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Patterns and management of crop multiple pathosystems

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Abstract

The study of multiple pathosystems has played a central role in the development of botanical epidemiology, leading to a number of approaches and concepts. Multiple pathosystems are facts, which are experienced by many non-cultivated, or cultivated, plant communities. The shapes and composition of multiple pathosystems vary in space and time because of their inherent structure of relationships, and also in response to management. Examples of variation in multiple pathosystems are given, of groundnut in Côte d'Ivoire, of wheat in Brittany, and of upland rice in northern Laos. Variation in the yield-reducing effects of multiple pathosystems is discussed, including interactions among disease elements, relationships with attainable performances, and linkages with production situations. Progress has been achieved in understanding the links between injury profiles, production situations, and attainable performances. Questions about the functioning and consequences of multiple pathosystems are central to defining the scientific bases for, the design of, and the implementing of IPM. The complexity of multiple pathosystems, however, remains a deterrent, not a challenge, to many plant pathologists. Progress achieved in designing production systems for hardy wheat in France, however, is very promising, because of the multidisciplinary science it involves, and because of the promise to deliver it carries. The concepts of epidemiological guilds and of guilds of harmful agents are offered as perspectives to address and manage syndromes of production and syndromes of disease.

Relevance of multiple pathosystems in botanical epidemiology

Multiple pathosystems as a research theme

The study of multiple pathosystems has played a central role in the development of botanical epidemiology. As a subject, it is the equivalent in botanical epidemiology of community ecology in general ecology. Research in the field has led to the development of a body of approaches, often statistical and multivariate, as the objective often has been mostly descriptive, rather than explanatory. Studies on multiple pathosystems (i) led to attempts to understand and manage them (e.g., Jörg et al., 1987; Daamen et al., 1989; Bastiaans and Daamen, 1994), (ii) resulted in analyses of case-studies, and efforts dealing with specific cases and contexts (e.g., Hamelink et al., 1988; Avelino, 1999), and (iii) often were perceived as practical endeavours only. Studies of multiple pathosystems, dealing with a complex subject, inviting complex analyses, and leading to complex interpretations, have arguably led to results that were difficult to share. Arguably, this type of research often addresses open-ended questions, not specific

hypotheses. Its value however is multifold: first, it provides a framework for other, hypothesis-specific, epidemiological research; second, it allows the description so furthering hypotheses for future research; and third, in some successful cases, it has allowed escape from idiosyncrasies and has generated some useful generalisations.

''*No one can be a good observer unless he is a good theorizer*'' (Charles Darwin, quoted from Zadoks, 1972). There are very few plant populations that are exposed to one disease only. Multiple pathosystems are where initial observations are made, where incipient hypotheses are borne, where these are tested, and sometimes result in success in managing diseases. This paper is not intended as a review of so vast a topic. Its aim is only to highlight some important points, research issues, and research avenues. Our purpose is to touch upon a limited number, but very different, aspects of the subject. References therefore are used only to illustrate research themes and approaches, and are given with no intention of offering a comprehensive overview.

Multiple pathosystems as facts

Multiple pathosystems consist of a series of disease elements that are present in the same host stand. Over time, e.g., during the cycle of a field crop, a number of diseases may appear, spread, decline, and interact among themselves and the growing crop. Figure 1 shows a series of principal component analyses on three very different multiple pathosystems. Principal component analysis is used here as one convenient means to provide a preliminary overview of very complex structures. For instance, the multiple pathosystem of groundnut in Côte d'Ivoire involves a series of fungal pathogens (Savary, 1987a) affecting the foliage (*Cercospora arachidicola*, *Cercosporidium personatum*, *Puccinia arachidis*), shoots, and stems (*Corticium rolfsii*, *Aspergillus niger*), and pods (*Botryodiplodia* sp.). Another principal component analysis highlights a series of wheat diseases in Brittany: eyespot, brown rust, septoria blotch and yellow rust. A third example illustrates an analysis on upland rice injuries in northern Laos (IRRI, 1998), which involves an array of injuries by insects (stem borers causing dead hearts and white heads, root injuries by white grubs), foliage injuries (caused by several species), disease injuries caused by fungi (neck and leaf blast caused by *Magnaporthe grisea*, brown spot caused by *Cochliobolus miyabeanus*, sheath blight caused by *Rhizoctonia solani*, sheath rot caused by *Sarocladium oryzae*), and weed infestation by a number of species. Not

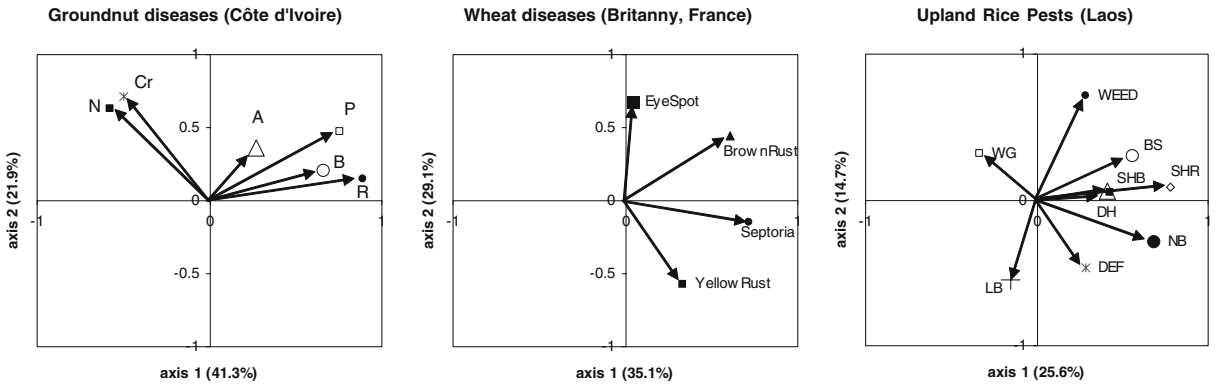

Figure 1. Three multiple pathosystems portrayed by principal component analyses: groundnut diseases in savanna and forest environments in Côte d'Ivoire, wheat diseases in Brittany, France, and upland rice in northern Laos. Left: Principal components analysis on 209 farmers' fields in several provinces (forest and savanna) of Côte d'Ivoire, 1982–1985; N: *Aspergillus niger* rot; Cr: *Corticium rolfsii* rot; A: *Cercospora arachidicola* leaf-spot; P: *Cercosporidium personatum* (*Phaeoisariopsis personata*) leaf-spot; B: *Botryodiplodia* pod rot; R: rust; Centre: wheat diseases in Britanny in a series of variety trials at varying levels of inputs, 2000, 2001, and 2003. Vectors indicate intensities of eyespot (*Tapesia yallundae*), of brown rust (*Puccinia recondita*), septoria (*Mycosphaerella graminicola*), and yellow rust (*Puccinia striiformis*). Data are from 180 individual plots (2.6×15 m) combining four crop management practices with five wheat cultivars in the replications over the 3 years. Right: upland rice pests in northern Laos, 1996 and 1997; LB: leaf blast; DEF: defoliating insects; NB: neck blast; DH: dead heart caused by stemborers; SHB: sheath blight, SHR: sheath rot; BS: brown spot; WEED: weed infestation; WG: white grub injury. Proportion of variances accounted for are indicated along each axis.

all the disease elements that actually were present in each of the three examples are represented. The three data sets used here correspond to different contexts for data acquisition (see legend of Figure 1). The groundnut data were collected during a multiyear survey in several provinces of Côte d'Ivoire, on 309 different farmers' fields (Savary, 1987a). The wheat data correspond to a series of varietal trials at different levels of inputs conducted over three different climatic years in Brittany (Rolland et al., 2003). The upland rice data were collected in a series of on-farm field experiments, where different fertiliser regimes were tested during two successive years (IRRI, 1998; Roder and Savary, unpublished data).

