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Short Title: Predicting combined efficacy

Integrated control of potato late blight: predicting the combined efficacy of host resistance and fungicides

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Abstract

Integrating cultivars that are partially resistant with reduced fungicide doses offers growers an opportunity to decrease fungicide input but still maintain disease control. To use integrated control strategies in practice requires a method to determine the combined effectiveness of particular cultivar and fungicide dose combinations. Simple models, such as additive dose models (ADM) and multiplicative survival models (MSM), have been used previously to determine the joint action of two or more pesticides. This study tests whether a model based on multiplicative survival principles can predict the joint action of fungicide doses combined with varieties of differing partial host resistance. Data from eight field experiments on potato late blight (*Phytophthora infestans*), where the severity of foliar blight was assessed and converted to AUDPC, were used to test the model. A subset of data, derived from the most susceptible cultivar, King Edward, was used to produce dose response curves from which parameter values were estimated, quantifying fungicide efficacy. These values, along with the untreated values for the more resistant cultivars, Cara and Sarpo Mira, were used to predict the combined efficacy of the remaining cultivar by fungicide dose combinations. Predicted efficacy was compared against observations from an independent sub-set of treatments from the field experiments. The analysis demonstrated that multiplicative survival principles can be applied to describe the joint efficacy of host resistance and fungicide dose combinations.

Introduction

There are many ways in which integrated control strategies for foliar plant pathogens can be deployed. Examples of using host resistance to limit damage or reduce dependence on fungicides include switching from susceptible to moderately resistant varieties for control of northern corn leaf blight of maize (*Exserohilum turicum*) and decreasing the number of fungicide applications on wheat varieties with resistance to tan spot (*Drechslera tritici-repentis*) (Debela *et al*., 2017, Jørgensen & Olsen, 2007). Decreasing fungicide inputs on moderately resistant potato cultivars compared with susceptible cultivars has been shown to be an effective control strategy against late blight, caused by *Phytophthora infestans* (Fry, 1978, Gans *et al*., 1995, Nærstad *et al*., 2007). Despite considerable research demonstrating the potential to optimise inputs by combining strategies, e.g. fungicide inputs and genetic resistance of varieties, no study has yet determined whether there is a predictable relationship describing the joint action of different components of integrated control.

Simple models to predict the joint action of two or more pesticides applied in mixture have been used in laboratory and field studies for invertebrate pests, weeds and diseases (Bliss, 1939, Scardavi, 1966, Colby, 1967, Rummens, 1975, Gisi *et al*., 1985, Paveley *et al*., 2003). In these simple models, the efficacy of each component of the mixture is quantified and used to predict the efficacy of the mixture. The efficacy of treatment combinations has been described as synergistic or antagonistic where the joint action of the mixture components exceeded or failed to achieve the level of control predicted. More recently, one such model has been applied to assess the joint action of host resistance genes (identified as quantitative trait loci) against diseases of winter wheat (Grimmer *et al*., 2015).

The two most frequently used models to determine the combined efficacy of two or more control methods have been defined as two broad types, additive dose models (ADM)

and the multiplicative survival models (MSM), also known as the Abbot and Wadley methods respectively. Their respective appropriateness for joint action comparisons has been reviewed previously (Morse, 1978). The ADM assumes that the action of one component can be directly substituted for the action of the other, i.e. that the dose of one mixture component can be expressed as an equivalent dose of the other component. It has been reported previously that the effects of host resistance and fungicides to control *Phytophthora infestans* were 'additive' (Fry, 1978). However, the assumption underlying the ADM cannot be met when the two components have fundamentally different modes of action, such as in the case described here where host resistance and fungicides are combined. MSM, however, calculates the proportion of the pathogen population which 'survives' the effect of each component separately, and then predicts the proportion of the population which would survive joint use of the control methods by multiplying the survivorship proportions. The underlying assumption is that the two components act independently, which is a plausible assumption for host resistance and fungicides. Hence, MSM is likely to be the more appropriate model for determining the joint action of host resistance and fungicide dose.

This study tested whether a simple multiplicative survival model can predict the joint action of fungicide dose and host resistance against *Phytophthora infestans* on potato and could provide an accurate prediction of the performance of those combinations under field conditions.

Materials and methods

Data derived from eight integrated control field experiments conducted in 2010 and 2011 were used in this study. The experiments were conducted on late blight (*Phytophthora infestans*) of potato (*Solanum tuberosum*) at two sites: Ayrshire and Ceredigion in the UK.

Two experiments were conducted at each site in each year: one with treatments applied during rapid canopy growth ('rapid canopy') and the other with treatments applied later in the seasons when canopy size was relatively stable ('stable canopy').

Experimental design

Treatments in each experiment consisted of all combinations of three cultivars and four fungicide doses, with an untreated control for each cultivar (Table 1). Integrated control treatments were considered to be the treatments where cultivars, which were moderately (Cara) or highly (Sarpo Mira) resistant, were combined with fungicide doses below the maximum permitted dose per application (the full label recommended dose). At both sites experiments were laid out as a randomised split-plot design with four replicates for each treatment. Fungicides were applied at the whole plot level and cultivars planted at the subplot level. Fungicides were randomised within each block and varieties randomised within each fungicide plot. In Ceredigion, main plots were four rows wide (each row = 0.9m wide) by 11m long. Main plots were separated by 1.5 m unplanted row length. Each main plot was divided into cultivar sub-plots of four rows by 3.0m long and separated by 1.0m unplanted row length. All cultivars were supplied as the same seed size (35 to 45mm) and planted at 30cm spacing. A single row of King Edward was planted between each of the blocks as an infector row.

