Differential Systems for Biology

Prepared for First-Year Master's Students

Master's Program in Dynamical Systems

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Main Topics

Modeling via Differential Equations

Infectious Diseases

Predator–Prey Systems

Competitive Species

This lesson is presented for first-year Master's students in Dynamical Systems. It introduces the basic principles of modeling via differential equations and develops three major biological applications: infectious disease models, predator–prey systems, and competition between species. The emphasis is on assumptions, model construction, equilibrium analysis, qualitative interpretation, and mathematical reasoning.

Prepared as a teaching document in English

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Contents

1. Introduction

This lesson is meant for first-year Master's students in Dynamical Systems. It serves as an introduction, but still keeps things rigorous, showing how differential systems come into play in biology. Differential equations are basically the go-to mathematical tools for describing change. Whenever something changes over time and you can write how it changes with derivatives, differential equations give you a natural and powerful way to model it. In biology, that means we get to see how populations grow, how diseases spread, how predators and prey chase each other around, or how two species scrap for the same resources.

The lesson has two big teaching goals. First, to help students go from everyday assumptions to an actual mathematical model. Second, to show that even when you can't solve a system exactly, you can still learn a lot about how it behaves just by looking at its overall structure.

Here's how the material is organized:

1. Kicking things off with modeling—how to use differential equations to capture real-world stories.
2. Studying infectious diseases with the SIR and SIRS models.
3. Exploring predator-prey dynamics, starting with the classic Volterra-Lotka model, then moving on to scenarios where growth hits limits.
4. Looking at competition between species, using broad qualitative ideas rather than specific formulas.

Learning outcomes

By the end of this lesson, you should be able to:

- Explain what the variables, parameters, and assumptions mean in a model.
- Build a differential equation or system based on biological ideas.
- Find equilibrium points and interpret what they mean in biological terms.
- Analyze nullclines and invariant regions in systems with two variables.
- Tell the difference between conclusions that only hold locally and bigger, global results in mathematical biology.
- Compare the biological significance of steady states (like disease-free or endemic situations), coexistence versus extinction cases, and even recurring cycles.

2. Modeling via Differential Equations

2.1. What is a mathematical model?

A mathematical model does not pretend to be a perfect snapshot of reality. Instead, it organizes the parts of a real world phenomenon that we actually care about. A model is not just about regurgitating data it helps us understand how things work, make predictions, and see what assumptions really matter.

The trick is finding the sweet spot between two things: keeping the model simple enough to work with, but also making sure it captures the important details of whatever you are studying.

A useful model must balance two demands

- it must be simple enough to analyze;
- it must remain faithful to the essential features of the phenomenon.

In mathematical biology, a model usually starts from verbal assumptions, such as:

- the rate of growth of a population is proportional to the population itself;
- infection occurs through encounters between susceptible and infected individuals;
- predator births depend on prey consumption;
- competition reduces the growth rate of each species.

The art of modeling lies in translating such statements into mathematical equations.

2.2. The standard modeling procedure

A standard modeling process can be summarized in three steps.

Three-step modeling strategy

Step 1: State the assumptions clearly. What biological mechanism is assumed to be active? What is neglected?
Step 2: Define variables and parameters. What depends on time? Which constants measure biological effects?
Step 3: Derive the equations. Translate “rate of change”, “proportional to”, “increases with”, or “decreases with” into mathematical relations.

These three steps are simple to state but often difficult to execute well. In practice, a good model requires repeated revision and comparison with biological intuition or experimental data.

2.3. Variables, parameters, and initial conditions

In most biological applications:

- the independent variable is time t;
- the dependent variables are populations or densities, such as $P(t)$, $S(t)$, $I(t)$, $R(t)$, $x(t)$, or $y(t)$;
- the parameters are positive constants such as growth rates, death rates, interaction coefficients, transmission rates, or carrying capacities.

The initial condition specifies the state of the system at the starting time, for example

$$P(0) = P_0, \qquad S(0) = S_0, \qquad I(0) = I_0.$$

It is the initial condition that determines which particular solution we are studying.

2.4. A first example: unlimited population growth

Suppose a population grows under the assumption that the rate of growth is proportional to the size of the population. Let $P(t)$ denote the population size. Then

$$\frac{dP}{dt} = kP,$$

where $k > 0$ is the growth-rate coefficient.

This equation is simple, but it already illustrates the modeling principles.

- The quantity P is the dependent variable.
- The parameter k measures reproductive intensity.
- The derivative dP/dt expresses the rate of change.

The solution of this initial value problem,

$$\frac{dP}{dt} = kP, \qquad P(0) = P_0,$$

is

$$P(t) = P_0 e^{kt}.$$

Thus the model predicts exponential growth. Biologically, this may be reasonable only for small populations in an environment with abundant resources.

Interpretation

The unlimited growth model is appropriate over short time intervals or in early stages of colonization. It is not appropriate when food, space, or other ecological constraints become important.

