

Chapter Eleven

Eco-Evolutionary Dynamics

11.1 CONTEXT

Life and death. Age-old questions for philosophers and scientists alike. So too, the study of how populations grow and decline, is perhaps the oldest of all branches in mathematical biology. In 1798, Malthus wrote his famous “Essay on the Principle of Population”, encapsulating what is now known as the Malthusian principle of population growth:

I say, that the power of population is indefinitely greater than the power in the earth to produce subsistence for man.

Population, when unchecked, increases in a geometric ratio. That is two become four, and four become eight, and eight become sixteen, and very soon the numbers are astronomical – far greater than the word astronomical could even convey, i.e., greater than the stars in the sky. The powers of 2 was of interest well before Malthus. The 13th century Islamic scholar Ibn Khallikān is thought to have proposed the following puzzle as a reward from the sovereign: his requests were modest, he only wanted one grain of rice on the first chessboard, two on the second, four on the third, eight on the 4th, and so on until all 64 squares are filled. The reward of course is not modest at all. By the end, it would take $2^{64}-1$ grains of rice to fill up the board or nearly 2 billion-billion grains! Exponentials are fast, and indeed, this ‘power’ – in the Malthusian sense – stands in stark contrast to the finite nature of resources. Malthus recognized this dichotomy:

Subsistence increases only in an arithmetical ratio. A slight acquaintance with numbers will shew the immensity of the first power in comparison of the second.

In modern parlance, this means that if populations grow as in $N(t+1) = rN(t)$ and the availability of food grows as $R(t+1) = R(t) + a$ then very soon $R/N \ll 1$. The balance of populations and their food, or consumers and resources, strikes to the very heart of ecological and evolutionary dynamics.

This chapter will provide an introduction, data, theory, and methods to consider how individuals making up a population interact, how such interactions drive changes in the abundances of population, and how over-time differential reproduction and survival can re-inforce trait differences that drive the evolution of complex and diverse communities. In doing so, it is important to state at the outset, what is different in this chapter than what is typically introduced as

the canonical view of ecological dynamics (see Figure 11.1). The modern origins of population dynamics is attributed to the work of Lotka and Volterra. As explained in the Preface, Alfred Lotka was a physicist and Vito Volterra was a mathematician. They both were concerned with the question of dynamic change of populations. Specifically: if populations were to oscillate in abundance did this necessarily mean that something external to the system were oscillating? Vito Volterra was inspired by inter-year variations in the yield of Adriatic fisheries. Perhaps oscillations with periods that spanned multiple years resulted from variation in some time-varying environmental property, like temperature, salinity, or mixing, that influenced the annual catch size. However, Volterra reasoned that changes in fishing pressure – a form of predation – could be sufficient to drive down yields which, in turn, would drive down incentives to fish, which could lead to an increase in fish and then the increase in fishing. The result – a model of coupled predator-prey dynamics to be introduced formally in this chapter – yields oscillations in which predators consume prey, driving prey downwards, then as prey decrease, predators decline, and given low levels of predators, the prey recover, leading to resurgence of predators and the cycle repeats.

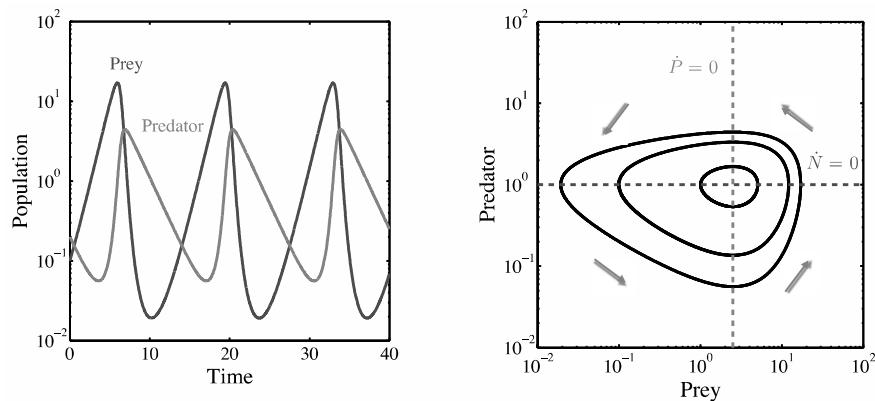


Figure 11.1: Canonical predator-prey dynamics in the original Lotka-Volterra model. (Left) Time dynamics; (Right) Phase plane view. In the phase plane, dynamics follow trajectories imprinted by distinct initial conditions as explained later in this chapter. The $\dot{N} = 0$ and $\dot{P} = 0$ dashed lines denote nullclines, with a single intersection denoting the coexistence point. The direction of dynamics is indicated by the arrows, it is counter-clockwise in the predator-prey plane.

Notably, such coupling between an ecological and a human social system has now remerged to become central to understanding modern ecosystems, at both local and global scales. The most visible evidence comes from studies of climate change, an issue to be revisited in the final chapter. But perhaps just as importantly are the ways in which human and ‘natural’ systems intersect, e.g., inspiring

the formation of national centers, including the “National Socio-Environmental Synthesis Center” (SESYNC), a NSF funded research center based in Annapolis, MD. Yet if one were to replace fisheries, *in toto*, with predators, then we have struck to the very core of theories of ecological dynamics. That is: interactions between population are sufficient to give rise to non-stationary dynamics, including oscillations, even given a “constant” environment. But these oscillations are unlikely to have the imprint of long-ago initial conditions – a feature of a model but not reality.

Indeed, we are not Lotka nor Volterra, and despite our debt to them, with one-hundred years of work, it would seem appropriate to aim for something more ambitious and, indeed, more realistic. The study of predator-prey dynamics sits squarely in the field of ecology. That is, measurements and models assume that changes due to predation (or consumption more generally) reflect changes due to ‘vital’ processes, i.e., life and death, between otherwise genetically, homogeneous populations. Such an assumption seems reasonable if one thinks that the change in the frequency of genotypes in a population varies over time-scales far slower than population rates of change. But what if that assumption is not reasonable? Indeed, perhaps one of the most exciting developments in predator-prey dynamics and consumer-resource dynamics more generally is the realization that evolution is rapid and can take place on time-scales similar to that of ecological dynamics (Hairston et al. 2005; Duffy and Sivars-Becker 2007; Cortez and Ellner 2010; Hiltunen et al. 2014). Indeed, if one doesn’t arrive to the field with the built-in bias of time-scale separation, or if one approaches the questions given a background in microbial dynamics, then it would seem apparent that the same forces that shape whether an organism lives or dies may also shape the frequency of genotypes in a population.

Understanding eco-evolutionary dynamics requires a nested approach. First, this chapter will introduce simple models of competition for a common resource and interactions between dynamic consumer and resource (e.g., as in predator-prey dynamics). These models are, in essence, the Lotka-Volterra models of competition and of predator-prey dynamics, respectively. Yet to bridge between classic and modern challenges in ecology, it is important to recognize that such classic models have many assumptions, perhaps none more important than that the consequences of interactions amongst individuals unfold on time-scales much faster than changes in the traits in the individuals in those populations.

