

## Predicting the combined efficacy of host resistance and fungicides

FAYE RITCHIE<sup>1</sup>, RUAIRIDH BAIN<sup>2</sup>, ALISON LEES<sup>3</sup>, TIMOTHY BOOR<sup>1</sup>, NEIL PAVELEY<sup>4</sup>

<sup>1</sup> ADAS Boxworth, Battlegate Road, Boxworth, CB23 4NN, England, UK

<sup>2</sup> SRUC, John Niven Building, Auchincruive Estate, Ayr, KA6 5HW, Scotland, UK

<sup>3</sup> James Hutton Institute, Invergowrie, Dundee, Scotland, DD2 5DA, UK

<sup>4</sup> ADAS High Mowthorpe, Duggleby, Malton, YO17 8BP, England, UK

### SUMMARY

Integrating strategies for the control of late blight caused by *Phytophthora infestans* on potato, such as epidemic rate limiting host resistance combined with reduced fungicide doses, has been demonstrated to successfully decrease foliar disease severity. A simple multiplicative survival model (MSM) was devised to predict disease severity on cultivars with differing disease resistance treated with different fungicide doses. AUDPC data were obtained from field experiments testing cultivar by fungicide treatment combinations. The effectiveness of the host resistance was expressed as the proportion of disease remaining on each of the more resistant cultivars compared with the most susceptible cultivar. The effectiveness of fungicide treatment was calculated using data from the most susceptible cultivar to parameterise a dose response curve function. This allowed calculation of the proportion of disease remaining at any given dose. The MSM model was then used to predict the AUDPC of other variety by fungicide dose combinations. A regression line fitted to the observed and predicted data explained 68% of the variation in AUDPC. Aspects of experimental design which could be addressed to improve predictive value are described.

### KEYWORDS

Late blight, *Phytophthora infestans*, foliar blight, host resistance, fungicides, integrated control, prediction, multiplicative survival model, MSM

### INTRODUCTION

Cultivar resistance, in combination with reduced fungicide input, has been shown to successfully reduce foliar late blight severity (Fry, 1978, Kirk et al., 2001, Kirk et al., 2005, Nærstad et al., 2007, Bain et al., 2011). A shift in the late blight population in GB towards more aggressive and virulent *P. infestans* genotypes, including 13\_A2 and 6\_A1, resulted in the foliar resistance ratings of several cultivars being downgraded from resistant (e.g. Cara with a rating of 7 in 2010) to moderately resistant (Cara with rating of 5 in 2012) (Lees et al., 2012). Sufficiently large differences in foliar resistance between cultivars are a key part of integrated control;

however, 99% of the potato area in GB consists of cultivars with a resistance rating of 5 or below.

Two broad types of simple models have been widely reported in the literature to determine the efficacy expected from the joint action of pesticide mixtures: the additive dose model (ADM) and the multiplicative survival model (MSM) (Morse, 1978). The MSM has been used to describe the joint efficacy of two active ingredients applied simultaneously and the joint action of host genes (Bliss, 1939, Gisi et al., 1985, Grimmer et al., 2015). It has also been used to determine whether particular mixtures are synergistic or antagonistic (Gisi, 1996). Synergy or antagonism may exist between varietal resistance and fungicide dose for control of late blight where disease control provided by this "mixture" is higher, or lower, than would be predicted from the disease control achieved from the cultivars and fungicide tested individually. The aim of this paper was to determine whether a simple model, based on multiplicative survival principles, could predict the joint action of fungicide dose and host resistance combinations.

