REVIEW ARTICLE

Interactions between foliar diseases: Concepts and epidemiological approaches

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ABSTRACT

This review deals with the phenomenon of plant disease interactions. The epidemiological implications of foliar diseases occurring simultaneously on the same crop are important because the establishment of disease management strategies depends on the knowledge of disease interactions. We discuss some concepts and the terminology related to the interaction studies and present related examples with special emphasis on interacting wheat diseases.
Key words: crop loss, disease dynamics, multiple diseases.

INTRODUCTION

The occurrence of two or more pathogens simultaneously on the same host is frequent in many production systems (Zadoks & Schein, 1979; Kranz & Jörg, 1989). This situation may be even more recurrent in many tropical areas, where environmental conditions are mostly favourable to the occurrence of diseases during all periods of the year. Little is known about the combined effects of diseases on crop yield and only a few epidemiological studies on this subject have been carried out. Estimates of disease effects on yield are usually made assuming that each disease acts independently. Interactions of diseases can increase crop damage and complicate the identification of primary causes of diseases and their control. The interactions may alter the occurrence and speed of epidemics. Weber et al. (1994), for instance, concluded for the two wheat pathogens *Septoria nodorum* and *Erysiphe graminis* that wherever the two pathogens occur together, neither their dynamics, nor the influence of external factors could be understood, if interspecific interactions are neglected. Interactions may have significant implications for assessing crop losses, diagnosing the causes of these losses and for selecting appropriate management strategies, as well as for forecasting, modelling and simulations of epidemics (Waller & Bridge, 1984; Bassanezi et al., 1998). Estimates of yield losses caused by several diseases made by adding of single disease yield-loss models are likely to be inaccurate if interactions are occurring (Zhou et al., 2000). Interactions may be important because the expected benefit from the control of one pathogen depends on the level of the other pathogens (Johnson et al., 1986) and

therefore multiple diseases can significantly alter economic decision criteria in comparison to single disease occurrence (Pinnschmidt, 1991). According to Newton et al., (2010), new approaches in agronomy, crop protection and breeding could be achieved by understanding the population dynamic balance between the organisms of the phyllosphere as an ecological system.

The objectives of studies in multiple-disease situations usually can be divided into understanding (i) the population dynamics of interacting diseases and (ii) the combined effect on crop yield or yield loss. Once the specific effects of combined pathogens are understood, the question shifts towards how to manage a crop grown under combined infection conditions (Johnson, 1990). The examples of interactions presented in literature are mainly dealing with their effects on yield. Epidemiological aspects of these interactions in terms of changes in the component analysis have rarely been studied (Zadoks & Schein, 1979; Weber et al., 1994). Here some aspects related to the dynamics and to crop losses of the interactions between diseases caused by aerial pathogens are discussed. The discussion will be restricted to the relationships between two or more aerial pathogens on the same host. Cross-protection and biological control will be not emphasized, although in these cases some kind of interactions can occur between the organisms involved.

TERMINOLOGY

Terms like interaction, association, interference and interrelationship have been used to describe relationships among diseases. In many cases these terms are used in an

improper manner (Wallace, 1983; Sikora & Carter, 1987). According to Wallace (1983), the various etiological agents can influence each other in their effects on the plant; that is, they interact. Consequently, the effect of the contribution of different pathogens on the same host may not be purely additive. As diseases can interact in their dynamics and/or in their effects on crop loss, it is necessary to classify the interactions for both aspects. Sometimes there is no significant interaction between two diseases with respect to yield, although one disease affects the development of the other (Simkin & Wheeler, 1974). For a given combination of pathogens, the type of interaction may change under different conditions or during successive stages in their life histories. Mixtures of synergistic and antagonistic interactions, creating usually unpredictable biological and epidemiological consequences, are likely to occur in plants, as Syller (2012) observed for interacting viruses.

Interactions concerning the disease dynamics

Several terms have been proposed to qualitatively describe interactions while quantitative methods seem to be used scarcely. The interactions between two and more diseases on a common host may produce antagonistic or protective, mutually exclusive, additive, or synergistic effects in the host (Damsteegt et al., 1993). An interaction between different pathogens can be antagonistic or protective when one inhibits or reduces the development of another (Latch & Potter, 1977). A mutually exclusive interaction occurs when the development of all involved pathogens is reduced (Jedlinski & Brown, 1965). An interaction is additive when the development of one pathogen is not altered in the presence of another and vice versa (Gordon & Schmitthenner, 1969). When there is some enhancement in the development of one or more interacting pathogens, the interaction is called synergistic (Beute, 1973).

In ecology, one population can influence a second one in different ways, whereby the effect can be positive (+), negative (-) or neutral (0). On the other hand, the second population can also affect the first one. Odum (1953) defined in an ecological sense that populations of two species may interact theoretically in six basic ways, corresponding to the six combinations of 0, +, and -, as follows: 00 (neutralism), -- (competition), ++ (mutualism or protocooperation), +0 (commensalism), -0 (amensalism), and +- (parasitism or predation). Although many types of direct effects of an organism on another can occur, indirect effects through the host plants seem to explain most of the cases of interacting diseases (Waller & Bridge, 1984). Sometimes it is even not clear whether there are any antagonistic or synergistic effects of interacting pathogens, like with *Alternaria porri* and *Stemphylium vesicarium* which often occur together in the same purple leaf blotch lesion on *Allium* species (Suheri & Price, 2000). In some cases the interactions between two diseases may not be detectable due to clear differences in time of disease onset or low disease levels, like for anthracnose (*Colletotrichum sublineolum*) and leaf

blight (*Exserohilum turcicum*) on sorghum in Kenya (Ngugi et al., 2000).

In situations involving the simultaneous occurrence of aerial pathogens on the same plant, terms "interactions between diseases" and "interactions between pathogens" are practically similar. However, approaches involving this subject should focus on the interactions between diseases, since, in many cases, there are indirect effects mediated by the host.

Interactions concerning crop losses

Similar to the definition of interactions with respect to disease dynamics, the interactions related to yield or yield loss are termed in different ways although the terms seem to be clearer. There are three outcomes of combined effects of diseases on crop loss (Waller & Bridge, 1984): the combined loss is equal to (no interaction, additive), more than (greater-than-additive, synergistic, positive interaction), or less than (less-than-additive, antagonistic, negative interaction) the sum of yield losses from individual diseases alone. According to the literature survey of Johnson (1990), studies of the effects of multiple pest and diseases on crop yield mostly report antagonistic interactions, which may result from competition between pathogen populations or from stimulation of active defence mechanisms in the host. Reports of synergistic interactions are relatively rare (Johnson, 1990). The synergistic interactions seem to operate through effects on host resistance permitting a pathological succession rather than by direct mutual synergism of pathogens (Waller & Bridge, 1984).

EXAMPLES OF DISEASE INTERACTIONS

Some data concerning interactions between aerial diseases are presented in the Tables 1, 2 and 3. In some pathosystems, the determination of the type of interaction concerning dynamics aspects and/or crop loss is demonstrated. Criteria utilized to classify the interactions in terms of dynamics and crop loss were made considering three categories – antagonistic, synergistic and additive interactions.

It is important to emphasized that two plant pathogens can interact as antagonists "in vitro", however the diseases as a result of their infections on the same host can present an additive effect. Thus, it is not appropriate to do generalizations.

CASE STUDY: RESULTS OBTAINED FROM PUBLISHED STUDIES ON WHEAT DISEASES

Most of the cases of interaction between foliar diseases reported in the literature is related to wheat. This crop can be infected by many pathogens singly or simultaneously. Although wheat is a crop of temperate regions, it has been cultivated in many tropical areas, which may contribute to increase the frequency of interaction cases. In Brazil, for

TABLE 1 - Selected examples of interactions related mainly to disease dynamics.