As a key motivating example, Figure 11.2 shows multiple examples of non-canonical phase relationships between predator and prey. This study examines the population and evolutionary dynamics of rotifers and algae. Rotifers are freshwater zooplankton, i.e., small aquatic organisms that eat phytoplankton, like algae (which are themselves photoautotrophs that fix carbon dioxide into organic carbon). These organisms were inoculated in chemostats. Chemostats are continuous culture devices, i.e., flasks with an input and output port. The input port enables new food to be added to the system while the output port allows all the components to be diluted enough – simulating a background mortality rate as well as providing opportunities for continuous and non-invading

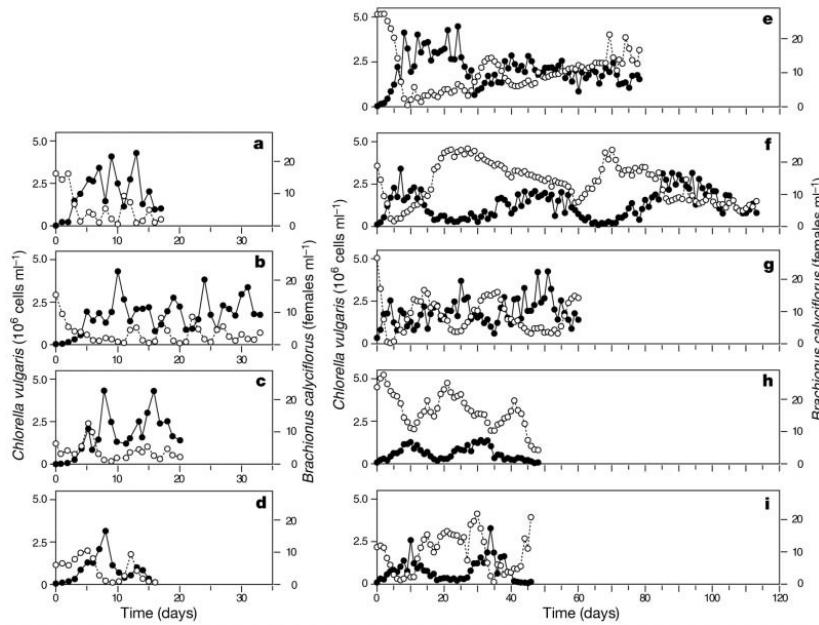


Figure 11.2: Rapid evolution drives ecological dynamics in a predator-prey system. Panels a-d denote rotifer-algal experiments with a single clone, such that the predator (solid, rotifer) has phase-shifted oscillations relative to the algal prey (dashed). In contrast, panels e-i show the dynamics of multi-clonal systems such that the total algal community is shown, albeit it is actually comprised of more than one algal type. In these, the oscillations tend to be anti-phase and not quarter-phase shifted as in classical theory. Reproduced from (Yoshida et al. 2003).

sampling. As is apparent in the left-panels, mixing rotifers and algae together leads to emergent oscillations, even though the chemostat is a continuous culture device and does not include exogenous changes in inflow or outflow. The experiments in panels (a-d) reveal oscillations, albeit short ones, in which the predator increase in abundance following peaks in prey abundance, leading to decreases in prey abundance, a decline in predators, and then a recovery of prey. In essence: classic Lotka-Volterra oscillations when combining a single predator with a single prey.

Yet, Figure 11.2's panels e-i reveal quite a different story. In these experiments the total algal population is plotted as prey vs. the rotifer, however the experiments themselves include multiple algal clones. Hence, rather than a single prey population, the algal population had intrinsic diversity. At the population scale, the dynamics include longer oscillations in which predator

and prey populations undergo “anti-phase” rather than phase-shifted oscillations. As was explained (and as we will explore in this chapter), this subtle signature is expected when evolutionary dynamics takes place on similar time scales to ecological dynamics. Such anti-phase relationships are simply impossible in canonical predator-prey dynamics; not only with the assumptions of Lotka and Volterra but with the typical modifications explored in the decades since. This is not just an example of an unusual exception. To the contrary, this subtle difference in the shape of cycles in fact common, occurring in many previously published consumer-resource dynamics datasets (see review in (Hiltunen et al. 2014)). Moreover, the emergence of such anti-phase cycles is possible when the frequency of clones changes over the same time scales as does the total population dynamics, i.e., when ecological and evolutionary dynamics are linked (Cortez and Ellner 2010). It was only through careful combination of mathematical modeling and experimental work that this new feature could supplant a prior paradigm.

Altogether, this chapter aims to address a central challenge in the modern study of populations and communities: the link between ecological and evolutionary dynamics. What are the key concepts that drive such changes? How can we start with simple models, extend them (in the right ways), and then connect mechanisms to experiments and observations? To do so, we must first introduce some classic models and their modern extensions, beginning from models preceding that of Lotka and Volterra – corresponding to simple growth in a finite environment.

11.2 CANONICAL MODELS OF POPULATION DYNAMICS

The construction of predator-prey dynamic models requires components that are then combined into representations of single predator-single prey system as well as more complex systems involving multiple types of predators and of prey. In doing so, these components of models are also relevant in the quantitative understanding of living systems, e.g., identifying universal growth curves for microbes or understanding competition from forest trees to fish. This section will focus on two components: (i) growth in a finite environment; (ii) competition between two populations of the same ‘trophic’ level. The former model corresponds to logistic growth dynamics, a context that is typically introduced in introductory classes as a means to highlight the saturating nature of growth. Here, we will use it to make the link between resource conditions and the expected levels of growth and saturation. The latter model is also critical to understanding predator-prey dynamics insofar as the prey population consistsens of multiple types (e.g., different algal clones as in Figure 11.2. As such, part of the challenge of understanding the ecology of predator interactions with diverse prey is to first assess the potential outcomes of interactions amongst the prey alone.

11.2.1 Logistic growth dynamics - from explicit to implicit dynamics

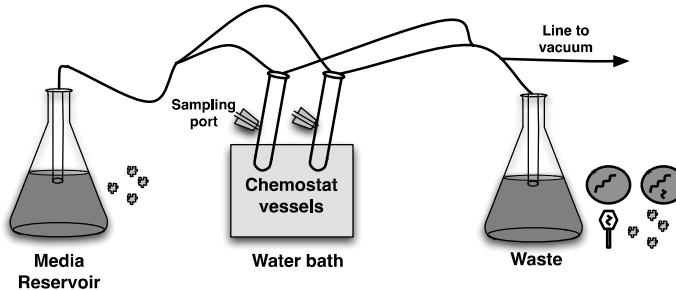


Figure 11.3: Schematic of a chemostat.

Populations grow exponentially, until they don't. The impact of the finite limits of space and resources can be accounted in extensions of exponential growth models. Perhaps the simplest is that the classic model of logistic growth

$$\frac{dN}{dt} = rN(1 - N/K), \quad (11.1)$$

where N is the abundance of a population, r is the maximum (or "Malthusian") growth rate, and K is the carrying capacity of the population. The use of implicit resource dynamics in modeling cellular growth is common. Yet, the particular form of logistic growth warrants examination as a particular limit of a mechanistic model of nutrient uptake and conversion into cell biomass. To explore this limit requires making the implicit explicit. To begin, consider a chemostat model in which a population of consumers, N , take up a non-reproducing resource with abundance R (see Figure 11.3):

$$\begin{aligned} \frac{dR}{dt} &= \omega J_0 - f(R)N - \omega R, \\ \frac{dN}{dt} &= \epsilon f(R)N - \omega N. \end{aligned} \quad (11.2)$$

Here, $f(R)$ is the functional response of the consumer, ϵ is the conversion efficiency of consumers, J_0 is the density of resources that is flowing into the chemostat, and ω is the washout rate, i.e., the rate at which both consumers and the resource flow out of the chemostat (see Figure 11.3).