In Ayrshire in 2010, fungicide treatment plots were four rows wide by 9.75 m long and separated longitudinally by 1.5 m of bare earth. Each cultivar sub-plot was four rows by 2.75 m long separated by 0.75 m unplanted row length with 25cm seed spacing. In 2011, seed spacing was 0.23 m. The fungicide treatment plots were 8.97 m long and the cultivar plots 2.3

m long. The unplanted row length was 1.61 m. A single row of King Edward was planted longitudinally between each of the blocks as an infector row.

One fungicide, mandipropamid [full recommended label rate 0.6 l ha^{-1} (250g l^{-1}) as Revus, Syngenta Ltd] was applied to cultivars at a range of doses, as proportions of the full recommended label rate (Table 1). Treatments were applied either during rapid canopy and or during stable canopy growth in separate experiments. For the rapid canopy experiments, first treatment fungicides were applied at the time of the first blight warning or when plants met within the rows, whichever was soonest. For the stable canopy experiments, chlorothalonil + propamocarb-hydrochloride [full recommended rate 2.5 l ha^{-1} (375 g l^{-1} + 375 g l^{-1}) as Merlin, Bayer CropScience] were applied to all plots at 7- or 10-day intervals until rapid canopy growth was complete (typically three applications at 10-day intervals per season). For all experiments, four fungicide applications for each treatment at 7-day intervals were planned, however, there was flexibility depending on the epidemic progress at different sites and for different seasons (Table 5). In Ceredigion, there were four applications of treatment fungicides in app experiments conducted in 2010 and 2011. In Ayrshire, there were four test fungicide applications in the 2010 rapid canopy experiment and five in the equivalent trial in 2011. Both stable canopy trials had six applications of test fungicides. Once treatment sprays had been applied, all plots were sprayed with between 1274g ha^{-1} and 1540g ha^{-1} mancozeb (as Dithane NT, Penncozeb or Laminator Flo depending on site and season) at 7-day intervals until desiccation. In Ceredigion, fungicide treatments were applied using a handheld Oxford Precision Sprayer in 250 litres of water per hectare operating at 200 kPa through 110° flat fan nozzles. In Ayrshire, treatments were applied in 200 litres of water per hectare, using a tractor-mounted modified AZO compressed air sprayer through Lurmark F03-110 flat fan nozzles at an operating pressure of 350 kPa.

Infector rows (cv. King Edward) were not sprayed with fungicide and were inoculated with isolate(s) of genotype 13_A2 grown on Rye B agar (Caten & Jinks, 1968) but sub-cultured several times on detached King Edward potato leaves prior to inoculation of experiments. These isolates were used to produce a sporangial suspension (a minimum of 1 x 10^4 spores ml^{-1}) in sterile distilled water which was applied to the spreader rows using a handheld mister. The isolates were supplied by the James Hutton Institute, Dundee, UK (Table 2).

Foliar blight was assessed at least weekly as the percentage of leaf area affected by *P. infestans*, with more frequent assessments when the epidemic was increasing rapidly, using the modified MAFF key 2.1.1: Potato Blight on the Haulm (Anon, 1976; Large, 1952).

Determining the effectiveness of cultivar and fungicide dose combinations

For each treatment the Area Under the Disease Progress Curve (AUDPC) was calculated from the foliar late blight severity scores (Campbell & Madden, 1990). To predict the effectiveness of host resistance and fungicide combinations, a multiplicative survival equation was derived incorporating a previously published exponential equation (1) describing the fungicide dose response curve, where D_d is disease severity at dose d and D_o is disease when fungicide dose = 0 (Paveley *et al*., 2000).

$$(1) \quad D_d = D_o[1 - b(1 - e^{-kdose})]$$

Data generated from the observed dose response curve of the most susceptible cultivar, King Edward, were used to calculate the parameters b and k, where b represents the amount of disease that might be potentially controlled with an infinite dose and k defines the rate of change of disease severity with dose. Curves were forced through the untreated values and

parameters calculated using the following equation in FITNONLINEAR in Genstat 16th edition (VSN International Ltd, UK).

The dose response equation and parameters were then used in the following equation (2) to predict the effectiveness of cultivar and fungicide dose combinations based on the principles of multiplicative survival:

$$(2) \quad D = D_o \left[\left(\frac{D_r}{D_s} \right) (1 - b(1 - e^{-kdose})) \right]$$

D is the predicted level of disease for the appropriate cultivar and fungicide dose combination, D_o is the untreated AUDPC of the standard susceptible cultivar (in this case the most susceptible cultivar King Edward). For the first analysis, D_s is the untreated AUDPC for the standard cultivar, D_r is the untreated AUDPC for the partially resistant test cultivar and *dose* is the proportion of the full fungicide dose.