Host	Involved pathogens	Authors	Type	Comments of the authors
Alfalfa	*Colletotrichum destructivum, C. dematium* f. sp. *truncate, C. trifolii*	Graham et al., 1976	Antagonism	Seedlings simultaneously inoculated with *C. trifolii* and *C. destructivum* or *C. trifolii* and *C. dematium* f. sp. *truncata* were significantly less damaged than those inoculated with *C. trifolii* alone.
Arabidopsis thaliana	Cucumber mosaic virus (CMV), Turnip crinkle virus (TCV)	Yang et al., 2010	Antagonism	Although TCV coat protein accumulates to a similar level in singly or doubly infected plants, CMV coat protein is significantly decreased in doubly infected plants, which develop symptoms similar to those of TCV infection alone.
Barley	Brome mosaic virus (BMV), Tobacco mosaic virus (TMV)	Hamilton & Nichols, 1977	Synergism	BMV functioned as a helper virus in TMV infection of barley.
Barley	*E. graminis* f. sp. *hordei*, Barley yellow dwarf virus (BYDV)	Potter & Jones 1981	Not clear	When infected with virus, cultivar Vada was significantly less susceptible to mildew at the 12-day scoring, in contrast to the 26-day score, when it was significantly more susceptible.
Barley	*Erysiphe graminis* f. sp. *hordei*, *Puccinia hordei*	Kiessling & Hoffmann, 1985a,b	Antagonism	Simultaneous inoculation with both pathogens diminished reactions, maybe due to a strong competition for assimilates among pathogens. In plants infected with powdery mildew, the development of uredia was inhibited, the beginning of fructification of rust delayed, the sporulation time shortened and the formation of uredsospores per uredium, day and per cm^2 reduced. *P. hordei* negatively affected the size of mildew pustules and the sporulation time of powdery mildew.
Barley	*Puccinia hordei, Septoria avenae* f. sp. *triticea*	Shearer et al., 1978	Synergism	Leaves infected with rust were more severely infected with *S. avenae* f. sp. *triticea*. Necrosis caused by rust and *Septoria* together was greater than the total necrosis caused by rust or *Septoria* alone.
Barley	*Pyrenophora teres, Rhynchosporium secalis,*	Cherif et al., 2007	Antagonism	Under low epidemic conditions, net blotch and scald developments were usually independent, but positively associated for tolerant lines for both diseases. Under high epidemic conditions, competition effects were obtained for susceptible and resistant genotypes.
Barley	*Pyrenophora teres, Rhynchosporium secalis*, Barley yellow dwarf virus (BYDV)	Varughese & Griffiths, 1983	Antagonism	Number of lesions produced by *P. teres* and *R. secalis* were reduced in the presence of the virus.
Celery	*Septoria apiicola*, Celery mosaic virus, Parsnip yellow fleck virus	Ataga et al., 1999	Antagonism	Inoculation with viruses decreased blight on leaves inoculated later with *S. apiicola*.
Citrus	*Geotrichum candidum, Penicillium digitatum*	Morris, 1982	Synergism	Synergism was greatest in mature fruit and occurred over a wide range of spore concentrations of *G. candidum* and *P. digitatum*.
Common bean	*Uromyces appendiculatus, Colletotrichum lindemuthianum*	Yarwood, 1977	Synergism	Rust predisposes bean plants to foliar infection of *C. lindemuthianum* in anthracnose-resistant bean varieties.
Common bean	*Uromyces appendiculatus, Colletotrichum lindemuthianum*	Kiel & Hau, 2000	Antagonism	Final severity of anthracnose was reduced after a pre-inoculation with rust, although the time of the pre-inoculation did not influence the progress curve of anthracnose.
Common bean	*Uromyces appendiculatus, Pseudocercospora griseola*, Bean line pattern mosaic virus (BLPMV)	Bassanezi et al., 1998	Antagonism	Plants infected with virus presented reduced final fungal disease severities.
Common bean	*Uromyces appendiculatus*, Bean line pattern mosaic virus (BLPMV)	Dalla Pria et al., 1994	Antagonism	Plants infected with BLPMV presented a lower severity of rust.
Common bean	*Uromyces appendiculatus, Xanthomonas axonopodis* pv. *phaseoli*	Finke et al., 1986; Defaria & Hagedorn, 1986	Additive	No antagonistic or synergistic interactions were observed on separate trifoliolate leaves.
Common bean	*Uromyces appendiculatus*, Bean common mosaic virus (BCMV)	Zaiter et al., 1990	Antagonism	Pre-infection of bean seedlings with the virus reduced the rust pustule size in 25%.
Common bean	*Xanthomonas axonopodis* pv. *phaseoli*; Bean common mosaic virus (BCMV)	Zaiter et al., 1990	Additive	Pre-infection of bean seedlings with BCMV did not affect the reaction to the bacteria.

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Host	Involved pathogens	Authors	Type	Comments of the authors
Common bean	*Xanthomonas axonopodis* pv. *phaseoli*, Bean common mosaic virus (BCMV)	Hedges, 1944; 1946a; 1946b; Panzer & Nickeson, 1959	Synergism	A synergistic effect of the two pathogens was observed only late in the growing season.
Common bean	*Uromyces appendiculatus*, Tobacco mosaic virus (TMV)	Wilson, 1958	Antagonism	Leaves infected with TMV presented resistance to rust, apparently due to inhibition of uredospore germination; leaves infected with rust presented resistance to TMV infection.
Common bean	*Uromyces appendiculatus*, Tobacco necrosis virus (TNV)	Kutzner et al., 1993	Antagonism	Preinoculation with rust induced resistance against the virus.
Common bean	*Pseudomonas phaseolicola*, *Achromobacter* sp.	Maino et al., 1974	Synergism	The number of lesions incited by *P. phaseolicola* increased from 2-to nearly 4-fold when inocula were mixed with *Achromobacter* sp.
Common bean	*Pseudomonas phaseolicola*, *P. marginalis*, *P. fluorescens*, *Achromobacter* sp.	Maino et al., 1974	Antagonism	The number of lesions of *P. marginalis* and *P. fluorescens* decreased when the pathogens were mixed with *Achromobacter* sp.
Cucumber and muskmelon	*Erysiphe cichoracearum* or *Sphaerotheca fuliginea* and *Mycosphaerella melonis*	Bergstrom et al., 1982	Synergism	Powdery mildew effectively predisposed the plants to *M. melonis*.
Cucumber	Cucumber vein yellowing virus (CVYV), Cucurbit yellow stunting disorder virus (CYSDV)	Gil-Salas et al., 2012	Synergism	Co-infection with CVYV and CYSDV leading to synergism.
Cucumber	*Sphaerotheca fuliginea*, Tobacco necrosis virus (TNV)	Conti et al., 1990	Antagonism	Preinoculation with TNV enhances peroxidase activity and lignification in cucumber, as a resistance response to *S. fuliginea*.
Cucumber	*Erysiphe polyphaga*, Cucumber mosaic virus (CMV)	Blumer et al., 1955	Antagonism	A distinct antagonism between the pathogens was observed.
Faba bean	*Uromyces viciae-fabae*, Bean yellow mosaic virus, Bean leaf roll virus (BLRV)	Omar et al., 1986	Antagonism	Prior infection with viruses decreased pustule density on leaves subsequently infected with *U. viciae-fabae*.
Faba bean	*Botrytis fabae*, *B. cinerea*, Bean yellow mosaic virus (BYMV), Bean leaf roll virus (BLRV)	Omar et al., 1986	Synergism	Prior infection with viruses increased host susceptibility to subsequent infection by *B. fabae* and *B. cinerea*.
Gladiolus	*Curvularia trifolii* f. sp. *gladioli*, Cucumber mosaic virus (CMV), Tobacco ringspot virus (TobRSV)	Beute, 1973	Synergism	The severity of *Curvularia* was increased by infection by viruses.
Groundnut	*Cercospora arachidicola*, *Phoma arachidicola*	Cole, 1982	Not clear	Cercospora leaf spot was not influenced by the amount of web blotch. The correlation matrices also showed the greater dependence of web blotch on Cercospora leaf spot.
Maize	*Helminthosporium maydis* race 0, Maize dwarf mosaic virus (MDMV)	Stevens & Gudauskas, 1982; 1983	Synergism	MDMV-infected plants were more susceptible to *H. maydis* than virus-free ones. *H. maydis* sporulated more abundantly on MDMV-infected leaves and the inoculum potential was greater than that of conidia produced on MDMV-free leaves.
Maize	*Peronosclerospora philippinensis*, *P. sorghi*, Maize streak virus (MSV)	Damsteegt et al., 1993	Additive	Neither pathogen affected the establishment of the other. Reduction in height, fresh weight and leaf area were greater (thought not significant) with pathogen combinations than with single pathogens.
Maize and sorghum	*Helminthosporium maydis* race 0, Maize dwarf mosaic virus (MDMV)	Beniwal & Gudauskas, 1974	Synergism	There was a positive association between MDMV infection and *H. maydis* susceptibility.
Nicotiana spp.	Helper tobamoviruses, Satellite tobacco mosaic virus (STMV)	Valverde et al., 1991	Antagonism	Yield of TMV was reduced in 32–48% by coinfection with STMV.
Nicotiana benthamiana	Tobacco necrosis virus (TNV), Turnip crinkle virus (TCV)	Xi et al., 2010	Antagonism	Accumulation of TNV (+)RNA as well as capsid protein in mixed infection were considerably lower than that of singly infected plants. There were also a slight reduction in the levels of TCV (+)RNA and capsid protein in doubly infected plants, which displayed the concentration of both viruses decreased in doubly infected plants.

