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Pathometric relationships reveal epidemiological processes involved in carrot cavity spot epidemics

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Abstract Carrot cavity spot (CCS) is one of the most important soilborne diseases affecting the carrot crop. The few epidemiological studies that have investigated the temporal and spatial dynamics of the disease have been based solely on diagrammatic scales or semi-quantitative indices. To reveal epidemiological processes involved in the development of CCS epidemics, we investigated pathometric relationships. To this end, standardised measurements were defined (disease incidence *i*, lesion density *d*, conditional lesion density *cd*, lesion size *ls*, and total diseased area *tda*). The evolution of a cohort of CCS lesions according to their size suggested that lesions can expand over time. Two pathometric relationships were tested: a first one, between *i* and *tda*, is given by the equation $i=100(1-\exp(-a(t)tda))$, where *t* is thermal time, and a second one, between *tda*, *d*, and *ls*, is given by the equation $tda=c(t)\pi d(ls/2)^2$. These relationships were validated for CCS epidemics in the case of field experiments, a survey in commercial fields, and a controlled-conditions experiment. The temporal linear decrease of the time-dependent parameter $a(t)$ in the first relationship suggested that CCS epidemics followed classical epidemiological phases driven by successive processes: (1) the mobilisation of soil inoculum leading to primary infection, (2) the spread of disease to neighbouring taproots (alloinfection), and (3) the intensification of disease on the taproot (autoinfection). This is consistent with complementary experimental results which demonstrated that auto- and alloinfections occur in CCS epidemics.

Keywords Incidence–severity relationship · Phytopathometry · Primary infection · *Pythium* spp. · Secondary infection · Soilborne disease

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Introduction

Carrot cavity spot (CCS) is one of the most important soilborne diseases affecting the carrot (*Daucus carota*) crop in Europe and other temperate countries. The

disease was first described in the 1960s (Guba et al. 1961; White 1986; Montfort and Rouxel 1988) and is characterized by small sunken elliptical lesions on the carrot taproots caused by a complex of *Pythium* spp. dominated by *P. violae* and *P. sulcatum* in France (Montfort and Rouxel 1988; Breton and Rouxel 1993; Guerin et al. 1994; Suffert and Guibert 2007). The lesions have a mean diameter of 2–15 mm, that may darken over time, and depending on edaphic conditions, may increase in size (Hiltunen and White 2002; Suffert 2006). One or more lesions occur anywhere on the surface of taproots and depreciate their quality. The identification of the epidemiological processes that favour the development of CCS is a crucial step in the optimisation of control methods. To this end, thorough assessments of the disease are required in order to quantify the epidemic progress in the field and to better understand CCS dynamics.

Much work has already been carried out on the aetiology of CCS; several measurements have illustrated an increase in overall symptom intensity over time. Perry (1983) found that the percentage of roots with lesions was higher in carrots harvested in November than in October. Maynard et al. (1963) found that the number of lesions per root increased as plants aged. Montfort and Rouxel (1988) reported the presence of small lesions on young carrots and a gradual increase in the frequency of root symptoms during the 4-month growing season. Perry and Harrison (1979) reported increases in the size of lesions over time. Some authors used a diagrammatic scale, as proposed by Sweet et al. (1986), and formalised aggregated disease indices. The ways to assess disease intensity are indeed prolific (see Suffert 2006 for a complete review); however, this makes comparisons very difficult. Moreover, the terminology used to assess CCS has not yet integrated the new concepts in phytopathometry introduced by Large (1966) and developed in following studies (e.g., Bald 1969; Kranz 1988; Nutter et al. 1991).

Some scientists have suggested a way to characterise the disease in order to have a better understanding of epidemiological processes. Guba et al. (1961) reported that at a given location some carrot roots had abundant lesions whilst others had few, if any, and that lesions tended to occur in clusters on individual roots. This aggregation at the root scale suggests that *Pythium* is involved in a pathogenic process, with new infections caused by infectious propagules distributed in local micro-aggregates (oosporic or mycelial form; Phelps et al. 1991). This may also suggest that CCS pathogens are involved in a reproductive process, possibly based on mycelial growth over root surfaces, with primary lesions producing secondary lesions. For other well-studied diseases, some mathematical equations have been proposed to describe the relationships between disease measurements at different scales (e.g., Seem 1984; McRoberts et al. 2003). Because the monitoring of symptoms on underground organs can only be done by destructive sampling, the understanding of cryptic processes involved in soilborne epidemics is generally based on hypotheses indirectly validated by experimental data. Some of these hypotheses can be investigated on the basis of the analyses of pathometric relationships. Unfortunately, due to insufficient normalized measurements in the case of CCS, such relationships have not yet been established.

Within this context, we set out to standardise measurements of CCS and to describe the evolution in number and size of lesions in field experiments after artificial soil infestation, in order to investigate and use pathometric relationships to reveal epidemiological processes involved in CCS epidemics, such as primary and secondary infections.

Materials and methods

Definitions of measurement for CCS assessment

R is the total number of roots in a sampling unit and I is the number of diseased roots (Table 1). N_r is the number of CCS lesions on the root $r \in [1; R]$. Roots with at least one CCS lesion were classified as diseased, whatever the size of the lesion. Lesions were graded according to their mean diameter $D_n = (a_1 + a_2)/2$, where a_1 and a_2 are major and minor axes of an ellipse corresponding to a lesion. Each lesion $n \in [1; N_r]$ was assigned to a diameter class Δ: Δ_0 when $D_n \leq 1$ mm, Δ_1 when $D_n \in]1; 3$ mm], Δ_3 when $D_n \in]3; 5$ mm], Δ_5 when $D_n \in]5; 10$ mm], and Δ_{10} when $D_n > 10$ mm. Δ_0 and Δ_{10} were chosen as lower and upper limits because the detection threshold of a CCS lesion is $D_n = 1$ mm and D_n was rarely >10 mm (Suffert 2006); Δ_5 was chosen because 5 is the median value of the interval]0; 10 mm] (i.e., (10–0)/2=5); Δ_3 was chosen because 3 is the median

