J. theor. Biol. (2001) **212,** 141–153
doi:10.1006/jtbi.2001.2363, available online at http://www.idealibrary.com on IDEAL®

Host-limited Dynamics of Autoparasitoids

SEBASTIAN J. SCHREIBER*†, NICHOLAS J. MILLS‡ AND ANDREW P. GUTIERREZ‡

**Department of Mathematics, Western Washington University, Bellingham, Washington* 98225, *U.S.A. and ‡Department of Environmental Science, Policy & Management, University of California, Berkeley, CA* 94720, *U.S.A.*

(*Received on* 15 *September* 2000, *Accepted in revised form on* 25 *May* 2001)

Autoparasitoids, an important class of intraguild predators used in classical biological control, have a unique biology. Females develop as primary endoparasitoids of scale insects and whiteflies. Males develop at the expense of conspecific or heterospecific parasitoid prepupae. To evaluate the effect of autoparasitism on host suppression, system stability, and parasitoid coexistence, stage-structured differential equation models are developed and analysed. For a host–parasitoid system, autoparasitism stabilizes host–parasitoid oscillations generated by developmental delays of the parasitoid. In host–autoparasitoid–primary parasitoid systems, a distinction between obligate (i.e. parasitoid only attacks conspecifics for the production of males) and facultative (i.e. parasitoid attacks conspecifics and heterospecifics for the production of males) autoparasitism is drawn. Coexistence between an obligate autoparasitoid and primary parasitoid occurs if and only if the autoparasitoid can invade at lower host densities than the primary parasitoid, and the primary parasitoid can suppress the host to a lower equilibrium density than the autoparasitoid. When coexistence occurs, the primary parasitoid determines the host equilibrium abundance. Interactions between facultative autoparasitoids and primary parasitoids can lead to a priority effect, and, less likely, to coexistence. When coexistence occurs, the invasion of the facultative autoparasitoid into the host–primary parasitoid system raises the equilibrium density of the host. In either coexistence scenario, the invasion of an autoparasitoid can stabilize an unstable host–primary parasitoid system. The analysis concludes by showing that the introduction of an autoparasitoid to a host–primary parasitoid system can improve host suppression in the short-term despite possible long-term disruption.

Introduction

Intraguild predation, a special case of omnivory in which a predator feeds on another member of the same guild (intraguild prey) as well as on the primary prey, is common among both vertebrate and invertebrate predators in ecological communities (Polis *et al.*, 1989). In general, omnivory has been found to destabilize the dynamics of simple food chain models (Pimm & Lawton, 1978), although McCann & Hastings (1997) have used a non-equilibrium approach to show that omnivory can provide local stability. Holt & Polis (1997) have similarly shown that unstable dynamics result from the more specific case of intraguild predation. In this case, coexistence is promoted by competitive superiority of the intraguild prey in exploiting the primary prey, and intraguild predation of a sufficient

†Author to whom correspondence should be addressed. E-mail: sschreib@cc.wwu.edu

0022–5193/01/180141 + 13 $35.00/0

magnitude to offset the competitive inferiority of the intraguild predator. However, coexistence is also influenced by the productivity of the primary prey population, low productivity leading to exclusion of the intraguild predator through exploitative competition and high productivity leading to exclusion of the intraguild prey through apparent competition.

The importance of intraguild predation to problems of applied ecology, such as biological pest control, has barely been addressed despite the widespread occurrence of intraguild predation among the natural enemies of arthropod pests (Rosenheim *et al*., 1995). This is somewhat surprising, given the extensive theoretical literature applied to biological control (Mills & Getz, 1996). However, this likely results from a focus of biological control theory on host–parasitoid models in contrast to the more frequent observations of intraguild predation among predators.

A particularly interesting form of intraguild predation among parasitoids occurs in the wasp family Aphelinidae, which contains several genera of autoparasitoids (Walter, 1983; Hunter & Woolley, 2001). Among the insect parasitoids, autoparasitoids have a unique biology: males and females develop on different hosts. Females develop as a primary endoparasitoid of a homopteran host, for example a scale insect or whitefly. Males develop either as an endoparasitoid or ectoparasitoid of conspecific females [obligate autoparasitoid (Walter, 1983)] or at the expense of either sex of any other endoparasitoid that uses the same homopteran host [facultative autoparasitoid (Walter, 1983)]. This form of male production differs from primary parasitoids whose males and females develop in or on the same host. Although the ecological and evolutionary significance of this reproductive strategy has been explored in some detail (Godfray & Waage, 1990; Godfray, 1994; Hunter & Godfray, 1995; Hunter & Woolley, 2001), the population dynamics of autoparasitoid–host interactions has not been adequately addressed.

Homopterans are among the most widespread of insect pests and frequently have been the targets for classical biological control programs, in which parasitoids from the region of origin of a pest are introduced for its control in a newly invaded region (Greathead, 1986; Mills, 2000). Successful biological control programs provide some of the best examples of food chain interactions that can be reduced to just two protagonist species: the host and the parasitoid (May & Hassell, 1981; Murdoch, 1990; Murdoch & Briggs, 1996). Despite this, biological control efforts often involve multiple parasitoid introductions (Huffaker & Messenger, 1976). Whether this is a sound strategy is a contentious issue that has been addressed by many practitioners (Turnbull & Chant, 1961; Huffaker & Messenger, 1976; Ehler, 1990) and theorists (May & Hassell, 1981; Kakehashi *et al*., 1984; Godfray & Waage, 1991; Briggs, 1993; Mills & Gutierrez, 1996). The underlying message in this controversy is that the particularities of the host's and parasitoid's biology and behavior can lead to different answers. As many of the more abundant parasitoids of scales and whiteflies are autoparasitoids, it is important to understand the dynamics of this form of intraguild predation in the context of biological control systems.

Autoparasitism has previously been investigated in the context of density dependence in the sex ratios of parasitoids (Hassell *et al*., 1983) and through a prospective simulation model of the biological control of whiteflies in cotton (Mills & Gutierrez, 1996). Here we generalize and extend these previous studies to focus on the conditions that permit coexistence between a primary parasitoid and autoparasitoid, and how this influences host suppression and system stability using analytical stage-structured models.