Some of the analyses involve more disease elements than others, and one of them involves more than just plant pathogens. Deriving conclusions on the overall importance of diseases in each pathosystem from the mere number of elements would of course be incorrect. These analyses only provide a view of possible associations, suggesting relationships, or absence of relationships, among disease elements. The relationships that seem to emerge from these summary analyses develop against the background of a large number of factors, including crop development stage, or crop management. For instance, a linkage appears in the groundnut multiple pathosystem between N (*A. niger*) and Cr (*C. rolfsii*); by contrast, there seems to be independence between Cr and A (*Cercospora arachidicola*) in the groundnut pathosystem, and independence between NB and LB (neck and leaf blast, respectively, both caused by *Magnaporthe grisea*) in the upland rice pathosystem. Collinearity or non-collinearity of (disease) vectors may lead to forwarding hypotheses, which in turn would require additional analyses.

Shapes of botanical pathosystems

Elements of multiple pathosystems

Multiple pathosystems have shapes, where individual diseases display a particular role. Several studies have shown that multiple pathosystems vary in shapes. Only two of the many reasons for change are illustrated here.

Change in age, i.e., development of the host stand, is one strong reason for change in the shape of multiple pathosystems. Figure 2 shows three separate principal component analyses on the groundnut–leaf-spot–rust pathosystem at three different ages of the groundnut stands. Although the analyses pertain to the same farmers' fields, comparison of analysis of Figure 2a (young stands), 2b (middle-age stands), and 2c (stands approaching or at harvest stage) indicate strong variation in relationships among variables. In young groundnut stands, a strong relationship between rust (R) and early leaf-spot (A) is apparent, both diseases being opposed to *A. niger* (N) wilt. In middle-age fields, a very strong association between rust (R) and late leaf-spot (P) is indicated, both diseases being opposed to early leaf-spot (A). In older fields, the relationship between rust and late leaf-spot has become loose, although both

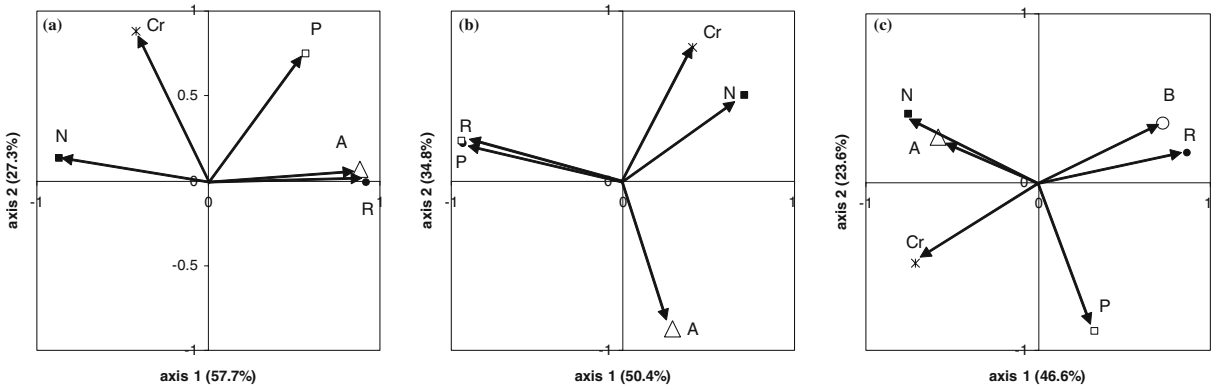

Figure 2. Patterns of change in multiple pathosystems over time and host development: principal component analyses of disease levels in farmers' fields in Côte d'Ivoire. (a) Groundnut field at early development stages (first trifoliate leaf – flowering); (b) groundnut field at medium development stages (flowering – pod filling); (c) groundnut field at final development stages (pod filling – harvest stage). Symbols for disease vectors are the same as in Figure 1.

remain opposed to early leaf-spot; and a linkage between *A. niger* and early leaf-spot is detected, which did not exist at earlier ages. These shifts in relationships are reflections of the respective dynamics of foliar diseases (rust and early leaf-spot usually establish earlier in a crop stand than late leaf-spot, while early leaf-spot generally declines as crop maturity approaches), and of environmental factors that favour certain diseases at certain stages of the crop (humid environment and contaminated seeds favour *A. niger* wilt and *C. rolfsii* in the early crop stages; dense, green canopies favour rust and late leaf-spot, whereas water stresses and poor soil fertility favour late leaf-spot in established stands; and more humid environments favour *Botryodiplodia* pod rot, late leaf-spot, and rust while drier environments favour *A. niger* wilt and early leaf-spot in older stands). One important factor that drives relationships among foliar diseases is disease-induced-defoliation. It will be addressed later in this discussion.

Crop management is another major reason for changes in shapes of multiple pathosystems. Figure 3 is an illustration of the effects of four crop management patterns in wheat experiments. Strong shifts in disease vectors are detected in Figure 3a–d, the transition from a to d corresponding to intensified wheat production. While in Figure 3a all diseases, except yellow rust, appear closely associated, an opposition between eyespot and both septoria leaf blotch and brown rust develops in Figure 3b, which persists in Figure 3c, but disappears in Figure 3d. Such sharp changes must be attributed to changes in fertiliser inputs,