Table 1 Glossary of symbols and formulae used to define CCS measurements

Disease measurement	Symbol	Formula	Definition	Unit
	R		Number of roots in the sampling unit	–
	I		Number of diseased roots in the sampling unit	–
	D_n		Mean diameter of the lesion $n \in [1; N_r]$ on the root $r \in [1; R]$	mm
	N_r		Number of lesions on the root r	–
	$N_{\Delta,r}$		Number of lesions assigned to the diameter class Δ on the root r	–
	D_M		Diameter value considered as 'median' for each diameter class Δ (D_M=1 for Δ_0, D_M=2 for Δ_1, D_M=4 for Δ_3, D_M=7.5 for Δ_5, D_M=10 for Δ_{10})	mm
Disease incidence	i	(1) $i = \frac{I}{R} \times 100$	Percentage of diseased roots in the sampling unit	–
Lesion density	d	(2) $d = \frac{1}{R}\sum_{r=1}^{R} N_r$	Mean number of lesions per root in the sampling unit	lesion per root
Lesion conditional density	cd	(3) $cd = \frac{1}{I}\sum_{r=1}^{R} N_r$	Mean number of lesions per diseased root in the sampling unit	lesion per root
Lesion size	ls	(4) $ls = \frac{1}{R \times N_r}\sum_{r=1}^{R}\sum_{\Delta_0}^{\Delta_{10}}\left(N_{\Delta,r} \times D_M\right)$	Mean diameter of lesions on the root	mm
Total diseased area	tda	(5) $tda = \frac{1}{R}\sum_{r=1}^{R}\sum_{\Delta_0}^{\Delta_{10}}\left(N_{\Delta,r} \times \pi\left(\frac{D_M}{2}\right)^2\right)$	Cumulative necrotic area at the surface of the root	mm^2

value of the interval]1; 5 mm] (i.e., 1+(5−1)/2=3). $N_{\Delta,r}$ is the number of lesions assigned to class Δ on the carrot root r.

Disease incidence (i) Disease incidence is defined as the percentage of diseased carrot roots (Eq. 1 in Table 1). Additionally, four truncated disease incidences i_Δ are defined as the disease incidence after a truncation at level Δ: i_1 (lesions are >1 mm, i.e., D_n> 1 mm), i_3 (D_n>3 mm), i_5 (D_n>5 mm) and i_{10} (D_n> 10 mm). Truncated measurement is justified, for example, to avoid overestimating disease incidence usually caused by diagnostic mistakes due to very small lesions.

Lesion density (d) Lesion density is the mean number of lesions per root in a sampling unit (Eq. 2 in Table 1).

Conditional lesion density (cd) Conditional disease is a measurement of disease that depends on the plants being infected (McRoberts et al. 2003). Conditional lesion density *cd* is defined as the mean number of lesions per diseased root (Eq. 3 in Table 1).

Lesion size (ls) Lesion size (or symptom intensity *si*) is an estimation of the size of the lesions on a diseased root in a sampling unit. The five diameter classes defined above (Δ_0, Δ_1, Δ_3, Δ_5, and Δ_{10}) are used for scoring purposes. The lesion size in a sampling unit was calculated using the median value D_M of each diameter class Δ, except for Δ_0 and Δ_{10} for the detection reasons stated above (Eq. 4 in Table 1).

Total diseased area (tda) Total diseased area is the cumulative necrotic area at the surface of a diseased root (Eq. 5 in Table 1). The ratio between the total diseased area *tda* and the total area *ta* of the taproot would be a definition of severity *sensu stricto* (Nutter et al. 1991) as it is a proportion of necrotic surface. However, the value of *tda*/*ta* remains very low even in high CCS intensities: usually <0.1 according to our

own field observations and the diagrammatic scale proposed by Sweet et al. (1986).

Data acquisition

Field experiment A field experiment was carried out in plots located at the INRA Station of Le Rheu (Ille-et-Vilaine, France; 48°01′ N, 1°43′ W) in 2001 and 2002 in the same area (Z1), and in 2003 in another area (Z2; Suffert 2007). The silt loam soil (16.3% clay, 62.5% silt, 21.2% sand, and 2.4% organic matter) was naturally contaminated with *P. sulcatum*, *P. intermedium*, *P. sylvaticum*, *P. coloratum*, and *P. ultimum* (Suffert and Guibert 2007) and was artificially infested with *P. violae* (strain Pv490, CBS 102.609) 3 weeks before sowing carrots. To produce inoculum, bags containing 240 ml of dry barley grains and 300 ml of distilled water were autoclaved twice at 120°C for 1 h at 24-h intervals, and inoculated with plugs of *P. violae* grown on carrot juice agar; these were incubated for 3 weeks at 20°C in a dark room. The experimental areas were sown with carrots (cv. Nanco) on 22 May 2001, on 13 June 2002, and on 20 June 2003. Infected barley grains were distributed manually in 2001 (Z1) and 2003 (Z2). The experiment was set up as a randomised block design (four blocks), with inoculum dose as one factor with four treatments (control, 5 g m^{-2}, 50 g m^{-2}, and 500 g m^{-2} of barley grain colonised by *P. violae*) to generate different CCS intensities. Experimental units were 2× 6 m plots and involved five rows 50 cm apart with about 80 plants per linear meter. All plots received the same treatments as described by Suffert (2007). Disease was assessed about every month on samples (8, 5, and 6 sampling dates in 2001, 2002, and 2003, respectively) consisting of all carrot roots present along a 50 cm segment of any of the three central rows of each plot; each sample typically included 30–40 roots. The number and size of CCS lesions were assessed on each root. Disease measurements *i*, *d*, cd, *ls*, and *tda* were then calculated.

