorburger (2018) for recent reviews). As parasitoids are frequently used as biocontrol agents for aphids infesting agricultural and horticultural crops, understanding the effects of parasitism resistance on pest population dynamics is crucial for improving parasitoid efficacy.

The cost of parasitism-resistance: aphids

The coexistence of parasitism-resistant and -susceptible aphids within aphid populations suggests that natural enemy resistance entails a fitness cost. Resistance trade-offs clearly exist: in closed systems, pea aphid, *Acyrthosiphon pisum*, phenotypes that harbour *H. defensa* reach high frequencies in the presence of parasitoids, but uninfected phenotypes dominate in the absence of parasitism (Oliver et al., 2008); a similar reduction in abundance of *H. defensa*-infected aphids when parasitism pressure is absent has been demonstrated in the cowpea aphid, *Aphis craccivora* (Dykstra et al., 2014). However, such changes in the frequency of infected aphids occurred over an extended period (weeks-months), probably because the fitness costs of resistance tend to be small or moderate for aphid life history parameters such as aphid lifespan and fecundity (Gwynn et al., 2005; Vorburger and Gouskov, 2011; Vorburger et al., 2013; Martinez et al., 2018) or only occur on certain plant species (Leybourne et al., 2020; Karley et al., 2017; Clarke et al., 2017). Given that the fitness costs of parasitism resistance are relatively small and/or context-dependent, it is unclear why resistant aphids do not dominate natural populations.

Where there is a cost to host resistance, we might expect oscillatory cycles in parasitoid and aphid abundance that are typical of eco-epidemiological systems (see Boots et al. (2008) for a review), namely that parasitoid numbers decline due to the presence of resistant hosts, allowing susceptible hosts to increase in frequency, which subsequently promotes parasitoid abundance. In field populations, changes in the frequency of resistant (symbiont-infected) aphids can occur over the course of 2-3 weeks (e.g. in *A. pisum* populations on alfalfa: Smith et al. (2015)). This is more rapid than in closed systems, (described above), which suggests that the fitness costs to aphids of being resistant are not sufficiently large to cause oscillation dynamics. Indeed, the detailed model developed by Kwiatkowski and Vorburger (2012) showed that an implausibly high constitutive fitness cost to hosting the symbiont, or a high cost of induced resistance, was required for coexistence of aphids with and without protective symbiont (*H. defensa*) infection. Together, these lines of evidence suggest that other mechanisms need to be invoked to explain short term changes in symbiont infection frequency in aphid populations.

The cost of parasitism-resistance: parasitoids

It is possible that an alternative mechanism could operate to regulate resistant and susceptible aphid frequencies, whereby the fitness cost is experienced by the parasitoid due to an effect on host discovery or handling. Ecological theory suggests that if parasitoids learn to handle frequently encountered (i.e. competitive) hosts more rapidly, less competitive (and therefore less common) hosts might be able to persist (Chase et al., 2002; Chesson and Kuang, 2008, 2010). Ishii and Shimada (2012) provided an example of this mechanism: they conditioned the generalist pteromalid wasp *Anisopteromalus calandrae* on a single bruchid beetle host species, either *Callosbruchus chinensis* or *C. maculatus*, and found a clear response to the conditioning in choice tests when both species were present. Taking account of this type of learning explained why the presence of this common parasitoid extended the period of coexistence of *C. chinensis* and *C. maculatus*, despite the weaker competitiveness of the former species in the absence of parasitism. The more efficient discovery or handling of common hosts can be interpreted as a switching penalty in the form of less efficient handling of, or searching for, less common hosts (see below). The existence of a switching penalty has not been investigated explicitly for aphid parasitoids but might be anticipated based on knowledge of parasitoid biology and the behaviour of parasitoids and/or their aphid hosts during attack. Generalist aphid parasitoids often show innate preferences for specific host species (Rehman and Powell, 2010). Host preference can be modified, however, by conditioning to the host types from which they were reared and by learning from ovipositional experience when parasitoids sample alternative host types (Chow and Mackauer, 1992; Rehman and Powell, 2010). Several studies have demonstrated the potential for parasitoids to experience a penalty - in the form of increased time for host location and handling - when switching between aphid hosts differing in quality (e.g. Slater et al., 2019) and illustrate that parasitoids can distinguish between parasitism-resistant and -susceptible types due to symbiont-conferred effects on aphid defensive behaviour or pheromone production susceptibility to parasitism (e.g. Dion et al., 2011; Oliver et al., 2012). These lines of evidence suggest that changes in oviposition behaviour could incur a cost to the parasitoid; we were intrigued, therefore, to find out whether this potential switching cost could help to explain coexistence of parasitism-resistant and -susceptible hosts in aphid populations.

Cereal aphid biocontrol in a changing climate

To test the possibility of this mechanism operating in an aphid-parasitoid system, we present a model based on the generalist hymenopterous parasitoid *Aphidius colemani* attacking the cereal-feeding aphid *Rhopalosiphum padi*. This model incorporates switching penalties and offers plausible explanation for coexistence of aphids with (resistant) and without (susceptible) the protective symbiont *H. defensa*. *R. padi* is a pest of global relevance to cereal crop production, transmitting plant viruses that can cause yield losses of 30% or higher (Perry et al., 2000; Finlay and Luck, 2011). Although seasonal dynamics of *H. defensa* infection frequencies are not yet available within *R. padi* populations, the symbiont is known to provide protection against parasitism by *A. colemani* (Leybourne et al., 2020) and we have a comprehensive description of this aphid-parasitoid system from our previous experimental work (Leybourne et al., 2019, 2020) that can be used for model parameterisation. Initial densities of resistant (*H. defensa*-infected) and susceptible (uninfected) aphids are equal in model simulations. In field conditions, resistant aphids tend to be more common when parasitoid pressure is high (using *H. defensa*-infection in *A. pisum* populations as an example: Smith et al., 2015). Adopting this scenario as a starting point, we therefore impose a cost incurred by the parasitoid when switching from resistant to susceptible aphids. First, we develop a single stage model, tractable to fixed point analysis, that we use to assess critical values of the switching penalty and conditions required for co-existence. We extend it to build a more realistic and more complex stage structured model incorporating an explicit juvenile stage to determine the effects of including different aphid life stages on coexistence of aphid hosts. The stage-structured model allows us to explore the effects of environmental stress, which can vary with aphid development stage. We were particularly interested in testing the effects of drought on coexistence as our previous research has shown that drought stress affects *R. padi* population structure (Aslam et al., 2013) and aphid fitness (Leybourne et al., unpublished), and this is supported by other studies demonstrating the effects of drought on aphid fitness and suitability as prey for natural enemies (Hale et al., 2003; Tariq et al., 2012; Wade et al., 2017). Climate change-imposed stresses are predicted to have dramatic effects on host-parasitoid interactions (e.g. Jeffs and Lewis, 2013); using drought as an example, we illustrate the model's utility for testing whether the switching penalty effects on aphid coexistence are robust under different environmental stresses. We discuss the implications of our findings for aphid-parasitoid dynamics and aphid biocontrol and identify modelling outcomes that might be generally applicable to host-parasitoid or predator-prey dynamics under fluctuating environmental conditions such as those imposed by a changing climate.