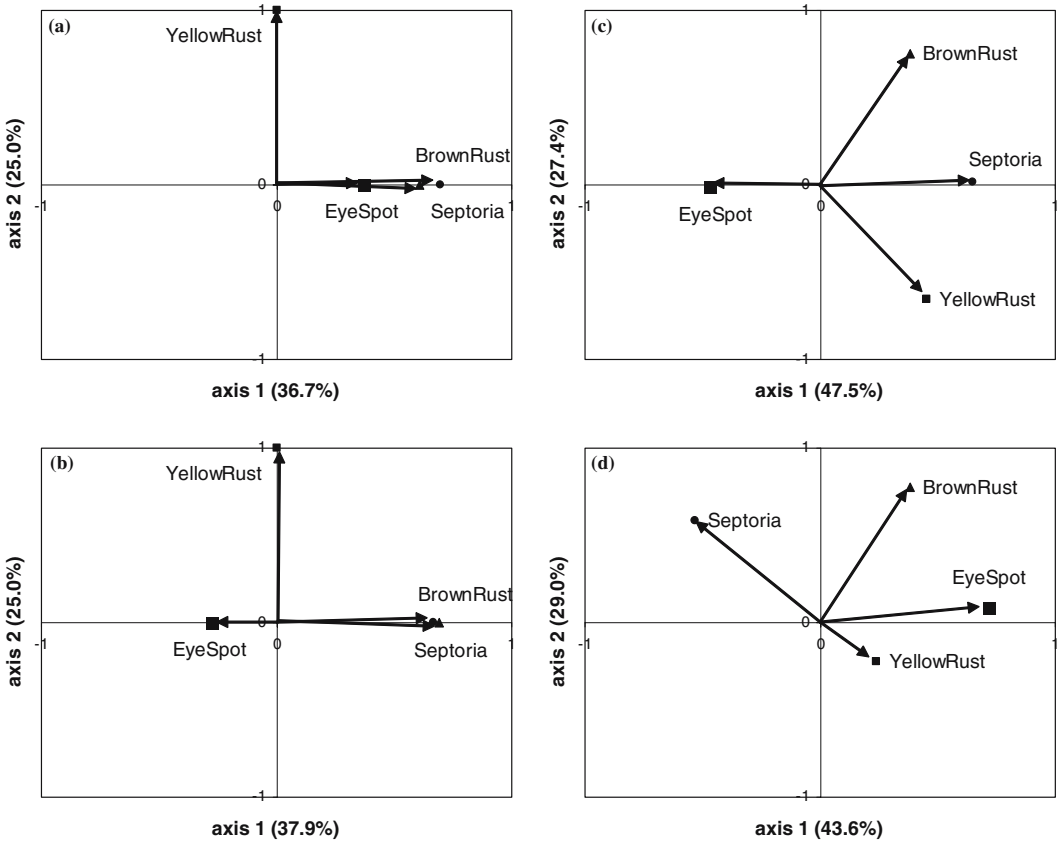

Figure 3. Patterns of change in multiple pathosystems over crop management practices: principal component analyses of disease levels in field trials in Britanny, France. (a) pattern of crop management A: fertiliser input with a target yield of 10 t ha^{-1}; seeding rate: 250 seeds m^{-2}; use of a crop growth regulator; three fungicide applications; (b) pattern of crop management B: fertiliser input with a target yield of 9 t ha^{-1}; seeding rate: 250 seeds m^{-2}; use of a crop growth regulator; two fungicide applications; (c) pattern of crop management C: fertiliser input with a target yield of 8 t ha^{-1}; seeding rate: 150 seeds m^{-2}; no growth regulator; one fungicide applications; (d): pattern of crop management D: fertiliser input with a target yield of 7 t ha^{-1}; seeding rate: 150 seeds m^{-2}; no crop growth regulator; no fungicide applications.

seeding rates, plant hormone use, and of course fungicide use. Crop management, involving pesticide use or not, has been found to be a major factor for changes in shapes of multiple pathosystems, in many diverse examples, including, e.g., wheat in Australia (Stynes, 1980) and the Netherlands (Daamen et al., 1989), lowland rice in Asia (Savary et al., 2000a), pea in Idaho (Wiese, 1982), groundnut in Côte d'Ivoire (Savary, 1987a), or coffee in Honduras (Avelino, 1999).

Shapes of multiple pathosystems in space

The spatial distributions of four different pathogens in the same crop stand are shown in Figure 4 (Lannou and Savary, 1991): rust (*Puccinia arachidis*), early leaf-spot (*Cercospora arachidicola*), late leaf-spot (*Cercosporidium personatum*), and web blight (*Rhizoctonia solani*) of groundnut. Several techniques, including geostatistical and multivariate, were used to show, as the maps strongly suggest, that (i) rust (Figure 2a) and web blight (Figure 2d) are spatially strongly associated, (ii) early leaf-spot (Figure 2b) is more severe where rust is less severe, and (iii) late leaf-spot (Figure 2c) does not intensify strongly where rust or early leaf-spot severities are extreme. The maps, which were drawn at the end of a cropping season, also show that two very different types of epidemics developed in the same stand, a typically focal epidemic (Figure 2d, web blight), and three general epidemics (Figures 2a, 2b and 2c, rust, early leaf-spot, and late leaf-spot, respectively) which did intensify locally.

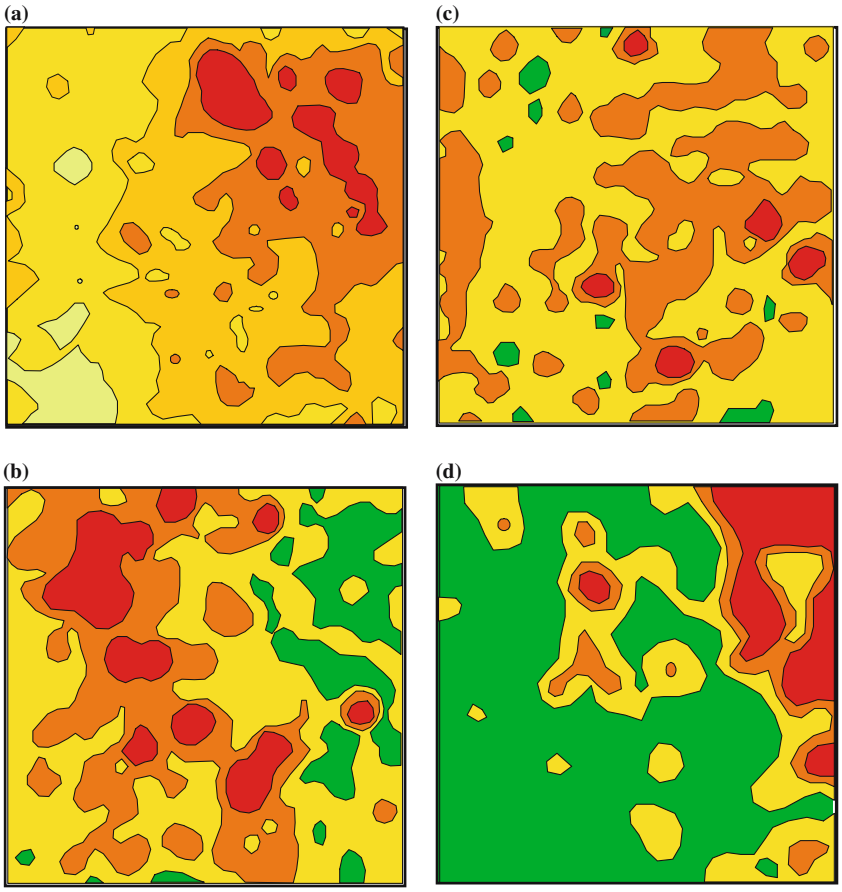

Figure 4. Patterns of change in multiple pathosystems over space: spatial distribution of four diseases a groundnut plot, Côte d'Ivoire (Lannou and Savary, 1991, modified). (a) Groundnut rust, *Puccinia arachidis*; (b) Early leaf-spot, *Cercospora arachidicola*; (c) Late leaf-spot, *Cercosporidium personatum* (*Phaeoisariopsis personata*), (d) Web blight, *Rhizoctonia solani*. Disease assessments were made at 90 days after sowing. Rust, early leaf-spot, and late leaf-spot: severity (% diseased leaf area) scales; web blight: incidence (% diseased plants) scale. From Lannou and Savary, 1991, modified.

Analysis of spatial patterns in multiple pathosystems may lead to a number of hypotheses, as in the case of multiple infection of hop stands by different viruses (Pethybridge and Turechek, 2003), which can lead to experimentally testing spatial co-occurrence and variation of infections in the host–vector–viruses system. In the case of groundnut diseases, much of the spatial co-variation of disease intensities may be attributed to competition towards vacant sites and defoliation of diseased tissues by some of the pathogens.

