Steps Towards "The Influence of Adaptive Movement on Coevolutionary Dynamics"

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Thanks to my collaborators (on related topics)

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Outline:

1. Why should we care?
-- Evolutionary stability (or not)
of biological control (bringing Holt and Hochberg 1997, Ecology up to date)

2. Proposition: Spatial heterogeneity and unstable dynamics can decouple the ecological and evolutionary impacts of a natural enemy on its host – adaptive movement can influence this

3. A few remarks about coevolution

Why do we care?

In addition to many issues in basic science, adaptive movement may influence significant applied problems, and help explain a "puzzle"

Control of pest species

"*Control*" = reduction in average abundance of a target species, N*, below the abundance K expected in the absence of control, i.e. $q = N^*/K \ll 1$

***Biological control* =**
"The control of pests... through the use of organisms that are natural predators, parasites, or pathogens"

vs. *Chemical control* = control of pests by the application of synthetic or naturally-derived pesticides

www.thefreedictionary.com

"When is biological control evolutionarily stable (or is it)?" **Holt and Hochberg Ecology 78:1673-1683 (1997)**

Taking the empirical literature at face value,

There is a *striking* contrast in evolutionary responses by target pest species to chemical, vs. biological control

The evolution of pesticide resistance is ubiquitous
-- hundreds (to thousands) of examples
-- some insects (e.g. diamondback moth) are now resistant to all known chemical pesticides

There are many fewer clear-cut examples of the loss of control due to evolution in biological control (and most of these involve pathogens, e.g., myxomatosis – and these involve e.g., loss of virulence)

Ten years later, this qualitative assertion still seems to hold...

# of Cases of evolved loss of control follow a clear pattern:

Chemical >> Pathogen > Parasitoid/predator
(and it is hard to find ironclad examples of latter)

Yet biological control agents would seem to impose very strong selection on target species

Assuming the pattern is real, why does it exist?

5 possible reasons for evolutionary stability of biological contrl

Genetic differences

1. Lack of genetic variation

2. Constraints on selection because of genetic correlations among traits

Ecological and organismal differences

3. Population dynamics in both host, and control agent

4. Behavior (including adaptive movements)

5. Coevolutionary arms races

1. Lack of genetic variation

-- many studies show genetic variation in traits that *could* lead to evolved resistance (e.g., in Drosophila, work of Alex Kraaijveld and Charles Godfray)

-- possible in some cases, but unlikely to be a general explanation across many species

2. *Genetic constraints* (correlated traits)

Maybe genetic constraints are more likely for evolution of resistance to biological control agents, than to chemicals

e.g., if resistance involves shifts in quantitative traits, such as body size or phenology, these will usually be correlated with other traits that also influence fitness, and so are "costly"

Resistance to chemicals may be "lock and key", with weaker (to no) correlations with other traits (e.g., a given pesticide may disrupt a single metabolic pathway, and this disruption can be countered with a low-cost change in a single enzyme)

3. *Population dynamics*

Predator-prey interactions are intrinsically unstable, particularly when predation (i.e., biological control) is effective at limiting prey.

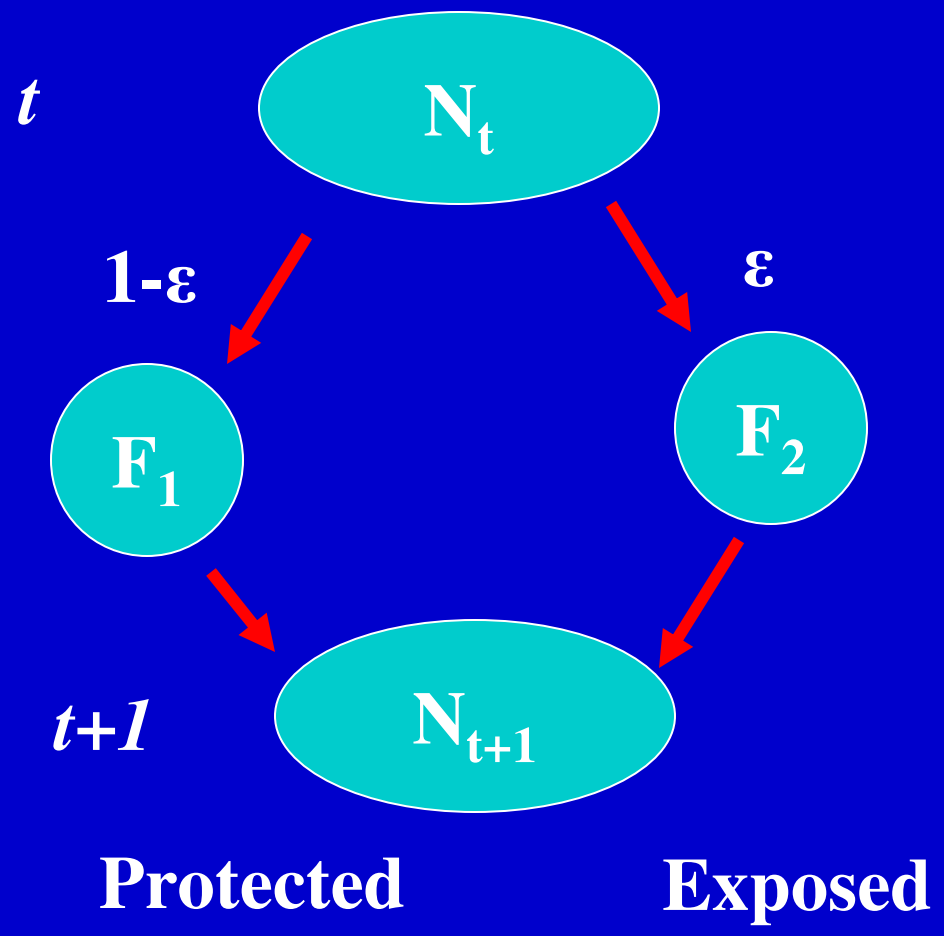
What permits persistence?
The most general mechanism appears to be spatial heterogeneity, e.g., refuges

How does this affect evolutionary dynamics?
What is the role of adaptive movement?

Conjecture:

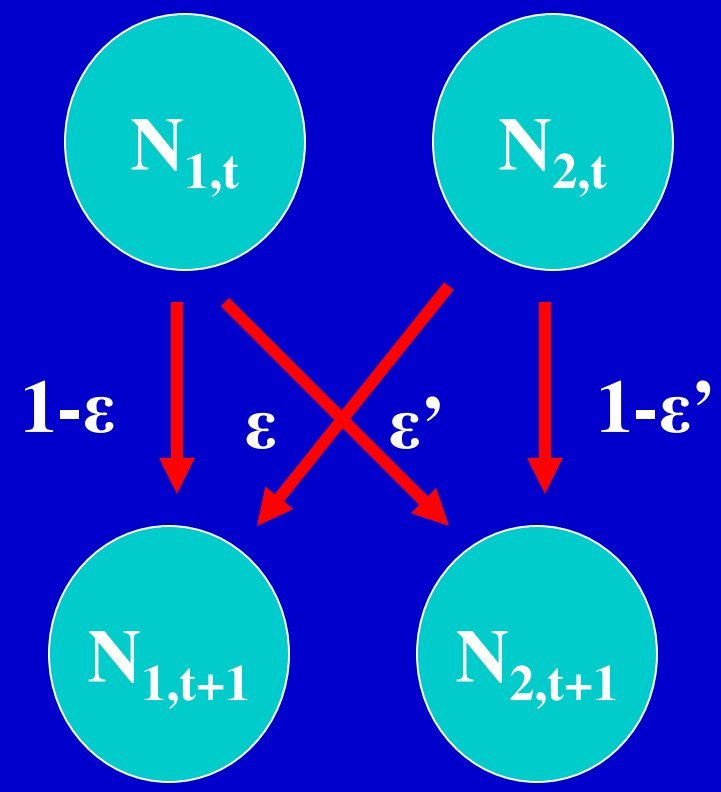
There can be a decoupling of predation as a factor limiting prey numbers, and its effectiveness as a selective agent on prey – and this decoupling can (sometimes) be enhanced by adaptive prey movement

One population, with refuge



Today's talk

Coupled populations



Start with a host that is initially in demographic equilibrium, and with an "ideal free" behavioral equilibrium, and assumes it moves quickly to a new equilibrium, after control is imposed.

We assume control measures only impact prey *outside* the refuge.

A "target" pest with a proportional refuge

$$N_{t+1} = (1-\varepsilon)N_t F_1(N_{1,t}) + \varepsilon N_t F_2(N_{2,t})$$

$$N_{1,t} = (1-\varepsilon)N_t \ , \ N_{2,t} = \varepsilon N_t$$

For demographic equilibrium: $N_{t+1} = N_t = N^*$

For behavioral "ideal-free" equilibrium

$F_1(N_1) = F_2(N_2)$, *fitness equlibration*

⇒Each patch equilibrates at
its respective "carrying capacity"
(N_i such that per capita fitness = 1)

=> $$\varepsilon = \frac{K_2}{K_1 + K_2}$$

How does evolution influence the ability to utilize the refuge and exposed habitats?

