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Running title: *The effect of fungicides on disease-induced yield loss in wheat*

A model of the effect of fungicides on disease-induced yield loss, for use in wheat disease management decision support systems

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Abstract

A model of the effect of foliar-applied fungicides on disease-induced yield loss is described, parameterised and tested. The effects of fungicides on epidemics of *Septoria tritici* (leaf blotch), *Puccinia striiformis* (yellow rust), *Blumeria graminis* f.sp *tritici* (powdery mildew) and *Puccinia triticina* (brown rust) on winter wheat were simulated using dose-response curve parameters. Where two or more active substances were applied together, their joint action was estimated using an additive dose model where the actives were of the same mode of action or a multiplicative survival model where the modes of action differed. By coupling the model with models of wheat canopy growth and foliar disease published previously, it was possible to estimate disease-induced yield loss for a prescribed fungicide programme. The difference in green canopy area, and hence interception of photosynthetically active radiation, between simulated undiseased and diseased (but treated) crop canopies was used to estimate yield-loss. The model was tested against data from field experiments across a range of sites, seasons and wheat cultivars, and was shown to predict the observed disease-induced yield loss with sufficient accuracy to support fungicide treatment decisions. A simple method of accounting for uncertainty in the predictions of yield loss is described. Fungicide product, dose and spray timing combinations selected using the coupled models responded appropriately to disease pressure and cultivar disease resistance.

Key Words: Winter wheat; foliar diseases; fungicides; yield-loss

Introduction

Fungicide treatment decisions are complex because there are thousands of possible fungicide product, dose and spray timing combinations which could be used, and product, dose and timing interact in their effects on disease-induced yield loss (Paveley 1999; Paveley *et al.* 2000; Bartlett *et al.*, 2002). Here we propose a model which, when coupled with foliar disease and crop canopy models published previously (Milne *et al.* 2003; Audsley *et al.*, 2005), describes the effect of foliar fungicide treatment on epidemic development, loss of green canopy area, absorption of insolation, and yield loss for a given scenario; where the scenario specifies sowing date, daily weather, cultivar, and fungicide program. Because yield-loss has an economic value and fungicides have an economic cost, the models can be used to rank treatment options by net economic benefit, as part of a decision support system (DSS) for crop managers. This use imposes certain requirements and constraints on model structure, considered below.

The model describes the effects of active substances, thus reducing substantially the task of parameterising fungicide efficacy, because the hundreds of commercially available fungicide products are formulated from approximately 30 active substances (either alone or in mixtures). Hence, a simplifying assumption is made here that the effects of differences in formulation on efficacy are small in comparison with the differences between active substance.

The biokinetics of different active substances vary substantially, even within the same class of fungicides (Bartlett *et al*., 2002), however, this variation can be broadly categorised as two types of effect on foliar disease development. Protectant effects reduce infection frequency, often by inhibiting spore germination and germ tube growth. Non-systemic active substances are predominantly protectant and therefore have to be applied soon after leaf emergence, before infections occur (Russell, 2005). Eradicant effects slow the rate of mycelial growth and hence the rate of formation of sporulating structures (Vyas, 1984). Many systemic fungicides have both protectant and eradicant properties, although some - for example the ergosterol biosynthesis inhibitors - have little effect on spore germination, as the pathogen obtains a supply of ergosterol or its precursors from reserves within the spore (Hanssler & Kuck, 1987). Eradicant fungicides can still be effective when applied after infection, so the best application timing depends on the active substance. To simulate these different effects of timing on efficacy, protectant and eradicant effects need to be modelled for each active substance.

Fungicides have a recommended maximum dose which must not be exceeded. However, because the cost of treatment rises linearly with dose, whereas efficacy is non-linearly related to dose, it is often cost effective to apply less than the recommended dose; particularly when inoculum pressure is low and rate-limiting disease resistance is constraining the epidemic. Hence, active substance efficacy needs to be modelled using representative dose-response curves.

For Type II pathosystems (Johnson, 1987), which cause crop damage (Nutter *et al*., 1993) predominantly through loss of green canopy area, the slopes of the relationships of yield on healthy area duration (HAD) or healthy area absorption of insolation (HAA) are reasonably consistent (Waggoner & Berger, 1987; Bryson *et al.*, 1997; Paveley,

1997). Hence, despite the difficulties of predicting yield *per se* (Landau *et al.*, 1999; Jamieson *et al.*, 1999), there is a reasonable prospect of predicting disease-induced yield loss, where disease and the effect of fungicides on disease, change HAD and HAA.

Given the uncertainty which surrounds any prediction, it would be unwise to make decisions on fungicide use solely on the basis of deterministic estimates of yield-loss. The unpredictability of weather means that future outcomes are variable. Ideally the coupled models should be run over a large number of future weather scenarios in order to estimate the resulting yield-loss variability. However, in a decision support application the models may need to be run thousands of times in a decision algorithm (Parsons & Te Beest, 2004), so a simple method to estimate yield-loss variability is proposed in order to minimise run time.

Model structure

The model reported here couples with canopy and disease simulation models reported in previous papers (Milne *et al,*. 2003; Audsley *et al.*, 2005). In summary, the canopy model simulates the growth and senescence of successive leaves on a single shoot as a function of day length and thermal time. Culm leaves are numbered down the shoot, so the flag leaf is leaf I, the next leaf down is leaf II, and so on. The foliar disease model simulates epidemics on the top six leaf layers. Disease is described as a series of daily infections which results in infectious lesions. The success of infection is dependent on pathogen species dependent weather variables such as rainfall, relative humidity and temperature. Lesion growth from an infection is modelled by a logistic function of thermal time.

Fungicide efficacy

Commercial fungicide products are formulated from one or more chemical active substances. Fungicide efficacy is modelled by simulating the effect of each active substance and then combining them.