Nelson and Campbell (1993) studied a far more complex multiple pathosystem, which involves eight fungal pathogens of white clover (*Rhizoctonia solani*, *Pseudomonas andropogonis*, *Stagonospora meliloti*, *Cercospora zebrina*, *Curvularia trifolii*, *Colletotrichum trifolii*, *Polythrincium trifolii*, and *Uromyces* sp.), in presence or absence of three virus diseases (alfalfa mosaic virus, yellow vein virus, and peanut stunt virus). They detected disease aggregation of the fungal disease complex at several scales (leaf, plant, population), which fluctuated over time as foliation and defoliation occurred, and which varied spatially as well. Changes over time in the spatial aggregation of the foliar disease complex were associated with changes in disease severity itself and defoliation. The background of varying, multiple virus infection did not seem to affect either the dynamics of the foliar disease complex, or its spatial pattern.

Shapes of multiple pathosystems over time: dynamics of multiple pathosystems

One approach to addressing the dynamics of multiple pathosystems is by means of linked differential equations. The equations themselves can assume a number of shapes, but are in many respects fairly similar. One important difference however among seemingly analogous systems of equations is the nature of the modelled diseases variables, which often are proportions (severities or incidences, i.e., disease densities) or less frequently (as in the model used below), amounts of diseased tissues. This difference has important consequences on the meaning of parameters used. Use of linked differential equations derives in botanical epidemiology from the approach Van der Plank (1963) introduced to the field, which itself is related to earlier ecological models, including especially the Verhulst-Pearl logistic equation and systems of equations of the Lotka-Volterra type (Pianker, 1983, cited in Madden et al., 1987). Use of this approach has been extensive in botanical epidemiology, and has been based on both the development of simple equations that adequately describe disease progress over time (see, e.g., Madden, 1980; Campbell and Madden, 1990), and on tools to numerically integrate sets of differential equations that constitute simulation models (Zadoks, 1971; Zadoks and Rabbinge, 1985).

The groundnut–leaf-spot–rust pathosystem represents a good example to illustrate the approach. This system is fairly simple, having only two disease components, rust and leaf-spot, but leads to considering several processes and interactions: (i) a biotrophic pathogen which multiplies only on healthy, green tissues, (ii) a necrotrophic pathogen (only *Cercosporidium personatum* is considered here, but both *C. personatum* and *Cercospora arachidola* could be considered collectively) which causes extensive defoliation, which (iii) compounds physiological (senescence) defoliation, (iv) competition between the two pathogens in their access to growing crop tissues, and (v) the ability of one of the two diseases (leaf-spot) to multiply from defoliated, infectious tissues (Savary and Servat, 1991). Such characteristics are very similar to another legume-based multiple pathosystem, the bean–angular leaf-spot–anthracnose–rust system (Gomes Carneiro et al., 2000; Bassanezi et al., 2001; de Jesus et al., 2001), which has been extensively studied. A series of linked differential equations representing the groundnut–leaf-spot–rust pathosystem are given in Table 1, corresponding to the overall model structure of Figure 5. Parameters for the model (relative rates of crop growth, of increase of both diseases, of physiological defoliation, and of disease-induced defoliation) were estimated (Savary and Servat, 1991) using a numerical integration procedure coupled with a sequence of two optimisation procedures (Rosenbrock, 1960; Nelder and Mead, 1964), applied to a set of 15 epidemics where levels of the both diseases were artificially manipulated (Savary and Zadoks, 1992a).

Simulated outputs using optimised parameters are shown in Figure 6a. A regular, logistic-shaped increase of leaf-spot is combined with a faster increase of rust, which declines in the later stage of the epidemic; these are coupled with a regular

Table 1. Equations used in a rust–leaf-spot–groundnut multiple pathosystem simulation model

Equation	Hypotheses
(1) Rate of increase of leaf area: $dL/dt = RRL * L\{1 - [(L + Y + Z + totD)/L_{max}]\}$	The rate of increase of leaf area is proportional to a relative rate and the amount of (healthy) leaf, corrected for the fraction of leaf growth, relative to a maximum. Leaf growth includes defoliated tissues
(2) Rate of rust increase: $DY/dt = RRY * Y\{1 - [(Y + Z)/(L + Y + Z)]\}$	The rate of rust increase is proportional to a relative rate and the amount of rust-diseased tissues, corrected for the fraction of relative growth, relative to the current total leaf tissues
(3) Rate of leaf-spot increase: $DZ/dt = (RRZ * Z + RRZDZ * DZ) * \{1 - [(Z + Y)/(L + Y + Z)]\}$	The rate of leaf-spot increase is proportional to both (1) a relative rate for standing diseased tissues and the amount of leaf-spot-diseased tissues and (2) a relative rate for defoliated diseased tissues and the amount of defoliated infected tissues, corrected for fraction of relative growth, relative to the current total leaf tissues
(4) Rate of defoliation (healthy tissues) $dD/dt = RRDS * L + RRDZ * [Z/(L + Y + Z)] * L$	The rate of defoliation of healthy tissues is the sum of senescence-induced (relative rate and healthy tissues) and indirectly leaf-spot-induced (relative rate, proportion leaf-spot-diseased, and healthy tissues)
(5) Rate of defoliation (leaf-spot-diseased tissues) $dDZ/dt = (RRDS + RRDZ) * Z$	The rate of defoliation of leaf-spot-diseased tissues is proportional to a relative rate (senescence and leaf-spot accumulated) and to the amount of leaf-spot-diseased tissues
(6) Rate of defoliation (rust-diseased issues) $dDY/dt = (RRDS + RRDZ) * Y$	The rate of defoliation of rust-diseased tissues is proportional to a relative rate (senescence and leaf-spot accumulated) and to the amount of rust-diseased tissues

Variables (dimensions in brackets): L: healthy leaf tissues [L^2]; Y: rust-diseased tissues [L^2] ; Z: leaf-spot-diseased tissues [L^2]; D: healthy, defoliated tissues [L^2]; DY: rust-diseased, defoliated tissues [L^2]; DZ: leaf-spot-diseased, defoliated tissues [L^2]; RL: rate of leaf growth [$L^2\ T^{-1}$]; RY: rate of rust increase [$L^2\ T^{-1}$]; RZ: rate of leaf-spot increase [$L^2\ T^{-1}$]; RD: rate of defoliation of healthy tissues [$L^2\ T^{-1}$]; RDY: rate of defoliation of rust-diseased tissues [$L^2\ T^{-1}$]; RDZ: rate of defoliation of leaf-spot diseased tissues [$L^2\ T^{-1}$]; RRL: relative (intrinsic) rate of leaf growth [T^{-1}]; L_{max}: maximum leaf growth [L^2]; RRY: relative rate of rust increase [T^{-1}]; RRZZ: relative rate of leaf-spot increase from non-defoliated tissues [T^{-1}]; RRZDZ: relative rate of leaf-spot increase from infected defoliated tissues [T^{-1}]; RRDS: relative rate of defoliation (senescence) of healthy tissues [T^{-1}]; RRDZ: relative rate of defoliation of (leaf-spot) diseased tissues [T^{-1}]; totD: accumulated defoliation [L^2].

accumulation of defoliated tissues, and a bell-shaped green leaf area curve. When the relative rate of rust increase is reduced (Figure 6b) or increased (Figure 6c), a strong rust reduction, or increase, respectively, is simulated, coinciding with opposite behaviour of the leaf-spot epidemic. When the relative rates of leaf-spot increase from both non-defoliated and defoliated tissues are reduced (Figure 6d), the leaf-spot epidemic is strongly reduced, while the rust epidemic is strongly increased. When both these parameters are increased (Figure 6e), the leaf-spot epidemic is strongly increased, the rust epidemic is nearly halved, and the amount of defoliation is increased. A last set of tests, where the relative rate of defoliation induced by leaf-spot is varied is also shown. When the leaf-spot induced-defoliation rate is decreased (Figure 6f), the amount of total defoliation is barely altered, the leaf-spot epidemic remains essentially unaffected, whereas the rust epidemic is slightly enhanced. Increase of this parameter (Figure 6g), on the other hand, leads to only a slight increase in total defoliation, an unaffected leaf-spot epidemic, but a reduction by about 8% of the rust epidemic.