2.5. Equilibrium solutions and long-term behavior

An equilibrium solution is a constant solution. For the equation

$$\frac{dP}{dt} = kP,$$

we have $P(t) \equiv 0$ as an equilibrium. The sign of the derivative tells us whether solutions increase or decrease:

- if $P > 0$ and $k > 0$, then $dP/dt > 0$ and the population increases;
- if $P < 0$, the mathematical solution decreases further, although negative populations have no biological meaning.

The important lesson is that qualitative information can often be extracted before solving explicitly.

2.6. The logistic population model

The exponential model ignores crowding and limited resources. To correct this, we assume:

1. if the population is small, growth is approximately proportional to population size;
2. if the population is too large, the growth rate becomes negative.

These assumptions lead to the logistic equation:

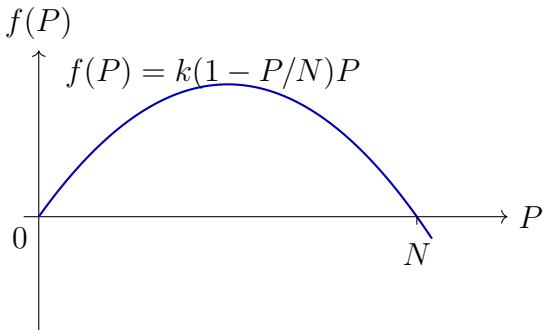
$$\frac{dP}{dt} = k\left(1 - \frac{P}{N}\right)P,$$

where $N > 0$ is the carrying capacity.

This equation has two equilibria:

$$P = 0 \qquad \text{and} \qquad P = N.$$

If $0 < P < N$, then $dP/dt > 0$, so the population increases. If $P > N$, then $dP/dt < 0$, so the population decreases. Hence positive solutions tend toward N.



The logistic model is one of the most important basic models in mathematical biology because it shows how a simple nonlinear term can change the long-term behavior from unbounded growth to saturation.

2.7. Qualitative analysis before explicit solving

In many nonlinear equations, the exact solution formula is less important than the structure of the vector field. For the logistic model, the sign of

$$f(P) = k\left(1 - \frac{P}{N}\right)P$$

provides immediate information:

- $P = 0$ is an unstable equilibrium for positive populations;
- $P = N$ is a stable equilibrium;
- every biologically meaningful solution tends to the carrying capacity.

This is a central methodological point: in applications, long-term qualitative behavior often matters more than exact formulas.

2.8. Predator–prey modeling as a first system

The next conceptual step is to study systems with more than one dependent variable. Suppose $R(t)$ is the prey population and $F(t)$ is the predator population. Under classical assumptions:

- prey reproduce at a rate proportional to prey abundance in the absence of predators;
- predators die out in the absence of prey;
- encounters between prey and predators are proportional to the product RF;
- prey consumption decreases prey and increases predator growth.

The resulting system is

$$\frac{dR}{dt} = \alpha R - \beta RF, \qquad \frac{dF}{dt} = -\gamma F + \delta RF.$$

This is a coupled first-order system. It already shows why systems are richer than single equations: the rate of change of each variable depends on both populations.

Why the product term RF?

The interaction term RF is the simplest algebraic expression that vanishes if either species is absent and increases when either population increases. It is therefore the most natural first approximation for encounter-based interaction.

2.9. Three complementary approaches

The modern study of differential equations uses three complementary approaches:

1. **Analytic approach:** derive explicit formulas if possible.
2. **Qualitative approach:** study equilibria, signs, nullclines, stability, and phase portraits.
3. **Numerical approach:** use computation when formulas are unavailable.

For biological systems, the qualitative approach is often the most informative because explicit formulas are rarely available for realistic models.

2.10. Pedagogical conclusion of the modeling section

At this stage, students should retain the following principles:

- every model begins with assumptions, not equations;
- parameters have biological meaning and should be interpreted;
- equilibrium analysis is often the first step in understanding the model;
- nonlinear terms typically encode important biological effects such as crowding, contagion, or competition;
- the biological interpretation of a solution is as important as its mathematical form.

3. Infectious Diseases

3.1. Biological motivation

Communicable diseases spread through contact between susceptible and infected individuals. A mathematical model should therefore distinguish at least between people who can become infected and people who are currently infected. In many diseases, a third group is important: people who have recovered and acquired immunity.

This leads to compartment models, where the total population is divided into classes. The most classical one is the SIR model.

3.2. The SIR model

Let:

- $S(t)$ be the number of susceptible individuals;
- $I(t)$ be the number of infected individuals;
- $R(t)$ be the number of recovered individuals.

The standard assumptions are:

1. the total population is constant;
2. infection is transmitted through encounters between susceptible and infected individuals;
3. recovery occurs at a rate proportional to the infected population;
4. recovered individuals do not become infected again.