Consider the dynamics of a clone which has habitat-specific fitnesses (when rare) differing from the resident clone by δ and δ'

The dynamics of the invading clone:

$$N'_{t+1} = (1-\varepsilon)N'_t(F_1(N_{1,t}) + \delta) + \varepsilon N'_t(F_2(N_{2,t}) + \delta')$$

Assuming the resident clone is at equilibrium,

$$\frac{N'_{t+1}}{N'_t} = \text{ fitness of invader } \lambda \ = (1-\varepsilon)\delta + \varepsilon\delta'$$

= fitness changes, weighted by movement rates

How does "control", and adaptive movement influence the rate of increase of this clone?

$$\lambda = (1-\varepsilon)\delta + \varepsilon\delta'$$

Assume movement rates are *fixed* at the pre-control ESS, then what matters in host evolution is that there is a potential coupling of selection across habitats

If chemical resistance ($\delta' > 0$), has little cost in refuge ($\delta \sim 0$), but resistance to natural enemies is usually costly ($\delta < 0$), selection is more likely to favor alleles that permit a given rate of increase in the impacted habitat, for chemical control.

If movement rates are *labile*, and again become ideal free, and control is effective (q <<1), because the new equilibrium has $N_2^* << K_2$

=> The new ideal free distribution has

$$\varepsilon' = \frac{qK_2}{K_1 + qK_2} << \varepsilon$$

⇒The novel mutant's growth rate = $\lambda' = (1-\varepsilon')\delta + \varepsilon'\delta' \approx \delta$

=> Its fate will be dominated by its effect in the *refuge*

⇒ An increase in (say) predation,
combined with adaptive prey movement
leading to an ideal-free distribution in and out of a refuge,
reduces the fraction of the prey exposed to predation,

=> This reduces the force of selection
for escaping predation.

This *could* contribute to the difference between
chemical and biological control.

This conjecture would seem to require prey to more
effectively distinguish "refuge" vs. "non-refuge" habitats
in biological control

This may be reasonable, as most prey species should have
experienced predation, parasitism, etc., in their history.

What about population *dynamics?*

Refuges may permit persistence in an attractor that is not a point equilibrium.

Example:

Nicholson-Bailey model with "proportional refuge"

Nicholson-Bailey model with refuge

Host recursion:

$$N(t+1) = N(t)[\varepsilon\lambda_1 e^{-aP} + (1-\varepsilon)\lambda_2] = W(t)N(t)$$

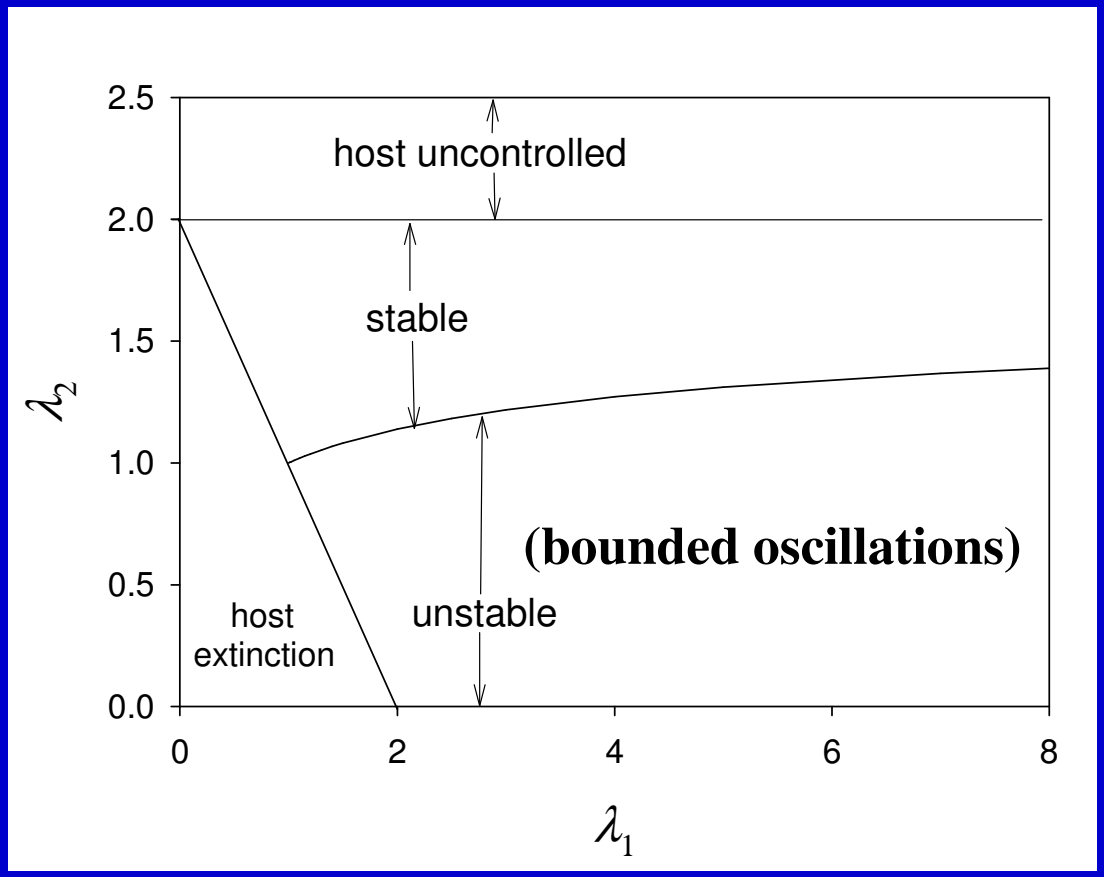
Parasitoid recursion:

$$P(t+1) = cN(t)\varepsilon(1-e^{-aP})$$

Assume "non-ideal" space use, i.e. fixed ε

Refuges that are intrinsic "sinks" tend to stabilize

Refuge hosts

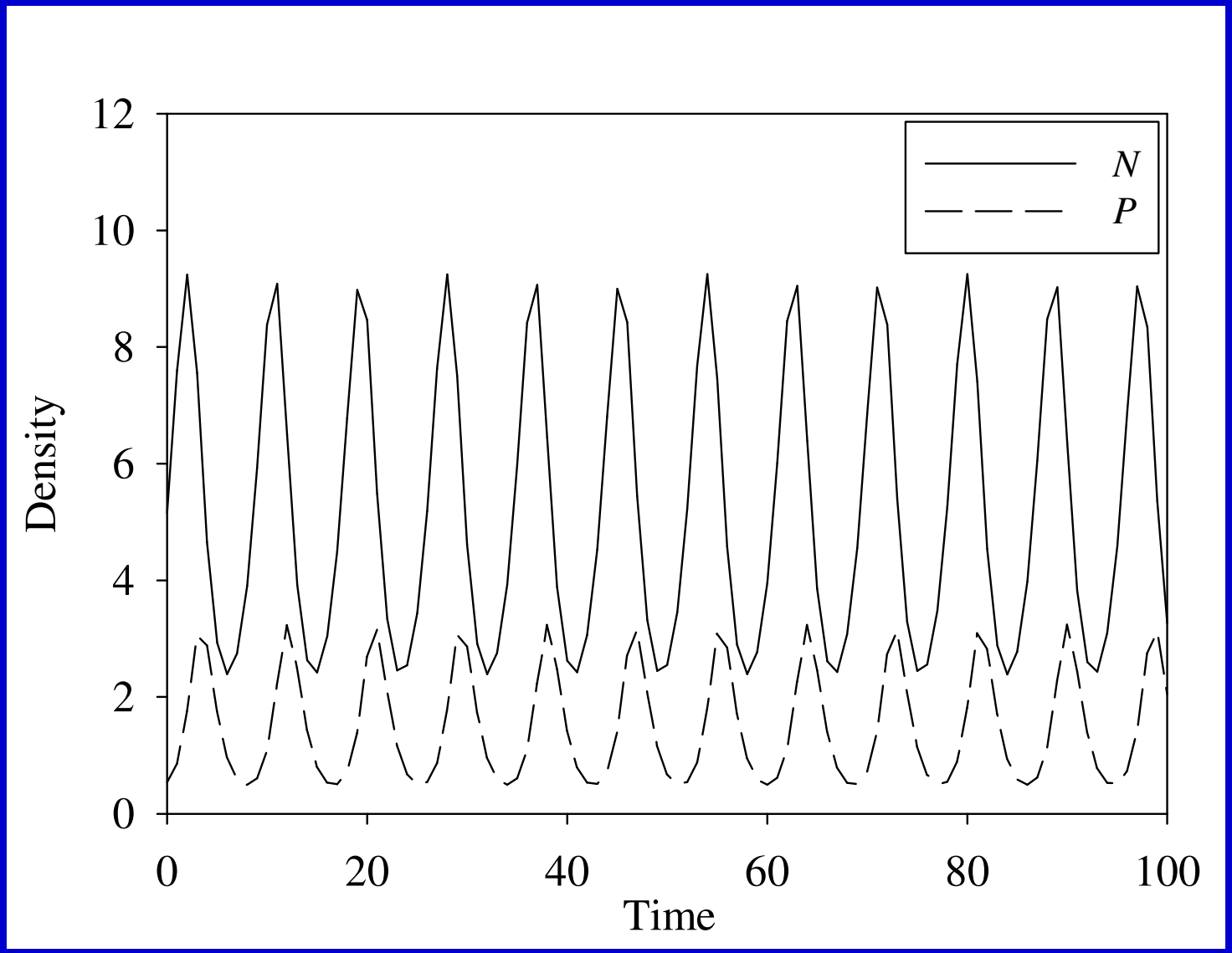


Non-refuge hosts

ε = .5

Holt and Hassell, J. Anim. Ecol. 1993

An example



How does unstable population dynamics affect adaptive evolution across the two habitats?