AUDPC values were logit transformed using an equation (3) modified from Grimmer *et al*. (2015):

$$(3) \quad LTS = In[s/(M - s)]$$

Where *LTS* is the logit transformed AUDPC, *In* is the natural logarithm, *s* is the observed or predicted disease severity and *M* is the maximum AUDPC achievable during the disease assessment period (e.g. if disease assessment period was 59 days then maximum AUDPC is 5900). The transformed observed severity was linearly regressed against the transformed predicted severity as advocated in Piñeiro *et al*. (2008). All analysis was done in Genstat 16th Edition (VSN International Ltd, UK).

Results

Parameter estimates for the dose response curves were derived from dose response curves generated using King Edward as the baseline. The fitted dose response curves were close to the observed AUDPC values (Figure 1). The percentage variance accounted for (as R^2) for all fitted curves ranged from 96% to 100% (Table 3). Observed and predicted disease severities, as the AUDPC for each cultivar and fungicide dose combination, for moderately resistant Cara and highly resistant Sarpo Mira cultivars were compared (Table 4). There was a highly signficant relationship (P<0.001; R^2 = 0.88) between the predicted and observed values for each fungicide dose and cultivar combination (Figure 2). When the combined effects of fungicide dose and cultivar were predicted, the resultant AUDPC values were generally higher than those observed in the experiments, regardless of cultivar resistance rating.

The percentage of foliar late blight present at the first and last fungicide application in each trial varied depending on the site and year (Table 5). In 2010, only the stable canopy trial in Ayrshire had foliar late blight present when first fungicides were applied. In contrast in 2011, most King Edward and Cara treatments had traces of foliar late blight when first fungicides were applied. At the time the final fungicide application was applied to King Edward, foliar late blight ranged from 0.7 to 94% leaf area affected and 5 out of the 8 trials had >90% foliar late blight. In comparison, for Cara, foliar late blight ranged from 0.5 to 85% and for Sarpo Mira 0.03 to 7.8% leaf area affected. Conditions were generally not favourable for disease development early in the season at the Ayrshire site in 2010. The epidemic was slower to start at the Ceredigion site in the rapid canopy trials in both 2010 and 2011.

Following the logit transformation, which took into account the differences between experiments in the duration of the disease assessment period (and hence differences in the maximum possible AUDPC; equation 3), the regression accounted for 76% of the variation in the logit transformed severity [P=<0.001, slope and intercept 95% confidence intervals

(0.7144, -1.569) and (0.5341, -2.324)] (Figure 3). T-values [36.01, -15.59] demonstrated that this line was significantly different from the slope of 1 and intercept of 0.

Discussion

The analysis presented here shows that multiplicative survival principles can be applied to derive a simple model to describe the efficacy of host resistance and fungicide dose combinations. To generate predicted AUDPC values and test the ability of the model to predict the joint action of host resistance and fungicide dose, there were two requirements: a fungicide dose response curve (including an 'untreated' control) for a susceptible cultivar (in this case King Edward) plus an 'untreated' control for each test cultivar. Using this method, there was a good relationship between observed AUDPC values and the multiplicative survival model (MSM) predictions.

In a previous study, dose response curves for different host resistance/fungicide dose treatments were compared and differences used to identify the contribution of host resistance. The contribution of host resistance was expressed as the equivalent dose of fungicide required to match the additional disease control provided by a more resistant cultivar (Fry, 1978). An alternative approach, using three dimensional regression, was used to estimate the equivalent fungicide dose equivalent to one point on a 1 to 9 scale (where 9 is most resistant) which defined cultivar resistance (Gans *et al*., 1995). Such approaches have the disadvantage of requiring dose response curves from all treatments to compare and quantify the benefits of host resistance. This paper demonstrates, for the first time, a method in which the performance of a cultivar in combination with different fungicide doses, could be predicted, without the need to include all the fungicide dose combinations in the experiment and in the absence of a cultivar resistance rating.

MSM models were used originally to determine whether the joint action of mixture components was synergistic. Experimental results are compared with the reference model which represents the joint action predicted from the efficacy of the components. Where the observed severity or AUDPC values were less than, or more than, the predicted values, the

229 combination may be considered synergistic or antagonistic, respectively (Kosman & Cohen,
230 1996). For the data presented in this paper, the majority of observed values were below their
231 corresponding predicted values. Cultivar and fungicide combinations therefore performed
232 better than predicted for the majority of host resistance and fungicide dose combinations.
233 Although this is a positive outcome for the value of integrated control, it is important to
234 consider whether this apparent synergy between host resistance and fungicide treatment may
235 in fact have been an artifact of the experimental method.