Active substance model

The protectant effect of an active substance is simulated by reducing the number of successful infections on a day to a proportion R_P. The eradicant effect is simulated by reducing the rate of growth of the infection to R_E. The logistic equation that describes the growth rate of a lesion from an infection (Audsley *et al.*, 2005) becomes

$$\frac{dy}{dT} = R_E k y(y-1) \text{ with } y(0) = a \qquad (1)$$

where y is lesion size, expressed as a proportion of its asymptote value, at thermal time T (°C days, base zero). Parameters a and k are disease dependant. The percentage symptom severity on a leaf on day t becomes

$$Y(t,n) = \sum_{i=1}^{t} P(i,n) R_P(i)\, y(T(t) - T(i)) \qquad (2)$$

where *P(i,n)* is the number of potential infections occurring on day *i* on leaf *n*, *T(t)-T(i)* is the thermal time accumulated over the period *i* to *t*.

Parameters $R_P(t)$ and $R_E(t)$ are calculated daily for each leaf layer from dose-response curves

$$R_P(t) = 1 - a_P\left(1 - \exp(-k_P d(t))\right) \text{ and } R_E(t) = 1 - a_E\left(1 - \exp(-k_E d(t))\right) \qquad (3)$$

where a_P, k_P, a_E and k_E are parameters dependent on the active substance and the disease that is being targeted and $d(t)$ is the effective daily dose, calculated as follows.

When a spray is applied, the dose D_n arriving on leaf n is a function of leaf position in the canopy and the extent to which it has emerged. This is modelled

$$D_n = D_0 A_n(t_s) \exp\left(-\tau \sum_{j=1}^{n-1} L_j(t_s)\right) \qquad (4)$$

where D_0 is the applied dose of the active substance, $A_n(t_s)$ is the proportion of leaf n that has emerged on the date of application t_s, $L_j(t_s)$ is the leaf area index (LAI) of leaf j on day t_s and τ is a constant parameter. Over time the efficacy of an applied active substance will decay. On day t, the effective daily dose $d(t)$ on leaf n is given by

$$d(t) = D_n \exp(-\mu(t - t_s)) \qquad (5)$$

where μ is the decay rate of the active substance.

Active substance mixtures

Mixtures of active substances can arise in two ways. Firstly, two or more active substances can be applied together, either within a single fungicide product formulation or as a tank mix of products. Secondly, fungicides may be applied in sequence and active substances from one application may still be present when the next is applied.

An additive dose model (ADM) is used to calculate the joint action of active substances from the same mode of action group of fungicides and a multiplicative survival model (MSM) for active substances with different modes of action (Morse, 1978).

Additive dose model: Consider combining the efficacy of the m active substances c_1, c_2, … c_m against a given disease on day t at effective doses of $d_1(t)$, $d_2(t)$, … $d_m(t)$ respectively. Firstly, the active substance with the greatest activity at full dose is identified; say active substance c_1. Using Eqn 3, for every other active substance calculate the effective daily dose $\hat{d}_j(t)$ of active substance c_1 that will give the same efficacy as active substance c_j, at effective dose d_j, where $j \in \{2,...m\}$. This is given by

$$\hat{d}_j(t) = -\frac{1}{k(1)}\ln\left(1-\frac{a(j)}{a(1)}\left(1-\exp\left(-k(j)d_j(t)\right)\right)\right) \quad (6)$$

where $a(j)$ and $k(j)$ are parameters of active substance c_j where $j \in \{1,...m\}$. The doses in terms of active substance c_1 are calculated for each of the active substances and summed together giving a combined effective dose for day *t.* This dose is substituted into Eqn 3 to give the combined efficacy $\hat{R}(t)$ for the *m* active substances

$$\hat{R}(t) = 1 - a(1)\left(1-\exp\left(-k(1)\sum_{j=2}^{m}\hat{d}_j(t) + d_1(t)\right)\right) \quad (7)$$

Multiplicative survival model: Active substances with different modes of action are assumed to work independently. Each active substance can be thought of as working on the pathogen population that has survived the effect of the other active substances. Therefore the combined efficacy $\hat{R}(t)$ of *m* active substances c_1, c_2, … c_m against a given disease on day *t* at effective doses of $d_1(t)$, $d_2(t)$, … $d_m(t)$ respectively is given by

$$\hat{R}(t) = \prod_{j=1}^{m} R(t;\, j) \quad (8)$$

where R(*t*; *j*) is given by $1 - a(j)\left(1-\exp(-k(j)d_j(t))\right)$ from Eqn 3.

Accounting for the variation in shoot development

The wheat canopy is modelled by considering a typical wheat shoot, where a leaf on the stem represents a layer of leaves over a field (Milne *et al.* 2003). In reality, tillers develop at slightly different rates, initially lagging behind the main stem, but tending to become more synchronous as the crop matures. Hence, a spray applied early in the canopy expansion will be intercepted by leaves of a given layer that vary more in age than those that would intercept a spray applied at later growth stages. This is likely to affect the performance of fungicides, in particular those with protectant effects because, for example, leaves which emerged earlier may already have become infected at the time of treatment. This effect is approximated by determining a parameter to reduce the protectant spray effect according to the equation

$$\bar{R}_P(t) = R_P - S(n-1) \quad (9)$$

where *S* is a disease dependant constant parameter, and *n* is the leaf number.

Grain yield loss model

Light interception

Each day light energy (MJ m^{-2}) is intercepted by leaves and ears. The energy $I_n(t)$

intercepted by a leaf *n* on day *t* is dependent on the shading from the leaves and ears above. Interception is calculated by analogy to Beer's law (Monteith & Unsworth, 1990)

$$I_n(t) = I_0(t)\left(1-\exp\left(-k_l L_n(t)\right)\right)\exp\left(-k_g G(t) - k_l \sum_{i=1}^{n-1} L_i(t)\right) \qquad (10)$$

where k_g is the ear extinction coefficient, k_l is the leaf extinction coefficient, $I_0(t)$ is the photosynthetically active radiation (PAR) incident on the canopy on day t, $L_n(t)$ is the simulated LAI of leaf n on day t, and $G(t)$ is the green area index of the ear on day t.

The energy intercepted by green leaf area $E_n(t)$ is reduced in proportion to the diseased area. The amount by which interception is reduced is a function of the disease type and depends partly on the extent to which green area loss exceeds symptom area. For example, a given leaf area affected by *B. graminis* lesions will cause less yield loss than the same area affected by *Mycosphaerella graminicola (Septoria tritici)* (Cook *et al.*, 1991). The reduced intercepted energy is given by

$$E_n(t) = \max\left(0, \left(1 - \sum_{i=1}^{f} \sigma_i Y_i(t,n)\right) I_n(t)\right) \qquad (11)$$

where $Y_i(t,n)$ is the percentage area of disease i on leaf n, σ_i is a disease dependent parameter which is usually greater than one (Paveley *et al.*, 2001) (intercepted energy is never allowed to be less than zero) and f is the number of foliar diseases modelled.

