PREFACE

This monograph describes the results of our gonorrhea modeling project which began in 1973. This research project was funded by Centers for Disease Control Contracts 200-76-0613 and 200-79-0949, by National Institutes of Health Grant AI-13233, and by National Science Foundation Grant MCS81-0217. Although some results have been described in journal articles (Yorke, Hethcote and Nold, 1978; Hethcote, Yorke and Nold, 1982), the details and mathematical basis of many results have not appeared before or have appeared only in reports to CDC and NIH.

For most communicable diseases it is understood how an infective can transmit the infection by contacts with others and how a disease spreads through a chain of infections. Because of the numerous complex interactions in a population, it is difficult to comprehend the large scale dynamics of disease spread without the formal structure of a mathematical model. An epidemic model uses a microscopic description (the role of an infectious individual) to predict the macroscopic behavior of disease spread through a community. The purpose of mathematical models is to achieve a better understanding of how the biological and sociological mechanisms influence disease spread. Fixed parameters which occur in the models must have a well understood epidemiological interpretation such as a contact rate or a duration of infection.

Comparisons can lead to a better understanding of the process of disease spread. It may be possible to compare different diseases in the same population, the same disease in different populations or the same disease at different times. One way of making these comparisons is to formulate models for the various situations and then to compare the parameter values. We have made comparisons involving measles, rubella, mumps, chickenpox and poliomyelitis in several papers (London and Yorke, 1973; Yorke and London, 1973; Yorke, Nathanson, Pianigiani and Martin, 1979; Hethcote, 1983).

Although planned experiments can be used to obtain information in many sciences, experiments with infectious diseases in human populations are generally not possible for ethical and practical reasons. The only data usually available is from naturally occurring epidemics or from the natural incidence of endemic diseases; unfortunately, even these data are not complete since many cases are not reported. Hence the most basic facts of transmission may be in doubt. Since repeatable experiments and accurate data are usually not available in epide-

miology, **mathematical models can be used to perform needed theoretical experiments** with different parameter values and different data sets.

In order to use epidemic models for a particular disease, the capabilities and limitations of the models must be realized. It is often not recognized that many important questions cannot be answered using a given class of models. **The most difficult problem for the modeler is finding the right combination** of available data, an interesting question and a mathematical model which can lead to the answer.

Since infectious disease models furnish a means of assessing quantitative conjectures and of evaluating control procedures, they can be the only practical approach to answering questions about which control procedure is the most effective. Quantitative predictions of communicable disease models are always subject to some uncertainty since the models are idealized and the parameter values can only be estimated. **Predictions of the relative merits of several control methods are often robust** in the sense that the same conclusions hold for a broad range of parameter values and a variety of models. Various control methods for gonorrhea are compared in Chapters 4, 5 and 6 of this monograph.

Although some of the results described in this monograph may be useful for other sexually transmitted diseases (STDs), we have focused our attention here on gonorrhea. The incidences of other STDs such as genital herpes, caused by herpes simplex virus, and nongonococcal urethritis, often caused by Chlamydia trachomatis, are increasing dramatically in North America and Europe. Because practical diagnostic tools, control methods and specific treatments are often lacking for these other STDs, their incidence is increasing faster than the incidence of gonorrhea. There has also been an increase in the sexual transmission of diseases with agents such as hepatitis B virus, cytomegalovirus and Group B streptococcus (NIAID, 1980). The incidence of syphilis has decreased dramatically so that it is much less than the incidence of gonorrhea. The models used here would generally not be suitable for syphilis since individuals with syphilis go through several stages.

We hope that the unified presentation in this monograph will be of use to epidemiologists, scientists, mathematicians and students interested in sexually transmitted diseases or in how mathematical models can contribute to the understanding of disease transmission and control. We also hope that this monograph will encourage other studies of specific diseases using mathematical models.

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