Field survey An epidemiological survey was carried out in the Créances region (Manche, France), where edaphic, cultural, and socioeconomic conditions are homogeneous. It is a traditional production area in which carrots are grown on small (on average 45 ha total cultivated area) and highly specialised farms. Soils are sandy and the organic matter rate is low (from 1% to 1.5%). CCS can be severe in the fields with repeated cultivation of carrots. *Pythium violae* and *P. sulcatum* are the species most frequently isolated from CCS lesions in this area (Breton and Rouxel 1993). The survey concerned commercial carrot crops mainly sown between 1 June and 20 June 2002 and harvested between 20 January and 10 February 2003. After harvest, carrots were loaded into trailers and sent to the cooperative factory to be washed, graded and packaged. Each trailer contained between 3 and 4 tons of carrots coming from a single homogeneous field. Ten samples of 30 carrots were randomly taken from each trailer. Overall, there were 55 trailers corresponding to 33 different fields. Symptoms of CCS were scored as described above.

Controlled-conditions experiment A steam-sterilised reconstituted soil (one half sand, one fourth compost, and one fourth organic soil) was artificially infested with infected carrot residues from a CCS epidemic mainly due to *P. violae* (Suffert and Guibert 2007). Typical lesions on 5 month-old carrots were cut to generate small fragments of necrotic tissues with sides 2–3 mm long, that were used to infest soil by evenly distributing them throughout the volume of 4 l pots just before sowing. The experiment was set up as a randomised block design (three blocks), with inoculum dose as one factor with four treatments as described by Suffert and Montfort (2007; 0.35 g, 1.75 g, 3.5 g, and 7 g of CCS-affected carrot root fragments per pot) to generate different CCS intensities. Seven carrot seeds (cv. Nanco) were sown in each pot immediately after infestation. Pots were placed in controlled-conditions (16 h of daylight at 20°C and 8 h of night at 12°C) and watered on the soil surface as needed to adjust soil moisture to the water-holding capacity. Carrot roots were harvested and washed 12 weeks after sowing and symptoms of CCS scored as described above.

Pathometric relationships and statistical analyses

James and Shih (1973), and then Rouse et al. (1981), modelled the relationship between disease incidence *i* and disease severity *s* (defined as the percentage of a plant surface affected by a disease) with a restricted exponential equation (Eq. 6) that describes the increase in disease incidence from a low value

(usually zero) towards an upper value b (usually 100 on a percentage scale) according to the increase in disease severity:

$$i = b.(1 - e^{-a \times s}) \quad (6)$$

Because Rouse et al. (1981) showed that the severity–incidence relationship can fluctuate over time, a modified version of the classical equation in which b=100 and t = thermal time (accumulated degree days from sowing, base 3.5°C; Eq. 7) was selected and fitted to data sets acquired in the three experimental conditions using the SAS statistical package, version 8.1 (SAS Institute Inc 2000):

$$i = 100.\left(1 - e^{-a(t)\,tda}\right) \quad (7)$$

The growth of both carrot and *Pythium* spp. responds strongly to the temperature of their environment. The 3.5°C threshold is the minimum temperature for carrot root growth (Tamet et al. 1993) and is close to the minimum growth temperature of the *Pythium* spp. involved in CCS (Van der Plaats-Niterink 1981; Suffert and Guibert 2007). Assessments of CCS were made in different locations and over long periods, including the winter period during which temperature was low (e.g., the minimal daily air temperature was <3.5°C for 57 days in the field experiment between 1 November 2001 and 28 February 2002); therefore, to cope with the effects temperature can have, thermal time was used (Lovell et al. 2004). It also best suits the overall investigation that included both field experiments and the controlled-conditions experiment. Because the development of CCS also responds to soil moisture (Hiltunen and White 2002), a hydrothermal time based on soil temperature and water potential (Dahal and Bradford 1994) would have been useful, but it was not practicable.

The second relationship (Eq. 8), in which *tda* is expressed as a function of *d*, *ls*, and thermal time *t*, was then fitted to data sets as follows:

$$tda = c(t)\pi d(ls/2)^2 \quad (8)$$

Parameters $a(t)$ in Eq. 7 and $c(t)$ in Eq. 8 were set constant for data from the survey and the controlled-conditions experiment, but varied with t for data from the field experiment because of the multiplicity of sampling dates. The linearity of the relationship between the parameter a and the thermal time t was tested fitting the model $a(t) = \alpha t + \beta$ to the field experiment data in 2001, 2002, and 2003.

Results

Illustration of the diversity of CCS intensities

Characteristics of CCS symptoms were gleaned from the field experiment data (2001–2003). Various distributions of lesions on roots were ranked according to disease density *d*, total diseased area *tda*, and lesion size *ls* (Fig. 1). Quantitative grades were used for *d* and *tda*, and mean diameter of lesions *ls* was expressed by qualitative grades (low, medium or high). Cases (a) and (d) showed isolated large lesions. Cases (e), (g), and (h) showed large lesions with an aggregated spatial distribution at the root scale. Cases (c), (f), and (i) showed an aggregated spatial distribution of some coalescent lesions with indistinct contours. Cases (a), (d) and (h) gave very different measurements of *d* and estimations of *ls*, while *tda* were similar.

Assessment of CCS progress and lesion expansion

CCS epidemics obtained after a soil infestation with two densities of *P. violae* (dose 5 g m^{-2} and dose 500 g m^{-2}) in field experiment 2001 are illustrated with four disease measurements (*i*, *d*, *ls*, and *tda*) plotted against time (Fig. 2). During the first part of the epidemics (June to September, i.e., 0 to 1,500 degree days) the four disease variables increased slowly. The CCS intensity, illustrated by the mean lesion density *d* (0 to 0.5 lesions per root) and the mean incidence *i* (0% to 20%), was stable over the summer before increasing quickly in autumn. The incidence *i* and the total diseased area *tda* soared between 1,500 degree days (September) and 2,200 degree days (November).