The overall picture this model gives is that the system is quite sensitive to comparatively small variation (10% or less) in parameter values. Leaf-spot is behaving as a very strong competitor for rust, especially through leaf-spot induced-defoliation. Rust on the other hand can rapidly take advantage of seemingly small increases in the amount of available healthy tissues. These interactions are strongly influenced by crop growth (and the effects of diseases on crop growth), and defoliation (especially through physiological senescence). One interesting response of the system is that a reduced retention (greater disease-induced

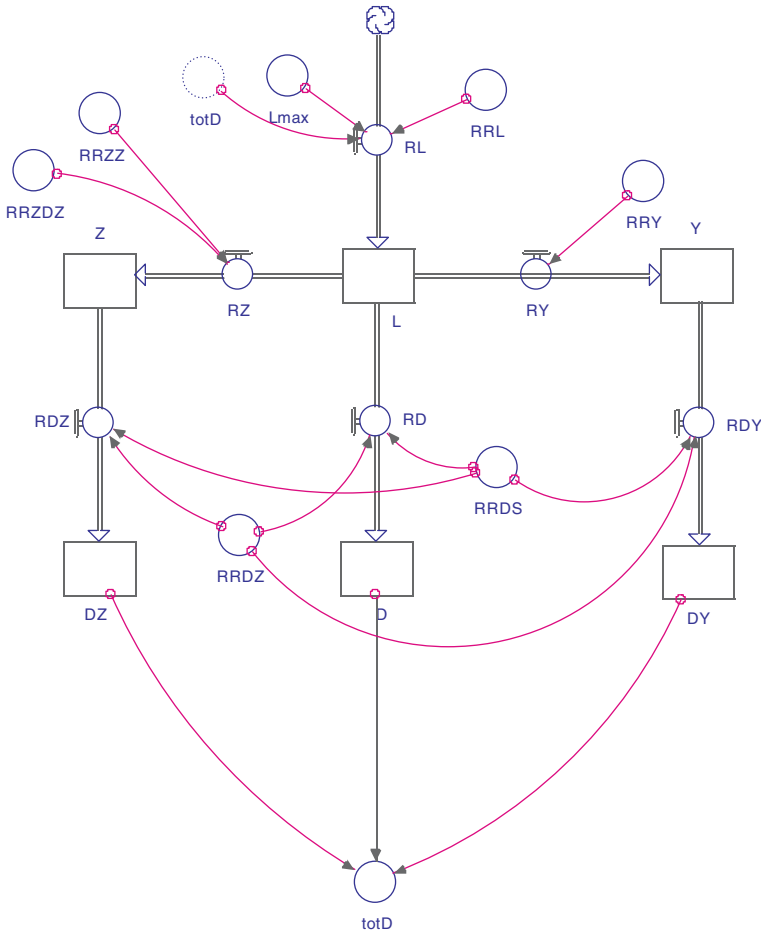

Figure 5. Overall structure of a mechanistic simulation model incorporating two foliar diseases, rust and leaf-spot on groundnut. State variables: L: healthy leaf tissues [L^2]; Y: rust-diseased tissues [L^2]; Z: leaf-spot-diseased tissues [L^2]; D: healthy, defoliated tissues [L^2]; DY: rust-diseased, defoliated tissues [L^2]; DZ: leaf-spot-diseased, defoliated tissues [L^2]; Rates: RL RL: rate of leaf growth [$L^2\ T^{-1}$]; RY: rate of rust increase [$L^2\ T^{-1}$]; RZ: rate of leaf-spot increase [$L^2\ T^{-1}$]; RD: rate of defoliation of healthy tissues [$L^2\ T^{-1}$]; RDY: rate of defoliation of rust-diseased tissues [$L^2\ T^{-1}$]; RDZ: rate of defoliation of leaf-spot diseased tissues [$L^2\ T^{-1}$]; Parameters: RRL: relative (intrinsic) rate of leaf growth [T^{-1}]; L_{max}: maximum leaf growth [L^2]; RRY: relative rate of rust increase [T^{-1}]; RRZZ: relative rate of leaf-spot increase from non-defoliated tissues [T^{-1}]; RRZDZ: relative rate of leaf-spot increase from infected defoliated tissues [T^{-1}]; RRDS: relative rate of defoliation (senescence) of healthy tissues [T^{-1}]; RRDZ: relative rate of defoliation of (leaf-spot) diseased tissues [T^{-1}]; totD: accumulated defoliation [L^2].

defoliation) of leaf-spot infected tissues (Figure 6a vs. 6g) leads to a reduced rust epidemic. Selecting varieties that shed their leaves at low leaf-spot severity might then be an efficient way of reducing rust epidemics.

The white clover–foliar fungal diseases–viruses system (Nelson and Campbell, 1993) renders the groundnut–rust–leaf-spot system a comparatively simple system to address. Nelson and Campbell question the relevance of the approach illustrated above, which in the case of the clover-based system would require a set of at least 10 equations. If this approach were to be taken, a complex model structure would have to be designed, a large number of parameters would have to be estimated, and numerical solutions would become difficult to interpret. The approach chosen by Nelson and Campbell (1993) in their field work, however, was not to consider each disease separately, but to quantify the leaf disease complex as a whole. This leads to the interesting avenue of perhaps considering groups of pathogens that share similar

