Epidemiological Analysis of Take-All Decline in Winter Wheat

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ABSTRACT

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Take-all dynamics within crops differing in cropping history (the number of previous consecutive wheat crops) were analyzed using an epidemiological model to determine the processes affected during take-all decline. The model includes terms for primary infection, secondary infection, inoculum decay, and root growth. The average rates of root production did not vary with cropping history. The force of primary infection increased from a low level in 1st wheat crops, to a maximum in 2nd to 4th wheat crops, and then to intermediate levels thereafter. The force of secondary infection was low but increased steadily during the season in first wheat crops, was delayed but rose and fell sharply in 2nd to 4th wheat crops, and for 5th and 7th wheat crops returned to similar dynamics as that for 1st wheat crops. Chemical seed treatment with silthiofam had no consistent effect on the take-all decline process. We conjecture that these results are consistent with (i) low levels of particulate inoculum prior to the first wheat crop leading to low levels of primary infection, low levels of secondary infection, and little disease suppression; (ii) net amplification of inoculum during the first wheat crop and intercrop period; (iii) increased levels of primary and secondary infection in subsequent crops, but higher levels of disease suppression; and (iv) an equilibrium between the pathogen and antagonist populations by the 5th wheat, reflected by lower overall rates of primary infection, secondary infection, disease suppression and hence, disease severity.

Take-all disease of winter wheat is caused by the soilborne fungal plant pathogen *Gaeumannomyces graminis* (Sacc.) Arx and Oliver. *tritici* Walker. There are no reliable sources of genetic resistance to the disease (25). Chemical control in the form of a seed treatment offers partial control of take-all (3,24). The action of silthiofam, one such treatment, is largely restricted to the protection of seminal roots during primary infection (3,24). It is possible however that such treatments may interfere with natural disease suppression during take-all decline.

Take-all decline, whereby disease builds from a low level in a first wheat crop to high levels in second, third, and sometimes fourth wheat crops, before returning to intermediate levels in subsequent crops, is one of the best known examples involving the natural suppression of a soilborne plant disease within a crop monoculture (4). Take-all decline is believed to evolve from the dynamical interactions between pathogen, host, and microbial antagonists in soil within and between a succession of consecutive winter wheat crops and is commonly perceived to be the result of a build-up in the natural antagonistic microbial population (7). The dynamics of take-all epidemics have been well described for single cropping seasons (1,24) involving consecutive phases of primary infection from particulate soil inoculum that survives intercrop periods, with multiple cycles of secondary infection as disease spreads from root to root. These processes lead to a disease progress curve in which the proportion of infected roots rises monotonically to an initial plateau that marks the end of primary infection and rises again thereafter as the epidemic is increasingly dominated by secondary infection (1).

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The pathogen survives the intercrop period as particulate soil inoculum that is formed from roots infected in the previous crop. The density and infectivity of the inoculum decays over time (14). It follows that the amount of primary and secondary infection in each successive crop depends upon the extent of infection in the previous crop, the survival of inoculum between crops and the activity of microbial antagonists (2). How the balance between primary infection and secondary infection changes during a sequence of successive wheat crops is not yet known.

In this paper, we analyze the development of take-all epidemics across a series of first, second, third, fourth, fifth, and seventh winter wheat crops. Specifically, we ask the following questions. (i) How do the processes of primary and secondary infection change during the growth of a sequence of consecutive wheat crops and the development of take-all decline? (ii) Are the changes in primary and secondary infection over a sequence of wheat crops affected by applications of the chemical seed treatment silthiofam?

MATERIALS AND METHODS

Experimentation. Data quantifying changes in the numbers of susceptible and infected roots over time for a series of consecutive wheat crops were obtained by assessment of epidemics of take-all in a field experiment performed at ADAS, Rosemaund in Herefordshire. The experiment was originally designed to analyze the effects of silthiofam on disease dynamics: the data are reanalyzed here to identify changes in epidemiological processes that may be associated with the phenomenon of take-all decline. The experimental site was on a deep, moisture retentive silty clay loam soil of the Bromyard series, representing moderate to high take-all risk. The experiment was initiated in 1997. A 1-year break from cereal cultivation was introduced in different years in order to generate a sequence of wheat crops with different cropping histories so that, in 2002, between one and six consecutive wheat

crops had been grown either with (+ silthiofam) or without (– silthiofam) seed treatment (Table 1). In 2003, to test for residual effects of silthiofam, plots were resown with winter wheat but plots previously treated with silthiofam were left untreated providing between one and seven consecutive wheat crops that had been grown either with or without previous seed treatment using silthiofam (Table 1). The plots had been sown to oilseed rape prior to continuous wheat cropping. Each treatment was replicated four times within a randomized block design, with a plot size of 3.5 by 24 m. In order to minimize soil and inoculum movement between plots during the course of the study the experimental area was ploughed across the direction of the plots and secondary cultivations done in the same direction as the plots. The wheat cultivar Equinox was used in all years. All treatments (rotation length × chemical control) received the same agronomic treatments according to good local practice. The fungicide Latitude (Montanto UK) was applied to plots receiving chemical treatment at a rate of 25 g of silthiofam per 100 kg of seed.

