CHAPTER 14

DISEASES CAUSED BY SOIL-BORNE PATHOGENS

P. LUCAS

14.1 INTRODUCTION

The soil is a favourable habitat for microorganisms and is inhabited by a wide range of bacteria, fungi, algae, viruses and protozoa. Soils contain large numbers of microorganisms – usually between one and ten million per gram of soil – with bacteria and fungi the most prevalent. Some microorganisms present in soil are also able to infect plants. These so-called soil-borne plant pathogens may complete their life cycle in the soil, or may spend part of it on the aerial parts of the plant (Bruehl, 1987).

Plant roots take up the mineral nutrients and water essential for plant growth, but they also release a wide range of organic compounds into the surrounding soil. Thus, the area of soil in contact with the plant root, the rhizosphere, is a site of intense microbial activity. Not surprisingly, many microorganisms are more frequent on the surface of plant roots and in the rhizosphere than in the bulk soil not influenced by the presence of roots. The rhizosphere is therefore a key soil habitat, in which numerous interactions occur between plant roots and soil microorganisms. These interactions determine growth conditions for both the plant and the microorganisms in the rhizosphere.

Soil-borne pathogens require a susceptible plant for the development of their parasitic phase, but they may persist in the soil as saprophytes on residues, or as resistant, dormant forms, from several weeks to several years, depending on their biology. Both parasitic and saprophytic phases may be affected by the physico-chemical and biological characteristics of the soil. Soil-borne pathogens generally affect the root system of plants or the base of the stem (foot), in some cases developing on upper parts of the plant through aerial dispersal from soil inoculum or via transport and/or growth in the vessels, leading to vascular diseases.

Such pathogens may cause extensive damage to crops by limiting water and nutrient uptake (root necrosis) and/or transfer towards the upper parts of the plant (vascular disease), or by reducing the quality of crop products developing underground (root or tuber rot, gall, proliferation, etc.). This damage has led to the focusing of considerable effort on improving our understanding of the biology and ecology of these diseases, with the aim of developing control methods.

This chapter, after having identified specific characteristics of soil-borne pathogens, will provide an overview of the research on these pathogens carried out to date. It will then deal with recent advances in epidemiology, bearing in mind that although soil microbiology has been an area of intense research, epidemiological studies have been developed to a lesser extent for soil-borne than for foliar diseases (McDonald, 1994).

B. M. Cooke, D. Gareth Jones and B. Kaye (eds.), The Epidemiology of Plant Diseases, 2nd edition, 373–386.

14.2 THE SOIL-BORNE DISEASE EPIDEMIC

Soil-borne plant pathogens affect crops throughout the world and have been extensively studied. Research efforts have been justified by the economic impact of these diseases on crop production and by specific difficulties associated with studying and controlling soil-borne diseases, mostly due to the complex environment in which these diseases occur (Lucas and Sarniguet, 1998).

14.2.1 A closed environment limiting dispersal

Soil is a closed environment in which propagules capable of initiating epidemics (e.g. spores, sclerotia, mycelia and hyphae) cannot disperse over long distances, with the exception of certain spores or bacteria transported in run-off water or in soil flowing within the soil matrix. It is rare for horizontal dispersal to extend beyond the field margins.

For some soil-borne pathogens, infection is also transmitted by the growth of the pathogen on or through the soil, from a source of inoculum to a susceptible host. This situation mostly applies to fungi, which form mycelia capable of growing through a heterogeneous medium of pores, cracks and aggregates, although this growth is affected by many other physical, biological and chemical factors (Otten and Gilligan, 1998).

Thus, during the crop cycle, soil-borne pathogen propagules are naturally dispersed over short distances (from a few centimetres to a few decimetres). For this reason, diseased plants show up as patches within a field at the start of epidemics (e.g. take-all of winter wheat caused by *Gaeumannomyces graminis* var. *tritici*) (Hornby *et al*., 1998; Cook, 2003). For soil-borne pathogens with an aerial phase, disease may extend to the whole field during the cropping season if climatic conditions are favourable for the disease (e.g. spore production and dispersal) as is the case for eyespot on wheat, caused by *Tapesia yallundae*.

Most soil-borne pathogens require oxygen. They are therefore mostly located towards the top of the soil profile and their vertical dispersal depends largely on water infiltration pathways and root progression.

14.2.2 A complex, opaque environment with intense biotic and abiotic interactions

Soil is a complex substance with solid, liquid and gaseous components. The organisation and interconnection of these components depend on soil texture, soil structure and external factors, such as climate. Soil structure, and its effects on the relationship between the liquid and gaseous phases, is a major feature determining microbial survival and development in soil. High soil moisture content generally favours microbial activity, but too much water may result in a high prevalence of water-filled pores, resulting in changes in the concentration of O_2, CO_2 or other gases, with consequences for the aerobic or anaerobic microbial communities of the soil microflora (McDonald, 1994).