This model of consumer-resource dynamics requires what is termed in the ecological literature a "functional response". The term functional response denotes the relationship between resource density and uptake for consumers – whether predators eating prey or herbivores consuming plants. To explore

one example, consider a consumer that moves “ballistically” through an environment with a velocity v . Moreover, assume the consumer can sense resources in a zone of radius r . Hence, the consumer will sense an area of approximate size $\pi r^2 + 2vT_s$ in a period T_s of searching. If resources are at a density R , then the potential number of encounters is $(\pi r^2 + 2vT_s)R$. Further if a fraction k of the resources are detected and of those f_c are consumed per unit time, then the total resource consumption is $(\pi r^2 + 2vT_s)k f_c R$ per consumer in a period of time T_s . This is true for all consumers, as long as they tend *not* to run into or near each other. Hence, the functional response should scale like

$$f(R) = cR$$

where the constant c approaches

$$\pi r^2 f_c k.$$

In practice, the velocity of the consumer ensures that over-consumption locally can lead the predator/consumer to a new resource patch, but in the limit of small sensing times does not markedly affect the scaling. This then completes the specification of this simple chemostat model:

$$\begin{aligned} \frac{dR}{dt} &= \omega J_0 - cRN - \omega R, \\ \frac{dN}{dt} &= \epsilon cRN - \omega N. \end{aligned} \tag{11.3}$$

This full model can be assessed as a two-dimensional coupled, nonlinear dynamical system. But, our aim here is slightly different.

To move from an explicit resource model to an implicit model we can make the assumption that resource dynamics in this system are much faster than changes in the population of consumers. If true, then we can assume that N is fixed. In other words, given a current population N , then we can apply a fast-slow assumption (as was utilized in the analysis of excitable dynamics earlier in this book). The equilibrium solution to the fast dynamics of the resource satisfies

$$\omega J_0 - cRN - \omega R = 0 \tag{11.4}$$

such that the resources should rapidly converge to

$$R^q(N) = \frac{\omega J_0}{\omega + cN} \tag{11.5}$$

where the superscript q denotes the fact that this represents a quasi-equilibrium of the consumer-resource system. As is apparent, resource concentration declines with an increasing level of consumers. Now that resource levels are implicitly defined in terms of consumer abundance, it is possible to rewrite the population dynamics strictly in terms of N

$$\frac{dN}{dt} = \frac{\omega \epsilon c J_0 N}{\omega + cN} - \omega N. \tag{11.6}$$

This assumption holds strictly in the limit that resource dynamics are much faster than consumer population dynamics, and otherwise should be recognized as an approximation. In the limit that $N(t) \ll \omega/c$, the dynamics can be further approximated as:

$$\frac{dN}{dt} = \overbrace{rN(1 - N/K)}^{\text{logistic growth}} - \omega N \quad (11.7)$$

where $K = \omega/c$ and $r = \epsilon c J_0$ (see Technical Appendices on the use of a Taylor approximation for this reduction). A similar approach may be taken with a Type-II response, such that $f(R) = \frac{\gamma R}{Q+R}$. In the limit that $Q \gg R$ then $f(R) = \gamma R/Q = cR$. In that case and in that limit $K = \omega Q/\gamma$ and $r = \epsilon \gamma J_0/Q$. It is important to keep in mind that the link between the mechanistic and phenomenological parameters only holds strictly in certain limits.

Nonetheless, let us examine the consequences of this slightly modified logistic model. Here, this one-dimensional dynamical system has two potential fixed points: $N^* = 0$ and $N^* = K(1 - \omega/r)$. These correspond to the extinction and persistence equilibria, respectively. As should be apparent, the density of the persistence equilibria is only positive when $r > \omega$. Hence, we maximum growth rates exceed the washout rate (here equivalent to local death), then a small population of consumers can grow to a large, steady state population of consumers. What is interesting here, is that by deriving the model from a resource-explicit uptake model, then one can identify the critical condition in this Type-II functional response model operating at saturation as:

$$\left(\frac{\epsilon \gamma J_0}{Q}\right) \times \frac{1}{\omega} > 1 \quad (11.8)$$

and for the Type-I model as:

$$(\epsilon c J_0) \times \frac{1}{\omega} > 1 \quad (11.9)$$

These may appear difficult to decipher. But, consider the following interpretation. Note that $1/\omega$ is the average residence time of the consumer in the chemostat. Whereas the first factor in parentheses corresponds to the division rate per time. Hence, this condition can be restated as: a small population of consumers will grow exponentially if their average number of new consumers produced in the lifetime of a consumer is greater than one. Does that resonate? It might, given that it is equivalent to the threshold condition or \mathcal{R}_0 in epidemic outbreaks, yet here the ‘outbreak’ is the spread of a consumer in an otherwise sterile environment. Further discussion of the basic reproduction number can be found in the subsequent chapter on outbreaks.

11.2.2 Consumer-resource models

There are two classic models of consumer-resource models attributed to Lotka and Volterra (Hastings 1997). One of these is the primary subject of this

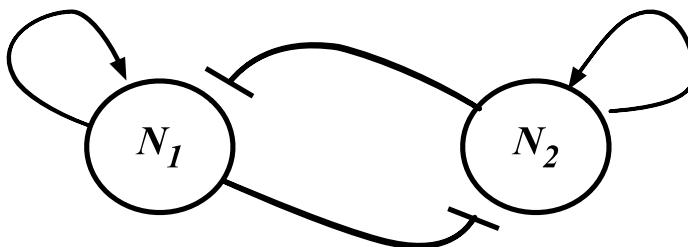


Figure 11.4: Competition between two species, including growth of each population due to intraspecific competition and inhibition of growth due to interspecific competition.

chapter: predator-prey dynamics. But to get there we need to assess the second: a model of competition for resources amongst two consumers, in which the resource is usually considered to be implicit (see Figure 11.4 for a schematic). Understanding the eco-evolutionary dynamics of predators and prey requires integrating both concepts together. In the LV model of competition, consumers are assumed to be at the same “trophic level”. The term trophic level denotes an equivalence in the kinds of foods that consumers may eat and is usually context-dependent when considering food webs (Cohen 1978). Putting aside complications of overlapping levels in food webs (or of the challenge of parasitism in food webs), the model can be assessed using the following dynamics:

$$\frac{dN_1}{dt} = r_1 N_1 \left(1 - \frac{N_1 + a_{21}N_2}{K_1}\right) \quad (11.10)$$

$$\frac{dN_2}{dt} = r_2 N_2 \left(1 - \frac{N_2 + a_{12}N_1}{K_2}\right) \quad (11.11)$$

Here the a_{12} and a_{21} coefficients denote the effect of the densities of each of the two populations on each other. Certain limits are apparent when we consider the case that a_{12} and a_{21} go towards 0. In this event, then each population acts effectively independently of the other. Hence, the two population each can grow to their carrying capacities, i.e., K_1 and K_2 , respectively. Moreover, if a_{12} or a_{21} becomes large when the other remains small, then dominant competitor can outcompete the other. The analysis of this system can be found in standard ecological texts (a brief review is presented in the Technical Appendices). There are potentially four equilibria in this system:

- Extinction: $(0, 0)$
- N_1 dominates: $(K_1, 0)$
- N_2 dominates: $(0, K_2)$
- Coexistence: (N_1^*, N_2^*)

Critically, the extinction point is always unstable as long as both $r_1 > 0$ or $r_2 > 0$. Experimentally, one can think of this condition as equivalent to the statement that a flask inoculated with bacteria will enable bacterial growth. Since we expect that both types can grow in the absence of competition, the real ecological question is whether or not the system ends up in a state dominated by a single competitor or in a coexistence state.