Grain yield loss calculation

The yield lost due to foliar disease which would otherwise be accumulated and partitioned to grain yield on day t is given by

$$\xi(t) = B\sum_{n}\left(UI_n(t) - \tilde{U}(t)E_n(t)\right) \qquad (12)$$

where the summation is over all leaf layers. The variable $I_n(t)$ is given by Eqn 10, $E_n(t)$ is given by Eqn 11, U is the radiation use efficiency (RUE) parameter, $\tilde{U}(t)$ is the adapted RUE parameter (described in the next section) and B is the dry matter partitioning constant. In general, yield-loss models based on HAD and HAA should be applied to the period when the harvested portion of the crop is growing rapidly (Johnson, 1987), in the case of wheat, during grain filling. However, some carbohydrate accumulated in the stem pre-anthesis can be translocated to grain post-anthesis (Austin *et al.*, 1977). To account for this contribution to yield from pre-anthesis photosynthesis, B is taken as zero before the plant reaches terminal spikelet, b_1 between terminal spikelet and anthesis and b_2 after anthesis. The total yield loss due to foliar disease ζ_f is given by summing $\xi(t)$ from terminal spikelet until plant senescence. The dry matter that can be accumulated prior to anthesis that is considered as contributing to grain yield is limited to 3t ha^{-1} to approximate the capacity for stem storage and re-distribution in modern wheat cultivars (Sylvester-Bradley *et al.*, 1997).

The effect of strobilurins on yield

As well as suppressing foliar diseases, strobilurin fungicides are thought to enhance yield (Bartlett *et al.*, 2002) via effects on the crop. Several mechanisms have been suggested to explain this effect including increased duration of green area, chlorophyll and radiation use efficiency (RUE) of the leaves.

Distinguishing between the yield gain due to disease suppression and yield gain due to any direct physiological effects is difficult in field experiments, so quantitative mechanistic evidence in representative environments is scarce. However the yield improvements are economically important and cannot be overlooked. Hence, arbitrarily, after a strobilurin fungicide has been applied, the RUE of each leaf which was present at the time of spray application is increased by an amount proportional to the dose of the spray reaching the leaf, up to a maximum of 0.25 of the full recommended dose. If a leaf emerges after spray application, the RUE for that leaf is increased by an amount proportional to the remaining dose. These increases remain until the leaf has senesced. This adapted RUE is notated $\tilde{U}(t)$ in Eqn 12.

Margin of grain value over fungicide cost

The margin over spray costs *M* is calculated

$$M = C_G(1-v)(W_E - \zeta_f - \zeta_s) - C_S \qquad (13)$$

where ζ_f is the yield loss due to foliar diseases, ζ_s is the yield loss due to stem diseases, C_S is the cost of fungicides (including application cost), C_G is the grain price and v is the proportional value loss due to ear diseases. Values for the stem and ear disease related variables can be calculated using a model such as the one described in Bailey (2000). An estimate of yield in the absence of disease (W_E) is entered by the user of the decision support system (DSS) and included so that the impact of disease on yield and margin can be reported to the user in relation to a yield which they consider to be realistic.

Parameter evaluation

Fungicide parameters

Two eradicant parameters and two protectant parameters describe the efficacy of each active substance against each disease. Since 1993, experiments have quantified the efficacy of the main fungicide active substances against the four predominant foliar diseases of winter wheat in the UK (Paveley & Clark, 2000; Clark, 2005); namely *S. tritici*, *Puccinia striiformis*, *B. graminis* and *Puccinia. triticina.* Randomised and replicated field plots of winter wheat were established at sites across the UK. Cultivars susceptible to the four diseases were chosen. Fungicides were applied to the plots between GS37 and GS39 at a range of doses between a quarter and twice the full recommended dose. Control plots were also grown where no fungicide was applied. Disease severity data were collected on leaves I, II and III during grain filling.

Dose-response curves were obtained by fitting the model

$$Y = \alpha + \beta \exp(-\kappa D) \tag{14}$$

to the data, where Y is the percentage symptom severity, D is the dose of the active substance and α, β, and κ are parameters. Dose-response data do not exist for all active substance and disease combinations, for example, where an active substance is not used primarily to treat a particular disease but may have some limited efficacy against it when applied to control another disease. In these cases expert opinion was used to estimate efficacy relative to active substances of known efficacy.

The active substance model parameters were fitted so that the coupled canopy (Milne *et al.*, 2003), disease (Audsley *et al.*, 2005) and fungicide models gave results reflecting the experimental and expert data. The half-life parameter μ, is correlated to parameters k_P and k_E, so to stabilise the parameter fitting process it was assigned a value based on expert knowledge of active substance decay. The experimental data provide ten points but those points are subject to large variability, so it was not possible to fit self-consistent active substance parameters directly to the data. Instead the parameters were fitted to smoothed experimental data from the dose-response curves.

The active substance parameter fitting was done by simulating similar field experiments to those carried out. The application timing of the active substance was simulated at the correct growth stage at the varying doses. A non-linear optimisation scheme (using a modified Levenberg Marquart method - Gill *et. al.*, 1981) was used to find the parameter values that minimised the difference between the simulated disease and the corresponding value of disease given by the experimental dose-response curve (Eqn 14).

For each disease, the eradicant, protectant and half-life parameters characterise a {dose by spray timing by disease} response surface for each leaf layer for a series of application dates (e.g. Fig. 1). Disease control increases as dose increases and spraying too early or too late reduces efficacy. Expert opinion was used to check the relative performance of active substances represented by the resulting dose–timing response surfaces. A few chemicals are known to have only protectant abilities (e.g. chlorothalonil). In these cases the eradicant parameters in the model were preset to zero and the protectant parameters fitted. Examples of parameter values for contrasting active substances are given in Table 1. Fig 2 shows the dose – response curves for these active substances on leaves I and II. The figure illustrates that applying chlorothalonil early in the leaf's life gives good control whereas delaying reduces its control relative to epoxiconazole. There were insufficient data available to fit parameter τ in Eqn 4 which characterises the reduction in dose arriving on leaves deeper in the canopy, so it was given a value 0.25 based on expert judgement.