The effects of the physicochemical characteristics of the soil on the behaviour of microorganisms have been investigated in detail but are still only imperfectly

understood. One problem with most of these studies is that the effects of single factors (pH, nutrients, oligoelements, etc.) are often assessed *in vitro* on culture media, making it difficult to account for interactions between these factors. Furthermore, measurements of the physico-chemical characteristics of the soil may lead to the calculation of mean values for a soil sample that mask the great variability between the niches in which specific microbial communities live. It is difficult to observe the microbe in its natural habitat whilst evaluating the environment, without causing a disturbance. New methods for the micro-scale measurement of soil characteristics, based on the use of microsensors, are becoming available (Meyer *et al*., 2002), as are new molecular techniques for identifying microorganisms and functions (Anderson and Cairney, 2004), and combinations of the two (Lüdemann *et al*., 2000). These advances should provide us with answers to some of our questions, improving our understanding of what occurs in the niches in which soil-borne pathogens survive before they reach the root cells; this work should also benefit from concepts used in ecology (Griffin, 1985; Reynolds *et al*., 2003).

The biological characteristics of the soil in relation to plant diseases have been extensively investigated over the last 40 years. One of the first major works published was a book on the ecology of soil-borne pathogens by Baker and Snyder (1965). It was later followed by a book on the biology and control of soil-borne plant pathogens edited by Bruehl (1975). More recently, Hornby (1990) published some of the contributions to the Soil-borne Plant Pathogens Section of the 5th International Congress on Plant Pathology. Making use of the diverse, high level of microbial activity in soil has been seen as a potential means of promoting the biological control of plant diseases. Many studies have concentrated on the identification, selection and application of biocontrol agents and few methods are currently available. Nevertheless, efforts are being made to increase the efficacy of candidate disease antagonists. These include genetic engineering to improve antibiotic production and exploring mechanisms that are important for their establishment in the courts or potential courts of infection by pathogens (Cook, 1993). Another, less well-studied approach is to manipulate soil management techniques such that naturally-occurring biological controls are conserved and can be exploited. Lucas and Sarniguet (1998) discuss these two approaches and suggest that managing the environment by stimulating naturally-occurring microorganisms and then enhancing their efficacy (if necessary and economically acceptable) by introducing specific biocontrol agents (into a more receptive environment) would be an effective complementary strategy (see also chapter 11).

14.2.3 An environment under human influence

Crop production is affected by a number of primary factors. Climate (e.g. sunshine and rainfall, conditioning light interception for photosynthesis and water uptake by the plant, and temperature, which drives crop growth and development) is difficult to modify and farmers simply have to deal with it. In contrast, soil (as a nutrient reservoir and matrix providing the root system with an ideal matrix for its development) is subject to a number of different management practices, from tillage

and fertiliser application to sowing or planting. These practices, which are designed to optimise plant growth, also have an impact on soil microbial activity. They therefore have a direct or indirect effect on soil-borne pathogens and may be considered to be a means of managing plant health, in addition to plant growth.

The first objective of soil fertilisation is to satisfy crop nutrition demands. Deficiencies in major and minor nutrients may affect plant physiology, increasing infection levels and exacerbating yield losses caused by the disease. It may also have direct effects on soil-borne pathogens and on the biological and physico-chemical characteristics of the soil. The impact of nitrogen fertilisation on take-all of wheat provides a good illustration of these complex interactions: the application of a source of ammonium reduces take-all in most situations whereas nitrate applications do not have the same effect (Huber *et al*., 1968). The uptake of NH_4^+ by roots decreases the pH of the rhizosphere. Smiley and Cook (1973) suggested that decreasing pH indirectly inhibits take-all by modifying the rhizosphere microflora at pH values between 5 and 7, and directly below pH 5. Smiley (1978a,b) subsequently reported that the application of a source of NH_4^+ increases the proportion of rhizosphere pseudomonads antagonistic to *G. graminis* var. *tritici in vitro* to a greater extent than the application of NO_3^-. Sarniguet *et al.* (1992a) showed, in pot bioassays and studies of fields cropped with take-all-infected winter wheat, that applications of ammonium-based fertiliser made the soil less receptive to take-all than applications of a nitrate or mixed (NH_4NO_3) fertiliser. The mixed fertiliser had an intermediate effect. Sarniguet *et al*. (1992b) demonstrated that the frequency of *in vivo* antagonistic fluorescent pseudomonads was higher in the NH_4^+-treated soil than in the NO_3^--treated soil. This work also demonstrated that the presence of rhizosphere pseudomonads can increase disease severity. These deleterious bacteria were more frequent in the nitrate-treated than in the ammonium-treated soil. Thus, antagonism observed *in situ* results from the overall effect of antagonistic and deleterious microorganisms, and nitrogen fertilisation (the form of nitrogen applied) affect these two biological components of soil receptivity to take-all.

Soil tillage affects soil structure, thereby affecting the behaviour of microorganisms. It also affects the distribution of crop residues in the soil profile. These residues remain in the top layers in no-tillage systems, but are buried by ploughing. This factor is important if successful infection requires the presence of infectious crop residues close to the soil surface, as is the case for eyespot on winter wheat, disseminated by spores carried over short distances by wind and rain drops (Colbach and Meynard, 1995). Soil tillage and other cultivation practices, including sowing, may also disperse inoculum within the field and even between fields. According to Truscott and Gilligan (2001), the observation that transmission distances within existing patches are frequently smaller than the expansion of patches between seasons suggests that there is a high level of mechanical inoculum dispersal during harvest and cultivation.