## MATERIALS AND METHODS

### *Field experiments*

In 2010 and 2011, four experiments were conducted to determine the effectiveness of integrated control treatments incorporating reduced fungicide inputs and cultivar resistance to control foliar late blight during rapid canopy growth: two were conducted in Ayrshire, Scotland and two in Ceredigion, Wales. Experiments were laid out in a randomised split plot design with four replicates. Each sub-plot consisted of either King Edward (foliar late blight resistance rating 3), Cara (5) or Sarpo Mira (7) and was four rows wide by c. 3m long, with seed spacing determined by tuber size. All foliar assessments were done on the centre two rows of each sub-plot. Treatment fungicide applications were started as soon as plants started to meet within the rows or earlier if late blight risk was high. One fungicide (Revus; 250g/L mandipropamid: full label rate 0.6 L/ha) was applied at 0, 25, 50, 75 and 100% of the recommended label dose at 7-day intervals. A maximum of five test fungicide applications were applied across sites. Dithane NT (mancozeb 75% w/w) at 2.0 Kg/ha was applied once treatment applications were completed.

**Table 2.** *Cultivars, fungicide treatments and GB foliar blight resistance ratings for all cultivars included in rapid canopy experiments in 2010 and 2011.*

Year	Doses applied (proportion of the full recommended label rate)	Spray interval	Cultivar (foliar blight resistance rating)		
2010 and 2011	0.00, 0.25, 0.50, 0.75 and 1.00	7 days	King Edward (3)	Cara (5)	Sarpo Mira (7)

Experimental sites were inoculated on 12 July 2010 and 3 July 2011 (Cilcennin) and 12 July 2010 and 8 July 2011 (Auchincruive) with one or several *P. infestans* isolates (genotype 13\_A2). At Cilcennin, fungicides were applied in 250 litres of water per hectare using a hand held Oxford Precision Sprayer operating at 2.0 bars (200 kPa) through F02-110 flat fan nozzles. At Auchincruive, fungicides were applied in 200 litres of water per ha using a tractor-mounted, modified AZO compressed air sprayer, operating at 3.5 bars (350 kPa) to give a medium/fine

spray quality using Lurmark F03-110 nozzles. The percentage leaf area destroyed by foliar blight was assessed at regular intervals during the epidemic using a modified version of the keys of Large (1952) and Anon (1976). Data were collected as the percentage of leaf area affected by foliar late blight and used to calculate the Area Under the Disease Progress Curve (AUDPC).

#### *Predicting the effectiveness of host resistance and fungicide combinations*

To determine whether the observed levels of late blight (as the AUDPC) could be used to predict the effectiveness of host resistance and fungicide combinations, a function was developed incorporating a simple exponential function describing the fungicide dose response curve (Paveley et al., 2000). Data derived from the dose response curve of the most susceptible cultivar, King Edward, were used to calculate the parameters  $b$  and  $k$  using FITNONLINEAR in Genstat. The parameters were then used in function (1), which was derived to predict the effect of host resistance and fungicide dose combinations from trial data based on the principles of the multiplicative survival model (MSM):