The reduction in protectant efficacy due to asynchrony of leaf emergence between shoots early in stem extension was parameterised by simulating the effect of fungicide on three tillers with phyllocrons 10°C days apart. The structure of the canopy model (Milne *et al.*, 2003) meant that flag leaves emerged over a period of 60°C days, and leaves on layer n emerged over a period of 20(n+2) °C days. The parameter S in Eqn 9 was fitted so that a simulation of a single shoot approximated to the simulation with three tillers. Values of S for *S. tritici*, *P. striiformis*, *B. graminis* and *P. triticina* are 0.135, 0.03, 0.075 and 0.0225 respectively.

Yield parameters

The parameters in Eqn 10 were taken from England (1987). The others were evaluated from experimental data from field experiments reported previously (Milne *et. al.* 2003, Audsley *et al.* 2005). In summary, winter wheat cultivars Riband, Apollo, Slejpner and Haven were grown in randomised and replicated plots at 12 sites across the England and Wales. A total of 37 experiments were completed during harvest years 1994 to 1997. Each replicate block consisted of fungicide treated and untreated plots of each cultivar; the former receiving a three-spray broad-spectrum fungicide programme, designed to give full protection to the upper leaves and ears. Growth stages, green leaf area and diseased leaf area were measured separately for each culm leaf weekly from GS 31 to maturity, and grain yield was measured. Daily weather data were collected from meteorological stations, within one kilometre of the site.

The RUE parameter was fitted to the undiseased yield by simulating undiseased growth at each site. The green leaf area loss parameters σ_i in Eqn 11 were fitted by simulating the canopy and disease development at each site where only one disease was observed, and matching the simulated disease loss with the observed. For each of the four diseases this was possible in approximately five cases per disease. To be risk-averse, the parameters were chosen so that the model gave higher than average yield loss. The yield partitioning coefficients in Eqn 12 were determined from data on the partitioning of dry matter (Paveley, unpublished). Limited data were available to estimate the strobilurin RUE increase parameter so the parameter value was estimated by peer assessed expert opinion. Table 2 summarises the yield model parameter values.

Variability

Because future weather is unknown and variable from season to season, disease risk will vary, leading to uncertainty surrounding the predicted effect of a grain fungicide treatment. An intensive fungicide programme will control disease well almost regardless of disease risk; hence there will be little variability. In contrast, the outcome from treatments which provide less effective control could vary widely depending on whether conditions are conducive to epidemic development. However, the spray programme itself does not provide a good explanatory variable for modelling, because there is no simple way of assessing the degree of control that will be achieved, other than using the model. Thus the explanatory variable used was yield-loss. The model was provided with 15 weather sets representing different years from two sites representing extremes within the cereal growing areas of the UK. A set of Monte Carlo simulations (Hammersley & Handscomb, 1979) were carried out in which 2080 spray programmes were generated at random, varying in chemical selection, dose and timing. Each one was applied to each weather set to obtain sets of 15 values from which the mean and standard deviation were calculated, giving 2080 mean-deviation pairs, shown in Fig. 3. The standard deviation tended to zero at zero yield loss and could be modelled using

$$s = qr^p(1-r)^p \tag{15}$$

where r is the mean relative yield loss and s is the standard deviation. The fitted model

is shown in Fig. 3, with $q = 0.27$ and $p = 0.606$. The yield range for a spray programme is calculated as the mean ±2 standard deviations. When the optimisation compares alternative programs it uses a risk-adjusted margin $\tilde{M}$ given by

$$\tilde{M} = M(1 - 0.3s) \tag{16}$$

Model validation

Materials and methods

The experiments used for validation were those reported by Audsley *et al.* (2005). In summary, during the season 1999/2000 randomised and replicated field plots of between one and three winter wheat cultivars were established at each of eight sites across the UK. The cultivars were selected to contrast for susceptibility to the four main foliar diseases. Each replicate block consisted of plots of each cultivar treated with a three-spray fungicide programme, a single spray or left untreated. Other treatments were included, but are not reported here. Leaf emergence dates and percentage disease were recorded weekly from each leaf layer that had emerged (ligule visible) on ten randomly sampled shoots from each of the three replicate plots of each cultivar. Mean yield loss for untreated and one-spray plots was estimated by subtracting the respective mean yields from the mean yield of the three-spray treatment plots.

The canopy and disease models reported in Milne *et al.* (2003) and Audsley *et al.* (2005) were coupled with the fungicide and yield loss model reported here. Two simulations of each experimental site were carried out. The first with no fungicides applied and the second with a single spray of fungicide applied. Weather data recorded at each site were used by the simulations, and canopy and disease observations were used to update the models at around GS 32 (as would be the case in practical use) so that they closely simulated the observed canopy and disease growth.

Results

In the field experiments, epidemics of *S. tritici*, *P. striiformis* and *P. triticina* occurred at a wide range of severities across the sites and varieties, resulting in a wide range of yield loss values. Fig. 4 compares the simulated and observed yield losses for the untreated plots and the plots that received a single spray application. The error bars show ± one standard error about the mean. The comparison tests both yield loss and active substance simulations. The simulated yield loss values were generally close to the observed values, often within the scope of one standard error.

Predicting the dose-response surface of a two-spray programme

Paveley *et al.*, (2003) reported experiments to determine dose-response surfaces for the joint action of two fungicide applications. In summary, two applications of

tebuconazole were applied to randomized replicate plots of winter wheat as leaves III and II emerged, at doses of 0.0, 0.25, 0.5, 1.0 and 2.0. A model of the effect of two spray programmes on disease was proposed and validated. Previously unpublished yield data from replicate plots were available for the ADAS Rosemaund site in harvest year 1997 (Fig. 5). The yield response surface shown in Fig. 5 is given by

$$W = \left(a + b\left[1 - \exp(-kd_1)\right]\right)\left(1 + c\left[1 - \exp(-kd_2)\right]\right) \quad (17)$$

where W is yield (t ha^{-1}), d_1 and d_2 are the doses of tebuconazole applied as leaves III and II emerge respectively, and a = 6.015, b = 0.528, c = 0.345 and k = 2.199 are model parameters. These parameters were estimated by fitting Eqn 17 to the yield data. The experiment was simulated using the model reported here and Fig. 6 shows actual mean yield loss (across three replicate plots) plotted against simulated yield loss for each treatment combination. The actual yield loss was calculated by subtracting the mean yield from 9.66 t ha^{-1}, which was the maximum recorded in the experiment. One standard error of the mean of the observed data was 0.25. The results show that the simulation behaved reasonably with two sprays, although actual yield loss increased more rapidly than simulated yield loss.

