European Journal of Plant Pathology (2005) 113:329–341
DOI 10.1007/s10658-005-4038-y

Mini-review

Epidemiological characteristics of angular leaf spot of bean: a systems analysis

Delphine Allorent[1] and Serge Savary[2,3,*]
[1]*IRD-CBGP, Campus International de Baillarguet, St-Gély-du Fesc cedex, CS 3001634988, Montferrier-sur-Lez, France;* [2]*IRD, Centre INRA, BiO3P, F-35653Le Rheu cedex, France;* [3]*UMR Santé Végétale, INRA, Centre de Bordeaux, 71 ave. Edouard Bourleaux, B.P. 81, 33883, Villenave d'Ornon cedex, France; *Author for correspondence (Phone: +33-5-57122617; Fax: +33-5-57122621; E-mail: ssavary@bordeaux.inra.fr)*

Accepted 7 October 2005

Key words: angular leaf spot of bean, defoliation, monocyclic process, *Phaeoisariopsis griseola*, polycyclic process, systems analysis

Abstract

In this review, available knowledge on angular leaf spot (ALS) of bean, caused by *Phaeoisariopsis griseola*, is analysed and synthesised. This is done through a systems-analytical approach, and successive flowcharts of the system, in order to identify knowledge gaps and guide further research. Six connected sub-models of the ALS monocycle are used as a framework: lesion establishment, lesion extension, defoliation, sporulation, spore liberation, and spore deposition. Each of the sub-models enables the linking of processes to various effects of environmental (physical and host) factors. Disease-induced defoliation is one feature of the pathosystem, leading to a depletion of infectious tissues from the canopy, which are transferred to the ground. Consequences of defoliation may include: strong reductions of the amount of inoculum and of vacant sites in the canopy, limited maximum disease severity, and progressive accumulation of inoculum below the canopy, which may become important a later stage of disease epidemics. These elements are hypothesised to explain the typical behaviour of ALS epidemics in the field, especially late onset, high apparent rate of disease increase, and low level of terminal disease severity in the standing canopy. Epidemiological consequences of lesion expansion, sporulation, and survival of spores deposited onto the canopy are other knowledge gaps in this pathosystem.

Introduction

Angular leaf spot (ALS) is caused by *Phaeoisariopsis griseola*, and is an important bean disease in the tropics and subtropics (Schwartz et al., 1981; Saettler, 1991). ALS causes typical symptoms on the leaves with angular shaped lesions as well as lesions on other aerial plant parts (stems, petioles, and pods) (Cardona-Alvarez and Walker, 1956). ALS epidemics are usually observed relatively late in the crop cycle (typically about the flowering stage) (Saettler, 1991; Allen et al., 1998). Lesion multiplication and extension on the foliage lead to defoliation, a prime mechanism leading to reduced physiological performance of the canopy (Bergamin Filho et al., 1997). ALS may be seen as a representative of many foliar, tropical diseases of legumes caused by hemi-biotrophic fungi.

The purpose of this review is to analyse and synthesise knowledge on ALS epidemiology. Although the overall quantitative behaviour of ALS epidemics is poorly understood, much is known on the underlying processes. Thus although the behaviour of ALS epidemics as a system is not well understood, the level of knowledge on components of the disease cycle is fairly well documented. One step towards a better epidemiological understanding is to collate current knowledge using a formal framework, and generate an overall view of the Bean – *P. griseola* system. This is done here

using a systems modelling approach, whereby processes are linked together, factors identified, and parameters accounting for the effects of factors on processes, outlined. The approach is that of Forrester (1961), applied to plant pathology (Zadoks, 1971; Teng, 1985). This allows (Rabbinge et al., 1989) (i) a scale-up of information from one level of integration (disease cycle) to the next (epidemic), (ii) identification of knowledge gaps, and (iii) guidance of further research.

ALS epidemics result from the functioning of a fairly complex system. This review is organised in six sub-models corresponding to different phases of the disease cycle. These sub-models are then connected to form an overall systems model that synthesises our quantitative knowledge on ALS epidemiology. This systems model is then used to discuss our understanding of ALS epidemics.

Monocyclic process: the infection cycle

Sub-processes of epidemics, sub-models, and couplers

The infection cycle, i.e., the building block of epidemics (Kranz, 1974; Zadoks and Schein, 1979) in angular leaf spot can be decomposed into five groups of processes (Figure 1):

1. Lesion establishment, corresponding to sub-model A;
2. Lesion extension, corresponding to sub-model B;
3. Defoliation of infected host leaves bearing lesions, corresponding to sub-model C;
4. Sporulation on infectious sites, corresponding to sub-model D;
5. Spore dispersal, corresponding to sub-models E (spore liberation) and F (spore deposition).

Mechanistic simulation models often consist of several sub-models, each representing different groups of processes. These can be linked by couplers (Zadoks and Rabbinge, 1985) which enable the dynamical connection of processes within the system. The different sub-models are shown in Figure 1, with their relationships materialised as couplers:

- coupler C_{AC} links lesion establishment to host defoliation, whereby removal from the canopy of infected sites (latent or infectious) brings also about the removal of healthy ones;
- C_{AB} links lesion establishment to lesion extension;
- C_{ACD} links infectious lesion establishment in the canopy (A) and on the ground (C) to sporulation in the system (D);
- C_{BD} links lesion extension to sporulation;
- C_{DE} links spore accumulation in the system (attached and detached leaves on the ground) to spore liberation;
- C_{EF} links flows of liberated spores from two sources (attached and detached infected leaves) to spore deposition onto the canopy, or spore loss; and,
- C_{FA} links spore deposition to the inflow of new efficient spores.

