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Size, shape and intensity of aggregation of take-all disease during natural epidemics in second wheat crops

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Point pattern analysis (fitting of the beta-binomial distribution and binary form of power law) was used to describe the spatial pattern of natural take-all epidemics (caused by *Gaeumannomyces graminis* var. *tritici*) on a second consecutive crop of winter wheat in plots under different cropping practices that could have an impact on the quantity and spatial distribution of primary inoculum, and on the spread of the disease. The spatial pattern of take-all was aggregated in 48% of the datasets when disease incidence was assessed at the plant level and in 83% when it was assessed at the root level. Clusters of diseased roots were in general less than 1 m in diameter for crown roots and 1–1·5 m for seminal roots; when present, clusters of diseased plants were 2–2·5 m in diameter. Anisotropy of the spatial pattern was detected and could be linked to soil cultivation. Clusters did not increase in size over the cropping season, but increased spatial heterogeneity of the disease level was observed, corresponding to local disease amplification within clusters. The relative influences of autonomous spread and inoculum dispersal on the size and shape of clusters are discussed.

Keywords: *Gaeumannomyces graminis* var. *tritici*, soilborne pathogen, spatial pattern, *Triticum aestivum*

Introduction

The analysis of plant disease spatial pattern provides insight into the spatial characteristics of epidemics and allows biological and environmental hypotheses to be proposed to account for the associations among pathogen propagules or diseased plants (Campbell & Madden, 1990). Spatial analysis of plant diseases has been used to describe spatial pattern (Chellemi *et al.*, 1988), improve sampling strategy (Turechek & Mahaffee, 2004), suggest hypotheses concerning the underlying biological processes responsible for disease spread (Larkin *et al.*, 1995; Rekah *et al.*, 1999; Roumagnac *et al.*, 2004) or propose improvements in control methods (Pethybridge *et al.*, 2005). Spatial patterns of different types of plant pathogens have been analysed: phytoplasmas (Madden *et al.*, 1995), viruses (Pethybridge & Turechek, 2003), bacteria (Roumagnac *et al.*, 2004), fungi (Savary *et al.*, 2001), parasitic plants (Aukema, 2004) and nematodes (Gavassoni *et al.*, 2001). A wide range of dispersal mechanisms have been encompassed in such analyses: vector spread (Dallot *et al.*, 2003), splash dispersal (Reynolds *et al.*, 1988;), wind dispersal (Pethybridge *et al.*, 2005) or dispersal within soil (Xiao *et al.*, 1997). For soilborne diseases, spatial pattern is typically found to be aggregated, with a spatial dependence ranging from one to several dozen metres [e.g. 1–4 m (Rekah *et al.*, 1999), 2 m (Chellemi *et al.*, 1988), 3 m (Savary *et al.*, 2001), >15 m (Larkin *et al.*, 1995), 23 m (Gavassoni *et al.*, 2001) or 70 m (Oliver *et al.*, 2003)].

Take-all disease of wheat, caused by *Gaeumannomyces graminis* var. *tritici* (Ggt) is a common disease of wheat worldwide (Cook, 2003). Most of the epidemiological studies to date have considered means over entire plots (e.g. Schoeny *et al.*, 2001), thus ignoring the spatial heterogeneity of the disease. However, take-all is known to occur in patches (Clarkson & Polley, 1981), but few studies have attempted to characterize the spatial pattern of take-all. The earliest studies (MacNish & Dodman, 1973; Cotterill & Sivasithamparam, 1989) dealt with inoculum survival and/or spatial pattern; both concluded that the disease was aggregated, but gave no estimate of patch size. More recently, a large-scale analysis of take-all index showed a spatial dependence over a range of 70 m, possibly linked to soil properties (Oliver *et al.*, 2003), but the large extent of the study (a 6-ha plot) came at the expense of resolution (the samples were taken at the vertices of a 24-m grid). A better knowledge of the spatial pattern of the disease at a finer scale would aid understanding of the mechanisms by which the disease spreads.

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The aim of the present study was to characterize the spatial structure of take-all at a fine scale (5 × 5 m with a 0·5-m grid), in order to (i) determine if the disease is aggregated and, if so, (ii) estimate the characteristic size of patches, (iii) study the relationship between disease incidence and aggregation and (iv) investigate the effect of cultivation practices on the size and shape of disease aggregation. The analysis was based on data collected in second wheat field plots during 2 years.

Materials and methods

Natural epidemics of take-all on second wheat crops were monitored over one cropping season at the INRA experimental station near Rennes (48°01′N, 1°43′W) in western France. The experiment was carried out in 2003 and then repeated in a different field in 2004. Both fields had a silty soil (15% loam, 65–70% silt and 15–20% sand).