As explained in the Technical Appendices, the condition under which there is coexistence can be summarized as

$$K_1 < \frac{K_2}{\alpha_{21}} \quad (11.12)$$

$$K_2 < \frac{K_1}{\alpha_{12}} \quad (11.13)$$

which implies that intraspecific competition is stronger than interspecific competition. The rationale is that if either of the species are at their own capacity, then their effect remains less than the self-limitation term, and so each can, in effect, invade the other type. More generally, in the event that we assume neither species has a growth advantage, i.e., $K_1 = K = K_2$, then this condition is equivalent to assuming that $\alpha_{12} < 1$ and $\alpha_{21} < 1$. Insofar as there is weak competition then both types can persist. This generic finding of coexistence belies the fact that explicit models with a single resource often lead to the exclusion of one competitor. This theory of competitive exclusion (Armstrong and McGehee 1980) posits that there must be as many resources as consumers, at least insofar as the habitat is homogeneous with relatively simply functional forms for uptake. Here, weak competition implies that there is, in effect, more than one resource type that enables the coexistence. However, once a predator is added to the system, we will see that competition for resources is not the only way that a consumer can persist. Not dying (i.e., by increasing defenses against predators) is also a key trait and can enable new mechanisms of coexistence.

11.3 PREDATOR-PREY DYNAMICS

Predator-prey systems are, in effect, a kind of consumer-resource system albeit in which the resource can also reproduce. The following sections build up quantitative models of predator-prey dynamics, beginning with Lotka and Volterra's premise and approaching the types of interactions necessary to integrate ecological and evolutionary dynamics together. Note that the Technical Appendices includes additional details on the analysis and also includes a summary review of linearization of this class of nonlinear dynamical systems.

11.3.1 Classic models

To begin, consider the following classic model of predator-prey dynamics that were first introduced by Lotka (Lotka 1925) and Volterra (Volterra 1926) (see

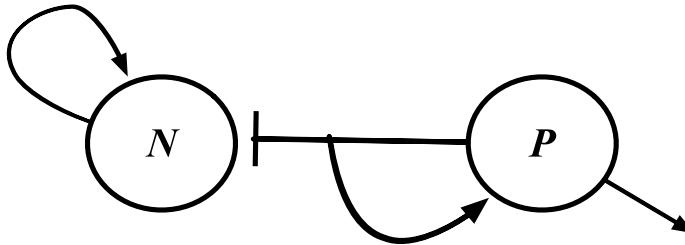


Figure 11.5: Predator-prey dynamics in which the prey grows in the absence of predators, predators consume prey leading to the growth of predators and predators experience a density-independent mortality.

sketch in Figure 11.5):

$$\frac{dH}{dt} = rH - cHP \quad (11.14)$$

$$\frac{dP}{dt} = \epsilon cHP - mP. \quad (11.15)$$

This is a model of predators, P , consuming prey, H in which predators would die exponentially in the absence of prey and prey would grow exponentially in the absence of predators. Note that the original model of Lotka and Volterra did not include the saturation of prey growth, e.g., a logistic like growth term that will be explored later in this chapter.

The original LV model of predator-prey dynamics leads to only two possible fixed points: $(0,0)$ and $(m/(\epsilon c), r/c)$. The former denotes the case where there are neither predator nor prey. The latter denotes the coexistence equilibria. As before, we can ask questions of the long-term dynamics by first investigating the stability properties of the internal equilibria. The Technical Appendices provides the details on the calculations which yield the following important result: the eigenvalues of the coexistence equilibrium have zero real part, i.e., are purely imaginary: $\lambda = \pm i\sqrt{m/r}$ where $i = \sqrt{-1}$. Now, this is not an imaginary system. Hence, the right (biological) way to interpret this finding is that an initial displacement will not grow nor decline. Instead a small deviation from the equilibrium will change like $u(t) \propto \cos(i\omega t)$ where $\omega = \sqrt{m/r}$ is the frequency of oscillation. Hence, just like a positive (negative) eigenvalue corresponds to exponential growth (decay), then a zero real component and non-zero imaginary component usually corresponds to neutral orbits.

That is precisely what happens. Without a logistic limitation then the dynamics including neutral oscillations, such that any initial condition (H_0, P_0) that is not at a boundary of the system nor at the unique interior equilibrium will continue to oscillate. Yet, such oscillations are not limit cycles in that they are not isolated periodic orbits. Indeed, there are an infinity of such orbits, associated with a conserved quantity. For example, by taking the ratio of the

prey and predator dynamics we find:

$$\frac{dH}{dP} = \frac{H}{(\epsilon cH - m)} \frac{(r - cP)}{P} \quad (11.16)$$

$$dH \frac{(\epsilon cH - m)}{H} = dP \frac{(r - cP)}{P} \quad (11.17)$$

$$(11.18)$$

such that after integration of both sides and re-arranging terms we find

$$\epsilon cH + cP = r \log P + m \log H + \text{const.} \quad (11.19)$$

What does this constant of motion mean? To physicists, such a constant of motion would be celebrated, as symmetries in systems usually lead to conserved quantities. Not so here.

In practice, the conserved quantity in the original formulation of the predator-prey dynamic model implies that given an initial value of (H_0, P_0) , the system will exhibit dynamics in a closed loop, on which Eq. (11.19) is satisfied. These are not limit cycles, but rather an infinite number of closed loops that retain their memory of the initial condition, forever. This is not good news for ecology, where the conserved quantity is a fragile feature of the particular choice of functions. One of the problems is that the model is over simplistic. Indeed, in the absence of predators, this model is unstable. Prey would grow exponentially – forever. That cannot be the case, and relaxing this assumptions is precisely the target of the next section. Of note, the finding of an infinite number of closed orbits is also consistent with the local stability analysis of the internal coexistence equilibrium.

Yet there is something to be learned from these dynamics as seen in Figure 11.6. The closed loops have an orientation, they run counterclockwise. This counter-clockwise feature is evident in the time-series in the bottom panels of the same figure. For example, beginning at the maximum prey density in any of the closed loops (in the top panel), then the dynamics move ‘up’ in the plane as predator thrive given high levels of prey. Given maximal predator densities, then prey densities decline. With prey densities declining (the left-most point) then so do predators. With fewer predators (the bottom-most point in the loop), the prey recover. Yet with prey recovery, the predators also thrive and the cycle repeats. Again, this constant of motion is fragile, it only applies when there is no logistic growth, but the relative ordering of the peaks and troughs does have a message that goes beyond this particular choice of functional form.