The model is

$$S' = -\beta SI, \qquad I' = \beta SI - \nu I, \qquad R' = \nu I,$$

where $\beta > 0$ is the transmission rate and $\nu > 0$ is the recovery rate.

Because

$$(S + I + R)' = 0,$$

the total population is constant. Therefore the three-dimensional system effectively reduces to a two-dimensional one once we know S and I.

3.3. Reduction to a planar system

Since R can be recovered from the conservation law, it is enough to study

$$S' = -\beta SI, \qquad I' = \beta SI - \nu I.$$

This system is simpler to analyze geometrically. The biologically meaningful region is

$$S \geq 0, \qquad I \geq 0.$$

The equilibria are all points on the S-axis, because $I = 0$ implies

$$S' = 0, \qquad I' = 0.$$

So every disease-free state is an equilibrium.

3.4. Nullclines and threshold phenomenon

The nullclines are:

- $S' = 0$ when $S = 0$ or $I = 0$;
- $I' = 0$ when $I = 0$ or $S = \nu/\beta$.

Thus the vertical line

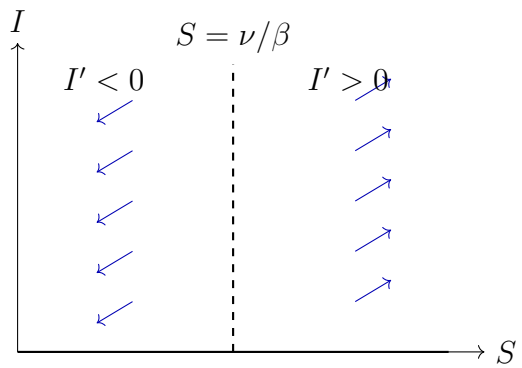
$$S = \frac{\nu}{\beta}$$

plays a decisive role. It is the threshold line.

If $S > \nu/\beta$, then

$$I' = I(\beta S - \nu) > 0,$$

so the number of infected individuals increases. If $S < \nu/\beta$, then $I' < 0$ and the number of infected individuals decreases. Therefore, the epidemic initially grows only if the susceptible population is sufficiently large.



3.5. A first integral and the geometry of solutions

For the SIR system, dividing I' by S' gives

$$\frac{dI}{dS} = \frac{I'}{S'} = -1 + \frac{\nu}{\beta S}.$$

Integrating with respect to S yields

$$I + S - \frac{\nu}{\beta} \log S = \text{constant}.$$

This means that solution curves lie on level sets of the function

$$H(S, I) = I + S - \frac{\nu}{\beta} \log S.$$

Geometrically, this gives the phase portrait in the (S, I)-plane. Biologically, it explains the typical epidemic trajectory:

- susceptible individuals decrease monotonically;
- infections first rise if S is above threshold;
- later infections decline as susceptibility falls below threshold.

3.6. Biological interpretation of the SIR model

The SIR model does not predict sustained oscillations or endemic infection in its simplest form. Rather, it describes an epidemic wave. The disease rises, peaks, and then fades out because the pool of susceptibles becomes too small to maintain transmission.

From a public-health perspective, the key message is the existence of a threshold. If interventions can reduce the effective susceptible pool below the threshold, the epidemic declines.

3.7. The SIRS model

Some diseases do not confer permanent immunity. After recovery, individuals may become susceptible again. This leads to the SIRS model:

$$S' = -\beta SI + \mu R, \qquad I' = \beta SI - \nu I, \qquad R' = \nu I - \mu R,$$

where $\mu > 0$ measures the loss of immunity.

Again, the total population

$$S + I + R = \tau$$

is constant. Eliminating R gives the planar system

$$S' = -\beta SI + \mu(\tau - S - I), \qquad I' = \beta SI - \nu I.$$

3.8. Equilibria of the SIRS model

This system has up to two equilibria:

- the disease-free equilibrium $(\tau, 0)$;
- the endemic equilibrium

$$(S^*, I^*) = \left(\frac{\nu}{\beta}, \frac{\mu(\tau - \nu/\beta)}{\nu + \mu}\right),$$

  provided that $\tau \geq \nu/\beta$.

The quantity ν/β is again the threshold level. When $\tau = \nu/\beta$, a bifurcation occurs: the endemic equilibrium is born from the disease-free equilibrium.

Threshold interpretation

If the total population lies below the threshold, the disease cannot become permanently established. If the total population exceeds the threshold, an endemic state may exist and be stable.

3.9. Invariant region

The biologically meaningful region is the triangle

$$\Delta = \{(S, I) : S \geq 0,\ I \geq 0,\ S + I \leq \tau\}.$$

This region is positively invariant: once a solution starts in Δ, it remains in Δ for all future time. This fact is mathematically important because it ensures that trajectories remain biologically meaningful.

3.10. Local stability and endemic persistence

At the disease-free equilibrium $(\tau, 0)$, the eigenvalues are $-\mu$ and $\beta\tau - \nu$. Hence:

- if $\beta\tau - \nu < 0$, the disease-free equilibrium is locally stable;
- if $\beta\tau - \nu > 0$, it becomes unstable in one direction.

