The evolution of host exploitation by parasitoid wasps: the timing of attack and consumption

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Abstract

In some parasitoid wasp species, larvae consume their host immediately after hatching ("idiobiont"), while in other species, larvae delay the consumption of the host until the maturation of the host ("koinobiont"). The delayed emergence is a life-history trait associated with numerous life-history traits including lifespan and body size. Consequently, the evolution of delayed emergence has been studied as an explanation for the diversity of life-history strategies in parasitoid wasps. Previous studies have provided support for the association between delayed emergence trait and other life-history traits such as fecundity and parasitoid-induced additional mortality. However, only poorly known is the adaptive significance of delayed emergence. As a result, we have little understanding of what the key factor in life-history diversity in parasitoid wasps is. In this study, we develop mathematical models to examine associations between delayed emergence trait and other life-history traits. Specifically, we studied the evolutionary dynamics of delayed emergence and resulting life-history characteristics of host exploitation. Our results predict that the reproductive values vary with which of the developmental stages of hosts they parasitize. Reproductive values thus determine the optimal target of attacking for parasitoids, with several empirical studies supporting the prediction. Additionally, the evolution of delayed emergence can profoundly alter the life-cycles of parasitoid wasps. For example, in species with delayed emergence, wasps attack young hosts that initially have low reproductive value for parasitoids, but only consume them after the hosts mature and their reproductive value increases. These findings suggest that delayed emergence may be a driver of the syndrome involving multiple traits related to host exploitation in parasitoid wasps. The high diversity of parasitoid wasps therefore provides a rich system for testing hypotheses about life-history syndromes. By linking delayed emergence to broader life-history strategies, this study lays a theoretical foundation for understanding life-history syndromes in parasitoid wasps.

1 Introduction

Understanding the evolution of resource exploitation, including its timings and allocation, within a life-history is a central research question in evolutionary biology. Parasitoids (wasps) are model systems to study resource-exploiter interactions, producing numerous studies on life-history evolution (Wajnberg et al., 2008). The life-histories of parasitoid wasps are particularly diverse (Godfray, 1994; Mayhew and Blackburn, 1999; Jervis et al., 2008; Jervis and Ferns, 2011; Quicke, 2014). For example, the order *Hymenoptera* comprises no less than 200,000 species in a conservative estimation (Pennacchio and Strand, 2006), probably each utilizing more or less different host species (mostly arthropods). Taking another example, there are life-history trait variations, by a factor of 18 in body-size (Jervis et al., 2003), more than one hundred in clutch size and lifetime potential fecundity, among wasp species (Jervis et al., 2008), with these variations presumably resulting from adaptation to host species. Because parasitoid life-history traits have significant impacts on the community structure of parasitoid and prey species (Lafferty et al., 2008), pest management (Wang et al., 2019), and coevolution (Amoroso and Antonovics, 2020), improving our understanding of the evolution of parasitoid wasps provides crucial implications for biodiversity and its applications.

Life-history trait variations are found typically in sequential timings of host exploitation, from attacking, hatching to emerging (Alonzo and Kindsvater, 2008). For example, some parasitoid wasps (e.g., *Apanteles kariyai*) attack only early juvenile hosts and emerge without the host's further development to late juvenile hosts (Hayakawa, 1991). Meanwhile, others (e.g., *Ichneumon promissorius*) lay eggs on late juvenile hosts and emerge immediately (Carpenter, 1995). Importantly, such differences in the exploitation timings may influence the phenotype at later life-history stages, by changing the degree to which parasitoids acquire resources from younger (and thus smaller) versus older (thus larger) hosts. In other words, the evolution of exploitation timings strongly influences the traits in the entire lifespan.

The delayed emergence, resulting from parasitoids not consuming their hosts immediately after the attack, is a key factor in creating diversity in the life-history characteristics of parasitoid wasps. (Jervis and Ferns, 2011). For example, paraistoids can be categorized into two groups according to this trait; namely, "idiophytic" species ('idio' means 'own/individual/personal' descending from Greek, indicating that hosts are personalized or monopolized by individual wasps) that consume the host immediately after the attack, and "koinophytic" species ('koino' means 'common', also from Greek, indicating parasitoids live with a living host) that allow hosts to continue to feed and grow after parasitization (Haeselbarth, 1978). By applying this criterion for a wider range of parasitoid wasp species, the major dichotomy is proposed in the larval developmental pattern of parasitoid wasps, namely idiobiont-koinobiont extremes (Askew, 1986). Parasitoid wasps can be therefore divided into two major groups based on delayed emergence trait (Godfray, 1994; Quicke, 2014; Askew, 1986), each with a different life-history characteristics.

To test the dichotomous hypothesis, the relationship between delayed emergence trait and other traits has been examined. Statistical analyses that control for phylogenetic correlations also provide some support for the

dichotomy hypothesis, suggesting possible mechanisms for the observed pattern of association between life-history traits (Mayhew and Blackburn, 1999). For example, koinobionts exhibit higher larval mortality, greater fecundity, and smaller egg sizes compared to idiobionts (Mayhew and Blackburn, 1999). This study suggests that these traits are explained by both selective pressures and developmental constraints: the higher mortality rate may result from the increased exposure or immaturity of the host, which in turn drives the evolution of higher fecundity. This increase in fecundity is traded-off against smaller egg sizes (Mayhew and Blackburn, 1999). Godfray (1994) predicts the selection pressures on parasitoid wasps based on the dichotomy hypothesis and suggest a diagram depicts chains of selective drivers of the diversity of life-history in parasitoid wasps, particularly focusing on idiobionts and koinobionts (Godfray, 1994). However, it is poorly known when natural selection favours delayed emergence in life-history strategies in parasitoids, which limits our understanding of the variations in life-history traits in parasitoid wasps.

A previous theoretical study showed that density-dependent factors, such as search efficiency and mortality during the delay period, influence the selection pressure for delayed emergence (Hackett-Jones et al., 2011). The study analyzed the evolution of delayed emergence in parasitoid–host systems where the host population has a homogeneous structure. In such models, the parasitoid's strategy is defined by its search efficiency and the timing of search initiation and termination, with the interval corresponding to the period of delayed emergence. However, this framework does not consider how host maturation affects the parasitoid's fecundity, because the model does not include a class structure of hosts.

In this study, we use mathematical models to explore the adaptive evolution of delayed emergence, with the aim of explaining the dichotomy. We specifically investigate the evolutionary dynamics of both the parasitoids' preference for hosts' developmental stages and that for delayed emergence, since parasitoid wasp can attack young hosts due to their ability to live in host body. Using adaptive dynamics theory (Dieckmann and Law, 1996), we determine the directions of natural selection on these traits and the stability of evolutionary consequences (i.e., the attainable strategies that no other rare mutant with small phenotypic differences can increase in frequency). As the host population is physiologically structured, we use Fisher's reproductive values (Fisher, 1930) to predict the optimal timing of emergence for parasitoid wasps.

2 Model

We first construct the population dynamics of stage-structured host-parasitoid system to investigate the evolution of delayed emergence. We consider a host population with a stage structure consisting of early juvenile hosts, late juvenile hosts, and adult hosts. Either stage of the juvenile hosts, but not adults, may be attacked by the parasitoid wasps and subsequently transits to the infected stage (Figure 1 for a diagram depicting the patterns of life-cycles). The variables and parameters are summarized in Table 1.