Host	Involved pathogens	Authors	Type	Comments of the authors
Nicotiana benthamiana	*Cucumber mosaic virus* (CMV), *Tobacco necrosis virus* (TNV)	Xi et al., 2007	Synergism	Mixed infections of *N. benthamiana* plants by CMV and TNV exhibit a synergistic interaction and result in symptom enhancement.
Nicotiana benthamiana and tomato	*Abutilon mosaic virus* (AbMV), *Tobacco mosaic virus* (TMV), *Tomato mosaic virus* (ToMV)	Pohl & Vege, 2007	Synergism	Both tobamoviruses exerted a negative effect on the DNA virus, resulting in a decrease in AbMV accumulation and significantly reduced infectivity in *N. benthamiana*. A striking synergistic enhancement in pathogenicity occurred with respect to stunting and necrosis.
Nicotiana benthamiana	*Potato virus X* (PVX), *Potato virus Y* (PVY), *Plum pox virus* (PPV)	Pacheco et al., 2012	Synergism	In comparison to single infections, co-infection of *N. benthamiana* with PVX and PVY or PPV resulted in increased systemic symptoms.
Nicotiana benthamiana and tomato	*Tomato rugose mosaic virus* (ToRMV), *Tomato yellow spot virus* (ToYSV)	Alves Júnior et al., 2009	Antagonism/ Synergism	ToYSV establishes a systemic infection and reaches a higher concentration earlier than ToRMV in both hosts. ToRMV negatively interferes with ToYSV during the initial stages of infection, but once systemic infection is established this interference ceases.
Orchids	*Cymbidium mosaic virus* (CyMV), *Odontoglossum ringspot virus* (ORSV)	Pearson & Cole, 1986; 1991	Not clear	The mean values also suggest that mixed infections were less serious than single infection by CyMV, but again the differences are not statistically significant.
Pea	*Bean yellow mosaic virus* (BYMV), *Cucumber mosaic virus* (CMV)	Blaszczak & Milchert, 1980	Synergism	Mixed infection caused a little more severe disease symptoms and somewhat greater decreases of plant weight than CMV alone.
Peanut	*Tomato spotted wilt virus* (TSWV), *Peanut mottle virus* (PMV)	Hoffmann et al., 1998	Additive	Dual infection did not cause greater symptom severity than infection with either virus alone.
Pepper	*Cucumber mosaic virus* (CMV), *Pepper mottle virus* (PMV)	Murphy & Kyle, 1995	Synergism	When plants were coinfected with PMV and CMV, antigen accumulation of PMV was no longer restricted temporally or spatially.
Pepper	Helper viruses (*Tobacco mosaic virus* – TMV-U2, *Pepper mild mottle virus* – PMMV) and *Satellite tobacco mosaic virus* (STMV)	Rodriguez-Alvarado et al., 1994	Synergism	Presence of STMV in coinfection with TMV-U2 caused increased chlorosis relative to the mosaic caused by TMV-U2 alone. Increased chlorosis was observed when TMV-U5 and PMMV were used as helper viruses.
Pepper	*Pepper huasteco virus* (PHC), *Pepper golden mosaic virus* (PepGMV)	Méndez-Lozano et al., 2003	Antagonism	In terms of symptom expression, antagonism was observed.
Pepper	*Potato virus Y* (PVY), *Tobacco mosaic virus* (TMV)	Sherwood et al. 1988	Synergism	TMV and PVY inoculated together caused severe stunting and systemic necrosis.
Ryegrass	*Puccinia coronata*, *Ryegrass mosaic virus* (RMV), *Barley yellow dwarf virus* (BYDV)	Latch & Potter, 1977	Antagonism (Pc-RMV) Additive (Pc-BYDV)	Two isolates of RMV suppressed the amount of crown rust emerging on leaves by up to 75% compared with virus-free plants. Severity of rust infection on BYDV infected plants generally did not differ significantly from that on virus-free plants.
Soybean	*Cercospora sojina*, *Colletotrichum truncatum*	Kunwar et al., 1985	Antagonism	In mixed infection, hyphae of *C. sojina* were more abundant than those of *C. truncatum*.
Soybean	*Cowpea mosaic virus* (CPMV), *Bean pod mottle virus* (BPMV), *Soybean mosaic virus* (SMV)	Anjos et al., 1992	Synergism	Plants dually infected with SMV and either CPMV or BPMV showed greatly increased symptoms severity over that induced by the individual viruses.
Soybean	*Soybean mosaic virus* (SMV), *Bean pod mottle virus* (BPMV)	Quinones et al., 1971; Ross, 1968; 1969; Hobbs et al., 2003	Synergism	Mixed virus infection on soybean with SMV and BPMV was reported to produce higher percentages of seed coat mottling than that caused by SMV alone, but not always.
Soybean	*Soybean mosaic virus* (SMV), *Phomopsis* spp.	Koning et al., 2001; 2003	Synergism	SMV infection predisposes soybean seed to *Phomopsis* spp. Seed infection and may induce seedcoat mottling.
Sugar beet	*Beet necrotic yellow vein virus* (BNYVV), *Beet soilborne mosaic virus* (BSBMV)	Wisler et al., 2003	Antagonism	BNYVV may suppress BSBMV in mixed infections.