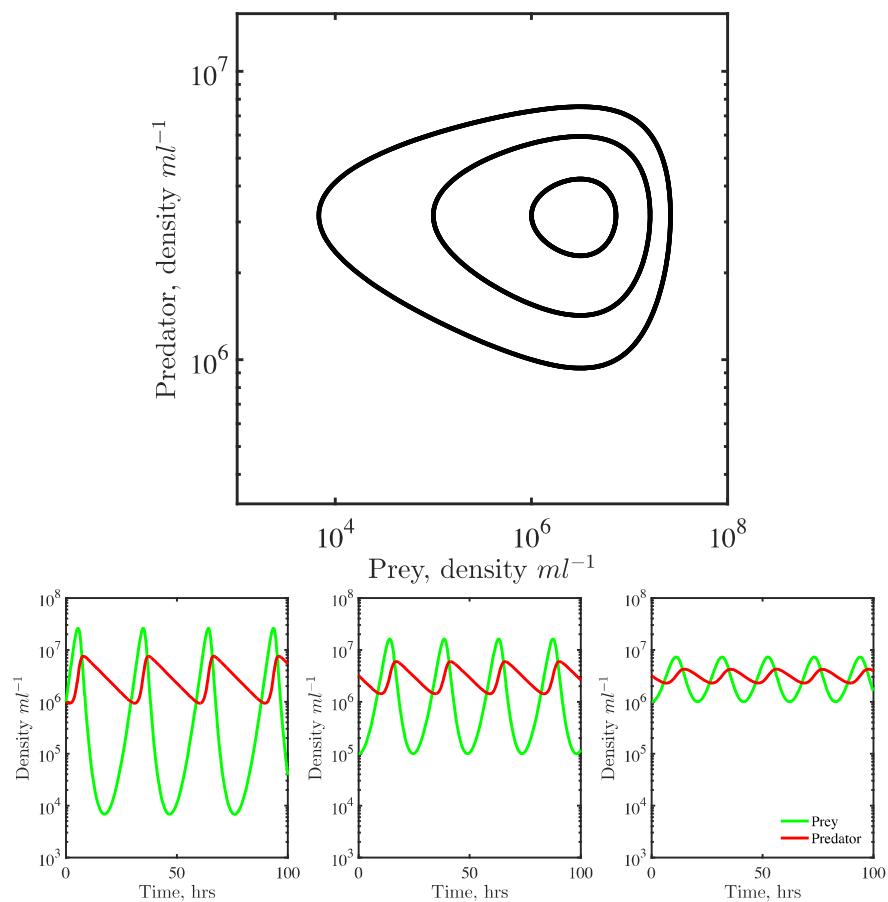


Figure 11.6: Predator-prey dynamics given the Lotka-Volterra model. (Top) Phase-plane dynamics where oscillations are counter-clockwise. (Bottom) Time dynamics, showing the quarter-phase shift of the peaks of predator (red) and prey (green).

11.3.2 Predator-Prey Dynamics with Limitations on Prey Growth

Here, let us once again revisit predator-prey dynamics by extending the original model to include density limitation in the growth of prey:

$$\frac{dH}{dt} = rH(1 - H/K) - cHP \quad (11.20)$$

$$\frac{dP}{dt} = \epsilon cHP - mP. \quad (11.21)$$

Given this new formulation there are now three fixed points of the system, rather than two:

- Extinction: $(0, 0)$
- Prey only: $(K, 0)$
- Coexistence: $(\frac{m}{\epsilon c}, \frac{r}{c} \left(1 - \frac{m}{\epsilon c K}\right))$

In this model, coexistence is possible whenever predators can increase in number given that prey are at their maximum, i.e., $\epsilon c K > m$. There is yet another interpretation, in that predators will live on average $1/m$ units of time. In that time, they will produce $\epsilon c K$ offspring per unit time. Hence, predators will proliferate if their average offspring exceeds 1 given the addition of a single predator into an otherwise prey-only system. This definition is termed the “basic reproduction number”, a concept that we will revisit in the epidemiological dynamics chapter. More details on the stability of this system can be found in the Technical Appendices accompanying this chapter. Finally, it is worth mentioning that the dynamics of predator and prey involve endogenous oscillations, whether permanently (as in the model without a carrying capacity) or transiently (as in the model with a carrying capacity).

Figure 11.7 reveals that the inclusion of density limitation fundamentally shifts the nature of the dynamics from persistent neutral orbits to spirals or arcs that converge to a fixed point. The spirals and arcs have the right ‘chirality’, in the sense that the dynamics appear counterclockwise in the phase plane, however given the parameters in this example, the oscillations are rapidly damped and the system converges to the stable equilibrium. Note that if predators exhibit a “Type-2” functional response, then the system can exhibit a limit cycle in which predator and prey oscillate together in a unique orbit, an issue explored next.

11.3.3 Predator-Prey Dynamics with Limitations on Prey Growth and Saturating Predation

The prior models both assume that the consumption of prey by predators is limited only by prey availability. In other words, if H increases, then predators can keep eating and eating and eating. Consumption takes time. For example, consider the case where predators can consume at most 1 prey in a time τ . In that case, the maximum rate of per-capita consumption should be $1/\tau$. Yet, when prey are scarce, then the rate of consumption will be limited by prey availability and not handling time. These dualing limits can be captured in the following model:

$$\frac{dH}{dt} = rH(1 - H/K) - \frac{cH}{1 + H/H_0}P \quad (11.22)$$

$$\frac{dP}{dt} = \frac{\epsilon c H}{1 + H/H_0}P - mP \quad (11.23)$$

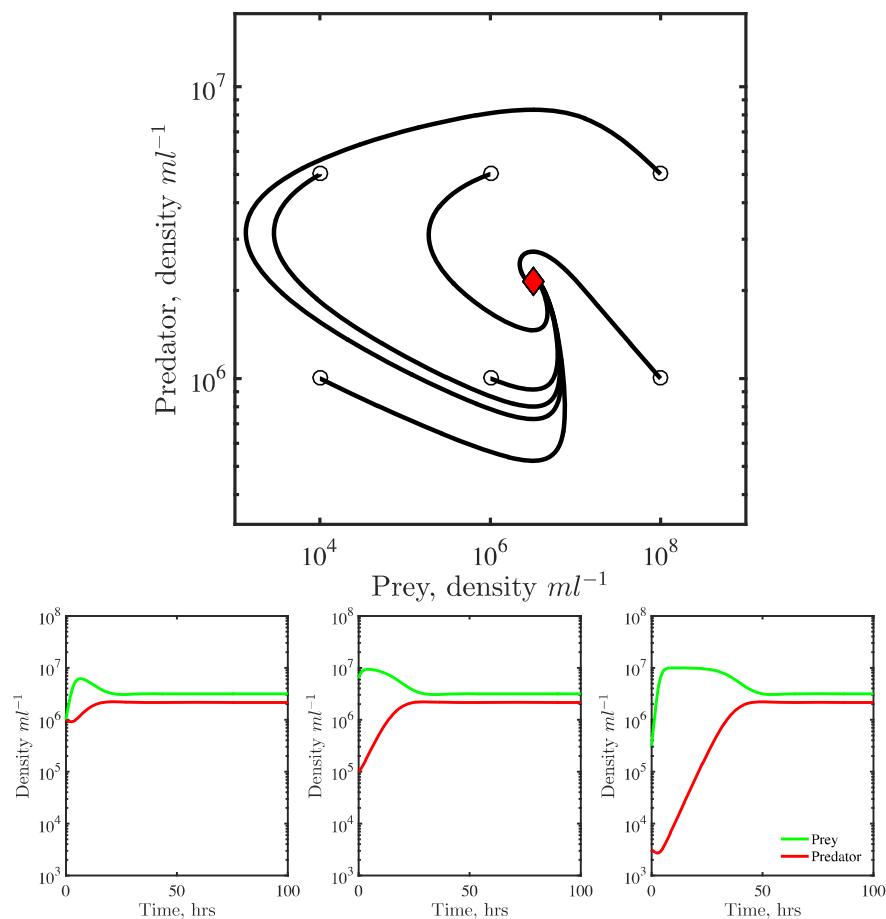


Figure 11.7: Predator-prey dynamics given the Lotka-Volterra model given logistic growth of prey in the absence of predators. (Top) Phase-plane dynamics where oscillations are counter-clockwise. (Bottom) Time dynamics, showing the quarter-phase shift of the peaks of predator (red) and prey (green). Parameters: $r = 1$, $c = 10^{-6.5}$, $\epsilon = 0.1$, $m = 0.1$, all in units of cells, mL, and days.

