Host-parasitoid interactions

Key concepts

- The hierarchy of natural enemies
- The parasitoid life-cycle
- Host defenses
- The Nicholson-Bailey model
- Stabilizing mechanisms

Parasitoids: their extent and utility

In recent readings, we've been looking at a range of organisms that can broadly be defined as *natural enemies*. We've looked in detail at predators and microparasites, and along the way we acknowledged that there are also large parasites like ticks and worms (which we often refer to as macroparasites). A commonality is that when these organisms interact with their prey or host, it is a +/- interaction, where there is a fitness gain to the predator or parasite and a fitness loss incurred by the prey or host. One way to consider what these natural enemy interactions have in common (and how they differ), is to jointly consider the relative body size of the natural enemy (relative to the partner in its antagonistic interaction) and the number of enemies per "host" (where here, we also consider prey as a host to its predator). This hierarchical concept also allows us to introduce a new natural enemy, the *parasitoid* (Fig. 2).

Parasitoids are insects that lay their eggs in or on a host. The emerging larvae feed on the host eventually killing it. Far from being an unusual strategy among insects, it is estimated that parasitoids represent around 10% of insect species (mostly *Hymenoptera* wasps and *Diptera* flies). Given that it has been estimated that 80% of the world's species are insects, then parasitoids are in fact very common. As with all antogonistic (+/-) interactions, we will aim to understand

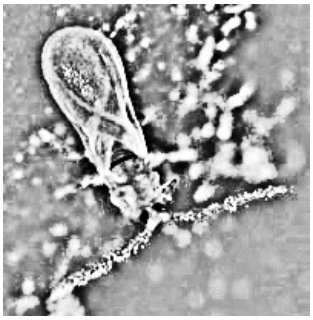

Figure 1: California red scale (*Aonidiella aurantii*) is an economically important pest of citrus crops. Control of this pest is partly through the release of parasitoids.

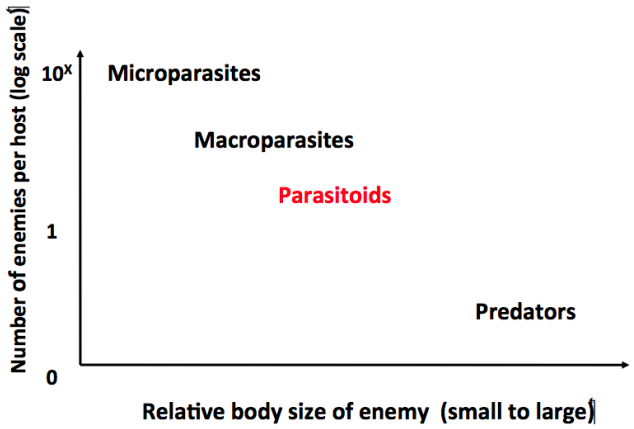

Figure 2: The heirarchy of natural enemies.

how this life style persists, as well as consider some practical uses of parasitoids as biocontrol agents.

Example life cycle

Dinocampus coccinellae is a brachonid wasp that acts as a parasitoid with its host: adult ladybug beetles.

1. First, the wasp searches for ladybug beetle hosts by probing with its antennae
2. On finding a potential host, the wasp stalks it with its ovipositor curved under its body ready to strike
3. The ladybug defends itself by running/flying away, and by crouching to prevent the ovipositor from accessing its soft underbody
4. If the defense is not successul, the wasp oviposits a single egg into the the underbody of the ladybug
5. Later the egg hatches and the larva begins to feed on non-essential parts of the ladybug (fatty tissue, ovarioles)
6. When ready to emerge, the larva bites through the nervous system, paralyzing its host
7. The larva spins a sticky cocoon under the still-living ladybug benefitting from host protection
8. Eventually the adult wasp emerges and the ladybug dies