Materials and Methods

Model Construction

Single Stage Model

We consider a model with one species of parasitoid P and two phenotypes of host aphids H_s and H_d where the only difference between the two is that H_d carries the symbiont *H. defensa* which confers resistance to parasitism, whereas H_s is susceptible. *H. defensa* is known to be maternally-inherited (Sandström et al., 2001) so we assume perfect vertical transmission and ignore any seasonal transmission dynamics. We further assume that other natural predators remain at a level sufficient to maintain a per capita rate of aphid consumption, δ_h and that parasitioids have a natural mortality rate of δ_p. Initially we will consider a model which excludes development time (age structure) and assume host reproduction is logistic with a maximum reproductive rate r and carrying capacity C.

Assuming that the parasitoids attempt to parasitise hosts of phenotype k at a rate $\Phi(H_k, H_l)$ (a function of the densities of both host types discussed below) and are successful on a proportion ϵ_k of attempts (where k and l are s or d), the system is modelled by the following system of ODEs:

$$\frac{dH_s}{dt} = rH_s\left(1 - \frac{\delta_h}{r} - \frac{H_s + H_d}{C}\right) - \epsilon_s\Phi\left(H_s, H_d\right)P \tag{1}$$

$$\frac{dH_d}{dt} = rH_d\left(1 - \frac{\delta_h}{r} - \frac{H_s + H_d}{C}\right) - \epsilon_d\Phi\left(H_d, H_s\right)P \tag{2}$$

$$\frac{dP}{dt} = \left[\epsilon_s\Phi\left(H_s, H_d\right) + \epsilon_d\Phi\left(H_d, H_s\right)\right]P - \delta_p P \tag{3}$$

We use a function for Φ proposed by Van Leeuwen et al. (2013) which is based on the Holling disc equation, but which allows the searching efficiency for a given aphid to differ depending on which phenotype of aphid was last parasitised. The Holling disc equation assumes that in a period of time T, the number of hosts of phenotype k parasitised by each parasitoid, N_k, depends on the discovery rate (or searching efficiency), α_k ($k = s, d$). It increases with the density of hosts H and the time available for searching (or hunting), T_h.

$$N_k = \alpha_k T_h H_k \tag{4}$$

The time available for searching is the total time available T, less that used for handling hosts. Thus if γ is the handling time for each host we have

$$T_s = T - \gamma\left(N_s + N_d\right). \tag{5}$$

Combining equations 4 and 5 and rearranging we get

$$N_k = \frac{\alpha_k H_k T}{\left(1 + \gamma \left(\alpha_s H_s + \alpha_d H_d\right)\right)} \tag{6}$$

and setting $T = 1$ yields the parasitism rate, $\Phi\left(H_k, H_l\right) = \Phi_k$, of hosts of phenotype k per unit time

$$\Phi_k = \frac{\alpha_k H_k}{\left(1 + \gamma \left(\alpha_s H_s + \alpha_d H_d\right)\right)} \tag{7}$$

where Van Leeuwen et al. (2013) modify this to incorporate a penalty for switching, s_{kl}, which is the reduction in searching efficiency due to switching from phenotype k to phenotype l.

$$\Phi\left(H_k, H_l\right) = \frac{\alpha_k H_k \left(s_{sk} \alpha_s H_s + s_{dk} \alpha_d H_d\right)}{\left(\alpha_s H_s + \alpha_d H_d\right)\left(1 + \gamma \left(s_{sk} \alpha_s H_s + s_{dk} \alpha_d H_d\right)\right)}$$

Without loss of generality, we assume that $s_{ss} = s_{dd} = 1$. Further, we assume that the two aphid phenotypes are identical except that one is more resistant to parasitoid attachment than the other. Thus $\alpha_s = \alpha_d$ and

$$\Phi\left(H_k, H_l\right) = \frac{\alpha H_k \left(s_{sk} H_s + s_{dk} H_d\right)}{\left(H_s + H_d\right)\left(1 + \alpha\gamma \left(s_{sk} H_s + s_{dk} H_d\right)\right)} \tag{8}$$

Assuming that reproduction is greater than background mortality ($r > \delta_h$) and non-dimensionalising with respect to r and δ_h we obtain the system:

$$\frac{d\hat{H}_s}{d\hat{t}} = \hat{H}_s\left(1 - \hat{H}_s - \hat{H}_d\right) - \epsilon_s\Phi\left(\hat{H}_s, \hat{H}_d\right)\hat{P} \tag{9}$$

$$\frac{d\hat{H}_d}{d\hat{t}} = \hat{H}_d\left(1 - \hat{H}_s - \hat{H}_d\right) - \epsilon_s\Phi\left(\hat{H}_d, \hat{H}_s\right)\hat{P} \tag{10}$$

$$\frac{d\hat{P}}{d\hat{t}} = \left(\epsilon_s\Phi\left(\hat{H}_s\right) + \epsilon_d\Phi\left(\hat{H}_d\right)\right)\hat{P} - \delta_p\hat{P} \tag{11}$$

where $\hat{t} = (r - \delta_h)\,t$, $\hat{H}_k = \frac{H_k}{C\left(1-\frac{\delta_h}{r}\right)}$, $\hat{P} = \frac{P}{C\left(1-\frac{\delta_h}{r}\right)}$ $\hat{\delta}_p = \frac{\delta_p}{r-\delta_h}$, $\hat{\alpha_k} = \frac{\alpha_k C}{r}$, $\hat{\gamma} = \gamma\,(r - \delta_h)$. This highlights that the dynamics of the system will be determined by the ratio of parasitoid mortality, attach rates and handling times to the intrinsic rate of increase of the aphid population. For ease of reading we will drop the hats from this point, and this non-dimensionalised system is assumed unless stated otherwise.

Stage-Structured Model

The stage structured model introduces an explicit juvenile stage where aphid nymphs of phenotype k are denoted by J_k and adults by H_k. Juvenile and adult aphids utilise the same resources so density dependent reproduction is a function of both nymphs and adults. However, parasitised nymphs, denoted I, move less and form mummies shortly after parasitism so they are excluded from density dependence and are not separated by phenotype. It is further assumed that adult parasitoids only parasitise juvenile aphid nymphs (Ives et al., 1999). Hence, the full stage-structured model is given by:

$$\frac{dJ_k}{dt} = rH_k\left(1 - \frac{\delta_j}{r} - \frac{H+J}{C}\right) - \epsilon_k\Phi\left(J_k, J_l\right)P \tag{12}$$

$$\frac{dH_k}{dt} = \int_{s=t-\tau_h}^{t} \dot{J}_k\left(t-\tau_h\right)ds \;-\; \delta_h H_k \tag{13}$$

$$\frac{dI}{dt} = \epsilon_s\Phi\left(J_s\right)P + \epsilon_d\Phi\left(J_d\right)P - \delta_i I \tag{14}$$

$$\frac{dP}{dt} = \int_{s=t-\tau_p}^{t} \dot{I}\left(t-\tau_p\right)ds \; - \delta_p P \tag{15}$$

$$k, l = s, d \tag{16}$$

where $H = H_s + H_d$ and J=$J_s + J_d$. The non-dimensionalised model is presented in supporting information.