Subjective analysis of suggested fungicide product, dose and timing combinations

The model described here was designed to form part of a decision support system of coupled models to suggest fungicide programmes that are appropriate for a particular crop; accounting for the host resistance of the cultivar and local disease pressure. The coupled canopy (Milne *et al.*, 2003), disease (Audsley *et al.*, 2005), fungicide and yield loss models were therefore tested through an optimisation routine (Parsons and Te Beest, 2004) which selects and ranks possible fungicide programmes for a prescribed scenario, based on their relative simulated risk-adjusted (Eqn 16) margins.

Three scenarios were used: Scenarios 1 and 2 were for cultivar Claire (*S. tritici* resistant but *B. graminis* susceptible) under moderate and high *B. graminis* pressure, respectively. Scenario 3 was cultivar Savannah (*S. tritici* susceptible but *B. graminis* resistant) under moderate *B.graminis* pressure. All of the suggested spray programmes in all scenarios include a fungicide treatment applied within a week of flag leaf emergence (typically around 22 May), supplemented by a treatment either around the emergence of Leaf III or the ear. The increase in disease pressure between scenario 1 and 2 resulted in an increase in the total dose units applied from 1.75 to 2.0. Despite fungicide treatment, the gross margin from Savannah was lower that that of Claire under equivalent disease pressure, due to the greater damage caused by *S .tritici* compared with *B. graminis.* A subjective analysis of the appropriateness of these treatments is presented in the discussion.

Discussion

The fungicide model described here provides a method of simulating the effect of different fungicide active substances, applied at any legal dose and timing, on disease, and hence on green canopy area. The yield model calculates disease-induced yield loss according to loss of green area which would otherwise intercept radiation. This

methodology couples with the canopy and disease models described previously (Milne *et al.*, 2003, Audsley *et al.*, 2005). The predicted yield loss was greater than an estimate of the observed yield loss when tested against data from eight experimental sites; although the reverse was found in the single experiment where two-spray programmes were tested. The observed yield loss at the eight sites was calculated by subtracting the mean diseased yield for each cultivar from the mean yield of plots of the same cultivar treated with a three-spray programme. Such plots would not be entirely disease free. Therefore we would expect a smaller observed yield loss than simulated. On average the simulated treated yield loss is 0.5 t ha^{-1} greater than the observed, whereas the untreated is 0.1 t ha^{-1} greater, indicating that the efficacy of fungicides is underrated in some of the simulated scenarios.

As the actual future weather and hence disease development is unknown at the time of the decisions, a comparison of the optimal spray programme and the post-harvest optimum is not appropriate, since the optimal spray programme is the best protection against all possible future outcomes. One test is whether a decision support system helps users get closer to the optimum more often than any other method of making decisions, given the information available at the time of the decision. With the large number of possible choices of fungicide product, dose and timing, it is infeasible to determine experimentally via every combination whether the coupled models are identifying the exact optimal spray programme. However, it was possible to assess subjectively that the inputs suggested were reasonable on average and responded appropriately to changes in disease risk and cultivar resistance. Pesticide usage survey data show that commercial wheat crops in the UK receive approximately three dose units of fungicide, typically applied as two- or three-spray programmes (Garthwaite *et al*., 2004). In contrast, the top treatments selected by the optimisation procedure running the coupled models, were between 1.0 and 2.75 total dose units, depending on disease pressure and cultivar. The pesticide usage survey data, together with data collected as part of the annual cereal disease survey (Hardwick *et al*., 2001), suggest that application timing and product choice are often poor in commercial crops. Field experiments could test whether the lower inputs suggested by the decision support system provide reliable control despite relatively low total dose inputs, by improving spray timing and product selection. The suggested treatments reported here show that this might be the case. All of the suggested spray programmes included a treatment within a week of flag leaf emergence – a key growth stage for treatment to protect the flag leaf which is vital for photosynthesis during the grain filling period (Paveley & Clark, 2000). Product choice responds appropriately to cultivar and disease risk. On the *B. graminis* susceptible cultivar Claire, active substances which are specifically active against *B. graminis*, such as metrafenone (as Flexity), quinoxyfen (as 'Fortress') or morpholines were included in mixtures with conazoles under high *B. graminis* pressure. On the septoria-susceptible cultivar Savannah, all of the suggested programmes included a mixture of a conazole to provide eradicant activity and a protectant active against *S. tritici* (such as cholorthalonil in Bravo, Alto Elite and Impact Excel, or boscalid in Tracker). The type of unconstrained optimisation described above (which allowed any combination of treatments within the legal constraints on product use described on the label) also provides a good test of the performance of the models. Any weakness in model logic or data would have scope to become apparent. However, further field testing of the complete decision support system is required across a wide range of cultivars and environments, to ensure that the treatments suggested provide consistently profitable

disease control.

