Modelling the effectiveness of Integrated Pest Management strategies for the control of *Septoria tritici*

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Abstract

Reducing reliance on pesticides is an important global challenge. With increasing constraints on their use, in recent years there has been a declining trend in pesticide use for arable crops in the UK. But with increasing disease pressures and global demand for food, there is a greater need for effective measures of pest and disease control.

These circumstances highlight the need for widespread adoption of sustainable alternative control measures. Integrated Pest Management (IPM) is one such solution, comprising a set of management strategies which focus on the long-term prevention, detection and control of pests, weeds and diseases. While many of these methods are acknowledged to offer effective control, their implementation has thus far been limited in practice.

As a case study we considered *Septoria tritici*, an economically important disease of wheat. We used epidemiological modelling techniques to investigate the potential of different IPM control strategies (crop residue burial, delayed sowing, variety mixtures and biocontrols). Combining existing data with a deterministic, compartmental infectious disease model of *S. tritici* transmission, we simulated the implementation of an IPM regime into the *S. tritici* disease system. We investigated the outcomes on disease prevalence and crop yield when comparing conventional and IPM control regimes. In a single field, for the individual implementation of IPM measures we found the starkest difference in potential yield outcomes between delayed sowing (greatest yields) and crop residue burial (lowest yields). We also found that the collective use of IPM measures has the potential to offer individual growers comparable control to a standard fungicide regime. For a multi-field setting, representing a community of crop growers, a high proportion of growers using IPM can reduce the level of external infection incurred by the growers who maintain a fungicide regime.

1 Introduction

Global demand for food is widely projected to increase in the coming decades, with many predicting a rise between 35% to 56% from 2010 and 2050 [1]. The agricultural industry faces the need to increase its production, while also working against mounting pressure from plant pests and diseases, which

are currently estimated to be the cause of up to 40% yield losses globally [2]. One such disease is *Septoria tritici* (commonly called 'septoria' or 'septoria leaf blotch'), a foliar disease that primarily affects wheat. The UK's Agriculture and Horticulture Development Board (AHDB) report *S. tritici* as the most important and damaging foliar disease on winter wheat [3]. The pathogen causes necrotic lesions to develop on the leaves, reducing the area of healthy green tissue available for photosynthesis, resulting in yield loss and reduced grain quality. AHDB state that crops severely affected by *S. tritici* can suffer losses of up to 50% [3].

To combat these threats to crop health, chemical control remains a staple of conventional crop protection. However, due to the negative ecological and environmental consequences of their use [4], there have been increasing constraints on pesticide use for pest and disease management (e.g. removal of some chemical pesticides from UK markets). These constraints have resulted in a recent declining trend in pesticide use on arable crops in the UK. The 'Arable crops in the United Kingdom' survey for 2022 reported, compared to 2018, a decrease in area treated with pesticides of 6% and a decrease in weight of pesticide applied of 14% [5]. These circumstances bring to the fore the need for widespread adoption of sustainable alternative solutions for crop pest and disease management.

Much focus for a sustainable alternative has been put on Integrated Pest Management (IPM). IPM is not a single control method, but a programme of coordinated, environmentally-sensitive control measures that can be used to prevent, detect and control crop pests or diseases [6]. It can comprise a wide range of potential strategies, such as the use of resistant varieties, biological controls, modification of sowing practices, and the targetted and controlled use of chemical pesticides. One of the main, generally agreed-upon, aims of IPM is to reduce reliance on the use of synthetic pesticides [7]. Nevertheless, despite long-term empirical evidence of the effectiveness of IPM, its adoption in practice has been limited. Growers face barriers to uptake in the form of access to existing tools and knowledge, risk, ease of implementation, and cost [8].

There remain several unanswered questions regarding IPM and its use in the control of *S. tritici*. Previous modelling work on *S. tritici* has investigated the rate of development of fungicide resistance under the use of high- or low-risk fungicides [9], the use of fungicide mixtures and alternation [10–12], and the timing of fungicide applications [13]. One study, by Taylor and Cunniffe [14], explored the use of resistant cultivars in combination with fungicide treatments, which would be considered an IPM strategy commonly in use by growers in the UK. Previous modelling work has not, however, investigated the use of IPM controls that are presently available to farmers but not commonly deployed in practice. Examples of such IPM controls that have had low uptake to date are variety mixtures, residue management, varied sowing dates, and biocontrols.

A crucial benefit that modelling can offer is the ability to test the effectiveness of IPM regimes and compare it to the current standard fungicide practices, without the use of real-world, long-term trials. This has been done previously with a number of different diseases and management strategies [15–17]. Nevertheless, at the time of writing we have not identified studies that have used modelling to contrast the possible impacts on yields of an IPM control strategy versus a conventional fungicide strategy. Moreover, such work can help identify where additional data to reduce parameter uncertainty can improve the robustness of model predictions. In this paper, we use modelling to address the following questions (with respect to yields):

- How does an individual IPM measure perform compared to either having no control measures, or using conventional fungicide treatment?
- Can the collective use of multiple IPM measures outperform a conventional fungicide regime?
- Considering a community of crop growers, is there an optimal balance of IPM farms with con-

ventional fungicide farms?

To provide insight into these questions, we present an infection transmission model for *S. tritici* whose novel contribution is its incorporation of IPM interventions. We initially acquired from the available literature plausible value ranges for the model parameters. We then performed model simulations to investigate the potential impact on yield of chemical control (i.e. fungicide use) and four different IPM control strategies (crop residue burial, delayed sowing, variety mixtures and biocontrols). From a single-field perspective, we found that collective use of multiple IPM measures has the potential to offer individual crop growers comparable control to a standard fungicide regime. For a multi-field system, we show circumstances where the presence of IPM-using fields can benefit all growers by boosting yields. By highlighting the potential for a 'free rider' situation (those using a chemical control regime benefitting from others using IPM), we anticipate our findings to be a starting point towards a combined epidemiological-behavioural model for growers' intentions towards disease control. The framework we present is also adaptable to other plant pathogens, allowing researchers to investigate the implications of IPM usage on infection burden and yield outcomes for other crops.

2 Methods

To carry out our investigation, we first developed a mathematical model of *S. tritici* infection dynamics that included fungicide and different IPM strategies as possible controls. We summarise here the core methodological components of our study. We begin with a description of our base *S. tritici* model that had no controls (Section 2.1), including how we sourced plausible model parameter values from the literature, followed by how we computed yield from a single growing season (Section 2.2). We then explain how we incorporated fungicide control into the model and its parameterisation (Section 2.3). Similar details are provided for different IPM control strategies (Section 2.4). Finally, we outline the model simulations that we performed to explore our stated research questions (Section 2.5).

2.1 Base *S. tritici* transmission model (with no controls)

We base the transmission dynamics of *S. tritici* on the model framework presented by Hobbelen *et al.* [9] (Section S1); this model is a system of ordinary differential equations (ODEs) describing the host-pathogen system of *Triticum aestivum* (winter wheat) and *Zymoseptoria tritici* (the causal organism of *S. tritici*). The pathogen is not explicitly featured in the model, which instead describes the relative area of leaf tissue in each disease state (Fig. 1). Note that the model by Hobbelen *et al.* [9] deals with resistance to fungicide, which means that it features two strains of infectious spores that are resistant and susceptible to fungicide, respectively. Our model assumes the presence of only the strain that is susceptible to fungicide. The model uses degree-day time, taking average temperature data recorded between 1984 and 2003 in Cambridgeshire, UK. In Hobbelen *et al.* [9] they processed the data to obtain an average temperature profile, identifying the degree-day and calendar dates of the average year that corresponded to the relevant growth stages of wheat. We define $T_{[X]}$ as the degree-day time when the plant reached growth stage [X] ('GS[X]'). Throughout our analysis we considered the system behaviour over one growing season.

Our model has six disease states, each measured in m^2 of leaf area per m^2 of ground. Although this is by definition a density, for simplicity it is referred to hereafter as the 'leaf area' because the ground area is fixed. Five of the disease states are related to the upper canopy leaves. Specifically, they describe the area of susceptible leaf tissue (labelled S), area occupied by latent (E) and infectious (I) disease lesions, the total leaf area that has died due to infection (D) and the total leaf area that has naturally senesced (R). Only tissue in the I class can transmit infection and only tissue in the S class can receive infection. We define the total leaf area, denoted by A, as $S + E + I + R + D$.

