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A Biologist's Guide to Mathematical Modeling in Ecology and Evolution

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CHAPTER 1

Mathematical Modeling in Biology

Chapter Goals:

- To develop an appreciation for the importance of mathematics in biology

Chapter Concepts:

- Variables
- Dynamics
- Parameters
- Principle of parsimony

1.1 Introduction

Mathematics permeates biology. Unfortunately, this is far from obvious to most students of biology. While many biology courses cover results and insights from mathematical models, they rarely describe how these results were obtained. Typically, it is only when biologists start reading research articles that they come to appreciate just how common mathematical modeling is in biology. For many students, this realization comes long after they have chosen the majority of their courses, making it difficult to build the mathematical background needed to appreciate and feel comfortable with the mathematics that they encounter. This book is a guide to help any student develop this appreciation and comfort. To motivate learning more mathematics, we devote this first chapter to emphasizing just how common mathematical models are in biology and to highlighting some of the important ways in which mathematics has shaped our understanding of biology.

Let's begin with some numbers. According to BIOSIS, 886,101 articles published in biological journals contain the keyword "math" (including math, mathematical, mathematics, etc.) as of April 2006. Some of these articles are in specialized journals in mathematical biology, such as the *Bulletin of Mathematical Biology*, the *Journal of Mathematical Biology*, *Mathematical Biosciences*, and *Theoretical Population Biology*. Many others, however, are published in the most prestigious journals in science, including *Nature* and *Science*. Such a coarse survey, however, misses a large fraction of articles describing theoretical models without using "math" as a keyword.

We performed a more in-depth survey of all of the articles published in one year within some popular ecology and evolution journals (Table 1.1). Given that virtually every statistical analysis is based on an underlying mathematical model, nearly all articles relied on mathematics to some extent. With a stricter definition that excludes papers whose only use of mathematics is through statistical analyses, 35% of *Evolution* and *Ecology* articles and nearly 60% of *American Naturalist* articles reported predictions or results obtained using mathematical models. The extent of mathematical analysis varied greatly, but mathematical equations appeared in almost all of these articles. Furthermore, many of the articles used computer simulations to describe changes that occur over time in the populations under study. Such simulations can be incredibly helpful, allowing the reader to "see" what the equations predict and allowing authors to obtain results from even the most complicated models.

TABLE 1.1

Use of mathematical models in full-length journal articles

Journal (in 2001)	Number of articles	General use of models ^a	Specific use of models ^b	Equations presented ^c
<i>American Naturalist</i>	105	96%	59%	58%
<i>Ecology</i>	274	100%	35%	38%
<i>Evolution</i>	231	100%	35%	33%

^aGeneral use: Used a mathematical model in the broadest sense, including statistical or phylogenetic analyses with a mathematical basis (e.g., ANOVA, regression, etc.).

^bSpecific use: Used a mathematical model to obtain results (excluding cases that involve only statistical or phylogenetic analyses); the model may or may not be derived in the paper.

^cEquations presented: Excluding standard statistical equations.

An important motivation for learning mathematical biology is that mathematical equations typically “say” more than the surrounding text. Given the space constraints of many journals, authors often leave out intermediate steps or fail to state every assumption that they have made. Being able to read and interpret mathematical equations is therefore extremely important, both to verify the conclusions of an author and to evaluate the limitations of unstated assumptions.

To describe all of the biological insights that have come from mathematical models would be an impossible task. Therefore, we focus the rest of this chapter on the insights obtained from mathematical models in one tiny, but critically important, area of biology: the ecology and epidemiology of the human immunodeficiency virus (HIV). As we shall see, mathematical models have allowed biologists to understand otherwise hidden aspects of HIV, they have produced testable predictions about how HIV replicates and spreads, and they have generated forecasts that improve the efficacy of prevention and health care programs.

