Interlude: What Has Been Omitted So Far?

The short answer to the above question is: quite a lot indeed. The purpose of the previous chapters was not to provide a comprehensive overview of methods and approaches in modern botanical epidemiology, which is provided in several references especially in Madden et al. (2007). What we tried instead is to provide a stepwise introduction to mechanistic simulation modeling, as one of the many methods available to botanical epidemiology. The choice of this approach was made because modeling attracts many plant scientists who, however, are deterred by the mathematics that might be involved. Note that much of the relationships we have used so far to analyze epidemiological systems are very simple. These relationships are closer to basic physical ones (thus the reference to dimensions in equations), rather than mathematical ones.

Yet many epidemiologists will rightly be prompt to point at the many gaps we left behind us. In the following elements, we briefly try to address at least some of these gaps from a practical modeling perspective. The points that are indicated below should not be seen as a check-list of gaps (there are more), but as a series of elements we feel are particularly important, especially the very last one.

Patterns of epidemics

Only one pattern of epidemics has been discussed in the prototype model developed from Chapter 4 onwards. This model implies that, in the course of a cropping season, each lesion produces a progeny of new lesions. Such epidemics are called polycyclic (e.g., Van der Plank, 1963; Zadoks and Schein, 1979). Many plant disease epidemics are not based on this pattern.

After all, the notion of successive, then overlapping, waves of sites that become infected, then infectious, and later removed is based on the quite anthropomorphic notions of a crop and of a cropping season. One may thus see a crop, from the (typical, but not sole) western point of view as a cohort, i.e., a population of plants whose development is nearly the same, having been established at the same time in a cultivated field. Many agricultural systems do not, however, consist of cohorts, nor are they homogeneous. In quite a few respects, they are thus closer to natural ecosystems. This point is briefly addressed below.

Some epidemics are caused by pathogens that produce only one wave (one generation) of propagules per season. Such epidemics are called monocyclic (e.g., Van der Plank, 1963; Zadoks and Schein, 1979). A typical example is that of some (specialized) diseases infecting flowers

(Ngugi and Scherm, 2001). Such epidemics can be of extreme importance, such as ergot (Agrios, 2005; Zadoks, 2008). There are different reasons why epidemics might be monocyclic. One is that the host is susceptible for a very short period of time, such as in the case of some flower diseases (Ngugi and Scherm, 2001); but another reason can be that the pathogen produces so very few propagules that only one wave of infection occurs in the course of a cropping season. The latter case prompted Zadoks and Schein (1979) and Ngugi and Scherm (2001) to compare such pathogens to K-strategists, as opposed to r-strategists, which cause polycyclic diseases, to bring us back to classical ecological concepts (May and McLean, 2007).

Confusing patterns of epidemics

Many diseases caused by soil-borne pathogens have been, and still are, often perceived as monocyclic diseases. This, actually, is often not true: even if there are few infections, even if the distance range of new infections from the mother lesion is small, many soil-borne diseases actually are polycyclic (e.g., Willocquet et al., 2008). In a key article, Pfender (1982) clarified the difference between deriving a model from the known biology of the disease, and the opposite: "*the error [is] that the nature of the disease cycle is being inferred from the disease progress curve. The nature of the disease must be determined before an appropriate model can be chosen."* This emphasizes the importance of focusing on processes and modeling them, as opposed to describing, even numerically, phenomena. In other words, the emphasis is on the importance of deriving models that truly have biology-related parameters (vs., for example, statistical models).

Many epidemic patterns may be considered

From an evolutionary perspective, one might admit that the notions of polycyclic or monocyclic epidemics are rather academic. As with any organism (Dodds, 2009), the point of view of the pathogen is to (1) survive, (2) multiply, (3) spread, and, in so doing, (4) adapt. Whether these processes happen during one or several cropping seasons, in fields cultivated with one or several plant species, in environments that are temporally and/or spatially uniform, does not really matter: failing to achieve any one of these steps means extinction. Phrased otherwise, the spatiotemporal diversity of agroecosystems is the context where these four events must take place; conversely, the spatiotemporal diversity of agroecosystems may, in some cases, be manipulated so that epidemics can be suppressed.