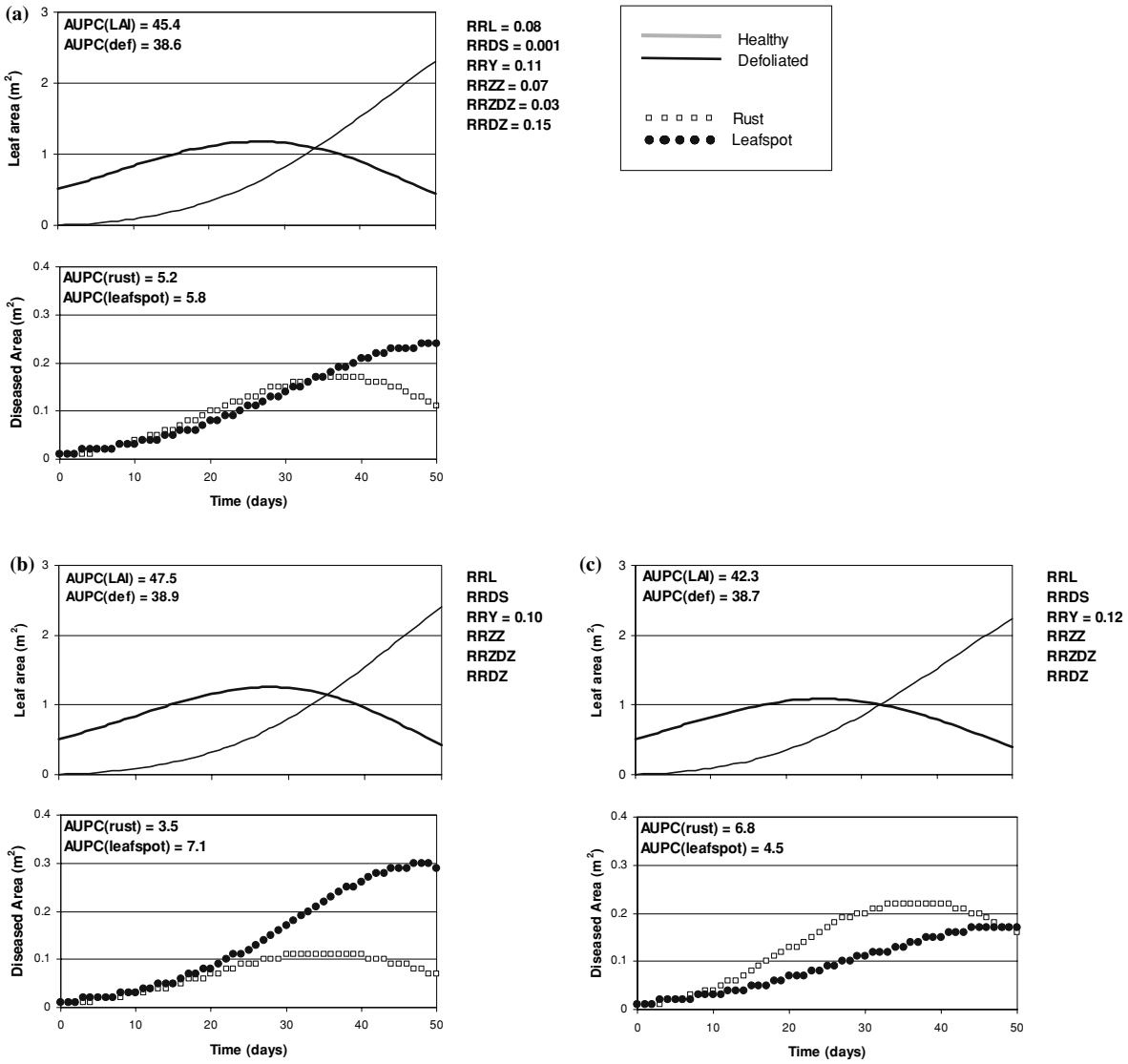

Figure 6. Two foliar diseases dynamically interacting, simulated rust and leaf-spot epidemics on groundnut. Each simulation is represented by two graphs of outputs, healthy and (total) defoliated area (upper half) and rust and leaf-spot severities (lower half). Numerical values of parameters are indicated in graphs b–g only when changes from reference (optimised) values (graph a) have been used. Calculated areas under progress curves (AUPC) of the rust epidemic, of the leaf-spot epidemic, of the leaf area index, and of the defoliated leaf area index are indicated. (a) simulated outputs with optimised parameter values for RRL (relative rate of leaf growth; [T^{-1}]), RRDS (relative rate of senescence defoliation of healthy tissues [T^{-1}]), RRY (relative rate of rust increase [T^{-1}]), RRZZ (relative rate of leaf-spot increase from non-defoliated tissues [T^{-1}]), RRZDZ (relative rate of leaf-spot increase from infected defoliated tissues [T^{-1}]), and RRDZ (relative rate of defoliation of leaf-spot diseased tissues [T^{-1}]). (b) simulated outputs for a reduced relative rate of rust increase (RRY). (c) simulated outputs for an increased relative rate of rust increase (RRY). (d) simulated outputs for reduced relative rates of leaf-spot increase (RRZZ and RRZDZ). (e) simulated outputs for increased relative rates of leaf-spot increase (RRZZ and RRZDZ). (f) simulated outputs for a reduced relative rate of defoliation induced by leaf-spot (RRDZ). (g) simulated outputs for an increased relative rate of defoliation induced by leaf-spot (RRDZ).

functional attributes in a community, and model the dynamics of guilds of pathogens, rather than of individual diseases.

One of the many criticisms of the linked differential equation approach (see, e.g., Nelson and Campbell, 1993) is its inability to account for