The numbers of infected (infected) and disease-free (susceptible) roots were assessed on six occasions in 2002 to 2003 on a replicated subset of plots chosen to represent each rotational sequence present in that year, again using stelar discoloration as a criterion for infection. Each take-all assessment was done on a sample of 20 to 25 plants/plot taken from each of four replicates of a 12 m length of the plot.

Modeling. For each plant, change in the number of susceptible (S) and infected (I) roots over time, t, (where t = degree-days > 4°C) was described by a compartmental model (1) referred to hereafter as the SIX model:

Susceptible roots
$$\frac{dS}{dt} = bN(t)\frac{(\kappa - N(t))}{\kappa} - (r_p X(t) + r_s(t)I(t))S(t) \quad (1)$$

Infected roots
$$\frac{dI}{dt} = (r_p X(t) + r_s(t)I(t))S(t) \quad (2)$$

Particulate inoculum
$$\frac{dX}{dt} = -r_d X(t) \quad (3)$$

Secondary infection
$$\frac{dr_s}{dt} = \alpha_s \exp(-0.5(\log(t/\gamma_s)/\beta_s)^2) \quad (4)$$

We assume that the total number of roots, N, increases logistically over time with a relative rate b, to a maximum, k, and where $N = S + I$ (equation 1). Disease is initiated by primary infection with rate, r_p (equation 2) from particulate inoculum, X, that decays exponentially with time at a rate, r_d (equation 3). Parameters for root production, κ and b, were estimated first and independently for each epidemic by summing S and I and hence fitting to trends in total root numbers (N) over time. The parameters κ and b were fixed at the estimated values for each epidemic during subsequent estimation of the epidemiological parameters (r_p, α_s, β_s, and γ_s). The precise times at which epidemics began are not well defined by these data. However previous experimentation suggests that field epidemics are initiated approximately twenty days after sowing and that particulate inoculum is largely exhausted by the spring (3). Moreover, the density of particulate inoculum, X, at the onset of each epidemic was not estimated experimentally and, for the purpose of modeling, is fixed at 1.0. Consequently, the SIX model (equation 1) was fitted (using Facsimile, MCPS Software Ltd., UK) to the monoculture data assuming all epidemics began at 200 degree-days and with a fixed decay, r_d, for inoculum, X, that led to less than 5% of particulate inoculum remaining at 800 degree-days. This means that any differences between epidemics with respect to the initial density or the decay of particulate inoculum are subsumed within the rate of primary infection, r_p. The pathogen spreads by secondary infection from infected to susceptible roots with rate r_s. Following preliminary analysis of the incremental changes in numbers of infected roots over time in individual epidemics, a log-normal model for r_s (equation 4) was used to accommodate a delay in the onset of secondary infection, followed by a rise and subsequent decrease in the rate of secondary infection over time. The parameters, α_s, β_s, and γ_s, of the log-normal function define the maximum, width and location of curve describing r_s over time (Table 2). Hence, four parameters, were estimated on final fitting, r_p, α_s, β_s, and γ_s, and as a measure of disease severity, change in the proportion of infected roots per plant was calculated as $I/(I + S)$. Disease progress curves are interpreted according to changes in the relative contributions of primary and secondary infection given by terms $r_p X(t)$ and $r_s(t)I(t)$, which represent the force of infection for each mode of transmission respectively.

TABLE 1. History of field plots used for the collection of wheat monoculture data in 2003[a]

1997	1998	1999	2000	2001	2002	2003
ww1	ww2+	ww3+	ww4+	ww5+	ww6+	ww7
ww1	ww2–	ww3–	ww4–	ww5–	ww6–	ww7
ww1	ww2+	ww3+	ww4+	osr	ww1+	ww2
ww1	ww2–	ww3–	ww4–	osr	ww1–	ww2
ww1	ww2+	ww3+	osr	ww1+	ww2+	ww3
ww1	ww2–	ww3–	osr	ww1–	ww2–	ww3
ww1	ww2+	osr	ww1+	ww2+	ww3+	ww4
ww1	ww2–	osr	ww1–	ww2–	ww3–	ww4
ww1	osr	ww1+	ww2+	ww3+	ww4+	ww5
ww1	osr	ww1–	ww2–	ww3–	ww4–	ww5
ww1	ww2+	osr	ww1+	ww2+	osr	ww1
ww1	ww2–	osr	ww1–	ww2–	osr	ww1

[a] ww = winter wheat; osr = oilseed rape; and + = treated with silthiofam.