Experimental treatments

Second winter wheat crops (second consecutive year of wheat culture) were sown with cv. Caphorn at a density of 300 seeds m^{-2} on 29 October 2002 and 13 October 2003. The seeds were treated with silthiofam (Latitude®) at 25 g a.i. per 100 kg seed because an early assessment of the disease at the beginning of 2003 showed that in the untreated plots disease incidence was already nearing 100%, which would have precluded any spatial analysis. Nitrogen fertilizer was applied twice, as ammonium solution (34% N) at a total rate of 120 kg ha^{-1}. Weeds, foliar and stem diseases and aphid populations were maintained below economic threshold levels, as recommended (Anonymous, 2003).

In order to generate different initial disease levels in the same field, three different management practices were performed during the fallow period between the harvest of the first wheat crop and the sowing of the second (studied) wheat crop (Ennaïfar *et al.*, 2005): host (wheat) or non-host (mustard) cover crops were sown, or the soil was kept bare. These treatments were designated W (wheat), M (mustard) and BS (bare soil). After a stubble break and a crosskill roller passage to firm the land at depth, the cover crops were direct-sown after harvesting of the first wheat and ploughed into the soil 77 and 46 days after sowing in 2003 and 2004, respectively. Bare-soil plots were maintained by glyphosate (Roundup; 5·5 L ha^{-1}; Monsanto Agriculture SAS) application to control weeds. In order to test the effect of cultural practices on the dynamics and spatial structure of the disease, two cultivation methods were used when sowing the second wheat: ploughing and circular spike-harrowing followed by line sowing with a shoe drill (thereafter referred to as P or ploughed plots) and conservation tillage accompanied by a broadcast sowing with a direct drill (referred to as CT or tilled plots).

Each year, the experimental field consisted of six plots (two soil cultivation methods × three cover crops). A 5- × 5-m observation grid was delimited in each plot and divided into one hundred 50- × 50-cm quadrats. This large number of quadrats enabled the geometry of epidemics to be explored at successive scales (Campbell & Madden, 1990).

Disease assessment

Three assessments were performed each year at the mid-stem elongation, booting and grain-milk stages (Table 1). For each assessment, three plants were randomly chosen in each quadrat and removed from the field with particular care not to trample the surrounding plants. It was decided to sample only three plants per quadrat as a compromise between the number of plants that could be removed from the field without disrupting the epidemic too much and the number of units needed to fit frequency distributions (this is why the smallest quadrat size for spatial analysis of plant disease incidence (see below) was two quadrats, i.e. six plants). The roots were water-washed, the numbers of diseased and healthy roots were counted on each plant and the percentage of the root system showing black stelar discoloration (Clarkson & Polley, 1981) was evaluated for each plant on a 0, 10, 20 ... 100 scale. This allowed observed values to be recorded for three variables: plant disease incidence (ratio of the number of diseased plants to the number of observed plants), root disease incidence (ratio of the number of diseased roots to the total number of roots) and disease severity (proportion of diseased root system). These variables were considered separately for the seminal and crown root systems, combined to obtain results for the whole root system.

Analysis of mean disease level

Plant disease incidence, root disease incidence and severity were transformed using the logit function. Normality of the transformed variables was verified using procedure UNIVARIATE of SAS (SAS Institute). The effects of assessment date, soil cultivation, cover crop and two-way interactions were analysed in each year separately with an analysis of variance using the procedure GLM of SAS; the Student-Newman-Keuls multiple range test was used to compare soil cultivation methods and cover crops.

Table 1 Sampling dates for measurement of wheat take-all (*Gaeumannomyces graminis* var. *tritici*) intensity

Year	Date	Growth stage[a]	Degree-days[b]
2003	8 April	32	1207
	5 May	43	1531
	2 June	75	1914
2004	5 April	33	1254
	4 May	39	1533
	1 June	75	1923

[a]Wheat growth stage according to Zadoks *et al.* (1974).
[b]Sum of growing degree-days (base 0°C) from sowing.

Distribution fitting

The beta-binomial (Hughes & Madden, 1993; Madden & Hughes, 1995) and the binomial distribution were fitted to the data (root or plant disease incidence) using the computer program BBD (Madden & Hughes, 1994). The binomial distribution has a single parameter π representing the probability for each plant or plant part to be diseased, independently of other plants or plant parts. The beta-binomial distribution has two parameters, p, the expected probability of disease, and θ, a measure of variation in disease incidence among quadrats. A good fit to the binomial distribution would suggest a random spatial pattern of disease incidence, while a good fit to the beta-binomial would suggest an aggregated spatial pattern of disease incidence (the greater θ, the more aggregated the disease) (Hughes & Madden, 1993; Madden & Hughes, 1995). Neyman's $C(\alpha)$ test was used to determine if the beta-binomial distribution provided a better fit to the data than the binomial distribution (Hughes & Madden, 1993).