The Model

Models of one or two parasitoid species competing for a common host are examined below. All the models are stage-structured ordinary differential equations. Similar models have been used to study predator–prey interactions with stage-structure (Hastings, 1983; McNair, 1987; Harrison, 1995) and forest–pest interactions with age-structure (Antonovsky *et al*., 1990). The models make the following assumptions about the biology of the parasitoids. The host and parasitoids have overlapping generations. In the absence of parasitoids, the host dynamics are given by the Logistic equation. The parasitoids have

two developmental stages: an immature stage with density I and an adult stage with density A. Adult parasitoids encounter and attack the host at a rate proportional to the host density. This assumption is justified if the parasitoids are host limited and not time or egg limited. After encountering a host, a female primary parasitoid lays a male or a female egg with fixed probabilities, $1-\theta$ and θ, respectively. On the other hand, a female autoparasitoid lays an egg of the appropriate sex into whatever host they encounter (Godfray & Waage, 1990). The host for female eggs are unparasitized hosts (here called the primary hosts) and the hosts for males (here called secondary hosts) are either conspecific female immatures or the immatures stages of other parasitoids in the system. Each parasitized host becomes an immature parasitoid that develops at a constant rate d to adulthood with an expected longevity of $1/m$.

One Parasitoid–One Host Systems

To understand the effect of obligate autoparasitism on host–parasitoid interactions, we compare obligate autoparasitism and primary parasitism in a single host–single parasitoid system (see Fig. 1). If H, I, and A denote the host density, immature parasitoid density, and adult female parasitoid density, respectively, then our

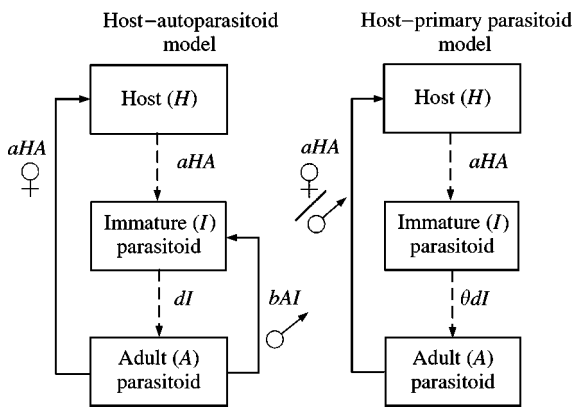

FIG. 1. Diagram illustrating the host–single parasitoid system. Dashed lines indicate the "flow of individuals": parasitized hosts become immature parasitoids that develop to become adult parasitoids. Solid lines indicate parasitoid attacks.

TABLE 1

Parameters and variables for the one host–one parasitoid system

H	Host density
I	Immature parasitoid density
A	Adult parasitoid density
r	Host's intrinsic rate of growth
K	Host's carrying capacity
a	Parasitoid attack rate on host
b	Autoparasitoid attack rate on immature parasitoids
d	Parasitoid developmental rate
m	Per-capita mortality rate of adult parasitoids
θ	Probability that a parasitoid lays a female egg on an encountered primary host

assumptions imply that the dynamics of the host–parasitoid system are given by

$$\begin{aligned}\frac{dH}{dt} &= rH\left(1-\frac{H}{K}\right)-aHA,\\ \frac{dI}{dt} &= aHA-dI-bIA,\\ \frac{dA}{dt} &= \theta dI-mA,\end{aligned} \tag{1}$$

where the parameters for the model are summarized in Table 1. For primary parasitoids, the parasitoid attack rate b on immature parasitoids is zero. Since autoparasitoids only produce females on primary hosts and always lay male eggs on secondary hosts (i.e. immature parasitoids), $\theta = 1$ and $b = a$ for autoparasitoids.

INVASION AND PERSISTENCE

When the invasion of a parasitoid is successful, the corresponding host–parasitoid system persists: there is a finite region of phase space (that is where the population vectors lie) bounded away from extinction in which every feasible population vector must ultimately lie. In Appendix A, we show that the success of a parasitoid invasion depends on the quantity $\theta aK - m$. When $\theta aK - m$ is positive, the parasitoid population grows exponentially when introduced at low densities into the host system. When $\theta aK - m$ is negative, the parasitoid population cannot invade and decays at an exponential rate.

THE HOST–PARASITOID EQUILIBRIUM

When a parasitoid is able to invade, there is a unique equilibrium that supports all populations. It is given by

$$H^* = \frac{Km(ad + br)}{bmr + a^2dK\theta},$$

$$I^* = \frac{m}{\theta d} A^*, \tag{2}$$

$$A^* = \frac{dr(\theta aK - m)}{bmr + a^2dK\theta}.$$

The stability of this equilibrium is determined by an inequality that is discussed in Appendix A. From this inequality, we derive two conclusions. First, the host–primary parasitoid system always exhibits the paradox of enrichment (Rosenzweig, 1971). Namely, enriching the system by increasing K always destabilizes the equilibrium at which the host and parasitoid coexist. Second, the equilibrium of a host–obligate autoparasitoid system (i.e. $a = b$ and $\theta = 1$) is always stable. Hence, obligate autoparasitism stabilizes host–parasitoid interactions.

In Fig. 2, we examine how the life-history traits of the host and parasitoid influence the stability of the host–parasitoid equilibrium. These bifurcation diagrams distinguish between three types of local behavior: unstable, in which a small perturbation from the equilibrium results in initially diverging oscillations and eventual periodic behavior; underdamped stability in which a small perturbation from the equilibrium results in exponentially decaying oscillations that return the system to the equilibrium; overdamped stability in which a small perturbation from the equilibrium is followed by an exponential decay back to the equilibrium. Figure 2 shows that hosts with high carrying capacities, parasitoids with high searching efficiencies, or parasitoids with long lifespans tend to destabilize host–parasitoid interactions. In the case of obligate autoparasitoids, the form of destabilization is weak as it involves a shift from overdamped to underdamped stability. On the other hand, Fig. 2 shows that hosts with a high intrinsic rate of increase stabilize host–parasitoid interactions.

Two Parasitoid–One Host Systems

Many autoparasitoids do not restrict their hyperparasitic activities to conspecifics. Consequently, when evaluating whether or not to release an autoparasitoid into a system, we are confronted with several questions. Under what conditions can an autoparasitoid invade a host–primary parasitoid system? If an autoparasitoid invades, will the two parasitoid species coexist or will the autoparasitoid displace the primary parasitoid? Does the establishment of an autoparasitoid lower the host equilibrium density? Will the establishment of an autoparasitoid preclude an invasion by a primary parasitoid that would suppress a host to lower density? Do invasions destabilize or stabilize the system? To address these questions, we consider a system consisting of a host, an autoparasitoid (A_1) and a primary parasitoid (A_2) (see Fig. 3). Under the assumptions outlined earlier, such a system is given by

$$\begin{aligned}
\frac{dH}{dt} &= rH\left(1 - \frac{H}{K}\right) - a_1A_1H - a_2A_2H,\\
\frac{dI_1}{dt} &= a_1A_1H - d_1I_1 - b_1A_1I_1,\\
\frac{dI_2}{dt} &= a_2A_2H - d_2I_2 - b_2A_1I_2,\\
\frac{dA_1}{dt} &= d_1I_1 - m_1A_1,\\
\frac{dA_2}{dt} &= \theta d_2I_2 - m_2A_2,
\end{aligned} \tag{3}$$

where the variables and parameters of the model are summarized in Table 2.