RESULTS

The epidemics of a wheat monoculture were well-described by the SIX model showing clear trends with respect to disease progression (Figs. 1 and 2). We summarize here the overriding epidemiological trends detected during wheat-crop monoculture. The final levels of disease (number or proportion of infected roots) did not vary much amongst wheat crops with different cropping history (Fig. 1) but the dynamics of disease progression were significantly affected: disease progress curves were characterized by rapid but short increases in disease in the 3rd and 4th wheat crops (Fig. 1C, D, I, and J), compared with a longer and more gradual development of disease in the other crops (Fig. 1A, B, E, F, G, H, K, and L). The rate of primary infection, r_p, and the timing, α_s, and maximum rate, γ_s, of secondary infection all increased to a maximum in epidemics of 3rd and 4th wheat crops before returning to intermediate levels in a 7th wheat crop (Fig. 3, Table 3). The opposite trend was observed for the duration of

TABLE 2. List of variables, parameters, and their meanings for the SIX model describing an epidemic of take-all in a single wheat crop

	Meaning
Variable	
S	Number of susceptible roots per plant
I	Number of infected roots per plant
X	Number of inoculum units per plant
r_s	Rate of secondary infection
Parameter	
b	Rate of root production
k	Maximum number of roots per plant
r_p	Rate of primary infection
α_s	Maximum rate of secondary infection
β_s	Duration of secondary infection
γ_s	Time of the maximum rate of secondary infection

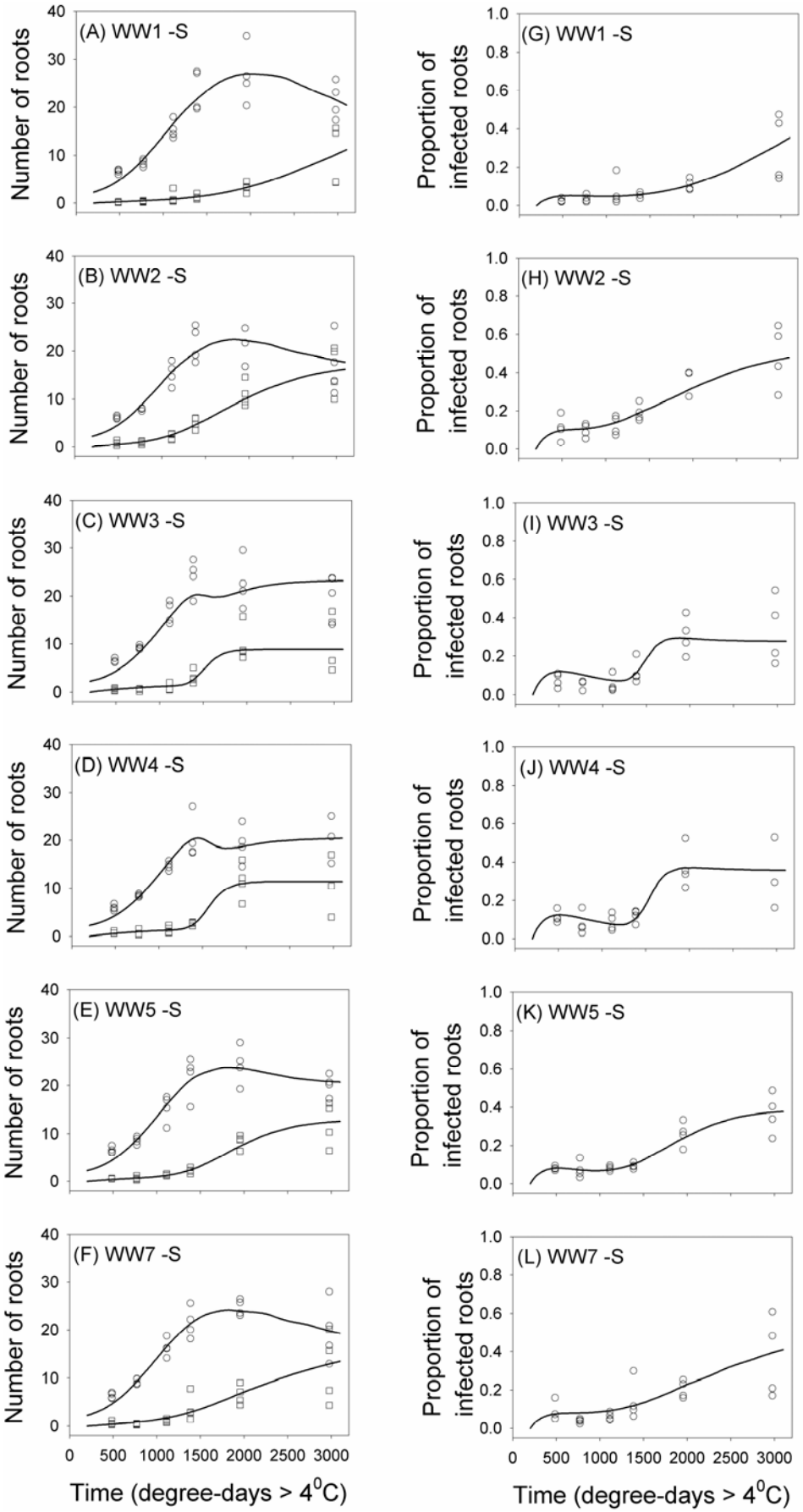

Fig. 1. Change in **A to F,** the number of infected (open squares) and number of susceptible roots (open circles) over time and **G to L,** the proportion of infected roots over time for untreated 1st to 7th wheat crops. Solid lines indicate the fit to data using the SIX model. Each square or circle represents the mean for 20 to 25 plants.