Anisotropy of the disease spatial pattern was detected by comparing θ values calculated with rectangular quadrats obtained by combining elementary sampling units so that the quadrats had either their long side (l) or their short side (s) parallel to the cultivation passes; quadrats were labelled '2 l' or '2 s' if they were obtained by adding two elementary sampling units and '8 l' or '8 s' if they were obtained by adding eight (2×4 or 4×2) elementary units. Wilcoxon's signed rank test was used to test if θ computed with quadrats oriented differently were significantly different, using procedure UNIVARIATE of SAS.

Comparison of the aggregation between quadrats of different sizes was done using the index of dispersion D (Hughes & Madden, 1993):

$$D = \frac{1 + n \times \theta}{1 + \theta} \quad (1)$$

where n was the number of units in a quadrat and θ the dispersion parameter of the beta-binomial distribution.

Several sizes of quadrat were used in order to determine the average size of clusters, as defined by Campbell & Madden (1990): one plant (i.e. n roots of one plant; for root disease incidence analysis only), one elementary quadrat (50×50 cm), or four, nine or 16 elementary quadrats. The peak in D indicated the scale at which most of the aggregation took place and was an estimate of the characteristic cluster size. In order to avoid a bias introduced by the choice of the point of origin on the grid, all possible quadrats were taken into account in the computation, that is, quadrats of sizes more than one were not adjacent, but were defined with a moving window. The distributions were fitted for the seminal-, crown- and whole root system.

Power law analysis

The binary form of Taylor's power law (Hughes & Madden, 1992) was used to relate heterogeneity to disease incidence. By taking the natural logarithm of the observed and theoretical (under the hypothesis of random distribution) variances, the power function was transformed into a linear function and adjusted to the data:

$$\ln(V_{obs}) = a + b \ln\left[\frac{p(1-p)}{n_h}\right] \quad (2)$$

where V_{obs} was the observed variance of the root disease incidence per plant over the 300 plants in a plot; a and b were the intercept and slope of a straight line, respectively; p was the ratio of the number of diseased roots to the total number of roots in the plot and n_h was the harmonic mean of the number of roots per plant (Madden *et al.*, 1995).

If a = 0 and b = 1, diseased roots would have a random spatial pattern that could be described by the binomial distribution. If b = 1 and a > 0, diseased roots would have an aggregated distribution, but the degree of aggregation would not depend on p. If a > 0 and b ≠ 1, the degree of aggregation (heterogeneity) would change systematically with incidence (Turechek & Mahaffee, 2004).

A covariance analysis was performed to determine the effect of cultivation method on the slope (b) and intercept (a) of the power law. The power law was considered the null hypothesis and the effect of cultivation method was added either as an intercept term or as an interaction term with the slope. Regression was performed with the procedure REG of SAS. The covariance analysis did not test whether the cultivation method had an influence on incidence or heterogeneity, but whether it affected the degree of heterogeneity after first correcting for the relationship between heterogeneity and p (Turechek & Mahaffee, 2004).

Results

Disease level

Plant disease incidence, root disease incidence and disease severity increased over the course of the growing season, both in 2003 and 2004 (Table 2). However, disease development was different between the 2 years: disease levels were higher in 2003 than in 2004. For example, 80% of plants were already diseased at the start of the study in 2003, whereas in 2004, plant disease incidence did not reach 60% at the last assessment. Analysis of variance showed no significant two-way interactions, except one interaction between assessment date and soil cultivation in 2003 in the case of disease severity. In 2003, cover crop had a significant effect (P = 0·0009, 0·0004 and 0·0001 for plant disease incidence, root disease incidence and severity, respectively): plots with a wheat cover crop had a higher level of disease than plots with mustard, which, in turn, were more severely diseased than plots where the soil was kept bare during the summer fallow period. In 2003, there was also an effect of soil cultivation for plant disease incidence (P = 0·0017) and severity (P = 0·0485), but ploughed plots had more diseased plants than those with conservation tillage, while severity was higher in the latter. In 2004, there was a significant effect of soil cultivation (P < 0·0001, < 0·0001 and = 0·0004 for plant

Table 2 Take-all (*Gaeumannomyces graminis* var. *tritici*) disease levels in each wheat plot at each assessment date