This is known as the MacArthur-Rosenzweig model (Rosenzweig and MacArthur 1963), and includes a ‘Type-II’ functional response, i.e., a saturating prey consumption function. Note that when $H \gg H_0$, then $cH/(1+H/H_0) \rightarrow cH_0 \equiv 1/\tau$.

Hence, another way to write this model is as follows:

$$\frac{dH}{dt} = rH(1 - H/K) - \frac{cH}{1 + c\tau H}P \quad (11.24)$$

$$\frac{dP}{dt} = \frac{\epsilon cH}{1 + c\tau H}P - mP \quad (11.25)$$

where the handling time dependence is now explicit. As a result there are once again three fixed points:

- Extinction: $(0, 0)$
- Prey only: $(K, 0)$
- Coexistence: H^*, P^*

where $\epsilon cH^* = 1 + H^*/H_0$ or $H^* = \frac{H_0}{H_0\epsilon c - 1}$ and $P^* = \epsilon r/mH^*(1 - H^*/K)$. This model reveals a new phenomena: stable limit cycles (see Figure 11.8). In this regime, the prey and predator continue to oscillate, but unlike in the original LV model, these oscillations do not have an infinite memory of the initial conditions. Instead, the oscillations either increase or dampen and then approach a stable limit cycle (see the phase plane image in Figure 11.8 for more detail). This implies that efforts to identify the signature of predator-prey interactions in natural systems should look for phase shifts between the peaks of predators which should follow those prey. How true this is, forms the core point of inquiry of this chapter, and the synthesis of ecological and evolutionary dynamics.

11.4 TOWARDS PREDATOR-PREY DYNAMICS WITH RAPID EVOLUTION

The prior sections have included a suite of models spanning the growth of a single population limited by resources, competition between populations of the same trophic level (i.e., interspecific competition), and finally predator-prey dynamics assuming one predator and one prey. These ingredients are precisely what is needed to take the next step and ask: what new kinds of ecological phenomena are possible when evolutionary dynamics occur rapidly. By evolutionary dynamics, we don't necessarily need to invoke *de novo* mutation. Instead, evolutionary dynamics here means that there is a change in the frequency of genotypes in a population. That condition is certainly met if a prey population is comprised of multiple clones – and as we shall see, the existence of clones or distinct strains that constitute subpopulations is precisely the driver of a potential fundamental shift in the shape of predator-prey cycles.

11.4.1 Multiple prey, and a single predator

There are multiple theories of eco-evolutionary dynamics. Yet, perhaps the most straightforward way to think about this link is to revisit the definition of

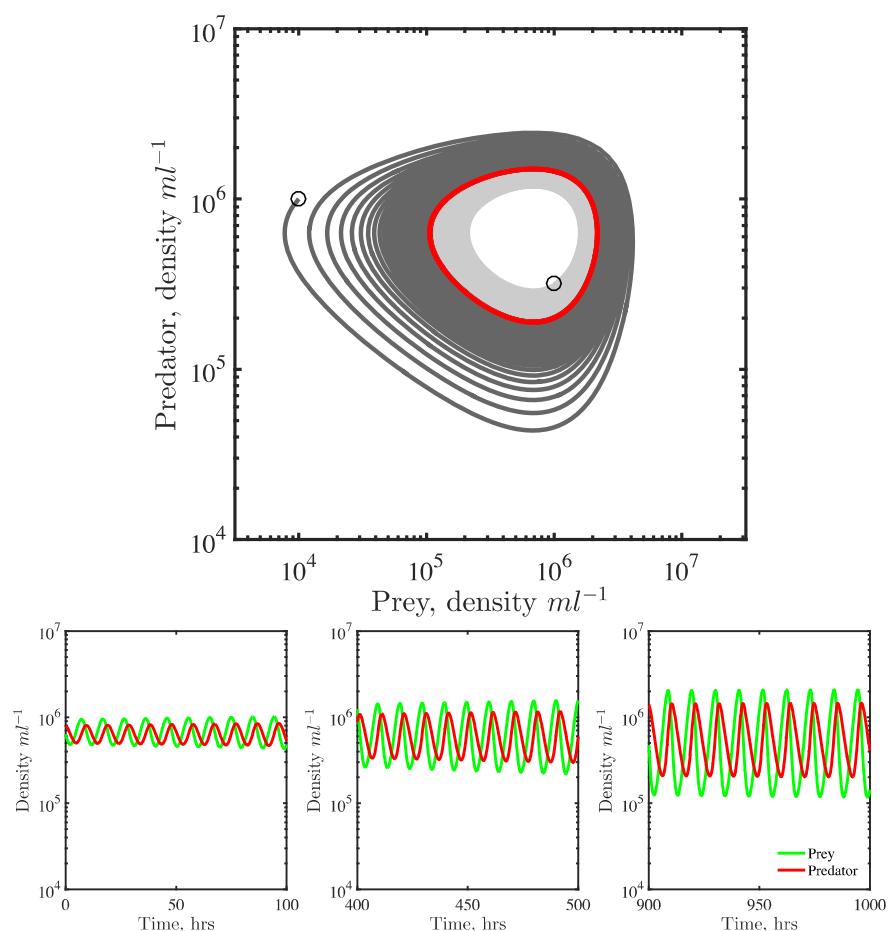


Figure 11.8: Predator-prey dynamics given the Lotka-Volterra model with prey saturation and predator handling of prey. (Top) Phase-plane dynamics where oscillations are counter-clockwise. Note that two trajectories are shown (dark and light grey) converging to a stable limit cycle (red line). (Bottom) Time dynamics, showing the quarter-phase shift of the peaks of predator (red) and prey (green) approaching a limit cycle. Parameters: $r = 1$, $c = 10^{-6.5}$, $K = 10^7$, $\epsilon = 0.1$, $m = 0.1$, all in units of cells, mL, and days.

evolution: “the heritable change in genotype frequencies from one generation to the next.” Hence, envision a prey population consisting of multiple genotypes of prey (i.e., distinct “clones”). It might not be possible to distinguish those clones based on the observational methods commonly used, even if those clones had distinct phenotypes. Moreover, consider a case where two clones had distinct

growth and palatabilities such that dynamics of the entire (unobserved) system was:

$$\frac{dH_1}{dt} = r_1 H_1 \left(1 - \frac{H_1 + a_{12} H_2}{K_1}\right) - \frac{c_1 H_1 P}{(1 + c_1 H_1 + c_2 H_2)} \quad (11.26)$$

$$\frac{dH_2}{dt} = r_2 H_2 \left(1 - \frac{H_2 + a_{21} H_1}{K_2}\right) - \frac{c_2 H_2 P}{(1 + c_1 H_1 + c_2 H_2)} \quad (11.27)$$

$$\frac{dP}{dt} = \left(\frac{\epsilon c_1 H_1 + \epsilon c_2 H_2}{1 + c_1 H_1 + c_2 H_2}\right) P - mP \quad (11.28)$$

However, if H_1 and H_2 are indistinguishable, then it is not evident what kind of dynamics might be observable in the $P - H$ phase plane. What can happen is something new: that the ratio of genotypes changes on the same time scale as changes in the total population of consumers. Hence, when the 3-dimensional system is projected back onto 2-dimensions, then new kinds of dynamics can occur, including antiphase cycles as shown in Figure 11.2.