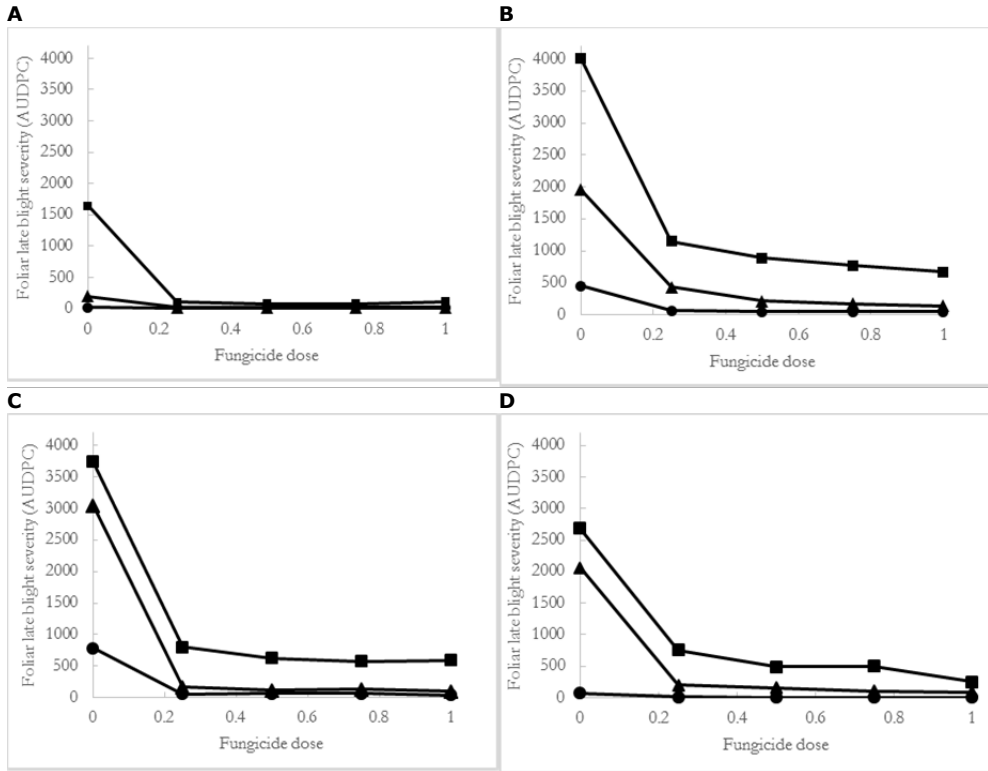
$$(1) \quad D = D_o \left[ \left( \frac{D_r}{D_s} \right) (1 - b(1 - e^{-kP})) \right]$$

$D$  is the predicted level of disease for the appropriate test cultivar and fungicide dose,  $D_o$  is the untreated AUDPC of the standard 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  $P$  is the proportion of the fungicide dose (e.g. 0.25 for  $\frac{1}{4}$  of the recommended label dose, 1 for the full recommended label dose). The constants  $b$  and  $k$  were calculated using the dose response curve for the standard cultivar (King Edward) in each experiment individually as described previously. Observed disease severity was linearly regressed against the predicted severity as advocated in Piñeiro et al. (2008). All analysis was done in Genstat 16<sup>th</sup> Edition (VSN International Ltd, UK).

## **RESULTS AND DISCUSSION**

### *Foliar late blight progress in the experiments in 2010 and 2011*

There were four contrasting foliar late blight epidemics in 2010 and 2011 (Figure 1). At the SRUC site in Scotland in 2010, there were 14 Smith periods during the season; 5 in July, 4 in August and 5 in September. Differences between cultivars, however, were less pronounced with the largest contrast between untreated cultivars and only small differences between fungicide-treated cultivars regardless of dose applied. At the ADAS site in Wales there were fewer Smith periods in 2010, with 4 in July, 4 in August and 3 in September giving a total of 11 for the growing season. The epidemic was more severe and there was greater separation of cultivars, particularly where fungicides had been applied. In 2011, the epidemic was more severe at the Scottish site. There were 11 Smith periods during the season; 3 in July, 6 in August, 1 in September and 1 in October. Differences between the untreated cultivars were clear, however, fungicide applications to Cara substantially decreased foliar blight to levels achieved on Sarpò Mira treated with fungicides. At the Welsh site, 7 Smith periods were reported during the growing season: 2 in July, 4 in August and 1 in September. Again, fungicides substantially decreased the epidemic on all cultivars at all doses tested and this has been demonstrated under GB conditions previously (Bradshaw and Bain, 2007, Bain et al., 2011, Bain et al., 2013).



**Figure 1.** Progress of foliar late blight (as the AUDPC for each fungicide dose and cultivar combination) in experiments conducted in 2010 and 2011. A = SRUC in 2010, B = SRUC in 2011, C = ADAS in 2010 and D = ADAS in 2011. To distinguish between cultivars, the following applies: King Edward (■), Cara (▲) and Sarpo Mira (●). Fungicide dose is the proportion of the full recommended rate (e.g. 1 = the full recommended label rate for a single application) applied.