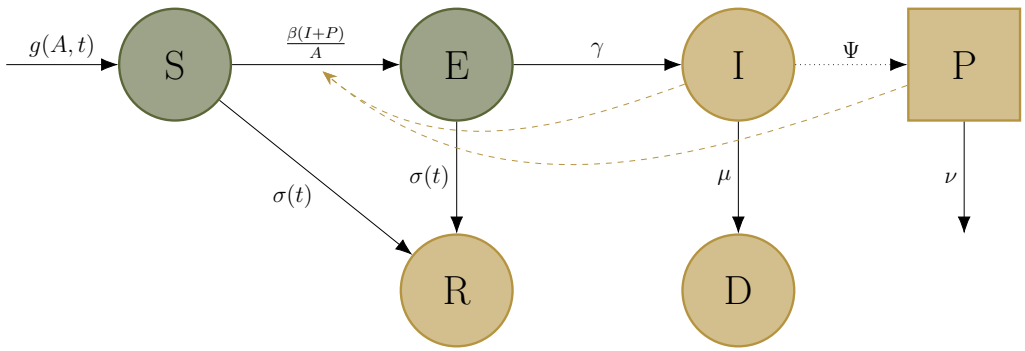


Fig. 1. Schematic for the base *S. tritici* dynamics model presented by Hobbelen *et al.* [9]. Circular nodes represent the states leaf area in the upper canopy can occupy. The square node (P) represents infectious leaf area in the lower canopy. Solid black lines represent the progression between states, labelled with the corresponding rates. The dotted black arrow shows between-season dynamics: infectious lesions deposit inoculum which initiates infection in the lower canopy the next year. Green nodes represent the states containing green leaf area: a healthy, uninfected state (S), and a latently infected state (E). Leaf area in these states are capable of photosynthesis, and contribute towards the yield computation. Later in the season these states experience natural senescence (R). Yellow nodes represent states that do not contribute to the yield computation. Infectious leaf area in the upper canopy (I) eventually dies due to infection (D), and infectious leaf area in both the upper and the lower canopy (I and P) transmit infection to healthy tissue, represented by yellow dashed arrows.

The sixth (and last) disease state, labelled P, represents the area of infectious lesions on the lower canopy of the plant. As disease in the upper canopy is typically initiated by infection with spores from the lower canopy, we include this lower canopy infection compartment to provide the initial infection to the plant. Infection in the lower canopy is initiated by inoculum from the previous year [11]. As defined by Hobbelen *et al.* [9], we assumed infection in the lower canopy has an initial condition of Ψ, and a fixed rate of exponential decay ν.

The model includes time-dependent transition rates for natural growth ($g(A,t)$) and senescence ($\sigma(t)$) of healthy leaf tissue, and constant rates of progression from latency to infectiousness (γ) and death from infection (μ). The force of infection term is comprised of transmission from both the I and P classes, in the form of $\frac{\beta(I+P)}{A}$.

2.2 Yield

We considered the yield to be proportional to the healthy area duration (HAD) over the period of grain filling; defined to be between GS61 and GS87 [9]. Given our assumption of latently infected tissue still being able to photosynthesise, we assumed the annual relative yield in our system to be

$$Y(t) = \frac{\int_{T_{61}}^{T_{87}} (S+E)dt}{Y_{\mathrm{DF}}},$$

where Y_{DF} is the yield obtained in the disease-free scenario (i.e. a fully healthy crop) [9, 11].

2.3 Summary of fungicide control

The existing *S. tritici* disease dynamics model presented by Hobbelen *et al.* [9] also included control with fungicide. For our analyses, we assumed that growers who used fungicide did not use any additional control methods. Although this is a simplifying assumption, it allows us to more distinctly contrast the two types of control regime (fungicide versus IPM). To incorporate fungicide control into our *S. tritici* transmission model, we used the 'high-risk' fungicide as defined in the original study [9]. The 'high-risk' fungicide represents the use of a quinone outside inhibitor (QoI), applied at GS32 and GS39, whose concentration over time is modelled by

$$C(t) = \begin{cases} 0 & \text{if } t < T_{32} \\ e^{-\delta(t-T_{32})} & \text{if } T_{32} \leq t < T_{39} \\ e^{-\delta(t-T_{32})} + e^{-\delta(t-T_{39})} & \text{if } t \geq T_{39}. \end{cases} \tag{1}$$

The parameters ω, θ and δ represent the maximum effect of fungicide, fungicide dose response curvature, and fungicide decay rate respectively (values listed in Table 1). The effect of this fungicide on both transmission, and on infection period, depends upon its concentration in the form of

$$\epsilon(C) = \omega(1 - e^{-\theta C}). \tag{2}$$

Therefore, the system of equations that govern *S. tritici* infection in a field under a fungicide regime (denoted with the subscript F) is

$$\begin{aligned} \frac{dS_F}{dt} &= g_F(A_F, t) - \sigma(t)S_F - S_F(1 - \epsilon(C))\lambda_F(t) \\ \frac{dE_F}{dt} &= S_F(1 - \epsilon(C))\lambda_F(t) - \sigma(t)E_F - \gamma E_F(1 - \epsilon(C)) \\ \frac{dI_F}{dt} &= \gamma E_F(1 - \epsilon(C)) - \mu I_F \\ \frac{dR_F}{dt} &= \sigma(t)(S_F + E_F) \\ \frac{dD_F}{dt} &= \mu I_F \\ \frac{dP_F}{dt} &= -\nu P_F, \end{aligned} \tag{3}$$

where $\lambda_F(t) = \frac{\beta(I_F+P_F)}{A_F}$, and g_F is identical to the function g in the base system [9] (Section S1). Parameter values, $\sigma(t)$, and initial conditions are the same as in the base system [9] (Table 1 and Section S1).

2.4 Summary of considered IPM controls

We considered the IPM measures described in the 2021 AHDB Research Review on *Enabling the uptake of integrated pest management (IPM) in UK arable rotations*, which identifies nine IPM measures applicable for the prevention of *S. tritici* in wheat [18]. Based on the existing research literature, the review assigned scores on a scale of 1-5 to each measure, across a number of qualities (Fig. 2). These

Table 1. *S. tritici* transmission model and fungicide parameter values. For these parameters we used the same values as used by Hobbelen *et al.* [9]. We use the notation 'dd' to represent degree days.

Parameter	Definition	Value
	Plant growth stages	
T_{emerge}	Time of GS31 (emergence of leaf 3)	1550 dd
T_{32}	Time of GS32	1668 dd
T_{39}	Time of GS39	1912 dd
T_{61}	Time of GS61 (senescence/harvesting begins)	2255 dd
T_{87}	Time of GS87 (senescence/harvesting ends)	3104 dd
	Plant growth parameters	
R	Host growth rate	1.26×10^{-2} dd^{-1}
k	Host carrying capacity	4.1
	Disease system parameters	
β	Infection rate	2.08×10^{-2} dd^{-1}
γ	Rate of becoming infectious	$1/266$ dd^{-1}
μ	Rate of recovering from infection	$1/456$ dd^{-1}
ν	Decay rate of primary inoculum	8.5×10^{-3} dd^{-1}
Ψ	Initial infected leaf area in lower canopy ($P(0)$)	0.0109
	Fungicide parameters	
ω	Fungicide maximum effect	1
θ	Fungicide dose response curvature	9.6
δ	Fungicide decay rate	1.11×10^{-2} dd^{-1}

qualities include the effectiveness of control, strength of evidence, economic factors, implementation factors, and current and potential use.

Of the IPM measures identified in the AHDB review, we restricted our examination to those that could be reasonably studied by our mechanistic system model (we expand on the selection of IPM measures for analysis in Section 4). As such, we define the 'IPM regime' in this study to be the use of crop residue burial, delayed sowing, variety mixtures, and biocontrols. We implemented each of these IPM measures into the *S. tritici* model based on a biological understanding of how the control acts and experimental data of their impacts.

2.4.1 Residue burial

Debris and stubble from the previous year's plants are commonly left in place between seasons. As a consequence, subsequent years' crops are planted among the residue from previous years. This practice offers certain benefits to growers, such as conserving soil moisture and reducing costs associated with clearing or tilling the field between seasons [18, 19]. However, if infection was present in the previous season, infected tissue can remain on the debris and contribute to the initial infection of the lower canopy in the next season [19–22]. Ensuring that the previous season's residue is buried before new crops are sown therefore has the potential to reduce the amount of inoculum at the beginning of the season, which could subsequently reduce the size or severity of the outbreak.

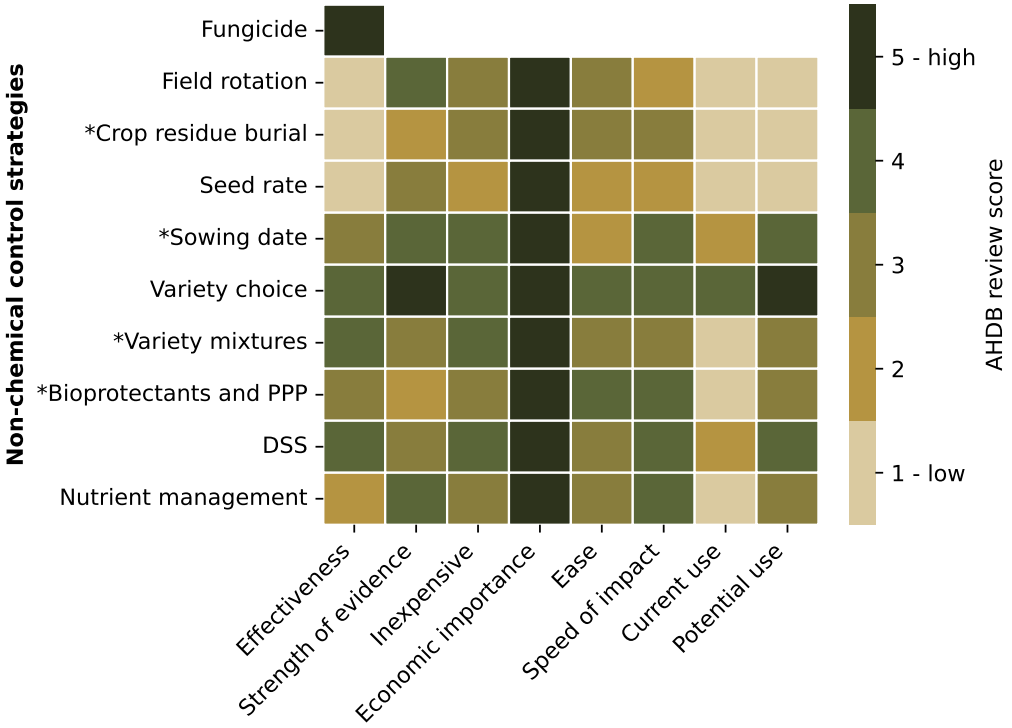


Fig. 2. IPM data for Septoria leaf blotch of wheat from the 2021 AHDB Research Review. Blake *et al.* [18] identified and scored IPM measures applicable for this disease by expert opinion on eight qualities, concerning effectiveness of the control, economic factors, and ease/speed of implementation. As in the original review, we display the effectiveness of fungicide for comparison. Control measures included in our study are indicated by a * preceding the control strategy name.