Table 1 lists the different state variables, rates, parameters and factors used in the six sub-models, and their dimensions.

Lesion establishment

Lesion establishment is considered here as the group of processes including spore germination until the end of the latency period. Effects of different factors on lesion establishment reported in the literature are summarised in Table 2.

Spore germination in *P. griseola* is strongly dependent on moisture. Spore germination on the leaf surface only takes place under moist conditions and occurs within three days after spore deposition (Monda et al., 2001). By contrast, the range of temperatures where spore germination is possible is very wide (5–33 °C), and so is the range of (near) optimal temperature (18–28 °C) (Cardona-Alvarez and Walker, 1956; Sindhan and Bose, 1980a). On the other hand, radiation does not appear to influence spore germination (Llanos, 1957; Santos-Filho et al., 1976).

Infection has often been recorded in the literature as a relative amount of successful lesion establishment, and so reported results incorporate spore germination. The range of temperature where infection occurs is wide (10–33 °C) (Cardona-Alvarez and Walker, 1956; Sindhan and Bose, 1980a; Bassanezi et al., 1997, 1998), but less so than for spore germination. However, disease development does occur at cool temperatures (16 °C), as reported by Inglis and Hagedorn (1986). Infection

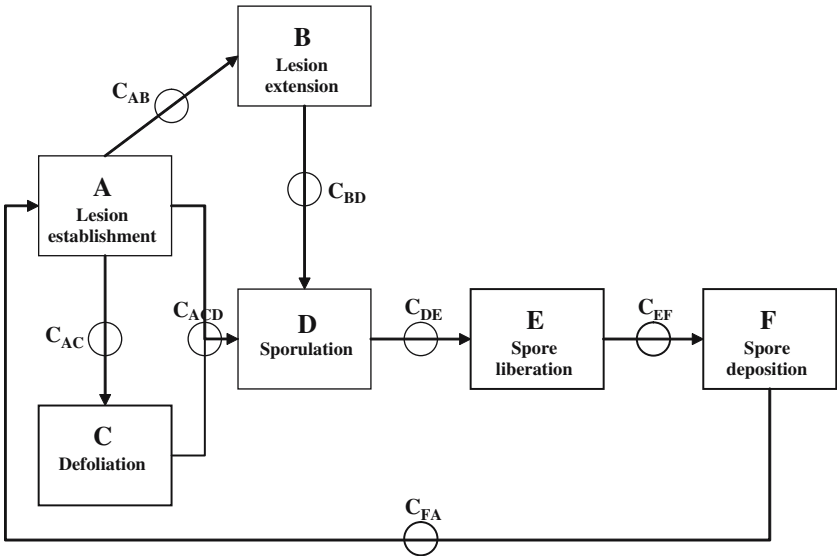

Figure 1. Monocyclic processes in angular leaf spot of bean synthesised as sub-models A, B, C, D, E and F, and the connecting couplers. C_{AB}: coupler linking lesion establishment to lesion extension, C_{AC}: coupler linking lesion establishment to host defoliation, C_{BD}: coupler linking lesion extension to sporulation, C_{ACD}: coupler linking lesion establishment in the canopy and on the ground to sporulation, C_{DE}: coupler linking spore accumulation in the system to spore liberation, C_{EF}: coupler linking liberated spore flows to spore deposition and loss, C_{FA}: coupler linking spore deposition to the inflow of new efficient spores.

efficiency (the ratio of established lesions to deposited spores) (Zadoks and Schein, 1979) was specifically studied by Willocquet et al. (2004), and was below 0.1 when inoculated plants were exposed to less than two consecutive nights of leaf wetness (i.e. two 16 h periods of continuous wetting at 70–100% RH with 8 h dry intervals at 50–80% RH). Optimum infection efficiency, about 0.5, was obtained after three to four consecutive wet nights, and infection efficiency was not increased by additional wet nights. These results concur with earlier reports concerning lesion establishment, where relative humidity in the air or leaf wetness duration (Cardona-Alvarez and Walker, 1956; Llanos, 1957; Campos-Avila and Fucikovsky Zak, 1980; Sindhan and Bose, 1980a) were varied. Recent results from Willocquet et al. (2004) also show that a single long continuous period of wetness is not necessary for infection to occur, because the authors exposed the inoculated plants to high humidity only during nights (16 h at 70–100% RH during nights and 8 h at 50–80% RH during days). Radiation does not appear to have a measurable effect on infection processes (Cardona-Alvarez and Walker, 1956). By contrast, host genotype by pathogen isolate effects are very strong, as illustrated by results reported by Correa-Victoria (1987), where two to 464 lesions per plant developed on a range of 17 Latin American isolates of *P. griseola* inoculated to 21 bean cultivars. Conversely, lesion density also varies according to the cultivar x isolate combination (from 0.47 to 5.24 lesions cm^{-2}) (Sartorato, 1989).