Effects of Drought Stress

We consider the possible effects on aphids carrying *H. defensa* (and hence those more resistant to parasitism) of developing on drought stressed plants in the stage-structured model. The first is to reduce the fecundity in the *H. defensa* carrying phenotype (a reduction in r_d). The non-dimensionalise equation for J_d is shown in the supplementary information.

The second possibility is a decrease in aphid development times which would involve decreasing τ_h either for one or for both phenotypes (See Results).

Model Parameterisation

The advantage of the model as formulated is that there are relatively few parameters and many of the raw parameters are obtainable from available empirical investigations. The key parameters for the raw (as opposed to non-dimensionalised) model are summarised in table 1 and their derivation is discussed below. We first consider the stage-structured model which most closely describes the lifecycles of aphids and parasitoids and then the adjustments necessary to parameterise a comparable single stage model

Laboratory experiments described in Leybourne et al. (2020) have indicated that the bird cherry-oat aphid, *Rhopalosiphum padi* has juvenile aphid development time τ_h=9 days.

There is no field data for *R. padi* and very limited data for other species of aphid. However, Leybourne et al. (2020) found almost no nymph mortality after 7 days in glasshouse conditions. In the field there is only data for caged aphids where Watt (1979) and Howard and Dixon (1992) found values of $87 - 95\%$ survival of cereal aphids on immature plants and $25 - 40\%$ on mature plants. Based on these data we assume that, in the field approximately 85% of juveniles *R. padi* survive to reproductive maturity after 9 days which implies a background natural mortality rate $\delta_j = 0.018$ per nymph per day.

There is no direct field data for adult survival. However, Leybourne et al. (2020) found average adult survivorship after 21 days of $10 - 20\%$ in glasshouse conditions and it is reasonable to assume that the same background field mortality that impacts juveniles will also apply to adults. The adult death rate of aphids (δ_h) is calculated from 2 components; a base rate of 0.087 per day so that 14.7% of individuals are alive after 21 days alongside a background field mortality rate of 0.018, calculated from juvenile survival. Therefore $\delta_h = 0.087 + 0.018 = 0.105$ so that after 21 days 9.7% of individuals are alive.

Leybourne et al. (2020) found that *R.padi* reproduces at a rate of approximately

5 nymphs per day for the first 9 days of reproductive maturity and approximately 1 per day thereafter which over an 18 day adult lifespan would equate to an average rate of 3 nymphs per day which is consistent with other studies such as Dean (1974). Therefore assume a base reproductive rate, of r=3 nymphs per adult per day. When investigating the possible effects of drought we assume aphid reproduction to be decreased under drought stress as previously reported (Hale et al., 2003; Johnson et al., 2011) with differential impacts on the two phenotypes of aphids.

The average development time of the parasitoid *A. colemani*, from egg to eclosion from an aphid host, is approximately 12 – 13 days under laboratory conditions (Kalule and Wright, 2005; Leybourne et al., 2020), but this was measured on a daily basis so those present at 12 days eclosed (i.e. emerged from the mummy case) at some point between 11 and 12 days old, similarly those present at 13 days eclosed between 12 and 13 days. Therefore we assume $\tau_p = 12$ days. Steenis (1993) found that juvenile mortality of *A. colemani* attacking *Aphis gossypii* was similar to that of the juvenile aphids so while the parasitised aphid nymph is alive its mortality rate will remain at $\delta_j = 0.087$ per day. *R. padi* forms mummies after approximately 6 days and approximately 90% of mummies will eclose to adult parasitoids 6 days later (*Leybourne pers. comm.*). We are not modelling the mummified stage separately so to translate the mortality rate into an average mortality rate, δ_i over 12 days we have the probability of a parasitoid egg eclosing to an adult

$$(1 - \delta_i)^{12} = 0.9\,(1 - \delta_j)^6$$

Therefore

$$\delta_i = 1 - 0.9^{1/12}\left(1 - \delta_j\right)^{1/2} = 0.0177.$$

Adult parasitoid longevity is approximately 20 days (Sampaio et al., 2008), similar to the aphids so we set $\delta_p = \delta_h = 0.105$ meaning that 90% of parasitoids have died after 20 days.

Aslam et al. (2013) found that in glasshouse conditions when individuals of another generalist parasitoid *Aphidius ervi* were restricted to searching for *R.padi* on a single plant the number of attacks was very variable. However, on average 17 aphid nymphs were parasitised in a 30 minute period which would equate to 34 in an hour, but all attacks occurred within the first 10 minutes suggesting that a parasitoid could theoretically parasitise over 100 nymphs an hour, but it is likely to be fewer. The handling time, γ, is assumed to be 0.001 days (equivalent to 86.4 seconds) allowing a parasitism rate of 41.7 nymphs per hour if there has been no switching.

A. colemani can successfully parasitise approximately 20% of aphids infected with *H. defensa* and $30 - 70\%$ of aphids uninfected with the symbiont (Leybourne et al., 2020) thus we assume that parasitism efficiency ϵ_d on the symbiont carrying host H_d is 0.2 and consider the two extreme values of ϵ_s, 0.3 and 0.7, on the uninfected (and so more susceptible) host H_s.

The efficiency of parasitoids in searching for aphids (sometimes known as the attack rate), α, is the discovery rate per unit time per unit density of the aphid population. It varies between parasitoid-host combinations, and is affected by aphid host plant, the presence of competitor parasitoids and environmental conditions (Ives et al., 1999; Chua et al., 1990). There is limited information available on the searching efficiency of parasitoids under field conditions and we use the model to explore the influence of α on model dynamics considering a range of 0.5 to 10 per day per unit density. The implication of $\alpha > 1$ is that a parasitoid will have the capacity to revisit aphids more than once in a day if it has previously failed to parasitise them or rejected them. It does not mean that an aphid can be host to more than one juvenile parasitoid.

Finally we consider the switching parameters. The higher the value of s_{kl}, the smaller the penalty for switching. Values are constrained to lie between 0, indicating an absolute refusal to switch, and 1 where there is no penalty for switching. Recall that without loss of generality we have set $s_{ss} = s_{dd} = 1$ because when no switching has occurred there can be no penalty. Switching penalties for moving from one host type to the other would depend on the life history of a parasitoid and, if based on learning, are likely to be higher for switching from the more common host phenotype to the rarer phenotype. To model learning explicitly would require a s_{sd} and s_{ds} to change depending on the life-history. However, if a phenotype with a competitive advantage (in this case H_d) is initially less common than the "weaker" phenotype (H_s) and therefore $s_{sd} < s_{ds}$ then H_d will rapidly become more dominant. If s_{ds} and s_{sd} were dynamic the relationship would then reverse and we would have $s_{ds} < s_{sd}$. It is reasonable therefore to assume that $s_{ds} < s_{sd}$ and, for simplicity we make the assumptions that s_{ds} is fixed and $s_{sd} = 1$ so that there is no penalty for switching from the (assumed rarer) susceptible aphid phenotype H_s to the *H. defensa* carrying phenotype H_d. These assumptions are conservative with respect to the stability of the system and we can therefore use the simpler model to make inference about systems which explicitly model learning. We then calculate the critical values of s_{ds} for coexistence of H_s and H_d in the single stage model and use a value close to this to explore the dynamics of the stage structured model.