Our implementation of residue burial into the model system assumed that this practice would reduce the amount of inoculum initiating *S. tritici* infection in the lower canopy, thereby reducing the level of initial infection in the P class. With Ψ being the base initial value of P (Table 1), we assumed that residue burial resulted in an initial value of $pq\Psi + (1-p)\Psi$, where p is the proportion of initial infection from debris in the field (rather than from other sources, such as other fields' debris or crops), and q is the contribution to inoculum which remains after debris is buried. Brokenshire [20] found that pycnidiospores on unburied debris had a survival rate of 65% after 50 days, while pycnidiospores on buried debris had a survival rate of less than 10% [23]. Based on these empirical data, we considered a range of values for q from 0 to $\frac{0.1}{0.65} = 0.154$ (to 3 significant figures).

The highest reduction in infected leaf area seen in the lower canopy due to debris removal is 75%, seen by Suffert and Sache [22] in the initial stages. However, this reduction is quickly overcome as external sources of infection become more important than debris in contributing to infection in the lower canopy, suggesting that the impact of debris removal could be transient in terms of long-term dynamics [22]. As such, we considered values of p that resulted in a disease reduction between 0 and 75%. Each resulting range for p depended on the choice of q (Fig. S1). The smallest possible range of p was $(0, 0.899)$, corresponding to $q = 0$ (when the burial of debris prevents all of its contribution to initial inoculum), and the widest possible range of p was $(0, 1)$, corresponding to $q > 0.101$. With no clear choice for the 'default' value of p, we chose $p = 0.1$ based on the strength of evidence that debris removal has a transient impact, and the AHDB review that scored its effectiveness as low (Fig. 2, [18]). We examine in the Discussion the implications of the uncertainty and wide parameter ranges

computed here (see Section 4).

2.4.2 Sowing date

Sowing date has been shown to have an effect on the severity of *S. tritici* outbreaks [24], with current AHDB guidance suggesting that later sowing increases a crop's level of potential resistance to *S. tritici* infection [25]. In many cases, while late sowing results in some level of yield penalty, it also allows the crop to avoid the peak level of inoculum that occurs earlier in the season. As a consequence, the severity of the outbreak can be reduced [25]. We do not account explicitly for reduced crop establishment due to late sowing, which is elaborated on in the Discussion (Section 4).

We implemented late sowing into the model system by introducing a scalar r_Ψ in $[0, 1]$. The scalar r_Ψ allowed us to adjust the magnitude of the initial value of infection in the lower canopy (state P), reflecting a reduced exposure to inoculum. We also shifted the initial time of leaf emergence in the model by the appropriate number of degree-days. Late sowing is described in terms of (calendar) days delay after 20th September, matching the assumed sowing date in the Hobbelen *et al.* [9] model. We assumed the default value for sowing delay to be 30 days late, reflecting the AHDB's average 'late sowing' date of 20th October, and that the upper limit for late sowing would be mid- (15th) November [26].

To compute the reduction in inoculum exposure over time, we fit an ODE population model to temporal data on the prevalence of immature, mature and fully discharged pseudothecia from 1995/6 [27]. We performed the fitting to these temporal data by minimising the difference between the data and the model output (Fig. S2). We assumed the prevalence of inoculum was proportional to the prevalence of mature pseudothecia over time. This assumption means the relative amount of inoculum exposure a plant has is proportional to the area under the mature pseudothecia curve during the post-sowing period. Late sown crops will therefore evade the early period of inoculum release, reducing their relative exposure (Fig. S3). We assumed that the level of inoculum exposure r_Ψ on the first possible sowing date (i.e. 0 days delay) was 1. Therefore, the values of r_Ψ for the default delay of 30 days, and the maximum delay of 56 days, were 0.856 and 0.303 respectively.

2.4.3 Variety mixtures

Monoculture farming is a very widely used practice, offering greater uniformity of product for consumers and often simplifying the large-scale growing and harvesting process. However, the genetic uniformity in a varietal monoculture puts crops at greater vulnerability for disease [18, 28, 29]. Increasing genetic diversity within a crop has been shown to reduce both the spread and severity of a disease outbreak [28]; these outcomes are achieved because of a reduction in the probability that infection is successfully transmitted from one plant to its neighbour [29].

To implement variety mixtures in our transmission model, we assumed the effect of sowing a field with a variety mixture (rather than in a monoculture) multiplied the transmission rate β by a scalar r_β in $[0, 1]$. As varietal diversity affects both the ability to receive infection (in the S state), and transmit infection (from the I and P state) we amended the transmission term to $\frac{r_\beta^2 \beta S(I+P)}{A}$. Note that the term (r_β^2) allows for a more concise way of writing expressions using only r_β in the later, multi-field simulations.

To quantify the reduction in *S. tritici* infection that can be achieved under variety mixtures, we used the results from Kristoffersen *et al.* [28]. In their meta-analysis of 406 trials that used varietal mixtures (of which the results from 221 trials were ultimately used), they found that the 'mixing effect' reduced disease severity by a mean of 9.0%, with lower and upper bounds of 1.0% and 16.3%, compared to the

most-grown varieties. For our base model representing an untreated outbreak, we selected the results for untreated trials. Under the assumption that the model presented by Hobbelen *et al.* [9] represents behaviour of a typically grown variety, we selected the results that compared the varietal mixture outcomes to those of the 'most-grown varieties'. Given that the use of resistant varieties is already widespread alongside the use of fungicides [18, 30] (Fig. 2), these 'most-grown varieties' are typically chosen for their resistant properties. As such, it would be unrealistic to contrast an IPM regime that uses resistant varieties with a fungicide regime that does not use resistant varieties. Accordingly, in our model we implicitly assumed the following two conditions: (i) that varieties planted by the fungicide-using growers were chosen for their resistance, and (ii) variety mixtures used in the IPM regime were a mixture of commonly-used resistant varieties.

We assessed disease severity in our model by integrating between GS69 and GS75, as this is when Kristoffersen *et al.* [28] state that the majority of trials carry out their disease assessment. Fitting r_β to the 1.0%, 9.0% and 16.3% disease reductions resulted in values of 0.997, 0.976 and 0.958, respectively. We took 0.976 as our default parameter value for biocontrol.

2.4.4 Biocontrols

A number of biofungicide and bioprotectant products are either commercially available, or identified as potentially effective in research studies [31–33]. Many of these products use either bacteria, lower-risk chemical compounds, or other biological agents, to enact either an actively fungicidal effect, or a protectant effect.

These are often applied using the similar methods as chemical fungicides, at similar growth stages, and act through similar mechanisms, though offer a lower level of control. As such, we implemented biocontrol in the model with an analogous form to fungicide (Eqs. (1) and (2)). We used experimental results from 2004 and 2006 reported by Kildea *et al.* [33]. The authors found that *Bacillus megaterium* was capable of inhibiting the development of *S. tritici* in small-scale field trials. Treatments were applied at GS31 and GS39, to align with conventional fungicide treatment procedures. We used the function

$$\hat{\epsilon}(B) = \hat{\omega}(1 - e^{-\hat{\theta}B}),$$

with *Bacillus megaterium* treatment solution concentration

$$B(t) = \begin{cases} 0 & \text{if } t < T_{31} \\ e^{-\hat{\delta}(t-T_{31})} & \text{if } T_{31} \leq t < T_{39} \\ e^{-\hat{\delta}(t-T_{31})} + e^{-\hat{\delta}(t-T_{39})} & \text{if } t \geq T_{39}, \end{cases}$$

where we fit for the parameters $\hat{\omega}$ (maximum reduction in disease), $\hat{\theta}$ (dose response parameter) and $\hat{\delta}$ (decay rate) by minimising the distance between the function and the data from Kildea *et al.* [33]. After fitting both the 2004 and 2006 experimental data, we chose the results from the 2004 data due to the quality of fit (Fig. S4). Our resultant parameter set was $(\hat{\omega}, \hat{\theta}, \hat{\delta}) = (0.696, 10.3, 0.0388)$. We acknowledge that for this IPM control we have a set of fixed parameter values only, without a potential range. Furthermore, the experimental data only looks at infection prevalence on the flag leaf (the top leaf), while our model would ideally be fit to prevalence in the whole upper canopy. Data limitations prohibited the acquisition of a potential range for these parameters, with further comment on these challenges given within the Discussion (Section 4).