We consider two independent traits subject to natural selection. One is preference for juvenile hosts, x, and

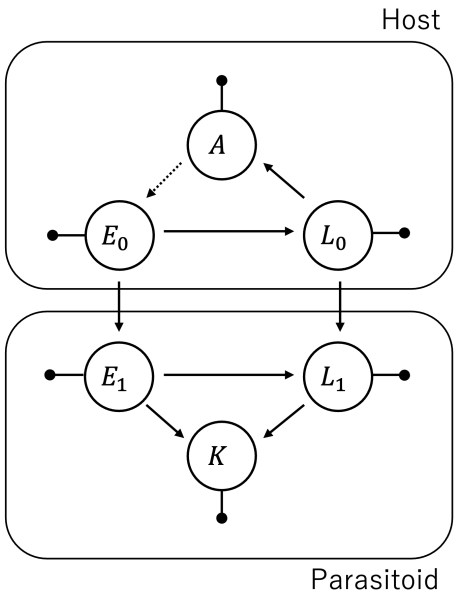


Figure 1: Diagram of host and parasitoid wasps. E represents the density of early juvenile hosts, L represents the density of late juvenile hosts, A represents the density of adult hosts, and K represents the density of parasitoid wasps. We use subscript 0 for uninfected and 1 for infected state, respectively. Black points represent death.

the other is probability of delayed emergence, y, with $0 \leq x \leq 1$ and $0 \leq y \leq 1$. For example, the case with $x = 1$ and $y = 0$ corresponds to the life-cycle graph which is depicted as Figure 2A. We use ordinary differential equations to describe the population dynamics of hosts and parasitoids:

$$
\begin{aligned}
\dot{E_0} &= rA - a_0E_0 - \lambda x E_0 K - d_E E_0 \\
\dot{L_0} &= a_0E_0 - \lambda(1-x)L_0K - b_0L_0 - d_L L_0 \\
\dot{A} &= b_0L_0 - d_A A \\
\dot{E_1} &= \lambda x E_0 K - y a_1 E_1 - ((1-y)\sigma_E + d_E + v_E)E_1 \\
\dot{L_1} &= y a_1 E_1 + \lambda(1-x)L_0K - (\sigma_L + d_L + v_L)L_1 \\
\dot{K} &= f_E(1-y)\sigma_E E_1 + f_L \sigma_L L_1 - d_K K
\end{aligned}
\tag{1}
$$

(with dot representing the time derivative). Here, from top to bottom, the ordinary differential equations represent the dynamics of non-parasitized early-juvenile host, non-parasitized late-juvenile hosts, adulst hosts, parasitized early juvenile host, paraasitized late juvenile host, and adult wasps (emerging from either early or late hosts). The key ingredients are the transitions in states due to (i) parasitism (e.g., from E_0 to E_1), and (ii) ontogeny (e.g., from E to L) (Figure 1). Surviving late juvenile hosts may develop into adults (with density A).

The first three lines in Equation (1) describe the population dynamics of non-parasitized hosts. With non-parasitized early juvenile hosts parasitized at rate $\lambda x K$ with searching efficiency of parasitoids λ and the probability of preferentially parasitizing early juvenile hosts x, the non-parasitized early juvenile hosts (with density E_0) are produced by adults at rate r, develop into late juvenile hosts at rate a_0 and die at rate d_E; they are

parasitized by wild-type parasitoids at rate λx. Non-parasitized late juvenile hosts (with density L_0) develop from non-parasitized early juvenile hosts, but may be parasitized at rate $\lambda(1-x)K$, develop into adults at rate b_0, or die at rate d_L.

The middle two lines in Equation (1), on the other hand, describe the population dynamics of parasitized hosts. Parsitized early juvenile hosts (with density E_1), having transited from the parasitism of early juvenile hosts (E_0), develop into late juvenile hosts, or otherwise die due either to (i) parasitoid emergence, at rate $(1-y)\sigma_L$ (ii) natural mortality at rate d_E, or (iii) parasitoid-induced additional mortality at rate v_E. Parasitized late juvenile hosts (with density L_1) develop from parasitized early juvenile hosts due to ontogeny or transit from non-parasitized late juvenile hosts due to parasitism, but die at rate σ_L due to emergence of the parasitoid, or at rate due to natural or parasitoid-induced additional mortality.

The last line in Equation (1), describes the population dynamics of adult parasitoids. Adult parasitoids emerge from either parasitized early juvenile hosts (at rate f_E) or late juvenile hosts (at rate f_L), where f_E or f_L represents the fecundity of adult parasitoids (per capita) emerging from early larvae or late larvae, respectively.

Table 1: Definitions of the parameters

Parameter	Definition
x	Preference for uninfected early juvenile hosts in parasitoid wasps (evolving trait)
y	Preference for delayed emergence in parasitoid wasps (evolving trait)
λ	Searching efficiency of parasitoid wasps
r	Birth rate of hosts
a_0	Growth rate of uninfected early juvenile hosts
b_0	Growth rate of uninfected late juvenile hosts
a_1	Growth rate of infected early juvenile hosts
d_E	Mortality rate of uninfected early juvenile hosts
d_L	Mortality rate of uninfected late juvenile hosts
d_A	Mortality rate of adult hosts
d_K	Mortality rate of parasitoid wasps
v_E	Parasitoid-induced additional mortality against early juvenile hosts
v_L	Parasitoid-induced additional mortality against late juvenile hosts
σ_E	Consumption rate of early juvenile hosts by parasitoid wasps
σ_L	Consumption rate of late juvenile hosts by parasitoid wasps
f_E	Per capita fecundity of parasitoid wasps emerging from early juvenile hosts
f_L	Per capita fecundity of parasitoid wasps emerging from late juvenile hosts

To investigate the long-term evolutionary dynamics of the preference for host stages in wasps and the preference for delayed emergence in wasps, we apply the adaptive dynamics theory (Dieckmann and Law, 1996). We first suppose that the resident population dynamics, Equation (1), have reached an endemic equilibrium, and introduce a rare mutant of the parasitoid wasp attempting to invade the resident equilibrium. We find there is a unique equilibrium of the resident dynamics (Appekdix D). To determine whether the mutant parasitoid wasp can invade the resident population, we derive the invasion fitness proxy R_{m} by using the next-generation

theorem (Van den Driessche and Watmough, 2002; Hurford et al., 2010):

$$
\begin{aligned}
&R_{\mathrm{m}}(x_{\mathrm{m}}, y_{\mathrm{m}}; x, y) \\
&= \underbrace{\frac{\lambda x_{\mathrm{m}} E_0^*}{d_K} \cdot \frac{f_E(1-y_{\mathrm{m}})\sigma_E}{y_{\mathrm{m}} a_1 + d_E + v_E + (1-y_{\mathrm{m}})\sigma_E}}_{\text{path (A)}} \\
&+ \underbrace{\frac{\lambda x_{\mathrm{m}} E_0^*}{d_K} \cdot \frac{y_{\mathrm{m}} a_1}{y_{\mathrm{m}} a_1 + d_E + v_E + (1-y_{\mathrm{m}})\sigma_E} \cdot \frac{f_L \sigma_L}{d_L + v_L + \sigma_L}}_{\text{path (B)}} \\
&+ \underbrace{\frac{\lambda (1-x_{\mathrm{m}}) L_0^*}{d_K} \cdot \frac{f_L \sigma_L}{d_L + v_L + \sigma_L}}_{\text{path (C)}} .
\end{aligned}
\tag{2}
$$

where E_0^* and L_0^* represent the density of uninfected early juvenile host and uninfected late juvenile host at the endemic equilibrium, respectively. In Equation (2), we partition the reproductive success of the mutant individual into that through each of the pathways presented in Figure 2. The first, 'path (A)' term represents the reproductive success by attacking and consuming early juvenile hosts (Figure 2A). The path (B) term represents the reproductive success by attacking on early juvenile hosts and consuming late juvenile hosts (Figure 2B). The path (C) term represents the reproductive success by attacking and consuming late juvenile hosts (Figure 2C).

The rare mutant can invade the resident population when $R_{\mathrm{m}} > 1$. By using this criterion, we examine a candidate evolutionary consequence, its evolutionary stability [i.e., whether the rare mutant can invade a population dominated by the wild type][]smith1982evolution, and also convergence stability (i.e., evolutionary dynamics convergence of the traits; Eshel et al., 1983).

