CHAPTER 22

Implications of Sanitation, Vectors, and Plant Susceptibility for Epidemic Development

Denis Fargette, Michael W. Shaw, and J. Michael Thresh

Preparation

These exercises can be performed individually or in small groups and can be completed within two to four hours. The main aim of the chapter is to help develop insights into disease progress curves by sketching freehand graphs to explore the effects of various factors on epidemic development. The spreadsheet program in Chapter 4 (Bowen) may be used; alternatively, a modelling package such as STELLA or SB ModelMaker may be used to supplement or extend the scope of this chapter.

Introduction

The exercises are intended to show how a simple epidemic model can be used to reason through the consequences of changes in a pathosystem. Three examples of practical importance are used: sanitation (without subsequent introduction of inoculum from outside); plant susceptibility to infection which varies over time; and disease vector populations which also vary through time.

Chapters 4 (Bowen), 6 (Nutter and Parker) and 7 (Neher et al.) describe various population growth models used to illustrate disease progress. Two of the simplest of these models are used here: monomolecular and logistic. Both incorporate a rate parameter to describe how fast disease increases per unit time. The focus of the exercises in this chapter is to link this description of disease progress to the biology of particular pathosystems.

Sanitation. One of the aims of sanitation is to reduce or eliminate initial inoculum from which epidemics start (Vanderplank, 1963). Its usefulness as a management technique depends on the type and rate of epidemic development, and the proportion of inoculum that can be eliminated. We shall mention three examples that show the diversity of biology connected with this technique. First, lettuce mosaic potyvirus, spread by aphids including *Myzus persicae*, is usually managed by planting seed with a very low virus incidence. To avoid yield losses, it is recommended that seed stocks should have less than one infected seed in 1000. Regulatory levels are often set lower than this (e.g., 1 in 70,000) to provide a safety margin and to allow for overlapping cropping patterns. Second, problems with African cassava mosaic geminivirus may be managed adequately in some ecological settings by ensuring that the cuttings used as planting material are free from virus. Third, *Rhizoctonia*- and *Verticillium*-induced wilts of tomato have been reduced by solar heating of the soil before planting (solarization).

However, sanitation techniques will not always be cost-effective, and reductions in disease can be small or transitory. In particular, if a pathogen is widespread and disperses long distances by wind or vector transport, gains from sanitation may be minute. This is because the initial inoculum in the field or planting material is less important than inoculum arriving from elsewhere. However, even when low inoculum densities comes from external sources, exploration of simple models can help to develop insights about the consequences of sanitation.

Changing vector populations. In almost all diseases spread by vectors, the vector populations vary through time. For example, one of the vectors of maize streak geminivirus in East and Central Africa, the leafhopper *Cicadulina mbila*, is rare at the end of the dry season in Zimbabwe, but multiplies throughout the wet season. Similarly, the whitefly *Bemisia tabaci*, vector of African cassava mosaic geminivirus, varies predictably between seasons.

Since vectors spread disease from plant to plant, variation in their numbers or habits will be reflected

in changes in the rate parameter of a model describing disease progress. If the main spread by vectors is from plant to plant within the field, insight can be gained from the logistic model; if inoculum is entirely from an external source, such as perennial weeds, the monomolecular model may more appropriate, since the inoculum present is independent of the proportion of diseased plants.

Changing host susceptibility. Susceptibility to disease varies commonly during the life of the host plant. For example, adult plant resistance to rust and mildew fungi is well-documented. Apple leaves become less and less susceptible to the apple scab pathogen, *Venturia inaequalis*, as the summer progresses. Likewise, potatoes become less susceptible to several well-known viruses as plants mature. Similarly, at least some cultivars of cassava become less susceptible to African Cassava Mosaic Virus as the plants mature. It is also possible for susceptibility to increase as plants or tissues mature; this is true for pathogens, such as *Stagonospora nodorum* of wheat and barley.

Procedure

Under each heading, several scenarios are suggested. In each case, try to give a quantitative answer by sketching disease progress curves for each scenario, and by comparing them with the given reference progress curve. The logistic and monomolecular models are simple to construct with software such as STELLA or SB-Modelmaker.