MUTUAL INVASIBILITY

Since biological control typically involves introducing small populations of natural enemies into a system, we use mutual invasibility as a criterion for coexistence. In our scenario, mutual invasibility means that a primary parasitoid can invade a host–autoparasitoid system and that the autoparasitoid can invade the host–primary

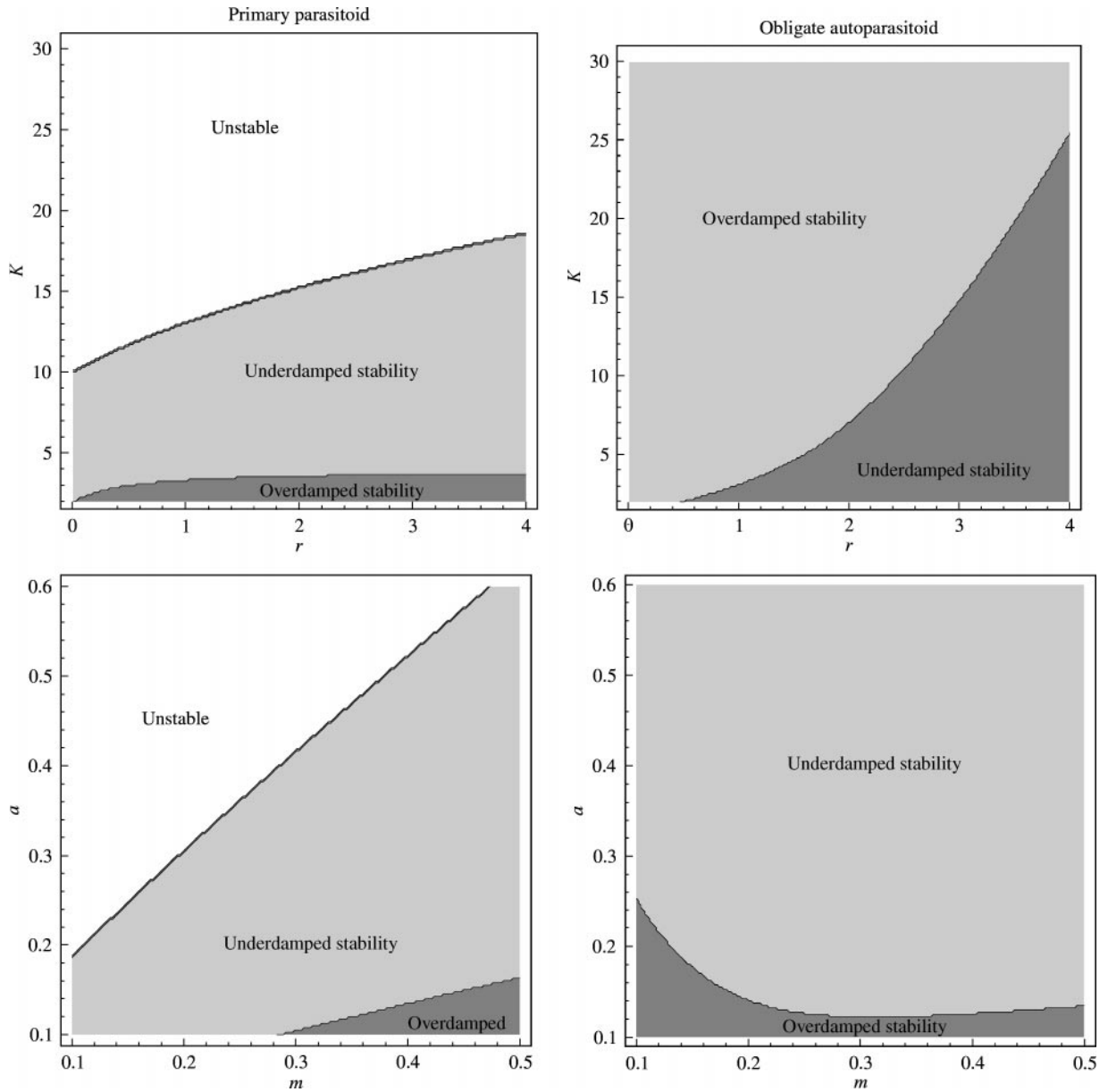

FIG. 2. Bifurcation diagrams for host–parasitoid systems. For the obligate autoparasitoid systems $a = b$ and $\theta = 1.0$, while for the primary parasitoid systems $b = 0$ and $\theta = 0.5$. In the top two diagrams, $a = m = d = 0.2$. In the bottom two diagrams, $d = m$, $r = 2$ and $K = 10$.

parasitoid system. Since testing for mutual invasibility only makes sense when each host–parasitoid subsystem is viable, we assume throughout this section that the carrying capacity of the host is sufficiently large to insure that each parasitoid population can invade the isolated host system (i.e. $a_1K - m_1 > 0$ and $\theta a_2K - m_2 > 0$). First, suppose we release an autoparasitoid into a host–primary parasitoid system that is at equilibrium. In Appendix B, we show that the success of an autoparasitoid's invasion attempt depends on the quantity, $a_1H^* - m_1$ where H^* is the equilibrium density of the host in the host–primary parasitoid system. When $a_1H^* - m_1$ is positive, the autoparasitoid can invade the host–primary parasitoid equilibrium. When $a_1H^* - m_1$ is negative, all invasion attempts fail. Hence, an autoparasitoid can invade a host–primary parasitoid system only if it has a positive growth rate at lower host densities than the primary

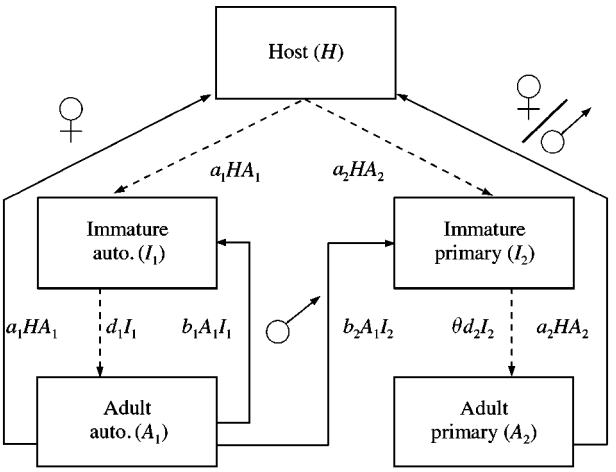

FIG. 3. Diagram illustrating the host–autoparasitoid–primary parasitoid system. Dashed and solid lines are as in Fig. 1.

