Fungicide Resistance Action Group UK (FRAG-UK)

Potato late blight: Guidelines for managing fungicide resistance

Introduction

Potato late blight, caused by *Phytophthora infestans*, has been the major foliar disease of potatoes since its introduction to the UK in 1845.

Fungicides continue to be an important component of late blight control and as many as 18 applications to individual crops have been recorded in a single season. The average number of applications per crop varies according to the season, depending on the perception of risk but is around 8-11.

Originally the sprays were of copper compounds but these were superseded by the dithiocarbamates and organotins in the 1960s. The introduction in the late 1970s of the phenylamides brought a new dimension to blight control but there was a rapid development of resistance in the blight pathogen. This quickly brought about a change in the number of recommended applications.

Until recently the number of active substances available has limited anti-resistance strategies. Questions have been raised regarding the toxicity of some of the older materials, e.g. the organotins, and they have been withdrawn from use.

This guide sets out actions to minimise the risk of resistance development to existing and new active substances for control of late blight.

Of the current approved fungicides resistance to the blight pathogen has developed only in the phenylamides. Resistance to the phenylamides in *P. infestans* was first identified in the UK in 1981. In response to this development, a resistance management strategy was devised. This strategy has been very successful and phenylamides remain an important component in the blight programme. These fungicides are now only available as co-formulations with fungicides that have different modes of action (e.g. Fubol Gold - metalaxyl-M+mancozeb) applied preventatively. The resistance situation has remained stable over a number of years.

Resistance in other pathogens has been identified to some of the other fungicide groups used for late blight control (e.g. copper, QoI and cymoxanil). Although dithiocarbamates have been superseded in efficacy by new chemistry, they have formed effective partners with other materials as an anti-resistance strategy.
General guidelines on fungicide use

1. Avoid over reliance on a single fungicide group.
2. Design spray programmes which utilise the major attributes of each group.
3. Make full use of fungicides that attack several metabolic pathways (multisite fungicides), e.g. dithiocarbamates, chlorothalonil, fluazinam.
4. Observe the manufacturers’ recommendations on dose, timing and spray interval.

What is fungicide resistance?

Resistance is generally first noted when a fungicide ceases to provide effective control of a disease. The pathogen causing the disease becomes so insensitive to a fungicide that its performance in the field is adversely affected.

There are two types of fungicide resistance:

1. where a single major gene is involved, pathogens are either resistant or sensitive and the disease is either controlled or not (e.g. the phenylamides). Increasing the dose of a fungicide will not affect control
2. with polygenic resistance, the pathogen population contains strains with a range of sensitivities and control may be improved, at least temporarily, by increasing the dose applied (e.g. the DMI fungicides used against cereal pathogens).

Resistance occurs by genetic mutation. The mutation may already be present in the pathogen population at a very low frequency when the fungicide is introduced, or it may arise subsequently. Either way, exposure of the pathogen population to the fungicide gradually selects for the resistant strains until the point when it becomes detectable by poor fungicide performance. In cases where sexual recombination is involved in the life cycle of the pathogen, some of the progeny produced may be less sensitive. Resistant pathogen strains are sometimes not as fit as the wild type and may decline in frequency if the selection pressure is removed by withdrawal of the active substance. This may allow for its re-introduction at a later date. In some situations there may still be some benefit in maintaining the active substance in use with a suitable partner.

Cross-resistance in a pathogen occurs when exposure to one fungicide from a group confers resistance to other fungicides in the same group. Multiple resistance occurs where a pathogen is resistant to a number of fungicides from more than one group as a result of multiple exposures to different fungicides.
General resistance management guidelines

To reduce the risk of resistance developing in a pathogen population it is essential to put in place an anti-resistance management strategy at the outset. Managing resistance once it has occurred may not be effective.

The first principle of any anti-resistance management strategy should be to reduce the risk from disease by attention to good agronomic practice.

1. One of the most effective methods of reducing the risk from late blight is to grow cultivars with as high a disease resistance rating as possible. However, this is difficult to achieve when customers demand a specific cultivar, which may be highly susceptible to late blight, e.g. Russet Burbank for processed French fries. Current disease resistance ratings for GB listed cultivars can be obtained from the British Potato Council’s Web-site at www.potato.org.uk.

2. Where possible, avoid growing large areas of highly susceptible cultivars, particularly in locations where there is a history of high risk from late blight. Not only are crops at greater risk from becoming infected early in the season but also, once they have become infected, they serve as an inoculum source for neighbouring crops.

3. Dumps are the most important source of early inoculum. Destroy all dumps of discarded potatoes. Make a note of where dumps are and, before the spring, make sure any growth is destroyed. Sheeting with heavy gauge black polythene can prevent haulm growth; or young haulm can be killed by spraying with paraquat or glyphosate. It is important to check the dumps throughout the season for regrowth.