Evaluation

1. **Sanitation.** First, consider an epidemic developing in an annual crop, where the inoculum comes exclusively from infected plants within the field and which is described adequately by a logistic model with a constant rate parameter. Cassava infected by African cassava mosaic geminivirus might in some settings be approximately described by such a model The proportion of infected plants is y. Time is denoted by t. When first observed, at time 0, the proportion of infected plants is y_0. The rate of increase per unit of disease in an almost completely healthy field is r. Then, these equations describe approximately how disease changes with time:

$$\frac{dy}{dt} = ry(1-y) \tag{22.1}$$

If r is constant in time, this implies that disease incidence at any time is given by

$$y = \frac{1}{1+[(1-y_0)/y_0]e^{-rt}} \tag{22.2}$$

[see Chapter 4 (Bowen)]. In this scheme, sanitation is represented by a reduction in y_0.

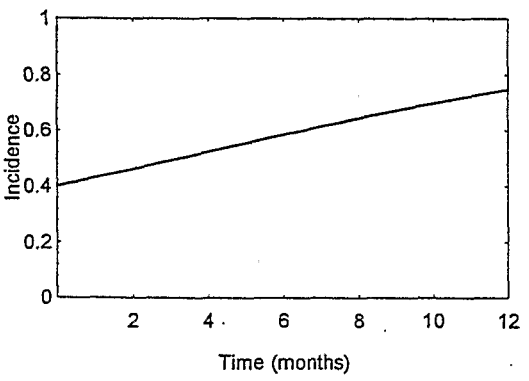

Fig. 22.1. Disease progress curve over 12 months according to a logistic model with rate parameter $r = 0.125$ per month, starting from an incidence of 40%.

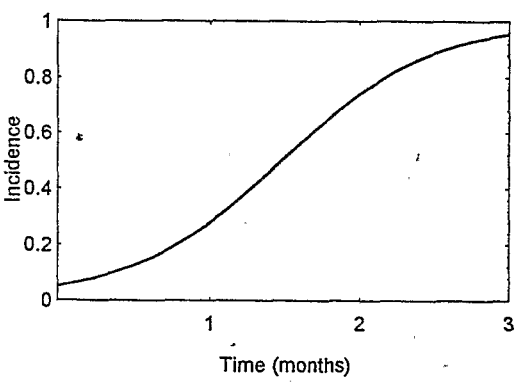

Fig. 22.2. Disease progress curve over three months according to a logistic model with rate parameter $r = 2.0$ per month, starting from an incidence of 5%.

Consider first an epidemic with a rate parameter, $r = 0.125$ per month, and with a 40% incidence of infection at the time of planting ($y_0 = 0.4$) (i.e., reference scenario). The disease progress curve for this over one year is illustrated in Fig. 22.1. Assume sanitation reduces initial inoculum to 20%, 10% or 5% infected plants. Without doing any calculations, sketch graphs to illustrate what the new curves will look like. Calculate disease levels at one month intervals using equation 22.2 starting from 5% infection (a spreadsheet program or programmable calculator will be useful). What will happen if the inoculum level is 10% or 20%?

Second, consider a faster developing epidemic such as might occur with lettuce mosaic virus, with a higher rate parameter, $r = 2$ per month, and with a 5% incidence of infection at planting ($y_0 = 0.05$) (i.e., reference scenario: Fig. 22.2). Assume that sanitation reduces inoculum to 1% disease incidence. Again, sketch the predicted outcome and perform calculations to verify the hypothesis. What happens if sanitation reduces inoculum to 0.1% ($y_0 = 0.001$)?

2. **Changing vector populations.** Consider an epidemic developing in an annual crop with a growing season of six months where the pathogen enters from external sources, and the inoculum generated during the epidemic makes a negligible contribution to disease progress. To stimulate ideas, we might compare maize streak disease in crops sown at different times of the year. As in the previous section, y is the disease incidence, y_0 is the disease incidence at the start of the epidemic, and t is the time. The rate parameter, r, has a different biological meaning to that in the previous section. From moment to moment, the changes in disease incidence are controlled by the equation:

$$\frac{dy}{dt} = r(1-y) \qquad (22.3)$$

If r were constant, this would imply that disease at any time was determined by

$$y = 1-(1-y_0)e^{-rt} \qquad (22.4)$$

[see Chapter 4 (Bowen)]. Variation in vector numbers or efficiency must be represented in this model by changes in r with time.

Consider the scenarios illustrated in Fig. 22.3. Note that in each scenario, the average number of vectors during the entire epidemic is identical; only the distribution over time is different. The initial disease incidence y_0 is 0; the inoculum arrives exclusively from external sources with the vector population. Fig. 22.4 shows the reference epidemic progress curve obtained with $r = 1$ per month regardless of time, as in Fig. 22.3(a).