Host	Involved pathogens	Authors	Type	Comments of the authors
Sugar beet	*Cercospora beticola, Beet yellows virus* (BYV)	Crane & Calpouzos, 1969	Synergism	In the virus-fungus treatment, the average number of *Cercospora* lesions increased 53%, while the average number of dead leaves increased 260% when compared to the treatment with fungus alone.
Sweet Potato	*Cucumber mosaic virus* (CMV), *Sweet potato feathery mottle virus* (SPFMV)	Cohen et al., 1988	Synergism	For replication of CMV in sweet potato, a factor was required that was provided by the presence of SPFMV.
Tobacco	*Pepper huasteco virus* (PHC), *Pepper golden mosaic virus* (PepGMV)	Méndez-Lozano et al., 2003	Synergism	In terms of symptom expression, synergism was observed.
Tobacco	*Potato virus X* (PVX), *Potato virus Y* (PVY)	Hoffmann-Wolf et al., 1990	Synergism	All the combinations of the viruses strains produced the same typical synergistic symptom.
Tobacco	*Pepper veinal mottle virus* (PVMV), *Potato virus Y* (PVY)	Marchoux et al., 1993	Synergism	In the presence of PVY, PVMV migrated systemically into the upper leaves of the tobacco plants.
Tobacco	*Potato virus X* (PVX), *Potato virus Y* (PVY)	Goodman & Ross, 1974	Synergism	Enhancement of the viruses amount occurs only in cells invaded by the two viruses within a relatively short period of time and the maximum enhancement results when critical stages in the replication cycle of each virus coincide.
Tobacco	*Tobacco etch virus* (TEV), *Tobacco vein mottling virus* (TVMV)	Madden et al., 1987	Additive	Neither virus had a consistent inhibitory effect on increase of the other.
Tobacco	*Erysiphe cichoracearum, Tobacco mosaic virus* (TMV), *Tobacco necrosis virus* (TNV)	Marte et al., 1993	Antagonism	TMV or TNV induced systemic resistance to subsequent inoculation with the powdery mildew fungus.
Tomato	*Potato virus Y* (PVY-SON41), *Cucumber mosaic virus* (CMV-Fny and CMV-LS	Mascia et al., 2010	Synergism	Mixed infection with the SON41 strain of PVY-SON41 increased accumulation of RNAs of strains Fny and LS of CMV-Fny and CMV-LS, respectively, and enhanced disease symptoms.

[a]See below selected examples for wheat.

TABLE 2 - Selected examples of interactions related mainly to crop loss.

Host[a]	Involved pathogens	Authors	Type	Comments of the authors
Barley	*Erysiphe graminis* f. sp. *hordei*, *Puccinia hordei*	Lim & Gaunt, 1986a;b	Synergism	Leaf rust severity after anthesis was most damaging when combined with earlier mildew epidemics. The mean yield after early mildew epidemics (568g/m^2) was similar to that after late leaf rust (573g/m^2), but greater than that after the combined epidemics (474g/m^2).
Common bean	Bean common mosaic virus (BCMV), Bean rugose mosaic virus (BRMV)	Castillo-Urquiza et al., 2006	Synergism	Single infection of 'Ouro Negro' and 'Novirex' beans caused a reduction in the total weight of pods per plant of 3.4% and 84.9%, respectively. Mixed infection with BCMV caused a reduction of pod weight per plant of up to 70.1% in 'Novirex' and up to 90.8% in 'Ouro Negro'.
Common bean	*Colletotrichum lindemuthianum*, *Pseudocercospora griseola*	Carneiro et al., 2000	Additive	No significant differences were observed between plants inoculated with both pathogens and those inoculated with each pathogen separately.
Common bean	*Uromyces appendiculatus*, *Colletotrichum lindemuthianum*	Lopes & Berger, 2001	Additive	There was no interaction on the impact of the photosynthesis, i.e., the presence of rust did not change the impact caused by anthracnose on leaf photosynthesis and vice versa.
Common bean	*Uromyces appendiculatus*, *Pseudocercospora griseola*	Jesus Júnior et al., 2001	Antagonism	There was no combined effect of the diseases on host growth, but there seems to be an antagonistic effect of the diseases on yield.
Common bean	*Uromyces appendiculatus*, *Pseudocercospora griseola*	Jesus Júnior et al., (data not published)	Antagonism	There was a clear reduction on crop leaf area, light interception, dry matter, RUE and harvest index due to the two pathogens, compared with plants infected with only one pathogen.
Cowpea	Blackeye cowpea mosaic virus (BICMV), Cucumber mosaic virus (CMV)	Pio-Ribeiro et al., 1978	Synergism	CMV alone reduced yield in 14.2%; BICMV alone in 2.5%; in combination in 86.4%.
Lettuce	Beet western yellows virus (BWYV), Lettuce mosaic virus (LMV), Cucumber mosaic virus (CMV)	Walkey & Payne, 1990	Synergism	The interaction resulted in a significantly greater yield loss than that caused by the viruses alone.
Maize	Maize dwarf mosaic virus (MDMV), Maize chlorotic mottle virus (MCMV)	Uyemoto et al., 1981	Synergism	Losses were heaviest on plants receiving the virus combination.
Oat	*Helminthosporium avenae*, Barley yellow dwarf virus (BYDV)	Sommerfeld et al., 1993	Additive	No interaction was observed.
Peanut	*Cercospora personata*, *Puccinia arachidis*	Harrison, 1973	Additive	Leaf rust can be nearly as destructive alone as when it occurs in combination with *Cercospora* leaf spot.
Ryegrass	Barley yellow dwarf virus (BYDV), Ryegrass mosaic virus (RMV)	Catherall, 1987	Additive	Both viruses together rarely caused greater damage than the most damaging virus on its own.
Soybean	*Pseudomonas glycinea*, *Septoria glycines*	Williams & Nyvall, 1980	Antagonism	*P. glycinea* alone reduced yield in17.9%; *S. glycines* alone in 17.4%; both pathogen together in 14.1%.
Soybean	*Pseudomonas syringae* pv. *glycinea*, *P. syringae* pv. *tabaci*, *Xanthomonas campestris* pv. *glycines*	Hwang & Lim, 1992	Additive	Effects of individual and multiple diseases on yield were not statistically significant.
Soybean	*Pseudomonas syringae* pv. *glycinea*, *Septoria glycines*	Basu & Butler, 1988	Not clear	The pathogens together were additive in the amount of foliage infection produced when disease levels were relatively low; otherwise the combined effect was not significantly greater than that of the *S. glycines* alone.

[a]See below selected examples for wheat.

TABLE 3 - Selected examples of interactions related to disease dynamics and crop loss.