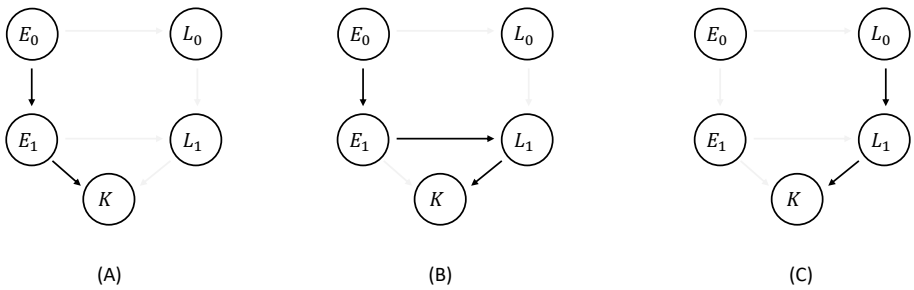


Figure 2: Three pathways involved in the invasion fitness. (A) Parasitoid wasps attack the early juvenile hosts and immediately prey on them. (B) Parasitoid wasps attack the early juvenile hosts and wait until they grow to late juveniles, and then prey on them. (C) Parasitoid wasps attack on the late juvenile host and immediately prey on them.

Although the results in the main text all assume the exponential growth of the parasitoid-free host population (as this assumption immensely simplify the analyses), we also analyze the case where host density follows logistic growth (i.e., density-dependent regulation of of host fecundity). Specifically, we conduct numerical, deterministic simulations (Appendix E).

3 Results

3.1 Evolution of delayed emergence

First, we show the evolution of the preference for delayed emergence y. Selection gradient (Appendix B) is given by:

$$\left.\frac{\partial R_{\mathrm{m}}(x_{\mathrm{m}}, y_{\mathrm{m}}; x, y)}{\partial y_{\mathrm{m}}}\right|_{x_{\mathrm{m}}=x, y_{\mathrm{m}}=y} = (\alpha F_L - F_E) \times (\text{positive term}). \tag{3}$$

where $\alpha = a_1/(a_1+d_E+v_E)$ is the probability of successful development from early to late juvenile for parasitized host, $F_L = f_L\sigma_L/(d_L + v_L + \sigma_L)$ or $F_E = f_E\sigma_E/(d_E + v_E + \sigma_E)$ is the product of (1)the probability of consumption of the host and (2)the fecundity(i.e., the expected conversion rate of parasitoid wasps preying on early or late juvenile hosts, respectively). While the selection gradient depends on both of the evolutionary traits x and y, the sign depends only on the parameters such as α and F_L. Thus, the preference evolves monotonically to either 0 or 1 (i.e. immediately emerge or delayed emergence) depending on the magnitude relationship between αF_L and F_E. The parameters F_E and αF_L represent the expected conversion rate of early juvenile hosts when parasitoid wasps prey on the host immediately (pathway A in Figure 2) and the conversion rate when parasitoid wasps allow the host to grow after the attack (pathway B in Figure 2), respectively.

Second, we assess the evolutionary stability. Since the candidate evolutionary consequence of y is at the boundary (0 or 1), the evolutionary stability can be simply implied by the convergence stability condition (Appendix C). We summarize the evolutionary consequences with respect to parameters in Figure 3.

We can interpret the monotonicity result of Equation (3) using the reproductive values (Fisher, 1930). Equation (3) represents the relationship between the reproductive value of early juvenile hosts when the parasitoid wasps prey immediately and wait for the host to develop into late juvenile hosts. The preference for delayed emergence is likely to be favoured when the product of (1) the growth probability of the parasitized early juvenile hosts and (2) the average conversion rate when wasps consume the late juvenile host, is higher than (3) the conversion rate of the early juvenile host. That is, in terms of the total reproductive value, late juvenile hosts are valuable than than juvenile hosts. This result suggests that the delayed emergence trait is biased to an extreme, leading to a clear division of parasitoid wasps into koinobionts and idiobionts depending on these conditions.

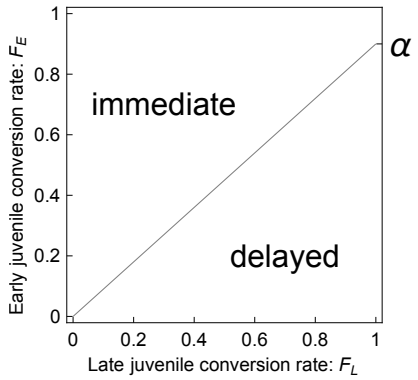


Figure 3: The evolutionary consequences of the preference for delayed emergence($\alpha = a_1/(a_1 + d_E + v_E)$: the growth probability of the infected early juvenile host, $F_E = f_E\sigma_E/(d_E + v_E + \sigma_E)$: the average fecundity rate of parasitoid wasps preying on the early juvenile host and $F_L = f_L\sigma_L/(d_L + v_L + \sigma_L)$: that on the late juvenile host)

3.2 Patterns of life-cycles

Now we examine the evolution of the preference for early juvenile hosts (x) in each scenario involving y. The selection gradient of the preference for host stages x is given by:

$$\left.\frac{\partial R_{\mathrm{m}}(x_{\mathrm{m}}, y_{\mathrm{m}}; x, y)}{\partial x_{\mathrm{m}}}\right|_{x_{\mathrm{m}}=x, y_{\mathrm{m}}=y} = \frac{\lambda E_0^*}{d_K}\left(\left(p_{E\to L}F_L + p_{E\to K}f_E\right) - \frac{L_0^*}{E_0^*}(x)F_L\right). \tag{4}$$

(Appendix B), where $p_{E\to L} = ya_1/(ya_1+d_E+v_E+(1-y)\sigma_E)$ represents the probability of transition from infected early juvenile hosts to late ones and $p_{E\to K} = (1-y)\sigma_E/(ya_1 + d_E + v_E + (1-y)\sigma_E)$ represents the probability of hosts consumption by wasps. The sign of Equation (4) is determined by both y and x. According to the previous section, y evolves to either 0 or 1.

Since the invasion fitness proxy is linear in x_{m}, the second derivative is 0. That is,

$$\frac{\partial^2 R_{\mathrm{m}}(x_{\mathrm{m}}, y_{\mathrm{m}}; x, y)}{\partial x_{\mathrm{m}}^2} = 0. \tag{5}$$

The point is neutrally stable, which thus cannot be judged by the linear stability analysis. We carry out simulations to show that the trait values converge to the candidate evolutionary consequence over time and remain stable (Appendix E).

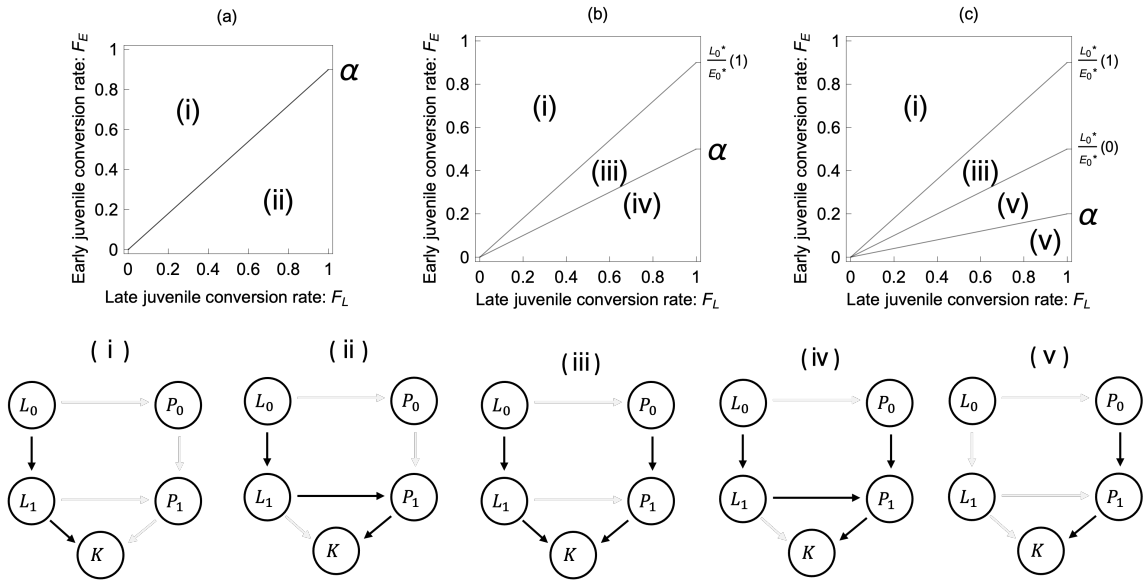


Figure 4: The patterns of delayed emergence. E or L represents early juvenile hosts or late juvenile hosts (respectively). (a) The singular point is 0 when the growth probability of the infected early juvenile host α is larger than the maximum of the density ratio of the uninfected late juvenile host to the early juvenile hosts L_0^*/E_0^*. (b) The singular point is between 0 and 1 (α is between the minimum and maximum of L_0^*/E_0^*). (c) The singular point is 1 (α is smaller the minimum of L_0^*/E_0^*).