236 As described in the methods, mancozeb was applied to all treatments, including
237 'untreated' plots, once treatment fungicide applications were completed. This over-spray was
238 designed to allow more time for differences in foliar late blight between treatments to
239 develop prior to defoliation. By the time the final treatment fungicides were applied, >90% of
240 the leaf area in untreated King Edward plots, in five out of eight of the experiments, was
241 infected with *P. infestans*. Mancozeb prevents spore gemination, but has limited effects on
242 established infections and mycelial growth (Bruck *et al.*, 1981, Kaars Sijpesteijn, 1982).
243 Mancozeb application where > 0.5% of leaf area was affected by *P. infestans* has been shown
244 previously to be insufficient to decrease epidemic growth rate immediately, with a delay of 8
245 to 10 days before established epidemics were slowed (Fry *et al.*, 1979). It is likely, therefore,
246 that the timing of mancozeb application, relative to epidemic severity and growth rate,
247 differed depending on cultivar and the fungicide dose applied in the experimental treatments.
248 It has been demonstrated previously that the order in which fungicides are applied can also
249 impact on the ability of particular fungicide products to influence the epidemic (Bain &
250 Bardsley, 2009). In the current study, the effect of mancozeb on the epidemic growth rate was
251 likely to be lower for treatments where disease was well established (e.g. on untreated King
252 Edward) compared with other treatments where disease was less established at the time of
253 mancozeb application (e.g. on untreated Sarpo Mira) leading to bias. Such bias could not be

254 excluded from this analysis, given the absence of a completely fungicide untreated control.
255 Given the apparent effect of oversprays on the epidemic and the potential for bias, it is
256 suggested that such over-sprays should be avoided, or completely untreated controls should
257 be included, for future experiments.

258 Generating up to date information on the likely efficacy of host resistance and
259 fungicide dose combinations is necessary, particularly when pathogen populations are
260 evolving rapidly towards aggressiveness, virulence or fungicide insensitivity. In Great
261 Britain, the dominance of 13_A2, one of the newer aggressive and more virulent genotypes,
262 resulted in the re-grading of cultivars, including Cara which was used in this experiment,
263 from highly resistant to moderately resistant (Lees *et al*., 2012). Similarly *P. infestans*
264 genotypes that are less sensitive to and less well controlled by the fungicide fluazinam have
265 been detected in Europe recently (Schepers, 2017). It has been demonstrated previously that
266 the rank order of partial resistance of cultivars exposed to *P. infestans* remains similar with
267 and without fungicide treatment (Bain *et al*., 2014), therefore using multiplicative survival
268 principles to explore the potential for using integrated control in the way described here
269 should provide a useful guide to the performance of integrated host resistance and fungicide
270 strategies.

271 Achieving effective control of late blight on potato using decreased fungicide doses
272 on moderately resistant cultivars has been demonstrated previously (Fry, 1975, Clayton &
273 Shattock, 1995, Gans *et al*., 1995, Bain *et al*., 2014) and, in some instances, such information
274 has been incorporated into models to guide fungicide applications (Nærstad *et al*., 2007, Liu
275 *et al*., 2017). For potato late blight, foliar resistance ratings are calculated for cultivars in
276 many countries (e.g. AHDB, 2017). For the cultivar resistance ratings reported in Europe, 1
277 to 9 ratings are based on AUDPC values for untreated test cultivars, expressed relative to
278 AUDPC values for one susceptible and one resistant reference cultivar. Cultivars with the

same 1 to 9 resistance rating will therefore have very similar relative mean AUDPC values. Consequently, one resistance rating in the equation should also give a useful indication of the combined efficacy of fungicide doses on all cultivars with the same rating, e.g. there are currently 11 cultivars in Great Britain rated 7 for foliar blight resistance.

The method defined in Equation 2 should be more generally applicable both for potato late blight worldwide and for integrated control in other pathosystems – although further experimental proof is required. The experimental data required to estimate parameters is relatively simple to obtain and analysis is straightforward. Models to describe the joint action of fungicides mixtures, based on MSM principles, have been included as a component in decision support system models for winter wheat disease control (Paveley *et al*., 2003, Milne *et al*., 2007) and models of pathogen evolution which consider integrated control (Carolan *et al*., 2017). MSM principles have proved to be remarkably generalisable in their application, provided the control methods for which joint action is being calculated are reasonably independent. The principles have proved useful for predicting joint action of fungicides, herbicides and insecticides, joint action of separate fungicide treatments in a spray programme (Paveley *et al*., 2003), joint action of host resistance QTL (Grimmer *et al*., 2015) and now for the joint action of host resistance and fungicides.

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References

AHDB, 2017. Potato variety database (Great Britain) [http://varieties.ahdb.org.uk/] accessed 7 June 2017.

Anon, 1976. Manual plant growth stages and disease assessment keys. MAFF Publications, Middlesex, UK.

Bain R, Bardsley E, 2009. The impact of fungicide spray order on foliar blight control in two growing seasons. In: Schepers HTAM, ed. *Proceedings of the Eleventh Euroblight Workshop, Hamar, Norway. PPO Special Report 13.* Wageningen, The Netherlands, 115-122.

Bain R, Ritchie F, Lees A, Dyer C, 2014. Impact of fungicide input on leaf blight (*Phytophthora infestans*) development on different cultivars. In: Schepers, HTAM, ed. *Proceedings of the Fourteenth Euroblight Workshop. PPO Special Report No. 16.* Wageningen, The Netherlands, 65-73.

Bliss C, 1939. The toxicity of poisons applied jointly. *Annals of Applied Biology* **26**, 585-615.