Latency period (LP, Table 1), the delay from spore deposition onto the host to production of a new generation of propagules (Zadoks and Schein, 1979; Campbell and Madden, 1990), is an important epidemiological parameter, as it determines the number of infection cycles a pathogen can cause during the life-cycle of its host. In the case of ALS, as in many hemi-necrotrophic foliar pathogens (Rapilly, 1983), the incubation period often coincides with LP as sporulation may be initiated as soon as the first lesions appear under high moisture conditions. Information pertaining to both the incubation period and the latency period is summarised here.

Incubation period in ALS is delayed at low temperatures (Cardona-Alvarez and Walker, 1956; Buchurara, 1983; Bassanezi et al., 1997, 1998), and varies from 12 to 15 days, 9 days, and 10 days at 16 °C, 24 °C, and 28 °C (Cardona-Alvarez and Walker, 1956), respectively. Large variation with the considered cultivar × isolate occur (Correa-Victoria, 1987; Buruchara et al., 1988; Sartorato,

Table 1. List of state variables, rates, parameters and factors used in sub-models A, B, C, D, E and F

Symbol	Meaning of symbol	Dimension[a]
State variables		
DSPO	Number of spores deposited on canopy leaves	$[N_{spores}]$
HSITE	Number of healthy sites	$[N_{sites}]$
INFC	Number of infectious sites on canopy leaves	$[N_{sites}]$
INFD	Number of infectious sites on defoliated leaves	$[N_{sites}]$
LAT	Number of latent sites	$[N_{sites}]$
LSPOC	Number of spores liberated from canopy leaves	$[N_{spores}]$
LSPOD	Number of spores liberated from defoliated leaves	$[N_{spores}]$
SPOC	Number of spores produced on canopy leaves	$[N_{spores}]$
SPOD	Number of spores produced on defoliated leaves	$[N_{spores}]$
Rates		
rAPP	Rate of appearance of infectious sites on canopy leaves	$[N_{sites}\ T^{-1}]$
rDEFh	Rate of defoliation of healthy sites	$[N_{sites}\ T^{-1}]$
rDEFi	Rate of defoliation of infectious sites	$[N_{sites}\ T^{-1}]$
rDEPC	Rate of deposition of spores from canopy leaves	$[N_{spores}\ T^{-1}]$
rDEPD	Rate of deposition of spores from defoliated leaves	$[N_{spores}\ T^{-1}]$
rINF	Rate of infection of healthy sites	$[N_{sites}\ T^{-1}]$
rEXT	Rate of extension of infectious sites on canopy leaves	$[N_{sites}\ T^{-1}]$
rLIBC	Rate of liberation of spores from canopy leaves	$[N_{spores}\ T^{-1}]$
rLIBD	Rate of liberation of spores from defoliated leaves	$[N_{spores}\ T^{-1}]$
rLOSC	Rate of loss of spores from canopy leaves	$[N_{spores}\ T^{-1}]$
rLOSD	Rate of loss of spores from defoliated leaves	$[N_{spores}\ T^{-1}]$
rREM	Rate of removal of infectious sites on defoliated leaves	$[N_{sites}\ T^{-1}]$
rSPOC	Rate of production of spores on canopy leaves	$[N_{spores}\ T^{-1}]$
rSPOD	Rate of production of spores on defoliated leaves	$[N_{spores}\ T^{-1}]$
Parameters		
LP	Latent period	[T]
PINC	Primary inoculum	$[N_{spores}]$
Calculated (dynamic) variable		
SEV	Severity	[–]
Factors		
C × I	Interaction between bean cultivar and P. griseola isolate	[–]
LW	Leaf wetness duration	[T]
RAD	Radiation	$[J\ L^{-2}\ T^{-1}]$
RAIN	Rainfall amount	[L]
T	Temperature	[K]
WIND	Wind speed	$[L\ T^{-1}]$

[a]Dimensions: [N]: numbers; [T]: time ; [J]: energy; [L]: length; [K]: temperature.

1989). Both periods have a range of 10 to 23 days in terms of incubation (Correa-Victoria, 1987) or of latency (Sartorato, 1989) period.

Figure 2 synthesises the lesion establishment process and highlights some of its factors. Primary inoculum (PINC, efficient spores) initiates the process whereby healthy sites (HSITE) are infected and latent sites (LAT) are produced through infection (rINF). A key factor for infection to occur is leaf wetness (LW), which does not appear to play a role in further lesion development (Cardona-Alvarez and Walker, 1956). Latent sites may become infectious (INFC) after a latency period, which may coincide with the rate of lesion appearance (rAPP). Temperature (T) and the cultivar × isolate (C × I) combination play an important role in INFC variation.

Lesion extension

The effects of different factors on lesion extension recorded in the literature are summarised in Table 2. Verma and Sharma (1984) reported that lesion size followed an optimum-type relationship with temperature, with maximum values observed at 15 °C, which concurs with field observations where larger lesions were measured during cooler (18–22 °C) compared to warmer (28–32 °C)

Table 2. Synthesis of literature on factors influencing epidemiological processes in angular leaf spot