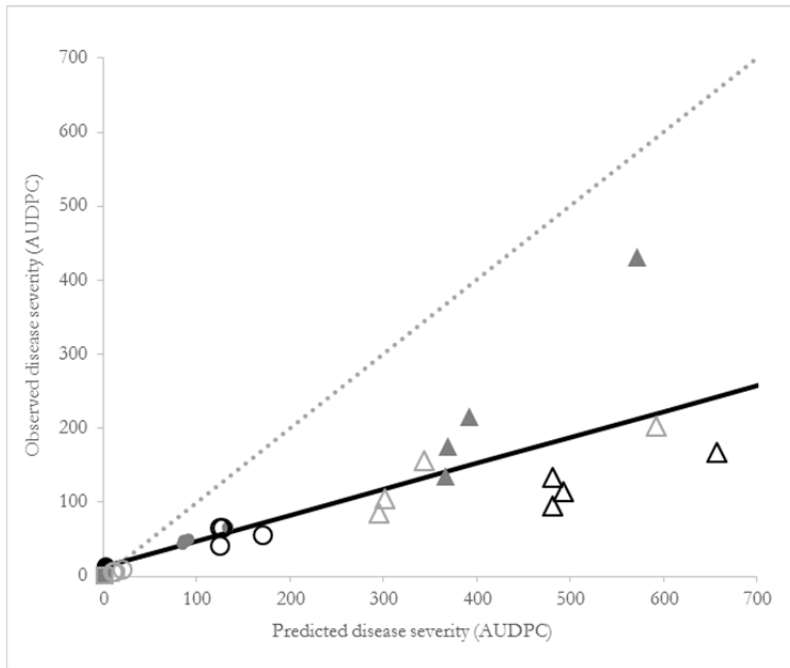
#### Determining predictive value using MSM principles

Generating up to date information on the likely contribution of host resistance and fungicide dose combinations to disease control is necessary, particularly following the dominance of more aggressive *P. infestans* genotypes. In Great Britain, the dominance of 13\_A2, one of the newer aggressive genotypes, resulted in the re-rating of cultivars, including Cara, from highly to moderately resistant (Lees et al., 2012).

The analysis suggests that there is a good relationship between the observed and predicted values, with 68% of the variation accounted for (Figure 2). However, most of the predicted values were overestimates of actual disease observed. Mancozeb was applied to all treatments including untreated plots once treatment fungicide applications were completed, to allow differences to develop between treatments prior to desiccation. Mancozeb application has been shown to require 8 to 10 days before it slows established epidemics (Fry et al., 1979). Disease in untreated plots ranged from 3% to 78% on King Edward, 0.9 to 49% on Cara and 0.1% to 3% on Sarpo Mira immediately prior to mancozeb application, therefore the timing of this

application, relative to the epidemic, would be different for each treatment. The effect of mancozeb on the epidemic growth rate on King Edward will be reduced compared to other treatments where disease was less established, leading to bias in predictions. Such bias could not be excluded from this analysis, but can be avoided for future experiments.

It has been demonstrated previously that the rank order of cultivars exposed to *P. infestans* remains similar with or without fungicide treatment, however, the contribution of host resistance to disease control is lower where plants remain unprotected by fungicide in the presence of these more aggressive strains (Bain et al., 2009). Therefore understanding the effectiveness of fungicide dose and cultivar combinations is necessary if accurate information is to guide agronomists or be incorporated into decision support systems.



**Figure 2.** Observed AUDPC plotted against predicted AUDPC for each dose and cultivar combination from 2 years of field experiments ( $R^2 = 0.68$ ). Data from individual years are shown as black (2010) and grey (2011). Sites are separated by solid markers (SRUC) and open markers (ADAS). Cultivars can be distinguished as Cara (▲) and Sarpo Mira (●). Dotted line shown has a slope of one. The solid line is the regression of predicted vs observed AUDPC values. Observed and predicted untreated AUDPCs are not included, as well as observed and predicted data derived from the standard cultivar King Edward.

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