Bruck R, Fry W, Apple A, Mundt C, 1981. Effect of protectant fungicides on the developmental stages of *Phytophthora infestans* in potato foliage. *Phytopathology* **71**, 164-6.

Campbell C, Madden L, 1990. *Introduction to plant disease epidemiology*. New York, USA: John Wiley & Sons, Inc.

Carolan K, Helps J, Van Den Berg F, Bain R, Paveley N, Van Den Bosch F, 2017. Extending the durability of cultivar resistance by limiting epidemic growth rates. *Proceedings of the Royal Society B* **284**, 20170828.

Caten C, Jinks J, 1968. Spontaneous variability of single isolates of *Phytophthora infestans.* I. Culture variation. *Canadian Jouranl of Botany* **46**, 329-48.

Clayton R, Shattock R, 1995. Reduced fungicide inputs to control *Phytophthora infestans* in potato cultivars with high levels of polygenic resistance. *Potato Research* **38**, 399-405.

Colby S, 1967. Calculating synergistic and antagonistic responses of herbicide combinations. *Weed Science* **15**, 20-2.

Debela M, Dejene M, Abera W, 2017. Management of Turicum leaf blight [*Exserohilum turicum* (Pass.) Leonard & Suggs] of maize (*Zea Mays* L.) through integration of host resistance and fungicide at Bako, Western Ethiopia. *African Journal of Plant Science* **11**, 6-22.

Fry W, 1975. Integrated effects of polygenic resistance and a protective fungicide on development of potato late blight. *Phytopathology* **65**, 908-11.

Fry W, 1978. Quantification of general resistance of potato cultivars and fungicide effects for integrated control of potato late blight. *Phytopathology* **68**, 1650-5.

Fry W, Bruck R, Mundt C, 1979. Retardation of potato late blight epidemics by fungicides with eradicant and protectant properties. *Plant Disease Reporter* **63**, 970-4.

Gans P, Carson W, N P, Owen L, 1995. Exploiting cultivar resistance to control potato blight (*Phytophthora infestans*). In: Dowley LJ, Bannon E, Cooke L, Keane T, O'sullivan E, eds. *Phytophthora infestans 150*. Dublin, Ireland: Boole Press Ltd, 345-50.

Gisi U, Binder H, Rimbach E, 1985. Synergistic interactions of fungicides with different modes of action. *Transactions of the British Mycological Society* **85**, 299-306.

Grimmer M, Boyd L, Clarke S, Nd P, 2015. Pyrimiding of partial disease resistance genes has a predictable, but diminishing, benefit to efficacy. *Plant Pathology* **64**, 748-53.

Jørgensen L, Olsen L, 2007. Control of tan spot (*Drechslera tritici-repentis*) using cultivar resistance, tillage methods and fungicides. *Crop Protection* **26**, 1606-16.

348 Kaars Sijpesteijn A, 1982. Mechanism of action of fungicides. In. Dekker J, Georgopoulos S,
349 eds. *Fungicide Resistance in Crop Protection.* Centre for Agricultural Publishing and
350 Documentation, Wageningen, The Netherlands, 32-45.

351 Kosman E, Cohen Y, 1996. Procedures for calculating and differentiating synergism and
352 antagonism in action of fungicide mixtures. *Phytopathology* **86**, 1263-72.

353 Large EC, 1952. The interpretation of progress curves for potato blight and other plant
354 diseases. *Plant Pathology* **1**, 109-117.

355 Lees A, Stewart J, Lynott J, Carnegie S, Campbell H, Roberts A, 2012. The effect of a
356 dominant *Phytophthora infestans* genotype (13_A2) in Great Britain on host resistance to
357 foliar late blight in commercial potato cultivars. *Potato Research* **55**, 125-34.

358 Liu Y, Langemeier M, Small I, Joseph L, Fry W, 2017. Risk management strategies using
359 precision agriculture technology to manage potato late blight. *Agronomy Journal* **109**, 562-
360 75.

361 Milne A, Paveley N, Audsley E, Parsons D, 2007. A model of the effect of fungicides on
362 disease-induced yield loss, for use in wheat disease management decision support systems.
363 *Annals of Applied Biology* **151**, 113-25.

364 Morse PM, 1978. Some comments on the assessment of joint action of herbicide mixtures.
365 *Weed Science* **26**, 58-71.

366 Nærstad R, Hermansen A, Bjor T, 2007. Exploiting host resistance to reduce use of
367 fungicides to control potato late blight. *Plant Pathology* **56**, 156-66.

368 Paveley N, Lockley D, Vaughan T, Thomas J, Schmidt K, 2000. Predicting effective doses
369 through observation of leaf emergence. *Plant Pathology* **49**, 748-66.

370 Paveley N, Thomas J, Vaughan T, Havis N, Jones D, 2003. Predicting effective doses for the
371 joint action of two fungicide applications. *Plant Pathology* **52**, 638-47.

Piñeiro G, Perelman S, Guerschman J, Paruelo J, 2008. How to evaluate models: observed vs. predicted or predicted vs. observed? *Ecological Modelling* **216**, 316-22.

Rummens F, 1975. An improved definition of synergistic and antagonistic effects. *Weed Science* **23**, 4-6.