To understand how such antiphase cycles might arise, consider cases where there is a large cost of prey defense given increases in growth rate (i.e., a growth-defense tradeoff). In that case, the increase in predator density selects for defense specialists which leads to suppressed abundances of prey. This leads to the fall-off of predators and concurrent rise of prey, including growth specialists. When prey at their peak and predators at their minimum, the dominance of growth specialists leads to the rapid return of predators and the decline of prey (as well as a switch back from growth to defense specialists). This rapid change on genotype frequency brings with it changes in population densities, i.e., the sum of H_1 and H_2 . This can appear like antiphase or even cryptic cycles depending on the magnitude of trade-offs.

The dynamics in Figure 11.9 illustrate this point, at least in part. This model includes two prey, one well-defended (that grows slower) and one that grows faster (but is easier to consume). The dynamics favor the fast-growing prey when predators are rare and the well-defended prey when predators are common. Yet, precisely because of the trade-off there is a switch in relative densities. When well-defended prey are abundant, this drives down predator densities which, eventually, leads to the rise and return of fast-growing prey. In turn, the increase of fast-growing prey provide a resource for predators to grow in abundance. What is critical is that the changes in total population densities are occurring *on the same timescale* as are changes in genotype frequencies. The phase plane dynamics (in the top panel) also reveal another feature: the crossing of phase lines. Because this is a deterministic model, it should not be possible to have phase lines cross in a two-dimensional dynamical system, precisely because the system should move in the same direction whenever it returns to the same point in phase space. Not so here. Instead, the phase line crosses itself – an indicator that the system is comprised of more than just two components. The phase plane is a projection of the 3D system onto 2D: total prey and predator densities. Here the dynamics seem anti-phase, i.e., when the

prey at their maximum, predators are at their minimum and vice-versa. This is possible precisely the genotype composition differences at the peaks and troughs of prey densities.

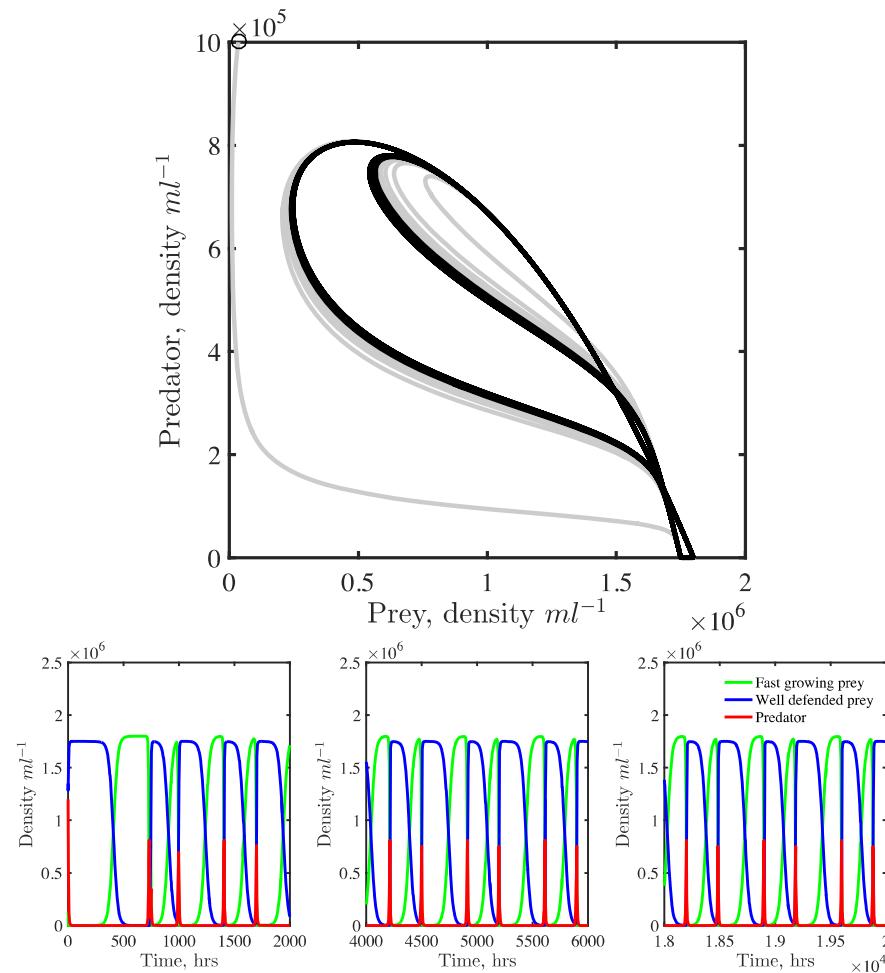


Figure 11.9: Predator-prey dynamics given the Lotka-Volterra model given logistic growth of prey in the absence of predators. (Top) Phase-plane dynamics where oscillations are counter-clockwise from the first half of the simulation (in gray) and the latter half (in black). (Bottom) Time dynamics, showing the dynamics of the fast-growing prey (green), well-defended prey (blue), and predator (red).

11.4.2 Multiple prey and multiple hosts

The dynamics above consider what happens when there are two prey and one predator, and when prey genotype frequencies change on the same timescale as total population dynamics. This rapid evolution can also extend to more than one predator. To see how, consider a model extension to the case of two predators and two prey, in which one prey is relatively well defended against both and the other grows faster. Likewise, consider the case where one predator is relatively the better consumer of both prey and yet also dies faster in the absence of prey (i.e., has a higher baseline energetic need). As a consequence, this entire set of predator and prey can coexist. Such a result may be hard to identify, in theory, but it is not surprising in practice. Complex communities in nature abound. Yet, what is surprising is the potential consequence for the qualitative patterns of total prey and predator density given the interaction of multiple clones.

The dynamics in Figure 11.10 illustrate the potential paradox (Cortez and Weitz 2014). When viewed in terms of the total number of prey and predators, it appears that the prey abundances peaks *after* that of predator. In essence, prey appear to eat predators, and not the other way around. Such a result is non-sensical, so how else do we interpret these seemingly contradictory findings. The answer lies in the phase-plane, where trajectories do in fact move in reverse, such that predator peaks are followed by prey peaks which are followed by predator troughs then prey troughs and finally predator peaks again. Yet in the process, the relative importance of predator and prey genotypes also change. These changes are denoted by the colored bars at multiple points along the clockwise phase plane trajectory.

The dynamics can be explained as follows, when prey abundances are high, this coincides with the state in which prey are not particularly vulnerable and the system has a balance of predator types. However, precisely because predator abundances are low, then prey can increase in number and indeed the prey type that is more vulnerable (but does not pay the cost for defense) can increase. Likewise, in order to attack the limited prey, then predators that are better in offense outcompete the other genotype. Therefore the high offense predators proliferate on the highly vulnerable prey. As predators increase, then the prey do not have advantages when they incur costly defensive strategies (there are simply too many high offense predators). Instead the prey switch to low offense types. As such, predators need not utilize high offense strategies. This shift in genotypes enables low vulnerability (and fast-growing) prey to proliferate during a period where predators are dominated by low offense types. At this point the population dynamics return to a high prey, low-predator dynamic, albeit having gone through a **clockwise** cycle in the phase plane. Such dynamics are simply not possible with a conventional predator-prey model (insofar as the functional response is a monotonically increasing function of the prey density). This analysis reveals the critical importance of how rapid evolutionary dynamics can lead to qualitative changes in population dynamics.

