growers' advice

Fight against Blight

Understanding Blight Populations: Step by Step Guide

Research into understanding blight populations has advanced considerably in recent years. As a result it has become increasingly more complicated to understand all the potential implications for blight control in Britain. This guide, based on current available knowledge, sets out the basics of blight populations, explains some of the associated terminology and outlines ongoing work in this area.

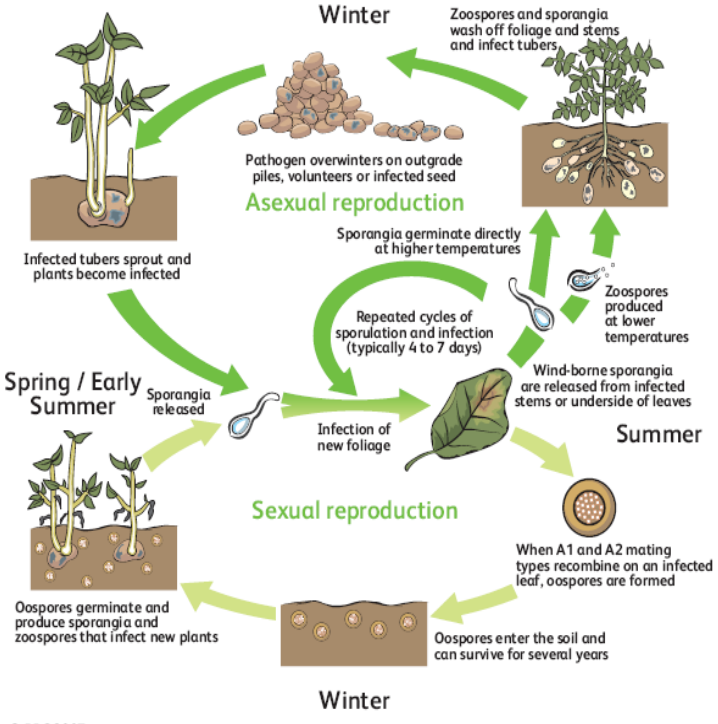
During the last 25 years there have been world-wide changes in the genetic make up of populations of the late blight pathogen *Phytophthora infestans*. These changes are thought to have occurred as a result of migrations of new strains of *P. infestans* from the Toluca Valley in Mexico where the blight pathogen is thought to have originated.

As a result of the changes in the *P. infestans* population, the life cycle of the pathogen is now more complex in some continental European countries (Figure 1).

The life cycle of blight pre-1980s

Before the 1980s, the European blight population was regarded as being asexual and consisting of one genetically similar clonal lineage of the **A1 mating type**. The life cycle of blight was fairly straightforward as the pathogen survived (over-wintered) from one season to another only on infected tubers. This emphasises the importance of healthy seed, and good control of outgrade piles and volunteers (see Figure 1). It is still thought that infected tubers remain the main source of late blight outbreaks.

Figure 1. Blight life cycle



The life cycle post-1980s - migrations & mating types

Genetic studies of blight populations were spurred on following the discovery in Mexico in the early 1950s of another mating type of blight which we call the **A2 mating type**. Where A1 and A2 occur together in an outbreak, they are able to mate and reproduce sexually (see sexual reproduction cycle of Figure 1).

SAC ADAS scri living technology

Since the 1980s the A2 mating type has been found in Europe, the USA and many other parts of the world. However, not only have the migrations resulted in the dispersal of the A2 mating type, there has also been a migration of more genetically diverse blight strains (called genotypes) of both A1 & A2 types. In some situations there is evidence that old A1 strains have been replaced by newer, genetically complex and possibly more aggressive A1 strains. The speed at which this displacement occurred indicates that the new strains of blight were fitter than the old ones.

Sexual reproduction (A1 and A2 recombining)

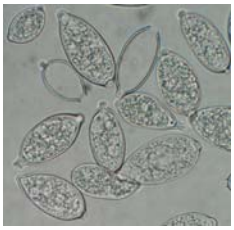
There are two key implications of sexual reproduction:

- It increases the genetic diversity of the blight population, potentially increasing the chances of more rapid adaptation to cultivar resistance and changes in traits like aggressiveness and fungicide sensitivity.
- The formation of oospores. (see 'Oospores' in section 'Terminology explained') Oospores are hardier than the air-borne sporangia and water-borne zoospores and may remain viable in soils between the shorter rotations used for some potato crops. As such, they could present British potato growers with the challenge of another source of inoculum each year.

Whilst there is no conclusive evidence to date of sexual recombination in Britain, it has been seen in other European countries, including The Netherlands and Scandinavia. Whilst oospores have been found in these countries, scientific understanding of oospore production, survival and possible control strategies, is still not clear.

Terminology explained

Sporangia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Sporangia are the airborne spores of the blight pathogen and are responsible for the rapid spread of the disease. Sporangia are small measuring 30 x 20 µm (1.2 x 0.8 thousandths of an inch). They are released into air currents for long distance spread or they may simply be splash dispersed by rain onto a nearby leaf. If washed into the soil they may also infect tubers. Delaying harvest by a minimum of 14 days after haulm death reduces this risk by allowing active sporangia to die.