Disease can only occur if susceptible crop plants are grown. Successful infection from soil inoculum is more likely to occur with high inoculum and plant densities. Disease propagation is then favoured by short distances between plants. However, in the case of strictly soil-borne diseases, dispersal within a crop has been shown to be very limited, and build-up of the disease to epidemic levels requires several

successive susceptible crops, and therefore several years in many cases. Such epidemics are described as polyetic (Zadoks, 1999). Crop rotation is recognised as the best way to keep levels of soil-borne diseases low, although it is not always acceptable to farmers for economic reasons. The mode of action of crop rotation was long thought to involve only the breakdown of pathogen inoculum build-up associated with decay of the inoculum during the cropping of non-host plants. However, different break crops have been shown to have different impacts on disease levels in the following host crop. In a study on the influence of crop rotation on foot and root diseases (take-all, sharp eyespot and eyespot) of wheat, Colbach *et al.* (1994) showed that host crops (wheat and barley) tended to increase the risk of the diseases whereas some non-host crops (alfalfa, peas, sunflower) decreased disease risk and others (maize, and sorghum for sharp eyespot) had an intermediate effect. These findings are consistent with those of Lucas *et al.* (1989), who showed that a soil cropped continuously with maize or cultivated under a wheat-maize rotation was far more conducive to take-all than the same soil cultivated under a wheat-beet rotation. The soil inhibiting disease development most strongly was wheat monoculture, providing evidence for take-all decline, which is known to be due to changes in soil microbial populations. The plant species grown is therefore a significant factor determining the composition of microbial soil communities living in soils and the rhizosphere. This applies not only to pathogens, but also to antagonistic and deleterious microbes. Lemanceau *et al*. (1996) demonstrated that two plant species, flax and tomato, modified in different ways the genetic and phenotypic diversity of the fluorescent pseudomonad community resident in the soil.

The cropping of resistant plants is limited by the fact that, curiously, cultivar selection has produced abundant examples of useful genetic resistance to above-ground but not to below-ground pathogens. Cook *et al.* (1995) suggested that the selection imposed by soil-borne pathogens may favour a different defence strategy which is for the plants to support and respond to populations of rhizosphere micro-organisms antagonistic to their pathogens. Attempts have been made to breed wheat cultivars able to react to hypovirulent strains of *G. graminis* var. *tritici* used as a biocontrol agent against take-all (Lemaire *et al*., 1982). However, more attention has been paid to selecting bacteria displaying a combination of efficient root colonisation and beneficial effects on the activity of a given plant (Kuiper *et al*., 2001) than to breeding plants able to exert beneficial selection pressure on microbial communities, although there are some reports of genotype-specific induction of soil microbial communities inhibiting soil-borne diseases such as rhizoctonia root rot in winter wheat cultivars (Mazzola and Gu, 2002; Mazzola, 2004). This approach is supported by studies in other areas demonstrating that the sensitivity of wheat (Rengel, 1997) and oat (Timonin, 1965) genotypes to manganese deficiency, for example, depends on the number of Mn-reducing microorganisms in the rhizosphere.

14.2.4 Consequences for the epidemiology of soil-borne diseases

The restricted dispersal of inoculum is a characteristic of soil-borne pathogens, accounting for the patchy distribution of diseased plants at the start of epidemics. Depending on whether these soil-borne pathogens have an aerial phase of development in their cycle, this patchiness may be observed only in part of the annual cycle or over several years of cultivation, with patches becoming larger before merging (Fig. 14.1). Truscott and Gilligan (2001) described this dynamic as a two-step process: (i) local amplification due to parasitic activity on the plants initially infected and transmission of the disease to neighbouring plants, (ii) dispersal of inoculum by water, wind or humans.

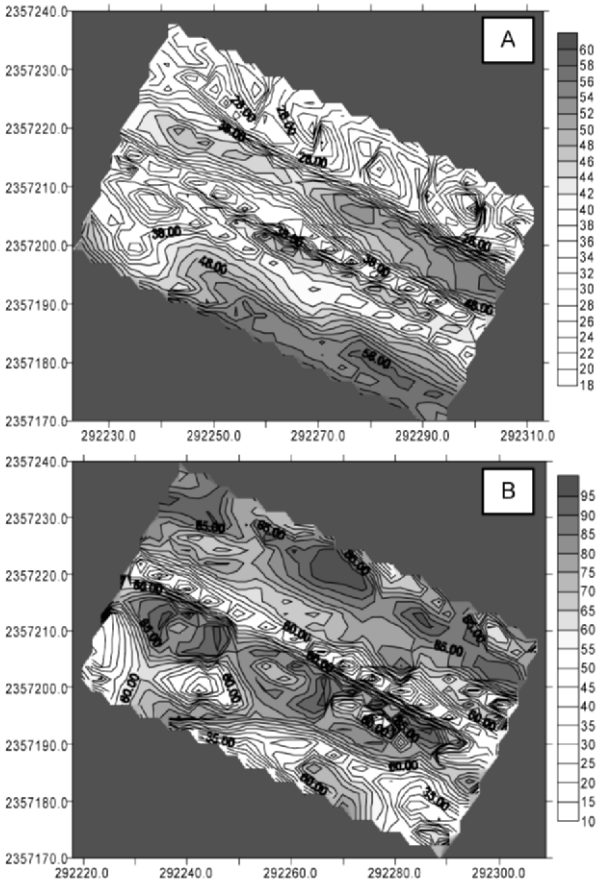

Figure 14.1. Yield (A, 10^{-1}t ha^{-1}) and take-all disease incidence (B, per 100 diseased plants) maps of a 3rd winter wheat field, showing patchiness of the disease (Le Rheu, France, 1999; from Lamkadmi et al.*, 2000).*

Local amplification depends on complex interactions between plants and soil-borne pathogens, regulated by the physicochemical and microbial characteristics of the soil. It is difficult to measure these interactions and characteristics due to soil 'opacity'. One way of relating the epidemiology of a soil-borne disease (take-all of winter wheat) to cultural practices known or thought to have an impact on these interactions and characteristics is described below.