The selection gradients on the preference for early juvenile hosts for both cases are given by:

$$\left.\frac{\partial R_{\mathrm{m}}(x_{\mathrm{m}}, y_{\mathrm{m}}; x, y)}{\partial x_{\mathrm{m}}}\right|_{x_{\mathrm{m}}=x, y_{\mathrm{m}}=y=0} \propto F_E - \frac{L_0^*}{E_0^*}(x) F_L, \tag{6}$$

$$\left.\frac{\partial R_{\mathrm{m}}(x_{\mathrm{m}}, y_{\mathrm{m}}; x, y)}{\partial x_{\mathrm{m}}}\right|_{x_{\mathrm{m}}=x, y_{\mathrm{m}}=y=1} \propto \left(\alpha - \frac{L_0^*}{E_0^*}(x)\right) F_L, \tag{7}$$

where $F_E = f_E \sigma_E/(d_E + v_E + \sigma_E)$ is the product of (1)the probability of consumption of early juvenile hosts and (2)the fecundity of them (i.e., the expected fecundity of parasitoid wasps preying on early juvenile hosts) and $F_L = f_L \sigma_L/(d_L + v_L + \sigma_L)$ is that on late juvenile host, $\alpha = a_1/(a_1 + d_E + v_E)$ is the growth probability of infected early juvenile host. A unique candidate evolutionary consequence x^* obtained analytically is evolutionary consequence of preference for host stages because the point is always convergence stable (i.e., natural selection leads the trait to evolve toward the point; Otto and Day, 2011). When the candidate evolutionary consequence is not between 0 and 1 (i.e., no interior points nullify the selection gradient), we carry out case-analyses to locate the singular point: the singular value is 0, between 0 and 1, or 1 (Appendix B.3). The evolutionary consequences in each case are shown in Figure 4. This result suggests that delayed emergence trait can generate the diversity of life-cycle of parasitoid wasps, including idiobiont and koinobiont.

3.3 Evolution of idiobiont and koinobiont

We define idiobiont as the parasitoid wasps to attack uninfected late juvenile hosts and prey on the host immediately. We also define koinobiont as the parasitoid wasps attack an uninfected early juvenile host and prey on the host after the host grows up to the late juvenile stage. Therefore, Figure 4v represents idiobiont and Figure 4ii represents koinobiont.

Both idiobiont and koinobiont are likely to evolve when early juvenile hosts is more valuable than late juvenile hosts ($F_L > F_E$). The evolution of idiobionts is more likely when the growth probability of infected early juvenile hosts is low, and the density of late juvenile hosts is higher than that of early juvenile hosts. Conversely, koinobionts are more likely to evolve when the growth probability of infected early juvenile hosts is high, and the density of late juvenile hosts is lower than that of early juvenile hosts.

All the analytical results presented here are based on the exponential growth of the host population, but we carry out numerical simulations assuming that the host population exhibits logistic growth (density-dependent regulation). We find consistent results between the two scenarios, confirming the robustness of our results against the logistic growth case (Appendix E).

4 Discussion

We have studied the evolution of delayed emergence as a life-history strategy. Our first key prediction is that the reproductive values of parasitoids determine the evolutionary consequences of the timing of emergence from the host (Fisher, 1930). Specifically, if the host's developmental growth does not increase the expected conversion efficiency (a measure of nutritional quality), the wasps are then selected to emerge from the host immediately. Otherwise, there occurs a variation in the timing of emergence, either early or late. In this case, the early emergence is selected for when the reproductive value of the host for wasps emerging early is higher than that for those waiting for host growth. These results can be understood as cost-benefit balance in delayed emergence (increased mortality versus increased resource quality). Overall, the results suggest that natural selection can shape the patterns of delayed emergence in parasitoid wasps, and such patterns are determined by the relative resource quality. Our predictions are consistent with a recent theoretical study on the evolution of parasite virulence in stage-structured host populations, including larvae and adults. This study indicates that the evolutionary consequences of virulence toward larval hosts depend on the interaction between host maturation rate and the infection pathway (Iritani et al., 2019). Specifically, virulence toward larval hosts evolves to be higher when either (i) the adult stage is relatively long and the transmission across host stages mostly occurs, or (ii) the larval stage is relatively long and the transmission across the same stage mostly occurs (Iritani et al., 2019). These results are similar to our findings, in which koinobiont evolves when early juvenile hosts are abundant and the probability of maturation under infection is high, and idiobiont evolves when late juvenile hosts are abundant and the probability of maturation is low. (Hackett-Jones et al., 2011) explicitly

incorporated a trade-off between emergence timing and the onset of parasitism to examine the evolution of emergence timing. In the model, early provides access to uninfected hosts, but also results in a longer the period of delayed emergence which increases the mortality. Thus, both the benefit of accessing host resources earlier and the cost due to increased mortality shape the evolution of emergence timing. In contrast, our study does not explicitly assume a trade-off between emergence timing and other traits. Instead, selection acts on the emergence timing through changes in the reproductive value as the host matures. This contrast reflects a different evolutionary mechanism whereas previous studies emphasized mortality risk and competition for host access, our study shows that the evolution of delayed emergence can be explained solely by temporal changes in host value.

Our second key insight is that the variation in the timing of emergence shapes the diversity in the life-cycle (Figure 2). The difference in emergence-timing results in the differences in the attack-timing (i.e., whether the target is early or late juvenile hosts). Since parasitoid wasps consume their hosts at the time of emergence, the timing of emergence, but not that of attack, determines their fitness. We considered two developmental stages of hosts available for the parasitoid wasps. In the model, infected early juvenile hosts are allowed to grow to a later developmental stage. As a result, the host utilization strategies of parasitoid wasps exhibit three distinct patterns as follows: attacking young hosts and hatching immediately (Figure 2A), attacking young hosts with prolonged emergence (Figure 2B), and attacking older host and consuming immediately (Figure 2C). This line of results suggests that the variation in the emergence timing produces diversity in the life-cycle of parasitoid wasps.

In our analysis, we found that the density ratio of late juvenile hosts to early ones affects the evolution of wasps' preference for hosts' developmental stages. For example, the greater the density ratio, the more the parasite evolves to attack late juvenile hosts. Intuitively, the relative resource quantity matters to the evolution of parasitoid wasps' exploitation strategy. Finding examples of the density ratio between two developmental stages of hosts can be difficult as the stage structures are more complex in nature. Nonetheless, some information is available about host-mortality, which significantly influences the density ratio. Idiobiont hosts are considered to have lower natural mortality than koinobiont hosts, presumably because idiobionts tend to attack hosts that have not been exposed to predation risk, as the host is immobile (Mayhew and Blackburn, 1999; Quicke, 2014). However, this remains a speculation, as empirical evidence supporting this assumption is lacking. In contrast, koinobionts can parasitize hosts that are exposed to predation because they do not paralize the hosts (Quicke, 2014). Consequently, koinobionts parasitize hosts with a high ratio of late to early juvenile stages, while idiobionts parasitize host with a low ratio, consistent with observed findings, although density ratios depend also on differences in fecundity and growth rates. Our finding provides a qualitative prediction for the effect of the relative availability of resource on the diversification of the parasitoid wasps' exploitation strategies.