Host	Involved pathogens	Authors	Type	Comments of the authors
Barley	*Erysiphe graminis*, *Puccinia hordei*	Simkin & Wheeler, 1974	Antagonism / Additive	Pre-inoculation with either fungus reduced the development of the other. The effects of mildew and rust in reducing grain yield are nearly additive.
Barley, oat	*Erysiphe graminis*, *Barley yellow dwarf virus* (BYDV)	Potter, 1980	Not clear / Additive	Prior infection by BYDV initially suppressed, then subsequently enhanced the amount of powdery mildew. No additional yield loss was caused by mildew infection on BYDV-infected plants.
Barley, oat	*Puccinia recondita* f. sp. *tritici*, *P. recondita* f. sp. *avenae* or *P. hordei* and *Barley yellow dwarf virus* (BYDV)	Potter, 1982	Not clear	The number of pustules on distal leaf portions was negatively correlated with the amount of yellowing of the leaf areas scored. The latent period o f rust on oats was not affected by BYDV. In barley, BYDV reduced the latent period of rust on leaf 5, but not on leaf 4, and reduced it on proximal, but not distal, leaf portions. When infected with both BYDV and rust, yield of oats was reduced by 91%.
Common bean	*Uromyces appendiculatus*, *X. axonopodis* pv. *phaseoli*	Zaiter et al., 1990	Synergism	Depending on the level of the host susceptibility to rust, the reaction of *X. axonopodis* pv. *phaseoli* remained confined within the rust pustule or spread beyond the pustule area, causing a necrosis of the entire leaf. Moreover, rust accentuated the damage caused to foliage by *X. campestris* pv. *phaseoli*.
Pea	*Mycosphaerella pinodes*, *Phoma medicaginis* var. *pinodella*	Le May et al., 2009	Antagonism / Synergism	The presence of the two pathogens on the same host plant organ limited the disease development and their reproduction. Damages caused by the two pathogens, however, increased when plants that had been previously inoculated were inoculated with the other species.
Sugar beet	*Beet yellows virus* (BYV), *Beet mosaic virus* (BtMV), *Beet western yellows virus* (BWYV)	Wintermantel, 2005	Synergism	Titers of all three viruses increased as a result of co -infection compared with single infections. The overall rate of appearance o f BWYV symptoms increased during co -infection with BtMV. Severe stunting, as measured by fresh plant biomass, was observed with mixed infections with BYV and BtMV, compared to single infections of these viruses.
Sugar beet	*Erysiphe betae*, *Beet mild yellowing virus* (BMYV)	Ahrens, 1981	Synergism / Not clear	*E. betae* increased virus yellowing. The mutual favouring of disease progress, different in extent according to variety, affected varietal yield below or above average.
Tomato	*Tomato chlorosis virus* (ToCV), *Tomato spotted wilt virus* (TSWV)	García-Cano et al., 2006	Synergism	Synergism in a tomato cultivar susceptible to both viruses resulted in a rapid death of plants. A pronounced enhancement of ToCV accumulation mediated by TSWV co -infection was observed with no evident egress of ToCV from phloem tissues. No consistent alteration of TSWV accumulation was detected.
Zucchini squash, melon	*Zucchini yellow mosaic virus* (ZYMV), *Cucumber mosaic virus* (CMV)	Wang et al., 2002	Synergism	Plants coinfected by ZYMV and either Fny-CMV (subgroup IA) or LS -CMV (subgroup II) displayed strong synergistic pathological responses, eventually progressing to vascular wilt and plant death.

aSee below selected examples for wheat.

example, wheat is being introduced gradually in the cerrado areas usually under irrigation, which may be favourable to the infection by foliar pathogens.

Different parts of the wheat plant can be infected by different pathogens at the same time (Bonfig-Picard & Kranz, 1984). These pathogens can naturally interact with each other. Interactions between diseases caused by aerial pathogens on wheat have frequently been demonstrated, mainly with respect to the dynamics of the pathogens. However, even for wheat, the crop that receives the most attention regarding interactions among diseases, there are little multi-disease studies concerning management or epidemiological approaches. Moreover, the observed effects of reported interactions (for instance synergism, antagonism or additive effect) are highly dependent on inoculum concentration, disease levels (Jörg, 1987; Weber et al., 1994), pre-infection by one of the interacting pathogens (Donchev et al., 1980), and climatic conditions (Weber et al., 1994). Cox et al. (2004) have demonstrated the potential of cultivar mixtures for the simultaneous management of tan spot (*Pyrenophora tritici-repentis*) and leaf rust of wheat (*Puccinia triticina*).

Johnston (1934) observed that uredospores of *Puccinia recondita* f. sp. *tritici* could develop on normally resistant varieties of wheat when the leaves were infected with *Erysiphe graminis* f. sp. *tritici* before the rust inoculation. Similarly, Manners & Gandy (1954) reported higher susceptibility to *P. recondita* of some wheat varieties infected with mildew. However, when mildew severity was high, the rust development was limited. On the other hand, the severity of mildew was significantly reduced on wheat plants previously inoculated with *P. recondita*, maybe due to biochemical changes in the plants (Donchev et al., 1980).

In many studies, it has been demonstrated that one pathogen is promoted by another. Plants infected with *Tilletia caries* are more susceptible to *P. striiformis* (Straib, 1938). In the presence of *Ustilago nuda*, the damage by *P. graminis* f. sp. *tritici* is higher (Hart, 1931 cited by Straib, 1938, Thomas & Chatarth, 1976). Raju et al. (1969) found that the number of pustules of *P. recondita* was increased in the presence of wheat streak mosaic virus (WSMV). In the same way, the number and the size of lesions caused by *Helminthosporium sativum* was higher when the plants were infected by WSMV (Adlakha & Raychaudhuri, 1975). Wainwright et al., (1986) observed that *S. nodorum* caused more damage than *T. caries*. According to Willingale & Mantle (1987) it was evident in the interaction involving *Claviceps purpurea* and *T. caries* that invasion by *C. purpurea* was essentially a displacement phenomenon, which characterize *C. purpurea* parasitism of healthy ovaries. The establishment of the sphacelium and subsequent differentiation to sclerotial tissue was more rapid in bunted rather than non-bunted ovaries.

One pathogen can also inhibit the development of another. Damage caused by *Urocystis agropyri* is reduced in the presence of *T. controversa* (Holton & Jackson, 1951).

The severity of Helminthosporium blight (*H. sativum*) was lower in plants infected with *U. nuda* (Thomas & Chatarth, 1976). Jones & Roane (1982) found that *Xanthomonas campestris* pv. *undulosa* reduced germination and the length of germinative tube of spores of *S. nodorum*. Potter (1982) observed that, in plants infected with barley yellow dwarf virus (BYDV), the severity of *P. recondita* was lower, although the latent period was not affected. When infected with both BYDV and rust, yield was reduced by 63%. Erasmus & Von Wechmar (1983) observed that wheat plants with brome mosaic virus (BMV) were found less susceptible to *P. graminis* f. sp. *tritici* infection than virus-free plants. Adee et al. (1990) found that competition occurred between *Pyrenophora tritici-repentis* and *S. nodorum*. Sporulation by *Puccinia triticina* was reduced substantially by the presence of *Pyrenophora tritici-repentis*; in contrast, the presence of *Puccinia triticina* sometimes increased sporulation of *Pyrenophora tritici-repentis* (Al-Naimi, 2003).

No interaction was observed for the combinations *P. striiformis* x *U. agropyri* (Purdy & Holton, 1963) and *S. nodorum* x *S. tritici* (Jenkins & Jones, 1981). Hyde (1981) observed no interaction for the combination *S. nodorum* x *P. striiformis* regarding seed weights, although the proportion of diseased leaves for the combination was less than the sum of effects of each pathogen occurring alone. Jones et al. (1981) observed that the apparent photosynthetic rate and transpiration rate of flag leaves did not differ statistically in the *X. translucens* f. sp. *undulosa* + *S. nodorum* treatment compared with *S. nodorum* alone. At lower incubation temperatures, combined inoculation had no effect compared with inoculation with either organism alone.