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Table 1. *Examples of parameter values for two fungicide active substances with contrasting modes of action. These values were valid for season 2003-2004.*

Active substance	a_P	k_P	a_E	k_E	μ
Epoxiconazole	0.71	6.00	0.50	7.01	0.069
Chlorothalonil	0.0	-	1.0	6.49	0.010

Table 2. *Yield-loss model parameter values* (*units are shown in the glossary, unless dimensionless*)

Eqn	Parameter	Value
10	k_l	0.44
	k_g	0.2
11	σ	2.3 (*P. triticina*)
		2.2 (*P. striiformis*)
		2.0 (*B. graminis*)
		3.15 (*S. tritici*)
12	b_1	0.375
	b_2	0.7
	$U(t)$	1.57 (no strobilurin applied)
		1.68 (max. value if a strobilurin applied)

Table 3. *Spray programs ranked in order of optimum risk-adjusted margin of grain value minus fungicide cost for three situations, showing effect of varietal resistance and difference in disease pressure on choice of fungicide dose and timing. Doses are given as the proportion of the maximum recommended dose for a single application.*

Date			1/5	8/5	15/5	22/5	29/5	12/6	19/6		
Fungicide Product			Dose units at each suggested spray date							Total dose units	Margin (£ ha^{-1})
Scenario 1 – Variety: Claire (resistance ratings *S.tritici* = 6, *B.graminis* = 3*);* moderate mildew disease pressure											
1st Spray	2nd Spray	3rd Spray									
Pro	Lan+Dia		1.0				0.5+0.25			1.75	548
Fan+Pro	Fol+SwG				0.5+0.25				0.75+0.25	1.75	551
Man+ Pro	Fol				0.25+0.5			0.75		1.5	546
Lan+Pro	AmP+Til		0.25+0.75				0.5+0.5			2.0	542
Opu+Fan	Fol				0.25+0.75				0.75	1.75	543
Uni+Tra	Lan		0.5+0.75			0.5				1.75	539
									Mean	1.75	545
Scenario 2 – Variety: Claire (resistance ratings *S.tritici* = 6, *B.graminis* = 3*);* high mildew disease pressure											
1st Spray	2nd Spray	3rd Spray									
Fle+Lan	Fol				1+0.5			1		2.5	489
Fle+Man	Fol				0.75+0.5			0.75		2.0	488
Fle+Man	Fol				0.5			0.75		2.25	483

Pro	OpT+For		0.75		0.5+0.75					2.0	479
Por-Bra	Pat+Man			0.5+0.25		0.25+0.25				1.25	483
									Mean	2.0	484
Scenario 3 – Variety: Savannah (resistance ratings *S.tritici* = 3, *B.graminis* = 7*);* moderate mildew disease pressure											
1st Spray	2nd Spray	3rd Spray									
Tra+Pro	Lan				0.5+0.25		0.5			1.25	523
Bra+Pro	Man				0.5+0.75		0.25			1.5	522
Tra+Epi	Pro+Man		0.5+0.5			0.5+0.25				1.75	516
Car+Tra	Man				0.25+0.5		0.25			1.0	515
AlE+Pro	Dia+AlE		0.25+0.75			0.25+0.5				1.75	509
ImE+Pro	Opu	Bra+Dia	0.25+0.5		0.75		0.75+0.5			2.75	498
									Mean	1.67	514

1

Fungicide product abbreviations

AlE = Alto Elite; AmP = Amistar Pro; Bra = Bravo; Car = Caramba; Dia = Diablo: Epi = Epic: Fan = Fandango; Fle = Flexity; Fol = Folicur: For = Fortress: ImE = Impact Excel; Lan = Landmark; Man = Mantra; OpT = Opus Team; Opu = Opus; Pat = Patrol

Pro = Proline; SwG = Swing Gold; Til = Tilt; Tra = Tracker; Uni = Unix

1 Glossary of variables

t	day number
n	leaf number
$A_n(t)$	proportion of leaf n that has emerged on day t
B	dry matter partitioning constant
C_S	spray plan cost including application cost (£)
C_G	grain price (£)
$d(t)$	effective active substance dose on day t
D_n	applied dose of the active substance
$E(t)$	total intercepted energy (MJ) over all leaf layers
$E_n(t)$	energy intercepted (MJ) by diseased leaf n on day t
$G(t)$	green area index of the ear on day t
$I_0(t)$	photosynthetically active substance radiation (MJ m^{-2}) incident on the canopy on day t
$I_n(t)$	energy (MJ) intercepted by undiseased leaf n on day t
$L_n(t)$	leaf area index of leaf n on day t
M	margin over spray costs
$P(t,n)$	number of potential infections
$R_P(t)$	reduced proportion of successful infections on day t caused by active substances protectant effect
$R_E(t)$	reduced growth rate of infection on day t caused by active substances eradicant effect
S	disease dependent reduction in protectant efficacy
$T(t)$	accumulated thermal time (°C days, base zero)
$U(t)$	radiation use efficiency (RUE) parameter
$Y(t,n)$	percentage symptom severity
$y(T)$	lesion size (%) expressed as a proportion of its asymptote value, at thermal time T (°C days, base zero)
W_E	expected yield (t ha^{-1})
$\xi(t)$	yield lost due to foliar disease on day t (t ha^{-1})

ζ_f yield loss due to foliar diseases (t ha^{-1})

ζ_s yield loss due to stem diseases (t ha^{-1})

V proportional value loss (%) due to ear diseases

Figure Legends

Fig. 1. A simulated dose – timing disease response surface for epoxiconazole against *S tritici* on the flag leaf. The *x*- axis is dose (L ha^{-1} of commercial product), the y- axis is day number since 1 September and the *z*- axis is the integral of percentage disease on the leaf over time (AUDPC). The leaf was fully emerged on day 250.

Fig. 2. Simulated dose-response curves for active substances epoxyconazole (⎯⎯) and chlorothalonil (⎯ ▪ ⎯) against *S. tritici* on leaf I and leaf II, applied when leaf I was fully emerged (GS 39). One dose unit is the full recommended dose for the commercial product.

Fig. 3. Plot of standard deviation versus mean of relative yield loss for 2080 different spray programmes simulated with 15 different weather sets. The solid line shows the fitted model (Eqn 15) for a relationship between the two.

Fig. 4.

Observed mean yield loss plotted against simulated mean yield loss for untreated (—) plots and single spray application treated (♦) plots at sites across the UK in season 1999/2000. The observed mean yield loss is calculated by subtracting the mean yield of the untreated or single spray replicate plots from the mean yield of the plots which were treated with a three spray programme. The line through each point shows ± one standard error about the mean. The dotted line has a slope of one.

Fig. 5. Dose-response surface for two applications of tebuconazole fitted to data from an experiment at Rosemaund in the 1996/1997 season. The first application was carried out as leaf III emerged and the second as leaf II emerged. The yield data points that the surface was fitted to (Eqn 17) are indicated by the solid dot (●). The dose is L ha^{-1} of commercial product.