In addition to the quantity, resource quality plays a role in the evolution of idiobiont and koinobiont strategies. Specifically, the host quality must increase with host growth for natural selection to favour idiobiont

and koinobiont strategies. Several empirical studies support this finding. In *Trissolcus mitsukurii* (egg parasitoid) and *Sclerodermus harmandi* (larval parasitoid), for example, host size positively correlates with parasitoids' body size and fecundity (Arakawa et al., 2004; Liu et al., 2011); female parasitoids emerging from larger hosts tend to have higher fertility in *Lysiphlebia mirzai* (nymphal parasitoid) and *Dirhinus giffardii* (pupal parasitoid) (Pandey and Singh, 1999; Wang and Messing, 2004). However, the relationship between host size and fecundity is not straightforward. For example, in *Campoplex dubitator* (larval parasitoid), while larger and more fertile females emerge from larger hosts, their developmental periods are also longer (Jenner and Kuhlmann, 2006). Despite these complexities, we might hypothesize that developmental stages reflect resource qualities that determine the adaptive evolution of exploitation strategies of parasitoid wasps. Future empirical studies may test the hypothesis and carry out comparative analysis to examine the predicted patterns in this study.

We theoretically studied the life-history evolution in parasitoid wasps based on the idiobiont and koinobiont dichotomy. Previous studies have used comparisons and meta-analyses of various traits associated with this dichotomy, suggesting correlations between the occurrence of specific traits. However, these correlations do not constitute a theory of life-history evolution (Mayhew and Blackburn, 1999), as these studies do not disentangle the causal effects of some trait's evolution on others. By focusing on the evolution of delayed emergence, the present study formulated the relationship between delayed emergence which is a dichotomous driver and other life-history traits (e.g. fecundity and parasitoid-induced additional mortality). Therefore, our study helps fill the knowledge gap in the correlated selection in life-history strategies.

The present study predicted the occurrence of "delayed emergence-syndrome" in parasitoid wasps. When convergent evolution involves the simultaneous appearance of multiple traits, it is referred to as "syndrome", defined as multiple traits evolving concurrently in response to common environmental pressures (Sinnott-Armstrong et al., 2022). For example, parasitoid wasps of the family Ichneumonidae that exploit wood-boring hosts are associated with six traits, such as teeth on the ovipositor and a long terebra (Legrand et al., 2016). In general, mechanisms for the simultaneous evolution of multiple traits include supergenes arising from chromosomal inversions that involve multiple genetic functions, as well as the multifaceted effects of single genes (or pleiotropy; (Raia et al., 2010; Thompson and Jiggins, 2014)). Highly diverse taxa, such as parasitoid wasps, may provide a suitable system for examining convergent evolution and syndromes (Dal Pos and Sharanowski, 2024). Because parasitoid wasps fully rely on their hosts to complete their life-cycle (Dal Pos and Sharanowski, 2024), natural selection favors various morphological traits associated with locating a host, laying eggs, developing on the host, and ultimately exiting the host (Quicke, 2014). Historically, the concept of syndrome has been considered to arise from adaptation to a single selective factor (e.g., primary pollinator, preferred habitat) (Sinnott-Armstrong et al., 2022). The idiobiont-koinobiont dichotomy can be therefore interpreted as a syndrome driven by delayed emergence. In particular, our analytical calculations of the evolutionary dynamics can allow for incorporating other factors (e.g. competition among wasps and non-linear searching efficiency of wasps). Thus, our study can analyze the relationship of selection pressure

between various life-history characteristics and delayed emergence.

To simplify the analysis, the possibility of multiple parasitisms is omitted from the model, despite their occurrences in nature. Because multiple parasitisms are assocaited with competition, such interactions may cause temporal niche differentiation in host developmental stages and promote the evolution of delayed emergence. Future studies may incorporate the effects of such competition into a model, which may provide testable predictions for the evolution of life-history variations in parasitoid wasps. Moreover, although our model does not consider the host evolution, hosts can evolve defense traits such as tolerance, potentially leading to coevolution. Age structure of hosts may further shape these dynamics, as shown in a model of tolerance–virulence coevolution (Buckingham and Ashby, 2024). Future extensions of the present model that incorporate host evolution could provide a useful theoretical framework for understanding how host–parasitoid coevolution drives variation in emergence timing and associated life-history traits.

In summary, our results show that host density and fecundity have a significant impact on delayed emergence evolution and that delayed emergence can alter the structure of the life-cycle graph. These results are supported, in part, by empirical studies. Our approach reveals a relationship between the delayed emergence trait and other traits in evolution that could not be explained by empirical studies. This study provides a theoretical basis to interpret the idiobiont-koinobiont dichotomy as a life-history syndrome of parasitioid wasps generated by the delayed emergence. Future studies could reveal the mechanism that produces the polarized traits of idiobiont and koinobiont by incorporating the effects of resource competition.

Appendix

A Next generation matrix and invasion fitness proxy

To study life-history evolution, we assess the invasion possibility of a rare mutant into the resident population assuming that the ecological dynamics has reached an equilibrium. The population dynamics of the rare mutant is given by:

$$\frac{\mathrm{d}\mathbf{X}_\mathrm{m}}{\mathrm{d}t} = (\mathbf{F}_\mathrm{m} - \mathbf{V}_\mathrm{m})\mathbf{X}_\mathrm{m} \tag{A-1}$$

where

$$
\begin{aligned}
\mathbf{X}_{\mathrm{m}} &= \begin{pmatrix} E_{1,\mathrm{m}} \\ L_{1,\mathrm{m}} \\ K_{\mathrm{m}} \end{pmatrix} \\
\mathbf{F}_{\mathrm{m}} - \mathbf{V}_{\mathrm{m}} &= \begin{pmatrix} -(y_{\mathrm{m}}a_1 + d_E + v_E + (1-y_{\mathrm{m}})\sigma_E) & 0 & \lambda x_{\mathrm{m}} E_0^* \\ y a_1 & -(d_L + v_L + \sigma_L) & \lambda(1-x_{\mathrm{m}})L_0^* \\ (1-y_{\mathrm{m}})f_E\sigma_E & f_L\sigma_L & -d_K \end{pmatrix} \\
\mathbf{F}_{\mathrm{m}} &= \begin{pmatrix} 0 & 0 & \lambda x_{\mathrm{m}} E_0^* \\ 0 & 0 & \lambda(1-x_{\mathrm{m}})L_0^* \\ (1-y_{\mathrm{m}})f_E\sigma_E & f_L\sigma_L & 0 \end{pmatrix} \\
\mathbf{V}_{\mathrm{m}} &= \begin{pmatrix} y_{\mathrm{m}}a_1 + d_E + v_E + (1-y_{\mathrm{m}})\sigma_E & 0 & 0 \\ -y_{\mathrm{m}}a_1 & d_L + v_L + \sigma_L & 0 \\ 0 & 0 & d_K \end{pmatrix}
\end{aligned}
\tag{A-2}
$$