Processes	Temperature (*T*) Range (optimum)	Moisture Relative humidity (RH) or leaf wetness (LW)	Radiation (RAD)	Interaction Cultivar × Isolate (C×I)	Disease severity (SEV)	Rain	Wind
Spore germination	8–32 °C (20–28) [1], 5–33 °C (18–24) [2]	None below 49%RH [2] Required 48 h at 61% and 6 h at 100%[2]	No effect [3] Little increase with light [4]				
Infection efficiency	16–28 °C (24) [1], 10–33 °C (24) [2]	Minimum of 3 h [1] or 24 h [2] of high RH required; none below 86% RH[2] Increases with periods of high RH [1,2,3,5, 6]	Increased with 12 h-photoperiod [1]	Varies depending on C×I [7,8, 12]			
Latency period (Incubation period)	16–28 °C (24) [1] 12–30 °C (24) [9,10], Delayed at low temperatures [1,9,10, 11]	Delayed with exposition period to high RH below 16 h [5]	No effect [1]	Varies depending on C×I [7,8]			
Lesion extension	12–30 °C (24) [9,10], 16–28 °C (24) [13], 10–30°C (15) [14]			Varies depending on C×I [7,15, 16]			
Host defoliation	16–28 °C (24) [1], Delayed at 16 °C 16–28 °C (24) [13], Lower at 16 °C				Increases with severity. Relative rate of defoliation of 0.23 day^{-1} for a severity of 18% [6]		
Sporulation	12–28 °C (16) [5], 16–32 °C (24) [4], 10–30 °C (21–24) [2]	None below 71% RH [2], 24 and 48 h of high RH to complete coremial production and abundant sporulation, repectively [1]	Increased with continuous darkness and 12 h-photoperiod [4]	Varies depending on C×I [7]			
Spore dispersal						Splash dispersal spreads spores from the soil [1]	Wind-blown spores : infections at up to 7m [1] Spores in soil particles: within 2m [1]

[1] Cardona-Alvarez and Walker, 1956; [2] Sindhan and Bose, 1980a; [3] Llanos, 1957; [4] Santos-Filho et al., 1976; [5] Campos-Avila and Fucikovsky Zak, 1980; [6] Willocquet et al., 2004; [7] Correa-Victoria, 1987; [8] Sartorato, 1989; [9] Bassanezi et al., 1997; [10] Bassanezi et al., 1998; [11] Buchurara, 1983; [12] Buchurara et al., 1988; [13] Inglis and Hagedorn, 1984; [14] Verma and Sharma, 1984; [15] Diaz et al., 1965; [16] Hocking, 1967.

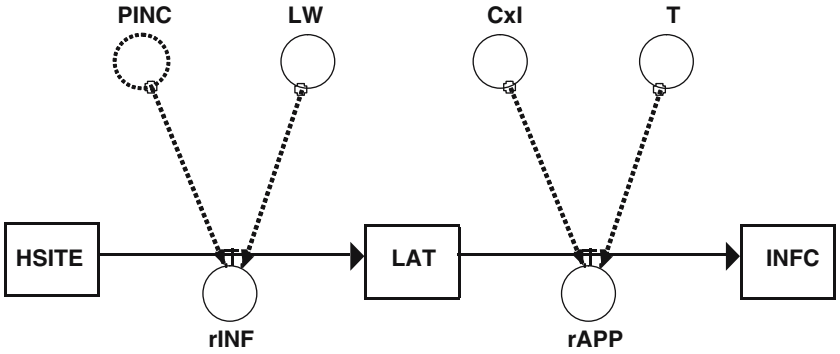

Figure 2. Sub-model A, lesion establishment in a bean canopy. Squares represent state variables, valves represent rates, continuous circles represent factors (fixed or variable), dotted circles represent calculated variables (e.g., severity) or parameters (set or computed), continuous arrows represent flows of sites, discontinuous arrows represent flows of quantitative information from parameters or factors. See Table 1 for meaning of abbreviations and dimensions.

periods. The largest lesion sizes (about 14 mm^2) and maximal rate of lesion extension (0.34 mm^2 day^{-1}) were observed at 24 °C (Inglis and Hagedorn, 1984; Bassanezi et al., 1997; Bassanezi et al., 1998). Such a rate of lesion extension is in the same range as for *Septoria nodorum* on wheat (Berger et al., 1997). It however is smaller than *Exserohilum turcicum* on sweet corn (25–43 mm^2 day^{-1}), *Phytophtora infestans* on potato (26–45 mm^2 day^{-1}), or *Fusarium moniliforme* on fig (225 mm^2 day^{-1}) (Berger et al., 1997).

For a given isolate, lesion size (range: 3.8–20.9 mm^2) strongly depends on the cultivar (Diaz et al., 1965; Correa-Victoria, 1987), and an atypical isolate produces circular lesions of 10 mm diam within 6 days after inoculation, with maximum diameters up to 20 mm (Hocking, 1967). A cultivar × isolate interaction on lesion extension has been reported (Diaz et al., 1965; Hocking, 1967; Correa-Victoria, 1987), the intensity of which varies with temperature.

A low negative but significant correlation ($r = -0.49$; $P = 0.05$) was found between average number of lesions and average lesion size by Correa-Victoria (1987), although Diaz et al. (1965) did not detect any significant correlation between these variables, possibly because of a defoliation effect.

Figure 3 summarises information from the literature on the lesion extension process in ALS in the simplest possible way: extension of infectious sites leads to a reduction of the number of healthy sites (HSITE), which may become immediately infectious (INFC). Two factors act on the rate of lesion extension, temperature (T) and the cultivar × isolate interaction (C × I).