Scardavi A, 1966. Synergism among fungicides. *Annual Review of Phytopathology* **4**, 335-48.

Schepers HTAM, 2017. Reduced sensitivity to fluazinam detected in the rapidly increasing *Phytophthora infestans* lineage EU-37 (Dark green 33). [goo.gl/cmCw3p]. Accessed 12 February 2018.

Figures

Figure 1. Dose response curves (lines) derived from data in the rapid canopy experiments and fitted to the original AUDPC values (points) for King Edward using the equation (1) derived from Paveley *et al*., 2000. Grey lines are the Ayrshire site and black lines the Ceredigion site. Solid lines are data from 2010 and dashed lines are data from 2011.

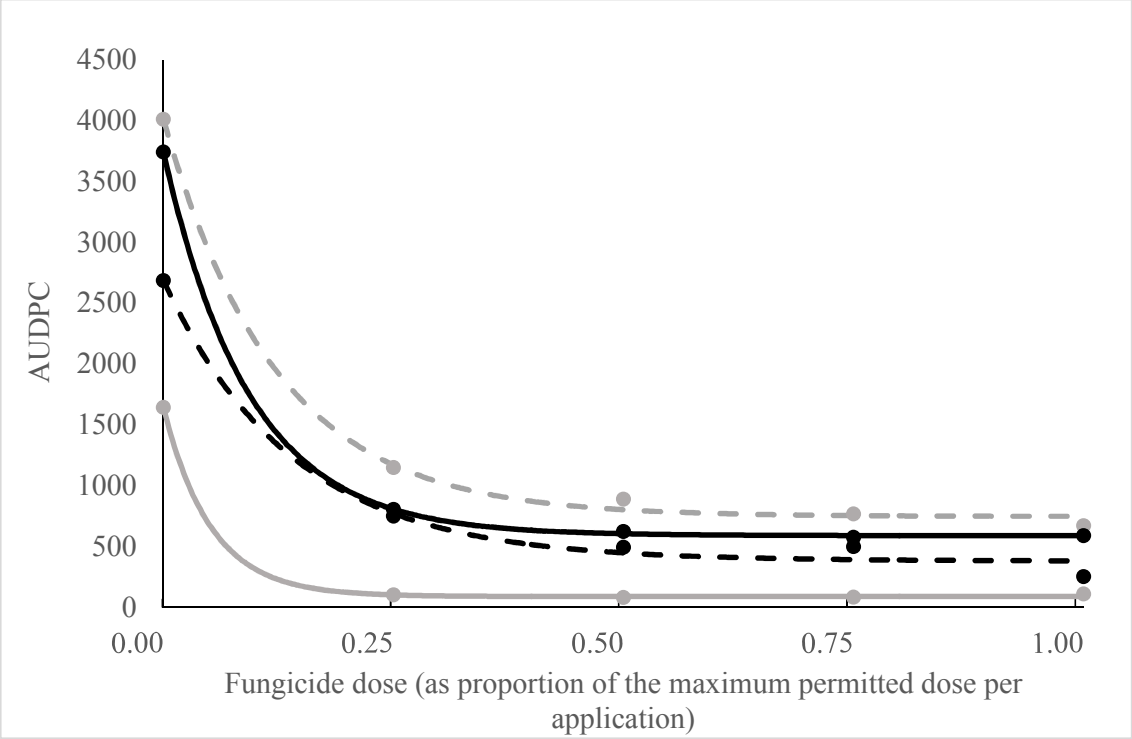
Figure 2. Relationship between observed and predicted disease severity for Cara (circles) and Sarpo Mira (triangles) from rapid and stable canopy trials conducted in 2010 and 2011. Black data points identify data points from Ceredigion and white data points from Ayrshire.

Figure 3. Relationship between logit transformed observed and predicted disease severity for Cara (circles) and Sarpo Mira (triangles) from rapid and stable canopy trials conducted in 2010 and 2011. Slope and intercept 95% confidence intervals [(0.7144, -1.569) and (0.5341, -