Consider strength of selection on each parameter in turn in equation for host fitness:

$$N(t+1) = N(t)[\varepsilon\lambda_1 e^{-aP} + (1-\varepsilon)\lambda_2] = W(t,p)N(t)$$

Strength of selection in stable population =

$$\left.\frac{dW}{dp}\right|_{W=1}$$

Substitute P*, evaluate above expression for W

[returning to Holt et al. 1999, in Theoretical Approaches to Biological Control]

What about unstable dynamics?

In unstable, persistent population fluctuating between bounds, long-term geometric mean growth rate ≈ 1

$$W_g = \left(\prod_{t=0}^{\tau-1} W(t,p) \right)^{1/\tau} \to 1$$

Strength of selection:

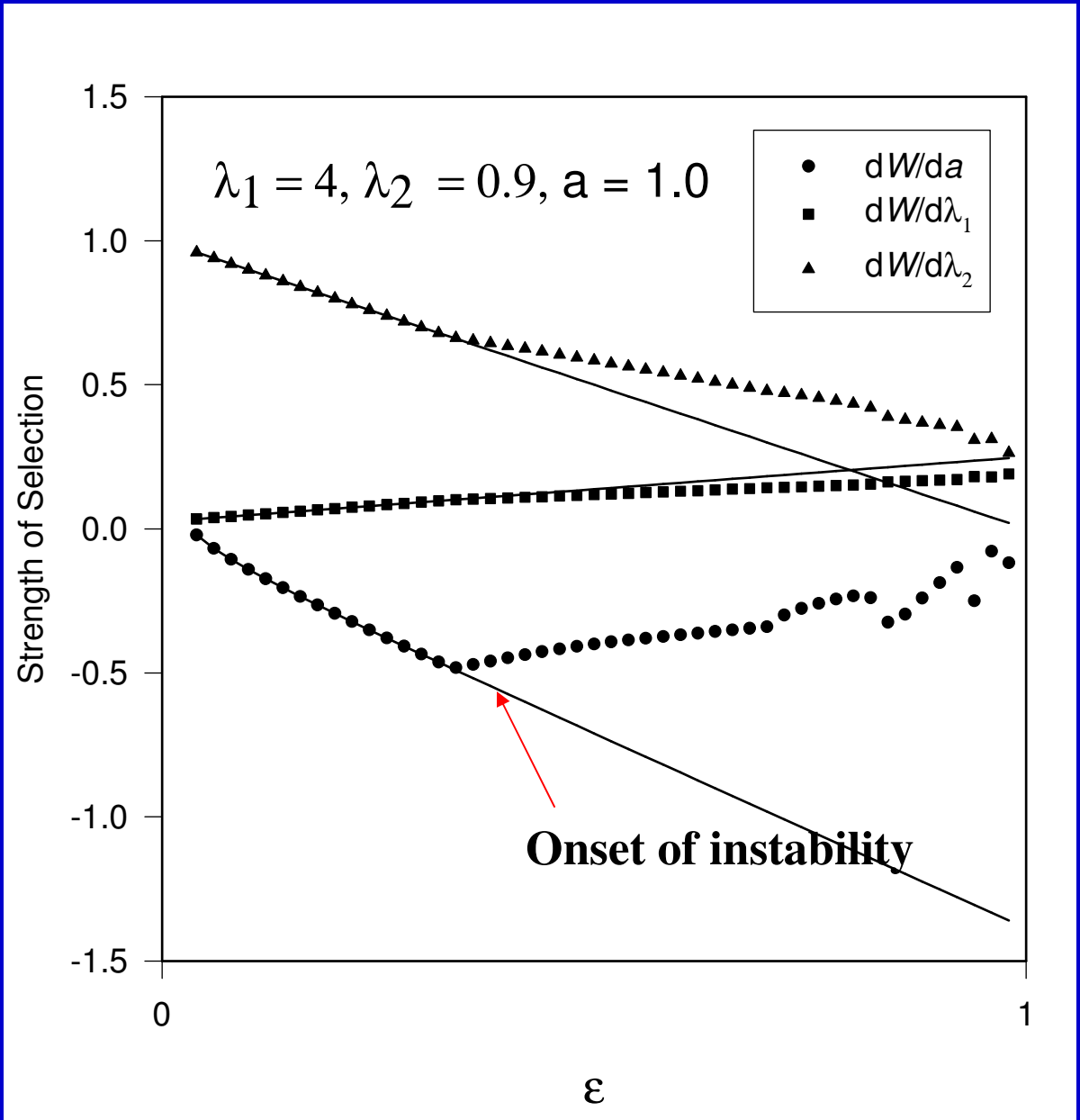
$$\left. \frac{dW_g}{dp} \right|_{W_g=1} = \left\langle \frac{1}{W(t)} \frac{dW(t)}{dp} \right\rangle$$