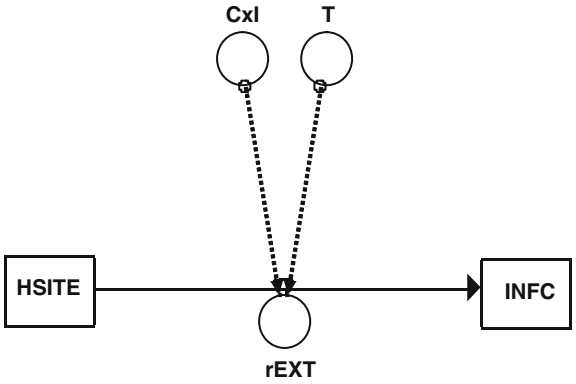

Figure 3. Sub-model B, lesion extension on canopy leaves. See Table 1 for meaning of abbreviations and dimensions and legend of Figure 2 for meaning of symbols.

Disease-induced defoliation

The effects of different factors on disease-induced defoliation reported in the literature are shown in Table 2. Disease-induced defoliation, which may reach 100% under glasshouse conditions begins first and is most rapid at 24 °C (Cardona-Alvarez and Walker, 1956; Inglis and Hagedorn, 1986), but is delayed at low temperatures (Cardona-Alvarez and Walker, 1956).

The correlation between defoliation and disease severity (proportion of diseased leaf area) is strong, with relatively low ALS severity already causing a substantial defoliation. For instance, a relative rate of defoliation of 0.23 day^{-1} was

estimated for a severity of 18% (Willocquet et al., 2004). Similar positive correlations were found in many leaf spot diseases of annual legumes, such as *Cercosporidium personatum (Phaeoisariopsis personata)* on groundnut (e.g., Watson et al., 1986).

Figure 4 provides a synthesis of the defoliation of infectious sites of a bean canopy. Infectious sites on infected leaves of the canopy (INFC) are defoliated (INFD), bringing about the defoliation of healthy sites (HSITE) belonging to the same infected leaves. The underlying hypothesis of Figure 4 is that defoliation of healthy sites is driven by that of infectious ones, and therefore that physiological defoliation in an infected canopy is negligible compared to disease-induced defoliation. Defoliated sites remain infectious (INFD) for an infectious period, and are then considered removed (rREM) from the system. The only factor acting on the rate of defoliation of infectious sites (rDEFi), and therefore on healthy ones (rDEFh) is the disease severity (SEV). As in any foliar disease, SEV, the proportion of infected sites, should be seen as the result of the dynamics of disease in the canopy, i.e., the balance between infection, defoliation, and host growth rates.

Sporulation

The effects of different factors on sporulation in ALS from the literature are identified in Table 2. The range of temperature where sporulation occurs is very wide (10–30 °C) (Santos-Filho et al., 1976; Campos-Avila and Fucikovsky Zak, 1980; Sindhan and Bose, 1980a). The relative humidity of the air is a very strong limiting factor of sporulation, which does not occur below 71% (Sindhan and Bose, 1980a). An early report by Cardona-Alvarez and Walker (1956) indicated that a 24 h period was required to complete the formation of coremia, and that an additional humid 48 h period or more was necessary for spore production. From results in controlled chamber work, it does not appear that sporulation is influenced by radiation (Santos Filho et al., 1976). A temperature–isolate interaction exists, with some isolates having an optimal sporulation temperature at 19 °C, whereas the optimum temperature for other isolates is 24 °C (Buruchara, 1983). Cultivar–isolate interactions occur too, with large effects on sporulation density (5–800 spores mm^{-2} of lesion area) (Correa-Victoria, 1987). Sporulation density, on the other hand, does not seem to vary with disease severity, number of lesions, lesion size and incubation period (Correa-Victoria, 1987).

Figure 5 distinguishes two sources and two rates of spore production in the sporulation process occurring in an ALS-infected bean stand. Infectious sites present in the canopy (INFC) and on defoliated leaves (INFD) both produce spores,

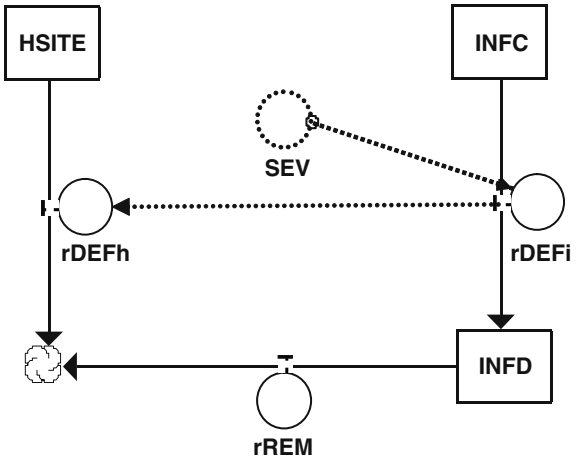

Figure 4. Sub-model C, defoliation of infected leaves bearing healthy and infectious sites. See Table 1 for meaning of abbreviations and dimensions and legend of Figure 2 for meaning of symbols.