The conditional lesion density was recorded according to lesion diameter at different sampling dates in the field experiment (S3 to S8 in 2001, S3 to S5 in 2002, S2 to S6 in 2003; Fig. 3). The data were expressed as the number of lesions arranged by diameter class (≤1 mm,]1; 3 mm],]3; 5 mm],]5; 10 mm], >10 mm), and as the percentage of lesions in each diameter class. At the beginning of the epidemic in 2001, roots were small and thin, and the diameter of

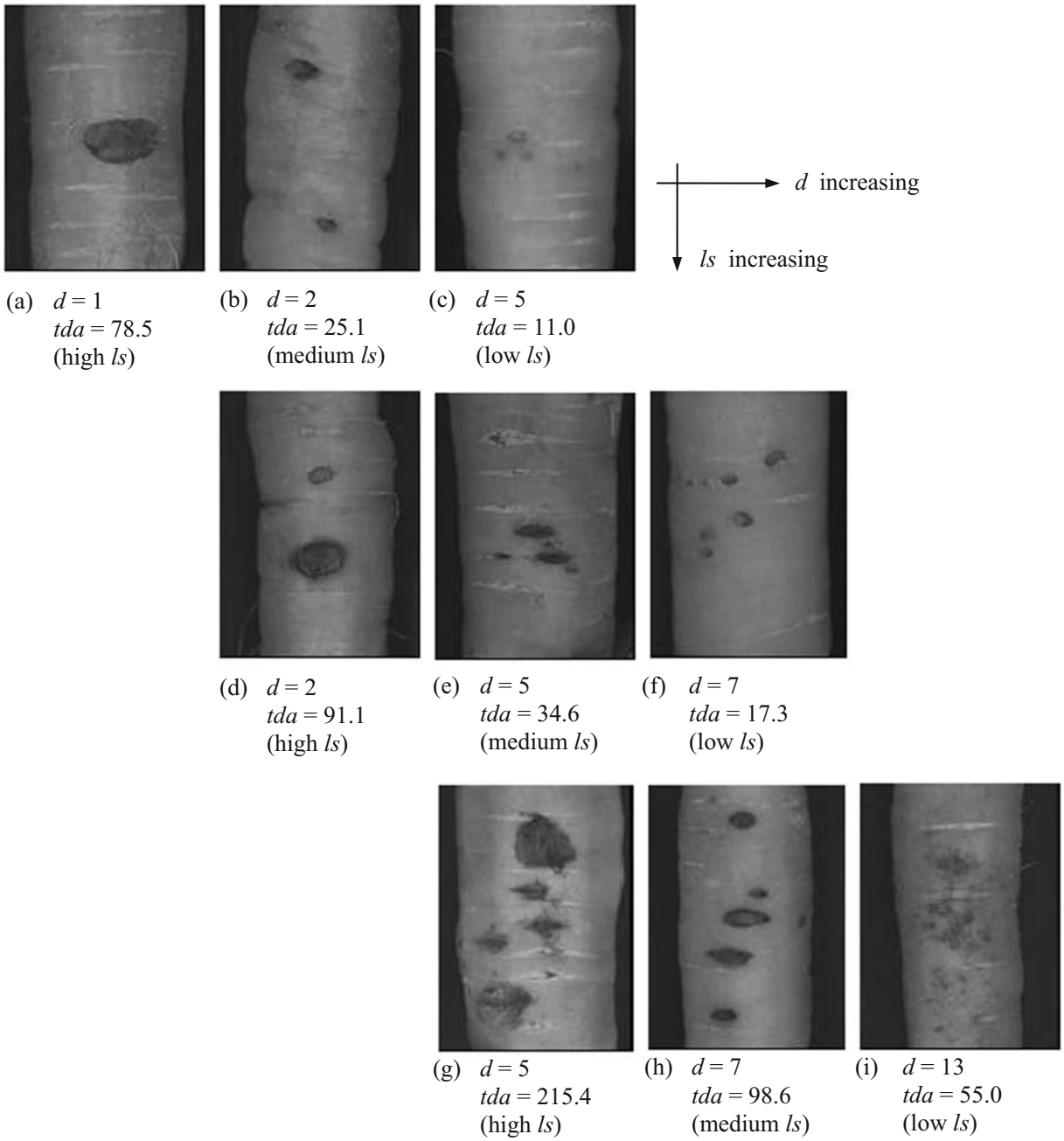

Fig. 1 CCS intensities on carrot taproots according to lesions density *d* (lesions per root), total diseased area *tda* (mm^2), and lesion size *ls* (low, medium, or high)

most lesions was <3 mm (Fig. 3a). Larger lesions (]5; 10 mm]) appeared after S6, while the proportion of small lesions (≤3 mm) decreased. At the end of winter (S6 to S8), the number and the percentage of small lesions again increased. Approximately the same pattern was observed during the 2002 epidemic (Fig. 3b): largest lesions (>5 mm) appeared in autumn (S4 and S5), while the proportion of small lesions (≤3 mm) decreased. In 2003 (Fig. 3c), the evolution of the cohort of CCS lesions according to their size