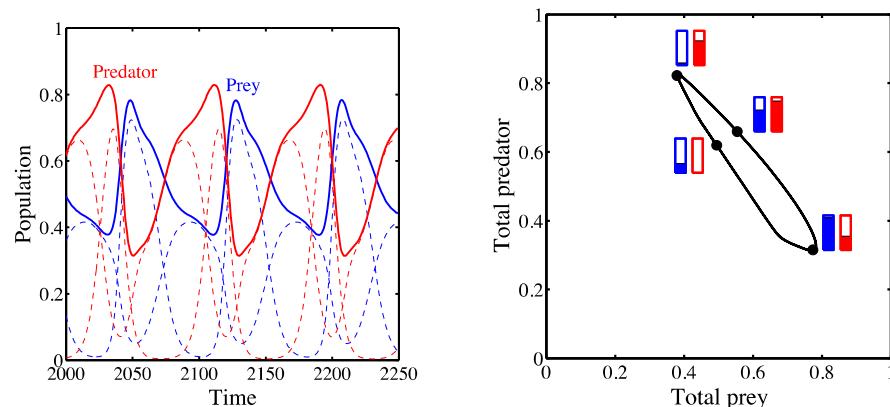


Figure 11.10: Predator-prey dynamics given a two-prey and two-predator model. (Left) Strain level dynamics and total population dynamics with time; (Right) Phase plane dynamics. Black curve denotes population dynamics whereas the bars denote the relative frequency of genotypes (blue, prey, and red predator). For prey, the shaded bar denotes low vulnerability and open bar denotes high vulnerability. For predators, the shaded bar denotes low offense, and the open bar denotes high offense.

Remarkably, such reversed cycles have been found in multiple datasets, including bacteriophage (acting as a predator) and *Vibrio cholerae* (the prey), mink (predator) and muskrat (prey) and gyrfalcon (predator) and rock ptarmigan (prey) systems (Cortez and Weitz 2014). Figure 11.11 shows five such examples in time (left panels) and in the phase plane (right panels). In each, the orientation is clockwise, rather than counter-clockwise. Of course, that may suggest that prey eat their predators (they do not), but instead a different interpretation is that these dynamics are hallmarks of the dynamic variation of subpopulations of predators and prey – projecting down (at least) a four-dimensional dynamical system into a two-dimensional plane. Notably, reverse cycles are not a potential outcome of cases where only the predator or the prey evolve. Instead, the existence of reverse cycles suggests coevolution’s impact on ecology. Indeed, in the case of the phage-bacteria system, there is additional evidence to suggest the importance of this multiclonal mechanism.

In the case of an experimental study of *V. cholerae* O1 and the bacteriophage JSF4, the study was conducted over a nearly month period with daily sampling (Wei et al. 2011). The time series were considered complex, and not necessarily consistent with conventional expectations of predator-prey dynamics. As considered elsewhere (Levin et al. 1977; Weitz 2015), virulent bacteriophage that exclusively infect host cells are expected to induce predator-prey like dynamics for precisely the same mechanism as explored in this chapter. Yet, in this case, the

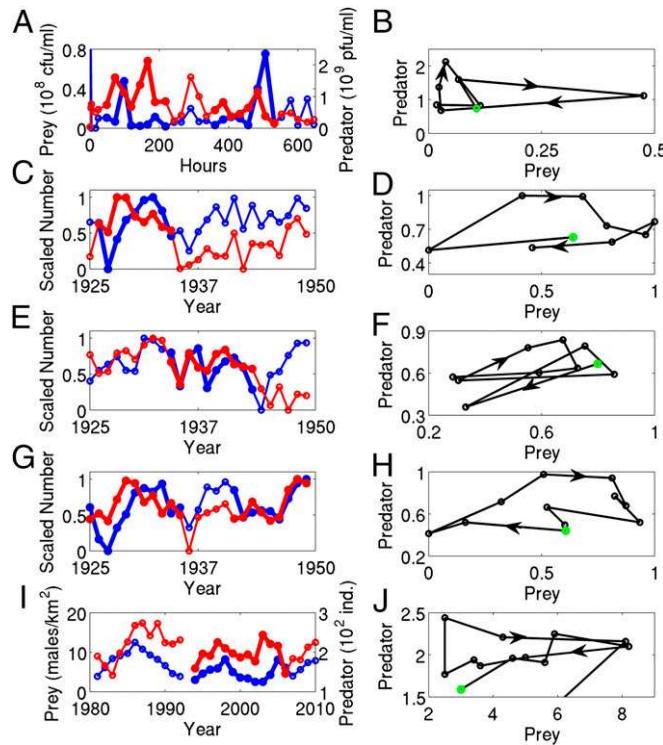


Figure 11.11: Clockwise predator-prey dynamics for *Vibrio cholerae* (prey) (A-B), mink (predator) and muskrat (prey) (C-D; E-F; G-H) and gyrfalcon (predator) and rock ptarmigan (I-J). Reproduced from (Cortez and Weitz 2014).

experimental team plated out multiple hosts and multiple phage strains from the chemostat experiment. In doing so, they found two key phage variants, what they termed phage T and phage B for ‘turbid’ and ‘big’ plaques, analogous to a less efficient and more efficient predator, given that the turbidity and size of plaques is a metric of efficient replication on the WT hosts. Likewise, the team also isolated multiple hosts, that they termed T^- and TB^- denoting largely resistant to the T phage and to both T and B phage, respectively. Rapid coevolution of viruses and bacteria in lab settings is a hallmark of the large population sizes; yet here the impact extends beyond insights into how evolution unfolds in a fixed ecological setting. Together, these findings suggest that evolution and ecology are entangled, mutually influencing a complex, joint dynamics.

11.5 TAKE-HOME MESSAGES

- The motivation for this chapter was a simple observation of anti-phase cycles between predator and prey. in a rotifer-algae system.
- Anti-phase cycles should not be possible in conventional predator-prey dynamics, instead, predator-prey models including those with and without density limitation exhibit phase-shifted cycles where prey peaks, and then predators, then prey reach their trough, then predators decline, and then finally prey recover and the cycle begins again.
- The particular outcomes of single predator-single prey population dynamics is influenced by functional responses.
- Attracting limit cycles are possible given density limitation of prey and Type II functional responses by the predator.
- In the event of a multi-clonal prey population then predator-prey dynamics can exhibit anti-phase and even cryptic dynamics (where one population appears fixed and the other changes with time).
- In the event of a multi-clonal prey and predator population then reverse cycles can appear – this does not imply that prey eat predators, but rather that the frequency of both genotypes are changing at the same time scale as ecological dynamics.
- Altogether, the chapter has provided a nested series of examples showing the impacts of rapid evolution on both ecological and evolutionary dynamics.

11.6 HOMEWORK PROBLEMS

These problems leverage the methodology in the accompanying computational laboratory. This particular laboratory has two distinct paths - that function as distinct modules. First, the laboratory treats the problem of the origin of functional responses, and how to move from an individual based model to an effective functional response to use at the population scale. Second, the laboratory covers multi-clonal dynamics, including the projection of dynamics from higher to lower dimensional systems.