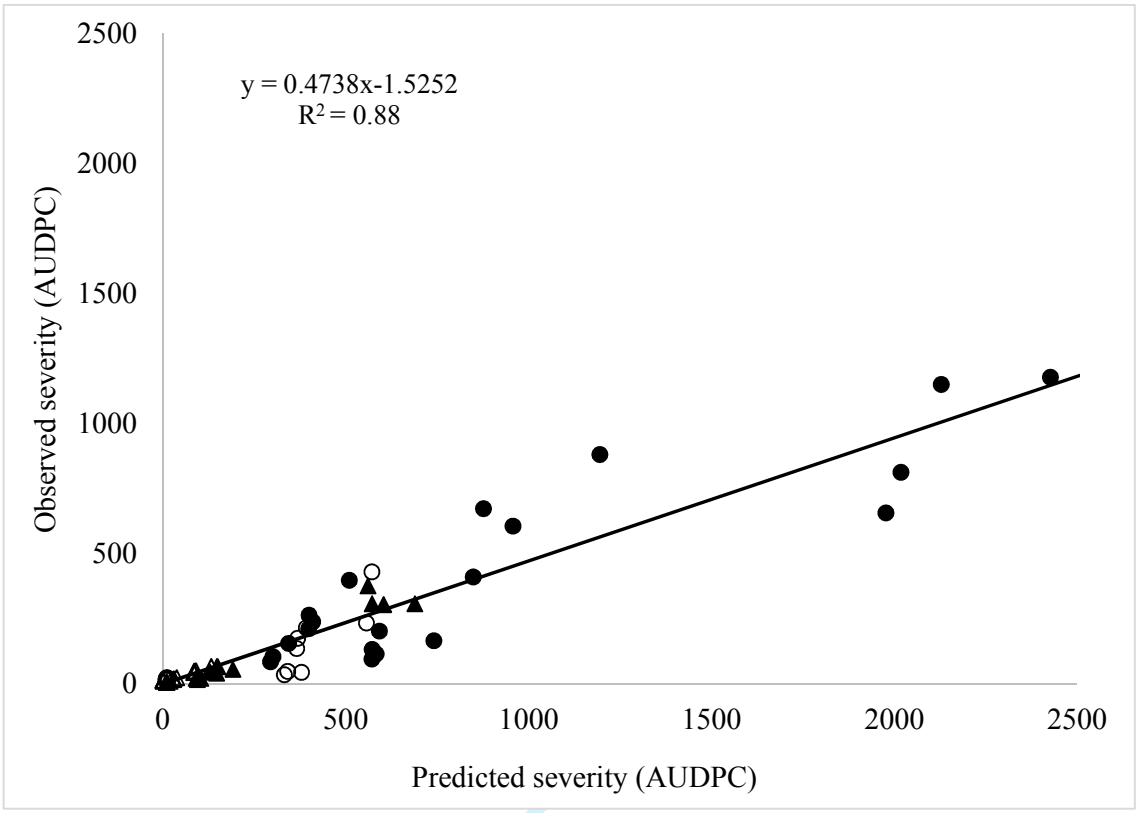
2.324)]. Solid black line represents the regression and dotted line the 1:1 line. Black data points identify data points from the Ceredigion site and white data points from the Ayrshire site.

Figures

Figure 1.



6 Figure 2



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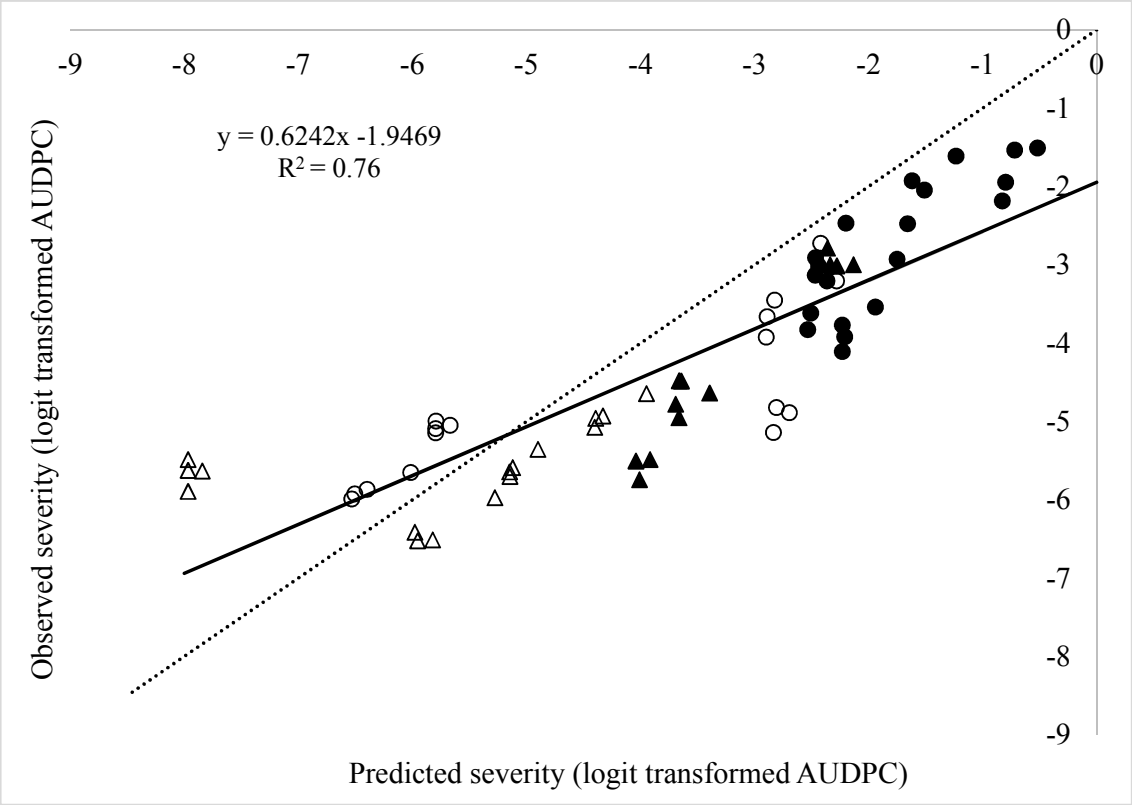
9 Figure 3



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Tables

Table 1. Cultivars, fungicide treatments, spray interval and UK foliar blight resistance ratings for all varieties included in the rapid canopy and stable canopy experiments.