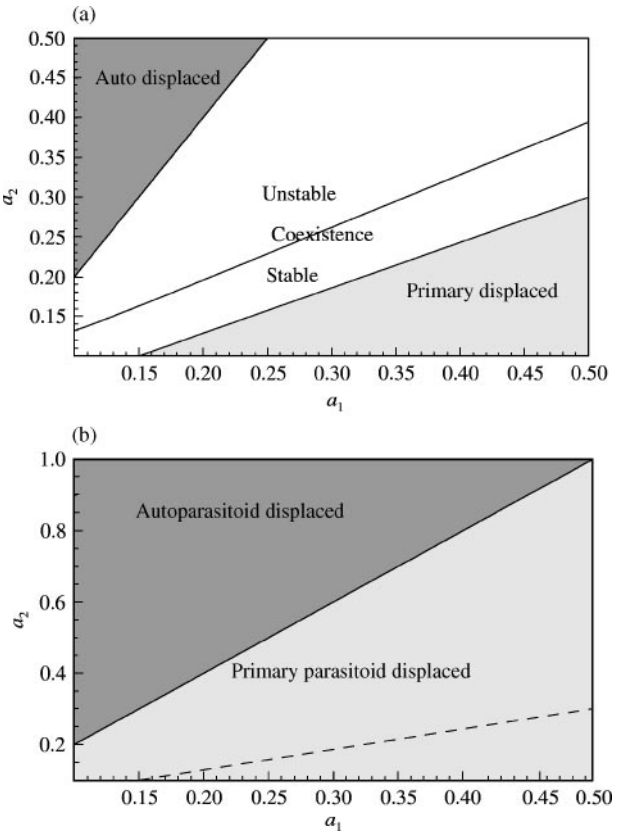

FIG. 4. Regions of coexistence and displacement in a_1-a_2 parameter space. In both diagrams, $r = 0.5$, $m_1 = m_2 = d_1 = d_2 = 0.2$, $\theta = 0.5$, and $K = 20$. In (a), the autoparasitoid is assumed to be obligate with $a_1 = b_1$ and $b_2 = 0$. In (b), the autoparasitoid is assumed to be facultative with $a_1 = b_1 = b_2$. The dashed line corresponds to where the host equilibrium abundance is equivalent for the host–autoparasitoid system and the host–primary parasitoid system.

parasitoid. On the other hand, suppose we release a primary parasitoid into a host–autoparasitoid system that is at equilibrium. The success of its invasion attempt depends on the quantity

$$\theta a_2 H^* - m_2 - \frac{b_2}{d_2} m_2 A_1^*, \tag{4}$$

where H^* and A_1^* are the equilibrium densities of the host and autoparasitoid, respectively, in the host–autoparasitoid subsystem. When eqn (4) is positive, the parasitoid can invade. When eqn (4) is negative, it cannot. One implication of eqn (4) is that a primary parasitoid can invade only if the autoparasitoid suppresses the host less effectively than it can.

TABLE 2

Parameters and variables for the one host–two parasitoid system. The subscript i is equal to 1 or 2 where $i = 1$ refers to the autoparasitoid and $i = 2$ refers to the primary parasitoid

H	Host density
I_i	Immature density of parasitoid i
A_i	Adult density of parasitoid i
r	Host's intrinsic rate of growth
K	Host's carrying capacity
a_i	Parasitoid i's attack rate on host
b_i	Autoparasitoid's attack rate on immatures of parasitoid i
d_i	Parasitoid i's developmental rate
m_i	Adult per-capita mortality rate of parasitoid i
θ	Probability that a primary parasitoid lays female egg on an encountered host

To understand the further implications of these invasion criteria, we consider separately the cases of an obligate autoparasitoid and a facultative parasitoid. When the autoparasitoid is obligate (i.e. $b_2 = 0$ and $a_1 = a_2 > 0$), the invasion criteria imply that the obligate autoparasitoid and primary parasitoid coexist if and only if the autoparasitoid can invade at lower hosts densities than the primary parasitoid, and the primary parasitoid can suppress the host to a lower equilibrium density than the obligate autoparasitoid.

We illustrate the regions of displacement and coexistence in Fig. 4(a). The white region of coexistence in Fig. 4(a) is divided into two subregions. The lower subregion corresponds to parameter values where all species coexist about a stable equilibrium, and the higher subregion corresponds to coexistence about a non-equilibrium attractor. Since the host–primary parasitoid systems corresponding to Fig. 4(a) are stable only if $a_2 < 0.12$, this figure implies that the invasion of an obligate autoparasitoid can stabilize an unstable host–primary parasitoid system.

On the other hand, if the autoparasitoid is facultative (i.e. $a_1 = b_1 = b_2 > 0$), then coexistence becomes less likely as illustrated in Figs. 4(b) and 5. For instance, if the developmental times of the autoparasitoid and primary parasitoid are the same, then we show in Appendix B that coexistence is impossible: one parasitoid species always displaces the other. There is a simple rule underlying this competitive exclusion principle, namely, that the parasitoid species that can invade at a lower host density dominates. We illustrate this competitive exclusion principle in Fig. 4(b). The dashed line in this figure corresponds to the parameter values at which the primary parasitoid and autoparasitoid suppress the host to the same equilibrium density. For parameter values below this dashed line, the autoparasitoid suppresses the host to lower equilibrium densities and for parameter values above this dashed line the primary parasitoid suppresses the host to lower equilibrium densities. As Fig. 4(b) illustrates, there is a broad range of parameter values at which facultative autoparasitoid displaces the primary parasitoid and results in a higher equilibrium abundance of the host. Finally, if the facultative autoparasitoid and primary parasitoid have different developmental rates as well as attack rates, then coexistence can occur if the primary parasitoid has a faster development rate than the autoparasitoid, or a priority effect (i.e. the species that establishes itself first excludes the other) can occur if the autoparasitoid has a faster developmental rate than the primary parasitoid as shown in Fig. 5. This figure also implies that the introduction of a facultative autoparasitoid can stabilize an unstable host–primary parasitoid system.