4. Control volunteers/ground-keepers. Although they tend to become infected later in the season and are, therefore, less likely to contribute to the early epidemics, they can still provide inoculum and infect crops as they approach harvest.

5. Source good quality seed and don’t be tempted to risk home-saved seed in years where there has been a high risk from tuber blight. Discard blight affected seed tubers. Only about one in 200 blighted tubers produces infected stems. However, 1% seed infection would produce about two primary infectors per hectare. Under warm moist conditions spores from these primary infectors will spread throughout the crop.

6. Make a timely start to spray programmes, when there is a warning of risk, or as crops meet along the rows, whichever is earlier. To ensure adequate protection ideally two sprays should have been applied to a crop before late blight would otherwise appear. Use forecast schemes and/or local knowledge to time applications more accurately.

7. Once spraying is underway, and where practical, adjust intervals according to risk (weather conditions/crop growth). Do not allow intervals to become too extended.

8. Do not apply fungicides when disease is well established in the crop, i.e. do not ‘chase’ the epidemic with fungicide, but consider burning off. This will not only help protect the crop from infection of the tubers but reduce late blight inoculum for neighbouring crops.

9. Use mixed formulations of active ingredients with different modes of action or from a different fungicide families, or target specific products in blocks to appropriate growth stages.
Table of active substances, their grouping and resistance risk
To date only resistance to the phenylamide group has been detected.

<table>
<thead>
<tr>
<th>Fungicide groups</th>
<th>Mode of action and mobility</th>
<th>Common name of active ingredient</th>
<th>Product name (example)</th>
<th>Resistance risk</th>
<th>Use</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benzamide</td>
<td>Single site inhibitor. Inhibits β-tubulin assembly in mitosis. Protectant, non-systemic</td>
<td>zoxamide</td>
<td>in Electis 75 WG</td>
<td>No resistance detected.</td>
<td>Can be used throughout the season at 7-14 day intervals with good activity against zoospore development. Formulated in mixture with mancozeb.</td>
</tr>
<tr>
<td>Copper</td>
<td>Multi-site inhibitor. Protectant, non-systemic</td>
<td>Bordeaux mixture copper oxychloride cupric ammonium carbonate</td>
<td>Wetcol 3 Cuprokylt Croptex Fungex</td>
<td>No resistance detected. Have been used since the early 1900s.</td>
<td>Currently can be used on organically grown crops. Limited use and can be phytotoxic. Have been superseded by the dithiocarbamates.</td>
</tr>
<tr>
<td>CAA</td>
<td>Mode of action unclear. Affects cell wall synthesis. Locally systemic.</td>
<td>dimethomorph, benthiavalicarb mandipropamid</td>
<td>in Invader in Valbon Revus</td>
<td>No resistance detected</td>
<td>Products containing a mixture of active substances or used in mixture with a fungicide with a different mode of action, up to 6 applications, making up no more than ½ the total number of sprays to control late blight may be made. When used alone, up to 4 applications, making up no more than ⅓ of the total number of sprays to control late blight may be made. No more than three consecutive applications of a CAA fungicide should be made.</td>
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<tr>
<td>Cyano-acetamide-oxime</td>
<td>Mode of action unclear. Prevents several cellular processes, including respiration, production of amino acids and cell wall permeability. Locally systemic.</td>
<td>cymoxanil</td>
<td>Sipcam C 50 in Curzate and Rhythm</td>
<td>Resistance described in other pathogens</td>
<td>Preventative and curative and can be used throughout the season on 10-14 day schedule. Short persistence used on own. Use with a suitable partner.</td>
</tr>
<tr>
<td>Cyanoimidazole</td>
<td>Single-site inhibitor. Inhibits fungal respiration and energy production at Qi site. Limited systemicity.</td>
<td>cyazofamid</td>
<td>Ranman TwinPack</td>
<td>No resistance detected</td>
<td>No more than three consecutive sprays recommended and should not form more than 50% of programme.</td>
</tr>
<tr>
<td>Diarylamine</td>
<td>Multi-site inhibitor. Stops cellular energy production. Protectant, non-systemic</td>
<td>fluazinam</td>
<td>Shirlan</td>
<td>No resistance detected.</td>
<td>Preventative. Strong action against spores. Best used at 5-10 day intervals. Used alone; so should not be used exclusively.</td>
</tr>
<tr>
<td>Dithiocarbamate</td>
<td>Multi-site inhibitor. Protectant, non-systemic</td>
<td>mancozeb</td>
<td>Dithane 945 Maneb 80 Manex</td>
<td>No resistance detected.</td>
<td>Can be used throughout the season, at 7-14 day intervals. A good partner for at risk active substances. Can be used alone.</td>
</tr>
<tr>
<td>Phenylamide</td>
<td>Single-site inhibitor. Interferes with synthesis of ribosomal RNA. Systemic.</td>
<td>benalaxyl metalaxyl-M</td>
<td>in Epok in Galben M in Intro Plus in Fubol Gold</td>
<td>A major resistance problem suddenly developed in 1981, with loss of blight control. A successful resistance management strategy was put in place and they still form an important component in spray programmes.</td>
<td>Best used at the beginning of the season. Maximum interval of 14 days. Only available in formulation with a partner of a different group. Apply a maximum of three sprays.</td>
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<tr>
<td>Phthalonitrile</td>
<td>Multi-site inhibitor. Protectant, non-systemic</td>
<td>chlorothalonil</td>
<td>Bravo 500 in Adagio in Merlin</td>
<td>No resistance detected.</td>
<td>Can be used throughout the season at 7-14 day intervals. A good partner for at risk active substances.</td>
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<tr>
<td>QoI</td>
<td>Single-site inhibitor. Inhibits fungal respiration at Qo site. Locally systemic.</td>
<td>azoxystrobin famoxadone fenamidone</td>
<td>Amistar in Tanos in Consento</td>
<td>A number of other plant pathogens have developed resistance to this chemistry; no resistance has been detected in late blight.</td>
<td>Use in partnership with a fungicide with a different mode of action. Maximum number of applications is six of which no more that three should be consecutive. In mixture can be used up to 50% of programme. Use alone as a pre-planting treatment (Amistar) does not contribute to the total number of applications, so the number of foliar treatments need not be reduced.</td>
</tr>
<tr>
<td>Acylpicolide</td>
<td>Delocalisation of spectrin-like proteins Protectant &amp; translaminar</td>
<td>fluopicolide</td>
<td>in Infinito</td>
<td>Resistance not known</td>
<td>Use after the rapid growth phase of the crop at 7-10 day intervals depending on risk. Formulated in mixture with propamocarb hydrochloride. Good activity on zoospores. Maximum number of sprays is 4 at full dose.</td>
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</table>
Impact on fungicide use of new strains of potato late blight