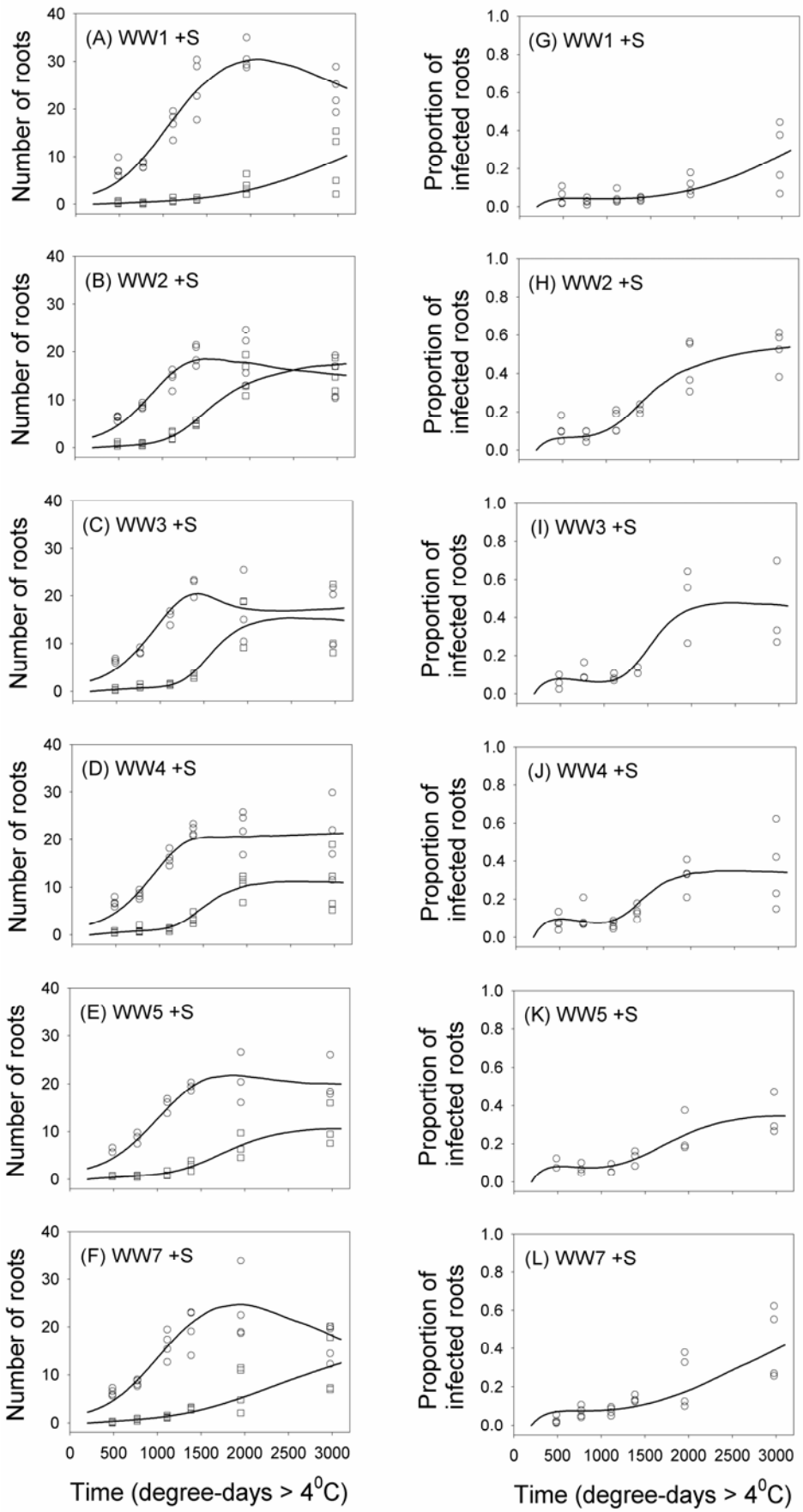

Fig. 2. Change in **A to F,** the number of infected (open squares) and number of susceptible roots (open circles) over time and **G to L,** the proportion of infected roots over time for 1st to 7th wheat crops treated with silthiofam. Solid lines indicate the fit to data using the SIX model. Each square or circle represents the mean for 20 to 25 plants.