Year	Date	Cover crop[b]	Plant disease incidence (300 plants)		Root disease incidence[a] (standard error)		Severity[a] (standard error)	
			P[c]	CT[c]	P	CT	P	CT
2003	8 April	W	0·96	0·92	0·21 (0·007)	0·22 (0·009)	1·47 (0·121)	2·91 (0·379)
		M	0·90	0·63	0·15 (0·006)	0·1 (0·008)	0·63 (0·06)	1·45 (0·37)
		BS	0·77	0·64	0·09 (0·005)	0·08 (0·006)	0·24 (0·023)	0·36 (0·09)
	5 May	W	0·97	0·91	0·3 (0·01)	0·25 (0·011)	4·58 (0·391)	4·05 (0·462)
		M	0·93	0·78	0·17 (0·007)	0·14 (0·009)	1·5 (0·188)	1·91 (0·367)
		BS	0·75	0·71	0·09 (0·005)	0·1 (0·007)	0·46 (0·079)	0·69 (0·135)
	2 June	W	0·99	0·96	0·53 (0·016)	0·43 (0·014)	22·66 (1·240)	15·62 (0·907)
		M	0·96	0·90	0·39 (0·014)	0·33 (0·015)	15·4 (0·975)	15·11 (1·089)
		BS	0·92	0·82	0·23 (0·011)	0·19 (0·013)	7·79 (0·564)	7·03 (0·772)
2004	5 April	W	0·14	0·31	0·01 (0·002)	0·03 (0·005)	0·01 (0·005)	0·27 (0·092)
		M	0·15	0·34	0·01 (0·002)	0·03 (0·003)	0·04 (0·013)	0·15 (0·057)
		BS	0·12	0·26	0·01 (0·002)	0·03 (0·004)	0·03 (0·015)	0·21 (0·064)
	4 May	W	0·29	0·57	0·02 (0·002)	0·07 (0·008)	0·04 (0·009)	1·04 (0·315)
		M	0·36	0·47	0·03 (0·003)	0·05 (0·006)	0·07 (0·024)	0·56 (0·177)
		BS	0·41	0·47	0·03 (0·003)	0·07 (0·008)	0·09 (0·025)	0·99 (0·299)
	1 June	W	0·40	0·72	0·06 (0·01)	0·18 (0·015)	1·7 (0·596)	6·56 (0·976)
		M	0·50	0·77	0·08 (0·01)	0·18 (0·015)	1·99 (0·511)	6·54 (1·053)
		BS	0·45	0·73	0·06 (0·007)	0·23 (0·017)	0·85 (0·249)	7·8 (0·975)

[a]Mean of 300 plants.
[b]Crops sown between the first and second wheat crops: W (wheat), M (mustard), BS (bare soil).
[c]Cultivation method: P (ploughing), CT (conservation tillage).

disease incidence, root disease incidence and severity, respectively) with a lower disease level in ploughed plots than in those with conservation tillage.

Fit to frequency distributions

The combination of 2 years, three assessments per year, two types of disease incidence (root and plant), two root systems, nine sizes of quadrats and six plots led to 1254 analyses. Of these analyses, 82·5% allowed a successful fit of the beta-binomial distribution through maximum likelihood estimation, and significant aggregation (as indicated by a significant C(α) test) was detected in 66·4% of the datasets. Aggregation was detected more often when root disease incidence was considered than when plant disease incidence was analysed (in 83·3% and 47·6% of the cases with root disease incidence and plant disease incidence, respectively). Aggregation tended to increase over time: at assessment 1, only 39·8% of the analyses based on plant disease incidence concluded that aggregation was significant, 50% at assessment 2 and 54·9% at assessment 3; for root disease incidence, 75·4% of the analyses showed significant aggregation at assessment 1, 82·5% at assessment 2 and 95% at assessment 3. Estimated θ ranged from 0 to 1·05, with a mean of 0·05 and a median of 0·02.

Anisotropy

Anisotropy was detected, with θ significantly ($P < 0·05$) higher when computed with quadrats oriented in the same direction as the cultivation passes (2 l and 8 l) than when computed with quadrats oriented perpendicularly (2 s and 8 s) (Table 3). Anisotropy was more marked in plots with conservation tillage than in ploughed plots: it was significant for all combinations of disease level (plant and root incidence), root system (seminal and crown) and quadrat size (2 and 8) for plots with conservation tillage, but was detected in four combinations out of eight only in the ploughed plots (Table 3).

Table 3 Probabilities (*P*) of Wilcoxon's signed ranks test for the difference between θ (dispersion parameter of the beta-binomial distribution) for vertical and horizontal quadrats (all assessment dates and cover crops pooled) for take-all disease (*Gaeumannomyces graminis* var. *tritici*) of wheat

System	Disease incidence	Cultivation method	2 l-2 s[a]	8 l-8 s[b]
Crown roots	Plant	Ploughing	0·041	0·261
		Conservation tillage	0·016	0·002
	Root	Ploughing	0·026	0·045
		Conservation tillage	0·010	0·016
Seminal roots	Plant	Ploughing	0·424	0·353
		Conservation tillage	0·058	0·021
	Root	Ploughing	0·005	0·531
		Conservation tillage	0·010	0·0003

[a]Two elementary quadrats oriented in the same direction as (2 l) and perpendicularly to (2 s) the cultivation passes.
[b]Quadrats made from eight (2 × 4) elementary sampling units oriented in the same direction as (8 l) and perpendicularly to (8 s) the cultivation passes.