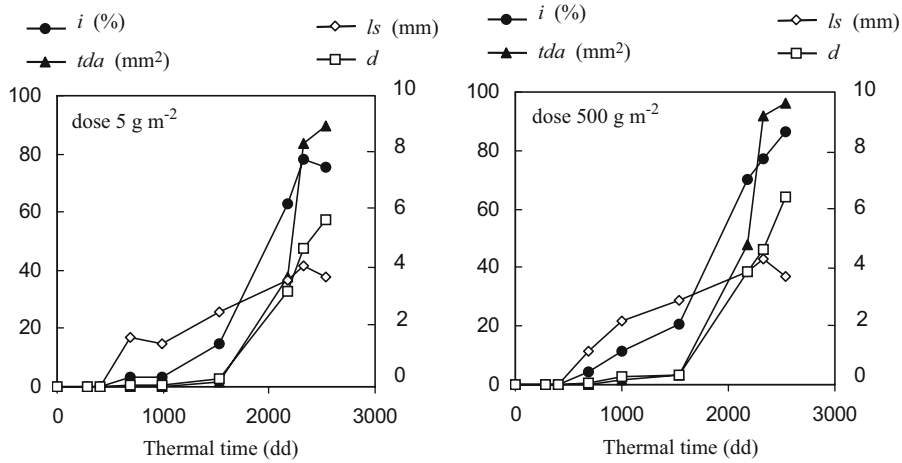

Fig. 2 CCS progression curves for four disease measurements (disease incidence *i*, lesion density *d*, lesion size *ls*, and total diseased area *tda*) plotted against thermal time (accumulated degree days from sowing, base 3.5°C) in the field experiment 2001. Curves correspond to two inoculum doses (5 g m^{-2} and 500 g m^{-2} of barley grain colonized by *P. violae*); 0–10 right scale corresponds to *ls* and *d* (*white symbols*), and 0–100 left scale to *i* and *tda* (*black symbols*)

was quite different: the number of smallest lesions increased over the whole epidemic, while the proportion of largest lesions was low and decreased in autumn (S4 to S6).

In 2001 and 2002 the proportion of small lesions (≤3 mm) decreased throughout the growth cycle, unlike the proportion of larger lesions (>5 mm), which increased (Fig. 3a and b). It was not possible to firmly establish that a given small lesion (e.g. ≤3 mm) enlarged (e.g. >5 mm) because new fresh CCS lesions could not be distinguished from older ones; however, the decrease in the number of lesions assigned to Δ_5 (]5; 10 mm]) between S6 and S7 was compensated by the increase in the number of lesions assigned to Δ_{10} (>10 mm; Fig. 3a). This suggests that lesions assigned to Δ_{10} in S7 could be those initially assigned to Δ_5 in S6, and that existing lesions may increase in size over time.

Pathometric relationships

Relationship between disease incidence i and total diseased area tda Fitting Eq. 7 to the field experiment data gave values of parameter $a(t)$ ranging from 0.012 to 0.067 (correlation coefficient R ranged from 0.659 to 0.940; $P<0.01$; Table 2). Figure 4 illustrates the relationship between *i* and *tda* at each sampling date in 2001, 2002 and 2003. For the survey and the controlled-conditions experimental data, $a=0.024$ ($R=0.893$; $P<0.01$) and $a=0.140$ ($R=0.929$; $P<0.01$), respectively. The correlation between *i* and *tda* was largely independent of the CCS intensity. A decreasing linear relationship between the parameter *a* and thermal time *t* was established for the field experiment in 2001 and 2002, with $\alpha = 3.3 \times 10^{-5}$ ($R=-0.974$; $P=0.045$) and $\alpha = 4.7 \times 10^{-5}$ ($R=-0.999$; $P=0.001$), respectively (Fig. 5). No similar decreasing linear relationship between *a* and *t* was found in 2003.

Relationship between total diseased area tda, lesion density d and lesion size ls Fitting Eq. 8 to the field experiment data gave values of parameter $c(t)$ ranging from 0.896 to 2.297 (correlation coefficient R ranged from 0.929 to 0.998 for the 3 years; $P<0.01$; Table 2); $c=1.238$ ($R=0.994$; $P<0.01$) for the survey data and $c=0.166$ ($R=0.954$; $P<0.01$) for the controlled-conditions experiment data. Contrary to $a(t)$, no significant linear relationship between *c* and *t* was established from the field experimental data.

Discussion

The four disease measurements (*i*, *d*, *ls*, and *tda*) allowed us to describe in full CCS symptoms and reveal epidemiological processes involved in CCS epidemics. From an empirical standpoint, an increase in disease incidence *i* concomitantly to a stabilisation of lesion density *d* suggests that CCS is spreading by alloinfection (root-to-root contamination). Conversely,