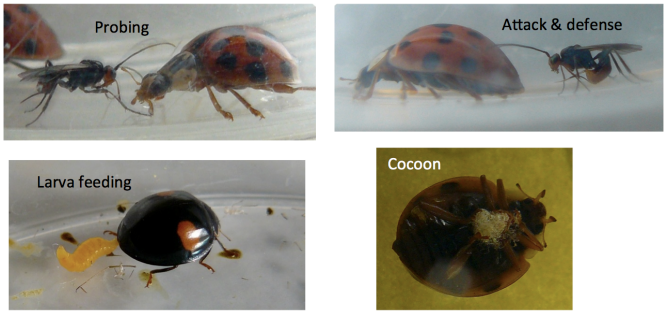

Figure 3: Images from the interactions of *Dinocampus coccinellae* wasps with their ladybug hosts.

Defenses

Given that parasitoids are hierarchically between predators and parasites, it is perhaps not surprising that host evasion from parasitoids covers a range of factors that span predator-prey and host-parasite interactions. Anti-parasitoid defenses include:

- Behavioral: hiding, evasion, and even predation!
- Physical: armor
- Chemical: defensive secretions, toxin sequestration
- Immune response: encapsulation (eggs surrounded by phagocytes and walled off from susceptible tissue)

These defenses are costly, whether it be the loss of time to forage for food or to find mates due to hiding and evasions, through to trade-offs between encapsulation and development. This cost of defense implies that their are other mechanisms at play facilitating the coexistence of parasitoids and their hosts.

Niogret et al. (2009) found that Indian meal moth *Plodia interpunctella* individuals that resisted the hymenopteran wasp *Venturia canescens* by successful encapsulation were significantly smaller than moths that were not challenged with a parasitoid

California red scale and evidence for coexistence

California red scale *Aonidiella aurantii* is an armored scale insect that can have a large, negative impact on citrus crops. It was first found in the US in the mid-to-late 19th century, possibly brought in unknowingly on infected plant material shipped from Australia. Along with selective use of pesticide, parasitic wasps (particularly in the *Aphytis* genus) continue to be used as a control method.

Given the variable success of biocontrol agents in the past, detailed scientific studies have been conducted to better understand the population dynamics of such host-parasitoid interactions. One of these, conducted by Murdoch and colleagues (Science, 2005), found that the presence of the parasitoid wasp would rapidly bring red scale populations under control and that the two populations would then coexist at relatively low, stable population densities (Fig. 4).

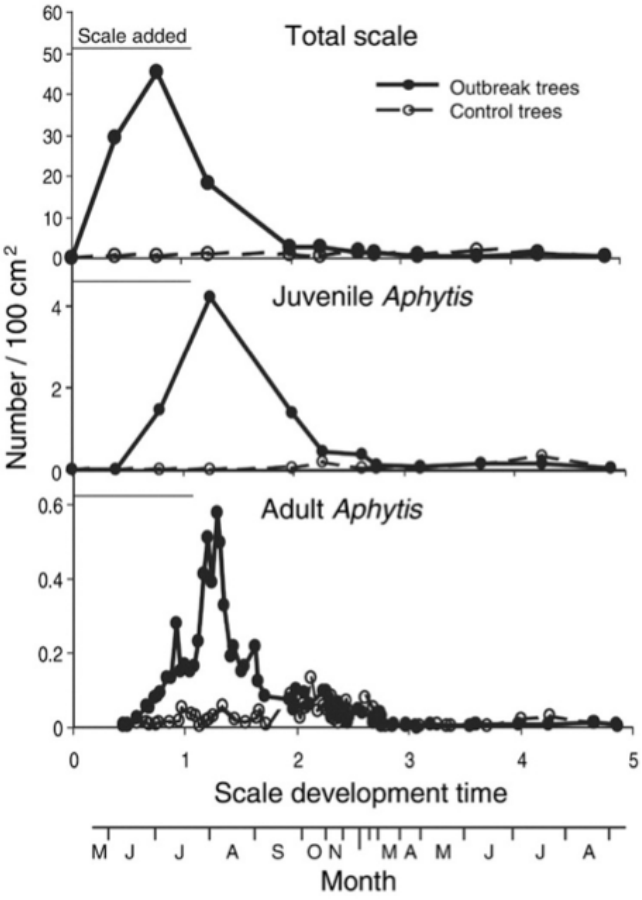

Figure 4: Data from Murdoch et al. 2005 (Science) showing mean densities of host (scale) and parasitoid (*Aphytis*) in four outbreak and 10 control trees over five scale development times (16 months).