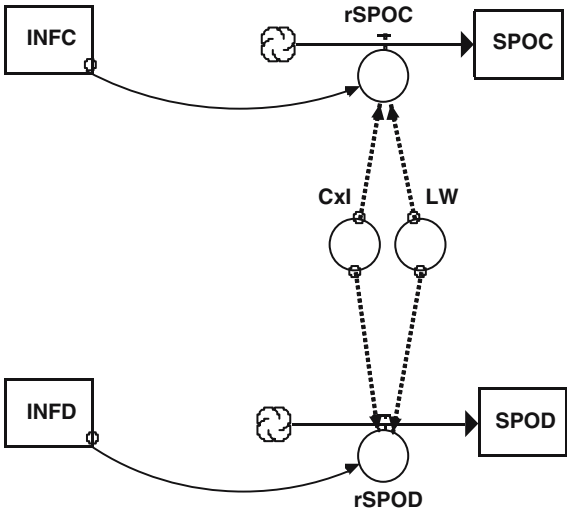

Figure 5. Sub-model D, sporulation of infectious sites on canopy and on defoliated leaves. See Table 1 for meaning of abbreviations and dimensions and legend of Figure 2 for meaning of symbols.

which lead to two stocks of spores, in the canopy (LSPOC) and on defoliated leaves (SPOD). Figure 5 indicates two variables acting on both the rates of sporulation, leaf wetness (LW) and the cultivar × isolate interaction (C × I).

Spore dispersal

Very little quantitative information is available from the literature on spore liberation and dispersal in *P. griseola*. The effects of different factors on spore dispersal are summarised in Table 2. Both rain and wind appear to liberate and disperse spores of *P. griseola*, and wind-blown particles from infested soil, wind-blown spores and rain droplet-borne spores are all effective agents of dissemination according to Cardona-Alvarez and Walker (1956).

Figure 6 therefore summarises the spore liberation from lesions in a diseased canopy (SPOC) and on defoliated leaves (SPOD) by rain and wind. Figure 7, in turn, represents the rates of deposition (rDEPC and rDEPD) from both stocks of spores which (daily) accumulate in a (combined) stock of spores deposited onto the canopy (DSPO), and the process of spore loss through two loss rates (rLOSC and rLOSD). Spore loss is assumed to occur through escape from the system in the atmosphere and deposition on the ground. All four rates are made dependent on rainfall and wind.

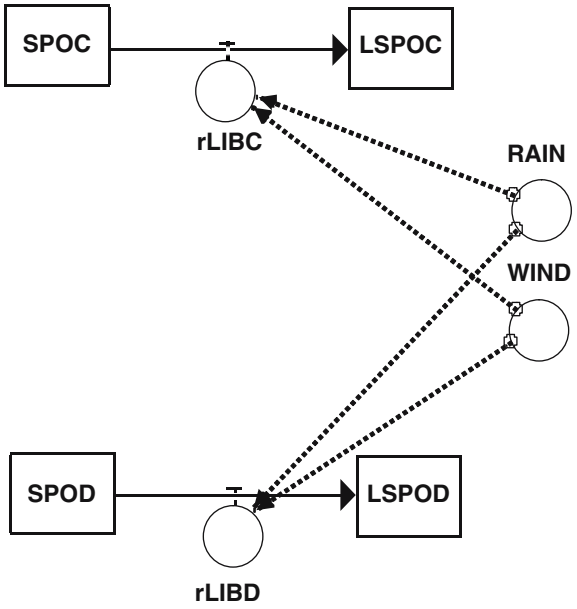

Figure 6. Sub-model E, spore liberation from infectious sites on canopy and defoliated leaves. See Table 1 for meaning of abbreviations and dimensions and legend of Figure 2 for meaning of symbols.

Overview and discussion

ALS epidemics: phenomenology

The dynamics of ALS epidemics have been described and analysed by several authors (e.g., Bergamin Filho et al., 1997) and may be characterised with the following attributes: (i) late onset, (ii) rapid increase towards the end of the cropping season, (iii) low maximum severity. Examples are provided in Figure 8, with disease progress curves derived from experiments conducted at EMBRAPA Center Arroz e Feijão (Goiania, Brazil) in 2004, where disease severity (Godoy et al., 1997) was monitored weekly in untreated bean plots (5.5×5.5 m, with 0.25 m spacing of cv. Roshina G2). Disease severity progress was slow at the beginning, but increased rapidly in the last third of the cropping seasons. The apparent rate of disease increase (Van der Plank, 1963) varied from 0.154 to 0.210 day^{-1}, these values being slightly higher than those estimated by Buchurara et al. (1988) in a susceptible cultivar ($r = 0.142$). Terminal severities were low, from 5 to 9% of diseased area, and are lower than terminal severities generally reported in the literature: from 5 (Jesus Junior et al., 2001), to 9% (Silva et al., 1998), 23% (Mora et al., 1985), 25% (Jesus Junior et al., 2003), 27% (Carneiro et al., 1997), 28% (Buchurara et al., 1988), and 29% (Bergamin-Filho et al., 1997).

