Fungicide Resistance Management in Potato Late Blight

FRAG - UK

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Introduction

Potato late blight, caused by *Phytophthora infestans*, has been the major disease of potatoes since its introduction to the UK in 1845 causing losses by destroying foliage and by infecting tubers. Fungicides continue to be an important component of late blight control with up to 15-20 applications being used per season.

Copper compounds were first used to control late blight in the 1890s. By the 1960s, these had been largely superseded (except in organic production) by other broad spectrum contact fungicides such as dithiocarbamates. The introduction in the late 1970s of the phenylamides brought a new dimension to late blight control, but was quickly followed by the development of phenylamide-resistant strains of the pathogen. In countries where phenylamides were applied as the sole active ingredient for late blight control, notably The Netherlands and the Republic of Ireland, this led in 1980 to a complete failure of disease control with concomitant crop losses. This experience highlights the need for measures to minimise the risk of resistance development.

In the UK, resistance to the phenylamides was first identified in 1981. In response to this development, a resistance management strategy was devised. Phenylamides are now only available as co-formulations with fungicides that have different modes of action (e.g. Fubol Gold, metalaxyl-M+mancozeb), numbers of applications are limited and they must be applied preventatively. This strategy was successful until the appearance of the aggressive phenylamide-resistant 13_A2 (Blue 13) genotype of the pathogen in the early 2000s. While phenylamides may still be applied as components in late blight control programmes, a wide range of fungicides with different modes of action is now approved and widely used for late blight control.

FRAC classifies *P. infestans* as a high risk pathogen for fungicides targeting the RNA 2 polymerase (*viz.* the phenylamides), but classifies it as medium risk for all other modes of action. Resistance has been identified to some of the other fungicide groups used for late blight control (e.g. CAA, QoI and cymoxanil), but only in pathogens other than *P. infestans.* However, in the past 10 years, genotypes of *P. infestans* associated with reduced effectiveness of fluazinam against late blight have been reported in mainland Europe. One of these, 37_A2 (Dark Green 37) appeared in the UK in 2016 and increased in frequency to 24% of samples collected via Agricultural and Horticultural Development Board (AHDB) population monitoring in 2017. The role of fluazinam in late blight control programmes therefore needs to be re-considered, particularly in locations where 37_A2 is common; field performance of the other late blight control fungicides does not appear to be impacted. Multi-site fungicides such as mancozeb remain important as effective mixture partners in anti-resistance strategies.

For further guidance on potato late blight management, access the AHDB website for reports and management tools potatoes.ahdb.org.uk/online-toolbox/fight-against-blight-tool).

Resistance terminology

Resistance occurs when a pathogen becomes so insensitive to a fungicide that the fungicide's field performance is impaired. Resistance can arise rapidly and completely so that disease control is lost in a single step. More commonly, resistance develops gradually so that the pathogen becomes progressively less sensitive. When this happens there is usually no initial detectable loss of control, but it may decline over time.

FRAG-UK

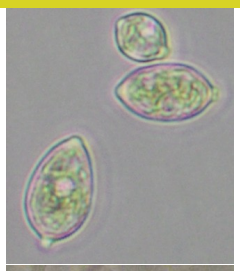
The Fungicide Resistance Action Group - UK (FRAG-UK) is a forum to look at fungicide resistance issues and to publish information and advice relevant to the UK. The group combines the expertise of industry with the independent sector to produce up-to-date information on the resistance status of important diseases in UK agriculture and to suggest ways of combating resistance.

Integrated Control and Resistance Management Guidelines

Adopt an integrated approach to disease and crop management to avoid over-reliance on fungicides, which increases the risk of selecting resistant pathogen strains.