While there are many theories for how antagonistic interactions such as host-parasitoid can persist, Murdoch et al. where able to identify two mechanisms that were critical to explaining their findings of host-parasitoid coexistence:

- An invulnerable adult life stage of the host
- Rapid development of the parasitoid

They confirmed their findings using an independently-parameterized population model.

The Nicholson-Bailey model

Although the sort of population models used by Murdoch et al. are fairly advanced, back in 1935, Nicholson and Bailey (respectively, entomologist and physicist) proposed a simple model for the interaction between a host and its parasitoid. Their broad assumptions were

- Host and parasitoid have a single generation per year, then die
- Host-parasitoid encounters occur at random
- The interaction can be captured as a discrete-time model tracking Host (H_t) and Parasitoid (P_t) density in year t

From studying discrete-time population dynamics we recall the following formula

$$N_{t+1} = \lambda N_t \tag{1}$$

Nicholson and Bailey started here and asserted that in year $t+1$:

The literature prefers *parasitized* rather than *parasitoidized* to refer the laying of eggs in a host by a parasitoid

- Unparasitized hosts each produce λ new hosts
- Parasitized hosts each produce c new parasitoids
- The probability of a host not being parasitized is $\phi = \phi(P_t)$, i.e., the probability is a function of parasitoid density
- The probability of a host being parasitized is $1 - \phi$

This results in the following equations

$$\begin{aligned} H_{t+1} &= \lambda H_t \phi \\ P_{t+1} &= c H_t (1 - \phi) \end{aligned} \tag{2}$$

At this point we need to stop and consider what the probability of not being parasitized, ϕ, is. We've made the plausible assumption that it depends on parasitoid density, P_t. But how?

Nicholson and Bailey started by assuming that parasitoids oviposit (lay their eggs) randomly into hosts, even if the host is already parasitized. Additionally, they assumed that each host is attacked at a rate aP_t, where a is the parasitoid's search efficiency. Processes like this (random encounters at some rate) are well-studied and described by the Poisson distribution:

$$\text{Prob. (attacked exactly } n \text{ times)} = \frac{(aP_t)^n e^{-aP_t}}{n!} \tag{3}$$

The symbol $n!$ reads as "n factorial" and is the product $1 \times 2 \times \cdots \times (n-2) \times (n-1) \times n$

We can now express the probability of not being parasitized as this is the same as being parasitized exactly zero times. Substituting $n = 0$ into equation 3 reveals

$$\phi = e^{-aP_t} \tag{4}$$

which we can substitute into our host-parasitoid interaction equations 2 to get

$$\begin{aligned} H_{t+1} &= \lambda H_t e^{-aP_t} \\ P_{t+1} &= cH_t(1 - e^{-aP_t}) \end{aligned} \tag{5}$$

where λ is the number of new hosts produced by one unparasitized host (equivalently $\lambda = e^r$ where r is the intrinsic growth rate), a is the parasitoid searching efficiency, and c is the number of new parasitoids produced by a parasitized host.

Dynamics of the Nicholson-Bailey model

In studying competition between species with the Lotka-Volterra equations, we learned that density-independent prey growth and random predator-prey encounters (type 1 functional response) led to neutral stability and population cycles. The Nicholson-Bailey model makes the same assumptions, but there is a delay (population sizes in year $t+1$ are determined by population sizes of the previous year). Generally, delays are de-stabilizing since populations can temporarily escape regulatory mechanisms. Indeed, for many plausible parameterizations of the Nicholson-Bailey model, populations cycle with increasing amplitude leading to population extinctions (Fig. 5).