secondary infection, β_s. This meant that the force of secondary infection switched from a steady increase in a 1st wheat crop to a delayed but intense phase of secondary infection in 2nd, 3rd, 4th, and 5th wheat crops and back again in 7th wheat crops (Fig. 4). This, in turn, accounts for disease progress curves describing changes in the numbers of infected roots over time that were almost sigmoidal in shape during the 1st, 2nd, 5th, and 7th wheat crops but clearly monomolecular during the primary infection phase and sigmoidal thereafter in 3rd and 4th wheat crops (Fig. 1A to F and Fig. 2A to F). Similarly, in 1st, 2nd, 5th, and 7th wheat crops the proportion of infected roots increased to a plateau following primary infection but produced a clear peak in the epidemics of 3rd and 4th wheat crops (Fig. 1G to L and Fig. 2G to L).

Previous applications of the chemical seed treatment, silthiofam, had no consistent effect on epidemic behavior (Fig. 2), on the force of primary or secondary infection (Fig. 4) or on their underlying parameters (Fig. 3, Table 3) although a marginal trend towards decreased primary infection was noted (Fig. 3A).

DISCUSSION

An epidemiological analysis of the dynamics of take-all within crops differing in cropping history (the number of previous consecutive wheat crops) is used here to examine the long-term effects caused by previous treatments with silthiofam. In particular, we were aiming at identifying changes in the contributions of primary and secondary infection and from these to infer changes in the net production and death of inoculum as well as the likely effects of disease suppression. Although the phenomenon of take-all decline has been long known (10,12), this is the first quantitative analysis of the underlying epidemiological processes.

Despite relatively few observations during the primary phase of the epidemic (from 200 to 400 degree-days), the data provided good resolution with which to identify the form of epidemiological model well-suited to describing the disease dynamics of take-all in a wheat monoculture. Here, as previously (3), we assumed a rapid decay in inoculum, *X*, consistent with field observations and responsible for a steady reduction in the contribution of primary infection during the course of the epidemic (1,14,26). This, in combination with the diluting effect of healthy root production, accounts for the initial plateau in the proportion of infected roots over time. It would not be possible to describe the disease progression in all crops using a model with a fixed rate of secondary infection (1). Such a model describes disease progression that is asymptotic towards 100% disease, equivalent to all roots becoming infected, and with secondary infection that begins at the onset of the epidemic. Whilst previous analysis, with a fixed rate for secondary infection, served well for improving our understanding of take-all epidemiology (1,3), here we propose that a rise and fall in the rate of secondary infection, arbitrarily defined by a log-normal function, is more appropriate. This, in-turn, allows more accurate estimates of primary infection and thus the comparison of primary and secondary infection during a sequence of epidemics in successive wheat crops. The log-normal