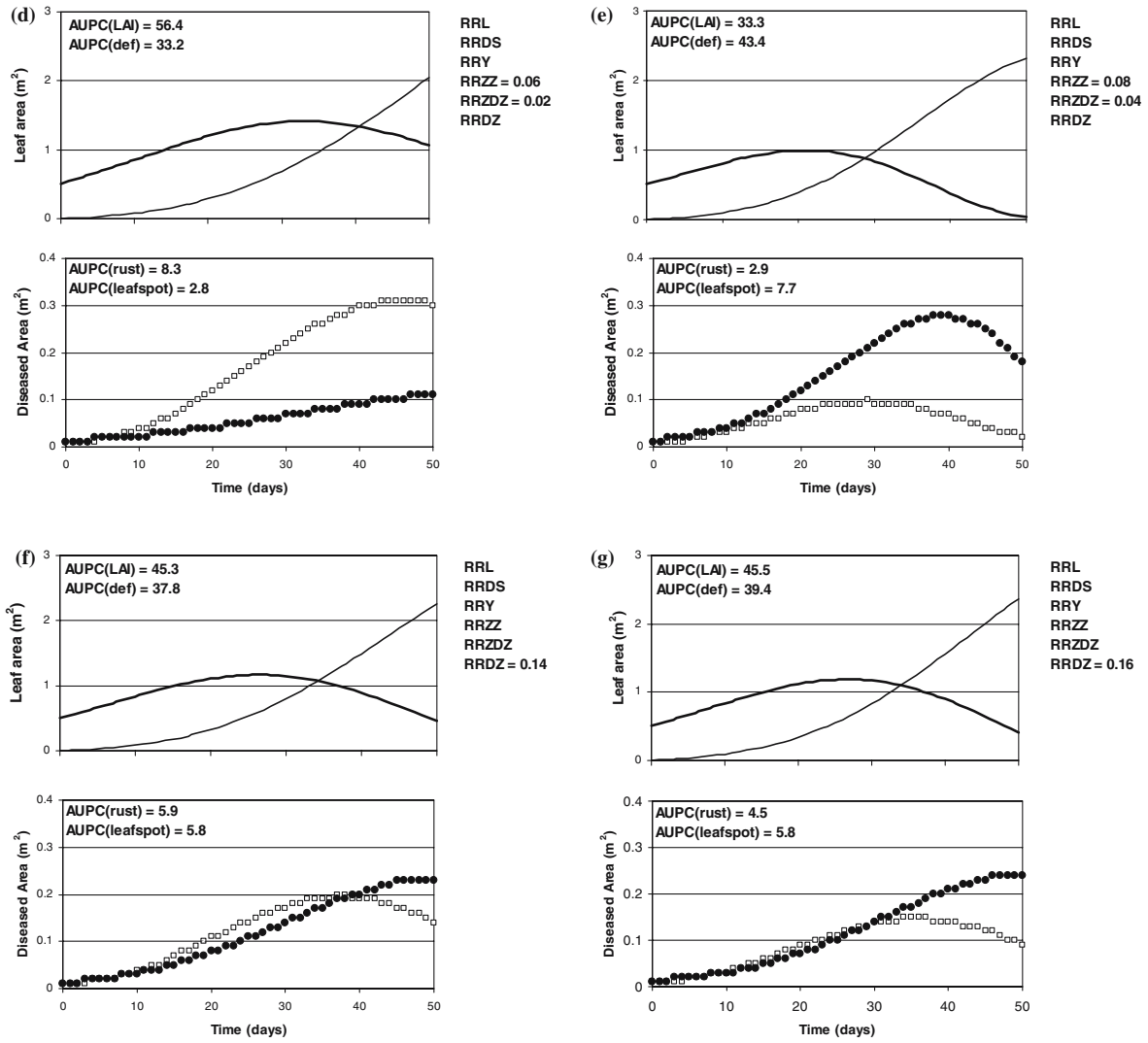

Figure 6. Continued

spatial patterns, and the essential effects of spatial patterns on disease dynamics. Figure 4 is sufficient evidence of the fact that the groundnut–leaf-spot–rust system is no exception. The model summarised here does however include implicit assumptions pertaining to the distribution of disease on host tissues. As in many pathosystems involving foliar diseases, a very strong vertical aggregation occurs in both the leaf-spot (Boote et al., 1980) and the rust (Savary, 1987b) pathosystems. Such aggregation of diseases (and defoliation) along the vertical dimension of a crop canopy must have very strong consequences on the behaviour of the multiple pathosystem. Considering the model outlined in equations of Table 1 and the flowchart of Figure 5, however, amounts to implicitly considering a growing canopy with two layers: (i) a (healthy) layer where defoliation is caused by senescence and distance effect of leaf-spot disease, and (ii) a (diseased) layer, where both rust and leaf-spot lesions occur, and where defoliation is caused by both leaf-spot and senescence effects.

The purpose of this type of model is to explore interactions within a framework of thinking defined, and limited by, a set of hypotheses. Expanding the model to address additional, albeit important, features of the considered system might prevent the interpretation of simulation results,

evaluation of the model, and its use. Further discussion on strategic models of this kind is given in McRoberts et al. (2003).

Multiple pathosystems and damage to crops

Multiple injuries and the resulting damage

Consideration of multiple pathosystem, i.e., pathogens dynamically interacting among themselves and a growing canopy, inevitably leads to questions regarding harmfulness of the multiple pathosystem. The extent of damage (*sensu* Zadoks, 1985, i.e., yield reduction) caused by a multiple pathosystem has scientific relevance of its own; it also has very practical implications with respect to the availability, efficiency, and deployment of management options. Plant pathologists did not study multiple pathosystems because of the interesting interactions among competing pathogens that may take place, and chose them because they were good examples for community ecology studies; they did not either address the issue with the prime objective of selecting plants that would be resistant against several diseases, or crops whose yields would be stable in the presence of several diseases. One simple, practical reason was the need to assess the damage caused by several injuries. Padwick (1956) made an early attempt to such quantification, with the formula:

$$\begin{aligned}&\text{Percent yield loss}\\&= \{100\\&\quad - [(100 - P1)(100 - P2)\\&\quad \times (100 - Pn)]/100n - 1\}\end{aligned}$$

where Pi is the percent yield loss caused by an individual injury i. Padwick's model assumes that the only interaction between diseases on yield is competition for the crop's resources. This amounts to forwarding the hypothesis that one disease cannot affect what other diseases have already injured (Johnson et al., 1986). Padwick's view strongly contrasted with several later analyses, whereby individual losses were merely accumulated in a 'loss profile' (see, e.g., Pinstrup-Andersen et al., 1976). Teng (1994) pointed to the fact that this latter reasoning may lead to the impossible result that diseases, or crop harmful agents in general, may cause losses exceeding 100%.

Quantification, analysis, and modelling damage is both the scientific-technical cornerstone for disease management (James, 1974; Chiarappa, 1980; Madden, 1983; Teng, 1983; Zadoks, 1985; Teng, 1987; Gaunt, 1995) and one of the very important entry points for disease management (see, e.g., Teng and Savary, 1992). This has been a very broad and active field of research for many decades. The subject has particular relevance when considering multiple pathosystems (Madden and Nutter, 1995), however, and a few points are discussed here.

Five directions

As opposed to an additive model for combined effects of injuries on damage, the model developed by Padwick was a useful starting point, from which several directions of thoughts were explored.

(i) A first direction concerns the nature of disease interactions in their yield-reducing effect: very often, a less than additive effect is observed, but some injuries may synergistically increase yield losses. Less than additivity has been demonstrated in one of the best documented studies on damage caused by a multiple pathosystem, the early blight–verticillium wilt–potato leafhopper of potato (Johnson, 1986; Johnson et al., 1986, 1987; Johnson and Teng, 1990). Injuries caused by diseases may, however, synergistically reduce yield. Synergies in yield-reducing effects are found in potato early dying caused by *Verticillium dahliae* and *Pratylenchus penetrans* (Francl et al., 1990), as well as in combinations of infections of wheat by *Septoria nodorum* and *Puccinia recondita* (Van der Wal et al., 1970). A first hypothesis therefore refers to the direction, positive or negative, of combined injuries on damage.

(ii) A second direction concerns the nature of competition, which may only be for 'resource', as Padwick's model refers, or may involve other mechanisms, resulting then in damage lower than expected from Padwick's model. Such is the case in the potato–early blight–verticillium wilt–leafhopper studied by Johnson (1986) and Johnson et al. (1986).

(iii) A third direction concerns the way damage measurements are expressed. Damage is commonly reported as percentage. The reported figures (percent losses) are therefore dependent on

the level of the uninjured yield reference, and their meaning will strongly depend on whether this yield reference is low or high. One of many alternatives to reporting damage as a proportion is expressing it as a biomass, and the choice will depend on the end-use of the information.

(iv) A fourth direction concerns the relationships that link damage to the various injuries (the damage function; Zadoks, 1985). The damage function may involve the yield pertaining to a given production situation as an explanatory variable (the attainable yield; Rabbinge and De Wit, 1989). As is the case with the damage caused by single harmful agents (see, e.g., Rossing, 1991, for the grain aphid on wheat, and Rabbinge et al., 1985, for powdery mildew in wheat), the amount of damage caused by a multiple pathosystem may depend on the level of attainable yield. This has been exemplified in the case of the multiple pathosystem of groundnut in Côte d'Ivoire (Savary and Zadoks, 1992b), and in the more complex and diverse multiple pathosystem of lowland rice in Asia (Savary et al., 2000b).

(v) A fifth direction of thought follows the realisation that diseases, and any harmful agent in general, belong to one or a few categories, based on the type of injury mechanisms they trigger. Rabbinge and Rijsdijk (1981) and Boote et al. (1983) defined the limited number of ways for a harmful organism to hamper the physiological performances of a growing canopy. This has particular relevance when considering multiple pathosystems for two reasons. First, it provides a basis for designing experiments, developing field survey procedures, and defining field measurements that refer not to specific diseases (or harmful agents) but to specific injuries. Diseases then are not measured with respect to how fast they intensify, but rather to how much they may affect the performances of a crop. Further, they need not necessarily be measured individually, but collectively, as Nelson and Campbell (1993) did. Second, this categorization provides a framework for modelling mechanistically the physiological interactions between a crop stand and a multiple pathosystem. This direction of thought has been underpinning research involving simulation models as tools for understanding damage caused by diseases and means to reduce them (Rouse, 1988; Rabbinge et al., 1989; Gaunt, 1995), including work conducted on multiple pathosystems such as potato–early blight–verticillium wilt (Johnson, 1986; Johnson et al., 1986, 1987; Johnson and Teng, 1990).