Such population dynamics have been observed in laboratory host-parasitoid systems, but we know from the red scale case study, that long-term stable interactions occur in nature. This leads us to the idea that there must be some stabilizing process not accounted for in the Nicholson-Bailey model.

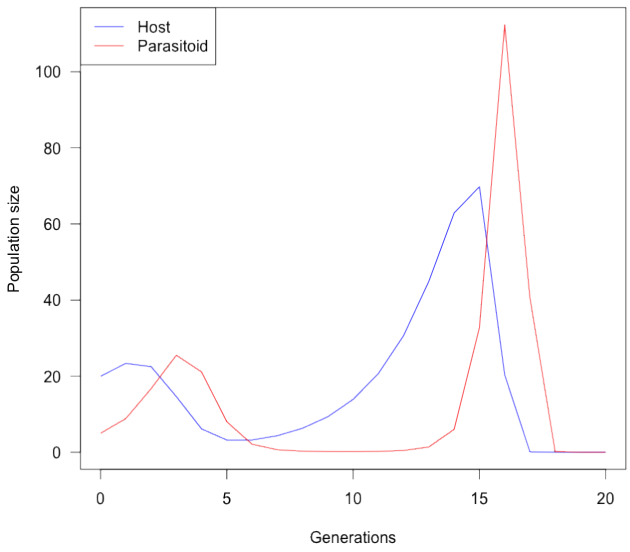

Figure 5: Dynamics of the Nicholson-Bailey model leading to mutual extinction of host and parasitoid. Model parameters are $\lambda = 1.5, a = 0.05, c = 2.0$. Initial conditions are $H_0 = 20, P_0 = 5$.

Stabilizing mechanisms: Host self-regulation

We have seen examples in competition and predator-prey interactions that self-regulation (caused by intraspecific competition leading to a carrying capacity) is stabilizing. We can include host self-regulation in the Nicholson-Bailey framework by modifying the host growth rate, λ (equivalently, e^r). If we use the e^r notation, then the Nicholson-Bailey model can be written as

In competition models (-/-) coexistence occurs when intraspecific competition is stronger than interspecific competition; also for predator-prey interactions (+/-) the cycles of the Lotka-Volterra model disappear with density-dependent prey growth

$$\begin{aligned} H_{t+1} &= e^r H_t e^{-aP_t} \\ P_{t+1} &= cH_t(1 - e^{-aP_t}) \end{aligned} \tag{6}$$

which can be simplified to

$$\begin{aligned} H_{t+1} &= H_t e^{r - aP_t} \\ P_{t+1} &= cH_t(1 - e^{-aP_t}) \end{aligned} \tag{7}$$

The addition of density-dependence can be achieved by replacing r with $r - \frac{rH}{K}$, where K is the host carrying capacity

$$\begin{aligned} H_{t+1} &= H_t e^{r - rH/K - aP_t} \\ P_{t+1} &= cH_t(1 - e^{-aP_t}) \end{aligned} \tag{8}$$

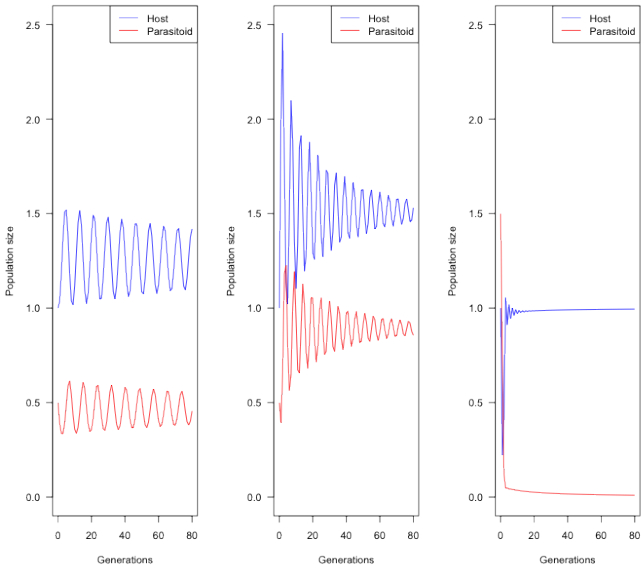

Figure 6: Examples of the variety of dynamics exhibited by the Nicholson-Bailey model with density dependence, equations 8. In all panels $a = 1.0, c = 1.0$. From left to right, (i) $r = 0.8, K = 3.0$, (ii) $r = 1.8, K = 3.0$, (iii) $r = 1.8, K = 1.0$. Note: low values of K reflect that population size is being estimated over a (small) spatial sample.