For the single stage model we adjust the reproductive rates of aphids to account for juvenile mortality and obtain an effective reproductive rate of $r_{singlestage} = (1-\delta_j)^{\tau_h} * r = 0.85 * 3 = 2.55$ new adult aphids per aphid per day. Note that there is not a direct translation from α in the single stage model to the stage structured model because in the latter model the adult host population is unavailable to adult parasitoids. Also in the single stage model, all successfully parasitised aphids are assumed to emerge as adult parasitoids.

Numerical simulations

Numerical simulations were run in R 3.4.3 (R Core Team, 2017) using the stagePop package (Kettle, 2015) described in Kettle and Nutter (2015). The single stage model was run for 200 days which more than covers the length of a summer season and the stage-structured model was run for 1000 days to allow any effect of initial conditions to be eliminated from the model and for trends to become apparent. Simulations were run to investigate the effect of altering the searching efficiency α and then to investigate the effects of drought described above (see Results section for details).

Results

Single Stage Model

Persistence of susceptible phenotype H_s

In the absence of the parasitoid, the two host aphid phenotypes have identical properties so the system will have an unstable equilibrium point where both aphid phenotypes are extinct and a neutrally stable equilibrium at (H_s^*,H_d^*) where $H_s^* + H_d^* = K$ and the proportion H_s^*/H_d^* is equal to the starting proportions H_s^0/H_d^0. However, the presence of a parasitoid changes the dynamics of the system removing the neutral stability and introducing 3 further equilibria - (0,H_d^*,P^*), (H_s^*,0,P^*) and a unique interior equilibrium (H_s^*, H_d^*, P^*) of coexistence. For the remainder of this paper we consider the existence and stability of the interior equilbrium. At this point we have

$$\hat{H_s}^* = \frac{\left(\epsilon_d - \hat{\gamma}\hat{\delta}_p - s_{sd}\left(\epsilon_s - \hat{\gamma}\hat{\delta}_p\right)\right)\hat{\delta}_p}{\hat{\alpha}\left(\epsilon_s - \hat{\gamma}\hat{\delta}_p\right)\left(\epsilon_d - \hat{\gamma}\hat{\delta}_p\right)(1 - s_{ds}s_{sd})} \tag{17}$$

$$\hat{H_d}^* = \frac{\left(\epsilon_s - \hat{\gamma}\hat{\delta}_p - s_{ds}\left(\epsilon_d - \hat{\gamma}\hat{\delta}_p\right)\right)\hat{\delta}_p}{\hat{\alpha}\left(\epsilon_s - \hat{\gamma}\hat{\delta}_p\right)\left(\epsilon_d - \hat{\gamma}\hat{\delta}_p\right)(1 - s_{ds}s_{sd})} \tag{18}$$

$$\hat{P}^* = \frac{\left(\hat{H_s}^* + \hat{H_d}^*\right)\left(1 - \hat{H_s}^* - \hat{H_d}^*\right)\hat{H_s}^*\left(1 + \hat{\alpha}\hat{\gamma}\left(\hat{H_s}^* + s_{ds}\hat{H_d}^*\right)\right)}{\hat{\alpha}\epsilon_s\left(\hat{H_s}^* + s_{ds}\hat{H_d}^*\right)} \tag{19}$$

For the parasitoid to persist, it is sufficient to satisfy the conditions for persistence when the susceptible phenotype (H_s) is extinct:

$$\frac{\epsilon_d \hat{\alpha}}{1+\hat{\alpha}\hat{\gamma}} > \hat{\delta} \tag{20}$$

Given that $\epsilon_d < \epsilon_s$, the interior equilibrium will exist with H_s^*, H_d^* and $P^* > 0$ if and only if the reduction in parasitoid searching efficiency s_{ds} due to parasitoid switching from infected to uninfected phenotypes is bounded above by

$$s_{ds} < \frac{\epsilon_d - \hat{\gamma}\hat{\hat{\delta}}_p}{\epsilon_s - \hat{\gamma}\hat{\hat{\delta}}_p} = \frac{\epsilon_d - \gamma\delta_p}{\epsilon_s - \gamma\delta_p} \tag{21}$$

If inequality (21) holds (i.e. the switching penalty is sufficiently severe) then both hosts will persist otherwise the presence of the parasitoid will cause H_s to go extinct. The handling time γ is small for parasitoid systems so the right hand side of equation (21) is dominated by the ratio of the efficiency of parasitoids on the infected phenotypes to the uninfected phenotypes. The greater the reduction in efficiency on infected aphids, the more severe must be the switching penalty (and hence the smaller s_{ds}) for the equilibrium to hold. This inequality therefore establishes our first key result, namely that in this two host system given a sufficiently high penalty for switching from H_d to H_s, the presence of the parasitoid can explain the coexistence of these phenotypes even in the absence of any costs associated with the *H. defensa* carrying, resistant, phenotype H_d. In the case where there is a single aphid phenotype present, the equilibrium is globally attracting and stable if

$$\epsilon\alpha\left(1-\frac{1}{\alpha\gamma}\right)-\delta\left(1+\frac{1}{\alpha\gamma}\right)<0 \tag{22}$$

Heuristically, the left hand side is a measure of the rate at which the parasitoid population can increase. In the case where $\alpha\gamma < 1$ the inequality will always hold and for systems such as parasitoids where the handling time is low, if the interior equilibrium exists it will remain stable unless the searching efficiency, α, is unrealistically high. The introduction of a switching penalty acts to decrease α when a parasitoid switches from one phenotype to another. Thus if inequality (22) holds for the susceptible aphid the interior equilibrium will be stable. For the parameter values of Table 1, the critical value of s_{ds} ranges from 0.667 (with $\epsilon_s = 0.3$) to 0.286 (with $\epsilon_s = 0.7$). Thus, as shown in Figure 1, taking $s_{ds} = 0.65$ yields stable coexistence of both host phenotypes at an approximate ratio of 20:80 symbiont-free:infected hosts consistent with up to $80-90\%$ aphids being symbiont-infected when parasitoid-induced mortality is high (Smith et al., 2015). However, parasitoids are at a higher density than would be expected being present at over twice the maximum host capacity (Figure 1) although this issue is addressed by the introduction of the stage-structured model (see below).

Sensitivity of results with respect to parasitoid attack rate α

Given the lack of knowledge about α, its impact on the system dynamics was assessed for α from 0.7 to 10 in increments of 0.01. With $\alpha \leq 0.80$ the parasitoid population does not persist. For $0.76 \leq \alpha \leq 1.59$ there is a stable equilibrium similar to that in Figure 1 with the density of parasitoids increasing with α, the density of hosts decreasing and the proportion of the host population present as the susceptible phenotype H_s decreasing. For $\alpha \geq 1.60$ there was still a stable fixed point, but the density of the parasitoid population decreased because of the low density of the host population. These results hold for both the case where $\epsilon_s = 0.3$, $s_{ds} = 0.65$ and where $\epsilon_s = 0.7$, $s_{ds} = 0.26$.