Zoospores

Temperatures in the range 9-13°C are optimal for the cell contents of sporangia to divide to produce between 10-12 zoospores. Zoospores are equipped with 'tails' (called flagella) which enable them to swim in water either on the leaf surface or in the soil. These 'tails' help them move through any free moisture in the soil and, together with their small size, make them more suitable for infecting tubers.


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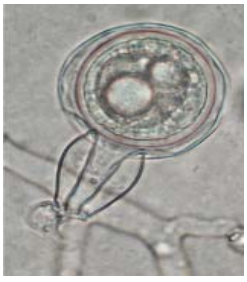
Oospores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Oospores are the product of the mating of the A1 and A2 mating types. When oospores are formed this allows the coupling and transfer of genetic material as happens in other plant and animal species where sexual reproduction occurs. These spores then lie dormant in the soil until they germinate. It is not known how long oospores survive in the soil in Great Britain, but it could be several years. However, oospores have not yet been found in British soils.

Oospores germinate in wet soils to produce a **sporangium** which then rapidly divides to release **zoospores**. Symptoms of disease arising from oospore (soil-borne) inoculum are very many lesions at the base of stems and on the lower leaves and the sudden death of patches of plants.


Oospore infection in Scandinavia Björn Andersson, SLU, Sweden

Genotype
Genotypes are individual 'blight strains' within the A1 and A2 mating types. Genotype refers to the individual DNA blueprint of each strain that is passed from one generation to the next. Each genotype/blight strain has different traits that are of interest to growers. These can include:

- Optimum temperature for spore production
- Fungicide sensitivity
- Aggressiveness
- Fitness

New molecular technology now allows the different genotypes or blight strains in Britain to be recorded. Once catalogued, the key traits can then be measured to help develop blight control strategies. With blight, different genotypes of the A1 and A2 mating types are often referred to by colour coding them. For example: 'Blue A2', 'Green A2' or 'Red A1'. Assessing which genotypes are present also allows change in blight populations to be accurately monitored. For example, if a large number of different genotypes with approximately equal numbers of A1 and A2 genes were routinely found, this would be a very strong indication that sexual reproduction and genetic recombination had taken place. New genotypes vary around the world and can be introduced through movement of infected tubers. Identifying any new genotypes will be important so that their specific traits can be understood.

Aggressiveness
Aggressiveness is an indicator of how quickly a blight strain causes a certain severity of foliar blight or a certain amount of tuber infection. Aggressiveness is a characteristic of the strain and is measured in comparison with that of other strains. For the blight pathogen, the situation is complicated by the fact that the disease affects both foliage and tubers and a strain that is particularly aggressive on leaves may not be on tubers due to the sometimes poor relationship between leaf and tuber resistance for cultivars.

Fitness
Fitness is a measure of how well a particular strain persists within the population and causes disease over an extended time period of several years. The fitness of a strain is influenced by many of its traits. For example, its abilities, under a range of diverse growing conditions, to cause enough tuber blight to ensure its own survival until the following growing season and to infect plants growing from infected tubers.

Aggressiveness and fitness are related but the relationship is not simple. For example, the most aggressive strains are not necessarily the fittest. They may destroy tubers before they can infect new plants. There are many different factors that affect both aggressiveness and fitness of blight strains.
A fuller account of aggressiveness and fitness is given in the document "Variation in aggressiveness in *Phytophthora infestans*: a review for the British Potato Council" written in 2006.

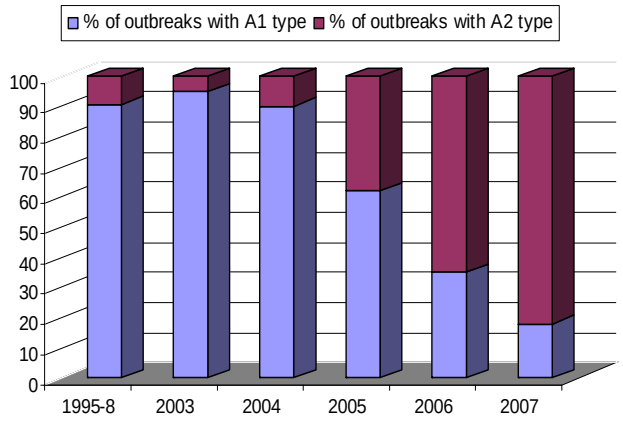
Population monitoring in Britain

Scientists have recently developed very sophisticated molecular diagnostic techniques to monitor changes in the genetic profile of blight populations. These techniques are similar to the genetic fingerprinting used in forensics that are common in modern policing.

In Britain, the A2 mating type was first detected in 1984 and the levels have been low (typically less than 10% of outbreaks tested). However, this started to increase dramatically in 2005 (see Figure 2).

In 2006, whilst 65% of outbreaks tested contained the A2 mating type, 22% of the outbreaks contained both A1 and A2.