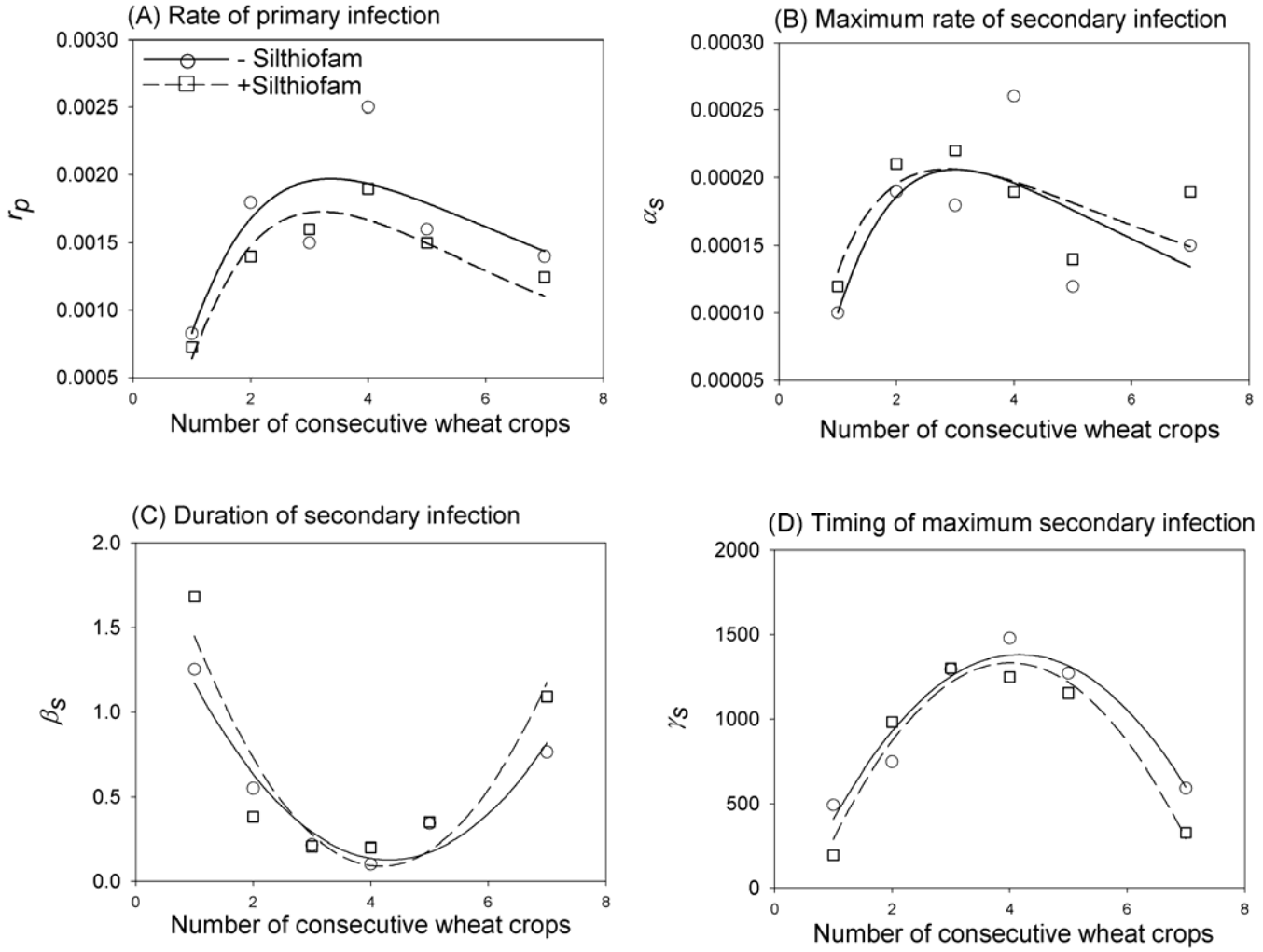

Fig. 3. Change in parameter estimates for **A,** the rate of primary infection, r_p, **B,** the maximum, α_s, **C,** the duration, β_s, and **D,** the timing of the maximum, γ_s, rate of secondary infection with numbers of consecutive wheat crops either with (squares, broken line) or without (circles, solid line) a previous history of silthiofam treatment.

function for secondary infection has previously been used by Otten et al. (19) to summarize the dynamics of secondary infection of radish (*Raphanus sativus*) by *Rhizoctonia solani*. Otten et al. (19) proposed several mechanisms to account for the nonmonotonic (log-normal) rate of secondary infection. These represent a latent period, an increase in infectivity as hosts grow and become a more potent source of infection, synergism (where transmission from an infected host is enhanced by another infected plant) or transmission beyond nearest neighbor. We do not know the precise mechanisms for a root pathogen such as *G. graminis* but a delay in the onset of secondary infection may conceivably be due to changes in root growth that affect the mixing between susceptible and infected roots and hence, the transmission of infection. Similarly, host and environmental factors may be responsible for a progressive reduction in the rate of secondary infection whereby changes in the susceptibility of the root (16) or in the soil environment (e.g., drying of the upper soil horizons, in which the pathogen is normally most active) may slow the transmission of the pathogen. Of these, soil microbial antagonism, already widely cited as a possible cause of take-all decline (5,6, 8,9,13,15,21–23,27), is a likely candidate.

Fitting the epidemiological model to disease data revealed clear trends in parameters associated with primary infection and secondary infection for a sequence of epidemics over the course of seven consecutive wheat crops (Fig. 3) and in changes of their contribution during each epidemic (Fig. 4). The low initial rates of primary infection detected in 1st wheat crops are consistent with low levels of particulate inoculum preceding the crop, whilst the low but steadily increasing force of secondary infection may reflect the presence of few initial disease foci and heterogeneous mixing for a pathogen with limited dispersal that is suffering no noticeable effects of disease suppression. Indeed, the wide confidence intervals estimated for parameters of secondary infection for 1st wheat crops suggest little evidence of a rise and fall. That little evidence of disease suppression could be detected in 1st wheat crops is consistent with the hypothesis that take-all decline is a consequence of the build-up of antagonists over a sequence of wheat crops (20). In particular, the continuing upward trend in disease progression at the time of harvest of a 1st wheat crop may result in the production of inoculum within the decaying root system even after harvest and account for higher levels of primary infection in 2nd wheat crops.