New developments have taken place, where these five points are considered in the case of lowland rice in Asia (Pinnschmidt et al., 1995; Willocquet et al., 2000, 2002, 2004). Simulation models have been developed that make use of the concept of guilds of injuries (Pinnschmidt et al., 1995; Willocquet et al., 2000, 2002) which have been used to analyse and understand the yield-reducing effects of several pathogens, insects, and weeds in the same crop. A modelling structure has been designed so that it can simultaneously handle production situations (as drivers of attainable crop performances) and injury profiles (as drivers of multiple injuries) in the very combinations where field characterisation had shown these (production situation) $\times$ (injury profile) associations occur (Willocquet et al., 2000, 2002)). Production situations and their associated injury profiles were then used as the modelling context where disease and pest management tools could be most efficiently deployed, and where progress should be expected, and so expressed in yield gains, instead of yield losses (Willocquet et al., 2004).

Multiple pathosystems and integrated pest management

A negative view

In his article on the functioning and performances of tropical ecosystems, Janzen (1973) was expressing his frustration at science not achieving its goals in vital fields of application: "*Nearly all research in tropical agriculture is highly reductionist, parochial, and discipline-oriented*". At the time when Janzen wrote, much of the synthesis tools that now are available to plant pathologists did not yet exist. A negative view, similar to Janzen's, could be expressed considering the very slow pace of progress that has been achieved in understanding, analysing, and managing multiple pathosystems. In spite of the availability of tools to address it, the complexity of these systems remains a deterrent, not a challenge, to many plant pathologists. But the primary reason why progress has been so slow is the weakness of communication among disciplinary fields (McRoberts et al., 2003).

Questions about multiple pathosystems are central to defining the scientific bases for, the designing of, and the implementing of IPM. Reductionism, in its many forms, and discipline-oriented science, are the very same reasons that hamper progress in IPM (Jeger, 2000), and it would seem that Joni Mitchell's song ("*Hey farmer farmer. Put that DDT now. Give me spots on my apples. But leave me the birds and the bees. Please.*"; (McRoberts et al., 2003)) does not seem to be fading away anytime soon.

A positive view

There has nevertheless been a change in the way disease management has been addressed, scientifically and technically, over the past 50 years – the time-span covering the cycle of the International Epidemiology Workshops. One good example appears to be the multiple pathosystem of wheat in wheat-based systems of western Europe.

Initial steps were taken in the Netherlands with the EPIPRE project which saw scientists sharing experience with farmers, adapting theories to practice, and farmers empowered in their disease management decisions from epidemiological and systems science (Zadoks, 1989).

This early farmers-driven project had a setting different from that of today. Much work has been accomplished since, and already was on its way then, to show through long-term experiments (Jordan et al., 1985; Webster, 1985; McRoberts et al., 2000) that integrated production systems for field crops tend to perform better financially than high-input systems when commodity prices are low (McRoberts et al., 2003). The notion that disease management depends on production situations – not only because multiple pathosystems are so sensitive in their composition to crop management, but simply because disease management is only part of crop management, and so, necessarily depends on the socio-economic dimensions of what a production situation is – was no longer a theory but a concept put into practice and a way to conduct research.

The old, simple, idea that stable yield, and stable yield characteristics, including multiple, incomplete, host plant resistance are criteria for selecting varieties was revisited by G. Doussinault (Doussinault, 1998; Doussinault et al., 2001). Selecting for maximum yield and maximum grain protein content under intensive production conditions, i.e., high (nitrogen) fertiliser inputs and a pesticide umbrella leads to ignoring a large fraction of genetic resources available in a germplasm, and to restricting progress within unrealistically favourable production situations. Hardy wheat varieties, which yield-wise may perform somewhat below, or nearly as well as, conventional high yielding cultivars, but exhibit a number of incomplete resistances and have fair performances with respect to protein contents of the grain, are being tested in a number of sites in France. Incomplete resistances concern eyespot (*Tapesia yallundae*) and leaf blotch (*Mycosphaerella graminicola*). Experiments do not involve varieties only, but patterns of crop management; they also lead to assessment of economic performances. So far (over 3 years, 1999–2002), reported results are very encouraging (Rolland et al., 2003): under a low input regime, hardy wheat cultivars yields are reasonably stable (within 10 to 8 t ha^{-1}), have acceptable grain protein contents (10.5–11.5%), and are produced at costs reduced from 400 to 150 euros ha^{-1} compared to an intensive production system. In 46 (i.e., 73%) of the 63 tested combinations of (year × crop management pattern × cultivar), low-input crop management with hardy varieties generated the highest net returns. Thus in about 73% of the cases, host plant resistance to the wheat multiple pathosystem is mobilised as a tool for system stabilisation – instead of pesticides for system perturbation, and yield-driven control. Interestingly, the effort appears to be led by breeders; that economists, agronomists, and pathologists contribute to designing production systems that suit a seed-based technology is making an experience of this kind very promising, not only because of the science it involves, but because of the promise to deliver it entails.

Perspectives

Epidemiological guilds

The example of approach used above to model the dynamics of simultaneous disease epidemics epidemics invites the question of how to best address the temporal and spatial structure of multiple pathosystems whose disease components may be

numerous, or variable. Nelson and Campbell (1993) addressed a very diverse multiple pathosystem. Aside from rendering their study doable, their approach to quantify and analyse several foliar diseases collectively is an important choice, which carries the simplifying, and new, assumption that several diseases may be considered as an aggregate, from an epidemiological standpoint. One approach to analysing complex, multiple pathosystems may thus be grounded on the consideration of epidemiological guilds, rather than of individual pathogens. Definition of these guilds and clustering individual disease components in them could make use of results achieved from comparative epidemiological work (Kranz, 1974, 1980). Simplification of this kind, if successful, would have the merit of accommodating quantitative epidemiological knowledge on individual disease components, while also generating a common framework for understanding and management of multiple pathosystems that differ in their biological components, but share common epidemiologically functional traits.

Guilds of harmful agents

This concept has proven useful. The notion that harmful organisms share common injury mechanisms (Rabbinge and Rijsdijk, 1981; Boote et al., 1983) enables us to bring together organisms that otherwise profoundly differ. This has found several applications, in designing field quantification methods, field experiments to measure damage, and of course, simulation models. These in turn have found strategic applications for research prioritisation towards multiple pathosystems, for assessing the impact of new crop management and changes in production situations, and of disease management tools. It also has allowed a shift of thinking, from assessment of damage, to projection of yield gains (Willocquet et al., 2004), that is, a shift from what has been lost because of current practices, to what could be gained from future options.

Syndromes of production, syndromes of disease

The term syndrome has two definitions (Babcock Grove, 1961): (i) a group of symptoms or signs typical of a disease, disturbance, condition, or lesion in animals or plants, and (ii) a set of concurrent things. Andow and Hikada (1989) used the term to both describe different patterns of management of rice in Japan, and the plant health consequences this difference in production situations has on rice diseases. Physicians do not differ in their use of the word (e.g., Peto, 2001; Zimmet et al., 2001). The detection of linkages between production situations and injury profiles is analogous to considering corresponding syndromes of production and syndromes of diseases. This represents an avenue towards improving plant health, i.e., better management of multiple pathosystems, via improvements of management. Considering epidemiological guilds and guilds of harmful agents might be an interesting direction to take towards that aim.

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