- **Cultivar choice:** growing cultivars with as high a disease resistance rating as possible is one of the most effective ways of reducing the risk from late blight, but is difficult if customers demand a specific highly susceptible cultivar. Disease resistance ratings for GB listed cultivars can be obtained from the AHDB Potato Variety Database varieties.ahdb.org.uk. If possible, avoid growing large areas of highly susceptible cultivars, particularly in locations prone to late blight. Very susceptible cultivars not only risk becoming infected early in the season, but may also act as sources of infection for neighbouring crops.
- **Outgrade piles:** these are an important source of early inoculum. Destroy all piles of outgrade potatoes, killing any growth before crop emergence. Sheeting with heavy gauge black polythene can prevent haulm growth or young haulm can be killed by applying an approved herbicide. Check outgrade piles throughout the season for re-growth.
- **Control volunteers/ground-keepers:** these can sometimes provide inoculum to infect crops relatively early in the growing season as well as later.
- **Seed:** source good quality seed and don't risk planting home-saved seed after years where there has been a high risk of tuber blight. Discard and destroy blighted seed tubers (see advice above for outgrade piles).
- **Start spray programmes promptly:** start when there is a warning of risk (1. weather-based, 2. local outbreak or 3. transmission from infected seed). The growth stage at which the fungicide programme should start will be dictated by the risk, e.g. crop emergence for 3. In the absence of risk, the timing of the first fungicide application should take account of crop-specific and local factors and the guidance on specific product labels. It's now common for the first application to be applied no later than the rosette stage; plants are most susceptible between crop emergence and when they have *c.* 10 leaves. Note that Smith Period criteria have been revised to take account of the current *P. infestans* population and replaced with Hutton criteria (see blightwatch.co.uk/professional/about/hutton-criteria).
- **Optimise application:** aim to maximise coverage of the canopy through selection of correct nozzles and use of water volumes appropriate for the growth stage.
- **Use appropriate spray intervals:** once spraying is underway, and where practical, adjust intervals according to risk (weather-based risk/crop growth rate/known local inoculum sources/Decision Support Systems); do not over-extend intervals and consider mobility and persistence of mixture partners where practical in order to protect the individual components of any fungicide mixture.
- **Avoid eradicant treatments:** do not apply fungicides when blight 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.
- **Make full use of fungicides with different modes of action:** avoid over-reliance on a single fungicide group, use co-formulations or tank mixes of different active ingredients, target specific products to appropriate growth stages and include multi-site fungicides e.g. mancozeb, chlorothalonil. Check product labels for manufacturers' recommendations on dose, timing and spray intervals, and restrictions on total and sequential numbers of applications.
- **Protect until the end of the season:** maintain protection of the foliage with fungicides until the foliage is dead. Where there is a risk of tuber infection, complete the spray programme with fungicides with tuber blight activity and different modes of action, apply a fungicide with the desiccant (check product labels for approved tank-mixes) and make further fungicide applications until the haulm is dead.
- **Minimise the risk of additional fungicide–resistant *P. infestans* genotypes being produced in the UK and contributing to epidemics:** the risk of further genotypes that combine aggressiveness and reduced fungicide sensitivity becoming established in the UK is higher if crops are infected by soil-borne oospores. Longer crop rotations can substantially reduce this risk, provided groundkeepers are effectively controlled.

Potato Late Blight (*Phytophthora infestans)*



The pathogen, *Phytophthora infestans*, can reproduce in two ways:

- Asexually by producing sporangia and zoospores.

  Asexual reproduction is very efficient: its rapid cycles are responsible for devastating epidemics and can lead to development of clonal lineages (genotypes).

- Sexually when the two mating types, designated A1 and A2, recombine and exchange DNA resulting in formation of oospores.

  Oospores, unlike sporangia and zoospores, can survive in the soil for several years in the absence of potatoes and if they germinate, give rise to new genotypes.

When potato late blight was imported on infected potatoes from the Americas to Europe in the 1840s it is believed that only the A1 mating type was introduced. The pathogen survived by asexual reproduction only, over-wintering as mycelium in seed, volunteers and outgrade piles. This applied worldwide (except in the pathogen's centre of origin) and for much of the 20th century it existed as a single A1 clonal lineage.

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

UK pathogen genotypes

Although in the UK new strains displaced the old clonal A1 late blight population entirely during the 1980s and 1990s, the proportion of A2 mating types remained low until 2005. In contrast, in some regions of Continental Europe levels of the A2 mating type approached 50%. In 2005, a new A2 genotype known as Blue 13 or 13_A2 was found in GB for the first time; this probably originated in Continental Europe. The occurrence of this genotype increased dramatically and by 2007 it was detected in 82% of GB outbreaks and was also found in Northern Ireland. This highly aggressive genotype dominated the GB late blight population until 2010 and was also dominant in Northern Ireland in 2009 and 2010. In recent years, its frequency has fluctuated and in GB another new genotype, 6_A1 or Pink 6, which probably also originated on the Continent, has been dominant in most years since 2011 (in Northern Ireland 6_A1 occurs more rarely and older A1 genotypes remain more common).