Structure of a systems model

The combination of the successive sub-components (processes) described in Figures 2–7 results in Figure 9, which gives an overview of the basic monocycle components (Teng and Close, 1978; Zadoks and Schein, 1979) of ALS epidemics. Factors influencing rates have been removed from the diagram in order to retain the main features of the processes themselves. Note no change in healthy sites (HSITE) over time, i.e. no host growth is included in the structure of Figure 9. Figure 9 is an expansion of Figure 1, where the

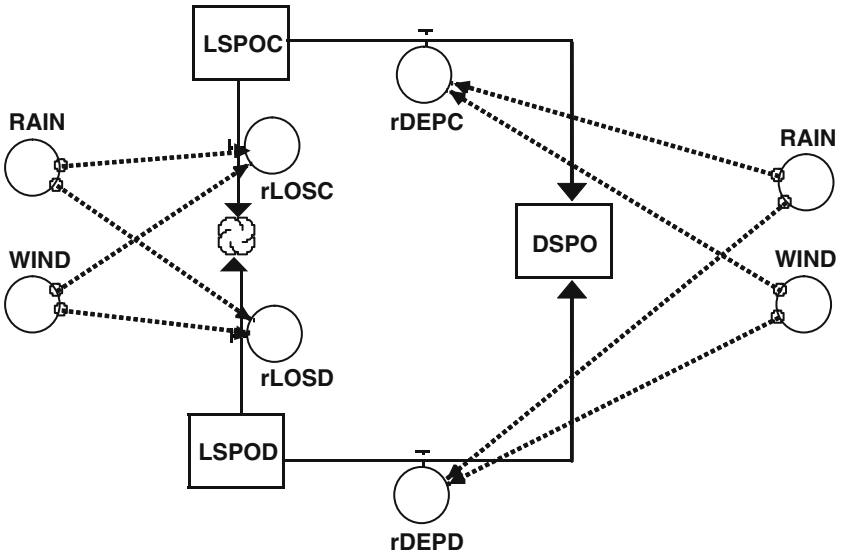

Figure 7. Sub-model F, loss and deposition of the liberated spores from canopy and defoliated leaves. See Table 1 for meaning of abbreviations and dimensions and legend of Figure 2 for meaning of symbols.

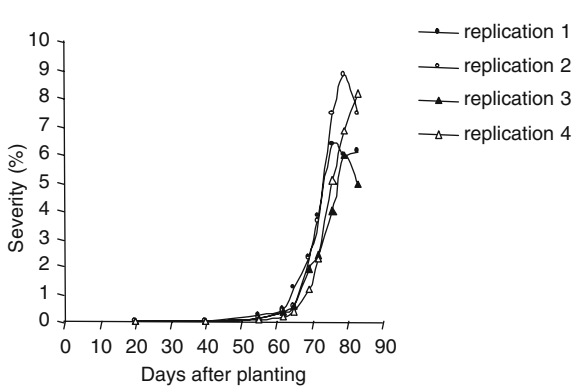

Figure 8. ALS severity progress curves in four untreated bean plots (EMBRAPA, Brazil, 2004).

main elements of Figures 2–7 have been retained, and where the couplers between sub-models have been replaced by rates, flows of sites, or flows of quantitative information, as appropriate. Many of the processes indicated in Figure 9 are classical components of plant disease epidemics affecting aerial plant parts (Van der Plank, 1963; Zadoks and Schein, 1979; Campbell and Madden, 1990), e.g. infection, latency, sporulation, spore dispersal, and deposition. This systems model may be qualitatively linked to the behaviour of ALS epidemics, in particular, their delayed onset, their rapid increase, and their low terminal severity.

Disease-induced defoliation and its epidemiological consequences

Disease-induced defoliation is one important feature of the ALS pathosystem, which can affect disease severity in several ways. First, defoliation leads to a loss of diseased leaves (including diseased and healthy sites, rDEFi, rDEFh, Figure 4) which cannot be assessed in disease measurements. Second, loss of diseased leaves may alter the crop microclimate, rendering it less favourable for disease intensification. Third, defoliation reduces growth, and thus the production of healthy sites available to infection. Consequences of defoliation may therefore include: (1) a reduction of inoculum present within the canopy and its transfer to the ground, (2) changes in the crop microclimate, (3) a reduction of LAI and so of vacant sites which otherwise could intercept dispersed spores, and (4) limited maximum disease severity. In addition to these four consequences, disease induced defoliation delays the increase in disease severity. This is discussed in the following section.

In many countries where bean is an important crop, selection for resistance is the main, often the only, practical direction for disease management. So far, selecting for complete resistance to this disease has proven extremely difficult (Correa and Saettler, 1987; Allen et al., 1998). Also, selection

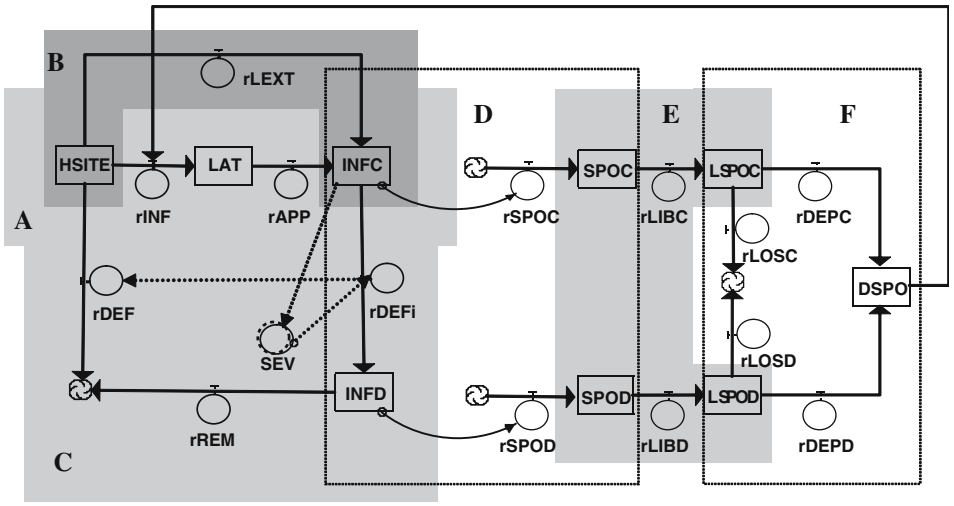

Figure 9. Synthesis flowchart representing the *P. griseola* cycle with sub-models A, B, C, D, E and F. A, B, C, D, E, and F: refer to sub-models shown in Figure 1. See Table 1 for meaning of abbreviations and dimensions and legend of Figure 2 for meaning of symbols. Not all relationships are shown (see text for details).