Stage Structured Model

When the juvenile stage is introduced parasitoid densities are at more sensible levels than in the single stage model and more complex dynamics are seen. Complete closed-form stability analysis of the stage-structured system is complex. However, within reasonable development times no further equilibria are introduced, and the range of dynamics for plausible parameter values is extended. For the parameterisation given in Table 1 we find that for $\alpha \leq 1.144$ the parasitoids go extinct. For $1.145 \leq \alpha \leq 1.67$ the system exhibits damped oscillations and converges to a stable steady state of coexistence (shown for $\alpha = 1.4$ in Figure 2). For $\alpha \geq 1.68$ coexistence is maintained, but the system exhibits limit cycles (shown for $\alpha = 1.75$ in Figure 3) which increase in amplitude with α. The aphid cycles are in phase, with the parasitoid lagging behind.

Drought Stress

We consider drought stress in the stage-structured system with a stable equilibrium (for the parameterisation of Table 1 when $\alpha = 1.4$). The two drought stress mechanisms described earlier are investigated namely reductions in the fecundity of resistant aphids (i.e. carrying *H. defensa*) and reduced development times for either one or both aphid phenotypes.

Firstly, as the resistant host reproduction rate r_d is reduced relative to that of the susceptible aphids, r_s, the latter aphid phenotype increases both in absolute density and as a proportion of the population. This means that the parasitoid spends more time on hosts which it is effective at parasitising and the stable equilibrium is tipped over into a limit cycle with a lower mean density of parasitoids (see Figure 4). If r_d drops sufficiently (relative to r_s) then the resistant (symbiont-infected) aphid phenotype (H_d) becomes extinct and the limit cycles remain. For higher values of α, the extinction of the resistant (*H. defensa*-infected) host phenotype occurs for higher values of r_d.

Secondly, altering the development time, τ_h, either for both phenotypes or for the resistant (symbiont-infected) phenotype had no effect on the dynamics of the system except to increase or decrease the period of the limit cycle as the development time increased or decreased.

Discussion

We have constructed a simple host-parasitoid model in which most parameters are empirically determined, and which includes parasitoid learning in the form of a 'switching penalty'. The inclusion of a switching penalty makes a parasitoid less efficient at finding a less common host phenotype and allows coexistence of two host phenotypes that differ only in their susceptibility to parasitism. Simulations showed that a switching penalty could provide a mechanism to explain coexistence of susceptible and resistant hosts in aphid populations at proportions observed in field populations (Smith et al., 2015) given a sufficiently high cost associated with parasitoid switching from resistant to susceptible aphids, even in the absence of any costs to the aphid of being resistant. Stable coexistence was maintained when stage-structuring was included. When aphid fecundity and development time were altered in response to drought stress, this led to reduced parasitoid abundance and, in some cases, tipped the system from stable coexistence into stable limit cycles, indicating that the switching penalty could destabilise biocontrol under conditions of abiotic stress.

The study provides novel insights into a previously overlooked mechanism capable of maintaining diversity within host populations, and represents an important advance in understanding the influence of symbiont-encoded fitness traits on population processes that regulate host-parasitoid dynamics. In aphids, including the cereal-feeding species *Rhopalosiphum padi* modelled in this study, effort has focussed on quantifying fitness costs to the aphid of parasitism resistance and not those experienced by the parasitoid. Fitness trade-offs for aphids are often small or context-dependent (Clarke et al., 2017, 2018; Leybourne et al., 2020) and not large enough to explain short-term (days-weeks) changes in aphid phenotype frequency (Smith et al., 2015; Kwiatkowski and Vorburger, 2012); mechanisms other than aphid fitness costs, therefore, need to be invoked, such as those experienced by parasitoids. Fitness costs imposed by parasitoid oviposition behaviour might also explain why susceptible aphid types persist at moderate to high frequencies across aphid populations (Henry et al., 2015; Zytynska and Weisser, 2016) and resistant aphids do not reach fixation despite appearing to be at a competitive advantage. Our study emphasises the importance of understanding different types of fitness costs to both organisms in the aphid-parasitoid interaction.

Potential causes of a switching penalty in aphid parasitoids

The behavioural and physiological causes of a switching penalty – a key assumption of our model - have not been investigated explicitly in parasitoids attacking aphids and remain speculative. Host selection by parasitoids involves behavioural decisions in response to chemical and physical cues that facilitate habitat and host location, host recognition, host acceptance and oviposition (Rehman and Powell, 2010). Innate host preferences can be altered by parasitoid conditioning and associative learning. Parasitoid exposure to certain host types can influence subsequent host choice or willingness to oviposit (Chow and Mackauer, 1992; van Emden et al., 2008; Rehman and Powell, 2010), demonstrating the potential for parasitoid oviposition choices to be influenced by learning. Our starting point was to assume that parasitoids are more efficient (e.g. due to faster host location and handling time) when attacking a familiar aphid (resistant) phenotype that is more common in the host population. We recognise that in situations where the susceptible aphid is initially more common, the penalty might operate in the other direction, although Ishii and Shimada (2012) demonstrated parasitoid conditioning on a time scale of several hours, which would be sufficiently rapid to prevent the extinction of susceptible aphids.

A small number of studies provide indirect evidence that parasitoids might experience a fitness cost when attacking resistant (symbiont-infected) or susceptible (uninfected) aphids due to symbiont-associated differences in parasitoid or aphid behaviour. For example, pea aphids infected with *H. defensa* show reduced aggressiveness and attenuated escape responses towards parasitoids compared with uninfected aphids (Dion et al., 2011) indicating that symbiont-infected aphids might present an easier target for attack with faster parasitoid handling time. Conversely, other studies suggest that resistant parasitoids will be more costly to attack. For example, *A. ervi* modified its oviposition behaviour in pea aphids in response to aphid *H. defensa* infection by selectively super-parasitising symbiont-infected aphids (Oliver et al., 2012), which would be predicted to increase the handling time compared with uninfected aphids. This behaviour was thought to be linked to higher levels of alarm pheromone emission by symbiont-infected aphids suggesting that parasitoids can modify host selection rapidly in response to volatile cues indicating host quality. Plant volatile emissions in response to aphid infestation could also contribute to parasitoid learning by altering the attractiveness of the plant, which is known to vary in response to infestation by symbiont-infected aphids (Frago et al., 2017). These studies provide initial evidence that a switching cost could cause aphid parasitoids to make rapid changes in oviposition behaviour through learning in response to the abundance of resistant/susceptible aphids. However, focussed experimentation is needed to confirm a switching penalty for aphid parasitoids and which aphid phenotype is likely to impose the greatest cost.