If irrigated maize is sown soon after the end of the wet season, vector numbers will be greatest just after sowing. Without detailed calculation, sketch the progress curve expected if vector activity is concentrated at the start of the season, decreasing linearly to zero, so $r = (2 - t/3)$ per month, as in Fig. 22.3(b). [Hints: Is the initial rate of disease increase faster or slower than with constant r? At

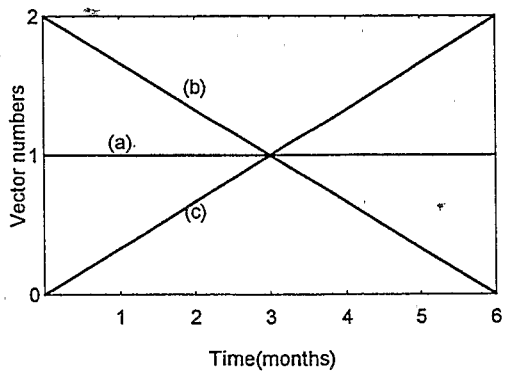

Fig. 22.3. Changing vector populations (arbitrary units) over six months: (a) constant, (b) decreasing linearly, and (c) increasing linearly.

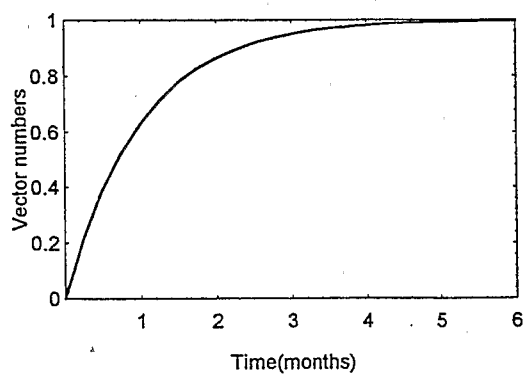

Fig. 22.4. Reference disease progress curve over six months according to the monomolecular model when $y_0 = 0$ and $r = 1.0$ per month.

what time of the season does $r = 1$ per month?] Now, using this experience, consider the situation for maize sown at the start of the wet season. Sketch the graph expected from linearly increasing vector activity, starting from zero ($r = t/6$ per month). As with many systemic viruses, the maize streak virus reduces maize yield greatly if it infects early, but scarcely at all if it infects late. With the assumptions made, what sowing date will minimize damage from the virus?

3. **Host susceptibility to infection.** Consider the same epidemic as in the previous section and the scenario in Fig. 22.4, with constant vector populations. Sketch what happens if host susceptibility were decreasing linearly through time ($r = 2 - t/3$). [Hint: What happens if vector numbers decrease linearly with time?!] Now sketch an expectation if susceptibility were decreasing exponentially over the season with $r = 2\exp(-t/3)$. [Hint: First calculate or sketch this pattern. When is r greater than in the reference case with constant r, and when is it less? Now sketch the disease progress curve.] Now, sketch an expectation if susceptibility decreased much faster, so $r = 2\exp(-2t)$. What model is a curve of this shape normally associated with?

Further Reading

Campbell, C. L., and Madden, L. V. 1990. Introduction to Plant Disease Epidemiology. John Wiley and Sons, New York.

Chan, M.-S., and Jeger, M. J. 1994. An analytical model of plant virus disease dynamics with roguing and replanting. J. Appl. Ecol. 31:413-427.

Doucet, P., and Sloep, P. B. 1992. Mathematical Modelling in the Life Sciences. Ellis Horwood, New York.

Gilligan, C. A. 1991. Modelling of crop pathogens. Aspects Appl. Biol. 26:111-121.

Kranz, J. 1990. Epidemics of Plant Diseases: Mathematical Analysis and Modelling. Springer-Verlag, Berlin.

Teng, P. S. 1985. A comparison of computer models of plant disease epidemics: A review of philosophy and methodology. J. Plant Dis. Prot. 88:49-63.

Thresh, J. M. 1988. Eradication as a virus disease control measure. Pages 155-194 in: Control of Plant Diseases: Costs and Benefits. B. C. Clifford and E. Lester, eds. Blackwell Scientific, Oxford.

Van der Plank, J. E. 1963. Plant Diseases: Epidemics and Control. Academic Press, New York.

Zadoks, J. C., and Schein, R. D. 1979. Epidemiology and Plant Disease Management. Oxford University Press, Oxford.