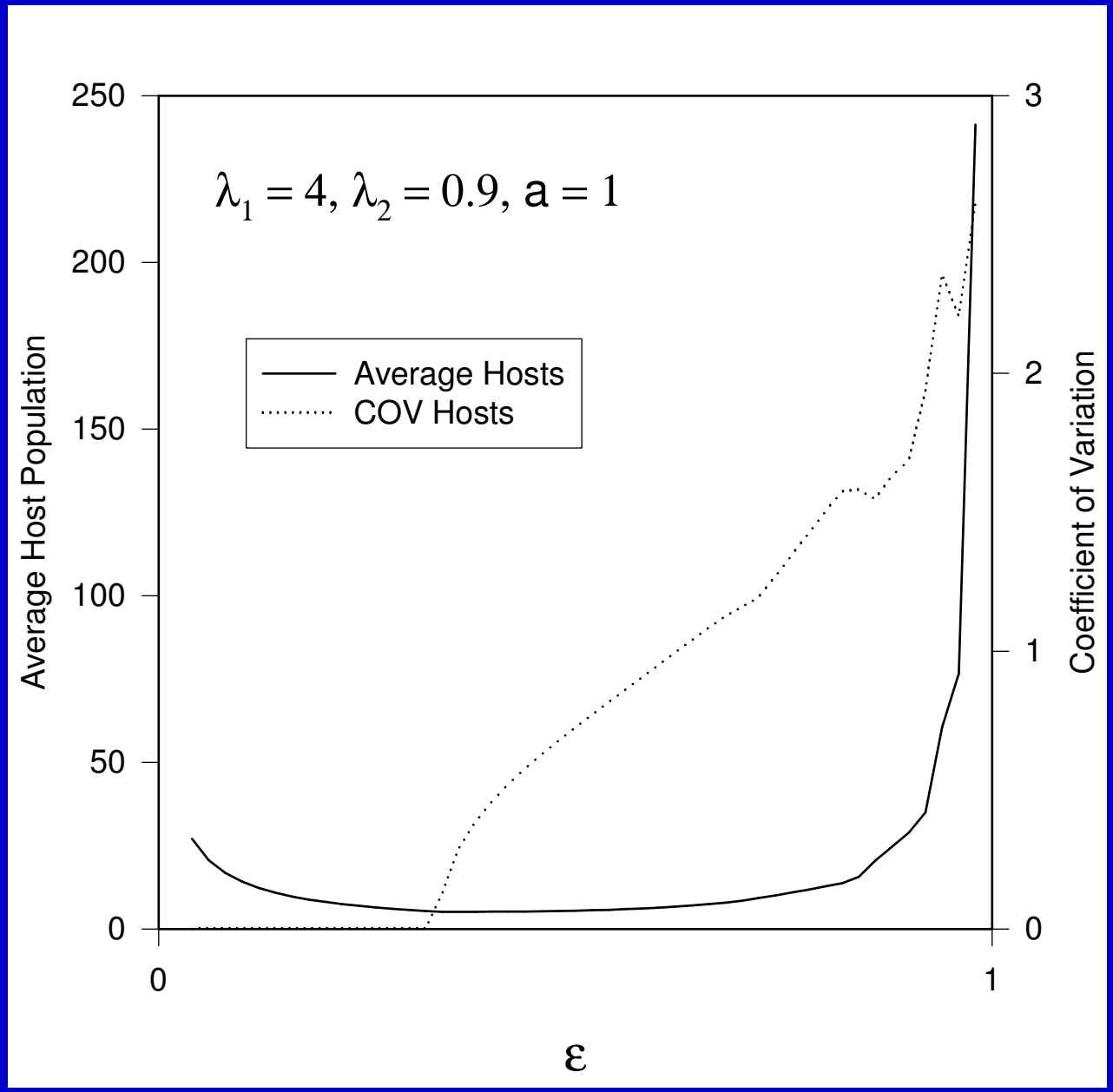
, a time-average

=> Selection weighted towards *times of low fitness*

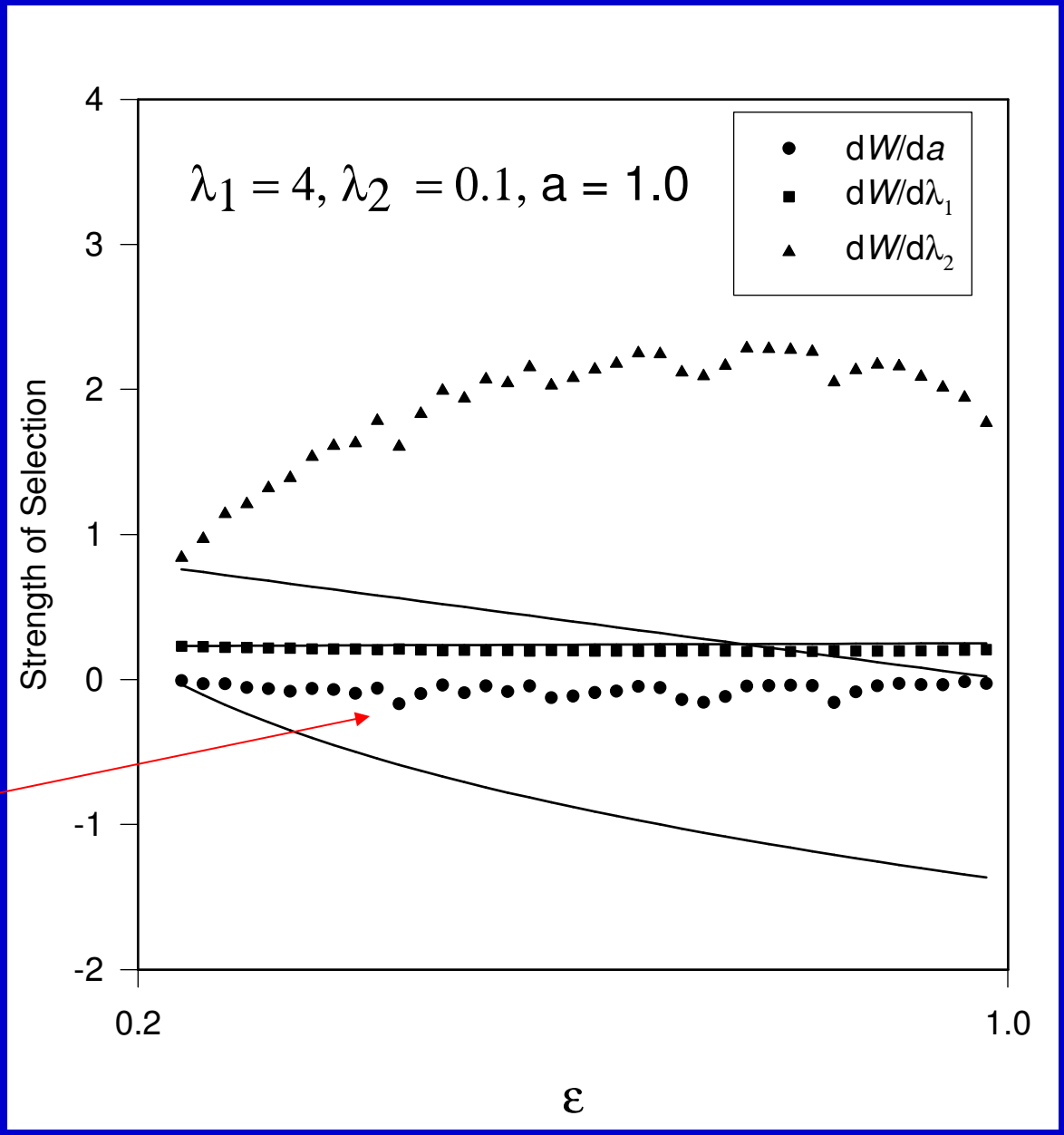
(Numerically, track the growth rate of a clone with slightly different parameter values from resident, when it is sufficiently rare that it does not perturb parasitoid)

Mild sink (solid lines, evaluated at equilibrium




$\lambda_1 = 4, \lambda_2 = 0.9, a = 1$
Average Hosts
COV Hosts
Average Host Population
Coefficient of Variation
250
200
150
100
50
0
3
2
1
0
0
1
ε

Poor sink (much more unstable)



Negligible selection on attack rate

Assume the system cycles between high and low parasitoid numbers, and that there is a large difference in host growth rates, in and out of the refuge

$$\left.\frac{dW_g}{dp}\right|_{W_g=1} = \left\langle \frac{1}{W(t)} \frac{dW(t)}{dp} \right\rangle \approx \frac{1}{W(high\ P)} \frac{dW(high\ P)}{dp}$$

$$W(t,P) = \varepsilon\lambda_1 e^{-aP} + (1-\varepsilon)\lambda_2 \approx (1-\varepsilon)\lambda_2$$

⇒Attack rate irrelevant, as is growth outside refuge

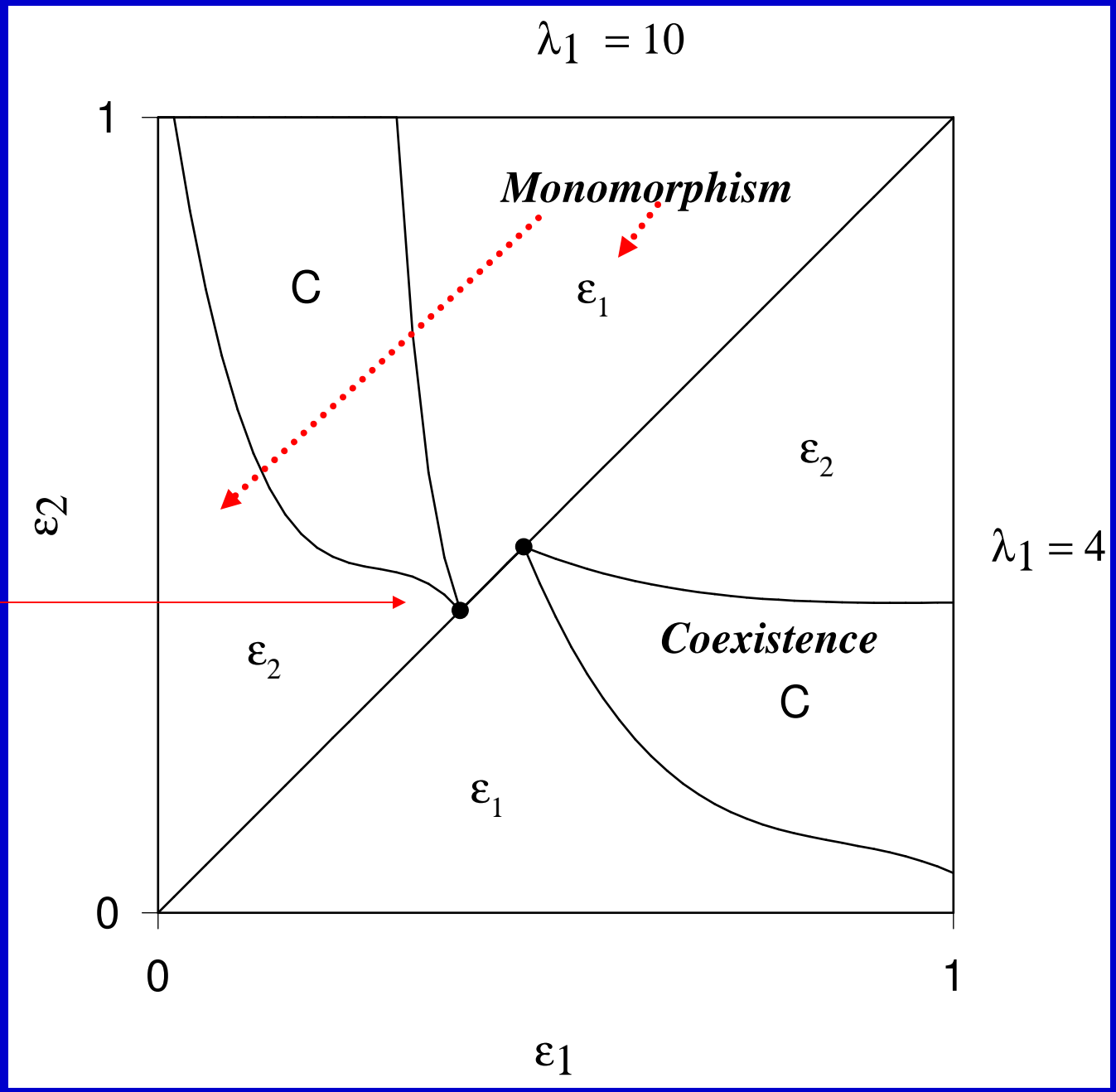
How does unstable dynamics influence the use of the refuge itself?

