Math 19. Lecture 1
Course Orientation

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1 Modeling in the Biological Sciences

- Mathematics consists of the study and development of methods of predictions.
- The goal of biology is to find useful and verifiable descriptions and explanations of phenomena in the natural world.

2 Population growth

The rate of growth of population is proportional to the size of the population,

$$\begin{aligned} \frac{dP}{dt} &= kP, \\ P(0) &= P_0. \end{aligned}$$

This is a good model for a population that grows without constraints.

3 Predator-Prey Model

Assumptions for how a population of rabbits, R, and foxes, F, interact.

1. If no foxes are present, the rabbits reproduce at a rate proportional to their population, and they are not affected by overcrowding.
2. The foxes eat the rabbits, and the rate at which the rabbits are eaten is proportional to the rate at which the rabbits and the foxes interact.
3. Without rabbits to eat, the fox population declines at a rate proportional to itself.

4. The rate at which foxes are born is proportional to the number of rabbits eaten by foxes, which by the second assumption, is proportional to the rate at which the foxes and rabbits interact.

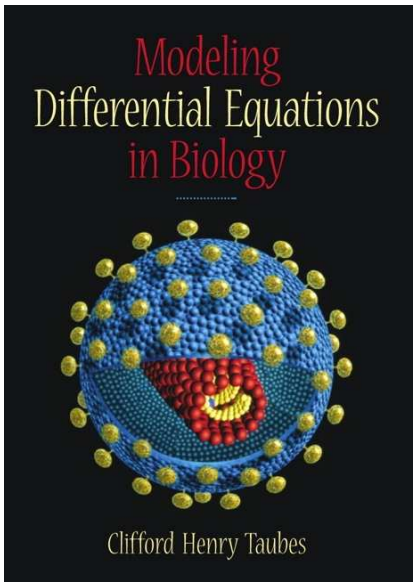
The system

$$\begin{aligned} \frac{dR}{dt} &= \alpha R - \beta RF \\ \frac{dF}{dt} &= -\gamma F + \delta RF \end{aligned}$$

is a model of how a predator population interacts with a prey population.

4 The Evolutionary Development of HIV-1

How does the HIV-1 virus evolve in the human body? Once infected with the HIV-1 virus, it can be years before an HIV-positive patient exhibits the full symptoms of AIDS. Does the virus lie dormant in patient's body? This was thought to be the case at one time.



Here is a model that tells us otherwise. Due to transcription errors during the replication process, quasispecies of the virus are created. These quasispecies are populations of closely related but distinct viral genomes of

the virus. We can base a mathematical model of this process on the following assumptions.

1. The virus can kill CD4-positive T-helper cells.
2. The continual evolution of new resistant viral mutants enables the total viral population to evade elimination by the immune system.
3. Subpopulations of CD4-positive T-helper cells specific to a particular viral direct immunological attack against that strain.
4. Each mutant can kill all CD4 cells, regardless of their specificity to a particular mutant.
5. Immunological responses to the virus are characterized by a specific response to individual strains and a non-specific general response that acts against all strains.

We assign the following variables.

- z is the population of nonspecific CD4-positive T-helper cells.
- v_i is the population of a virus strain, where $i = 1, 2, \ldots, n$.
- x_i is the population to strain-specific CD4-positive T-helper cells, where $i = 1, 2, \ldots, n$.

We are now ready to construct out basic model. We do this in terms of systems of equations.

- Each strain of the virus will grow at a rate

$$\frac{dv_i}{dt} = rv_i - szv_i - px_iv_i,$$

  where r, s, and p are constants.

- The population of nonspecific CD4-positive T-helper cells will behave according to the equation

$$\frac{dz}{dt} = k'v - uvz,$$

  where $v = \sum v_i$ and k' and u are constants. Note that the immune cells are produced at a rate $k'v$ proportional to the density of antigens. The term uvz tells the rate at which the virions are destroyed.

- The population of specific CD4-positive T-helper cells will behave according to the equation

$$\frac{dx_i}{dt} = kv_i - uvx_i,$$

where k and u are constants.

The problem with modeling such a system is that n keeps increasing. We do not know how to solve such a system. One way to deal with this difficulty is to consider a finite system

$$\begin{aligned} \frac{dx_i}{dt} &= kv_i - uvx_i \\ \frac{dz}{dt} &= k'v - uvz \\ \frac{dv_i}{dt} &= rv_i - szv_i - px_iv_i + M(v), \end{aligned}$$

where $M(v)$ is a term representing the appearance of new viral strains.

Homework

- None

Readings and References

- C. Taubes. *Modeling Differential Equations in Biology.* Prentice Hall, Upper Saddle River, NJ, 2001.
- P. Blanchard, R. Devaney, and G. Hall. *Differential Equations*, second edition. Brooks/Cole, Pacific Grove, CA, 2002, pp. 4–8, 11-13.
- M. Nowak, R. May, and R. Anderson. "The evolutionary dynamics of HIV-1 quasispecies and the development of immunodeficiency disease," *AIDS*, 1990, Vol 4, No 11.