In 2013, a new, highly aggressive genotype 37_A2 (Dark Green 37) emerged in the Netherlands. This spread in 2014 and 2015 and was detected in GB crops in 2016 including in blighted tubers. In 2017, the frequency of 37_A2 increased to 24% of samples collected via AHBD population monitoring; it was identified in 44 outbreaks, all in England. It has not so far been detected in Northern Ireland, but this is based on limited data. Another new genotype (36_A2), first identified in mainland Europe in 2014, constituted nearly 5% of the mainland European population in 2016 and was detected in Eastern England in 2017. There are concerns that this genotype too might be associated with reduced fungicide sensitivity, but information on this is not yet available.

In recent years, although most UK *P. infestans* continues to belong to such clonal genotypes, the occurrence of miscellaneous types particularly in Scotland suggests the possibility that the pathogen is sexually reproducing and that oospores are acting as inoculum. There are two reasons why this is a concern: 1. sexual reproduction can generate novel pathogen genotypes, and 2. the resulting oospores may remain viable in the soil between crops and lead to early outbreaks of the disease. Work on this aspect of pathogen epidemiology is ongoing in GB funded by AHDB Potatoes and the Scottish Government.

Implications of pathogen genotypes for fungicide use

New genotypes such as 13_A2 and 6_A1 can impact on control. Studies have indicated that 13_A2 is fitter, more aggressive and able to overcome the resistance of some potato varieties. Genotype 6_A1 is also highly aggressive and may be favoured by slightly warmer temperatures than 13_A2. The presence of such genotypes requires closer attention to spray intervals.

Some clonal genotypes may be associated with either resistance or sensitivity to specific fungicides. Genotype 13_A2 is almost invariably phenylamide-resistant and 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. In contrast, 6_A1 is phenylamide-sensitive.

Newer genotypes of *P. infestans* such as 13_A2 are highly aggressive.

Genotypes 33_A2 (Green 33) and 37_A2, first found in The Netherlands in 2010 and 2013, respectively, are associated with reduced efficacy of fluazinam. In the absence of fluazinam, 33_A2 is out-competed by other *P. infestans* genotypes; its incidence declined rapidly following the adoption of modified recommendations for fluazinam use and it is now very rare in mainland Europe. This genotype did not become established in the UK: a few isolated occurrences were detected in GB in 2011 and 2012 followed by single incidents in the English Fens in 2016 and 2017. However, subsequently the more aggressive 37_A2 appeared and has become established in England. Genotype 37_A2 has shown reduced sensitivity to fluazinam in laboratory studies and has been associated with reduced performance of fluazinam in some field trials and commercial crops in several areas of the UK. With the emergence of this genotype, fluazinam should be used with caution and should not form a major component of blight control programmes. In areas where 37_A2 is present, the effectiveness of fluazinam will be reduced and fluazinam is not recommended for late blight control. In other areas, fluazinam should only be used in two- or three-way mixtures with a partner(s) with a different mode of action and with comparable persistence. The use of fluazinam at any point in the season will potentially select for resistant strains. Its use at the start of the season may select for a high proportion of such strains if 37_A2 is present in the seed and its use at the end of the season could leave a legacy of resistant strains of this genotype persisting as tuber blight. Its use as a sequence partner in late blight control programmes should be limited, and it should always be alternated with a product containing fungicides from a different group(s) (see Table). The other major late blight fungicides remain effective against this genotype, although its phenylamide sensitivity has not so far been reported.

Fungicide Groups for control of potato late blight

Fungicide Group (FRAC Code)	Active ingredient(s)	Mobility	Resistance risk	Use
Benzamides (pyridinylmethyl-benzamides) (43)	fluopicolide	Translaminar, protectant	No resistance detected	Use after the rapid growth phase of the crop at intervals suited to risk. Formulated as mixture with propamocarb hydrochloride. Good activity on zoospores. Maximum number of sprays is 4 at full dose.
Benzamides (toluamides) (22)	zoxamide	Non-systemic, protectant	No resistance detected (FRAC: Low to medium risk, resistance management required)	Can be used throughout the season. Good activity against zoospore development. Formulated as mixtures with fungicides with different modes of action. Maximum number of sprays depends on product.
CAA-fungicides (40)	dimethomorph benthiavalicarb-isopropyl mandipropamid	Translaminar, locally systemic	Resistance known in *Plasmopara viticola,* not *P. infestans*. Cross resistance between all members of the CAA group. Low to medium risk	When used in mixture with a fungicide with a different mode of action, up to 6 applications. When used alone, up to 4 applications, making up no more than 33% of the intended total number of sprays. No more than 3 consecutive applications of a CAA fungicide should be made.
Carbamates (28)	propamocarb hydrochloride	Systemic	FRAC: Low to medium risk. Resistance management required. No resistance detected.	Best used during period of rapid haulm growth. Use with a suitable partner.
Chloronitriles (M05)	chlorothalonil	Non-systemic, protectant	Multi-site inhibitor. No resistance detected.	Formulated as a mixture with cymoxanil. Maximum number of applications is 2 at full dose.
Cyanoacetamide-oxime (27)	cymoxanil	Translaminar, locally systemic	Resistance claims described. Low to medium risk. Resistance management required.	Recommended for use throughout the season. Short persistence used alone. Use with a suitable partner.
Dithiocarbamates (M03)	mancozeb	Non-systemic, protectant	Multi-site inhibitors. No resistance detected. Used since the 1960s	Can be used throughout the season. A good partner for at risk active substances. Can be used alone.
Phenylamides (4)	benalaxyl metalaxyl-M	Systemic	High risk: major resistance developed suddenly in 1980 in Ireland and The Netherlands with loss of late blight control.	Only available in formulation with a partner of a different group. Best used early season. The 13_A2 (Blue 13) *P. infestans* genotype common in the UK since 2006 is associated with phenylamide resistance. Check with manufacturers for advice on recommended numbers of sprays per crop.
Qil fungicides (21)	cyazofamid amisulbrom	Limited systemicity	No resistance detected. Resistance risk unknown but assumed to be medium to high. Resistance management required.	No more than 3 consecutive sprays recommended and should not form more than 50% of the intended programme.

Fungicide Groups for control of potato late blight

Fungicide Group (FRAC Code)	Active ingredient(s)	Mobility	Resistance risk	Use
QoI fungicides (11)	famoxadone fenamidone	Locally systemic	Resistance known in various species, but not detected in *P. infestans*.	Use in partnership with a fungicide with a different mode of action. Maximum number of applications 6 of which no more than 3 should be consecutive. In mixture can be used up to 50% of programme. Note that foliar applications of azoxystrobin for control of early blight (caused by *Alternaria* spp.) contribute to the total number of QoI applications, but a pre-planting treatment does **not**.
QoSI fungicides (45)	ametoctradin	Limited systemicity	Not cross resistant to QoI fungicides. Resistance risk assumed to be medium to high (single site inhibitor). Resistance management required.	Used in mixture with a fungicide with a different mode of action, up to 4 applications of any one product containing ametoctradin, no more than 3 consecutively.
Uncouplers of oxidative phosphorylation (29)	fluazinam	Non-systemic, protectant	Reduced efficacy has been associated with specific *P. infestans* genotypes detected in mainland Europe and the UK; 2015-2017, 37_A2 has increased in frequency in the UK during 2017 and is now quite widespread in England.	In areas where 37_A2 is present, the effectiveness of fluazinam will be reduced and fluazinam is not recommended as a component of late blight control programmes.
OSBPI oxysterol binding protein homologue inhibition (49)	oxathiapiprolin	Systemic	Resistance risk is medium to high (single site inhibitor)	Use in mixture with a fungicide with a different mode of action. No more than 3 applications should be made consecutively, with a maximum of 4 applications per crop. Minimum interval between applications is 7 days.

Acknowledgements & Disclaimer

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This leaflet is available at potatoes.ahdb.org.uk/blight
Further information on resistance is available from the FRAG-UK website cereals.ahdb.org.uk/frag.

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