The Next-Generation Theorem (Van den Driessche and Watmough, 2002) states that if the dominant eigenvalue of the next-generation matrix $\mathbf{G}_{\mathrm{m}} = \mathbf{F}_{\mathrm{m}} \cdot \mathbf{V}_{\mathrm{m}}^{-1}$ exceeds 1, then the mutant can invade and drive the resident individuals extinct. The squared, dominant eigenvalue $\rho(\mathbf{G}_{\mathrm{m}})$ is given by:

$$
\begin{aligned}
\left(\rho(\mathbf{G}_{\mathrm{m}})\right)^2 =& \frac{\lambda x_{\mathrm{m}} E_0^*}{d_K} \cdot \frac{y_{\mathrm{m}}a_1}{y_{\mathrm{m}}a_1 + d_E + v_E + (1-y_{\mathrm{m}})\sigma_E} F_L \\
&+ \frac{\lambda x_{\mathrm{m}} E_0^*}{d_K} \cdot \frac{f_E(1-y_{\mathrm{m}})\sigma_E}{y_{\mathrm{m}}a_1 + d_E + v_E + (1-y_{\mathrm{m}})\sigma_E} \\
&+ \frac{\lambda(1-x_{\mathrm{m}})L_0^*}{d_K} F_L
\end{aligned}
\tag{A-3}
$$

where

$$
\begin{aligned}
\mathbf{G}_{\mathrm{m}} &= \begin{pmatrix} 0 & 0 & \dfrac{\lambda x_{\mathrm{m}} E_0^*}{d_K} \\ 0 & 0 & \dfrac{\lambda(1-x_{\mathrm{m}})L_0^*}{d_K} \\ p_{m,E\to L}F_L + p_{m,E\to K}f_E & F_L & 0 \end{pmatrix} \\
p_{m,E\to L} &= \frac{y_{\mathrm{m}}a_1}{y_{\mathrm{m}}a_1 + d_E + v_E + (1-y_{\mathrm{m}})\sigma_E} \\
p_{m,E\to K} &= \frac{(1-y_{\mathrm{m}})\sigma_E}{(y_{\mathrm{m}}a_1 + d_E + v_E + (1-y_{\mathrm{m}})\sigma_E}
\end{aligned}
\tag{A-4}
$$

where $p_{m,E\to L}$ represents the probability that mutant wasps' larvae can transit from infected early juvenile hosts to late ones and $p_{E\to K}$ represents the probability that mutant wasps' larvae consume the host. Since $\rho^2 \gtrless 1$ is equivalent to $\rho \gtrless 1$, we use ρ^2 for the invasion proxy.

B Selection gradient

In this section, we derive the stable class distribution, reproductive values and selection gradient using the next-generation matrix.

B.1 Stable class distribution

The right eigenvector associated with the dominant eigenvalue of the next-generation matrix gives the stable class distribution (Taylor, 1990; Otto and Day, 2011), which represents the asymptotic distribution of mutant alleles. It is given by:

$$\mathbf{G}\vec{u} = \rho(\mathbf{G})\vec{u}, \tag{A-5}$$

where

$$\vec{u} = \begin{pmatrix} \dfrac{\lambda x E_0^*}{d_K} \\ \dfrac{\lambda(1-x)L_0^*}{d_K} \\ \rho(\mathbf{G}) \end{pmatrix} \tag{A-6}$$

represents the stable distribution of the mutant allele.

B.2 Reproductive value of each class

The left eigenvector associated with the dominant eigenvalue of the next-generation matrix gives the reproductive values of each of these classes (Fisher, 1930; Otto and Day, 2011). The reproductive values represent the contributions of individuals in each class to future reproduction after sufficiently long generations, given by:

$$\vec{v}^\top \mathbf{G} = \rho(\mathbf{G})\vec{v}^\top \tag{A-7}$$

where

$$
\begin{aligned}
\vec{v}^{\top} &= \begin{pmatrix} p_{E\to L}F_L + p_{E\to K}f_E \\ F_L \\ \rho(\mathbf{G}) \end{pmatrix} \\
p_{E\to L} &= \frac{ya_1}{ya_1 + d_E + v_E + (1-y)\sigma_E} \\
p_{E\to K} &= \frac{(1-y)\sigma_E}{ya_1 + d_E + v_E + (1-y)\sigma_E}
\end{aligned}
\tag{A-8}
$$

$p_{E\to L}$ represents the probability that wasps' larvae can transit from infected early juvenile hosts to late ones and $p_{E\to K}$ represents the probability that mutant wasps' larvae consume the host.

B.3 selection gradient of x

From the calculations above, we have $\vec{u}\vec{v} = 2\rho^2(\mathbf{G}) = 2$. Let $c_i = u_i v_i$ be class reproductive value of class i; the selection gradient of x is given by:

$$
\begin{aligned}
\frac{\partial}{\partial x_{\mathrm{m}}}\left(\frac{\vec{v}\mathbf{G}_{\mathrm{m}}\vec{u}}{\vec{v}\vec{u}}\right)\Bigg|_{x_{\mathrm{m}}=x} =& \frac{1}{2}c_1\left(\frac{\partial g_{11}}{\partial x_{\mathrm{m}}} + \frac{v_2}{v_1}\cdot\frac{\partial g_{21}}{\partial x_{\mathrm{m}}} + \frac{v_3}{v_1}\cdot\frac{\partial g_{31}}{\partial x_{\mathrm{m}}}\right) \\
&+\frac{1}{2}c_2\left(\frac{v_1}{v_2}\cdot\frac{\partial g_{12}}{\partial x_{\mathrm{m}}} + \frac{\partial g_{22}}{\partial x_{\mathrm{m}}} + \frac{v_3}{v_2}\cdot\frac{\partial g_{32}}{\partial x_{\mathrm{m}}}\right) \\
&+\frac{1}{2}c_3\left(\frac{v_1}{v_3}\cdot\frac{\partial g_{13}}{\partial x_{\mathrm{m}}} + \frac{v_2}{v_3}\cdot\frac{\partial g_{23}}{\partial x_{\mathrm{m}}} + \frac{\partial g_{33}}{\partial x_{\mathrm{m}}}\right) \\
=& \frac{1}{2}c_3\left(\frac{v_1}{v_3}\cdot\frac{\partial g_{13}}{\partial x_{\mathrm{m}}} + \frac{v_2}{v_3}\cdot\frac{\partial g_{23}}{\partial x_{\mathrm{m}}}\right)
\end{aligned}
\tag{A-9}
$$

where

$$
\begin{aligned}
\frac{\partial \mathbf{G}_{\mathrm{m}}}{\partial x_{\mathrm{m}}}\Bigg|_{x_{\mathrm{m}}=x} &= \begin{pmatrix} \frac{\partial g_{11}}{\partial x_{\mathrm{m}}} & \frac{\partial g_{12}}{\partial x_{\mathrm{m}}} & \frac{\partial g_{13}}{\partial x_{\mathrm{m}}} \\ \frac{\partial g_{21}}{\partial x_{\mathrm{m}}} & \frac{\partial g_{22}}{\partial x_{\mathrm{m}}} & \frac{\partial g_{23}}{\partial x_{\mathrm{m}}} \\ \frac{\partial g_{31}}{\partial x_{\mathrm{m}}} & \frac{\partial g_{32}}{\partial x_{\mathrm{m}}} & \frac{\partial g_{33}}{\partial x_{\mathrm{m}}} \end{pmatrix}\Bigg|_{x_{\mathrm{m}}=x} \\
&= \begin{pmatrix} 0 & 0 & \frac{\lambda E_0^*}{d_K} \\ 0 & 0 & -\frac{\lambda L_0^*}{d_K} \\ 0 & 0 & 0 \end{pmatrix}
\end{aligned}
\tag{A-10}
$$

$$c_3 = \rho^2(\mathbf{G}) = 1$$

$$v_1 = p_{E\to L}F_L + p_{E\to K}f_E$$

$$v_2 = F_L$$

$$v_3 = \rho(\mathbf{G}) = 1$$

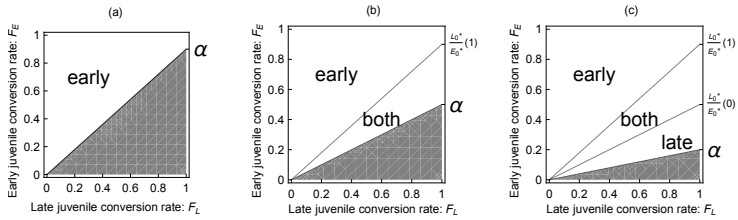


Figure 5: Evolutionary consequences of the preference for host stages when wasps emerge immdiately. Immediately emerge ($y = 0$) is convergence stable in the white area, but not in the gray area. Parasitoid wasps attack only early juvenile hosts in the "early" area, attack both of early and late juvenile hosts in the "both" area, and attack late juvenile hosts in the "late" area. (a) The singular point is 0 (the growth probability of the infected early juvenile host α is larger than the maximum of the density ratio of the uninfected late juvenile host to the early one L_0^*/E_0^*). (b) The singular point is between 0 and 1 (α is between the minimum and maximum of L_0^*/E_0^*). (c) The singular point is 1 (α is smaller the minimum of L_0^*/E_0^*).