Let us consider the successive growing seasons of a vegetable crop, and a soil-borne disease, caused by, for example, *Rhizoctonia solani*. Let us further assume that the epidemic is monocyclic during a given growing season. The very fact that seasons follow after seasons will lead individual monocyclic epidemics to be concatenated in what was termed by Zadoks and Schein (1979) a polyetic epidemic. Each epidemic contributes to increasing the stock of soil-borne primary inoculum. Modeling this accumulation, its remobilization, and the effects of various crop management components, represents a very useful exercise to both understand and manage a difficult case. Difficulties arise due to few options for disease management, the pathogen has an extremely wide host range, and vegetable growers cannot easily shift to new lands, especially in peri-urban areas where vegetable growing is a most profitable activity. Such polyetic epidemics have so far been very poorly studied (e.g., Amorim and Bergamin Filho, 1991), but one might want to consider them as a particular form of polycyclic epidemic, characterized by having, at the beginning of each growing season (1) a new cohort of host plant (or host tissues, for perennials) is established and (2) the pathogen population has been reduced, and/or underwent a phase during which it had to survive without hosts.

We believe that *complicated* models do not need to be developed to gain a better understanding of patterns in *complex* systems. The case of household gardens highlights this point well. Household gardens are very important systems, agriculturally, socially, culturally, and from the point of view of sustainable food security, both in the developing and the developed worlds (Niñez, 1987). At times of economic and food crises, such systems may play a critical role. Plant pathology, and in general, epidemiology, ecology, and agronomy (as well as the Social Sciences and Medicine), might both have an important role to play, as well important lessons to learn, in household gardens. These systems often are very complex (e.g., Conway, 1994); yet, we believe that much insight could still be gained using un-complicated models.

The landscapes where epidemics occur may provide opportunity, or hindrance, to disease spread. This has been at the center of fundamental thinking for decades. For instance, Heesterbeek and Zadoks (1987) devised the notions of order 0 (focal), order 1 (general), and order 2 (pandemics) epidemics. In agricultural systems such as the Mekong Delta of Vietnam or the Central Plain of Thailand, up to seven rice crops are grown over two years, providing "green bridges" for pathogens to spread from one older cohort (field) to a younger one. The epidemiology of plant diseases in such environments has been studied in much detail, especially with respect to viral diseases, and the models derived by Chancellor, Azzam, and Holt (Azzam

and Chancellor, 2002; Chancellor et al., 2006) exemplify a success story based on clear understanding of processes, their quantification, their implementation in models, and leading to true application to disease management at the farm and landscape scales.

The critical importance of space-time interactions

The so-called 'mean field' hypothesis has been mentioned across the successive examples discussed so far. In other words: all the model structures discussed so far *implicitly* assumed that each healthy site, at any point of time, was accessible to infection. Conversely, they also implied that each propagule, be it for instance a viruliferous vector or a bacterial spore, had the same chance to infect, meaning a propagule that has been through the sequence of dispersal steps successfully, and has been lucky enough to (1) not land on bare ground, or on an unsuitable host, or in a hostile soil environment, and (2) encounter a site which is not already infected (i.e., a very lucky propagule indeed). The 'mean field' hypothesis is suitable for some thinking, but is admittedly inadequate for many systems.

Spatial and temporal processes cannot be distinguished and represent a mainstream area of investigation in Ecology (e.g., Renshaw, 1991; May and McLean, 2007), and in Botanical Epidemiology (Madden et al., 2007). A keystone article by Jeger (1982) provided a first analytical framework of plant disease epidemics in time and space.

Each epidemic is a unique event. We have not (and will not) speak of stochasticity in this module. Xu and Ridout (1996) demonstrated in a very elegant way how the inherent variability in the dispersal characteristics of a pathogen, and to a lesser degree, of the initial conditions of the modeled pathosystem (amount of primary inoculum) may affect the final spatial distribution and the final amount of disease. Being stochastic, their model simulates epidemics as unique successions of dispersal and infection events. Simulation outputs show expanding foci as propagules are dispersed and as epidemics unfold, sometimes giving rise to secondary foci, or, on the contrary, to general epidemics. Their model, in short, matches reality so well that is shows how difficult accurately measuring disease can be.

This transition chapter in neither the right place for expanding on this subject, nor on several other aspects we have left behind. Let us simply put it this way: the spatial structure of epidemics is very closely associated to their temporal ones. The spread (or extensification) of disease in the host population goes hand in hand with its local multiplication (intensification). The respective weights of extensification and intensification depend on the pathosystem

considered. Without extensification, the pathogen would locally intensify at one or a few sites, rapidly reach a local carrying capacity, and die. Without intensification, the pathogen would simply be unable to reproduce itself. From the pathogen viewpoint, extensification implies risks — from total propagule loss (e.g., deposition on the ground) to the encounter of unsuitable or resistant hosts; but it also entails opportunities: encountering more susceptible host sites. Aggregated (focal) epidemics lead to low levels of terminal disease, whereas random (general) epidemics lead to high levels of terminal disease (Xu and Ridout, 1996). Further, and critically, extensification offers the chance to the pathogen to evolve and adapt to different genotypes of its host. This is particularly true in complex agrosystems (Lenné and Jeger, 1994).

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