Cluster size

The index of aggregation D was in general higher in conservation tillage plots than in ploughed plots, in particular for plant disease incidence (see Fig. 1 for crown root system, results from seminal root system were similar and are summarized in Table 4). In most cases, D increased and then decreased as quadrat size increased. The peak in D was an estimate of average cluster size. These estimated sizes are given in Table 4. Clusters of diseased roots typically ranged from two to four quadrats (1 m in diameter) in the case of crown roots (Fig. 1 a1–a3, b1–b3 and Table 4)

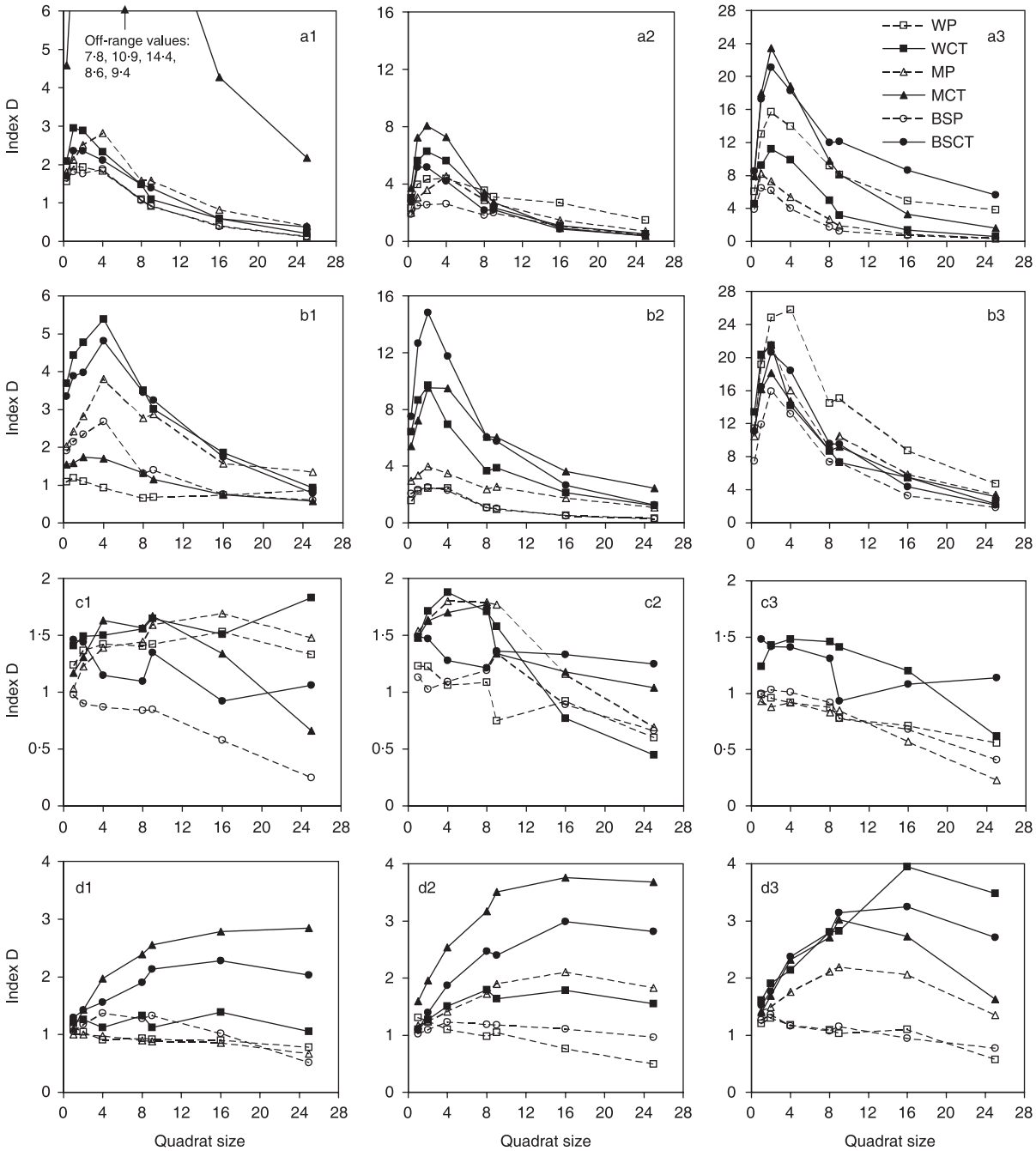

Figure 1 Relationship between index D and quadrat size (expressed as number of 50 × 50 cm sampling units) for natural wheat take-all (*Gaeumannomyces graminis* var. *tritici*) epidemics. a and b, root disease incidence observed on crown root system; c and d, plant disease incidence observed on crown root system a and c, 2003; b and d, 2004; a1, b1, c1 and d1, stem elongation; a2, b2, c2 and d2, booting; a3, b3, c3 and d3, grain-milk growth stage. square symbols: wheat cover crop; triangles: mustard cover crop; circles: bare soil; empty symbols and dashed line: ploughing; filled symbols and solid line: conservation tillage.