B.3.1 Evolution of preference for host stages when imediate emergence occurs.

The selection gradient on the preference for early juvenile hosts is

$$\left.\frac{\partial R_{\mathrm{m}}(x_{\mathrm{m}}, y_{\mathrm{m}}; x, y)}{\partial x_{\mathrm{m}}}\right|_{x_{\mathrm{m}}=x, y_{\mathrm{m}}=y=0} \propto F_E - \frac{L_0^*}{E_0^*}(x) F_L. \tag{A-11}$$

where $F_E = f_E\sigma_E/(d_E + v_E + \sigma_E)$ is the expected fecundity of parasitoid wasps preying on early juvenile host and $F_L = f_L\sigma_L/(d_L + v_L + \sigma_L)$ is that on late juvenile host. A unique candidate evolutionary consequence x^* can be obtained analytically. The point is always convergence stable because L_0^*/E_0^* is monotonically increasing function of x^*. Evolutionary consequences of the preference for host stages are always continuously stable in the case that parasitoid wasps consume hosts immediately after they attack the hosts. According to result section, we obtain the evolutionary consequences in three cases (Figure 5). Equation A-11 represents the relationship between the class reproductive value of uninfected early juvenile hosts and that of late ones when the parasitoid wasps prey immediately. We can interpret Equation A-11 as the evolution of delayed emergence such that the hosts' class reproductive value maximizes. When parasitoid wasps consume hosts immediately after they attack hosts, the preference for early juvenile hosts is likely to evolve if the average conversion rate and the density of late juvenile hosts are higher than that of early juvenile hosts. In addition, the lower the growth probability of infected early juvenile hosts, the greater the diversity of host stages can evolve.

B.3.2 Evolution of preference for host stages when delayed emergence occurs.

The selection gradient on the preference for early juvenile hosts is

$$\left.\frac{\partial R_{\mathrm{m}}(x_{\mathrm{m}}, y_{\mathrm{m}}; x, y)}{\partial x_{\mathrm{m}}}\right|_{x_{\mathrm{m}}=x, y_{\mathrm{m}}=y=1} \propto \left(\alpha - \frac{L_0^*}{E_0^*}(x)\right) F_L. \tag{A-12}$$

where $\alpha = a_1/(a_1 + d_E + v_E)$ is the growth probability of infected early juvenile host, $F_L = f_L\sigma_L/(d_L + v_L + \sigma_L)$ is the expected fecundity rate of parasitoid wasps preying on late juvenile host. A unique candidate

evolutionary consequence x^* can be obtained analytically. The point is always convergence stable because L_0^*/E_0^* is monotonically increasing function of x^*. Evolutionary consequences of the preference for host stages are always continuously stable in the case that parasitoid wasps do not consume hosts immediately after they attack the hosts. According to result section, we obtain the evolutionary consequences in three cases (Figure 6). Equation A-12 represents the relationship between the class reproductive value of uninfected early juvenile hosts and that of late ones when wasps wait for their maturation. We can interpret Equation A-12 as the evolution of delayed emergence such that the hosts' class reproductive value maximizes. Therefore, in the case that parasitoid wasps do not consume hosts immediately after they attack the hosts, the preference for the uninfected early juvenile host is likely to evolve under two conditions: (1) the growth probability of the infected early juvenile host is high, and (2) the density of the late juvenile host is lower than that of early juvenile hosts.

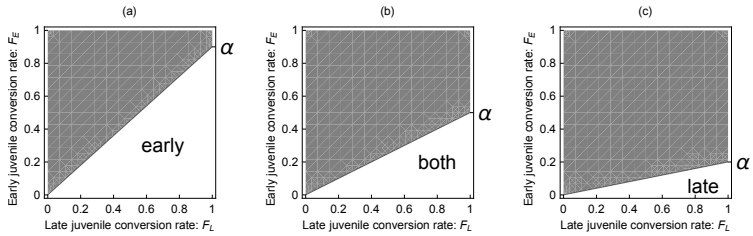


Figure 6: The evolutionary consequences of preference for host stages when delayed emergence occurs. Delayed emergence ($y = 1$) is convergence stable in the white area, but not in the gray area. Parasitoid wasps attack only early juvenile hosts in the "early" area, attack both early and late juvenile hosts in the "both"area, and attack late juvenile hosts in the "late" area.

B.4 selection gradient of y

Selection gradient of y is given by:

$$\begin{aligned}\frac{1}{2}\vec{v}\left.\frac{\partial \mathbf{G}_{\mathrm{m}}}{\partial y_{\mathrm{m}}}\right|_{y_{\mathrm{m}}=y}\vec{u} =& \frac{1}{2}c_1\left(\frac{\partial g_{11}}{\partial y_{\mathrm{m}}}+\frac{v_2}{v_1}\cdot\frac{\partial g_{21}}{\partial y_{\mathrm{m}}}+\frac{v_3}{v_1}\cdot\frac{\partial g_{31}}{\partial y_{\mathrm{m}}}\right)\\ &+\frac{1}{2}c_2\left(\frac{v_1}{v_2}\cdot\frac{\partial g_{12}}{\partial y_{\mathrm{m}}}+\frac{\partial g_{22}}{\partial y_{\mathrm{m}}}+\frac{v_3}{v_2}\cdot\frac{\partial g_{32}}{\partial y_{\mathrm{m}}}\right)\\ &+\frac{1}{2}c_3\left(\frac{v_1}{v_3}\cdot\frac{\partial g_{13}}{\partial y_{\mathrm{m}}}+\frac{v_2}{v_3}\cdot\frac{\partial g_{23}}{\partial y_{\mathrm{m}}}+\frac{\partial g_{33}}{\partial y_{\mathrm{m}}}\right)\\ =& \frac{1}{2}c_1\frac{v_3}{v_1}\cdot\frac{\partial g_{31}}{\partial y_{\mathrm{m}}}\end{aligned} \tag{A-13}$$

where

$$
\begin{aligned}
\left.\frac{\partial \mathbf{G}_{\mathrm{m}}}{\partial y_{\mathrm{m}}}\right|_{y_{\mathrm{m}}=y} &= \left.\begin{pmatrix} \frac{\partial g_{11}}{\partial y_{\mathrm{m}}} & \frac{\partial g_{12}}{\partial y_{\mathrm{m}}} & \frac{\partial g_{13}}{\partial y_{\mathrm{m}}} \\ \frac{\partial g_{21}}{\partial y_{\mathrm{m}}} & \frac{\partial g_{22}}{\partial y_{\mathrm{m}}} & \frac{\partial g_{23}}{\partial y_{\mathrm{m}}} \\ \frac{\partial g_{31}}{\partial y_{\mathrm{m}}} & \frac{\partial g_{32}}{\partial y_{\mathrm{m}}} & \frac{\partial g_{33}}{\partial y_{\mathrm{m}}} \end{pmatrix}\right|_{y_{\mathrm{m}}=y} \\
&= \begin{pmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ \frac{a_1(d_E+v_E+\sigma_E)}{\left(ya_1+d_E+v_E+(1-y)\sigma_E)^2\right)}F_L - \frac{f_E\sigma_E(a_1+d_E+v_E)}{\left(ya_1+d_E+v_E+(1-y)\sigma_E\right)^2} & 0 & 0 \end{pmatrix} \qquad \text{(A-14)} \\
c_1 &= \frac{\lambda x E_0^*}{d_K}\left(p_{E\to L}F_L + p_{E\to K}f_E\right) \\
v_1 &= p_{E\to L}F_L + p_{E\to K}f_E \\
v_3 &= \rho(\mathbf{G}) = 1
\end{aligned}
$$

C Evolutionary stability

By examining the sign of the second derivative given that the candidate evolutionary consequence is in the interior of the phenotypic space (which is the square rectangle), we assess the evolutionary stability of x using the adaptive dynamics theory.