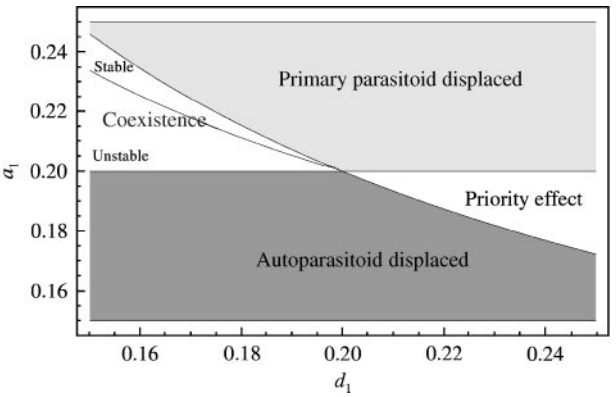

FIG. 5. Regions of coexistence, displacement, and priority effect in d_1-a_1 parameter space with $r = 0.5$, $m_1 = m_2 = d_2 = 0.2$, $\theta = 0.5$, $K = 30$, $b_1 = b_2 = a_1$, and $a_2 = 0.4$.

EQUILIBRIUM HOST DENSITY

When mutual invasibility occurs, the equilibrium host density equals

$$H^{**} = \frac{m_1 m_2 (b_2 d_1 - b_1 d_2)}{a_1 b_2 d_1 m_2 - a_2 b_1 d_2 m_1 \theta}. \qquad (5)$$

To evaluate how parasitoid coexistence influences host suppression, we observe that H^{**} is an increasing function of $b_2 d_1 / b_1 d_2$. Therefore, host suppression (in an equilibrium sense) is maximized by coexisting auto and primary parasitoids when the autoparasitoid is obligate (i.e. $b_2 = 0$). In this circumstance, the host equilibrium density is determined by the primary parasitoid and equals $m_2/\theta a_2$. On the other hand, a facultative autoparasitoid (i.e. $b_2 > 0$) that coexists with a primary parasitoid results in the host density at equilibrium increasing whenever the autoparasitoid invades the host–primary parasitoid system. However, primary parasitoids with faster developemental rates than autoparasitoids can marginalize this disruptive effect.

SHORT-TERM VS. LONG-TERM TRENDS

Thus far, we have only examined the limiting (long-term) dynamics of the host and not considered the transient (short-term) dynamics. Since what is often observed and documented in ecological systems is short-term behavior, it is important to ascertain when these dynamical trends are or are not in agreement (Hastings &

Higgins, 1994; Schreiber, 2001). To understand the short-term behavior of eqn (3) when a host–parasitoid subsystem is at a stable equilibrium and the other parasitoid is introduced, it is sufficient to examine the population eigenvector corresponding to exponential growth. Each entry of this vector indicates the rate of increase or decrease for the various populations of the system in response to an invasion. For example, if the entry corresponding to the host population is negative, then an invasion leads to an exponential decrease (in the short-term) of the host population at a rate determined by the magnitude of the corresponding eigenvalue. It is important to note that the magnitude of this effect is always bounded below by a quantity that is *independent* of the size of the introduced population. Consider the case of an autoparasitoid (I_1, H_1), invading a stable equilibrium of a host–primary parasitoid system. It is possible to show that the population vector, $(H, I_1, I_2, A_1, A_2)^\top$, associated with this invasion has the sign pattern

$$\begin{pmatrix} - \\ + \\ - \\ + \\ - \end{pmatrix}$$

whenever an autoparasitoid's attack rate on heterospecific hosts is small (see Appendix B). Hence, following the introduction of an autoparasitoid, host and primary parasitoid (adult and immature) populations decline as the autoparasitoid's population increases despite the fact that in the long-term the host population may increase.

Discussion

We have shown that developmental delays in primary parasitoids result in host–parasitoid oscillations and the paradox of enrichment (Rosenzweig, 1971). Similar destabilizing effects of stage-structure occur in delay-differential equation models of host–parasitoid interactions (Murdoch *et al.*, 1987, 1992) and predator–prey interactions (van den Bosch & Gabriel, 1997). Our models show that obligate autoparasitism stabilizes the population oscillations generated by parasitoid stage-structure and that this stabilization occurs even when the host is not resource limited. Since autoparasitism can be viewed as a form of cannibalism (i.e. immature males cannibalize immature females), this conclusion reinforces the view that cannibalism by predators can stabilize both externally generated (consumer-resource) as well as internally generated (age-structure) fluctuations in predator–prey systems (Kohlmeier & Ebenhöh, 1995; van den Bosch & Gabriel, 1997).

The multiple parasitoid species model predicts that primary parasitoids and autoparasitoids coexist only if the primary parasitoid suppresses the host to a lower equilibrium density than that of the autoparasitoid. Hence, when coexistence is possible, the introduction of a facultative autoparasitoid raises the equilibrium host density, while the introduction of an obligate autoparasitoid leaves the host equilibrium density unchanged. These predictions are consistent with the work of Polis *et al.* (1989), Polis & Holt (1992) and Holt & Polis (1997) on intraguild predation and Mills & Gutierrez's (1996) simulation study of autoparasitism, but run contrary to the predictions of May & Hassell (1981). The main reason for this discrepancy is that in the May and Hassell model aggregation acts independently on the two parasitoid species. Consequently, coexistence in the May and Hassell model occurs when aggregation limits population growth more than autoparasitism. Under these conditions, the host equilibrium density when both parasitoids are in the system can be lower than the host equilibrium density when only a single parasitoid is in the system.

For facultative autoparasitoids and primary parasitoids with comparable developmental rates, our analysis asserts that coexistence is impossible: the parasitoid that can invade at lower host densities dominates. This simple rule is similar to another simple rule, the R^* rule, that concerns exploitative competition between predators for a common resource: the predator that suppresses the resource to a lower equilibrium density dominates (Tilman, 1990; Holt *et al.*, 1994). However, since a facultative autoparasitoid always suppresses the host to an equilibrium density that is greater than the host density

at which it can invade, the invasion rule and R^* rule are not equivalent. When the developmental period of the facultative autoparasitoid is longer than that of the primary parasitoid, the model predicts that coexistence is possible. Alternatively, when the developmental period of the facultative autoparasitoid is shorter than that of the primary parasitoid, only displacement or a priority effect is possible. Currently, there exists very little data about the developmental rates of facultative autoparasitoids. One notable exception is the work of Hunter & Kelley (1998). They found that the developmental period of the immature stages of the facultative autoparasitoid *Encarsia transvena* is a little shorter at 13.95 days than that of the primary parasitoid *Eretmocerus eremicus* at 17.13 days.