The aim of this work was to propose cropping strategies limiting disease risk. In order to evaluate these strategies based on their ability to decrease epidemics and to increase yield, a dynamic approach to the relationship between the kinetics of the disease and crop growth and development is also presented.

Inoculum dispersal has received less attention, especially that occurring between the harvesting of one crop and the sowing of the subsequent crop. Cultivation leads to the redistribution of inoculum. This may increase the likelihood of invasion, but it may also dilute the inoculum to levels below the threshold required for symptom development in the next crop (Truscott and Gilligan, 2001). The third part of the next section, will illustrate how current or possible management of this intercropping period can affect disease incidence in the next crop.

14.3 MODELLING SOIL-BORNE DISEASE EPIDEMIOLOGY

14.3.1 Relationship between cropping practices and disease dynamics

Much attention has been given to the use of non-linear models to describe the temporal progress of disease (Madden, 1980; Gilligan, 1985). Brasset and Gilligan (1989) compared the use of several non-linear models to describe the increase in the absolute number of diseased roots infected with *G. graminis* var. *tritici* in first and second wheat crops. They concluded that a model incorporating components of primary and secondary infection, together with inoculum decay, described the data in a manner consistent with biological constraints.

Colbach *et al.* (1997a) simplified one of these models and used it to assess the impact of crop management on the primary and secondary infection cycles of take-all epidemics. Origin of inoculum and infection rates are the central elements of this model. Inocula may be found in soils, on plant debris or on the roots of the living plant. Each inoculum is associated with an infection rate. Rate (c_1) corresponds to the capacity of the soil reservoir inoculum to cause infection and disease. The rate of secondary infection (c_2) is a measure of the capacity of infected roots to spread disease to other roots (or from a diseased plant to other plants). The percentage of diseased plants is given by the following equation, where time t is expressed as cumulative degree days (basis 0°C) since sowing:

$$y = \frac{1 - e^{-(c_1+c_2)t}}{1 + \frac{c_2}{c_1} e^{-(c_1+c_2)t}} \qquad (14.1)$$

This equation was first successfully tested ($r^2 = 0.99$) on a plot assessed every two weeks after growth stage 30 (Zadoks *et al.*, 1974). It was then fitted to data concerning take-all build-up for each experimental treatment at three sites (three regions of France), where different cultural practices (sowing date, sowing density, total nitrogen dose, nitrogen fertiliser form, burial or removal of preceding crop residue) were tested. The parameters c_1 and c_2 were estimated for each experimental treatment at each site.

A linear model was tested to interpret c_1 and c_2 for each set of estimates at each site as a function of the factors analysed and co-variables measured. Sowing date always affected c_1 (e.g. primary infections) whereas c_2 (e.g. secondary infections) was influenced by sowing date only at the most favourable sites for disease (e.g. those with the highest infection rates, due to favourable climatic conditions). Early sowing systematically increased c_1. This is consistent with previous results (Hornby *et al.,* 1990) and the fact that early sowing provides a longer period for infection before winter. The effect of early sowing on c_2 was variable, positive for one experimental site, negative for another.

A positive correlation was found between plants m^{-2} and parameter c_1, but only at the most favourable sites. Plant density, like sowing date, had a variable effect on c_2. A high plant density at early stages, when the roots are still few in number and short, probably increases the chance of contact between the soil inoculum and living roots, whereas it has a less predictable effect when the root system is well developed.

High levels of nitrogen application increased c_1 and decreased c_2 but both these parameters were decreased by applications of nitrogen in the form of ammonium. As reported by Sarniguet *et al.* (1992a,b), nitrogen can stimulate both the pathogenic and the antagonistic microflora. Increases in the antagonistic microflora early in the infection of seminal roots facilitate the development of fluorescent pseudomonads on necrotic tissue. These pseudomonads then interfere with pathogen expansion, particularly if nitrogen fertiliser is applied in the form of ammonium.

The hierarchy of and interactions between various factors were shown to be important. Factors other than sowing date were generally significant only if sowing date was also significant. Sowing date may therefore be considered the dominant factor, and its interactions with other factors as the most important. This type of interaction is very similar to that observed for site: several factors had a stronger influence or were only significant if the site was favourable for disease development. Each factor seemed to amplify the risk due to the other effects and factors with a weak effect influenced disease only if factors with a strong effect were also present.

This model was used to assess the efficacy of new methods of control, such as the use of fungicidal seed treatments (Fig. 14.2). It was found that, in an early epidemic, the fungicide significantly reduced take-all incidence during all or most of the cropping season whereas, in late epidemics, it decreased incidence only moderately. Seed treatment was shown to reduce incidence by delaying primary infection (Schoeny and Lucas, 1999).