Versions of the Nicholson-Bailey model with host density dependence display a wide range of behaviors, with examples shown in Fig. 6: (i) sustained cycles (a form of coexistence), (ii) damped oscillations to coexistence, (iii) parasitoid extinction, host persistence. In addition to those examples, for appropriate parameterizations, we may also observe (iv) coexistence with aperiodic dynamics as well as (v) alternative stable states - where either coexistence or parasitoid extinction will occur, determined solely by initial conditions. Studying models like these helps us to understand why the outcome of biocontrol via parasitoid introduction is so variable.

Stabilizing mechanisms: alternatives

To the observations of Murdoch and colleagues, we can add some additional mechanisms

- Invlunerable adult stage in hosts
- Rapid parasitoid development
- Type 3 functional response
- Parasitoid self-regulation
- Spatial heterogeneity

The study of these mechanisms requires additional model complexity.

Summary

- Parasitoids are a ubiquitous natural enemy of insects
- Since generations are non-overlapping, delays lead to inherent instability (Nicholson-Bailey)
- Including prey self-regulation is stabilizing, but more complex dynamics possible (hence variable success of parasitoids as biocontrol)

Test yourself

- In what ways do host-parasitoid interactions resemble host-parasite interactions and predator-prey interactions?
- What were Murdoch and colleagues trying to explain, and what were their main findings?
- Starting from $H_{t+1} = \lambda H_t$, extend this in the style of Nicholson-Bailey's original model, to include a parasitoid that oviposits one egg per host, and in which 50% of the hosts become parasitized.
- What are the possible outcomes of the model of Nicholson-Bailey with host density-dependence included?
- Name at least four mechanisms that can stabilize the interaction between hosts and parasitoids.

Further reading

- Murdoch, W., Briggs, C.J. and Swarbrick, S. 2005. Science. "Host Suppression and Stability in a Parasitoid-Host System: Experimental Demonstration"
- Niogret, J., Sait, S.M. and Rohani, P. 2009. Ecological Entomology. "Parasitism and constitutive defense costs on host life history traits in the parasitoid-host interaction *Venturia canescens* - *Plodia interpunctella*"

Homework

1. In the following sentences, choose one word from each set of [square brackets] so that the paragraph makes most sense to you.

   [Generalist/Specialist] parasitoids may undergo host switching when the original host becomes low in abundance. Host switching can best be modeled by a type [1/2/3] functional response. Such a functional response is likely to [stabilize/destabilize] the host-parasitoid interaction.

2. Which mechanism is responsible for the last sentence in Question 1?

   (a) Imperfect tracking of the host population by the parasitoid population, leading to unregulated host growth and boom and bust cycles

   (b) Self-regulation of the host-population which then grows to a carrying capacity

   (c) Intra-specific competition between parasitoids

   (d) The parasitoid prefers an abundant host which prevents extinction of hosts at low abundances

3. If only a fraction ϕ of individuals reproduce, and the multiplitcative growth rate is λ such that $H_{t+1} = \lambda H_t \phi$, write λ as a function of ϕ assuming the population is at equilibrium.

4. Imagine the term $\phi = e^{-aP_t}$, equivalenty $\phi = \exp(-aP_t)$ were replaced by $\phi = \exp(-aP_t^{1-m})$ with $0 > m > 1$. Sketch the relationship between the new version of ϕ (y-axis) and m (x-axis) and write down a plausible biological explanation for the parameter m.