Despite leading to possibly higher host equilibrium abundance, the invasion of an autoparasitoid into a host–primary parasitoid system can stabilize the systems dynamics. Hence, the introduction of an obligate autoparasitoid into a host–primary parasitoid system may benefit biological control efforts by stabilizing the system without raising the host equilibrium abundance. This result is consistent with the prediction of Mills & Gutierrez (1996), and juxtaposes the observation of Holt & Polis (1997) that intraguild predation can lead to instability, even when all pairwise interactions are inherently stable. This juxtaposition stems from the fact that the intraguild predators considered by Holt & Polis (1997) exhibit no self-limiting dynamics.

Rosenheim *et al.* (1995) in a recent survey of intraguild predation among biological control agents reviewed the literature on autoparasitism and its effect on host suppression. The survey was inconclusive. Some studies indicated that the presence of an autoparasitoid delayed the establishment of a primary parasitoid and reduced its efficiency (Nguyen *et al.*, 1983; Thomspon *et al.*, 1987). In other studies (Heinz & Nelson, 1996; Bogran & Heinz, 2000) autoparasitism appeared to enhance biological control. While there are many factors that explain this discrepancy, our analysis suggests that two factors may be responsible for the autoparasitoids stabilizing the system's dynamics, and the difference between short- and long-term trends.

The predictions from the models should be viewed with some caution as they rely on several simplifying features including exponential distribution of parasitoid maturation times, host-limited parasitoids, and the absence of parasitoid mating dynamics. The exponential distribution of parasitoid maturation times follows from the use of coupled ordinary differential equations to model the stages of the parasitoid (MacDonald, 1978). An alternative approach is to use delay-differential equations to model constant maturation times. Unfortunately, a direct comparison of our host–primary parasitoid model to previously studied delay-differential equation models of host–primary parasitoid interactions (Murdoch *et al.*, 1987, 1992; Briggs, 1993) is impossible as the stabilizing mechanisms for these models are different: host density dependence in our model, and an invulnerable host stage in the delay-differential equation models. However, delay-differential equation models of predator–prey interactions with cannibalistic and stage-structured predators show that cannibalism can suppress or even completely eliminate internally generated population oscillations (van den Bosch & Gabriel, 1997). Therefore, even with constant parasitoid maturation rates, autoparasitism is likely to be stabilizing, but possibly not to the extent suggested by our model. It is difficult to evaluate how parasitoid egg-limitation and including mating dynamics would effect our conclusions. Both additions would increase the complexity of the model significantly: including egg-limitation changes the functional response of the parasitoid and results in a density-dependent sex-allocation strategy for autoparasitoids (Hunter & Godfray, 1995), while including mating dynamics requires extra state variables. However as these additional biological complexities play important roles in these systems, it would be useful to understand their implications in any future analysis of host–autoparasitoid dynamics.

In conclusion, the remarkable biology of autoparasitoids may account for their success in previous biological control programs (Huffaker & Messenger, 1976; Penagos & Williams, 1995). However, careful evaluation is needed before introducing an autoparasitoid into a system where a pest population is already suppressed below an economic threshold by a primary parasitoid.

This research was supported in part by the National Science Foundation Grant DMS-0077986 to SJS.

REFERENCES

ANTONOVSKY, M. YA., FLEMING, R. A., KUZNETSOV, YU. A. & CLARK, W. C. (1990). Forest-pest interaction dynamics: the simplest mathematical models. *Theor. Popul. Biol.* **37,** 343–367.

BRIGGS, C. J. (1993). Competition among parasitoid species on a stage-structured host and its effect on host suppression. *Am. Nat.* **141,** 372–397.

BOGRAN, C. E. & HEINZ, K. M. (2000). Interspecific interactions among parasitoids in agroecosystems. XXI International Congress of Entomology, Abstracts, Book 1, p. 208.

EHLER, L. E. (1990). Introduction strategies in biological control of insects. In: *Critical Issues in Biological Control* (MacKauer, M., Ehler, L. E. & Rohland, J., eds), pp. 111–134. Andover: Intercept.

GODFRAY, H. C. J. (1994). *Parasitoids*: *Behavioral and Evolutionary Ecology*. Princeton, NJ: Princeton University Press.

GODFRAY, H. C. J. & WAAGE, J. K. (1990). The evolution of highly skewed sex ratios in aphelinid wasps. *Am. Nat.* **136,** 715–721.

GODFRAY, H. C. J. & WAAGE, J. K. (1991). Predictive modelling in biological control: the mango mealy bug (*Rastrococcus invadens*) and its parasitoids. *J. Appl. Ecol.* **28,** 434–453.

GREATHEAD, D. (1986). Parasitoids in classical biological control. In: *Insect Parasitoids* (Waage, J. & Greathead, D., eds), pp. 289–318. London: Academic Press.

HARRISON, G. W. (1995). Comparing predator–prey models to Luckinbill's experiment with Didinium and Paramecium. *Ecology* **76,** 357–374.

HASSELL, M. P., WAAGE, J. K. & MAY, R. M. (1983). Variable parasitoid sex ratios and their effect on host–parasitoid dynamics. *J. Anim. Ecol.* **52,** 889–904.

HASTINGS, A. (1983). Age-dependent predation is not a simple process. I. Continuous time models. *Theor. Popul. Biol.* **23,** 347–362.

HASTINGS, A. & HIGGINS, K. (1994). Persistence of transients in spatially structured ecological models. *Science* **263,** 1133–1136.

HEINZ, K. M. & NELSON, J. M. (1996). Interspecific interactions among natural enemies of *Bemisia* in an inundative biological control program. *Biol. Control* **6,** 384–393.

HOLT, R. D. & POLIS, G. A. (1997). A theoretical framework for intraguild predation. *Am. Nat.* **149,** 745–764.

HOLT, R. D., GROVER, J. & TILMAN, D. (1994). Simple rules for interspecific dominance in systems with exploitative and apparent competition. *Am. Nat.* **144,** 741–771.

HUFFAKER, C. B. & MESSENGER, P. S. (1976). *Theory and Practice of Biological Control.* New York: Academic Press.

HUNTER, M. S. & GODFRAY, H. C. J. (1995). Ecological determinants of sex allocation in an autoparasitoid wasp. *J. Anim. Ecol.* **64,** 95–106.

HUNTER, M. S. & KELLY, S. E. (1998). Hyperparasitism by an exotic autoparasitoid: secondary host selection and the window of vulnerability of conspecific and native heterospecific hosts. *Entomol. Exp. Appl.* **89,** 249–259.

