

Mathematical Model Predicts AIDS Spread

The AIDS epidemic among heterosexuals may die out of its own accord

THE AIDS epidemic, says Robert May of Princeton University, is much less predictable than people think. Although it is possible to project how many people will develop AIDS in the next year or two, for example, there is no way of knowing how many will be infected in 5 years. Crucial data are missing, but it may be possible to estimate some important features of the epidemic indirectly from mathematical models of how the disease spreads.

May and his colleague Roy Anderson of Imperial College in London have developed a model of the AIDS epidemic which not only provides rough estimates of such things as the length of time between infection with the AIDS virus and onset of actual disease, but which also suggests questions about the epidemic that can lead to a much clearer picture of how AIDS will spread, particularly among heterosexuals. They reported on their model in the 12 March issue of *Nature*.

Among their conclusions are that it is by no means clear that the AIDS epidemic will be the Black Death of our age. AIDS may die out naturally. In particular, May and Anderson conclude that whether an AIDS epidemic among heterosexuals can be sustained depends on how long infected people remain infectious, how likely it is that an infected man will give the disease to a woman, and how likely it is that an infected woman will give AIDS to a man. If everyone infected dies within 10 years, for example, the epidemic will look quite different than if only 30% die and the rest go on infecting others for the rest of their sexually active lives.

The AIDS epidemic, says May, has taken people unaware because mathematicians have tended to study epidemiology in the abstract, unencumbered by any need to study messy but realistic data, and public health specialists have not had many true epidemics to deal with. In the 6 years since AIDS was first recognized as a disease, molecular biologists have learned an incredible amount about the AIDS virus, but epidemiologists have learned relatively little about how AIDS spreads in populations.

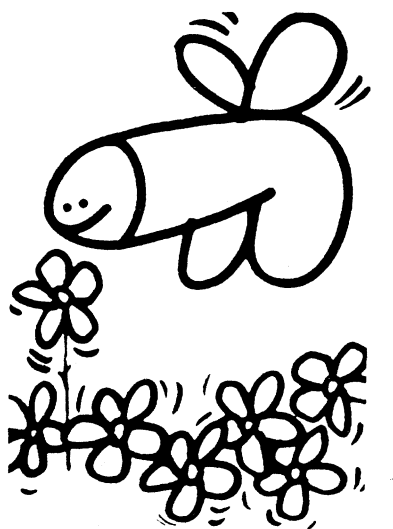
The precedent for the current AIDS model is a model of gonorrhea proposed in the mid-1970s by James Yorke of the University of Maryland. Yorke's model led him to

conclude that gonorrhea was being maintained in the population by a core of sexually active infected people and that the most effective strategy to control the disease would be to identify and treat that core group. The AIDS epidemic is similar, May says. "It is just essential that you recognize the great degree of sexual activity of the group at risk," he remarks.

But gonorrhea was endemic in the population—but the rates of infection were neither increasing nor decreasing. The problem was how to bring it under control.

AIDS is an epidemic, and so May and Anderson's model focuses on the rates at which people are being infected and on how to predict the total number of cases.

The model brings into sharp focus a number of important unanswered questions about AIDS. For example, it is not known for certain how long people are infectious. Nor is it certain what fraction of those who are infected will eventually get AIDS. And it



Kondom

Swedish condom symbol. *The Swedes have been promoting condoms for more than a decade to slow the spread of sexually transmitted diseases. The mathematical model of the AIDS epidemic indicates that if condom use can decrease by a factor of 5 the chance that a man with AIDS will infect a homosexual partner, the epidemic will die out in the homosexual community.*

is unclear whether people who are infected but never get AIDS will be infectious for the rest of their sexually active lives. Yet, says May, "The long-term view of what's going to happen is crucially dependent on these answers, and we just don't have them."

The AIDS epidemic is still in a phase of exponential growth, but May and Anderson predict that it will slow down and the number of cases will increase linearly; in fact, there is evidence from San Francisco that this is already happening. At first, when essentially everyone in the sexually active homosexual populations of large cities such as San Francisco was vulnerable, the AIDS infection rate took off. But now, those populations are already infected and it takes longer for the virus to spread to less sexually active groups. For this reason, the epidemic is slowing.

The problem in modeling this epidemic is that it is nonlinear and no simple mathematical formula predicts how the number of cases will increase. Public health officials who are trying to predict the AIDS epidemic curve-fit—they use today's data to decide what will happen in the next few years. But because the shape of the curve is changing, this approach will not be accurate for projections of even 5 years in the future, May says. The only way to make long-term predictions is to have answers to some of the crucial questions about the course of the disease.

Nonetheless, May says, "we can use the model to estimate combinations of parameters that by themselves may not be amenable to observation." For example, the rate at which the incidences of infection doubled in the initial phase of the epidemic is known. This is proportional to the probability that an infected person will infect a sexual partner multiplied by the number of sexual partners that person has. If it is assumed that the average number of partners is ten, the chance of transmitting the disease to a partner is one chance in ten.

The model also leads May to conclude that it is "rubbish" to argue from the African data that it is as likely that AIDS is transmitted from man to woman as it is from woman to man. In Africa, equal numbers of men and women are infected. But, says May, "The sex ratio in Africa is more in the nature of a problem to be explained than a proof that the virus is equally likely to be transmitted to men as women." One possible explanation, he says, is that most African men are relatively promiscuous and most women are relatively monogamous except for a core group of prostitutes. It could easily be that it is less likely for a woman to give AIDS to a man than vice versa, but AIDS infections among these prostitutes would be such an important reason for the spread of the AIDS

virus that the sex ratio of AIDS infections in Africa could still be 1:1.