At the endemic equilibrium, the trace is negative and the determinant is positive, so the equilibrium is asymptotically stable whenever it exists.

Biologically, this means that if immunity is temporary and the total population is above threshold, the disease may persist indefinitely at a stable endemic level rather than disappearing after one epidemic wave.

3.11. Comparison between SIR and SIRS

Feature	SIR model	SIRS model
Recovered class	Permanent immunity	Temporary immunity
Long-term disease behavior	Tends to die out after epidemic wave	May persist endemically
Threshold role	Determines initial growth or decline of epidemic	Determines existence of endemic equilibrium
Main qualitative picture	Single outbreak dynamics	Possible stable persistent infection

3.12. Pedagogical discussion

The epidemic models teach several essential ideas of mathematical biology:

- conservation laws reduce dimension;
- threshold quantities determine biological regimes;
- equilibria represent biologically meaningful states;
- stability has direct epidemiological interpretation;
- compartment models are structurally simple yet conceptually rich.

3.13. Exercises on infectious diseases

Exercise 3.1. *For the SIR model, explain why $S(t)$ is always nonincreasing.*

Exercise 3.2. *Show that the infected population increases exactly when $S > \nu/\beta$.*

Exercise 3.3. *Interpret biologically the endemic equilibrium of the SIRS model.*

Exercise 3.4. *Suppose vaccination instantly reduces the susceptible population from S_0 to qS_0 with $0 < q < 1$. Explain qualitatively how this changes the epidemic threshold condition.*

4. Predator–Prey Systems

4.1. The biological problem

Predator–prey systems describe ecological interaction between two populations when one species feeds on the other. Typical examples are foxes and rabbits, wolves and deer, sharks and fish, or insects and pest species. The mathematical challenge is to capture two opposite effects simultaneously:

- prey support predator growth;
- predators reduce prey abundance.

4.2. The Volterra–Lotka model

Let $x(t)$ denote the prey population and $y(t)$ the predator population. The classical assumptions are:

1. prey grow exponentially in the absence of predators;
2. predator population decreases in the absence of prey;
3. encounters are proportional to xy;
4. prey are harmed by encounters and predators benefit from them.

This gives the system

$$x' = x(a - by), \qquad y' = y(-c + dx),$$

where $a, b, c, d > 0$.

4.3. Nullclines and equilibrium points

The nullclines are:

$$x' = 0 \iff x = 0 \text{ or } y = \frac{a}{b}, \qquad y' = 0 \iff y = 0 \text{ or } x = \frac{c}{d}.$$

Therefore the system has an interior equilibrium

$$Z = \left(\frac{c}{d}, \frac{a}{b}\right).$$

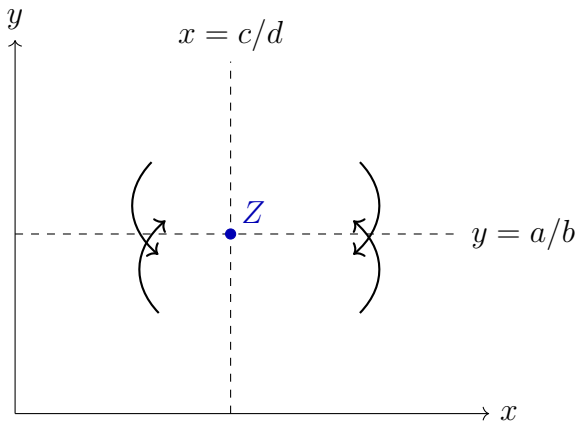
The coordinate axes are invariant because if one species is absent, it remains absent.

4.4. Qualitative motion in the phase plane

The nullclines divide the first quadrant into four regions. In each region, the signs of x' and y' are fixed:

Region	Sign of x'	Sign of y'
$x > c/d,\ y < a/b$	$+$	$+$
$x < c/d,\ y < a/b$	$+$	$-$
$x < c/d,\ y > a/b$	$-$	$-$
$x > c/d,\ y > a/b$	$-$	$+$

This sign structure causes trajectories to circulate around the interior equilibrium.



4.5. Closed orbits and cyclical oscillation

A remarkable property of the classical Volterra–Lotka model is that every solution in the first quadrant, except the equilibrium and the axes, is a closed orbit. Hence predator and prey populations oscillate cyclically forever.

Biologically, this means:

- prey increase first when predators are few;
- the abundance of prey later allows predator growth;
- increased predation reduces prey;
- scarcity of prey then reduces predators;
- the cycle repeats.

The model predicts neutral cycles rather than convergence to equilibrium.

4.6. Limitations of the classical model

Although elegant, the Volterra–Lotka model is too idealized for many real ecosystems. It does not include environmental carrying capacity, self-limitation, or saturation of predation. As a consequence, it predicts perpetual oscillations of constant amplitude, which are rarely observed in nature.