Fig. 6. Actual mean yield loss (t ha^{-1}) across three replicate plots of a field experiment plotted against simulated yield loss for each dose and spray timing combination. The actual yield loss was calculated by subtracting the mean yield from 9.66 t ha^{-1}, which was the maximum recorded in the experiment. One standard error mean of the observed data was 0.25. The dotted line has a slope of one.

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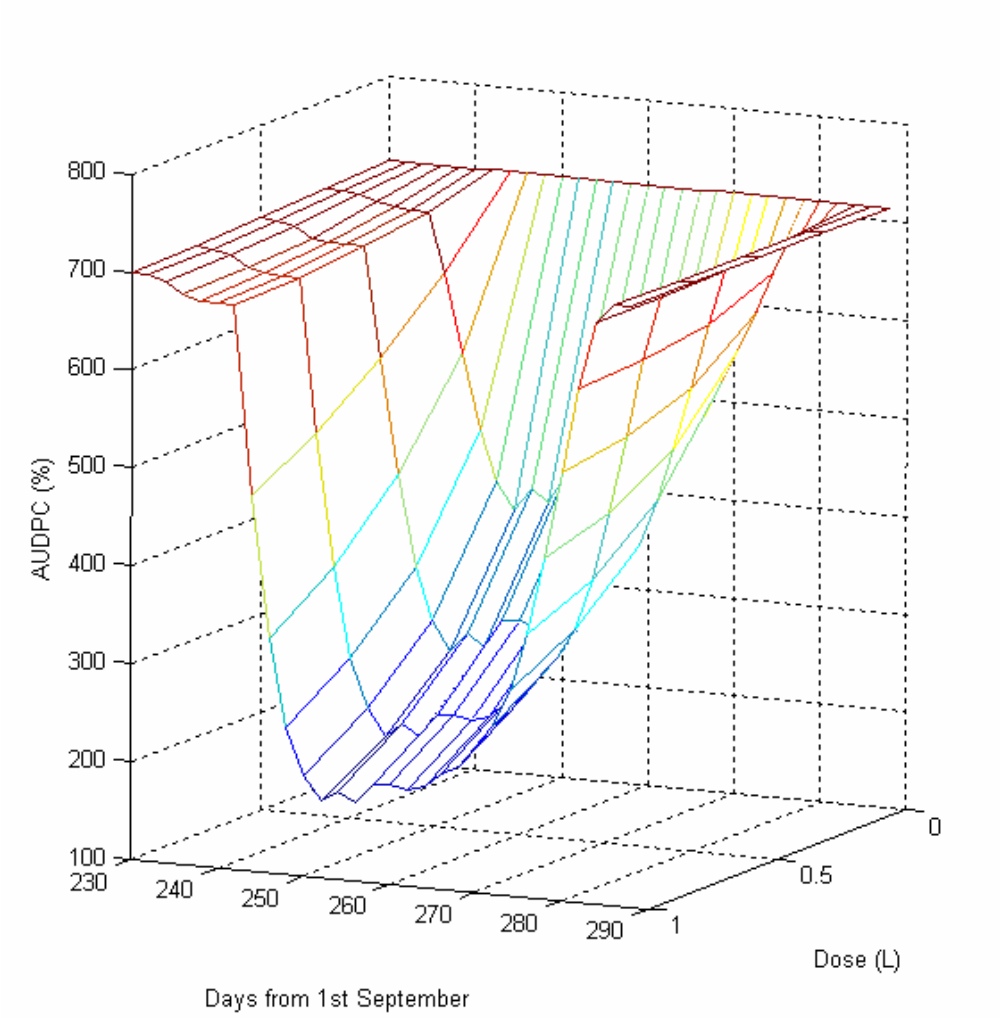

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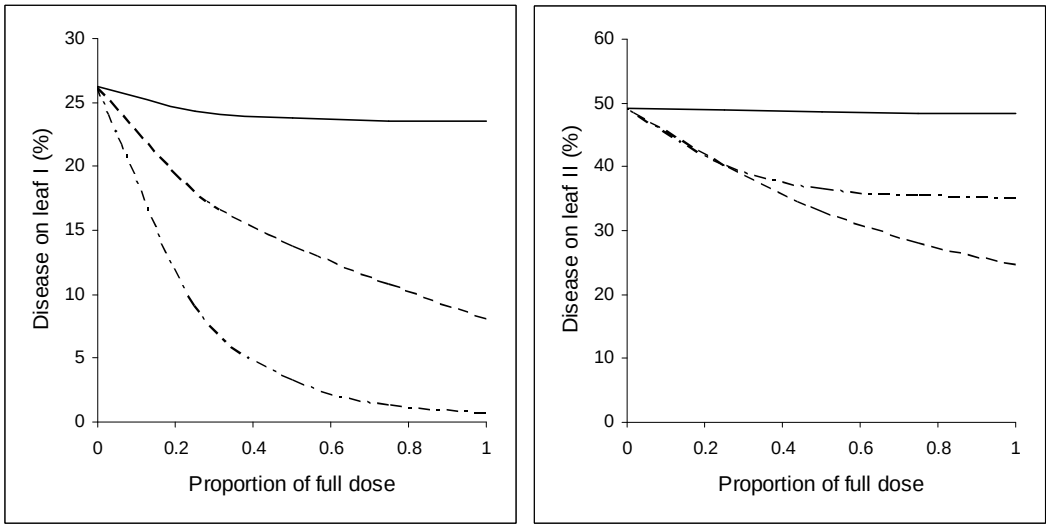