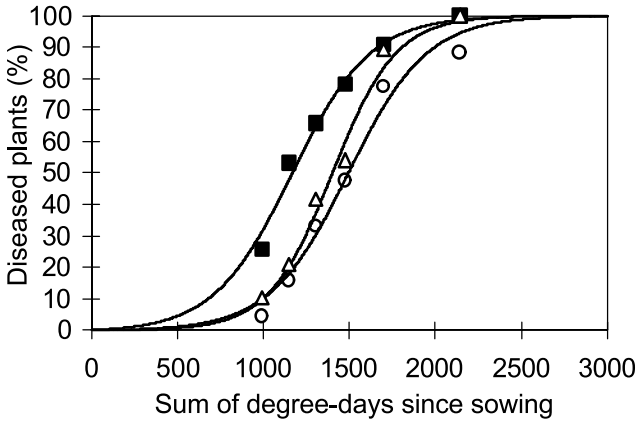

Figure 14.2. Illustration of take-all progress curves for various levels of an experimental seed treatment in a field experiment conducted in 1995, at Le Rheu, France. Symbols represent observed means for each experimental treatment (control ■ and two rates of fungicide Δ, ○). (From Schoeny and Lucas, 1999).

Similar approaches, using a similar type of model, have been applied to other soil-borne diseases, including some with an aerial phase, such as eyespot (caused by *T. yallundae*) and sharp eyespot (caused by *Rhizoctonia cerealis*) on winter wheat (Colbach and Saur, 1998; Colbach *et al.,* 1997b). For both diseases, the impact of the major components of cropping systems (crop rotation, soil tillage, wheat management) was assessed by means of field trials, with several disease assessments during wheat growth. Early sowing consistently increased disease incidence through the primary infection cycle, whatever the disease. The frequencies of eyespot and sharp eyespot were increased by high plant density and/or small numbers of shoots per plant during the primary infection cycle. In contrast, in the secondary infection cycle, the frequency of these diseases was decreased by small shoot number per plant, that reduced late disease development at high density. For both diseases, high doses of nitrogen increased disease levels through both infection cycles. However, nitrogen fertiliser in the form of ammonium (*vs.* ammonium nitrate, i.e. 'mixed' fertiliser) decreased eyespot levels as it does for take-all, but had the opposite effect on sharp eyespot.

For eyespot, the model was also used to analyse the influence of crop residue distribution on disease development and infection cycles (Colbach and Meynard, 1995). Differences in the amount and placement of crop residues were achieved by varying crop rotation and soil tillage before the assessed winter wheat crop. When the previous crop was a host crop preceded by a non-host crop, soil inversion resulted in the burial of host residues, thereby decreasing primary infection risk. However, if the previous crop was a non-host crop preceded by a host crop, soil inversion carried the host residues back to soil surface, thereby increasing primary infection risk. Secondary infection was not correlated with crop succession or soil tillage.

14.3.2 Relationship between disease dynamics and yield components

Disease-yield loss relationships (see also Chapter 2) must be determined to assess the agronomic efficacy and economic benefits of control methods. Few studies have focused on the relationships between soil-borne disease progress curves and crop response, for three main reasons. First, studies on the development of soil-borne disease epidemics must include a large number of representative samplings, because of the patchy distribution of most of these diseases. Second, disease assessment for soil-borne pathogens involves destructive sampling, which leads to discontinuities in the dynamic representations of both disease and crop growth. Third, it is not always possible to compare, in equivalent conditions, a healthy situation with various levels of disease in the crop.

The question of yield losses due to take-all has been addressed by comparing situations involving different crop rotations (Slope and Etheridge, 1971), sowing dates (Bateman *et al*., 1990), or artificial inoculations with different amounts of fungal inoculum (Rothrock, 1988) or with the same amount of inoculum incorporated at different depths (Hornby and Bateman, 1990), in order to generate differences in epidemic patterns. These approaches have generally focused on total yield at harvest, but rarely, the various yield components formed successively during the wheat cropping season (Meynard and Sebillotte, 1994) have been investigated: ear number per square meter (sowing to mid-stem elongation), grain number per ear (floral initiation to flowering), grain number per square meter (sowing to flowering), and 1,000 grain weight (flowering to maturity). The impact of the disease is likely to depend on when disease occurs and, consequently, on the nature of the yield components affected. However, most studies have simply established correlations between damage (i.e. yield reduction) and disease level at flowering (Bateman *et al.*, 1990), grain filling (Slope and Etheridge, 1971; Hornby and Bateman, 1990), or harvest (McNish and Dodman, 1973), and have taken no account of the link between disease dynamics and crop growth dynamics.

Schoeny and Lucas (1999) carried out a series of experiments in which a fungicidal seed treatment was used to generate different disease incidence and severity progress curves at a single location, with identical cultural practices and climatic conditions. Schoeny *et al.* (2001) then investigated the effects of various take-all epidemics on yield formation as a function of disease progression. Simple linear regression models involving various disease variables were compared and their ability to account for and predict the losses of yield components was assessed. Yield losses at harvest were strongly linked to the area under the disease progress curve (AUDPC) for disease incidence calculated between sowing and flowering (Fig. 14.3). The observed losses were larger for plots to which low rates of fertiliser were applied than for plots to which high rates of fertiliser were applied. Losses in terms of ear number per square meter, grain number per ear, and grain number per square meter were mainly related to cumulative disease incidence, calculated as AUDPC, during periods corresponding to yield component formation (sowing to mid-stem elongation, floral initiation to flowering, and sowing to flowering, respectively). In contrast, 1,000 grain weight losses were linked to disease incidence at mid-stem elongation (i.e. at a growth stage before the formation of this yield

component, grain filling). This relationship is particularly interesting because of its predictive nature. It can be interpreted as an early effect of take-all on nitrogen and carbon assimilates, limiting re-mobilisation from stems and leaves during grain filling.