Higher estimates for primary infection and delayed, shorter periods of secondary infection, albeit at a higher level, in epidemics of 2nd to 4th wheat crops are consistent with amplification and carry-over of particulate inoculum from the first wheat crop and the onset of disease suppression. The occurrence of a significant delay in the onset of infection, together with a shorter period of secondary infection in 2nd to 4th wheat crops suggests that, at this stage in the cropping sequence, the antagonist population has achieved densities and levels of dissemination that allow it to affect the initiation of take-all lesions. A key question, at this point, concerns the mechanism for the suppression of secondary infection. This could be due to a reduction in the probability of lesion initiation or to subsequent lesion extension. The first equates with a reduction in root susceptibility, the second with a net reduction in lesion infectivity. The behavior of the antagonist population as either generalists (able to grow on root exudates and to reduce the susceptibility of a disease-free root), or specialists (requiring interaction with the pathogen and able only to reduce the infectivity of an already infected root) has important implications for the persistence and management of the antagonist population in long-term wheat cropping. Previous work showing that antagonists have no effect on the numbers of lesions (8) suggests a specific relationship between antagonists and the pathogen or disease expression. In our analysis, the rate of primary infection reflects both the quantity and infectivity of particulate inoculum. Either or both of these may well be affected by the behavior of the antagonist population between wheat crops, which means that the high levels of inoculum produced during the growth of a 2nd, 3rd, or 4th wheat crop may be subject to colonization by antagonists and hence be less capable of infecting the next wheat crop. Earlier work suggests that take-all decline does not result from loss of pathogenicity in the population of *G. graminis* (6).

Finally, the reduced primary infection in 5th and 7th wheat crops is most likely the result of restricted amplification of inoculum by secondary infection in the preceding 2nd, 3rd, and 4th wheat crops whilst the increased duration of secondary infection suggests an equilibrium between disease and its suppression

TABLE 3. Parameter estimates (average ±95% confidence intervals) associated with root growth, b and k, and primary, r_p, and secondary, α_s, β_s, and γ_s, infection obtained from fitting sequentially (i.e., first estimating parameters for root growth and then estimating parameters for the spread of disease) the SIX model to take-all epidemics at different stages of rotation in a wheat monoculture (NB, the rate of inoculum decay; r_d is set to 0.0374 for all crops)[a]

Treatment	Wheat crop	b	k ($\times 10^3$)	r_p ($\times 10^2$)	α_s ($\times 10^3$)	β_s	γ_s
– silthiofam	WW1	31.64	0.100	0.083	0.10	1.25	492.7*
		(29.16–34.33)	(0.088–0.115)	(0.014–0.50)	(0.01–1.00)	(0.21–7.27)	
	WW2	34.05	0.093	0.180	0.19	0.55	744.1
		(31.72–36.55)	(0.084–0.104)	(0.078–0.41)	(0.13–0.28)	(0.35–0.87)	(586.8–943.3)
	WW3	32.65	0.100	0.148	0.18	0.21	1290.2
		(29.03–36.72)	(0.080–0.125)	(0.022–1.00)	(0.08–0.42)	(0.08–0.50)	(920.1–1809.3)
	WW4	32.03	0.092	0.246	0.26	0.10	1479.5
		(31.26–32.80)	(0.091–0.093)	(0.091–0.67)	(0.03–2.28)	(0.01–0.87)	(1272.5–1720.0)
	WW5	34.28	0.089	0.156	0.12	0.34	1266.9
		(32.22–36.49)	(0.080–0.098)	(0.041–0.60)	(0.05–0.27)	(0.18–0.62)	(761.2–2108.3)
	WW7	33.16	0.098	0.140	0.15	0.76	607.0*
		(30.89–35.60)	(0.088–0.109)	(0.086–0.23)	(0.13–0.18)	(0.65–0.88)	
+ silthiofam	WW1	34.69	0.090	0.073	0.12*	1.68	197.5*
		(31.94–37.68)	(0.077–0.105)	(0.029–0.18)		(1.44–1.97)	
	WW2	34.05	0.093	0.137	0.21	0.38	981.3
		(31.71–36.55)	(0.084–0.104)	(0.012–1.56)	(0.05–0.96)	(0.19–0.76)	(413.5–2328.7)
	WW3	32.48	0.099	0.158	0.22	0.21	1296.6
		(28.67–36.79)	(0.081–0.012)	(0.011–2.21)	(0.08–0.59)	(0.08–0.55)	(879.3–1911.9)
	WW4	32.48	0.097	0.186	0.19	0.20	1244.5
		(28.35–37.20)	(0.076–0.124)	(0.043–0.80)	(0.11–0.34)	(0.08–0.52)	(914.6–1693.4)
	WW5	34.28	0.089	0.153	0.14	0.35	1151.0
		(32.21–36.49)	(0.080–0.098)	(0.014–1.61)	(0.03–0.64)	(0.10–1.22)	(400.1–3311.0)
	WW7	31.58	0.102	0.125	0.19	1.09	327.2*
		(28.53–34.95)	(0.085–0.123)	(0.013–1.23)	(0.02–1.91)	(0.53–2.25)	

[a] * Indicates that parameter is not well determined.