4.7. Predator–prey model with limited growth

To make the model more realistic, we introduce crowding in the prey population and self-limitation in the predator population:

$$x' = x(a - by - \lambda x), \qquad y' = y(-c + dx - \mu y),$$

with $a, b, c, d, \lambda, \mu > 0$.

Now:

- in the absence of predators, prey follow the logistic equation $x' = x(a - \lambda x)$;
- in the absence of prey, predators satisfy $y' = -cy - \mu y^2$, so they decay to zero.

4.8. Nullclines in the limited-growth model

The nontrivial nullclines are the lines

$$L : a - by - \lambda x = 0, \qquad M : -c + dx - \mu y = 0.$$

Two cases arise.

4.8.1. Case 1: the lines do not intersect in the first quadrant

If L and M do not intersect in the biologically relevant region, then every positive trajectory tends to the equilibrium

$$\left(\frac{a}{\lambda}, 0\right).$$

In this case the predator population becomes extinct, while the prey population approaches its carrying capacity in the absence of predators.

This is a mathematically precise expression of an ecologically intuitive outcome: predators cannot survive if the environmental or interaction parameters do not allow coexistence.

4.8.2. Case 2: the lines intersect in the first quadrant

If L and M intersect in the first quadrant, then there is an interior equilibrium Z. In that case, trajectories remain bounded in a positively invariant rectangle. By the Poincare–Bendixson theorem, every positive trajectory approaches either the equilibrium Z or a limit cycle surrounding Z.

Hence the long-term dynamics can settle into one of two biologically meaningful behaviors:

- stable coexistence at constant population levels;
- persistent oscillation around coexistence.

Ecological interpretation

In the limited-growth model, the long-term behavior is bounded. No population can grow without bound, and the system ultimately settles into either an equilibrium regime or a periodic regime.

4.9. Invariant rectangles and boundedness

One of the important mathematical tools in this section is the construction of a rectangle in the first quadrant that all trajectories eventually enter and never leave. This proves boundedness. Such invariant regions are especially important in biological models because they guarantee that populations remain finite in the long run.

4.10. Comparing the two predator–prey models

Feature	Volterra–Lotka	Limited-growth model
Prey in absence of predators	Exponential growth	Logistic growth
Predators in absence of prey	Exponential decline	Decline with self-limitation
Interior dynamics	Closed orbits	Equilibrium or limit cycle
Long-term boundedness	Yes, but by conserved geometry	Yes, via invariant region
Ecological realism	Idealized	More realistic

4.11. Why the Poincare–Bendixson theorem matters

For planar systems, the Poincare–Bendixson theorem is a fundamental tool. It says, roughly, that a bounded trajectory in the plane cannot behave chaotically: its omega-limit set must be an equilibrium, a closed orbit, or a collection built from equilibria and connecting orbits. In predator–prey systems, this theorem explains why periodic solutions and stable coexistence are the principal bounded possibilities.

4.12. Exercises on predator–prey systems

Exercise 4.1. *For the Volterra–Lotka model, determine the signs of x' and y' in each region separated by the nullclines.*

Exercise 4.2. *Explain biologically why the term $-by$ appears in the prey growth factor and why the term dx appears in the predator growth factor.*

Exercise 4.3. *For the limited-growth model, explain why $(a/\lambda, 0)$ represents prey survival with predator extinction.*

Exercise 4.4. *Give a biological interpretation of a limit cycle in a predator–prey model.*

5. Competitive Species

5.1. From specific equations to qualitative assumptions

Competition models study the interaction of two species that depend on a common limited resource. In contrast with the earlier sections, the book adopts a more general strategy here. Instead of starting with one explicit algebraic form, it considers a broad class of systems and assumes only qualitative properties.

This is pedagogically important. It shows students that mathematical biology is not only about solving given formulas; it is also about understanding the geometric consequences of biologically reasonable assumptions.

5.2. General form of the model

Let $x(t)$ and $y(t)$ denote the populations of two competing species. Their growth equations are written as

$$x' = M(x, y)x, \qquad y' = N(x, y)y,$$

where M and N are the growth rates. The factors x and y ensure that the coordinate axes are invariant.

The nullclines are therefore:

$$x = 0, \quad M(x, y) = 0, \qquad y = 0, \quad N(x, y) = 0.$$

5.3. Qualitative assumptions on the growth rates

The general competitive framework assumes:

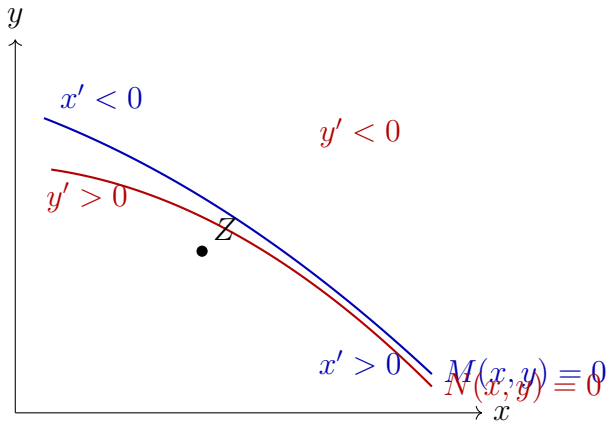
1. each species can grow when its own population and the competitor population are both sufficiently small;
2. growth is reduced by increasing either population;
3. for large enough values of a population, its own growth becomes negative;
4. competition acts by decreasing the effective growth rates M and N.