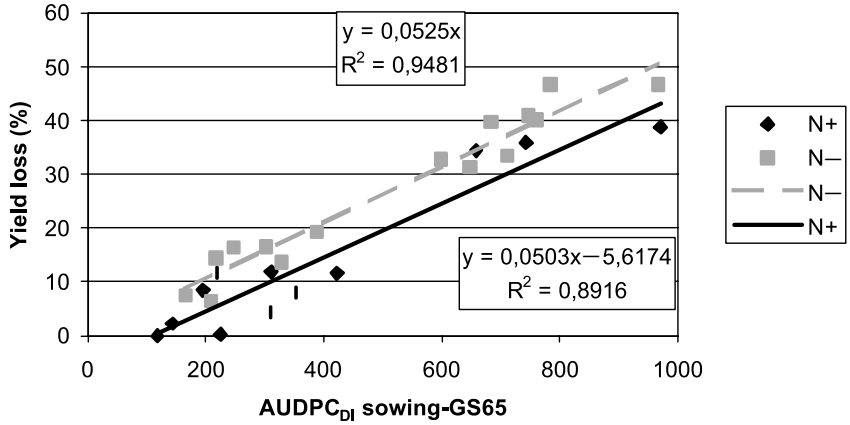

Figure 14.3. Disease-yield loss relationships established from field experiments on winter wheat crops infected with take-all. Yield loss function of cumulative disease incidence between sowing and flowering is established for low (N−) and high (N+) levels of nitrogen fertilisation. (From Schoeny et al.*, 2001).*

This was confirmed in a subsequent study showing that although wheat plants with severe take-all infection took up more nitrogen per unit of efficient root than uninfected plants, this compensatory response was insufficient to give nitrogen accumulation levels equivalent to those in healthy plants (Schoeny *et al.,* 2003). Thus, split applications of nitrogen with the amount of nitrogen adapted to the lower capacity of infected root systems to absorb nitrogen, as proposed by Lucas *et al.* (1997), might be of value.

14.3.3 Importance of cultivation management between crops

The role played by the intercrop period has not received adequate attention from plant pathologists in the analysis of soil-borne pathogen epidemics. The severity of rhizoctonia root rot on wheat has been linked to the presence of volunteers and weeds growing in the field between harvest and planting of the subsequent crop. These plants act as a 'green bridge', maintaining or increasing the potential inoculum of many plant pathogens, such as *R. solani* AG-8 in particular (Smiley *et al.,* 1992). Dulout *et al*. (1997) compared the effects of wheat volunteers, blackgrass (*Alopecurus myosuroides*) and bare soil on soil infectivity and soil conduciveness to take-all. They showed that both wheat volunteers and blackgrass maintained soil infectivity at a higher level than bare soil. Soil conduciveness was reduced by wheat volunteers whereas bare soil and blackgrass were highly

conducive to the disease. Intermediate cropping (e.g. growing of a crop between harvest of the previous cash crop and sowing of the subsequent crop), already recommended for limiting soil erosion and nitrate leaching, should therefore also be considered as a way of improving soil health (Ennaïfar *et al.,* 2005).

14.4 CONCLUSION

Soil-borne pathogens are difficult to control with pesticides because it is difficult to target the niches in which they are found without treating the whole soil profile. Soil disinfection is no longer acceptable, for environmental reasons, and never was acceptable for some crops for economic reasons. Most alternative methods are only partly effective at controlling these diseases, but may act on different phases of the disease development cycle. As soil-borne diseases often develop more slowly than air-borne diseases, this provides an opportunity to time actions throughout the crop cycle, and even between crops, which may be useful given the polyetic characteristic of these epidemics.

This approach requires accurate description of the processes underlying disease epidemics, damage and resulting yield losses, comprehensive organisation of these processes in time and space, identification of the determinants affecting these processes and the identification of possible ways to control epidemics and minimise yield losses. Epidemiology and modelling are central to this kind of approach, (i) describing the behaviour, dynamics and damage of soil-borne pathogens at a range of ecological scales and (ii) making this information available to farmers and advisors to enable them to implement integrated crop protection strategies.

REFERENCES

Anderson, I.C. and Cairney, J.W.G. (2004) Diversity and ecology of soil fungal communities: increased understanding through the application of molecular techniques. *Environmental Microbiology,* **6**(8), 769-779.

Baker, R.R. and Snyder, W.C. (1965) *Ecology of soil-borne plant pathogens – Prelude to biological control*. University of California Press, Berkeley, Los Angeles. 571 pp.

Bateman, G.L., Hornby, D. and Gutteridge, R.J. (1990) Effects of take-all on some aspects of grain quality of winter wheat. *Aspects of Applied Biology,* **25**, 339-348.

Brasset, P.R. and Gilligan, C.A. (1989) Fitting of single models for field disease progress data for the take-all fungus. *Plant Pathology,* **38**, 397-407.

Bruehl, G.W. (1975) *Biology and control of soil-borne plant pathogens*. The American Phytopathological Society, St Paul, Minnesota. 216 pp.

Bruehl, G.W. (1987) *Soilborne plant pathogens*, Macmillan Publishing Company, New York.

Colbach, N., Lucas, P. and Cavelier, N. (1994) Influence des successions culturales sur les maladies du pied et des racines du blé d'hiver. *Agronomie,* **14**, 525-540.