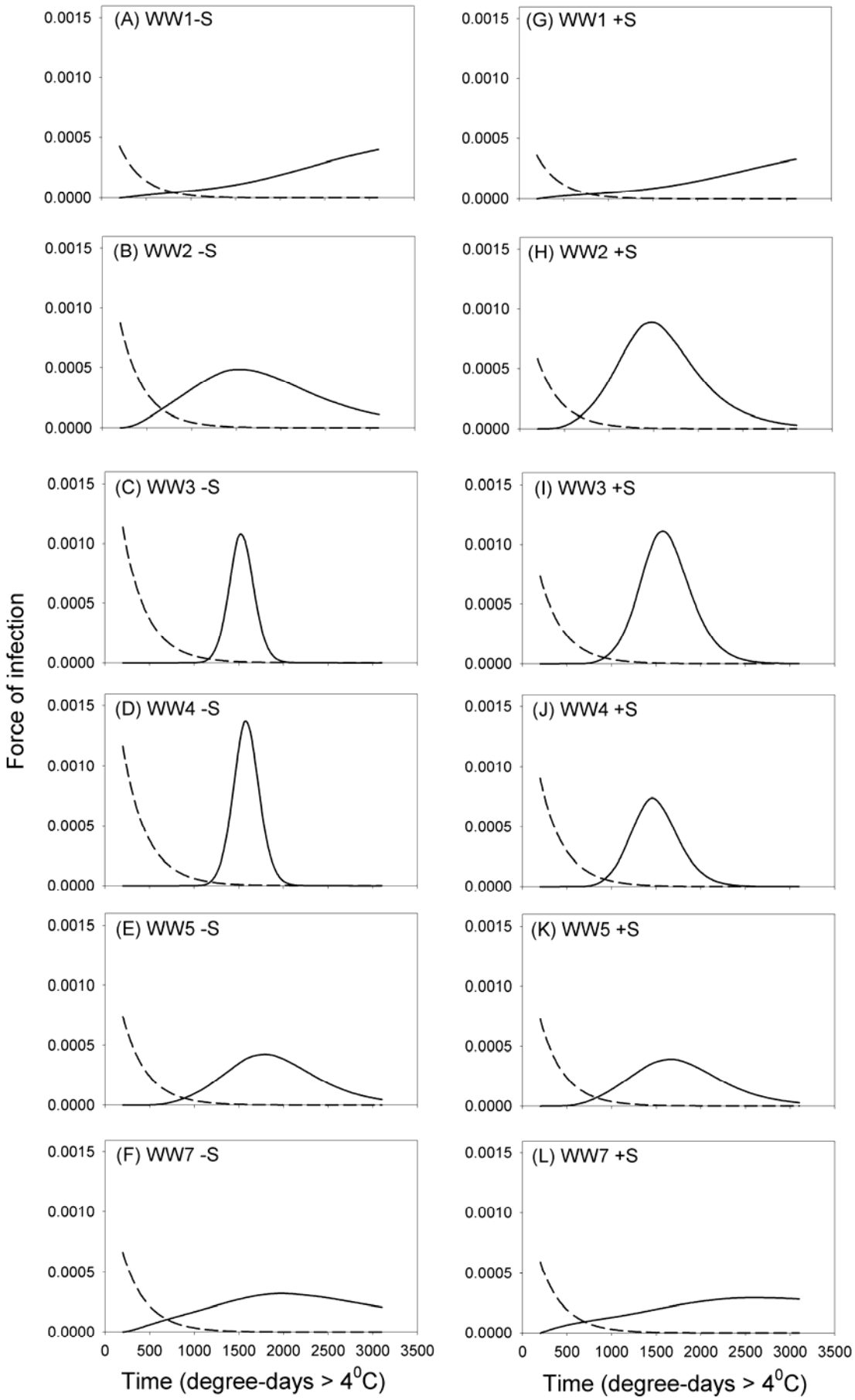

Fig. 4. Change in r_pX, the force of primary infection (broken lines) and r_sI, the force of secondary infection (solid lines) over time derived from fitting the SIX model to the 2002-2003 epidemics of 1st to 7th wheat crops either **A to F,** with or **G to L,** without a previous history of silthiofam treatment.

by the antagonist population. More recently, however, studies of the effect of cropping history on changing pathotype frequency, whereby first wheat crops are dominated by less aggressive pathotypes, second, third and fourth wheat crops by more aggressive pathotypes, and subsequent crops again by higher frequencies of the less aggressive pathotypes (11,17,18), suggest that changes in the genetic structure of the population may also contribute to take-all decline.

The treatment of previous wheat crops with silthiofam had no consistent effect on primary and secondary infection and did not prevent take-all decline. Indeed, if the action of the antagonist population is to respond to take-all infection (i.e., that the antagonist populations build-up in direct response to the development of a take-all lesion) and is thus restricted to the control of secondary, root to root, spread, then we might conjecture that silthiofam, being itself restricted to the control of primary infection (3), provides a complementary treatment against take-all disease.

In conclusion, by using a mechanistic model to quantify changes in the balance of primary and secondary infection within epidemics that occur in a sequence of wheat crops, this work provides an important analysis of the underlying dynamics of inoculum and, by inference, of changes in the antagonist population during wheat monoculture.

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