These assumptions imply that the curves $M(x, y) = 0$ and $N(x, y) = 0$ are decreasing graphs in the first quadrant under suitable monotonicity conditions.

5.4. Biological meaning of nullclines

The curve $M(x, y) = 0$ separates the region where species x increases from the region where it decreases. Likewise, $N(x, y) = 0$ separates the region where species y increases from the region where it decreases.

Hence nullclines play the role of ecological thresholds. They tell us where each species can persist and where it declines.



5.5. Possible equilibrium configurations

Because of the multiplicative form of the system, there are always boundary equilibria on the axes corresponding to extinction of one or both species. The most interesting question concerns interior equilibria, where both species coexist.

Several qualitative cases are possible depending on the relative position of the nullclines.

5.5.1. Case A: no interior intersection

If the nontrivial nullclines do not intersect in the first quadrant, then coexistence is impossible. One species eventually outcompetes the other. Depending on the arrangement of the curves, either:

- species x survives and species y becomes extinct; or
- species y survives and species x becomes extinct.

This is the mathematical expression of the competitive exclusion principle.

5.5.2. Case B: one interior equilibrium

If the nullclines intersect at an interior point Z, then coexistence is possible. However, the nature of Z matters:

- if Z is stable, both species coexist in the long run;
- if Z is a saddle, then coexistence occurs only for carefully chosen initial conditions, while generic trajectories tend to one of the boundary equilibria.

5.6. Stable coexistence versus bistability

One of the most important biological distinctions is the difference between:

a) **stable coexistence**: the interior equilibrium attracts nearby trajectories, and the two species persist together;

b) **bistability**: the interior equilibrium is unstable, and long-term survival depends on initial conditions.

In bistability, the basin of attraction is split by the stable manifold of a saddle equilibrium. Two communities with the same parameters but different initial abundances may evolve to completely different outcomes.

5.7. Geometric interpretation in the phase plane

Competition models are especially well suited to nullcline analysis. In each region of the first quadrant, the signs of x' and y' determine whether trajectories move up, down, right, or left. The global phase portrait can often be understood by combining:

- the positions of the nullclines;
- the location and type of equilibria;
- positive invariance of the first quadrant;
- the impossibility of crossing invariant axes.

5.8. Boundary equilibria and ecological interpretation

Boundary equilibria correspond to the survival of one species and extinction of the other. For example:

$$(x^*, 0) \quad \text{or} \quad (0, y^*).$$

A stable boundary equilibrium means that one species is the superior competitor under the model assumptions. The mathematical conclusion is extinction of the weaker competitor.

5.9. Coexistence and resource partitioning

A stable interior equilibrium suggests that the two species may coexist, at least in the coarse-grained sense of the model. Biologically, this can represent partial niche separation, different efficiencies of resource use, or a balance of interspecific and intraspecific competition.

5.10. Why a qualitative framework is valuable

Explicit competition models can be difficult to solve and may depend heavily on arbitrary algebraic choices. By imposing only qualitative conditions on M and N, we learn which conclusions are robust. This is a major strength of the dynamical-systems approach.

Main ecological outcomes of competition models

Depending on the nullcline arrangement and local stability of equilibria, competition can lead to:

- extinction of both species only in degenerate situations;
- competitive exclusion of one species by the other;
- stable coexistence of both species;
- bistability, where the winner depends on initial conditions.

5.11. A conceptual comparison with predator–prey systems

Competition and predator–prey systems differ in structure:

- in predator–prey models, one population helps the other while being harmed by it;
- in competition models, both populations inhibit each other.

This sign difference has major dynamical consequences. Predator–prey systems often produce oscillations. Competition systems more often produce convergence to equilibria and exclusion phenomena.

5.12. Exercises on competitive species

Exercise 5.1. *Explain why the axes are invariant in the model* $x' = M(x,y)x$, $y' = N(x,y)y$.

Exercise 5.2. *Give a biological meaning for a stable interior equilibrium in a competition model.*

Exercise 5.3. *What is the ecological meaning of a saddle interior equilibrium?*

Exercise 5.4. *Compare competition and predator–prey interactions in terms of the signs of the interaction effects on each equation.*

6. Synthesis of the Three Biological Themes

The three families of models studied in this lesson may now be compared at a conceptual level.