Year	Fungicide doses applied (as percentage of full recommended label rate)	Spray interval	Cultivar (foliar blight resistance rating)*		
2010 and 2011	0, 25%, 50%, 75% and 100%	7 days	King Edward (3)	Cara (5)	Sarpo Mira (7)

*from the Potato Variety Database (AHDB, 2017).

Table 2. *P. infestans* isolate(s) and inoculation dates for each site by year.

	Site			
	Ayrshire		Ceredigion	
Year	inoculation date(s)**	isolates	inoculation date	isolates
2010	15, 19, 28 July 17 August	2009_7654A	3 July	2009_7654A
2011	8, 13, 18, 28 July 8, 16 August	07/39, 2009_7654A, 2006_3928A, 2008_6082F	12 July	2009_7654A

** There were multiple trials at the site, inoculated on different dates.

Table 3. Untreated AUDPC values for King Edward, Cara and Sarpo Mira plus parameter estimates (refer to text for definitions) derived from dose response curves (cv. King Edward/fungicide dose) and R^2 for the fitted dose response curves by site, experiment and year.

			Untreated AUDPC			Parameter estimates		
Experiment	Site[a]	Year	King Edward	Cara	Sarpo Mira	*b*	*k*	R^2
Rapid canopy	AYR	2010	1643	196	22	0.94	19.40	1.00
		2011	4008	1957	431	0.81	8.17	1.00
	CER	2010	3741	3042	786	0.81	10.73	1.00
		2011	2686	2058	71	0.86	7.10	0.99
Stable canopy	AYR	2010	3112	1371	36	0.76	6.10	1.00
		2011	2419	1568	116	0.75	9.42	1.00
	CER	2010	3828	3234	918	0.40	3.97	0.96
		2011	2094	1885	204	0.56	4.28	0.98

[a]AYR = Ayrshire site, CER = Ceredigion site.

Table 4. Observed and predicted disease severity (as AUDPC) of foliar blight for rapid canopy and stable canopy experiments. Fungicides were applied to rapid canopy experiments early in the season when plants are actively growing and once canopy expansion was complete for stable canopy experiments. Dose is expressed as a proportion of the maximum permitted dose per application and cultivar is expressed as a 1 to 9 ranking, where 1 is most susceptible and 9 is most resistant to *P. infestans*.

				Trial			
				Rapid Canopy		Stable Canopy	
Site	Year	Cultivar (resistance rating)	Dose	Observed	Predicted	Observed	Predicted
Ayrshire	2010	Cara (5)	0.25	23	12	234	557
			0.5	24	11	45	380
			0.75	22	11	48	341
			1.0	21	11	35	333
		Sarpo Mira (7)	0.25	13	1	21	15
			0.5	13	1	17	10
			0.75	10	1	16	9
			1.0	15	1	15	9
Ayrshire	2011	Cara (5)	0.25	431	572	399	510
			0.5	216	392	239	409
			0.75	176	369	264	400
			1.0	136	366	213	399
		Sarpo Mira (7)	0.25	67	133	24	38
			0.5	50	91	19	30
			0.75	49	86	17	30
			1.0	43	85	18	30
Ceredigion	2010	Cara (5)	0.25	167	742	1180	2427
			0.5	115	584	1152	2129
			0.75	133	573	814	2018
			1.0	96	572	658	1977
		Sarpo Mira (7)	0.25	57	192	309	689
			0.5	66	151	305	604
			0.75	66	148	309	573
			1.0	41	148	377	561
Ceredigion	2011	Cara (5)	0.25	203	592	883	1195
			0.5	156	344	607	958
			0.75	105	302	674	877
			1.0	85	294	411	849
		Sarpo Mira (7)	0.25	10	20	44	129
			0.5	6	12	22	103
			0.75	6	10	17	95
			1.0	7	10	22	92

Table 5. The percentage leaf area affected by foliar late blight (%) in untreated varieties at the first (First) or within 7 days of the last (Last) treatment application and the number of test fungicides applied to each trial.

Experiment	Site[a]	Year	Percentage of leaf area affected by foliar blight (%)						No. of treatment applications	No. of mancozeb applications
			King Edward		Cara		Sarpo Mira			
			First	Last	First	Last	First	Last		
Rapid canopy	AYR	2010	0.0	0.7	0.0	0.5	0.0	0.3	4	6
		2011	5.3	100.0	1.3	47.5	0.5	7.3	5	4
	CER	2010	0.0	17.5	0.0	22.0	0.0	0.3	4	8
		2011	0.0	9.0	0.0	0.7	0.0	0.0*	4	4
Stable canopy	AYR	2010	0.2	95.0	0.1	33.0	0.0	1.0	6	2
		2011	0.1	97.0	0.0*	60.0	0.0	4.3	5	2
	CER	2010	0.0	90.0	0.0	62.5	0.0	7.8	4	5
		2011	0.1	93.8	0.1	85.0	0.0	7.5	4	2

[a]AYR = Ayrshire site, CER = Ceredigion site. * = 0.03