In estimating how many will die from AIDS, investigators often assume that the more likely it is that an infected person will develop full-blown AIDS, the higher the death rate. But May and Anderson's model leads them to take issue with that assumption.

Suppose, says May, that you assume that everyone infected with the AIDS virus will die within 10 years. And then suppose you consider an alternative scenario—that 30% will die within 6 years and the rest will remain infectious for the rest of their sexually active lives, for about 30 to 40 years. "In the second case, the total number who will get infected is larger. The virus will continue to penetrate the less sexually active group of the population for 30 to 40 years. You can't say intuitively if there will be more deaths from 100% of a smaller number of people or a smaller percent of a larger number," May says. "I think this is very relevant to any long-term view in developed countries of heterosexual transmission of AIDS."

May explains that if the AIDS epidemic can take off through heterosexual transmission of the virus, it will, of course, begin among the most promiscuous heterosexuals. But, he says, "if all these people are dead within 10 years, the epidemic will likely run its course. If 70% of those infected remain infectious, there is more chance for the epidemic to spread more slowly." What will happen, he says, "depends on knowledge we don't have on how many will die and how long people remain infectious. We just don't know."

But May can use the data at hand on homosexual infections to estimate how difficult it might be to contain the AIDS epidemic in that group. The argument hinges on a parameter called R_0 , which is the basic reproductive rate of an epidemic—the number of new infections each person with the virus is likely to initiate. If R_0 is less than 1, an epidemic can't take off. And the greater than 1 R_0 is, the harder it is to contain an epidemic. Since 50 to 70% of homosexual men in San Francisco are infected with the AIDS virus, R_0 must be greater than 5, according to May. "That leads to a rather depressing epidemic," he says. "The only way to halt the epidemic is to change R_0 by a factor of 5." It would mean that infected individuals must have one-fifth as many sexual partners as they had in the past or that they practice safe sex, with condoms, so as to decrease by at least a factor of 5 the chance that they will infect their partners. And, like gonorrhea, any efforts at reducing R_0 must concentrate on the core group of sexually active men who are keeping the

epidemic alive. For heterosexual transmission, it is unknown whether R_0 is greater than or less than 1, according to May.

The mathematics of estimating R_0 among homosexual men also leads May and Anderson to an independent estimate of the incubation time of the virus. According to their model and the San Francisco data on infection incidences, it takes more than 5 years from infection with the virus to the disease

AIDS—a time that agrees with other estimates that come from clinical data.

In the end, May and Anderson's model raises more questions than it answers. But its importance lies in its new approach to the AIDS epidemic. Says May, "Population models can give insights into the dynamics of an epidemic and can help people think about the disease more clearly." ■

GINA KOLATA

Life Thrives Under Breaking Ocean Waves

Biological productivity in intertidal communities that are constantly battered by waves greatly exceeds what is achieved by even the most fruitful tropical rain forests

“ANYONE who has felt the force of a large wave breaking on an ocean beach knows that it dissipates a large amount of energy when it crashes against the shore. Can intertidal organisms put this energy to use?”

This question was posed by Egbert Leigh, of the Smithsonian Tropical Research Institute in Panama, and his colleagues Robert Paine, James Quinn, and Thomas Suchanek. The answer, it seems, is yes. In fact, say these researchers, many intertidal communities that are subject to constant wave battering are several times more biologically productive than the world's lushest rain forests, which are usually thought of as being at the apogee of the living world. The means by which wave energy is converted into living material is, however, rather indirect: the key to the story is not so much raw energy as disturbance.

In addition to trying to unravel the network of influences that shape the composition of biological communities, ecologists are interested in what governs the use of energy within them. In other words, why is the biological productivity of one type of zone different from that of another? Availability of nutrients is clearly one important factor, as is the source and flow of usable energy. No wonder then that tropical rain forests are so very fruitful, given their propitious location on the globe.

It was something of a surprise, therefore, when Leigh first noticed that some intertidal kelp communities appeared to be even more productive than rain forests. He joined

forces with Paine, Quinn, and Suchanek to analyze the energy flow in such communities in more detail. Most of their work was done on Tatoosh Island, Washington, which has long been a popular spot for ecological research by Paine and his associates.

The energy delivered to the shoreline by ocean waves in this region is enormous, and greatly exceeds that from solar radiation. On average, waves deliver 0.335 watt per square centimeter of energy to the coastline, which is about 15 times more than comes from the sun. Even during the calmest months, wave energy exceeds solar energy by more than 100%. So, the potential for enhanced productivity is great, if only the energy can be harnessed.

Leigh and his colleagues soon found that among the individual species in intertidal communities, those with the highest standing crops are restricted to the most wave-beaten areas. These species are the lower intertidal mussels, sea palms, and shrubby kelps. Now, although rain forests have enormous standing crops, much higher than in intertidal communities, a very large proportion of it is locked up in woody tissue, which is essentially dead. By contrast, a comparison of leaf (or frond) area between the two types of community gives a different perspective. For instance, the shrubby kelp in the Tatoosh intertidal zone bears around 20 square meters of frond per square meter of growing surface compared with around 8 square meters per square meter for tropical rain forests.

In terms of productivity, which is mea-