If the system is stable, use of the refuge is maladaptive...

But instability favors some refuge use

Conduct pair-wise competition between clones differing only in ε (and among three clones, if coexistence is observed)

For
$\lambda_2 = .9$



ESS
refuge use

Back to adaptive movement

Assume that host exposure varies with parasitoid abundance, adaptively (not optimally), according to a logistic function

***b* = strength of behavioral response**

$$\varepsilon = \frac{(1-2\varepsilon_{\min})(P/P_e)^{-b}}{1+(P/P_e)^{-b}} + \varepsilon_{\min}$$

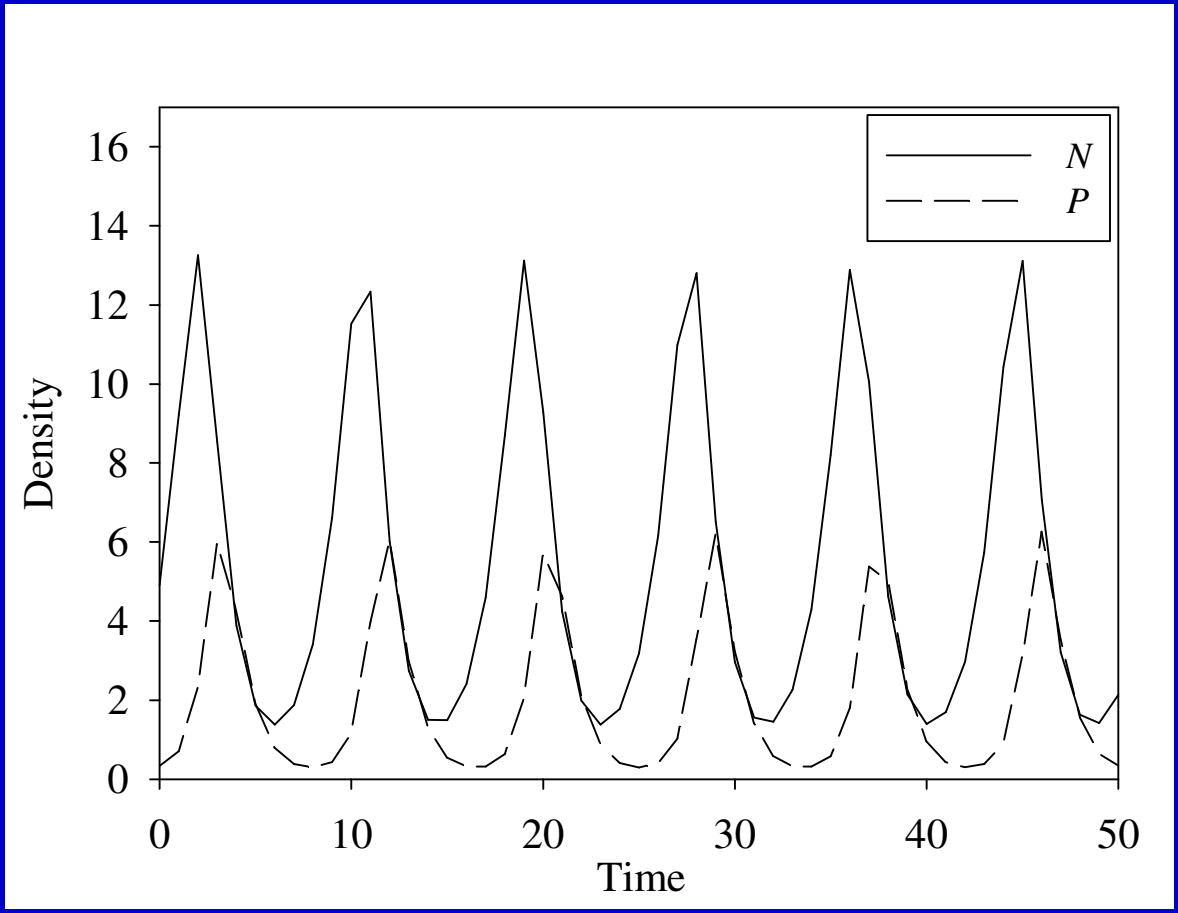
$$\lim_{P->\infty} \varepsilon = \varepsilon_{\min}$$

P_e is parasitoid density at which 2 habitats have equal host fitness, gives $\varepsilon = 0.5$.

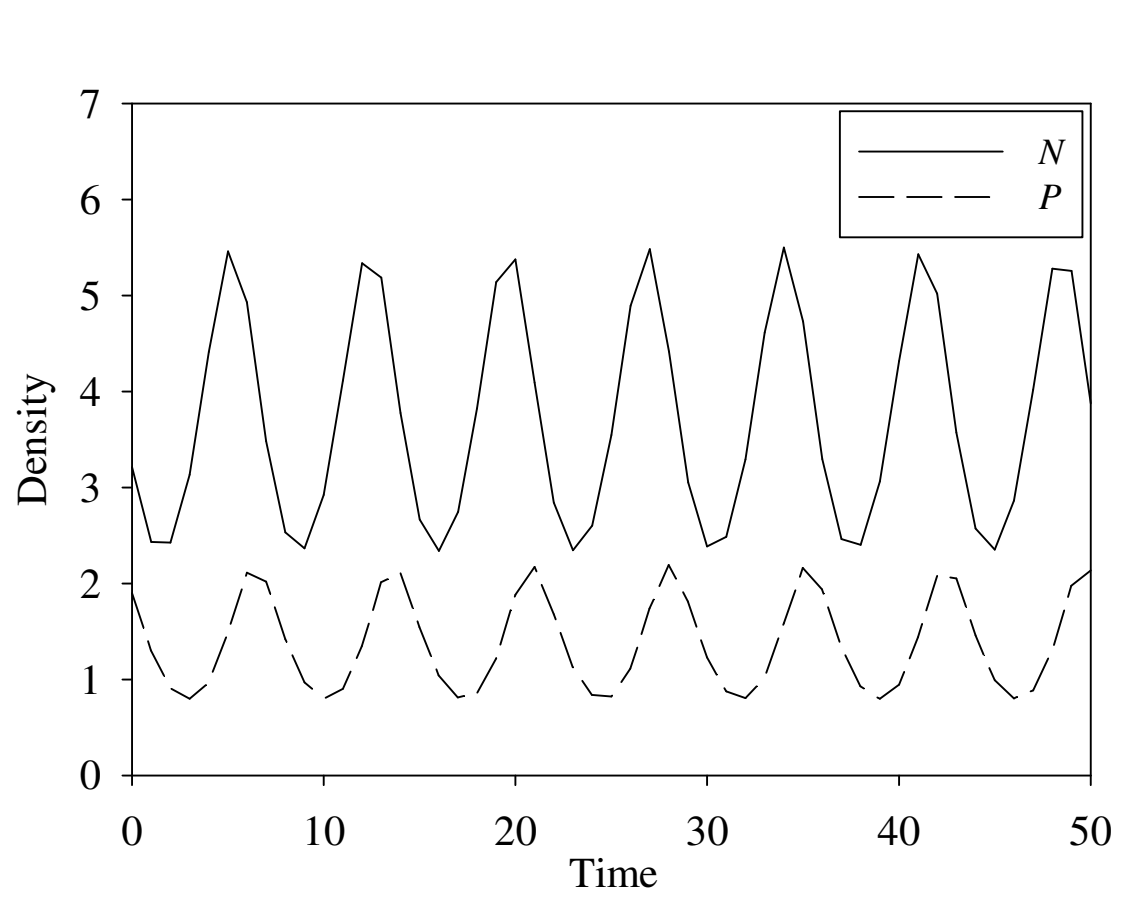
$$P_e = \ln(\lambda_1 / \lambda_2) / a$$

First, ecology, then, selection...