6.1. A unified view

All three models are built from the same ingredients:

- populations as dependent variables;
- rates of change as derivatives;
- interaction mechanisms encoded by nonlinear terms;
- long-term behavior analyzed through equilibria and phase portraits.

Yet the biological meaning of the nonlinear interaction differs greatly.

Model	Main variables	Key nonlinear term	Typical long-term behavior
SIR / SIRS	S, I, R	SI	Epidemic wave or endemic equilibrium
Predator–prey	x, y	xy	Oscillation, coexistence, or limit cycle
Competition	x, y	interaction through M, N	Exclusion, coexistence, or bistability

6.2. Thresholds, coexistence, and extinction

A recurrent theme is the existence of threshold structures.

- In epidemic models, thresholds determine whether infection grows or declines.
- In predator–prey systems, the crossing of nullclines determines oscillatory or convergent behavior.
- In competition systems, the relative positions of nullclines determine whether coexistence or exclusion occurs.

Thus, thresholds are not restricted to one application; they are a structural idea in dynamical systems.

7. Methodological Toolbox for Students

7.1. How to read a biological system of differential equations

When a student first sees a system such as

$$x' = f(x, y), \qquad y' = g(x, y),$$

the following checklist is recommended:

1. Identify the variables and parameters.

2. Determine the biologically meaningful region.
3. Find the equilibria.
4. Compute or sketch the nullclines.
5. Determine the sign of each derivative in each region.
6. Interpret the resulting arrows biologically.
7. Study local stability by linearization when appropriate.
8. Draw qualitative conclusions about long-term behavior.

7.2. Frequent mistakes

Common student errors

- Confusing variables and parameters;
- ignoring the biological region and discussing negative populations;
- finding equilibria algebraically but not interpreting them;
- computing nullclines without using them qualitatively;
- assuming that oscillation in time always implies a limit cycle;
- forgetting that local stability does not automatically imply a global conclusion.

7.3. What should be justified in an exam?

In a written solution, a student should not merely state conclusions. They should justify them using sign arguments, nullcline analysis, eigenvalue criteria, invariance, or biological interpretation. For example, it is not enough to write "the disease dies out". One should say why: because the disease-free equilibrium is stable, or because $I' < 0$ once $S < \nu/\beta$.

8. Extended Worked Examples

8.1. Worked example 1: logistic growth

Consider

$$P' = 0.4P\left(1 - \frac{P}{230}\right).$$

The equilibria are $P = 0$ and $P = 230$. For $0 < P < 230$, the derivative is positive, so the population increases. For $P > 230$, the derivative is negative, so the population decreases. Therefore every positive solution tends to 230.

Biologically, this says that 230 is the carrying capacity and the long-term population stabilizes there.

8.2. Worked example 2: epidemic threshold

Suppose the SIR parameters satisfy $\beta = 0.002$ and $\nu = 0.5$. Then

$$\frac{\nu}{\beta} = 250.$$

If initially $S(0) > 250$, then the infected class initially grows. If $S(0) < 250$, then it declines immediately. This threshold computation is simple but biologically crucial.

8.3. Worked example 3: interior predator–prey equilibrium

For the Volterra–Lotka system

$$x' = x(a - by), \qquad y' = y(-c + dx),$$

the interior equilibrium is

$$Z = \left(\frac{c}{d}, \frac{a}{b}\right).$$

This equilibrium exists because predator persistence requires enough prey and prey control requires enough predators. The formulas themselves already encode an ecological balance.

8.4. Worked example 4: competition outcome

Suppose species x and y satisfy a competitive system and the nullclines are arranged so that the only stable equilibrium in the first quadrant boundary is $(x^*, 0)$. Then any initial condition in the interior, except possibly those on a separatrix, leads to extinction of species y and survival of species x.

This is a precise example of competitive exclusion.

9. Review Questions

1. What is the difference between a variable and a parameter in a differential-equation model?
2. Why is the logistic model more realistic than the exponential growth model?

3. In the SIR model, why is the line $S = \nu/\beta$ important?
4. What is the biological interpretation of the endemic equilibrium in the SIRS model?
5. Why do interaction terms often appear as products such as SI or xy?
6. What makes the Volterra–Lotka model mathematically elegant but biologically limited?
7. What is the role of nullclines in studying planar systems?
8. What is meant by positive invariance of a region?
9. In a competition model, what does a stable interior equilibrium mean?
10. In a competition model, what does a stable boundary equilibrium mean?

10. Suggested Homework Set

1. Derive the logistic equation from the assumptions of proportional growth for small populations and negative growth for populations larger than carrying capacity.
2. For the SIR model, show that if $I(0) = 0$, then $I(t) = 0$ for all t.
3. For the SIRS model, compute the endemic equilibrium and state the condition for its existence.
4. Draw the nullclines of the Volterra–Lotka system and explain the sign of each derivative in each region.
5. Explain why a predator–prey model with limited prey growth may admit a stable equilibrium or a limit cycle.
6. Give an example of a biological interpretation of bistability in a competition model.
7. Compare the meaning of the terms “disease-free equilibrium”, “coexistence equilibrium”, and “extinction equilibrium”.
8. Write a one-page reflection on why qualitative analysis is essential in mathematical biology.