Although the exact value of a host switching penalty would be hard to quantify empirically, it should be possible to establish plausible ranges using targeted experiments, and the critical value for coexistence is straightforward to calculate. It depends primarily on the relative efficiencies of parasitising the different host phenotypes (ϵ), and additionally on the parasitoid death rate (δ_p) and the handling time of each aphid parasitized (γ). The effects of the latter two parameters on the critical value of the penalty are small relative to the larger effect of the ratio of the parasitism efficiencies for susceptible and resistant hosts (see equation 21). Once the switching penalty is determined, it reduces the attack rate, α, on an aphid of a different phenotype to the one last attacked (see equation 8). In a simple non-spatial model, the switching penalties are fairly high, although although they are reduced when parasitoid efficiency on the susceptible host is at the lower end of the plausible range ($s_{ds} = 0.667$ which equates to a reduction in attack rate of about one-third). Parasitoid learning may not be enough to fully stabilise coexistence in a system alone, and indeed in the laboratory system investigated by Ishii and Shimada (2012) the period of coexistence of two beetle species was prolonged, but not stabilised, by the presence of a parasitoid. However, spatial heterogeneity is known to have a stabilising effect at the population level (Hastings, 1977; Murdoch, 1977; Holt, 1984; Briggs and Hoopes, 2004) and, in a heterogeneous field situation, parasitoid learning may well have significant explanatory power for the coexistence of multiple phenotypes of aphids operating in the same ecological niche.

The introduction of an explicit juvenile stage allowed the preference of parasitoids for juvenile aphids to be included in the model and led to emergence of more complex population dynamics with limit cycles likely to occur unless parasitoid attack rates were low. As attack rates increased, the system maintained limit cycles of approximately the same period, but with higher mean parasitoid population density. However, above an optimum attack rate the host population was suppressed, leading to a decline in the mean parasitoid population density. Further, the non-dimensionalized equations show that system stability is related to the ratio between reproductive rate of hosts and attack rate of parasitoids. As the aphid reproductive rate decreases, the rate of parasitoid attack at which the system starts to cycle and the optimum rate of attack also decrease. Host switching might also influence the sex of the emerging offspring. Parasitoid wasps used for biological control of insect herbivores often exhibit the typical hymenopteran haplo-diploid mode of sex determination, where fertilized eggs become diploid females, but unfertilized eggs become haploid males (Heimpel and de Boer, 2008). In our study, we assumed that all emerging parasitoids were mated females, which led to high parasitoid abundance. While this assumption is reasonable for asexual (thelytokous) parasitoid populations, the model could be modified to incorporate male and female offspring production (e.g. by preventing a proportion of the offspring from being able to oviposit to account for males). This would allow study of the effects of changing sex ratio and mother wasp decisions on sex-specific allocation of offspring to different quality aphid hosts (reviewed in Rehman and Powell (2010)).

Model application in aphid-parasitoid systems

If switching penalties are a significant determinant of the outcome of pest-natural enemy interactions, the model could be used to devise optimum parasitoid abundances for augmenting local populations to control pests and to predict when changes in parasitoid behaviour due to conditioning and/or learning might alter the stability of the system. Integrated pest management systems incorporating biocontrol are receiving greater attention as more pesticides are withdrawn due to concerns about toxicity beyond the intended target (e.g. Birch et al., 2011). However, as climate change increases the risk of drought stress in crop production areas, consideration should be given to how future climate conditions might change the outcome of biocontrol measures. Under drought, the rates of aphid development and aphid growth could increase (Aslam et al., 2013; Wade et al., 2017) and the fitness costs of carrying the protective symbiont *H. defensa* may become more apparent, for example by a reduction in the fecundity of aphids carrying the symbiont (e.g. Leybourne et al. (2020)). The former scenario was tested and found to have little impact on system dynamics. However, the latter effects could well tip the system from stability into cycling on a timescale similar to the development time of hosts and parasitoids. Alternatively, symbiont protection might be attenuated by environmental stress, as seen in the breakdown of *H. defensa*-encoded pea aphid resistance to parasitism under heat stress (e.g. Bensadia et al., 2006; Guay et al., 2009). If this effect was combined with a reduction in fecundity it may result in the extinction of the phenotype. The potential impact of changing host fecundity on population dynamics, and the evidence for a greater impact of poor quality plants on endosymbiont-infected aphids (Karley et al., 2017; Leybourne et al., 2020), highlights the importance of elucidating the modulating effects of drought and other environmental stresses on host-parasitoid dynamics if biocontrol is to be used for pest management under a changing climate (e.g. Thomson et al., 2010). Further work is currently underway to investigate this effect in the cereal aphid-parasitoid model system used here.

Model insights for predator-prey systems under fluctuating environmental conditions

The model, with re-parameterisation, is applicable to a wide variety of consumer-resource interactions (including host-parasitoid and predator-prey systems). Our findings showed that the model dynamics are robust to variation in absolute and relative development times of the host and parasitoid, provided the background mortality rates are not so high that the population goes extinct, and they are also robust to handling time (which is small relative to the attack rate). The switching parameter is applicable to other consumer-resource systems where resource phenotype or quality can vary. It could, therefore, be used to investigate the impact of switching on the stability of other systems provided the consumer is subject to conditioning (through learning or acclimatisation) and that conditioning occurs rapidly relative to the life cycle of the resource, otherwise the initial conditions will have a large effect on model outcomes and the more common consumer could become extinct. Sensitivity analyses indicated that low values of the attack rate, α, could be critical for population dynamics, suggesting that accurate estimates of this parameter would be needed when modelling other systems. Further, the findings of this study indicate that the model is suitable for investigating the effect of any stress or change in the external conditions on consumer-resource dynamics. The analysis presented here focussed on the effects of a specific stress (drought) on reproductive rate of the resistant aphid phenotype but would be applicable to any abiotic or biotic stress which affects the ratio of consumer attack rate to resource reproductive rate. This might include stress conditions that affect resource vulnerability to attack by the consumer, for example through a change in morphology (e.g. stress-induced changes in aphid body colour that alter their location by predators: Losey et al., 1997; Wang et al., 2019) or effects on emission of volatile signals such as herbivore-induced plant volatiles used for host location (see Stenberg et al., 2015).

Conclusions

Our study shows the powerful stabilising effect of a switching penalty on host-parasitoid population dynamics, demonstrating the relevance of ecological theory to practical situations such as pest biocontrol and highlighting that this type of fitness effect should be considered more widely when constructing complex predator-prey models. In most aphid species, little is known about within-season dynamics of defensive symbiont infection, aphid resistance/susceptibility and the consequences for natural enemies. The simple aphid-parasitoid model incorporating a switching penalty introduced here could be used to identify and mitigate against scenarios that lead to dominance by resistant aphid phenotypes and has significant potential for application in other pest-natural enemy systems.

Author Contributions and Data Availability Statement

K.F. Preedy contributed to the development of and executed the mathematical aspects of the modelling and analysis, D.J. Leybourne contributed to the biological development and parameterisation of the models; M.A.J. Chaplain and G. Marion contributed to mathematical aspects of model development and analysis; A.J. Karley contributed to the biological development and parameterisation of the models. All authors contributed to writing the manuscript. Models have been parameterised from published studies cited in the paper. The Modelling has been performed in a standard package, stagePop, cited in the paper, a copy of the R code can be supplied on request.