Table 4 Cluster sizes[a] estimated from the plot of index D against quadrat size for take-all disease (*Gaeumannomyces graminis* var. *tritici*) of wheat

Year	Root system	Assessment number	Plot	Plant disease incidence		Root disease incidence	
				Ploughing	Conservation tillage	Ploughing	Conservation tillage
2003	seminal	1	wheat	4	16–25	9	4–9
			mustard	1	1–4	4	4–9
			bare soil	4	9	4	1 and 9
		2	wheat	9–16	1 and 9	9	1 and 9
			mustard	1–9	1	4–9	1–4
			bare soil	4	1	9	1 and 9
	crown	1	wheat	4 and 16	9 and 25	1–4	1
			mustard	16	4–9	4	4
			bare soil	0	1, 9 and 25	1–4	1
		2	wheat	1	4	1–4	1–4
			mustard	4–9	4	4	1–4
			bare soil	9	1	1–4	1
		3	wheat	0	4–16	1–4	1–4
			mustard	0	1 and 25	1	1–4
			bare soil	1–4	1 and 25	1	1–4
2004	seminal	1	wheat	0	0	9–16	0·3 and 16
			mustard	0	16	1	9–16
			bare soil	0	16	4–8	9
		2	wheat	0	0	1	9–16
			mustard	16–25	9	16	4–9
			bare soil	0	16	1	9–16
		3	wheat	4	1 and 16	9–16	16
			mustard	9–25	>25	9–16	16
			bare soil	1–4	16	1	9
	crown	1	wheat	0	1 and 16	0	4
			mustard	0	>25	4	4
			bare soil	4–9	9–25	4	4
		2	wheat	1	16–25	1–4	1
			mustard	16	16–25	1–4	4
			bare soil	0	16–25	1–4	1–4
		3	wheat	1 and 16	16	4	1
			mustard	9–16	9–16	1	1
			bare soil	1 and 9	9–25	1–4	4

[a]Sizes are expressed as number of 50 × 50 cm elementary sampling units; a size of 0 indicates that no significant aggregation could be detected whatever the size of the quadrat.

and up to nine (in 2003) or 16 (in 2004) quadrats (1·5–2 m in diameter) in the case of seminal roots (Table 4). Clusters of diseased plants were more variable in size, and in some cases no aggregation was detected (Fig. 1 c1–c3, d1–d3 and Table 4).

Evolution of aggregation during the cropping season

There was no visible trend in the spatial structure of diseased plants during the season as the index D remained almost constant over the three successive assessments each year (Fig. 1 c1–c3 and d1–d3). For example, for quadrat size '4', i.e. quadrats 1 × 1 m, mean D over the six plots for the three consecutive assessments was 1·26 (Fig. 1 c1), 1·32 (Fig. 1 c2) and 1·2 (Fig. 1 c3) in 2003; 1·24 (Fig. 1 d1), 1·47 (Fig. 1 d2) and 1·51 (Fig. 1 d3) in 2004. Furthermore, there was neither consistent increase nor decrease in cluster size when plant disease incidence was analysed (Table 4). On the contrary, the spatial pattern of diseased roots became more aggregated during the season (note the change of scale in the y-axis in Fig. 1, from a1 to a3 and from b1 to b3): mean D over the six plots for quadrat size '4' was 3·96 during stem elongation (Fig. 1 a1), 5·18 at booting (Fig. 1 a2) and 11·73 at the grain-milk stage (Fig. 1 a3) in 2003; and 3·36 (Fig. 1 b1), 5·37 (Fig. 1 b2) and 15·86 (Fig. 1 b3), respectively, in 2004. However, this increase in D was not associated with an increase in cluster size (Table 4), indicating that clusters became more saturated as time went by, but did not change in size: the increase of disease aggregation corresponded to disease intensification within clusters.

Relationship between disease heterogeneity and incidence

The binary form of Taylor's power law provided a good fit to the data for both years (Table 5, Fig. 2a,b). The estimated parameters for slope and intercept were significantly

Table 5 Estimated parameters of the binary power law[a] for incidence of take-all disease (*Gaeumannomyces graminis* var. *tritici*) on wheat roots

Year	R^2	a	SE(a)[b]	ProbF[c]	b	SE(b)[b]	ProbF[c]
2003	0·77	3·60	1·00	0·0024	1·51	0·20	0·0239
2004	0·94	6·28	0·70	<0·001	1·76	0·11	<0·001

[a] $\ln(V_{obs}) = a + b \ln[p(1 - p)/n_h]$ with p the mean root disease incidence and n_h the harmonic mean of number of roots per plant.
[b] SE is the standard error of the parameter.
[c] ProbF is the probability associated with the F-test with H_0: a = 0 and b = 1.

(α = 5%) greater than 0 and 1, respectively, according to the *F* test, indicating that heterogeneity changed systematically with disease incidence. Covariance analysis indicated that the factor cultivation method had no effect on the parameter estimates (Table 6).