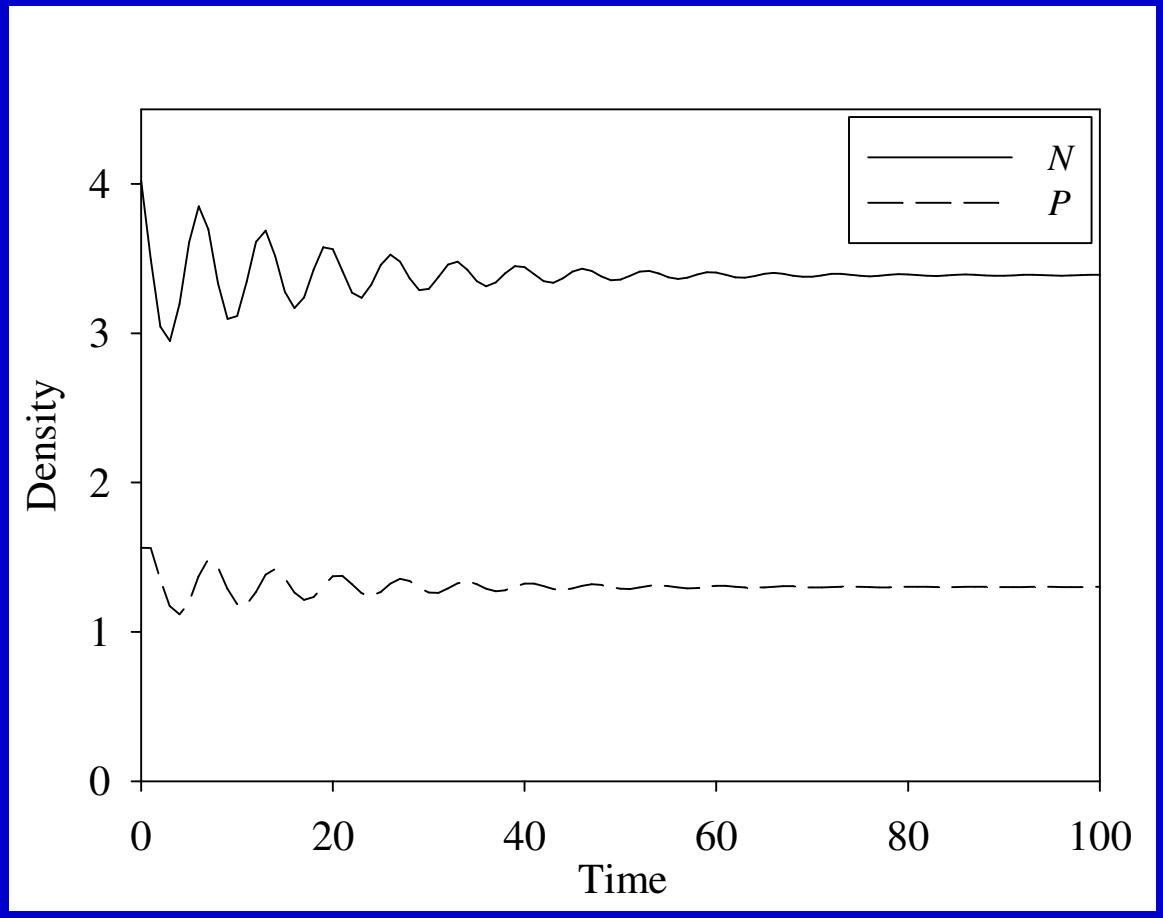
$b = 0$ (fixed, equal habitat use; mild sink)



b = .5 => more moderate cycles

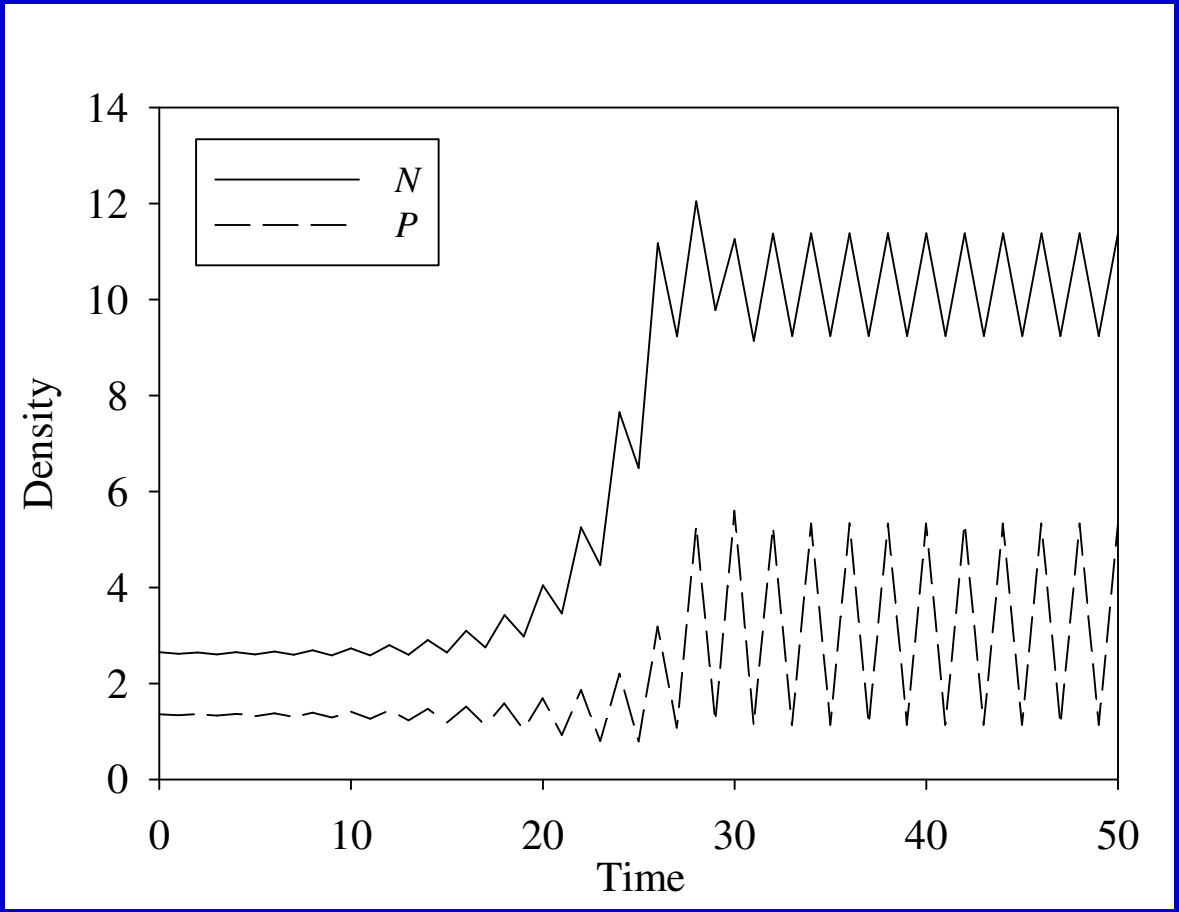


$b = 1$ (stronger switching)