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Table captions

Table 1. Summary of parameters and their values. The derivation is primarily based on data for *Rhopalosiphum padi* from Aslam et al. (2013) and Leybourne et al. (2020) and is discussed in Materials and Methods. Recall that without loss of generality we assigned $s_{ss} = s_{dd} = 1$

Table 1:

Parameter	Description	Units	Single Stage	Stage Structure	Drought Effect
α	attack rate	(day^{-1})	$0.5 - 10$	$0.5 - 10$	
r	host reproduction	(day^{-1})	2.55	3	H_d reduced
γ	handling time	(day)	0.001	0.001	
δ_j	J_k background mortality	(day^{-1})		0.018	
δ_h	H_k background mortality	(day^{-1})	0.15	0.15	
δ_i	I background mortality	(day^{-1})		0.0177	
δ_p	P background mortality	(day^{-1})	0.15	0.15	
τ_h	J development time	(day)		9	H_s, H_d reduced
τ_p	I development time	(day)		12	
ϵ_s	parasitism success on H_s	-	0.3, 0.7	0.3	
ϵ_d	parasitism success on H_d	-	0.2	0.2	
s_{ds}	switching penalty J_d to J_s	-	0.65, 0.26	0.65	

Figure captions

Figure 1. Co-existence in the Single stage model. A. Shows the density of the two adult aphid (Host) phenotypes, the red dotted line represents the susceptible phenotype without *H. defensa* and the dash-dot black line represents the phenotype which carries *H. defensa* and is resistant to parasitism. B. shows the total host density (dashed red line) and parasitoid density (solid black line). Parameters are as in Table 1 with $\epsilon_s = 0.3$, $\epsilon_d = 0.2$, $\alpha = 1$ and $s_{ds} = 0.65$ which is above the persistence threshold in equation 21.

Figure 2. Co-existence in the stage structured model: stable equilibrium. A.and B. show the density of the juvenile and adult aphid (Host) pheno-

types respectively. The black dash-dot line represents the phenotype which carries *H. defensa* and is resistant to parasitism and the red dotted line the susceptible phenotype. C. shows the total densities of the aphids (Hosts) with the solid black line representing adults and the dashed red line juveniles. D. shows densities of juvenile (dashed red line) and adult (solid black line) parasitoids. The choice of attack rate $\alpha = 1.4$ (with other Parameters as in Table 1) ensures the system reaches a stable equilibrium.

Figure 3. Co-existence in the stage structured model: limit cycles. A. and B. show the density of the juvenile and adult aphid (Host) phenotypes respectively. The black dash-dot line represents the phenotype which carries *H. defensa* and is resistant to parasitism and the red dotted line the susceptible phenotype. C. shows the total densities of the aphids (Hosts) with the solid black line representing adults and the dashed red line juveniles. D. shows densities of juvenile (dashed red line) and adult (solid black line) parasitoids. The choice of attack rate $\alpha = 1.75$ (with other Parameters as in Table 1) ensures the system enters a limit cycle.

Figure 4. Drought induced population cycles in the stage-structured model. A. and B. show the density of the juvenile and adult aphids (Host) phenotypes respectively. The black dash-dot line represents the phenotype which carries *H. defensa* and is resistant to parasitism and the red dotted line the susceptible phenotype. C. shows the total densities of the aphids (Hosts) with the solid black line representing adults and the dashed red line juveniles. D. shows densities of juvenile (dashed red line) and adult (solid black line) parasitoids. Pa-

rameters are as in Figure 2, namely the attack rate $\alpha = 1.4$ and other parameters are as in Table 1 except that whilst the reproductive rate of susceptible aphids, $r_s = r = 3$, the reproductive rate of the symbiont carrying phenotype is reduced to $r_d = 1.4$ ensuring the system reaches a limit cycle rather than a stable equilibrium.

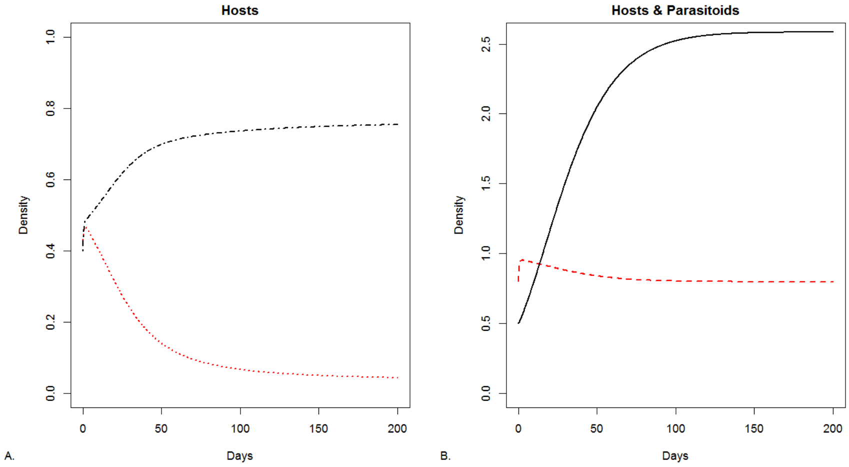
Hosts
Hosts & Parasitoids
Density
Density
Days
Days
0.0
0.2
0.4
0.6
0.8
1.0
0.0
0.5
1.0
1.5
2.0
2.5
0
50
100
150
200
0
50
100
150
200
A.
B.

Figure 1:

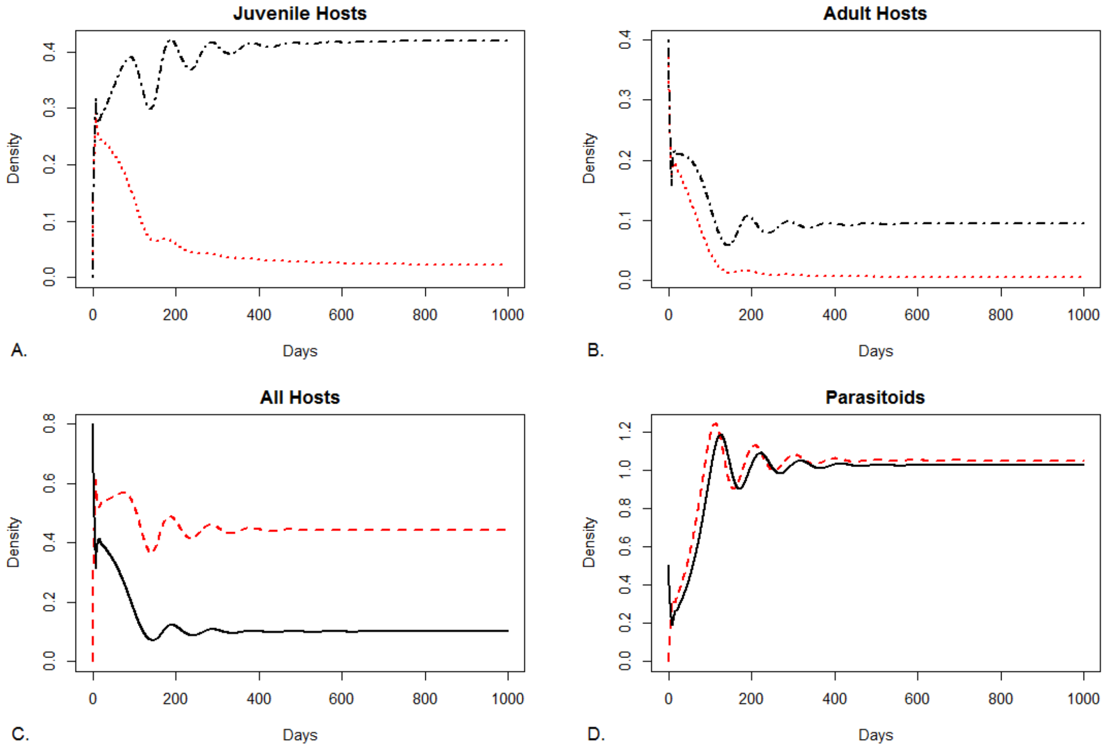
Juvenile Hosts
Adult Hosts
All Hosts
Parasitoids
Density
Days
0.0
0.1
0.2
0.3
0.4
0.6
0.8
1.0
1.2
0
200
400
600
800
1000
A.
B.
C.
D.