Discussion

The spatial structure of take-all disease of winter wheat was analysed in natural epidemics of second consecutive wheat crops under different cultural practices. The disease levels (severity and root- and plant-disease incidence) were influenced by cultural practices and differed between the 2 years. In 2003, the epidemics started earlier than in 2004, 80% of the plants being diseased in April, while only about 20% were diseased at the same point in 2004 (Table 1). The results concerning the effect of cover crops on disease levels were consistent with previous reports (Dulout *et al.*, 1997; Ennaïfar *et al.*, 2005), which showed that primary inoculum and/or primary infections were affected by the type of crop preceding the wheat: the highest inoculum (or initial disease) level was obtained with wheat volunteers and the lowest with bare soil, other crops or weeds such as blackgrass, mustard or oats having an intermediate effect. The use of different cultural practices allowed different epidemics to be generated within the same year in order to study the spatial pattern and the relationship between aggregation and disease level.

The spatial structure of take-all was often found to be aggregated, which is in agreement with previous reports (Clarkson & Polley, 1981). Anisotropy of the spatial pattern was detected in both years, as a higher degree of aggregation was observed for quadrats oriented in the direction of cultivation passes (Table 3). This anisotropy could not be attributed to host anisotropy (i.e. a possibly increased spread along rows) because it was observed in plots with broadcast sowing as well as plots with line sowing. The hypothesis then is that the anisotropy was

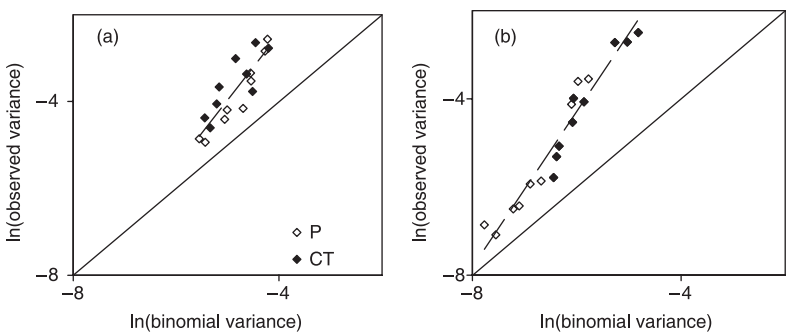

Figure 2 Relationship (on a log scale) between the observed variance of the incidence of wheat roots with take-all disease (*Gaeumannomyces graminis* var. *tritici*) per quadrat and the expected variance under the hypothesis of a random distribution of diseased roots among quadrats, (a) 2003 epidemic, (b) 2004 epidemic. Empty symbols: ploughing; filled symbols: conservation tillage. Each dot represents the observed variance of root disease incidence among the 300 plants of a plot at a given assessment date.

Table 6 Deviances and chi-square tests for the covariance models testing for the effect of soil cultivation on the intercept or slope parameters of the binary power law[a] for root incidence of take-all (*Gaeumannomyces graminis* var. *tritici*) on wheat in 2003 and 2004

				Intercept			Slope		
Year	Model	df1[b]	df2[b]	Dev.	Diff[c]	Probchi[d]	Dev.	Diff[c]	Probchi[d]
2003	Power law	16	–	2·28	–	–	2·28	–	–
	+ Soil cultivation	15	1	1·71	0·57	0·45	1·67	0·62	0·43
2004	Power law	16	–	2·18	–	–	2·18	–	–
	+ Soil cultivation	15	1	1·73	0·45	0·50	1·75	0·43	0·51

[a] $\ln(V_{obs}) = a + b \ln[p(1 - p)/n_h]$ with p the mean root disease incidence and n_h the harmonic mean of number of roots per plant.
[b] df1 = degrees of freedom for the model deviance; df2 = degrees of freedom for soil cultivation factor.
[c] Difference between the deviance for the power law model and the model with the soil cultivation factor included as either an intercept term or as an interaction with the slope term. If the deviance was reduced by χ^2_{df2}, the factor was considered to improve the model significantly.
[d] Probability that the difference would be more than observed under the hypothesis of no effect of soil cultivation.

caused by soil cultivation. Soil cultivation can displace inoculum propagules of Ggt up to 2·5 m, but more frequently 0·9 m (Prew, 1980b). The displacement of inoculum propagules from an inoculum point source (e.g. the roots and crown particles from one, or a group of a few, infected plants from the first wheat crop) could thus account for the anisotropy detected in this study. Cluster anisotropy was more pronounced in plots with conservation tillage than in ploughed plots, which may have been because ploughing can displace inoculum particles transversely as well as longitudinally, as observed for weed seeds (N. Colbach, INRA Dijon, France, personal communication), thus reducing anisotropy.