2.5 Simulation outline

We produced all model simulation scripts and figures in Python 3.9.12. Code and data for this study are available at https://github.com/emrvincent/IPM_s_tritici_modelling.

2.5.1 Individual IPM measures

To allow us to assess the relative effectiveness of each IPM measure, as well as the potential variability in outcomes between IPM measures and due to parameter uncertainty, we first ran simulations with each IPM measure implemented individually, in an isolated field. We performed model simulations for each of the four IPM measures using the base ODE system (given in Eq. (5)) with modifications relevant to that IPM measure, as described in Sections 2.4.1 to 2.4.4.

For each control measure we output both the infection prevalence over time (defined as the percentage of leaf area infected) and the relative yield. For comparative purposes, we also generated the same metrics for a 'no control regime' and a 'fungicide regime'. Note that although the no control regime is not typically used in practice, we considered it here for comparative purposes. We implemented the fungicide regime as described in Section 2.3.

2.5.2 Full IPM regime

We next ran simulations to compare three possible field regimes, each employed in an isolated field: the 'no control regime', a 'fungicide regime' (implemented as described in Section 2.3) and a collective 'IPM regime'. The IPM regime corresponded to the simultaneous implementation of all four of our IPM control measures; residue burial, sowing delay, variety mixtures, and biocontrols.

For each regime we output the infection prevalence over time and the relative yield. The system of ODEs describing the model under the IPM regime is

$$\begin{aligned}
\frac{dS_M}{dt} &= g_M(A_M, t) - \sigma(t)S_M - r_\beta S_M(1-\hat{\epsilon}(B))\lambda_F(t) \\
\frac{dE_M}{dt} &= r_\beta S_M(1-\hat{\epsilon}(B))\lambda_F(t) - \sigma(t)E_M - \gamma E_M(1-\hat{\epsilon}(B)) \\
\frac{dI_M}{dt} &= \gamma E_M(1-\hat{\epsilon}(B)) - \mu I_M \\
\frac{dR_M}{dt} &= \sigma(t)(S_M + E_M) \\
\frac{dD_M}{dt} &= \mu I_M \\
\frac{dP_M}{dt} &= -\nu P_M,
\end{aligned} \tag{4}$$

where $\lambda_M(t) = \frac{\beta r_\beta (I_M + P_M)}{A_M}$. As in the single field fungicide model, g_M is identical to the function g in the base system, whilst parameter values and $\sigma(t)$ are also the same as in the base system [9] (Table 1 and Section S1). In this case, the initial conditions at the beginning of a year's growing season were $(S, E, I, R, D, P) = (0.05, 0, 0, 0, 0, r_\Psi(pq\Psi + (1-p)\Psi))$, with the initial condition for P modified by the control measures. All IPM parameters that were derived in Sections 2.4.1 to 2.4.4 are listed in Table 2, and we ran this simulation using the default values of all four control measures.

2.5.3 Multi-field setting

In the multi-field setting, we explored the scenario in which fields are not isolated, but receive infection from all other fields in the system (Fig. 3). In each simulation, we assumed a fixed proportion ρ of fields were treated using the IPM regime, while the remaining proportion $(1-\rho)$ of fields were treated using the fungicide regime. To implement this, we amended the growth terms g_F and g_M so that the maximum leaf area in each field type was scaled by $(1-\rho)$ and ρ respectively;

Table 2. IPM parameter values.

Parameter	Definition	Default	Possible range		Ref
r_β	Scalar for transmission using variety mixtures	0.976	(0.958,0.997)		[28]
r_Ψ	Scalar for initial ascospore exposure	0.856	(0.303,1)		[27]
p	Debris contribution to initial infection	0.1	(0,1)		[22]
q	Infection remaining after debris burial	0.154	(0,154)		[20]
$\hat{\omega}$	Maximum reduction in disease from biocontrol	0.696	(-)		[33]
$\hat{\theta}$	Shape parameter of the dose response curve	10.3	(-)		[33]
$\hat{\delta}$	Biocontrol decay rate	0.0388	(-)		[33]
	IPM regime*	**Low**	**Medium**	**High**	
-	r_β	0.997	0.976	0.958	-
-	Days delayed (r_Ψ)	15 (1)	30 (0.856)	38 (0.668)	-
-	p	0	0.1	0.899	-
-	q	N/A	0.154	0	-
-	Time (s) of biocontrol application	None	T_{31}	T_{31}, T_{39}	-

* Parameter sets used for the low, medium, high-intensity IPM regimes.

$$g_F(A,t) = \begin{cases} 0 & \text{if } A = 0 \\ R(k(1-\rho) - A) & \text{otherwise,} \end{cases}$$

and

$$g_M(A,t) = \begin{cases} 0 & \text{if } A = 0 \\ R(k\rho - A) & \text{otherwise.} \end{cases}$$

We also applied the same scaling to the initial conditions of each field type. To implement between-field infection, we amended the infection terms for both field types to

$$\lambda_M(t) = \lambda_F(t) = \frac{\beta r_\beta (I_M + P_M) + \beta(I_F + P_F)}{A_M + A_F},$$

thereby assuming that contact was well-mixed between both field types.

We compared the relative yields of each field type for ρ ranging between 0.02 and 0.98, with an increment of 0.02. We output the results for the default IPM parameter set (and under the base values for the fungicide parameters), as well as exploring the system behaviour as we varied the intensity of both regimes (fungicide and IPM). Specifically, we considered all combinations of three different fungicide regime intensities ($\omega = 0.5$, $\omega = 0.75$, $\omega = 1$) and three different IPM regime intensities (low, medium, high), giving a total of nine fungicide-IPM intensity combinations.

We subjectively defined our three IPM regimes, informed by the range of potential values that our fitting produced for each intervention (selected parameter values for each regime are given in Table 2).

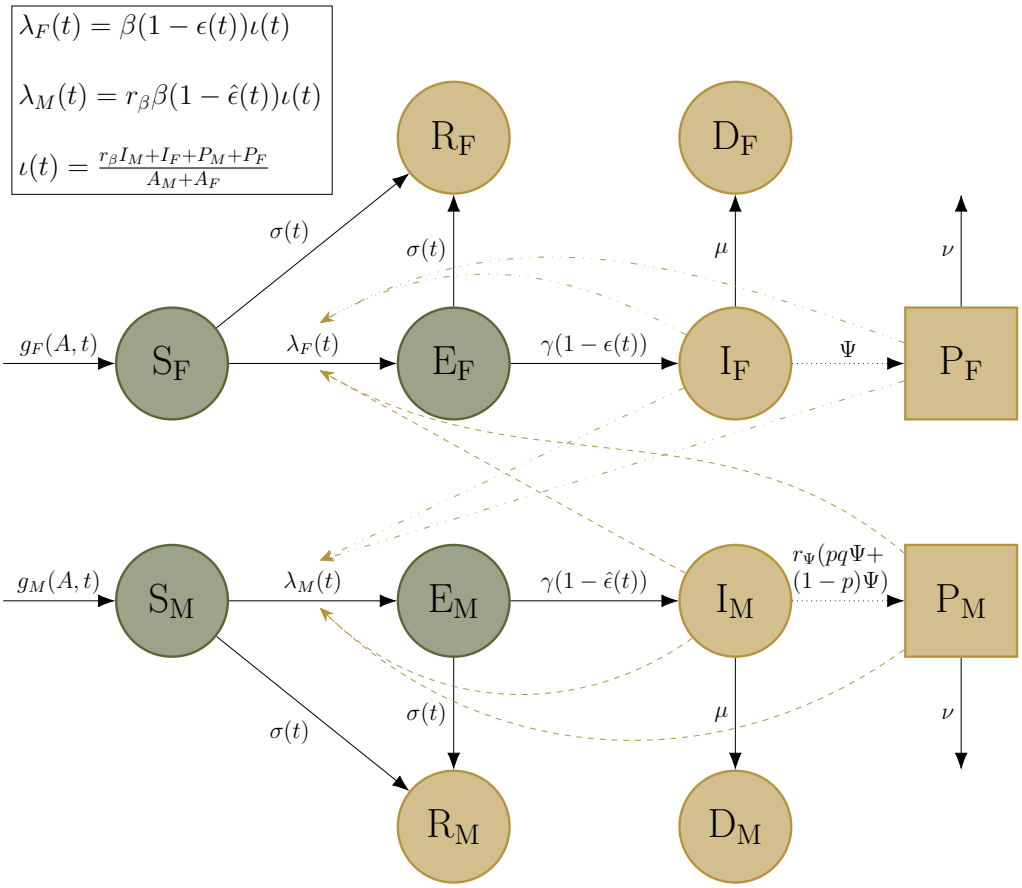


Fig. 3. Schematic for the multi-field *S. tritici* dynamics model. States are divided into fields under the fungicide regime (F subscript) and those under the IPM regime (M subscript). As a result of the control strategies, compared to the base model the two regimes have different rates on the solid transition arrows and on the dotted between-season arrows. We also introduce transmission between the two field groups.