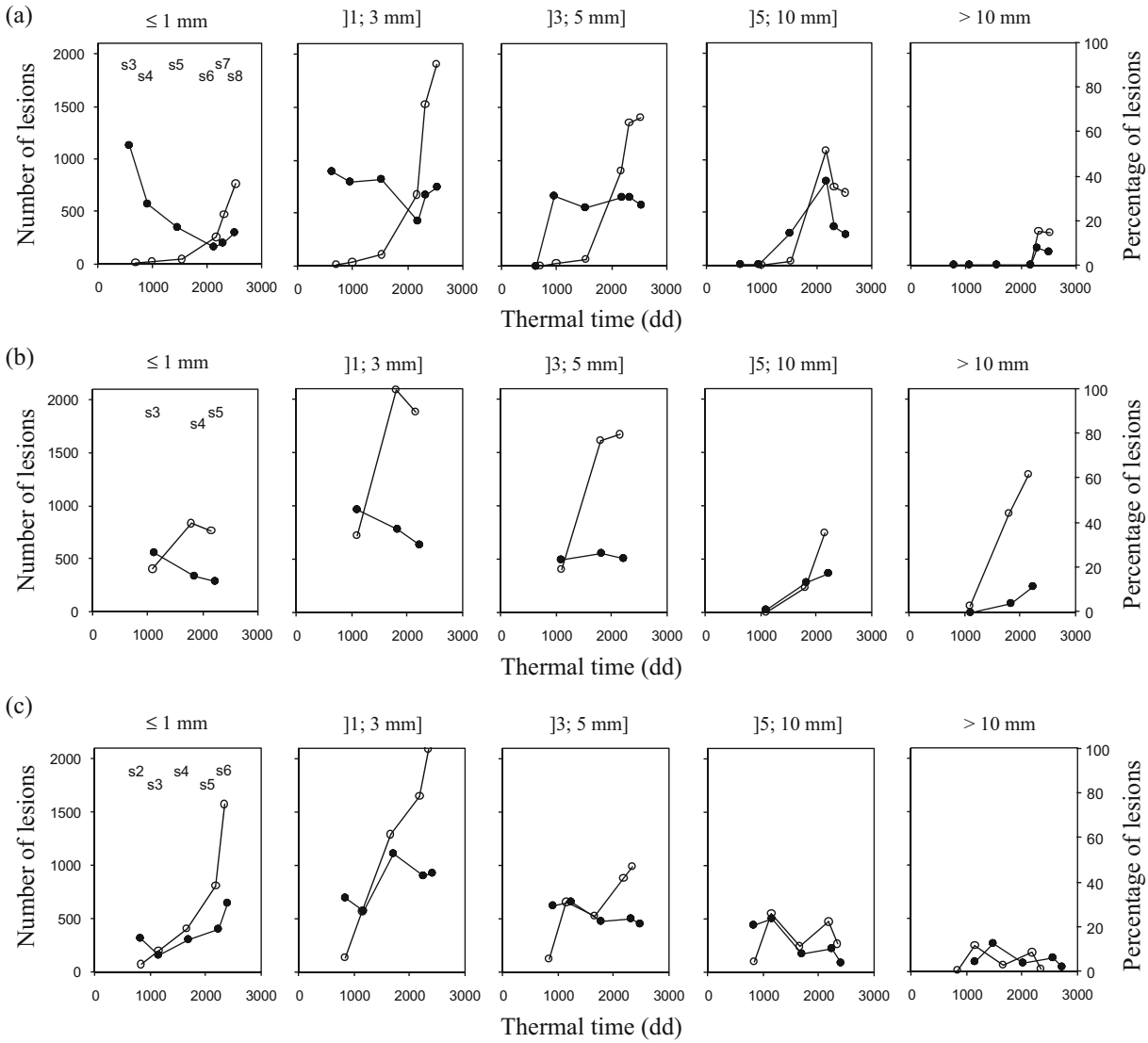

Fig. 3 Evolution in number and proportion of CCS lesions according to their mean diameter during three years of epidemic in the field experiment; **a** 2001, **b** 2002, **c** 2003. Horizontal scale is thermal time (accumulated degree days from sowing, base 3.5°C). *White circles* represent the number of lesions (extrapolated to 1,000 carrot roots) pertaining to the same cohort size, (left vertical scale); *black circles* represent the percentage of lesions pertaining to the same cohort size (right vertical scale). Curves were plotted with three inoculum doses pooled (5, 50, and 500 g m^{-2} of barley grain colonized by *P. violae*)

an increase in lesion density *d* concomitantly to a stabilisation of disease incidence *i* suggests that the disease becomes more intense at the root scale by autoinfection (contamination from a lesion on the same root). An invariability of lesion density *d* concomitantly to an increase in total diseased area *tda* suggests that the size of existing lesions was increasing, as expressed by Eq. 8. This is consistent with our analysis of a cohort of CCS lesions according to their size, which showed that a lesion may increase in size over time. As in the case of aerial plant diseases (Kranz 1988), we need to assume that each CCS lesion corresponds to a single initial infection, disregarding the occurrence of large lesions generated by the coalescence of the smallest (Suffert 2006). Changes in the cohort of CCS lesions according to their size were similar in 2001 and 2002, but differed in 2003: this suggests an impact of edaphic

Table 2 Relationship between disease incidence i and total diseased area tda, given by the equation $i=100(1-\exp(-a(t)tda))$ fitted to CCS measurements (from field experiment, survey, and controlled-conditions experiment), and relationship between total diseased area tda, lesion density d and lesion size ls, given by the equation $tda = c(t)\pi d(ls/2)^2$, where t is the thermal time (accumulated degree days from sowing, base 3.5°C)

Equation			$i=100(1-\exp(-a(t)tda))$		$tda = c(t)\pi d(ls/2)^2$		Size of sample
	Date	Accumulated degree days	a	R	c	R	
Field experiment							
2001							
S4	30/07/01	997	0.067	0.940*	0.957	0.991*	28/48
S5	03/09/01	1,535	0.058	0.798*	0.896	0.929*	40/48
S6	05/11/01	2,175	0.036	0.934*	1.047	0.995*	48/48
S7	08/01/02	2,325	0.021	0.930*	1.405	0.992*	48/48
S8	25/02/02	2,534	0.021	0.927*	1.440	0.985*	47/48
Pooled data			0.027	0.957*	1.257	0.979*	211
2002							
S3	12/08/02	1,087	0.067	0.808*	1.953	0.988*	56/57
S4	08/10/02	1,795	0.034	0.782*	2.204	0.998*	57/57
S5	02/12/02	2,162	0.017	0.738*	1.886	0.992*	57/57
Pooled data			0.046	0.863*	2.211	0.995*	170
2003							
S2	15/07/03	827	0.035	0.722*	2.297	0.992*	48/48
S3	04/08/03	1,154	0.012	0.659 *	1.968	0.991*	48/48
S4	02/09/03	1,663	0.038	0.819*	1.755	0.980*	48/48
S5	15/10/03	2,183	0.029	0.858*	2.197	0.996*	48/48
S6	09/12/03	2,351	0.039	0.747*	2.096	0.986*	48/48
Pooled data			0.028	0.775*	1.947	0.988*	240
Survey							
2003	15/01/03 to 30/01/03	2,000 to 2,200	0.024	0.893*	1.238	0.994*	55
Controlled-conditions experiment							
2001	–	1,160	0.140	0.929*	0.166	0.954*	10

Values of the parameters a and c were optimized by the method of minimisation of sums of squares; R is the correlation coefficient between experimental data and simulated data; size of sample is the number of rows with $i\neq0$ in the field experiment (maximum 48 in 2001 and 2003, and 57 in 2002), the number of trailers in the survey, and the number of pots in the controlled-conditions experiment.