for incomplete resistance is confronted with an extremely variable pathogen (Sartorato, 1989; Sartorato and Rava, 1992). Selection could take advantage of new knowledge gained on the functioning of the pathosystem, in particular, the dynamic relationship between severity, defoliation, and inoculum remobilisation. For instance, bean cultivars that shed their leaves rapidly after being infected would not allow the pathogen to build inoculum sources, either in the canopy or at its base, thus delaying epidemics. Excessive defoliation would of course reduce yields so strongly that a balance between healthy area duration (Bergamin Filho et al., 1997), yield accumulation, and disease progress is to be sought. Simulation models using structures derived from Figure 9 could be useful to achieve that aim.

Delayed epidemics

An hypothesis to explain delayed epidemics is the progressive accumulation of infectious tissues on the ground through defoliation (Figure 4, rDEFi) in a first phase, followed by the progressive mobilisation of this inoculum in a second phase (sporulation on lesions on shed leaves: Figure 5, rSPOD; spore liberation from this source: Figure 6, rLIBD; deposition of spores of this source on the canopy: Figure 7, rDEPD). Another hypothesis is that the microclimate (especially leaf wetness) may be unfavourable for lesion establishment (Figure 2, LW and rINF) until canopy closure. A third hypothesis to explain delayed epidemics is related to the availability and amount of primary inoculum (Figure 2, PINC). Five sources of primary inoculum are considered in the literature: (i) infected seeds (on which the pathogen might survive up to nine months) (Orozco-Sarria and Cardona-Alvarez, 1959; Diaz et al., 1965; Sohi and Sharma, 1974; Sindhan and Bose, 1979; Dhingra and Kushalappa, 1980; Sengooba and Mukiibi, 1986), (ii) infected plant debris over successive seasons (Cardona-Alvarez and Walker, 1956; Barros et al., 1958; Sindhan and Bose, 1979; Sengooba and Mukiibi, 1986; Correa and Saettler, 1987; Rodrigues et al., 1999), (iii) infected soil (Sindhan and Bose, 1979), (iv) infected volunteer plants, and (v) off-season crops (Sengooba and Mukiibi, 1986). Depending on cropping practices, crop rotation, and crop regimens, the relative importance of these sources may vary greatly. Information on change in host susceptibility over development is very scarce (Cardona-Alvarez and Walker, 1956; Sindhan and Bose, 1980b) and controversial.

Life strategy of the pathogen: lesion extension, sporulation, and infection efficiency

Other features of ALS epidemics are indicated in Figure 9, including lesion extension and sporulation. Lesion extension enables progress to vacant sites, where sporulation may rapidly be

initiated. This process is one way for the pathogen to intensify, and is less strongly influenced by environmental factors, compared to infection via propagule liberation, transport, deposition, and germination (Berger et al., 1997). Data on spore production in *P. griseola* is scarce, but suggests that it is actually limited (propagules infectious area^{-1}), and strongly depends on environmental factors (relative humidity and leaf wetness). For epidemics to occur, a comparatively low spore production would have to be compensated by a very high infection efficiency, combined with a strong survival ability of deposited or germinating spores. Such characteristics concur with observations by Monda et al. (2001), and have been quantitatively described in other pathosystems (e.g. Kaizer and Lukezic, 1966; Eversmeyer and Burleigh, 1968; Van Hees-Boukema and Zadoks, 1986; Becker and Burr, 1994), but have not been quantitatively documented in the case of ALS.

Knowledge gaps and further research

Most of the parameters and rates in the structure described in Figure 9 remain to be quantified. Among them are the rates of spore liberation and deposition, and their relationships with environmental (weather) variables. Similar questions arise when considering infection efficiency, spore production and its duration on infectious tissues, and factors that may influence them. Some of the rates and parameters of Figure 9 are also under the influence of aggressiveness of the isolate on a given host genotype (host-isolate interaction), and of plant age. A basic question remains to assess the epidemiological significance of the presence of two distinct sources of inoculum in the system. Another generic question pertains to the life strategy of the pathogen (Zadoks and Schein, 1979), which possibly might compensate a comparatively low spore production by a strong survival ability of deposited spores and of lesions themselves (lesion extension) (Berger et al., 1997) and by a high infection efficiency. These questions need further quantitative documentation and testing.

Acknowledgements

This research was partly funded by the European Project INCO n°ICA4-CT-2000–30004. We thank L. Willocquet (INRA-IRD) for her assistance in reviewing the manuscript, and A. Sartorato (EMBRAPA Arroz e Feijão) for his support in field work.

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