Our assumptions for the parameter values selected for each regime intensity were as follows. For variety mixtures, the medium intensity regime used the default value (0.976), and the low and high intensity regimes used the boundary values (0.997 and 0.958, respectively). For delayed sowing, in the low intensity regime we assumed only half of the default delay (15 days) would be implemented, in the medium intensity regime used the default value of a 30 day delay, and for the high intensity regime we used the value that optimised yield (38 days delay). For residue burial, implementation of the low intensity regime assumed that residue contributed negligibly ($p = 0$) to the initial inoculum, the medium intensity regime used the default values ($p = 0.1$, $q = 0.154$), and the high intensity regime assumed that residual burial contributed the maximal value our fitting allowed ($p = 0.899$, $q = 0$). Finally, for biocontrol we chose to vary the number of applications in each regime, with the number of applications a commonly varied parameter value for that type of control [31]. We assumed the low intensity regime had zero applications, the medium intensity regime had one application and the high intensity regime had both experimental application times.

3 Results

3.1 Individual IPM measures: Sowing date has greatest impact on yield

In our initial analysis we modelled four different IPM control measures (residue burial, delayed sowing, variety mixtures, biocontrol) applied to an isolated field. We were particularly interested in how their use may impact expected yield and variability in yield (resulting from parameter uncertainty) over a single growing season,

Residue burial
When implementing residue burial as the sole control measure, under our default parameter set ($p = 0.1$, $q = 0.154$) it offered little improvement compared to having no infection controls ($p = 0$); we observed 22.3% infection at peak prevalence versus 21.6% in the no control scenario (Fig. 4(a)), and a relative yield of 0.731 (Fig. 4(e)). Under the best-case parameters ($p = 0.899, q = 0$), the peak level of infection was reduced to 6% and it gave a relative yield of 0.946.

Delayed sowing
The second IPM control strategy we considered was delayed sowing. Delaying sowing by 30 days substantially decreased the peak prevalence of infection, to less than 5% (Fig. 4(b)). It also resulted in a final relative yield above 0.95 (Fig. 4(e)). A sowing delay of 38 days resulted in the highest possible relative yield of 0.967, and a peak of infection below 2.5%. The minimum sowing delay of 0 days (equivalent to the no control scenario) gave the lowest yield from this set of scenarios (0.717). Whereas for the maximum sowing delay we considered, 56 days, we observe an infection prevalence below 1%. Although this is the lowest infection prevalence achieved across our considered parameter range, it did not maximise yield (having a relative yield of only 0.884). The low prevalence and lower yield is a consequence of the extreme delays to sowing, meaning the plants do not spend enough time experiencing growth before senescence to reach their maximum (healthy) tissue area. Delayed sowing also resulted in a delay to the peak infection time. However, the impact of delaying by one extra day is larger early in the season than late in the season, because the number of degree-days lost to each calendar day decreases later in the year.

Variety mixtures
For the variety mixture control measure, the default parameterisation had a peak infection prevalence of 20.2%. The best-case scenario had a 1.6% lower peak prevalence of 18.6%. The worst-case scenario had a 1.9% higher prevalence at its peak of 22.1% (Fig. 4(c)). When considering the yield outcome, the default parameterisation results in a relative yield of 0.749. The best- and worse-case parameterisation increased the relative yield by 0.023 and decreased the relative yield by 0.028 respectively (Fig. 4(e)).

Biocontrols
The last IPM control strategy we considered was biocontrol. Unlike the three other control measures, we ascertained the default scenario to be the best-case scenario (with two sprays being applied); each of the other two scenarios investigated only involved one spray. Additionally, this was the only IPM measure in our analysis that had discrete options (as we assumed the two sprays will either be applied, or not applied, at fixed times). Applying the biocontrol at only GS31 gave a peak infection prevalence of 17.0%. This contrasts with applying the biocontrol at only GS39, which gave a peak prevalence of 19.3%. A two spray application lowered the peak infection prevalence to 14.1% (Fig. 4(d)). The corresponding relative yields for one spray at GS31 and GS39 were 0.809 and 0.771, respectively. The relative yield was higher given two spray treatments (0.851).

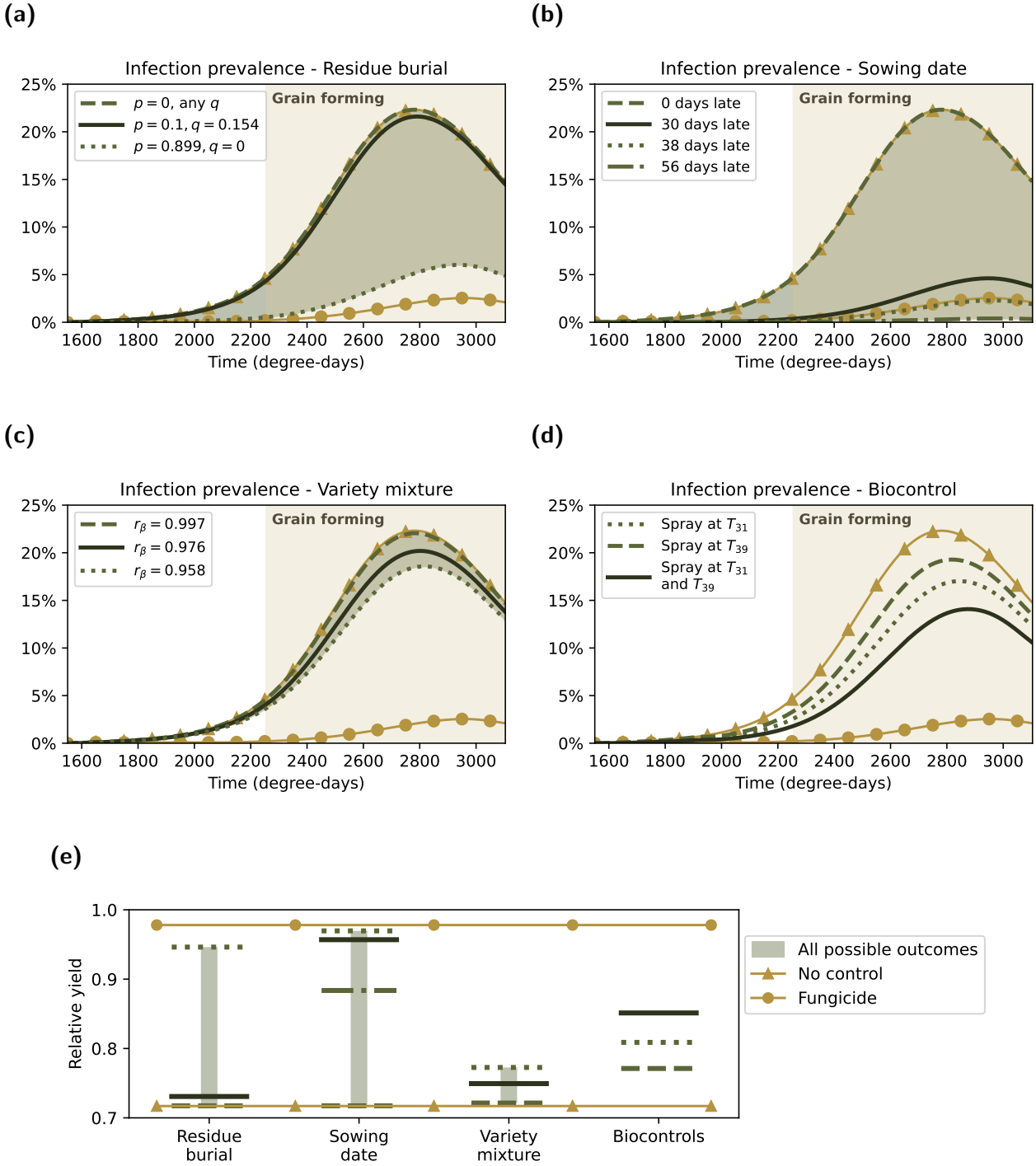


Fig. 4. Single season outcomes for each IPM measure implemented individually. For each control, we show the results for the default parameter set by a dark green solid line, the boundary or alternative-implementation results in medium green dashed and dotted lines and, where applicable, the continuous range of possible outcomes the shaded pale green ribbons (we list parameter values and ranges in Table 2). We also show results for the 'no control' regime and 'fungicide' regime (implemented as described in Section 2.3). Panels **(a-d)** show the infection prevalence (% leaf area infected) with the following sole IPM control measure applied: **(a)** residual burial; **(b)** delayed sowing; **(c)** variety mixtures; **(d)** biocontrol. Panel **(e)** displays the yield at the end of a season under each of the individual IPM control measures. The legend outside panel **(e)** contains information relevant to all subfigures **(a-e)**, while the legends contained within panels **(a-d)** contain information applicable to each particular IPM measure.