Colbach, N. and Meynard, J.M. (1995) Soil tillage and eyespot: influence of crop residue distribution on disease development and infection cycles. *European Journal of Plant Pathology,* **101**, 601-611.

Colbach, N., Lucas P. and Meynard J.M. (1997a) Influence of crop management on take-all development and disease cycles on winter wheat. *Phytopathology,* **87**, 26-32.

Colbach, N., Lucas, P., Cavelier, N. *et al.* (1997b) Influence of cropping system on sharp eyespot in winter wheat. *Crop Protection,* **16**, 415-422.

Colbach, N. and Saur, L. (1998) Influence of crop management on eyespot development and infection cycles of winter wheat. *European Journal of Plant Pathology,* **104**, 37-48.

Cook, R.J. (1993) Making greater use of introduced microorganisms for biological control of plant pathogens. *Annual Review of Phytopathology,* **31**, 53-80.

Cook, R.J., Tomashow, L.S., Weller, D.W. *et al*. (1995) Molecular mechanisms of defense by rhizobacteria against root disease. *Proceedings of the National Academy of Sciences*, **92**, 4197-4201.

Cook, R.J. (2003) Take-all of wheat. *Physiological and Molecular Plant Pathology,* **62**, 73-86.

Dulout, A., Lucas, P., Sarniguet, A. *et al*. (1997) Effects of wheat volunteers and blackgrass in set-aside following a winter wheat crop on soil infectivity and soil conduciveness to take-all. *Plant and Soil,* **197**, 149-155.

Ennaïfar, S., Lucas, P., Meynard, J-M. *et al.* (2005) Effects of summer fallow management on take-all of winter wheat caused by *Gaeumannomyces graminis* var. *tritici*. *European Journal of Plant Pathology,* **112**, 167-181.

Gilligan, C.A. (1985) Construction of temporal models: III. Disease progress of soil-borne pathogens, in *Advances in Plant Pathology, Volume 3, Mathematical Modelling of Crop Disease*, (ed. C.A. Gilligan), Academic Press, London, pp. 67-105.

Griffin, D.M. (1985) Soil as an environment for the growth of root pathogens, in *Ecology and Management of soilborne plant pathogens* (eds C.A. Parker, A.D. Rovira, K.J. Moore, P.T.W. Wong and J.F. Kollmorgen), American Phytopathological Society, St Paul, Minnesota, pp. 187-190.

Hornby, D. (1990) *Biological Control of Soil-borne Plant Pathogens*. C.A.B. International, Wallingford, 479 pp.

Hornby, D. and Bateman, G.L. (1990) Artificial infestation of soil with *Gaeumannomyces graminis* var. *tritici* to study the relationship between take-all and wheat yields in field experiments. *Soil Use Management,* **6**, 209-217.

Hornby, D., Bateman, G.L., Gutteridge, R.J. *et al*. (1990) Experiments in England and France on fertilisers, fungicides and agronomic practices to decrease take-all. *Brighton Crop Protection Conference – Pests and Diseases* 1990, **2**, 771-776.

Hornby, D., Bateman, G.L., Gutteridge, R.J., *et al.* (1998) *Take-all Disease of Cereals: A Regional Perspective*. CAB International, London, 400 pp.

Huber, D.M., Painter, C.C., McKay, H.C., *et al.* (1968) Effect of nitrogen fertilization on take-all of winter wheat. *Phytopathology,* **58**, 1470-1472.

Kuiper, I., Bloemberg, G.V. and Lugtenberg, B.J. (2001) Selection of a plant-bacterium pair as a novel tool for rhizostimulation of polycyclic aromatic hydrocarbon-degrading bacteria. *Molecular Plant Microbe Interactions*, **14**, 1197-1205.

Lamkadmi, Z., Fouad, Y., Nicolas, H. *et al.* (2000) Potentialités de la télédétection rapprochée, dans les domaines optique et thermique pour la cartographie d'attaques parasitaires sur culture, in *Agriculture de précision: avancées de la recherche technologique et industrielle. Actes du colloque CEMAGREF – ENESAD, Dijon, 29 et 30 mai 2000,* Educagri Editions, pp. 223-234.

Lemaire J.M., Doussinault G., Lucas P., *et al.* (1982) Possibilités de sélection pour l'aptitude à la prémunition dans le cas du piétin échaudage des céréales, *Gaeumannomyces graminis*. *Cryptogamie Mycologie,* **3**, 347-359.

Lemanceau, P., Corberand, T., Laguerre, G. *et al.* (1996) The composition of fluorescent pseudomonad populations associated with roots is influenced by plant and soil type. *Applied and Environmental Microbiology,* **62**, 2449-2456.

Lucas, P., Sarniguet, A., Collet, A. *et al.* (1989) Réceptivité du sol au piétin-échaudage (*Gaeumannomyces graminis* var. *tritici*): influence de certaines techniques culturales. *Soil Biology and Biochemistry,* **21**, 1073-1078.

Lucas, P., Jeuffroy, M.-H., Schoeny, A. *et al*. (1997) Basis for nitrogen fertilisation management of winter wheat crops infected with take-all. *Aspects of Applied Biology,* **50**, 255-262.

Lucas, P. and Sarniguet, A., (1998) Chapter 9: Biological control of soil-borne pathogens with resident versus introduced antagonists: Should diverging approaches become strategic convergence? in *Conservation Biological Control,* (ed. P. Barbosa), Academic Press New-York, pp. 351-370.