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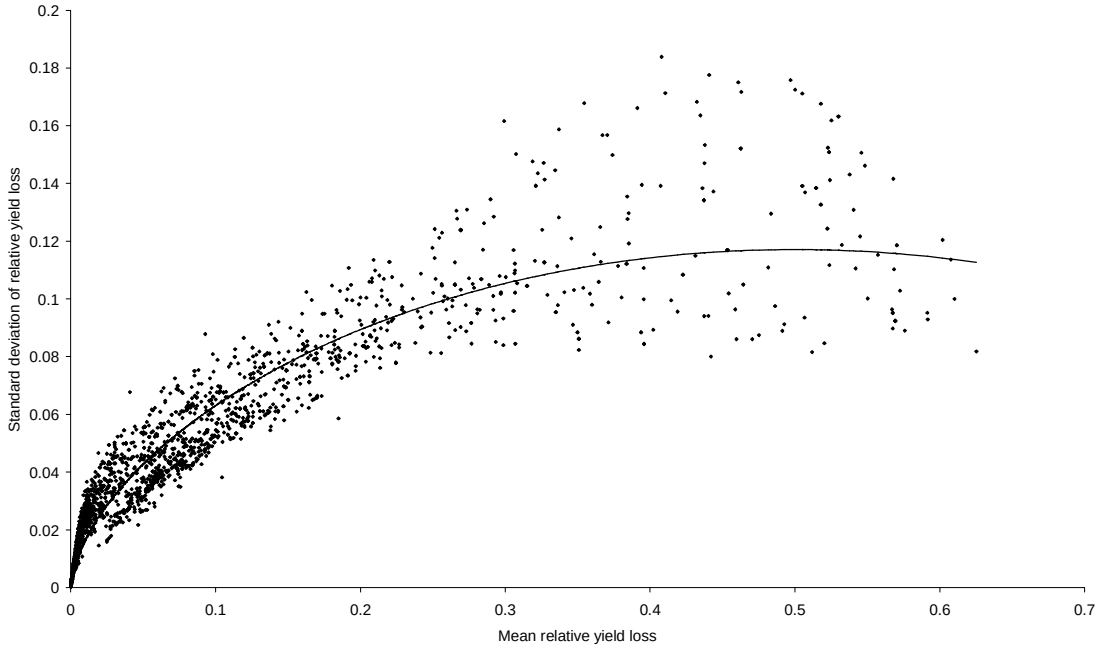

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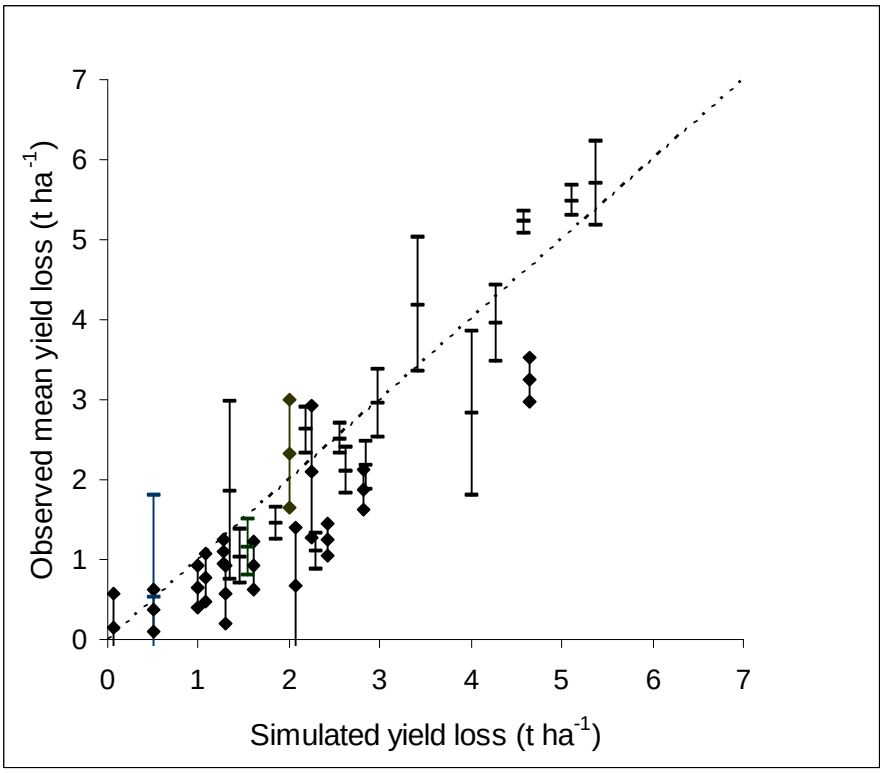

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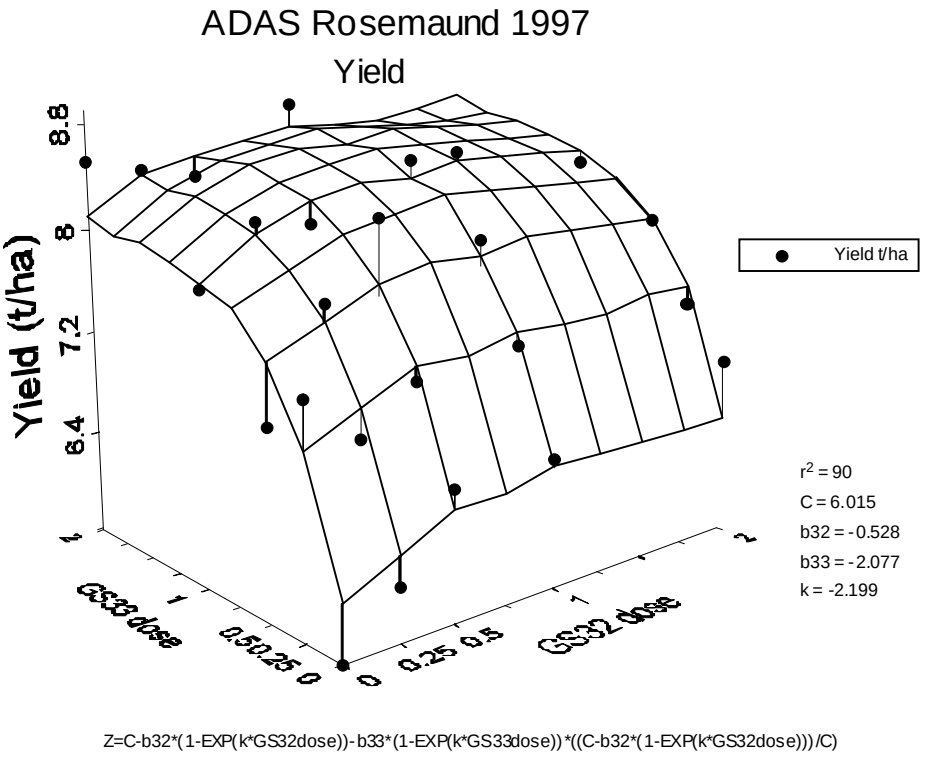

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