Van der Wal et al. (1970) and Van der Wal & Cowan (1974) observed synergistic effect on crop losses when they studied the combination *P. recondita* x *S. nodorum*. Van der Wal et al. (1970) observed also that on plants infected by *P. recondita*, the intensity of glume blotch symptoms caused by *S. nodorum* was greater than on not inoculated plants. They observed also that, in the presence of *S. nodorum*, the production of uredospores of *P. recondita* was reduced while the production of teliospores was stimulated. The loss caused by the interacting fungi is significantly larger than the calculated sum of the losses caused by each fungus alone. Wheat plants infected by *P. recondita* were predisposed to infection by *S. nodorum* (Van der Wal & Cowan, 1974). According to these authors the effect of both pathogens together on the dry weight of the heads was greater than the sum of the effects of each of the pathogen separately. However, no interaction was observed when the infection by *S. nodorum* occurred before *P. recondita* infection (Hyde, 1978). Spadafora & Cole (1987) found an inverse relationship between the severities of *P. recondita* and *S. nodorum*.

Broscious et al. (1982) and Bonfig-Picard & Kranz (1984) have observed a competition between populations of *S. nodorum* and *E. graminis* f. sp. *tritici*. Geuting (1984) found that in the presence of *S. nodorum* the number of

mildew colonies was lower and the colonies were smaller compared to the control plants. The number of conidia/colony was also lower. The latent period of *S. nodorum* was shortened in the presence of *E. graminis* f. sp. *tritici*. Jörg (1987) confirmed under field conditions the results of Geuting (1984), although he found no influence of *E. graminis* on *S. nodorum*. The development of mildew was inhibited in the presence of *S. nodorum* and the effect was more pronounced at high severity of the diseases. Similarly to Geuting (1984), Brokenshire (1974) observed reduced latency duration of *S. tritici* in the presence of *E. graminis*. Resistant plants were susceptible to *S. tritici* in the presence of mildew. There was an inverse relationship, based on individual replicate values, between the pre-inoculation mildew treatments and the latent period of *S. tritici*, but a significant positive correlation for the sporulation index. For this combination, Madariaga & Scharen (1984) observed that in the presence of *S. tritici*, the effect of *P. striiformis* was always reduced by the presence of *M. graminicola*. The two pathogens could colonize the same leaf simultaneously, and the diseased area was similar or smaller than the area affected by each pathogen separately. A smaller amount of leaf tissue was colonized by *P. striiformis* when *M. graminicola* was present. *M. graminicola* acted as a hypostatic parasite.

Weber et al. (1994) observed that, in greenhouse, *S. nodorum* reduced the severity of mildew. On the other hand, under field conditions, *E. graminis* increased the final intensity of *S. nodorum*. The apparent contradiction was explained as a result of different climatic conditions, which allowed secondary infections of *S. nodorum* in the field. Based on the studies of Brokenshire (1974), Geuting (1984) and Jörg (1987), Weber et al. (1994) concluded that these pathogens couldn't strictly be described as competitors *sensu* Odum (1953). The relation between *E. graminis* and *S. nodorum* is better described, *sensu* Powell (1979), as "predisposition with dominance of the secondary pathogen", with *E. graminis* as primary, and *S. nodorum* as secondary pathogen (Jörg, 1987; Weber et al., 1994).

Tatineni et al. (2010) studied the double infection of wheat cultivars with *Triticum mosaic virus* (TriMV), the type member of the *Poacevirus* genus, and *Wheat streak mosaic virus* (WSMV), the type member of the *Tritimovirus* genus, both of the family *Potyviridae*. They found that double infections in wheat cvs. Arapahoe and Tomahawk induced disease synergism with severe leaf deformation, bleaching, and stunting, with an increase in accumulation of both viruses over single infections at 14 days post inoculation.

QUANTIFYING INTERACTIONS

Interactions in disease dynamics

As mentioned above, the interactions between pathogens or diseases can be of various natures, for instance characterised as competition, symbiosis, parasitism, etc. Independent of the kind of interactions, the effect is reflected in changes of the disease progress curves compared to the situation in which each disease is occurring alone. Some research has been published showing the effect of the pre-infection of one disease on a second disease. A detailed study for this approach to interactions was presented by Bassanezi et al. (1998) who investigated the effect of pre-infection with *Bean line pattern mosaic virus* (BLPMV) on some elements of *Uromyces appendiculatus* and *Phaeoisariopsis griseola* over a wide range of temperatures. Other studies have applied the de Wit replacement series technique to investigate the outcome of competitive interactions between two plant pathogens using the conidial production (Adee et al., 1990; Nolan et al., 1999). In order to compare epidemics of interacting diseases, the area under the disease progress curve have been calculated and analysed using analyses of variance (Savary & Zadoks, 1992a).

In many publications the disease dynamics of single diseases is modelled, but only a few examples have been published in which the progress of epidemics in a multiple disease situation has been quantified and modelled. This approach will be discussed in more detail, starting with the classical Lotka-Volterra competition model. Madden et al. (1987) applied the classical Lotka-Volterra competition equations to model the disease progress curves of the disease incidence of tobacco etch virus (TEV) and tobacco vein mottling virus (TVMV). The model is based on the assumption that the disease progress curves are logistic functions if one disease would be there alone. The competition model is given by the following system of differential equations for the disease severity or incidence of two diseases y_1 and y_2 given as proportions with values between 0 and 1:

$$\mathrm{d}y_1/\mathrm{d}t = r_1 \, y_1 \, (1 - [y_1 + \alpha a_{12} \, y_2] / K_1)$$
$$\mathrm{d}y_2/\mathrm{d}t = r_2 \, y_2 \, (1 - [y_2 + \alpha a_{21} \, y_1] / K_2) \tag{1}$$

The parameters r_1 and r_2 are the apparent infection rates and K_1 and K_2 the maximum disease levels of both diseases in absence of the other disease. The two parameters αa_{12} and αa_{21} are the coefficients of competition. The coefficient αa_{12} indicates the competitive effect of disease 2 on disease 1. The system of differential equations (eq. 1) can be re-arranged to point out the mutual effects of the diseases on each other:

$$\mathrm{d}y_1/\mathrm{d}t = r_1 \, (1 - \alpha a_{12} \, y_2 / K_1) \, y_1 \, (1 - y_1 / [K_1 \, (1 - \alpha a_{12} \, y_2 / K_1)])$$
$$\mathrm{d}y_2/\mathrm{d}t = r_2 \, (1 - \alpha a_{21} \, y_1 / K_2) \, y_2 \, (1 - y_2 / [K_2 \, (1 - \alpha a_{21} \, y_1 / K_2)]) \tag{2}$$

The new system (eq. 2) clearly shows that the actual apparent infection rate and the actual maximum disease level of each disease are linearly decreasing with increasing severity of the other disease. Moreover, the reducing effect is identical to the rate and the capacity.

In the example of Madden et al. (1987) the dynamics of two tobacco virus diseases TEV (*Tobacco etch virus* = 1) and TVMV (*Tobacco vein mottling virus* = 2) occurring

at the same time in tobacco fields were modelled. Disease incidence progression was quantified by fitting the Lotka-Volterra equations (eq. 1), resulting in the following parameters values for the data set in 1984 B without insecticide usage: $r_1 = 0.223$/day, $r_2 = 0.261$/day, $K_1 = 0.175$, $K_2 = 1.00$, $\alpha a_{12} = 0.06$ and $\alpha a_{21} = 1.67$. From eq. 2 it can be concluded that the actual rate and the maximum disease level of TEV are reduced by 0.34% when the disease incidence of TVMV increases 1%. The reduction of TVMV is 1.67% per 1% increase of TEV. Due to the interaction modelled as competition, the maximum disease levels of both diseases are reduced.

Ngugi et al. (2001) used the Lotka-Volterra equations (eq. 1) to simultaneously describe the disease progress curves of sorghum anthracnose (caused by *Colletotrichum sublineolum*) and leaf blight (caused by *Exserohilum turcicum*). In most cases the competition coefficients were not significantly different from 0 so they concluded that interactions between both diseases did not occur.