Figure 2. Mating type analysis in GB 1995-2007



(Note: Data collected prior to 2003 are from different surveys. Data collected from 2003 onwards are from analysis of samples submitted by blight scouts as part of the BPC blight mapping service. One lesion per outbreak was tested in 2003-05 with multiple lesions per outbreak from 2006 onwards).

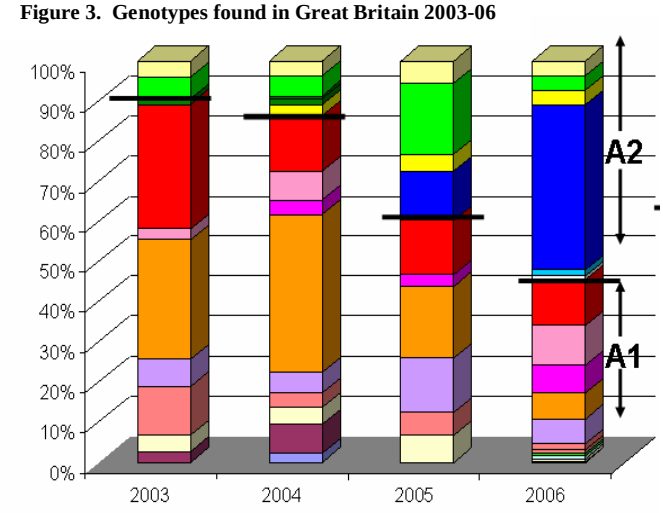
Differences within mating types

Within the A1 and A2 mating types there are different 'genotypes' (see terminology section).

Results to date in Great Britain have indicated that the population is currently dominated by fewer than 10 genotypes (see Figure 3), with little evidence of novel genotypes that might suggest sexual reproduction of A1 and A2 genotypes. The increase in the A2 mating type is explained by the dramatic spread of a single A2 genotype, referred to as '**Blue A2'.**

'Blue A2', first discovered in Britain in 2005, has displaced resident genotypes across much of the country.

Figure 3. Genotypes found in Great Britain 2003-06



What is happening in the rest of Europe?

Recent analyses of the A2 mating type across Europe paint a mixed picture.

Nordic surveys have disclosed that the frequency of A2 is nearer 50%. There is also confirmed oospore production in Scandinavian potato crops.

Researchers in The Netherlands investigated the source of 184 outbreaks of blight in four regions of the country between 1999 and 2005. For the four regions, oospores were considered responsible for 0, 0, 8 and 33% of the outbreaks.

In Northern Ireland, no A2 mating types have been detected since 1995 and in the Republic of Ireland the incidence has also been low and generally less than 5%.

Other research groups are reporting marked increases in A2 frequency. For example, in northern France the frequency of A2 strains has dramatically increased from 6% in 2003 to 35 % in 2005 and 75% in 2006.

What do we need to understand in Britain?

- Will the proportion of A2 in the population stabilise? If it does what will be the proportion of A2?
    - Will it be dominated by the A2 strain (a reverse of the situation in the 80s and 90s), again making sexual recombination and oospore production less of a risk?
    - Will A1 and A2 exist in more equal numbers therefore increasing the risk of sexual recombination and oospores?
- How are the new genotypes that we have now different from the old ones?
    - Are they more aggressive?
    - Is there any change in fungicide sensitivity?
    - Is there any substantial change in the resistance of varieties?
    - Are the new genotypes more active at lower and/or higher temperatures and/or relative humidities?
- Under the right conditions could oospores be produced by the genotypes of A1 and A2 that we have in Britain?
- If so, how long could they survive?

What are the possible implications of population changes for British potato production?

It is difficult at this stage to know what the implications would be in Great Britain. Even in countries with oospores, control strategies are still being investigated. However, the following outcomes could be expected.

- Earlier start to spray programmes.
- Closer intervals between sprays.
- The need for longer and cleaner rotations.
- Increased cost of control.
- The need for a soil test to assess the presence or absence of oospores to aid field selection.
- It may not be advisable to grow early crops under fleece in oospore-infested fields.

What is the BPC doing?

The BPC are consulted in the development of government funded research programmes to ensure all research in to blight is complimentary with BPC funded research. This includes blight research from the Scottish Government Rural and Environmental Research and Analysis Directorate (RERAD).

To help gain a full understanding of blight populations in Britain the BPC have commissioned a project consortium led by SCRI and including SAC, ADAS, CSL and Sárvári Research Trust to research the following areas:

- Determining levels of A2 mating type of blight in 2006, 2007 and 2008 potato crops.
- Examining specific early outbreaks to determine the likely source of inoculum with particular emphasis on the roles of oospores and seed-borne blight over the 2006, 2007 and 2008 seasons.
- Determining genetic diversity of British blight populations in 2006, 2007 and 2008 seasons and compare to that in previous years and other states.
- Analysing oospore production and survival under British conditions.
- Assessing the implications of, and risks arising from, the observed blight population structure and oospore prevalence and survival (incorporating information from other studies).

To keep up to date with current blight populations and best practice see
www.potato.org.uk/blight