Comparison across individual IPM measures
Our considered range for the variety mixture parameter gave less variability in possible outcomes compared to the variation across the potential scenarios using residue burial or delayed sowing. Across all considered scenarios, we found a maximum relative yield of 0.967 by delaying sowing for 38 days.

Comparison with no-control and fungicide regimes
We initially note that the worst-case implementations of residue burial and sowing date were identical to predicted relative yield for the no control scenario (Table 2), and therefore produced the same modelled relative yield outcomes. Outside of these two IPM measure parameterisations, all yield values attained by all individual IPM measures were greater than the yield with no control, but less than the yield attained under a fungicide regime (Fig. 4(e)). In terms of infection prevalence, almost all scenarios (that were not equivalent to the no control scenario) result in lower infection prevalence than the no control case, but consistently higher infection prevalence compared to the fungicide control regime (Figs. 4(a) to 4(d)). A notable exception was when sowing was delayed by around 37 days or more, which resulted in the a projected infection prevalence that was consistently lower than that attained under a fungicide control regime (Fig. 4(b)).

3.2 Single field setting: Full IPM regime offers comparable control to a fungicide regime

We next considered the implications for infection prevalence and yield for three different field regimes (as if implemented on an isolated field): a 'no control regime', a 'fungicide regime' (implemented as described in Section 2.3) and an 'IPM regime' (with simultaneous implementation of four IPM control measures; residue burial, sowing delay, variety mixtures, and biocontrols).

In terms of infection burden, the default parameter IPM regime (Table 2) returned a lower peak infection prevalence (1.33%) than the fungicide regime (2.54%). Both of these control regimes vastly outperformed the no control scenario, which had a peak infection prevalence of 22.3% (Fig. 5(a)). The time of the peak in infection prevalence was also slightly delayed under the control regimes compared to the no control regimes.

For relative yield, the IPM regime estimate of 0.984 marginally outperforms the fungicide regime (relative yield of 0.978) and markedly outperforms the no control regime (relative yield of 0.717) (Fig. 5(b)). Looking at temporal dynamics of the S class under the three regimes well-illustrates both the loss in yield under the no control regime, and the similarity in yield between the fungicide and IPM regimes (Fig. S5). As yield was computed by the area under the susceptible leaf tissue (S) curve during the grain forming period, the much higher infection prevalence in the no control regime results in less area under the susceptible leaf tissue (S) curve in this scenario. We also saw that, despite the delayed emergence of the upper canopy under the IPM regime, the susceptible leaf area grows to almost the same maximum as seen in the fungicide regime, just as the grain forming period begins. As such, the shape of the susceptible leaf tissue (S) curve (and thus the area under it) during the grain forming period is very similar in both regimes (Fig. S5).

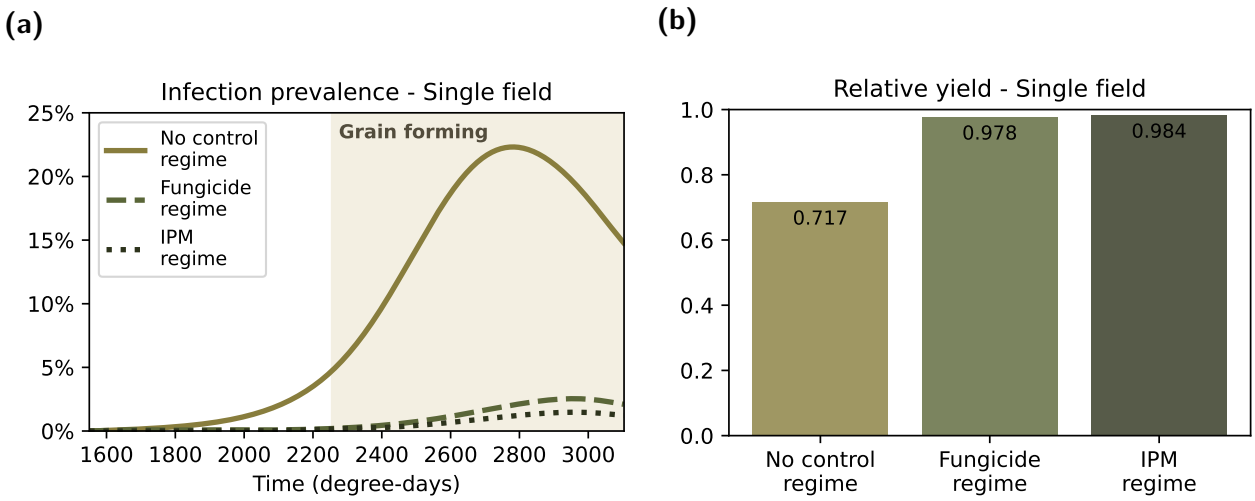


Fig. 5. Single season outcomes under three different control regimes. Our three control regimes were 'no control', 'fungicide', and 'IPM' (as described in Section 2.4). **(a)** Infection prevalence (% leaf area infected) over time under each regime; **(b)** Relative yield at the end of a season under each regime.

3.3 Multi-field setting: Presence of IPM users can improve yield outcomes for all growers

The final component of our analysis involved looking at the outcomes in a system with multiple fields. The multi-field setup allowed for transmission of infection between fields. We examined the effects on the system dynamics of having a varied proportion of fields use an IPM control regime (ρ) and the remainder ($1 - \rho$) adopting a fungicide control regime.

For values of ρ between 0.02 and 0.92, the fields using the IPM regime achieved a higher relative yield than the fields using the fungicide regime. As ρ increased, the marginal advantage of IPM over fungicide diminished, and for $\rho \geq 0.94$, farms using the fungicide regime achieved a higher yield than farms using the IPM regime. At our initial value of $\rho = 0.02$, the IPM and fungicide fields had approximate relative yields of 0.979 and 0.978 respectively, whilst at our final value of $\rho = 0.98$ the respective relative yields were both 0.984 (to 3 significant figures). As ρ increases from 0.02 to 0.86, increasing the proportion of farms using IPM increased the relative yield for both field types. In contrast, relative yields decreased from $\rho = 0.88$ onward for the IPM fields and from $\rho = 0.9$ onward for the fungicide fields (Fig. 6(a)).

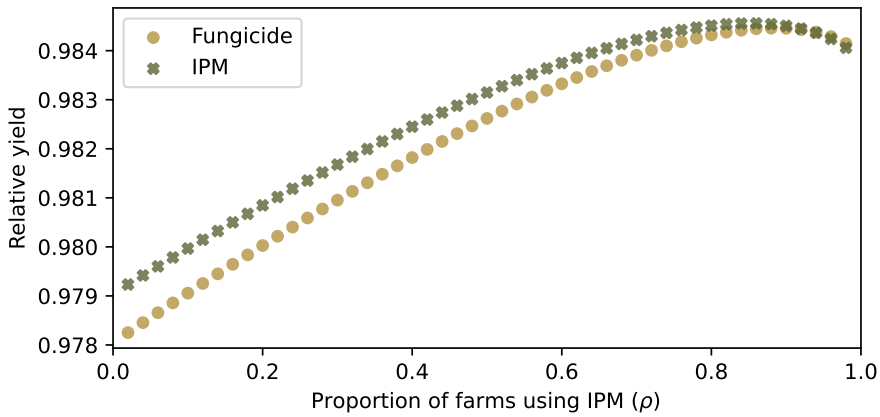
To conclude, we investigated the relative yield obtained with different intensities of the two control regimes. At a low level of IPM intensity, and for all three levels of fungicide performance ($\omega = 0.5$, 0.75, 1), increasing ρ decreased yields under either form of control (Fig. 6(b), left column). However, a higher intensity fungicide regime improved yields for both field types, particularly at lower values of ρ. Increasing ω from 0.5 to 1 gave estimated ranges of relative yield from the IPM fields between 0.885-0.905 (when $\omega = 0.5$) and 0.890-0.976 (when $\omega = 1$), and estimated ranges of relative yield from the fungicide fields between 0.886-0.909 (when $\omega = 0.5$) to 0.907-0.978 (when $\omega = 1$).

For the medium intensity IPM regime, in combination with either a low or medium intensity fungicide regime (Fig. 6(b), middle column, middle and bottom row) increasing ρ resulted in greater yields for both field types. Furthermore, for all tested values of ρ, IPM fields had higher yields compared to fields using fungicide. We found differing yield outcomes with respect to ρ when the medium IPM regime was combined with a high fungicide intensity (Fig. 6(b), middle column and top row). Firstly, for all tested values of ρ, fields applying fungicide had greater yields than fields using IPM. Secondly, only when increasing ρ up to and including $\rho = 0.78$ was there an increased yield for both control types. When ρ exceeded 0.8 the additional uptake of IPM resulted in a reduced yield for the IPM fields, and when ρ exceeded 0.82 a similar decrease occurred in the yield for the fungicide fields (a similar phenomenon occurred in the default scenario).