Although the Lotka-Volterra competition equations have been successfully applied in these examples, the general disease progression resulting from the model may not reflect interacting plant disease epidemics in a real situation. The equations allow a decrease in disease intensities, which is not possible without regarding changes of the host plant. Even under adverse conditions to the pathogen and the disease, the leaf area covered by disease symptoms cannot decrease and thus the disease levels in the worst case remain constant. Thus it is logical to demand that in the Lotka-Volterra equations the changes of the disease severity must be equal or greater than 0 ($dy_1/dt \geq 0$ and $dy_2/dt \geq 0$). This can be achieved by introducing the maximum function max$(0; x)$ which is zero if x is below 0. The Lotka-Volterra equations can then be replaced by the following model:

$$dy_1/dt = \max(0; r_1 y_1 (1 - [y_1 + \alpha a_{12} y_2] / K_1))$$
$$dy_2/dt = \max(0; r_2 y_2 (1 - [y_2 + \alpha a_{21} y_1] / K_2)) \qquad (3)$$

It must be pointed out that this more biological approach has a disadvantage as the equilibrium values of the interacting diseases are not fixed, like in the original Lotka-Volterra model, but depend on the initial disease values.

Weber (1996) used this approach to describe disease progress curves of wheat powdery mildew (*E. graminis* f. sp. *tritici*) and leaf blotch disease (*S. nodorum*) and their interactions. In addition to this change of the Lotka-Volterra equations, he included a promoting effect as possible interaction between diseases, thus going beyond a competition model. For the interactions between *E. graminis*, a biotrophic pathogen, and *S. nodorum*, a perthotrophic fungus (which initiates infection as a biotroph but spends most of its life cycle as a necrotroph), he assumed an inhibiting effect of *S. nodorum* on *E. graminis* and a disease-promoting effect of *E. graminis* on *S. nodorum*. This led to the following model

for the interactions between the diseases whereby y_M and y_S represent the disease severities (as proportions) of powdery mildew and *Septoria* leaf blotch, respectively:

$$dy_M/dt = \max [0; r_M y_M (1 - y_M / K_M - a_s y_S)]$$
$$dy_S/dt = r_S y_S (1 - y_S / K_S + a_M y_M) \qquad (4)$$

Again, r_M and r_S are infection rates, K_M and K_S the maximum severity levels of mildew and *Septoria* leaf blotch, respectively, without mutual influences. The interaction term a_M (> 0) gives the influence of mildew on *Septoria* disease, and the coefficient a_s (> 0) the influence of *Septoria* leaf blotch on mildew. Here the *Septoria* disease is modelled adversely to mildew, as a competitor for infection places. However, the effect of mildew is incorporated as a factor delaying the density regulation of *Septoria* disease by the positive of sign. The function max prevents a decline of the mildew growth rate which can be biologically interpreted as the exclusion of the overgrowing of mildew lesions by *Septoria* leaf blotch.

To show the similarity to the Lotka-Volterra equations, Weber's system (eq. 4) can be re-arranged resulting in the following equations (eq. 5):

$$dy_M/dt = \max \{0; r_M (1 - a_s y_S) y_M (1 - y_M / [K_M (1 - a_s y_S)])\}$$
$$dy_S/dt = r_S (1 + a_M y_M) y_S (1 - y_S / [K_S (1 + a_M y_M)]) \qquad (5)$$

The effect of *Septoria* leaf blotch on mildew is reflected in the reduced infection rate as well as in the decreased maximum disease level of mildew. On the other side, increasing mildew severity raises the actual infection rate and the maximum disease level of *Septoria* disease. The relative changes of the rate and capacity parameter values as a result of the interaction are identical, negative for mildew, but positive for *Septoria* leaf blotch.

For data of disease progression in 1991, the following parameter values were estimated by Weber (1996): $r_M = 0.2$ / day, $r_S = 0.19$ / day, $K_M = 0.07$, $K_S = 0.334$, $a_M = 17.13$ and $a_s = 4.32$. Thus, an increase of *Septoria* disease by 1% reduces the mildew parameter values by 4.32%, while a 1% increase of mildew raises the *Septoria* parameter values by 17.13%. Simulated disease progress curves of both diseases in 1991 are calculated according to the modified Lotka-Volterra equations (eq. 5) and under the assumption of no disease interaction.

In a second model, Weber (1996) assumed that mildew is not changing the maximum disease level of *Septoria* leaf blotch so that the model can be written as:

$$dy_M/dt = \max \{0; r_M (1 - a_s y_S) y_M (1 - y_M / [K_M (1 - a_s y_S)])\} \qquad (6)$$
$$dy_S/dt = r_S (1 + a_M y_M) y_S (1 - y_S / K_S)$$

In contrast to the previous interaction models, the rate and capacity parameters are now not affected in the same way, as the capacity of *Septoria* leaf blotch remains unchanged in presence of powdery mildew. Weber (1996)

also fitted this model to the data of disease progression in 1991 and determined the following parameter values: $r_M =$ 0.29 / day, $r_S = 0.14$ / day, $K_M = 0.066$, $K_S = 0.40$, $a_M = 57.83$ and $a_S = 3.29$. When the disease incidence of *Septoria* leaf blotch increases by 1%, the actual rate and the maximum disease level of mildew are reduced by 3.29%. A 1% increase of mildew increases the rate of *Septoria* disease by 57.83%, however, without changing the maximum disease level of *Septoria* leaf blotch.

Following Weber's approach by assuming that in an interaction the rate and the capacity parameters are affected in the same direction but to a different extent, a general interaction model can be constructed:

$$\mathrm{d}y_1/\mathrm{d}t = r_1 \, (1 - \alpha a_{12} \, y_2 \, / \, K_1) \, y_1 \, (1 - y_1 \, / \, [K_1 \, (1 - \beta b_{12} \, y_2 \, / \, K_1)]) \tag{7}$$
$$\mathrm{d}y_2/\mathrm{d}t = r_2 \, (1 - \alpha a_{21} \, y_1 \, / \, K_2) \, y_2 \, (1 - y_2 \, / \, [K_2 \, (1 - \beta b_{21} \, y_1 \, / \, K_2)])$$

The model would be more flexible because of additional parameters. The coefficients αa_{ij} describe the mutual effects on the rates and the βb_{ij} on the maximum values. However, to our knowledge this approach has never been tested in explaining disease dynamics.

The models discussed so far describe the interactions of plant disease epidemics without taking into consideration the host plant. Host growth, however, can change the dynamic of diseases, leading for instance to a decrease in disease severity if the host is growing faster than the disease is progressing. Thus the host influences the disease dynamics. On the other hand, a disease can affect host growth in different ways, as classified by Boote et al. (1983). The mutual effects of host and diseases form another important interaction demanding an additional equation in interaction models to account for changes of the host plant. Waggoner (1986) and Jeger (1986) have dealt with analytical models to describe the dynamics of interacting host and disease. Hau & Meier (1998) included the host dynamics when modelling the progression of different leaf diseases (*U. appendiculatus*, *P. griseola* and *C. lindemuthianum*) on *Phaseolus* beans. The coupling of pest and disease models with crop growth models forms an essential element in understanding the interactions among diseases and between diseases and host plants, especially with respect to the combined yield losses caused by several diseases (Boote et al., 1983; Rouse, 1988; Béasse et al., 2000).