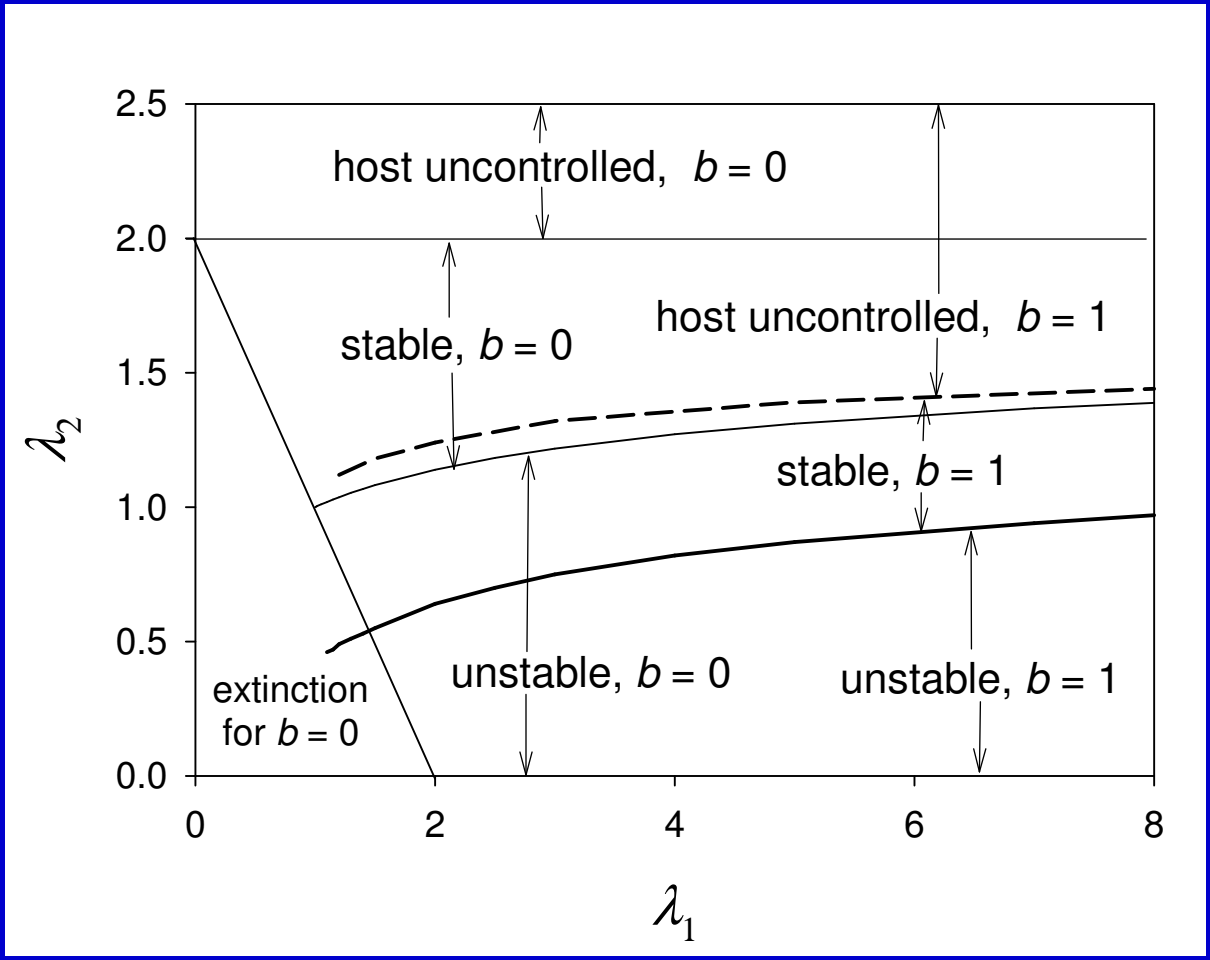


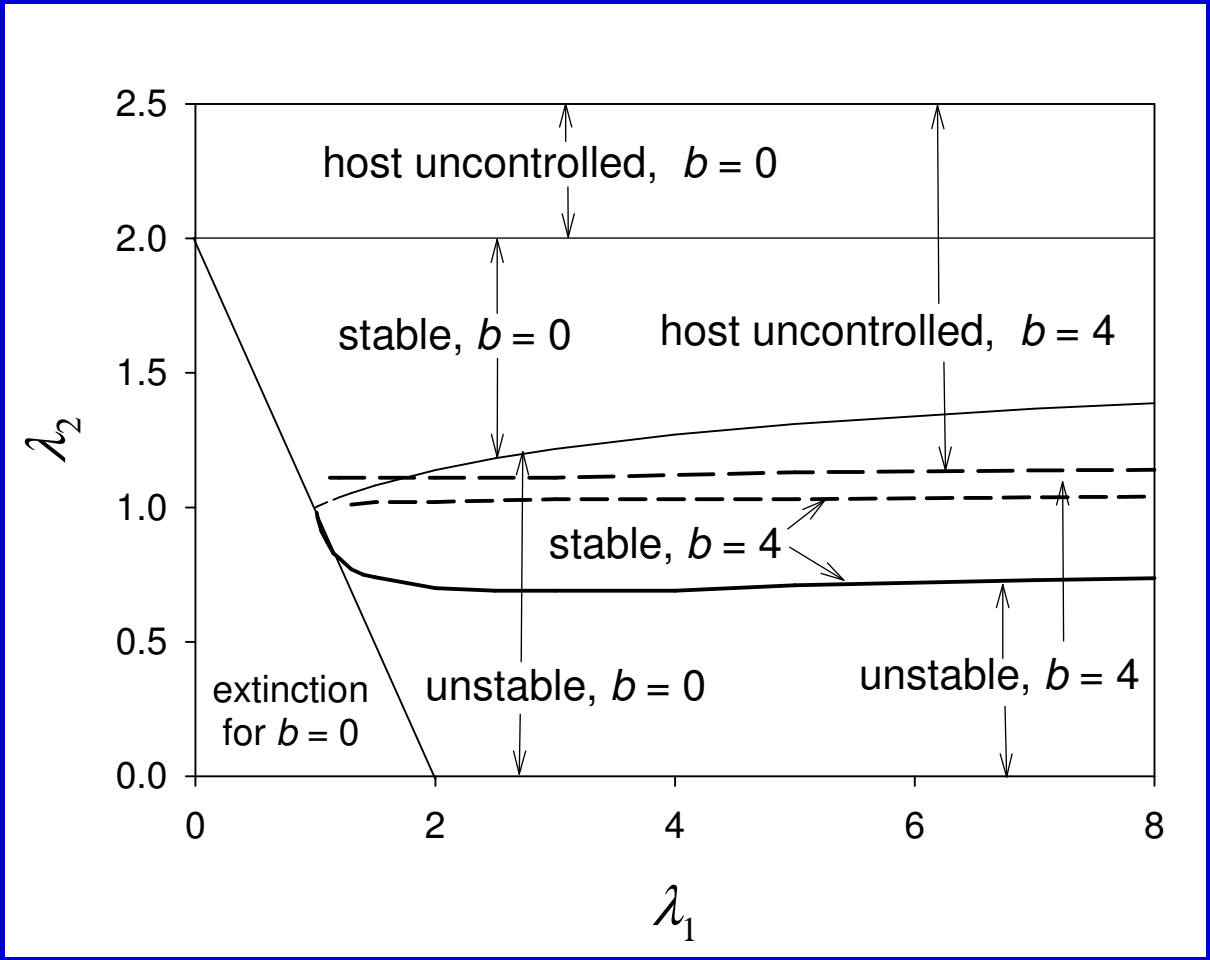
Note: outcome depends on starting conditions