*$P<0.01$

conditions on the processes involved in a CCS epidemic. It was recently confirmed that a deficit of soil moisture reduced primary infections in the field and promoted the healing of lesions, thereby limiting lesion expansion and the potential for alloinfections (Suffert et al. 2008).

The time-dependent parameter $a(t)$ in the pathometric relationship $i=100\cdot(1-\exp(-a(t)\cdot tda)$ decreased linearly over time in 2001 and 2002, but was constant in 2003. This difference can most likely be explained by the exceptionally hot and dry climatic conditions in summer 2003 (mean of mean daily temperatures in August of 21.5°C in 2003, in comparison to 18.9°C in 2001 and 17.5°C in 2002, and mean of maximal daily temperatures in August of 29.7°C in 2003, in comparison to 24.5°C in 2001 and 23.7°C in 2002). The decrease in $a(t)$ illustrated temporal changes in epidemiological processes involved in CCS epidemics (Fig. 6): parameter $a(t)$ decreased traditionally with increasing disease aggregation at the root population scale. In 2001 and 2002, the decrease of $a(t)$ over time illustrated the intensification of symptoms at the root scale ($a<0.04$; increase of d and tda), after the first phases of mobilisation of soil inoculum ($a>0.06$) and spread of disease ($0.04<a<0.06$; increase of i; Fig. 5). This hypothesis implies a random distribution of inoculum in the soil.

Hughes et al. (1997) formulated some theoretical relationships between disease measurements made at two levels in a spatial hierarchy. Willocquet and

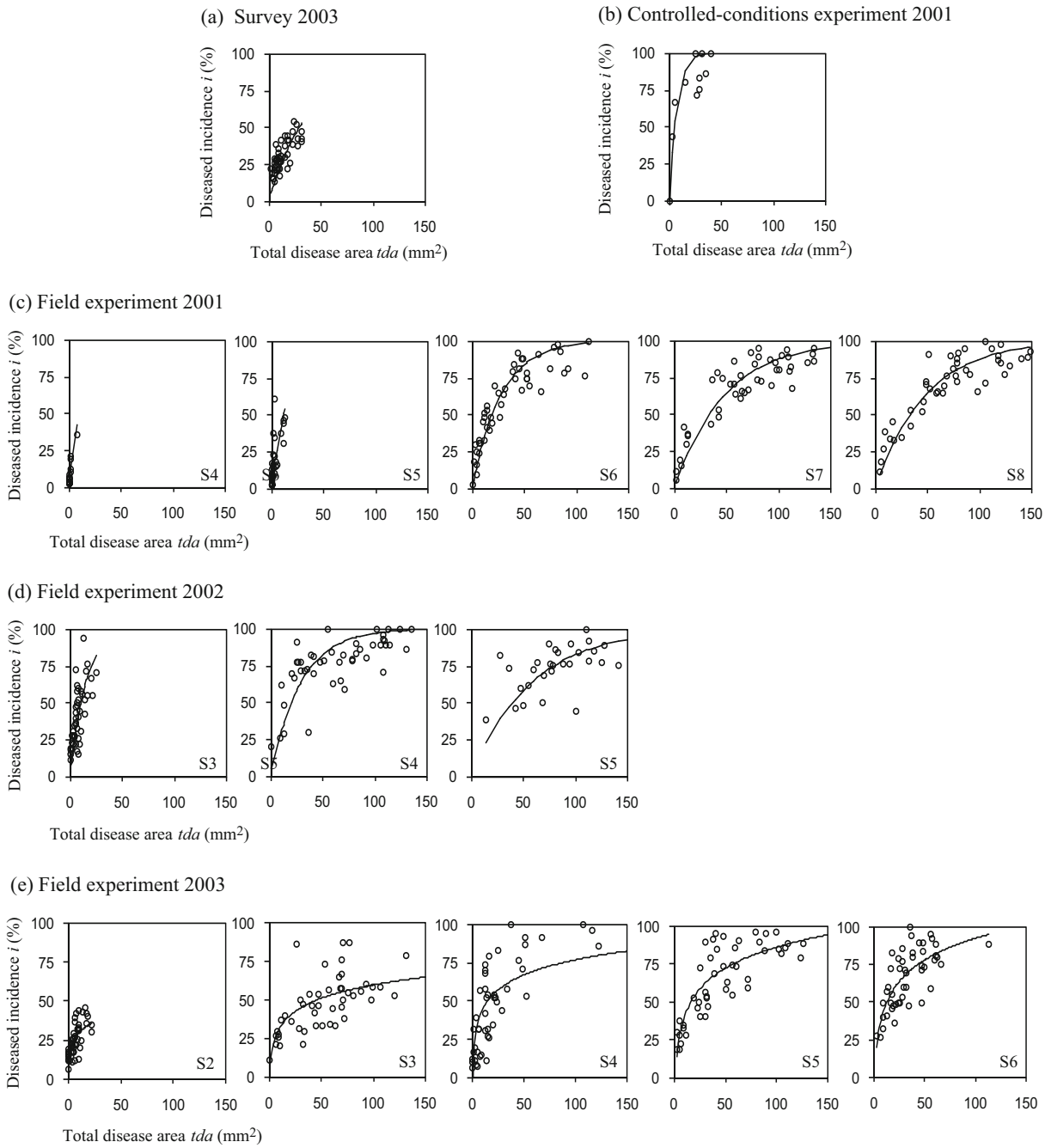

Fig. 4 Experimental relationship between disease incidence i and total diseased area tda given by the equation $i=100(1-\exp(-a(t)tda))$, where t is thermal time, fitted to CCS measurements from survey, controlled-conditions experiment, and field experiment (at each sampling date from S4 to S8 in 2001, from S3 to S5 in 2002, and from S2 to S6 in 2003; see Table 2)