With a high IPM regime intensity, yields increased when there was a greater proportion of growers using IPM (Fig. 6(b), right column). However, with respect to an increase in the fungicide intensity ω, for an increasing proportion of growers using IPM (i.e. higher ρ) those growers using fungicide had greater relative increases in yield compared to growers using IPM. This dynamic meant that for a low intensity fungicide regime, although IPM fields had a greater relative yield than fungicide for all values of ρ, when $\rho = 0.98$ their relative yields became almost equal (at 0.986 and 0.983; see Fig. 6(b), right column, bottom row). For the medium intensity fungicide regime ($\omega = 0.75$), for $\rho = 0.32$ and above there was a transition from higher yields in the IPM fields to higher yields in the fungicide fields (Fig. 6(b), right column, middle row). In the high intensity fungicide regime ($\omega = 1$), fungicide fields had higher yields than IPM fields, irrespective of the value of ρ (Fig. 6(b), right column, top row).

Lastly, we note in all cases of the high intensity IPM regime, and almost all cases of the medium intensity IPM regime (except for high values of ρ when $\omega = 1$), a greater presence of IPM fields improved the relative yield for both field types (Fig. 6(b), middle and right columns).

(a)



(b)

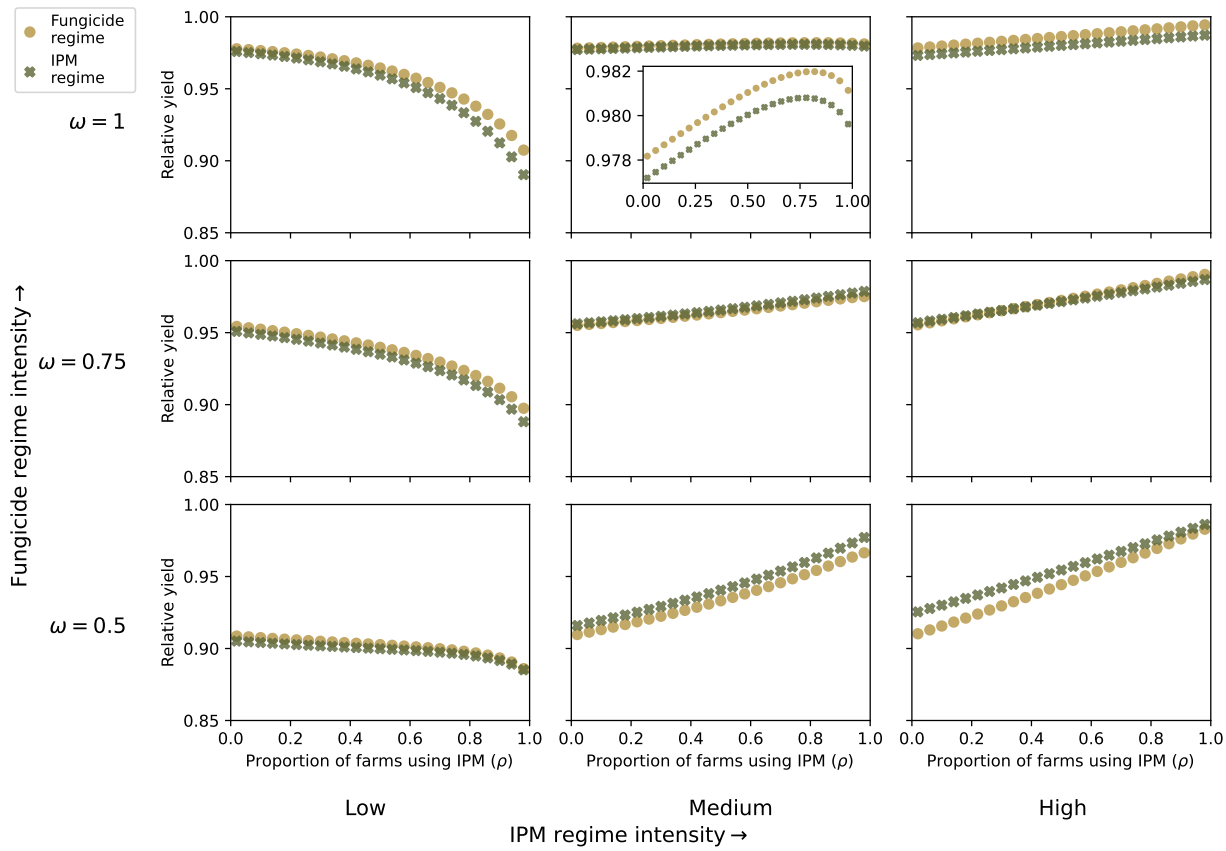


Fig. 6. Single season yield outcomes in the multi-field setting. We display end-of-season relative yield for an individual field, within a multi-field system. The parameter ρ corresponds to the proportion of fields using the IPM control regime, with the remaining proportion of fields (1 - ρ) using a fungicide control regime. We present relative yield outcomes versus ρ for: **(a)** An IPM control regime using the default values from Table 2 and fungicide control regime using the base parameters from Table 1; **(b)** Three different intensities of IPM control regime and three different intensities of fungicide control regime. Low, medium, and high intensity IPM regimes are defined in Table 2. Low, medium, and high intensity fungicide regimes are defined by $\omega = 0.5$, $\omega = 0.75$ and $\omega = 1$.

4 Discussion

4.1 Findings

With greater constraints on the use of fungicide, the effectiveness of IPM as a control for arable diseases has become increasingly relevant. Previous work has identified IPM measures that are effective at controlling *S. tritici* and explored their implementation in practice [24, 31, 33, 34]. By incorporating these existing data into an epidemiological model, we explored the potential outcomes of these IPM measures on the overall disease system. We found that individual IPM measures offered differing levels of control, but that an 'IPM regime' consisting of the simultaneous implementation of multiple methods offered comparable disease control and yield to a standard fungicide regime. Furthermore, when IPM was implemented at least at medium intensity, the presence of IPM-using fields benefitted all growers in a multi-field system. An additional finding was that, in general, improved data on different IPM measures is critical to the ability to judge its effectiveness as a regime, and which measures to deploy concurrently.

For the use of single IPM controls, the most striking findings were for delayed sowing and debris burial. Our results suggest that when implemented independent of other IPM measures, delayed sowing has the potential to reduce the severity of *S. tritici* outbreaks. This outcome corroborates experimental studies [35], in addition to the AHDB review (Fig. 2, [18]) that assigned 'sowing date' an effectiveness score of 3/5 (with 4/5 being the maximum effectiveness score given to any IPM control of *S. tritici*). In contrast to the study by Bekele and Kassaye [35], which found that the decrease in disease severity had no effect on yield, we found an increased yield. Our model findings for delayed sowing contrast with those for the implementation of residue burial, where it offered little control over the size of the final outbreak. This finding is in agreement with the findings of Morais *et al.* [36], Suffert and Sache [22] and the AHDB review data (Fig. 2, [18]). Residual burial providing little control over amount of infection during an outbreak appears to result from the fact that even when the burial of debris is very effective at eliminating inoculum from this source ($q = 0.154$ in our default parameter set), larger-scale sources of primary inoculum are likely to play a bigger role in initiating an outbreak [22]. Furthermore, the amount of local initial inoculum is unlikely to meaningfully limit infection development, as the presence of larger-scale sources of inoculum during the emergence of the first leaf mean that its infection is likely to occur almost immediately upon its emergence, regardless of the presence of early within-field inoculum [36]. It is important to note that the effectiveness of residue removal varies a lot depending on how important debris is as a contributor of inoculum. Although its contribution is believed to be low [22, 36, 37], it is known to be extant [20, 38], and has not been well-quantified. We therefore encourage the collection of additional empirical data to help quantify this process.

In the single field setting, we found that IPM was able to serve as an effective measure of control against *S. tritici*, in agreement with previous theoretical and experimental findings about *S. tritici* [28, 35, 39] and other diseases [39–41].

When considering the multi-field setting, we found that increasing the presence of IPM regimes within the system can offer increased yields to all growers. This was the case if the IPM regimes are sufficiently intense; that is, our definitions of medium or high intensity (a range which also includes our default parameterisation). We hypothesise these yield benefits arise when more growers use IPM control because, while the fungicide regime offers protection only to the target field, the IPM regime offers control to other fields in the system by several mechanisms: (i) reducing the force of infection it emits due to the use of variety mixtures (r_β); (ii) reducing the initial infection due to debris removal; (iii) reduced overall ascospore exposure, and; (iv) the asynchronous timing of initial infections between the two farm types.

We also identified circumstances ($\omega = 1$ and either the medium intensity, or our default IPM regime) where if the proportion of fields using IPM was already at a high level (above 0.8), then further increasing the proportion of farms who use IPM could result in a decrease in yield. This appears to occur due to the fact that under these circumstances, the proportion of farms using fungicide becomes too small to realise the benefits that the fungicide control provides; that is, to reduce infection in those fields, which occurs just prior to the emergence of the IPM fields. In other words, having few farms using fungicide in the scenarios where fungicide is very effective ($\omega = 1$) reduces the possible benefit this control offers to the system, while also disrupting the 'asynchronicity' effect, as the majority of infection emerges with the delayed-sown IPM fields.