HUNTER, M. S. & WOOLLEY, J. B. (2001). Evolution and behavioral ecology of heteronomous aphelinid parasitoids. *Annu. Rev. Entomol.* **46,** 251–290.

KAKEHASHI, M., SUZUKI, Y. & IWASA, Y. (1984). Niche overlap of parasitoids in host–parasitoid systems: its consequences to single versus multiple introduction controversy in biological control. *J. Appl. Ecol.* **21,** 115–131.

KOHLMEIER, C. & EBENHÖH, W. (1995). The stabilizing role of cannibalism in a predator–prey system. *Bull. Math. Biol.* **57,** 401–411.

MACDONALD, N. (1978). *Time Lags in Biological Models.* Berlin, New York: Springer-Verlag.

MAY, R. M. & HASSELL, M. P. (1981). The dynamics of multiparasitoid–host interactions. *Am. Nat.* **117,** 234–261.

MCCANN, K. & HASTINGS, A. (1997). Re-evaluating the omnivory-stability relationship in food webs. *Proc. R. Soc. London, Ser. B: Biol. Sci.* **264,** 1249–1254.

MCNAIR, J. N. (1987). A reconciliation of simple and complex models of age-dependent predation. *Theor. Popul. Biol.* **32,** 383–392.

MILLS, N. J. (2000). Biological control: the need for realistic models and experimental approaches to parasitoid introductions. In: *Parasitoid Population Biology* (Hochberg, M. E. & Ives, A. R., eds), pp. 217–234. Princeton, NJ: Princeton University Press.

MILLS, N. J. & GETZ, W. M. (1996). Modelling the biological control of insect pests: a review of host–parasitoid models. *Ecol. Model.* **92,** 121–143.

MILLS, N. J. & GUTIERREZ, A. P. (1996). Prospective modelling in biological control: an analysis of the dynamics of heteronomous hyperparasitism in a cotton–whitefly–parasitoid system. *J. Appl. Ecol.* **33,** 1379–1394.

MURDOCH, W. M. (1990). The relevance of pest-enemy models to biological control. In: *Critical Issues in Biological Control* (Mackauer, M., Ehler, L. E. & Roland, J., eds), pp. 1–24. Andover: Intercept.

MURDOCH, W. M. & BRIGGS, C. J. (1996). Theory for biological control: recent developments. *Ecology* **77,** 2001–2013.

MURDOCH, W., NISBET, R. M., GURNEY, W. S. C. & REEVE, J. D. (1987). An invulnerable age class and stability in delay-differential equation parasitoid–host models. *Am. Nat.* **129,** 263–282.

MURDOCH, W., NISBET, R. M., LUCK, R. F.,GODFRAY, H. C. J. & GURNEY, W. S. C. (1992). Size-selective sex-allocation and host feeding in a parasitoid-host model. *J. Anim. Ecol.* **61,** 533–541.

NGUYEN, R., BRAZZEL, J. R. & POUCHER, C. (1983). Population density of the citrus blackfly, *Aleurocanthus woglumi* Ashby (Homoptera: Aleyrodidae), and its parasites in urban Florida in 1979–1981. *Environ. Entomol.* **12,** 878–884.

PENAGOS, D. I. & WILLIAMS, T. (1995). Important factors in the biology of heteronomous hyperparasitoids (Hym.: Aphelinidae): agents for the biological control of whiteflies and scale insects. *Acta Zool. Mexicana* **66,** 31–57.

PIMM, S. L. & LAWTON, J. H. (1978). On feeding on more than one trophic level. *Nature* **275,** 542–544.

POLIS, G. & HOLT, R. (1992). Intraguild predation: the dynamics of complex trophic interactions. *Trends Ecol. Evol.* **7,** 151–154.

POLIS, G., MEYERS, C. A. & HOLT, R. D. (1989). The ecology and evolution of intraguild predation: potential competitors that eat each other. *Annu. Rev. Ecol. Syst.* **20,** 297–330.

ROSENHEIM, J. A., KAYA, H. K., EHLER, L. E., MAROIS, J. J. & JAFFEE, B. A. (1995). Intraguild predation among biological-control agents: theory and evidence. *Biol. Control* **5,** 303–335.

ROSENZWEIG, M. L. (1971). Paradox of enrichment: destabilization of exploitation ecosystems in ecological time. *Science* **171,** 385–387.

SCHREIBER, S. J. (2001). Chaos and sudden population disappearances in simple ecological models. *J. Math. Biol.* **42,** 239–260.

THOMPSON, C. G., CORNELL, J. A. & SAILER, R. I. (1987). Interactions of parasites and a hyperparasite in biological control of citrus blackfly, *Aleurocanthus woglumi* (Homoptera: Aleyrodidae), in Florida. *Environ. Entomol.* **16,** 140–144.

TILMAN, D. (1990). Mechanisms of plant competition for nutrients: the elements of a predictive theory of competition. In: *Perspectives on Plant Competition* (Grace, J. B. & Tilman, D., eds), pp. 117–142. San Diego: Academic Press.

TURNBULL, A. L. & CHANT, D. A. (1961). The practice and theory of biological control of insects in Canada. *Can. J. Zool.* **39,** 697–753.

VAN DEN BOSCH, F. & GABRIEL, W. (1997). Cannibalism in an age-structured predator-prey system. Bull. *Math. Biol.* **59,** 551–567.

WALTER, G. H. (1983). 'Divergent male ontogenies' in Aphelinidae (Hymenoptera: Chalcidoidea): a simplified classification and a suggested evolutionary sequence. *Biol. J. Linn. Soc.* **19,** 63–82.

APPENDIX A

Invasion and Persistence

To determine whether a parasitoid can invade in eqn (1), it is sufficient to evaluate the eigenvalues of the Jacobian matrix evaluated at the equilibrium $(K, 0, 0)$:

$$\begin{pmatrix} -r & 0 & -aK \\ 0 & -d & aK \\ 0 & d\theta & -m \end{pmatrix}.$$

The eigenvalues of this matrix are given by $-r$ and the eigenvalues of the matrix

$$M = \begin{pmatrix} -d & aK \\ \theta d & -m \end{pmatrix}.$$

For the parasitoid to invade (and, thus, persist) it is necessary and sufficient that this matrix has an eigenvalue with positive real part. Since the trace of M is negative, this occurs if and only if the determinant of M is negative. In other words, $\theta aK - m > 0$.