b = 10 (sharp switching) => destabilizing

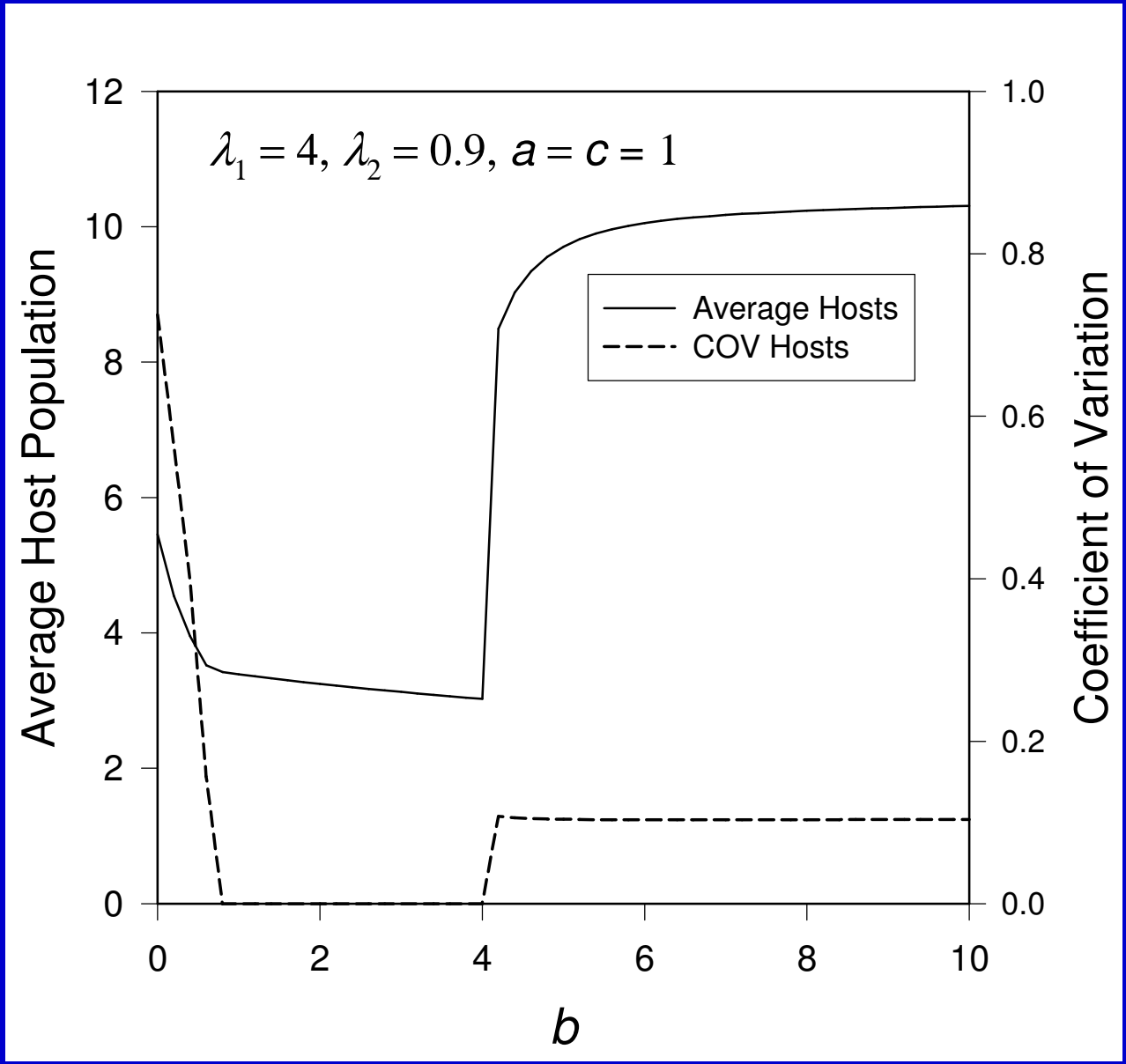


Note: shorter cycles

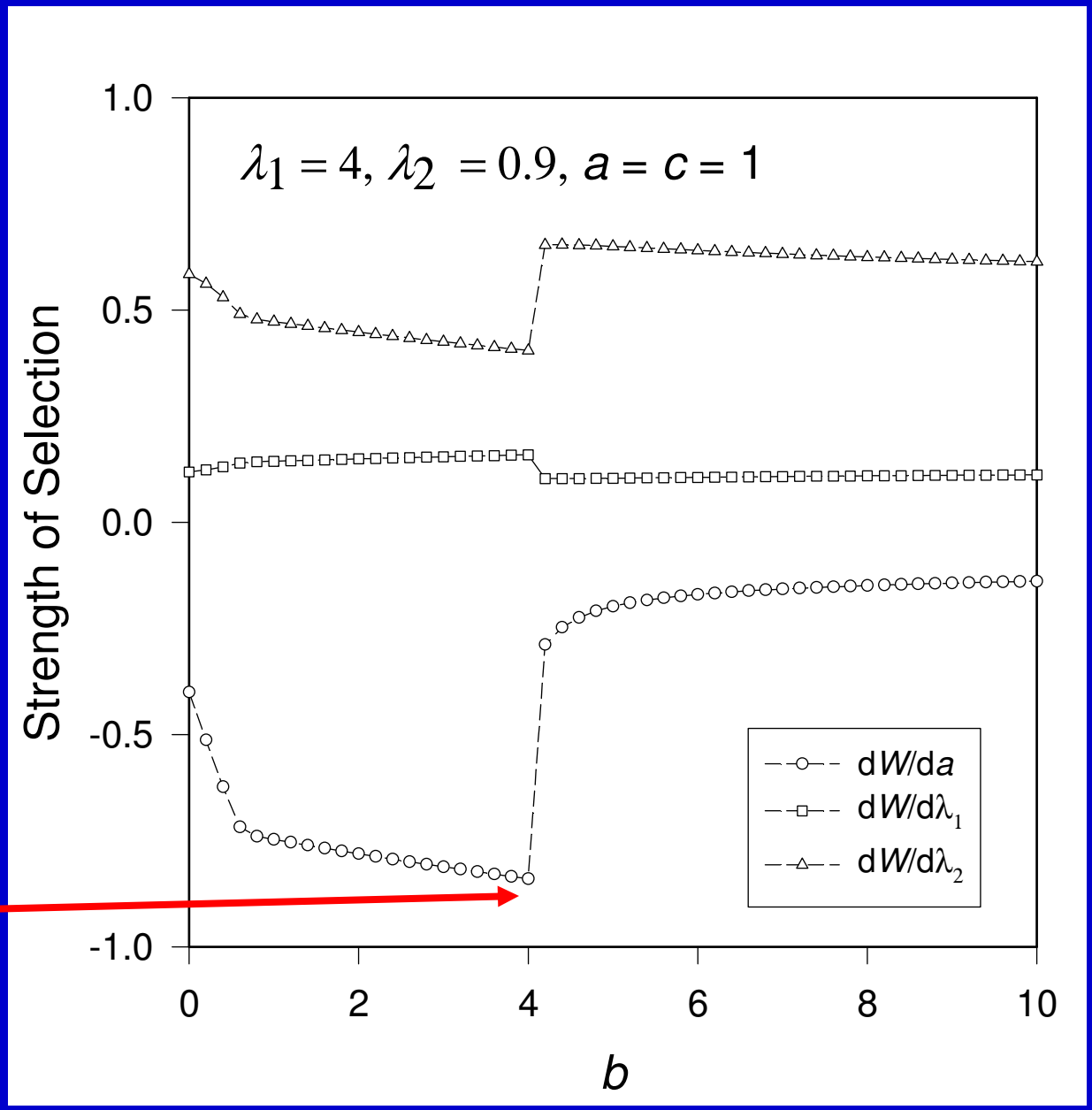

2.5
2.0
1.5
1.0
0.5
0.0
0
2
4
6
8
λ_2
λ_1
host uncontrolled, $b = 0$
host uncontrolled, $b = 1$
stable, $b = 0$
stable, $b = 1$
unstable, $b = 0$
unstable, $b = 1$
extinction
for $b = 0$


2.5
2.0
1.5
1.0
0.5
0.0
0
2
4
6
8
λ_2
λ_1
host uncontrolled, $b = 0$
stable, $b = 0$
host uncontrolled, $b = 4$
stable, $b = 4$
extinction
for $b = 0$
unstable, $b = 0$
unstable, $b = 4$

Mild sink

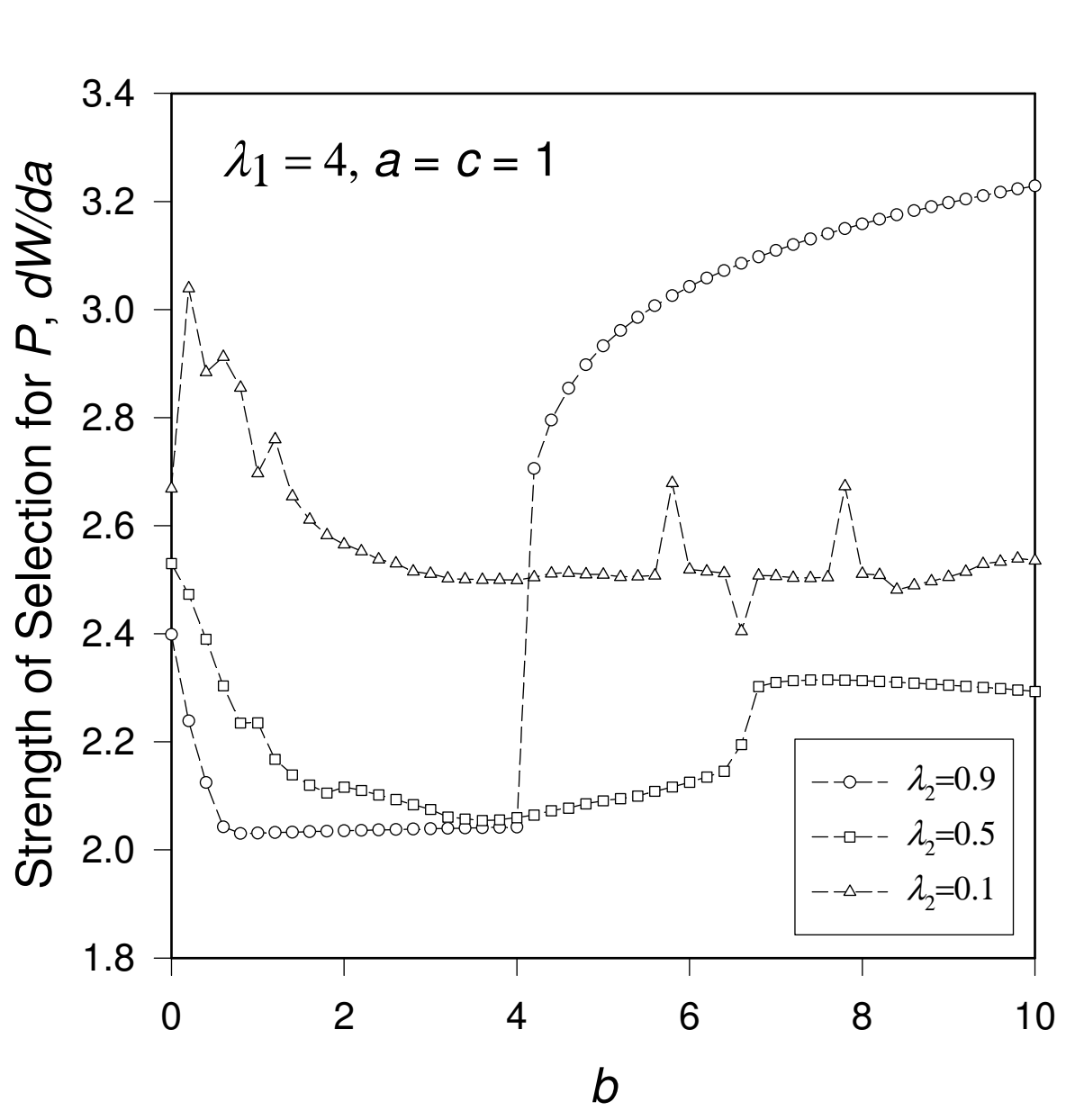


Strength of behavioral habitat "switching"



Onset of instability

For coevolution, we must have selection in parasitoid, too...



Note: all much higher than for host

Conclusions I:

Population dynamics can have large impacts upon evolutionary dynamics (a general proposition going well beyond the specifics of this talk)

Unstable dynamics tends to “focus” host selection within refuges, possibly at the expense of selection to escape the parasitoid outside

Adaptive movement has complex effects, but at least sometimes leads in same direction

Conclusions II:

Unstable dynamics and adaptive movement in heterogeneous landscapes may help weaken evolutionary responses in the targets of biological control,

Helping to explain the seeming evolutionary stability of biological control, relative to chemical control

What about coevolution? An open question....

THANK YOU FOR YOUR ATTENTION