Figure 2:

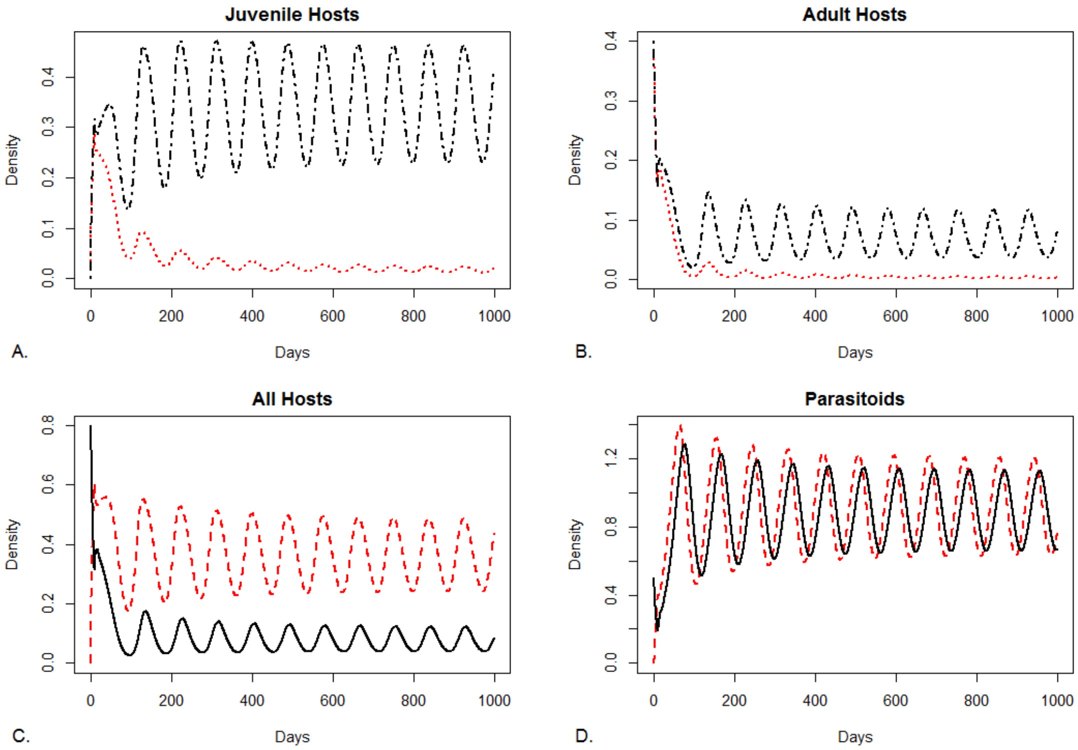
Juvenile Hosts
Density
0.0
0.1
0.2
0.3
0.4
0
200
400
600
800
1000
Days
A.
Adult Hosts
Density
0.0
0.1
0.2
0.3
0.4
0
200
400
600
800
1000
Days
B.
All Hosts
Density
0.0
0.2
0.4
0.6
0.8
0
200
400
600
800
1000
Days
C.
Parasitoids
Density
0.0
0.4
0.8
1.2
0
200
400
600
800
1000
Days
D.

Figure 3:

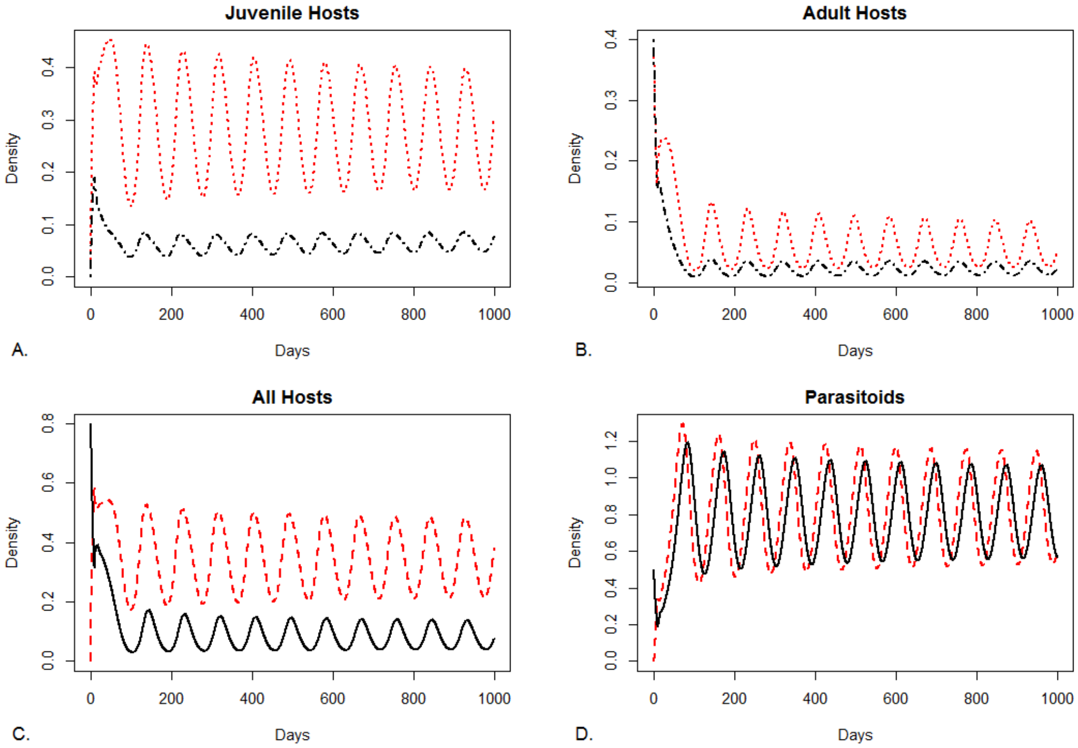
Juvenile Hosts
Adult Hosts
All Hosts
Parasitoids
Density
Days
0.0
0.1
0.2
0.3
0.4
0.6
0.8
1.0
1.2
0
200
400
600
800
1000
A.
B.
C.
D.

Figure 4: