Fungicide Resistance Action Group UK (FRAG-UK)

Potato late blight: Guidelines for managing fungicides 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.

In addition to a range of cultural counter-measures, fungicides continue to be an important component of late blight control. Whilst the average number of applications per crop is around 8-11 the actual number varies according to the prevailing weather and perception of risk; exceptionally up to 18 sprays/season have been recorded previously.

Originally the sprays were of copper compounds but these were superseded by the dithiocarbamates and organotins in the 1960s.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have been raised regarding the toxicity of some of the older materials, e.g. the organotins, and they have been withdrawn from use.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, the blight pathogen has developed resistance only to the phenylamides. Phenylamide resistance was first identified in the UK in *P. infestans* in 1981. In response to this development, a resistance management strategy was devised and phenylamides are now only available as co-formulations with fungicides that have different modes of action (e.g. Fubol Gold - metalaxyl-M+mancozeb) applied preventatively. This strategy was successful and phenylamides remained an important component in the blight programme, but since 2006 there has been an increase in one specific blight strain within the population in Great Britain and this is associated with an increase in phenylamide resistance. While the current levels of phenylamide resistance are high, they are not unprecedented. It remains vital that phenylamides are used as part of a resistance management strategy and, due to the current situation, the number of sprays should be restricted. Refer to the manufacturers for the latest advice.

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 and continue to play a critical role in anti-resistance strategies. Recent changes to EU legislation put their continued availability in question and may have serious consequences for resistance management in the future.

**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 improve 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 an approved dessicant. 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 produce 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. To ensure adequate protection two sprays should ideally 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 group</th>
<th>FRAC code</th>
<th>Common name of active ingredient</th>
<th>Product name (example)</th>
<th>Mode of action and mobility</th>
<th>Resistance risk</th>
<th>Use</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benzamides (pyridinylmethyl-benzamides)</td>
<td>43</td>
<td>fluopicolide</td>
<td>in Infinito</td>
<td>Delocalisation of spectrin-like proteins transaminar, protectant.</td>
<td>No resistance detected.</td>
<td>Use after the rapid growth phase of the crop at 7-10 day intervals depending on risk. Formulated as mixture with propamocarb hydrochloride. Good activity on zoospores. Maximum number of sprays is 4 at full dose.</td>
</tr>
<tr>
<td>Benzamides (toluamides)</td>
<td>22</td>
<td>zoxamide</td>
<td>in Electis 75 WG</td>
<td>Single site inhibitor. Inhibits $\beta$-tubulin assembly in mitosis. Protectant, non-systemic</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>CAA-fungicides</td>
<td>40</td>
<td>dimethomorph, bentiavacarb, mandipropamid</td>
<td>in Invader, Valbon, Revus</td>
<td>Phospholipid biosynthesis and cell wall deposition (proposed) Locally systemic.</td>
<td>Resistance known in Plasmopara viticola (vine downy mildew): no resistance detected in late blight</td>
<td>When used in mixture with a fungicide with a different mode of action, up to 6 applications, making up no more than $\frac{1}{2}$ the intended total number of sprays may be made. When used alone, up to 4 applications, making up no more than $\frac{1}{3}$ of the intended 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>Carbamates</td>
<td>28</td>
<td>propamocarb hydrochloride</td>
<td>in Consento, Merlin and Tattoo</td>
<td>Affects cell membrane permeability, fatty acids. Systemic.</td>
<td>No resistance detected.</td>
<td>Best used during period of rapid haulm growth. Use with a suitable partner.</td>
</tr>
<tr>
<td>Chloronitriles</td>
<td>M5</td>
<td>chlorothalonil</td>
<td>Bravo 500 in Adagio in Merlin</td>
<td>Multi-site inhibitor. Protectant, non-systemic</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>Copper</td>
<td>M1</td>
<td>Bordeaux mixture copper oxychloride copper ammonium carbonate</td>
<td>Wetcol 3 Cuprokylt Croptex Fungex</td>
<td>Multi-site inhibitor. Protectant, non-systemic</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>Cyano-acetamide-oxime</td>
<td>27</td>
<td>cymoxanil</td>
<td>Sipcam C 50 in Curzate and Rhythm</td>
<td>Mode of action unclear. Prevents several cellular processes, including respiration, production of amino acids and cell wall permeability. Locally systemic.</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>Dithiocarbamates</td>
<td>M3</td>
<td>mancozeb maneb</td>
<td>Dithane 945 Trimangol 80</td>
<td>Multi-site inhibitor. Protectant, non-systemic</td>
<td>No resistance detected. Have been used globally since the 1960s.</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>Phenylamides</td>
<td>4</td>
<td>benalaxyl metalaxyl-M</td>
<td>in Galben M in Intro Plus in Epok in Fubol Gold</td>
<td>Single-site inhibitor. Interferes with synthesis of ribosomal RNA. Systemic.</td>
<td>Major resistance developed suddenly in 1980 in Ireland and the Netherlands with loss of blight control.</td>
<td>Only available in formulation with a partner of a different group. Best used early season. Maximum interval 14 days. One blight strain has dominated the population in Great Britain since 2006; this is associated with phenylamide resistance. Check with manufacturers for advice on recommended numbers of sprays per crop.</td>
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<tr>
<td>Qil fungicides</td>
<td>21</td>
<td>cyazofamid/amisulbrom</td>
<td>Ranman TwinPack/Shinkon</td>
<td>Single-site inhibitor. Inhibits fungal respiration and energy production at Qi site. Limited systemicity.</td>
<td>No resistance detected</td>
<td>No more than three consecutive sprays recommended and should not form more than 50% of the intended programme.</td>
</tr>
<tr>
<td>Qol fungicides</td>
<td>11</td>
<td>azoxystrobin/famoxadone/fenamidone</td>
<td>Amistar in Tanos/Consento</td>
<td>Single-site inhibitors. Inhibit fungal respiration at Qo site. Locally systemic.</td>
<td>Resistance known in various species; 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. Used alone as a pre-planting treatment (Amistar) does <strong>not</strong> contribute to the total number of applications, so the number of foliar treatments need not be reduced.</td>
</tr>
<tr>
<td>Uncouplers of oxidative phosphorylation</td>
<td>29</td>
<td>fluazinam</td>
<td>Shirlan</td>
<td>Multi-site inhibitor. Stops cellular energy production. Protectant, non-systemic</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>
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</table>
Impact on fungicide use of new races of potato late blight

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

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

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.

Implication for control

*A2 mating strains*

The overall proportion of A2 mating types in the UK remained low for many years, in contrast to Continental Europe where there are indications of levels as high as 50%. However, the frequency of GB blight outbreaks in which the A2 mating type was found increased from 35% to 82% from 2005 to 2007. 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, oospores have not been shown to be a significant source of primary inoculum UK potato crops.

*Implications for control*

The population in Great Britain is now dominated by an A2 genotype (termed genotype 13_A2) that has spread rapidly since 2005 and displaced the previous population. This genotype was also identified in Northern Ireland in 2007, but is less common than in Great Britain. Studies have indicated that this genotype is fitter, more aggressive and able to overcome the resistance of some potato varieties. Close attention to spray intervals is therefore essential and reports from growers and advisors support this. An increase in levels of phenylamide resistance associated with this genotype has been reported. This has influenced the manufacturers’ advice on phenylamide products and they have reduced the recommended number of applications. Refer to the manufacturers for their latest advice.
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.
- Adjust spray interval and select appropriate fungicides to suit local conditions and blight risk.
- 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