The size of clusters of diseased plants or roots was estimated through plots of indices of aggregation D against size of quadrat (Fig. 1). The index D was more suitable than the θ parameter of the beta-binomial distribution for this kind of approach because θ is dependent on the number of units per quadrat (n) (Madden & Hughes, 1995; Madden *et al.*, 1995) and typically decreases when n is increased. Conversely, there can be a peak in D if θ first decreases faster than n increases and then slower, as can be seen from Eqn 1. The rate of change of θ with change in n will dictate the value of n where max(D) occurs, which is a good empirical measure of scale of pattern (L. Madden, Ohio State University, USA, personal communication). The estimated cluster sizes were similar in 2003 and 2004, despite the other observed differences between the epidemics during the 2 years: clusters ranged in size from 50 × 50 cm to more than 2·5 × 2·5 m, but were usually less than 1 × 1 m when based on crown-root disease incidence, 1·5 × 1·5 m for seminal roots and up to 2·5 × 2·5 m for plant disease incidence (Table 4).

In a previous study of take-all spatial pattern, Cotterill & Sivasithamparam (1989) concluded that take-all inoculum was aggregated on the basis of visual assessment of infectivity maps (i.e. disease severity on bait seedlings grown in soil cores), but they did not statistically test for aggregation. A spatial analysis of their published results was performed in the present study, using the D index of aggregation (by considering their 5-class severity scale of bait wheat seedlings as an incidence based on four elements). This analysis showed aggregation of Ggt inoculum with an average cluster size of 2 × 2 m after sowing of the second wheat crop (Fig. 3), which was within the range of sizes found in the present study.

There was no increase in average cluster size over the cropping season (Table 4), indicating that secondary infections do not contribute to a large extent to disease spread within a cropping season. This result was in agreement with previous work indicating that within-season disease spread from a focal source of inoculum was less than 20 cm (Prew, 1980a; Willocquet & Lebreton, 2005). Conversely, signs of changes in the scale of disease patterns were observed between April and June assessments in a second wheat crop at Rothamsted, UK, (Hornby *et al.*, 1989), but the disease patches in that field were only about 30 × 40 cm in June, smaller than in the present study.

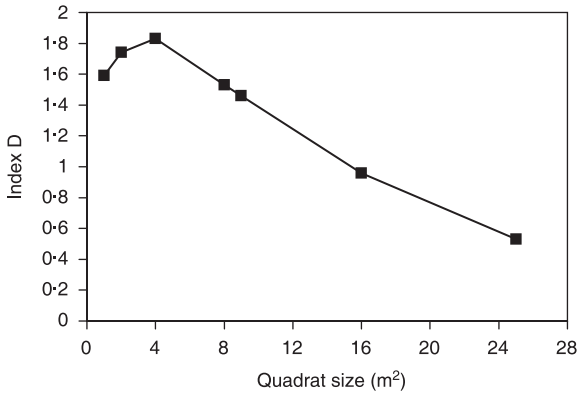

Figure 3 Plot of D index of take-all (*Gaeumannomyces graminis* var. *tritici*) aggregation against quadrat size (in m^2) just after sowing of a second wheat crop (data from Cotterill & Sivasithamparam (1988), computations based on a 5-class disease index: 0, 1–25, 26–50, 51–75 and 76–100% of bait seedlings' root systems diseased).

Large clusters (up to 2·5 m in diameter) were detected as early as April in both years in second wheat plots. As no increase in cluster size was observed within the growing season, this initial spatial pattern must have originated from other processes. As in the case of anisotropy, the effect of soil cultivation is suggested here as the factor responsible for the spread of inoculum propagules before sowing of the second wheat and thus for the initial patchy spatial structure of take-all. The importance of soil cultivation in the spread of Ggt was demonstrated in the case of artificially introduced inoculum in a first wheat crop (Prew, 1980b). The increase with time in θ and D indices computed from root-disease-incidence data (Fig. 1a,b) suggests disease intensification within clusters during the growing season, building up aggregated primary inoculum for the following years.

The power law analysis also showed that heterogeneity of root disease incidence (i.e. aggregation of diseased roots among plants) was related to mean disease level (Fig. 2). The inclusion of soil cultivation did not improve the power law model significantly, indicating that the relationship between aggregation and disease level was not affected by soil cultivation.

The results on take-all spatial structure indicated that disease intensifies within clusters and that cluster size does not increase over the course of the epidemic in second wheat crops (Fig. 1; Table 4). This could not result from the seed treatment because it has been shown that silthiofam has an effect on primary infection, but not on secondary infection (Bailey *et al.*, 2005). The analysis of spatial anisotropy also suggested that soil cultivation, through mechanical displacement of inoculum particles, may play an important role in take-all spatial structuring. These results are in agreement with the general view of successive processes of inoculum amplification during epidemics, followed by inoculum spatial dilution between cropping seasons, in the case of soilborne diseases (Truscott & Gilligan, 2001).

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