Lüdemann, H., Arth, I. and Liesack, W. (2000) Spatial changes in the bacterial community structure along a vertical oxygen gradient in flooded paddy soil cores. *Applied and Environmental Microbiology,* **66**, 754-762.

MacDonald J.D. (1994) The Soil Environment, in *Epidemiology and Management of Root Diseases*, (eds C. Lee Campbell and D. Michael Benson), Springer-Verlag Berlin Heidelberg pp. 82-116.

MacNish, G.C., and Dodman, R.L. (1973) Relation between incidence of *Gaeumannomyces graminis* var. *tritici* and grain yield. *Australian Journal of Biological Sciences,* **26**, 1289-1299.

Madden, L.V. (1980) Quantification of disease progression. *Protection Ecology,* **2**, 159-176.
Mazzola, M. and Gu, Y.-H. (2002) Wheat genotype-specific induction of soil microbial communities suppressive to to *Rhizoctonia solani* AG-5 and AG-8. *Phytopathology,* **92**, 1300-1307.
Mazzola, M. (2004) Assessment and management of soil microbial community structure for disease suppression. *Annual Review of Phytopathology,* **42**, 35-59.
Meyer, R.L., Kjaer, T. and Revsbech, N.P. (2002) Nitrification and denitrification near a soil-manure interface studied with a nitrate-nitrite biosensor. *Soil Science Society of America Journal,* **66**, 498-506.
Meynard, J.-M. and Sebillotte, M. (1994) L'élaboration du rendement du blé, base pour l'étude des autres céréales à talles, in *Un point sur… Elaboration du rendement des principales cultures annuelles*, (eds L. Combe and D. Picard). INRA Editions, Paris, pp. 31-51.
Otten, W. and Gilligan, C.A. (1998) Effect of physical conditions on the spatial and temporal dynamics of the soil-borne fungal pathogen *Rhizoctonia solani*. *New Phytologist,* **138**, 629-637.
Rengel, Z. (1997) Root exudation and microflora populations in the rhizosphaere of crop genotypes differing in tolerance to micronutrient deficiency. *Plant and Soil,* **196**, 255-260.
Reynolds, H.L., Packer, A., Bever, J.D., *et al*. (2003) Grassroots ecology: Plant-microbe-soil interactions as drivers of plant community structure and dynamics. *Ecology,* **84**, 2281-2291.
Rothrock, C.S. (1988). Relative susceptibility of small grains to take-all. *Plant Disease,* **72**, 883-886.
Sarniguet, A., Lucas, P. and Lucas, M. (1992a) Relationships between take-all, soil conduciveness to the disease, populations of fluorescent pseudomonads and nitrogen fertilizers. *Plant and Soil,* **145**, 17-27.
Sarniguet, A., Lucas, P., Lucas, M. *et al.* (1992b) Soil conduciveness to take-all of wheat: Influence of the nitrogen fertilizers on the structure of populations of fluorescent pseudomonads. *Plant and Soil,* **145**, 29-36.
Schoeny, A. and Lucas, P. (1999) Modeling of take-all epidemics to evaluate the efficacy of a new seed-treatment fungicide on wheat. *Phytopathology,* **89**, 954-961.
Schoeny, A, Jeuffroy, M.H. and Lucas, P. (2001) Influence of take-all epidemics on winter wheat yield formation and yield loss. *Phytopathology,* **91**, 694-701.
Schoeny, A., Devienne-Barret, F., Jeuffroy, M.H. *et al.* (2003) Effect of take-all root infections on nitrate uptake in winter wheat. *Plant Pathology,* **52**, 52-59.
Slope, D.B. and Etheridge, J. (1971) Grain yield and incidence of take-all (*Ophiobolus graminis* Sacc.) in wheat grown in different crop sequences. *Annals of Applied Biology,* **67**, 13-22.
Smiley, R.W. and Cook, R.J. (1973) Relationships between take-all of wheat and rhizosphere pH in soils fertilized with ammonium vs nitrate nitrogen. *Phytopathology,* **63**, 882-890.
Smiley, R.W. (1978a) Antagonists of *Gaeumannomyces graminis* from the rhizoplane of wheat in soils fertilized with ammonium or nitrate nitrogen. *Soil Biology and Biochemistry,* **10**, 169-174.
Smiley, R.W. (1978b) Colonization of wheat roots by *Gaeumannomyces graminis* inhibited by specific soils, microorganisms and ammonium-nitrogen. *Soil Biology and Biochemistry,* **10**, 175-179.
Smiley, R.W., Ogg, A.G. and Cook, R.J. (1992) Influence of glyphosate on Rhizoctonia root rot, growth, and yield of barley. *Plant Disease*, **76**, 937-942.
Timonin, M.I. (1965) Interaction of higher plants and soil microorganisms, in *Microbiology and Soil Fertility*, (eds C.M. Gilmore and O.N. Allen). Oregon State University Press, Corvallis, pp. 135-138.
Truscott, J.E. and Gilligan, C.A. (2001) The effect of cultivation on the size, shape, and persistence of disease patches in fields. *Proceedings of the National Academy of Sciences,* **98**, 7128-7133.
Zadoks, J.C., Chang, T.T. and Konzak, C.F. (1974) A decimal code for the growth stages of cereals. *Weed Research*, **14**, 415-421.
Zadoks, J.C. (1999) Reflections on space, time, and diversity. *Annual Review of Phytopathology,* **37**, 1-17.