The second derivative with respect to x_{m} is given by:

$$
\left.\frac{\partial^2 R_{\mathrm{m}}(x_{\mathrm{m}}, y_{\mathrm{m}}; x, y)}{\partial x_{\mathrm{m}}^2}\right|_{x_{\mathrm{m}}=x=x^*, y_{\mathrm{m}}=y} = 0. \qquad \text{(A-15)}
$$

On the other hand, the candidate evolutionary consequence of y is located on the boundary of the phenotypic space. We here examine whether the convergence stability implies the evolutionary stability without taking the second derivative of the fitness function.

When $y = 0$ is convergence stable and the candidate evolutionary consequence of x has an interior solution x^*, the selection gradient is given by:

$$
\left.\frac{\partial R_{\mathrm{m}}(x_{\mathrm{m}}, y_{\mathrm{m}}; x, y)}{\partial y_{\mathrm{m}}}\right|_{x_{\mathrm{m}}=x, y_{\mathrm{m}}=y} \propto \alpha F_L - F_E < 0. \qquad \text{(A-16)}
$$

which always holds (i.e., never positive, implying that selection pushes the phenotype (preference for delayed emergence) towards zero). Substituting this equation into the Taylor expansion of the fitness function around

$y = 0 (0 < \delta << 1)$, we obtain

$$R_{\mathrm{m}}(x^*, \delta; x^*, 0) = R_{\mathrm{m}}(x^*, 0; x^*, 0) + \delta \left. \frac{\partial R_{\mathrm{m}}(x^*, y_{\mathrm{m}}; x^*, 0)}{\partial y_{\mathrm{m}}} \right|_{y_{\mathrm{m}}=0} \tag{A-17}$$

$$< R_{\mathrm{m}}(x^*, 0; x^*, 0). \tag{A-18}$$

Thus, $y^* = 0$ is evolutionarily stable (i.e., the strategies that no other rare mutant with small phenotypic differences can increase in frequency) because it attains a local maximum. When the singular point has an interior solution, the selection gradient is 0, i.e., the second term of the Taylor expansion vanishes. Thus it is necessary to take the second derivative to evaluate the sign of the third term. We can apply a similar argument to the case where $y^* = 1$, and find that $y^* = 1$ is also evolutionarily stable because it attains a local maximum.

D An unique equilibrium of the population dynamics

We can obtain the density ratio of L_0^* to E_0^* as follows:

$$\frac{L_0^*}{E_0^*}(x) = \frac{-d_A(b_0 + d_L)x + d_A(a_0 + d_E)(1 - x) + \sqrt{(d_A(b_0 + d_L)x + d_A(a_0 + d_E)(1 - x))^2 + 4rd_A a_0 b_0 x(1 - x)}}{2rb_0(1 - x)} \tag{A-19}$$

which is a monotone increasing function of x. Here, we will prove the uniqueness of the equilibrium of the population dynamics (Equation (1)). From the third line, we obtain

$$A^* = \frac{b_0}{d_A} L_0^* \tag{A-20}$$

From the third line, we obtain K^* analytically

$$K^* = \frac{1}{\lambda x} \left(-(a_0 + d_E) + r \frac{L_0^*}{E_0^*} \right) \tag{A-21}$$

From the fourth line and the fifth line, we obtain

$$L_1^* = \frac{1}{\sigma_L + d_L + v_L} \left(\frac{\lambda x y a_1}{y a_1 + (1 - y)\sigma_E + d_E + v_E} + \lambda(1 - x) \right) K^* E_0^* \tag{A-22}$$

From the sixth line, we obtain

$$E_0^* = \frac{d_K}{\frac{\lambda x f_E (1-y)\sigma_E}{y a_1 + (1-y)\sigma_E + d_E + v_E} + \frac{f_L \sigma_L}{\sigma_L + d_L + v_L} \left(\frac{\lambda x y a_1}{y a_1 + (1-y)\sigma_E + d_E + v_E} + \lambda(1 - x) \right)} \tag{A-23}$$

From this equation, we can obtain all the rest equilibrium L_0^*, A^*, E_1^*.

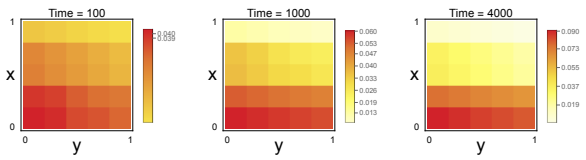
$$L_0^* = \frac{L_0^*}{E_0^*} E_0^* \tag{A-24}$$

$$A^* = \frac{b_0}{d_A} \frac{L_0^*}{E_0^*} E_0^* \tag{A-25}$$

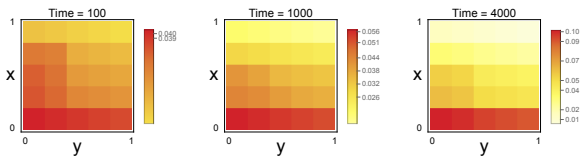
$$E_1^* = \frac{\lambda x}{y a_1 + (1-y)\sigma_E + d_E + v_E} K^* E_0^* \tag{A-26}$$

E Simulation

We conduct deterministic evolutionary simulations and then compare the results of exponential growth with those of logistic growth. First, we divide the parasitoid wasps population into 25 strains based on the evolutionary traits and construct a mathematical model that represents the population dynamics of these strains. Second, we numerically solve the population dynamics to determine which strain survives over an extended period. Finally, we compare the outcomes of evolutionary simulations of the case where the host has logistic growth with that of the case where the host has exponential growth. We show that our analytical results are robust against this modified assumption.

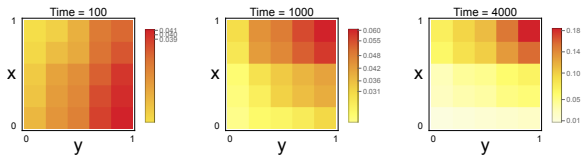


(a)Time variation of frequency of each strain

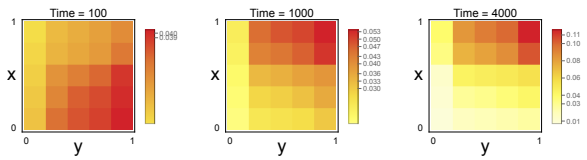


(b)Time variation of frequency of each strain

Figure 7: Evolutionary simulations: (a) The host density follows linear growth. (b) The host density follows logistic growth (carrying capacity $c = 100$). Both represent the case where wasps evolve to idiobiont. Wasps attack late juvenile hosts when $x = 0$, so the preference for delayed emergence y is neutral. ($\lambda = 1$, $r = 4$, $a_0 = 1.5$, $b_0 = 0.5$, $d_E = 1$, $d_L = 1$, $d_A = 0.5$, $d_K = 0.5$, $v_E = 0.5$, $v_L = 0.5$, $\sigma_E = 0.5$, $\sigma_L = 1$, $f_E = 0.5$,$f_L = 0.6$)



(c)Time variation of frequency of each strain



(d)Time variation of frequency of each strain

Figure 8: Evolutionary simulation:(a)The host density follows linear growth. (b)The host density follows logistic growth(The carrying capacity c=100). Both of them represent the case that wasps evolve to koinobiont($\lambda = 0.1$, $r = 0.5$, $a_0 = 0.16$, $b_0 = 0.2$, $d_E = 0.1$, $d_L = 0.1$, $d_A = 0.1$, $d_K = 0.1$, $v_E = 0.1$, $v_L = 0.1$, $\sigma_E = 1$, $\sigma_L = 1$, $f_E = 1$,$f_L = 2$).

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