Stability

To check the stability of this equilibrium, we examine the characteristic polynomial of the variation matrix evaluated at (H^*, I^*, A^*). It is of the form, $c(x) = x^3 + a_3x^2 + a_2x + a_1$. The Routh–Hurwitz stability criterion asserts that (H^*, I^*, A^*) is locally stable if and only if $a_1 > 0$, $a_3 > 0$ and $a_1a_2 - a_3 > 0$. The quantities a_1 and a_3 are given by

$$a_1 = \frac{bmr(m+r) + a^2dK(d+m)\theta + adr(m+bK\theta)}{bmr + a^2dK\theta}$$

and

$$a_3 = \frac{dmr(ad + br)(-m + aK\theta)}{bmr + a^2dK\theta}.$$

Since $\theta aK - m > 0$ whenever the equilibrium is feasible, a_1 and a_3 are positive whenever the equilibrium is feasible. Hence the equilibrium is linearly stable if and only if $a_1a_2 - a_3 > 0$. In general, the expression for $a_1a_2 - a_3$ is quite complex. However, from it two conclusions can be drawn. First, when $b = 0$,

$$\frac{\partial}{\partial K} a_1a_2 - a_3 =$$

$$-\left(\frac{mr(ad^2K\theta + m^2(2r + aK\theta) + dm(2r + 3aK\theta))}{a^2K^3\theta^2}\right)$$

is always negative, and

$$\lim_{K\to\infty} a_1a_2 - a_3 = -dmr.$$

Hence, the equilibrium at which the host and primary parasitoid coexist is unstable for K sufficiently large and otherwise stable. Second, when $a = b$, $a_1a_2 - a_3$ reduces to a quantity which is always positive. Hence, the host–autoparasitoid equilibrium is stable.

APPENDIX B

Invasions

To determine whether the primary parasitoid can invade, we need to evaluate the Jacobian

matrix of eqn (3) at the equilibrium $(H, I_1, A_1, I_2, A_2) = (H^*, I_1^*, A_1^*, 0, 0)$ as follows:

$$\begin{pmatrix} r - \dfrac{2rH^*}{K} - a_1A_1^* & 0 & -a_1H^* & 0 & -a_2H^* \\ a_1A_1^* & -b_1A_1^* - d_1 & a_1H^* - b_1I_1 & 0 & 0 \\ 0 & d_1 & -m_1 & 0 & 0 \\ 0 & 0 & 0 & -b_2A_1^* - d_2 & a_2H^* \\ 0 & 0 & 0 & d_2\theta & -m_2 \end{pmatrix}.$$

The primary parasitoid can invade if the lower right 2×2 minor

$$\begin{pmatrix} -b_2A_1^* - d_2 & a_2H^* \\ d_2\theta & -m_2 \end{pmatrix}$$

of this Jacobian has an eigenvalue with positive real part. Since the trace of this minor is negative, this can only occur if its determinant is negative. Namely,

$$d_2a_2\theta H^* - d_2m_2 - m_2b_2A_1^* > 0.$$

Similarly, the autoparasitoid can invade the equilibrium (H^*, A_2^*, P_2^*) of the host–primary parasitoid system if and only if the determinant of the 2×2 minor

$$\begin{pmatrix} -d_1 & a_1H^* \\ d_1 & -m_1 \end{pmatrix}$$

of the Jacobian is negative. Namely, $a_1H^* - m_1 > 0$.

To show that mutual invasibility is atypical for a facultative autoparasitoid and primary parasitoid, some algebraic manipulations of the invasion criteria show that mutual invasion occurs when $b_1 = b_2 = a_1$ if and only if

$$a_2 < \frac{a_1m_2}{m_1\theta}$$

and

$$a_2 > \frac{m_2((d_2 - d_1)m_1r + a_1d_1K(d_2 + r))}{d_2Km_1(d_1 + r)\theta}.$$

When the developmental times of both parasitoids are the same (i.e. $d_1 = d_2$), these inequalities reduce to $a_2 < a_1m_2/m_1\theta$ and $a_2 > a_1m_2/m_1\theta$. Hence, mutual invasion is impossible when $a_1 = b_1 = b_2$ and $d_1 = d_2$.

Short-term Behavior

To prove our assertion concerning the possible discrepancy between short- and long-term behavior, we introduce the following change of variables, $Z_1 = A_1 + I_1$ and $Z_2 = A_2 + \theta I_2$, and begin by assuming that $b_2 = 0$. In the H, Z_1, Z_2, A_1, A_2 coordinate system with $b_2 = 0$, eqns (3) become

$$\begin{aligned} \frac{dH}{dt} &= rH\left(1 - \frac{H}{K}\right) - a_1A_1H - a_2A_2H, \\ \frac{dZ_1}{dt} &= a_1A_1H - m_1A_1 - b_1A_1(Z_1 - A_1), \\ \frac{dZ_2}{dt} &= \theta a_2A_2H - m_2A_2, \\ \frac{dA_1}{dt} &= d_1(Z_1 - A_1) - m_1A_1, \\ \frac{dA_2}{dt} &= d_2(Z_2 - A_2) - m_2A_2. \end{aligned} \tag{A.1}$$

Under the assumption that an autoparasitoid can invade the host–primary parasitoid equilibrium, the variation matrix for eqn (A.1) at the host–primary parasitoid equilibrium, is of the form

$$M = \begin{pmatrix} - & 0 & 0 & - & - \\ 0 & 0 & 0 & + & 0 \\ + & 0 & 0 & 0 & 0 \\ 0 & + & 0 & - & 0 \\ 0 & 0 & + & 0 & - \end{pmatrix},$$

where $+$ denotes a positive term and $-$ denotes a negative term. Since the autoparasitoid can invade, M has a positive eigenvalue $\lambda > 0$ with an eigenvector $v = (v_1, v_2, v_3, v_4, v_5)$ that satisfies $v_2 > 0$ and $v_4 > 0$. To show that $v_1 < 0$, we argue by contradiction. Suppose, on the contrary, that $v_1 \geqslant 0$. Since $\lambda > 0$ and $Mv = \lambda v$, the sign pattern of the third row of M implies that $v_3 \geqslant 0$. Since $v_1 \geqslant 0$, $v_4 \geqslant 0$, $\lambda > 0$, and $Mv = \lambda v$, the sign pattern of the first row M implies that $v_5 < 0$. Since $v_3 \geqslant 0$, $v_5 < 0$, $\lambda > 0$, and $Mv = \lambda v$, the sign pattern of the fifth row of M implies that $v_5 > 0$ which is impossible. Hence, we have shown that $v_1 < 0$, and v has the following sign pattern:

$$\begin{pmatrix} - \\ + \\ - \\ + \\ - \end{pmatrix}.$$