Our modelling approach is similar to that taken in several other studies in considering a mechanistic, S-I system of equations where multiple strategies are present, and infection is spread between all fields in the system [17, 42]. Although we were not able to identify any studies that have previously implemented this type of method to compare an IPM-only strategy with a fungicide-only strategy, Murray-Watson and Cunniffe [17] investigated the choice between resistant and tolerant varieties (which would be considered IPM measures), and McQuaid *et al.* [42] considered the use of clean planting material in the growing of cassava (which would also be considered an IPM measure). Murray-Watson and Cunniffe [17] found that increasing the proportion of growers using resistant varieties decreased the infection level in both users and non-users of the control. We observed a similar phenomenon in our system; an increasing proportion of IPM growers offers increased benefits to other farmers in the system, in addition to the users themselves. However, the opposite trend was present in their results for the use of tolerant varieties; in this case, the increased presence of tolerant varieties resulted in higher overall infection presence for control users. This was because tolerant varieties still harbour the pathogen, in constrast to resistant varieties which do not.

The previously mentioned studies focus primarily on grower behaviour dynamics in the system. We were able to identify few similar studies that have studied purely epidemiological modelling outcomes in the context of IPM. The system on which our model was based has been used to investigate the epidemiological outcomes of different control strategies for *S. tritici*. While much of this work investigates the development of resistant strains of infection under various regimes of fungicide control [9–12], one study by Taylor and Cunniffe [14] does consider the implementation of an IPM strategy. They found that disease resistant cultivars were able to decrease infection and increase yield, and we found improvements in the same outputs under the use of an IPM regime; although our IPM regime did not include resistant varieties, the IPM measure considered in this study. Nonetheless, our approach differs to theirs in that we found IPM to be an effective control in the absence of fungicide, while Taylor and Cunniffe [14] showed that an IPM measure can be used to reduce the quantity of fungicide which is needed for control. Other notable studies that take a modelling approach to the use of IPM are those by Tang *et. al* [40, 43, 44]. Similarly to our work, they found that sufficiently effective or well-implemented IPM regimes were capable of effectively suppressing pest population. These studies differ from ours in methodology in a number of ways however: in their consideration of pests and ours of infectious disease; their approach being that of dynamical system analysis and ours of numerical solutions; and theirs of considering an IPM regime which includes the use of chemical pesticide, while we consider the use of IPM and fungicide in the absence of one another.

4.2 Limitations

The model we have presented is necessarily a simplified representation of reality. It is therefore important that we consider the modelling assumptions made and their potential limitations. We elaborate here on four items. These are the implications of considering IPM and fungicide to be two completely distinct regimes, the exclusion of certain IPM measures from our modelling framework,

the potential compounding effect of concurrent implementation of multiple IPM controls, and the variability in IPM parameter ranges used in our model construction.

First, to make the comparison between the fungicide and IPM regimes more straightforward, we excluded any use of fungicide from our IPM regime, and vice versa. Although we recognise this as a simplifying assumption, and resulted in the omission of Decision Support Systems (DSS) - the strategic, limited use of chemical fungicide controls [45] - as a potential IPM measure in our study, it allowed us to more clearly contrast different IPM options with a common control method that did not use IPM. As addressed in Section 2.4.3, to further limit the overlap between these two regimes we also omit the explicit consideration of resistant varieties.

Second, we chose to include only a subset of the IPM measures identified in the AHDB review (Fig. 2, [18]) as having the potential to control *S. tritici* of wheat, in our model. This was done, as in the case of DSS, due to the unsuitability of including certain measures in our model. Field rotation was omitted because our model contains only a population-average, rather than characterising individual fields; and seed rate and nutrient management are omitted as these measures would affect plant population/leaf density in a way that cannot be readily implemented into our model. Given that each of these measures (DSS, field rotation, seed rate, and nutrient management) are identified as reducing overall infection in the system (at varying levels of effectiveness), it seems likely that were these additional control methods included in the system, they would offer at least as much suppression of infection as our present model.

For our implementation of delayed sowing we also did not explicitly account for a reduced yield due to failures in establishment. Although it is acknowledged that later sowing reduces the rate of successful seed establishment, this is typically accounted for by increasing the seed rate sown [26, 46, 47]. That being said, delayed sowing can still implicitly result in yield reductions within our model. Based on the date the crop is sown, some degree-day penalty is incurred to upper canopy emergence, ultimately reducing the growth period of the plant before senescence (Fig. S3).

Third, we did not have sufficient data to account for any compounding effect that these IPM measures would have when acting in combination. Nonetheless, we made subjective considerations for the implementation of each IPM measure when used in conjunction with other IPM measures. For example, we assumed that when sowing was delayed, the time at which biocontrol was applied shifted by the corresponding number of degree days. We also assumed that the reduction in inoculum due to sowing delay would act on both the $pq\Psi$ component, and the $(1-p)\Psi$ component of the initial condition. In other words, we assumed that late sowing would affect exposure to inoculum from both debris and other sources.

Lastly, we note that there is a high amount of variability in the certainty of some IPM parameters in our model construction. In the case of debris burial, although the relative contribution of local debris to the initial pool of inoculum has been qualitatively noted [22, 23], it has not been quantified in a way that allowed it to be integrated into our model. Suffert and Sache [22] do numerically compute the reduction in disease severity early in an outbreak, attributable to chopping or removing debris. However, we deemed these data as not suitable for our purposes as these methods of debris control may have different impacts compared to burial. Additionally, the experimental data in Suffert and Sache [22] was taken only in the lower canopy and at timepoints outside of our model timeframe. Similarly, there was limited experimental data to quantify the level of ascospore presence in fields throughout the season. Morais *et al.* [36] collected data this type, but only in the case of fields with debris (not those without), and it was not of a high enough resolution for use in our model (only 2 data points between October and December). These factors necessitated our assumption that the level of ascospore exposure was proportional to the presence of mature pseudothecia. In both of these

aspects, we would encourage the collection of further data, which would improve our understanding of the relative contributions of sources of inoculum over time during the initial stages of an *S. tritici* outbreak. In the case of biocontrols, there was almost no experimental literature that we could identify that quantified the reduction in disease severity due to the use of biocontrol on *S. tritici* on a field-scale, at multiple timepoints, although many have done this in-vitro [39, 48], or in-vivo on an individual plant level [39, 49, 50], at a small number of timepoints. Perelló *et al.* [32] and Mann *et al.* [31] are the only studies we have identified at the time of writing that have quantified the field-level impact of a biocontrol on *S. tritici* severity. These studies are themselves limited as both assessed disease severity at only one timepoint, which made them unsuitable for fitting the three necessary parameters in our model. To accurately predict their ability to control disease when employed by growers in practice, we advocate for the need for collection of experimental data on the effectiveness of biocontrol measures on a field-scale.

4.3 Further work

In the present work we considered a purely epidemiological model of the disease system. We have yet to account for any social or behavioural factors that would influence the choice of control adopted. Across the literature, it is acknowledged that profitability (of which one may consider cost, yield, time, and ease of implementation) is a major factor in growers' choice of control using IPM, or other methods [18, 51, 52]. The AHDB review data used in the present study (Fig. 2, [18]) also acknowledges the varying ease of implementation and cost that characterises different IPM measures. Deepening our understanding of adoption of plant disease controls and grower behaviour towards disease management is an inherently interdisciplinary challenge. There is consequently an urgent need to pursue interdisciplinary approaches to understand the epidemiological and behavioural factors behind such decision making.

In light of this, future work will focus on combining this epidemiological model with a behavioural model for growers intentions towards disease control. Development of a conjoined epidemiological-behavioural model will enable us to extend the analysis beyond a single growing season, as the initial conditions for subsequent seasons will be defined by both infection prevalence and the perceived success of each control strategy by growers in the previous season. This work would be in line with previous literature considering the interactions between grower behaviour and the control of a disease system [16, 17, 42].

4.4 Conclusion

In this study, we have parameterised a novel model of *S. tritici* transmission, fungicide control and IPM measures to investigate the potential impact on yield of different control strategies. We found that the presence of fields using IPM may offer indirect control to all growers in the system. We also identified where detailed empirical studies will help reduce uncertainty in model parameterisation and improve the robustness of model projections; specifically, quantifying contribution of crop debris to inoculum and the collection of experimental data on the effectiveness of biocontrol measures on a field-scale. Overall, the results suggest that wheat growers can consider IPM as a viable alternative to a conventional fungicide regime whilst maintaining yield, particularly as the use of chemical fungicides comes under increasingly tighter constraints. As growers not participating in IPM control could indirectly benefit from its presence, this has interesting implications for incentivisation of IPM uptake, as we see a free-rider effect that has been observed in other cases of crop disease management [15–17].

Author contributions

Elliot M.R. Vincent: Data curation, Formal analysis, Methodology, Software, Validation, Visualisation, Writing - Original Draft, Writing - Review & Editing.

Edward M. Hill: Conceptualisation, Methodology, Supervision, Validation, Visualisation, Writing - Original Draft, Writing - Review & Editing.

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Data availability

All data utilised in this study are publicly available, with relevant references and data repositories provided.

Code availability

The code repository for the study is available at:
https://github.com/emrvincent/IPM_s_tritici_modelling.

Archived code: https://doi.org/10.5281/zenodo.14974774.

Competing interests

All authors declare that they have no competing interests.

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