1.2 HIV

On June 5, 1981, the Morbidity and Mortality Weekly Report of the Centers for Disease Control reported the deaths of five males in Los Angeles, all of whom had died from pneumocystis, a form of pneumonia that rarely causes death in individuals with healthy immune systems. Since this first report, acquired immunodeficiency syndrome (AIDS), as the disease has come to be known, has reached epidemic proportions, having caused more than 20 million deaths worldwide (Joint United Nations Programme on HIV/AIDS 2004b). AIDS results from the deterioration of the immune system, which then fails to ward off various cancers (e.g., Kaposi’s sarcoma) and infectious agents (e.g., the protozoa that cause pneumocystis, the viruses that cause retinitis, and the bacteria that cause tuberculosis). The collapse of the immune system is caused by infection with the human immunodeficiency virus (Figure 1.1). HIV is transmitted

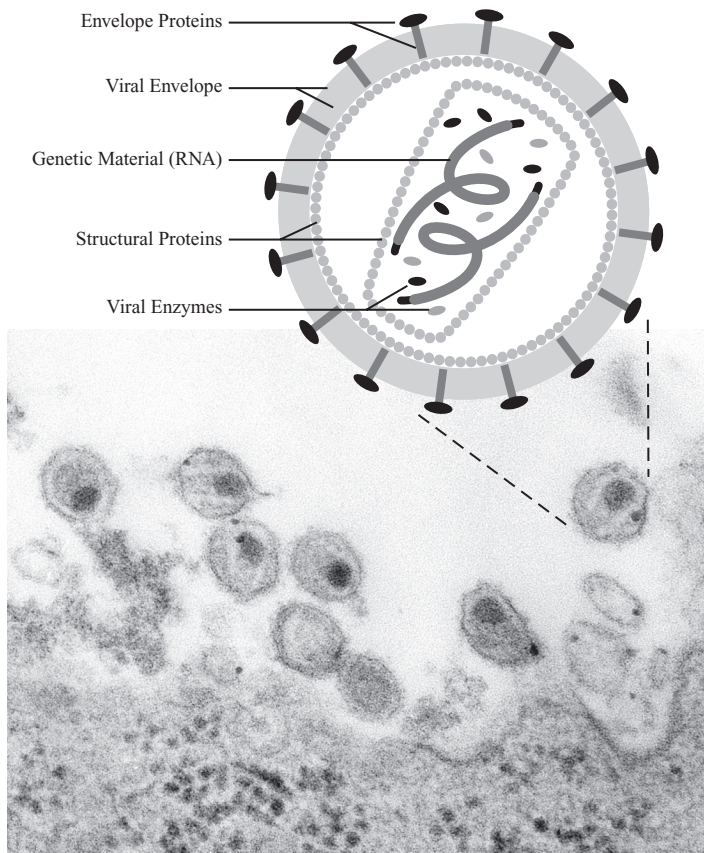


Figure 1.1: The human immunodeficiency virus. Electron micrograph shows HIV co-cultivated with human lymphocytes (courtesy of CDC; A. Harrison, P. Feorino, and E. L. Palmer).

from infected to susceptible individuals by the exchange of bodily fluids, primarily through sexual intercourse without condoms, sharing of unsterilized needles, or transfusion with infected blood supplies (although routine testing for HIV in donated blood has reduced the risk of infection through blood transfusion from 1 in 2500 to 1 in 250,000 [Revelle 1995]).

Once inside the body, HIV particles infect white blood cells by attaching to the CD4 protein embedded in the cell membranes of helper T cells, macrophages, and dendritic cells. The genome of the virus, which is made up of RNA, then enters these cells and is reverse transcribed into DNA, which is subsequently incorporated into the genome of the host. (The fact that normal transcription from DNA to RNA is reversed is why HIV is called a retrovirus.) The virus may then remain latent within the genome of the host cell or become activated, in which case it is transcribed to produce both the proteins necessary to replicate and daughter RNA particles (Figure 1.2). When actively replicating, HIV can produce hundreds of daughter viruses per day per host cell (Dimitrov et al. 1993), often killing the host cell in the process. These virus particles (or virions) then go on to infect other CD4-bearing cells, repeating the process. Eventually, without treatment, the population of CD4⁺ helper T cells declines dramatically from about 1000 cells per cubic millimeter of blood to about 200 cells, signaling the onset of AIDS (Figure 1.3).