Interactions of diseases on yield or yield loss

Compared to the quantification of interactions on the epidemic level, more information is available on the combined effect of multiple attacks on yield loss. A good example is the study of Johnson et al. (1986, 1987), in which the yield reduction in potato caused by early blight (*A. solani*), Verticillium wilt (*Verticillium dahliae*) and potato leafhopper (*Empoasca fabae*) was investigated. The principal conclusion from the field studies was that combined infestations resulted in yield and foliage

losses that were less than the sum of losses from solitary infestations of each organism. In another study, Savary & Zadoks (1992a, b, c) analysed the crop losses of groundnut due to a combined attack of rust (*P. arachidis*) and late leaf spot (*Cercosporidium personatum*). The overall result indicated that the effects of the two diseases on damage were less than additive. Similarly, injuries caused by rice pathogens, insects and weeds were less than additive in their yield-reducing effects (Savary et al., 2000).

The effects of interacting pathogens on yield loss have been modelled using multiple regression equations (Savary & Zakoks, 1992a; b), analysis of variance of factorial designs (Johnson et al., 1986; 1987), discriminant analysis (Francl et al., 1987), principal component analysis (Savary et al., 2000) or correspondence analysis (Savary & Zadoks, 1992c; Savary et al., 2000).

Empirical yield-loss equations for a complex of diseases are often obtained via multiple regression analyses with yield or yield loss as the dependent variable. Disease parameters, used directly or after a transformation, serve as independent variables. For two diseases, the general equation for the total yield loss (*YL*) predicted is given as:

$$YL = b_1 f_1(y_1) + b_2 f_2(y_2) + b_{12} \, g(y_1 y_2)$$

The two disease parameters, y_1 and y_2, could be disease severities which can be transformed with the functions f_1 and f_2. The coefficients b_1 and b_2 are the respective damage coefficients for the transformed values of y_1 and y_2. The third term is the interaction term of the model depending on the function g of both disease severities. If the coefficient b_{12} is equal 0, the diseases reduce yield independent from each other. For $b_{12} > 0$ the total yield loss is higher than the sum of the individual yield losses, for $b_{12} < 0$ the effects of y_1 and y_2 are less than additive.

If the disease parameters represent disease incidence or disease severity at one time in the season, this equation reflects a so-called "single point" or "critical point model". The disease parameters could also be the areas under disease progress curves (AUDPC). A special case of this equation would be the well-known linear regression equation with an interaction term involving the multiplication of the disease parameters:

$$YL = b_1 y_1 + b_2 y_2 + b_{12} \, y_1 y_2$$

For the application of this model as a critical point model, the disease severities of both diseases at a certain point in time of the season must be known. These disease levels are a result of the dynamics of epidemics of the two interacting diseases. Thus the understanding of interactions of diseases with respect to their dynamics is a prerequisite to understand their combined effect on yield loss.

For some diseases it has been shown that yield or yield loss are not related to disease parameters, like

the disease severity in a critical stage or the area under disease progress curve, for instance for angular leaf spot (*P. griseola*) on *Phaseolus* beans (Bergamin Filho et al., 1997). Therefore, it can be expected that an equation involving an interaction term by multiplying disease severities may also not be useful in predicting yield loss. Disease severity is a relative measure, which doesn't contain information on the total amount of leaf area. The latter, however, or other host parameters like the area under leaf area progress curve (AULAPC), may be strongly related to yield (Waggoner & Berger, 1987). In such a case, useful predictions of yield loss can only be achieved by realistic estimations of the available leaf area. The dynamics of the leaf area is influenced by the disease progress, which in turn is affected by the leaf area available for infection. Thus without understanding the interactions between host dynamics and progression of epidemics, no appropriate yield loss prediction can be achieved. Consequently, crop growth simulation models, coupled with disease models have been applied to estimate crop losses (e.g. Johnson, 1992; Batchelor et al., 1993; Pinnschmidt & Teng, 1994; Pinnschmidt, 1997) by identifying coupling points as described by Boote et al. (1983). Teng & Johnson (1988) pointed out that "crop-pest models may be the only realistic way to understand or predict the effects of multiple pests on yield", but the application of this approach is rather limited still today.

Zhang et al. (2006) analyzed the relationship between winter wheat cultivar susceptibility to four main fungal diseases (*Septoria tritici* blotch, brown rust, yellow rust and powdery mildew), multiple disease systems, and yield loss (YL) levels. According to authors five potential disease profiles (PDP) were obtained. For all five PDP, cultivar susceptibility profiles (CP) 1 and CP3 (susceptible to *Septoria tritici* blotch and brown rust) consistently made a major contribution to YL, whereas CP8 (most resistant to diseases) consistently contributed little to YL. The impact of CP5 (high susceptibility to *Septoria tritici* blotch and medium to high susceptibility to yellow rust) on YL is higher among the cultivar profiles for PDP5 (with the occurrence of yellow rust and *Septoria tritici* blotch) and also for PDP3 and PDP4 (no yellow rust but high intensity of Septoria tritici blotch), but is comparatively lower in the case of PDP1 and PDP2 (no yellow rust and nil to medium intensity of *Septoria tritici* blotch). Authors concluded that these results could be used to improve the disease module of an agronomic model for wheat aimed at designing "cultivar-crop management" combinations for a given environment and cost/price ratio.

In a previous review (Paula Júnior et al. 2010), it was discussed some aspects related to the epidemiology of interactions among diseases and concluded that there are only few studies that emphasize epidemiological aspects and they are not merely descriptive. We also assumed that although studies involving interactions between diseases still remain quite new, it is obvious for many pathosystems that significant progress in recommending the implementation of appropriate strategies of disease management can only be achieved by careful consideration of all implications related to the interactions.

FINAL REMARKS

It is evident that the interactions of different diseases occurring on one host are a very complex phenomenon. Although effects of some disease combinations on specific hosts, like Septoria disease and mildew on wheat, are known and intensively studied, effects of interactions in general cannot be anticipated. Nevertheless, it has been reported that combinations involving obligate pathogens and viruses frequently result in antagonism (Blumer et al., 1955; Wilson, 1958; Latch & Potter, 1977; Potter, 1982; Erasmus & Von Wechmar, 1983; Omar et al., 1986; Conti et al., 1990; Zaiter et al., 1990; Kutzner et al., 1993; Marte et al., 1993; Dalla Pria, 1994; Bassanezi et al., 1998). Conversely, combinations involving non-obligate pathogens and viruses often result in synergism (Hedges, 1944; 1946a; b; Panzer & Nickeson, 1959; Crane & Calpouzos, 1969; Beute, 1973; Beniwal & Gudauskas, 1974; Adlakha & Raychaudhuri, 1975; Stevens & Gudauskas, 1982; 1983; Omar et al., 1986). For most combinations that may occur in the nature, results will depend on several factors. Methods of experimentation and assessment are important factors in interpreting results in interactions studies (Hyde, 1981; Sikora & Carter, 1987), since sites and timing of inoculations, level of infection, host age, and many other host and pathogen characters may affect the disease outcome (Hyde, 1981).

Disease development under glasshouse conditions where environmental conditions may differ from those prevalent in the field can produce artificial or forced interactions. Normally the inoculum concentration, temperature, nutrition, relative humidity, wetness and others variables used in glasshouse experiments may not occur in the field or, if they occur, other interactions could make the interpretation of the data difficult. This review emphasizes the necessity of studies at field conditions to understand possible interactions.

Interaction between foliar diseases is a subject that should be not neglected in breeding programs, especially in tropical regions. Investigations should include inoculations of mixtures of inoculum from different pathogens and incorporation of genes that confer resistance to multiple pathogens.

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