11. Conclusion

This lesson has shown that differential equations are not only computational tools; they are conceptual tools for organizing biological reasoning. Starting from assumptions about

contagion, predation, or competition, we obtained systems of differential equations whose structure already contains biological insight.

The SIR and SIRS models illustrate threshold behavior and the distinction between epidemic fade-out and endemic persistence. Predator–prey systems show how opposite interaction effects can generate cyclic or bounded coexistence. Competitive-species models show that qualitative assumptions alone can produce strong conclusions about exclusion, coexistence, and dependence on initial conditions.

For first-year Master's students, the central message is this: mathematical biology begins with model construction, gains depth through qualitative analysis, and reaches interpretation through the language of dynamical systems.

Final take-away

Whenever you face a new biological application, ask four questions:

1. What are the mechanisms?
2. Which quantities vary in time?
3. Which equations express those mechanisms?
4. What do equilibria, nullclines, and trajectories mean biologically?

These four questions form the bridge between biology and differential equations.

12. Glossary of Core Concepts

Term	Meaning in this lesson
Model	A mathematical representation of selected features of a real biological phenomenon.
Dependent variable	A quantity that changes with time, such as a population or an infected class.
Parameter	A fixed constant that measures a biological mechanism, such as a transmission rate or a carrying capacity.
Initial condition	The state of the system at the initial time.
Equilibrium	A state in which all derivatives vanish, so the system remains constant in time.
Nullcline	A curve in the phase plane where one component of the vector field is zero.
Invariant region	A set that, once entered by a trajectory, cannot be left in forward time.

Threshold	A critical value separating qualitatively different biological regimes.
Endemic equilibrium	A steady state where the disease persists with a positive infected population.
Coexistence equilibrium	A steady state where two species survive together with positive populations.
Limit cycle	An isolated periodic orbit that describes sustained oscillatory behavior.
Competitive exclusion	The long-term survival of one species together with extinction of the competitor.
Bistability	A situation in which different initial conditions lead to different long-term outcomes.
Linearization	The approximation of a nonlinear system near an equilibrium by its Jacobian matrix.

13. Mini Project Topics for Master 1 Students

The following short projects can be assigned individually or in pairs.

Project A: Vaccination and epidemic thresholds

Start from the SIR model and introduce a vaccination fraction $p \in [0, 1]$ that reduces the initial susceptible population. Study how the condition for initial epidemic growth changes. Explain the public-health meaning of the resulting threshold.

Project B: Predator control and ecological consequences

Modify a predator–prey model by introducing human hunting of predators at a constant rate or at a rate proportional to predator abundance. Discuss qualitatively how this changes the nullclines and the possible long-term outcomes.

Project C: Invasion of a competing species

Consider a competition model in which species x is initially established and species y is introduced with small initial abundance. Use phase-plane reasoning to discuss when the new species can invade successfully and when it fails.

Project D: From equations to biological language

Choose one of the systems in this lesson and write a two-page explanation addressed to biology students with minimal mathematics background. The goal is to translate each mathematical statement into biological language without losing correctness.

14. Sample Examination Problems

Exercise 14.1. *Consider the logistic equation $P' = rP(1 - P/K)$ with $r, K > 0$.*

a) Find all equilibria.

b) Determine their stability for positive populations.

c) Explain the biological meaning of the carrying capacity K.

Exercise 14.2. *Consider the SIR system $S' = -\beta SI$, $I' = \beta SI - \nu I$, $R' = \nu I$.*

a) Show that $S + I + R$ is constant.

b) Show that I increases exactly when $S > \nu/\beta$.

c) Explain why the epidemic cannot grow forever in this model.

Exercise 14.3. *For the Volterra–Lotka system $x' = x(a - by)$, $y' = y(-c + dx)$:*

a) Find the interior equilibrium.

b) Draw the nontrivial nullclines.

c) Use sign analysis to explain why trajectories circulate around the equilibrium.

Exercise 14.4. *In a competition model $x' = M(x, y)x$, $y' = N(x, y)y$, explain the biological difference between a stable boundary equilibrium and a stable interior equilibrium.*

15. Final Recap

The lesson can be summarized in four sentences.

1. Modeling begins by choosing assumptions and ends by interpreting solutions.
2. Nonlinear interaction terms encode biological mechanisms such as contagion, predation, and competition.
3. Equilibria, thresholds, nullclines, and invariant regions are the main qualitative tools for understanding biological systems.
4. The real success of a model lies not only in its equations, but in the biological meaning extracted from its dynamics.

Pedagogical Note

This teaching document is intentionally written in a detailed expository style for graduate-level classroom use. It is designed to support lecture presentation, student reading, tutorial sessions, and exam preparation.