Savary (2004) illustrated similar effects of the deposition pattern on the rate of aerial epidemics at different scales in a theoretical model: the relationships between disease incidence at a larger scale and disease incidence at a lower scale showed that the slope of the curves decreased as the ratios of allo-leaf- and allo-plant-deposition decreased. The slope of the incidence-severity curve increased with alloinfection processes, as well as when disease aggregation decreased: greater aggregation of disease was charac-

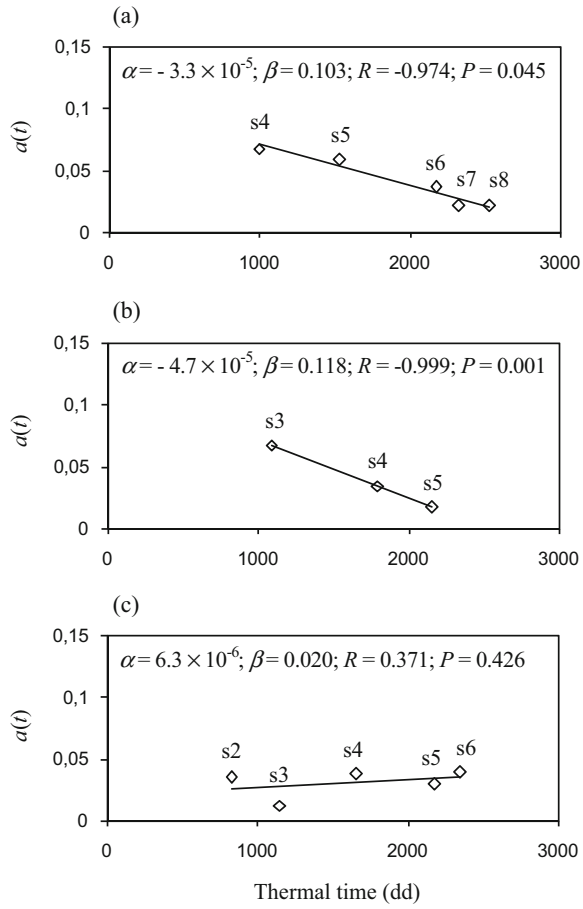

Fig. 5 Linear relationship between the parameter a and thermal time t given by the equation $a(t) = \alpha t + \beta$ fitted to data from three CCS epidemics from field experiment; **a** 2001, **b** 2002, **c** 2003

terised by a lower disease incidence at a given level of disease density (Hughes et al. 1997; McRoberts et al. 2003). Current and prior experimental results (Guba et al. 1961; Phelps et al. 1991) are consistent with theoretical data based on Eq. 7: the decrease in $a(t)$ over time and the increase in the aggregation of lesions on a root was probably caused first by the increase in the ratio secondary infection/primary infection, and secondly by the increase in the ratio autoinfection/alloinfection. The bilogistic model of Hau and Amorim (Hau et al. 1993) and the model of Brassett and Gilligan (1988), which back the existence of primary and secondary infections, were correctly fitted to CCS incidence progress curves (Suffert 2007). The occurrence of alloinfection by *P. violae* in CCS epidemics was verified by a soil infestation method, in which an artificially infected carrot taproot (donor) was transplanted close to healthy roots (receptors; Suffert and Montfort 2007; Suffert and Lucas 2008).

The commercially acceptable level of CCS is variable, can change during the harvesting season, and depends on market demand; consequently, producers need disease standards and quality indicators to adapt the quality of carrots to their harvesting date (and vice-versa) and to the market demand. The CCS measurements defined in this article also have a high epidemiological significance: roots with numerous and large lesions can generate a higher inoculum potential for secondary infections than roots with fewer and smaller lesions. These disease measurements have been used to establish the effects of different cropping practices on primary infection, on alloinfection (as assessed indirectly by i), on autoinfection (as assessed indirectly by d), and on lesion expansion (as measured by ls). Pathometric relationships could help to assess CCS field management options, especially by analysing the effect of some cropping factors on time-dependent parameters. Effects of timing of fungicide application and soil moisture content on $a(t)$ in Eq. 7 have been established with the prospect of an integrated disease management system based on a combination of different cropping practices (Suffert et al. 2008).

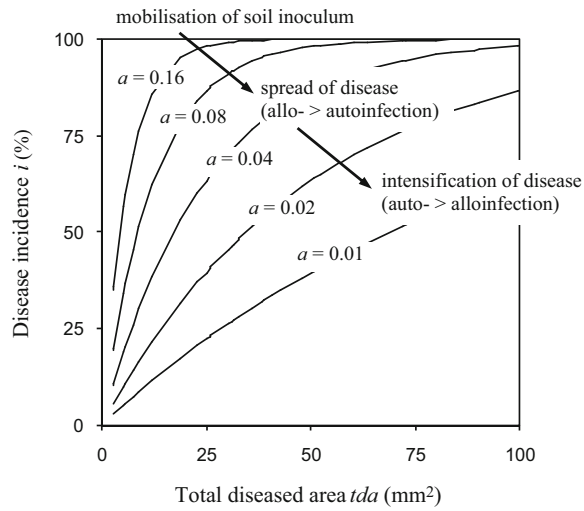

Fig. 6 Theoretical relationships between disease incidence i and total diseased area tda given by the equation $i=100(1-\exp(-a(t)tda))$, where t is thermal time. The temporal decrease of $a(t)$ suggests traditionally a change of epidemiological phases (McRoberts et al. 2003; Willocquet and Savary 2004). *Arrow* on the graph indicates increasing aggregation of CCS lesions at the root population scale

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