The pathogen, *P. infestans*, can reproduce in two ways:

1. asexually by producing sporangia (spores)
2. sexually when two mating types, designated A1 and A2, combine and exchange DNA.

When potato late blight was imported on infected potatoes from the Americas to Europe in the middle of the 1800s only the A1 mating type was introduced. The pathogen survived by asexual reproduction only, over wintering as mycelium in seed, volunteers and dumps.

In the mid-1970s, new strains of the pathogen of both A1 and A2 mating type were introduced into Europe in a quarantine-breaking shipment of tubers from Mexico. This provided the pathogen with the opportunity for sexual reproduction. Further migration during international trade impacted on the population dynamics of the pathogen in Continental Europe and the UK. The new strains have, over subsequent years, replaced the original population of A1 mating types.

‘New’ versus ‘old’ blight

In the USA, blight was reported to be more serious and to be occurring in areas where it was not previously a problem. There is no evidence for this in the UK. The ‘new’ population has been in the UK for over 25 years, so that current fungicides have all been tested against the ‘new’ blight population. There is also no evidence to suggest that ‘stem’ blight is a different disease or has increased over the years.

A2 mating strains

The proportion of A2 mating types in Great Britain (but not in Northern Ireland) has increased dramatically in the last two years: the A2 mating type was found at over 60% of sites sampled in 2006. An increase has also occurred in France and the Netherlands in recent years. The implications of this change are being investigated in BPC-funded research. The concern is not only that there is the opportunity for sexual reproduction to occur but that the resulting oospores will remain viable in the soil between crops and lead to early outbreaks of the disease. To date, no oospores have been found from fields cropped with potatoes in the UK.

Implication for control

Although there have been major changes in the structure of the blight population in the UK, at present there is no need to change current strategies for control.
Summary

- Where possible use a blight resistant cultivar.
- Eliminate sources of blight (infected seed, ground-keepers and dumps).
- Monitor local weather conditions.
- Apply fungicides as protectants.
- Select fungicides to suit local conditions.
- Do not chase the epidemic with fungicides, burn off early if blight levels are significant and delay lifting until the haulm has been dead for at least 14 days.

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The Fungicide Resistance Action Group – UK (FRAG-UK) was formed in 1995 to look at fungicide resistance issues and to publish information and advice relevant to the UK. The Group combines the expertise of industry with the independent sector to produce straightforward, up-to-date information on the resistance status of important disease in UK agriculture and to suggest way of combating resistance once it has occurred.

This leaflet can be downloaded from www.pesticides.gov.uk and www.potato.org.uk