Phytophthora infestans – the plant destroyer

With the arrival of new genotypes and increasing fungicide resistance, control of *Phytophthora infestans* continues to be a challenge.

While fungal, bacterial and viral pathogens continue to shape global societies, there are few plant-specific pathogens that can be regarded as having impacted the 19th and 20th centuries in the way *Phytophthora infestans* has. Aptly named, this plant destroyer is the cause of late blight of both potato and tomato, and its arrival in Ireland over 175 years ago contributed to the Irish famine, with the ensuing consequences still felt to the present day. In Ireland, while we no longer depend as heavily on the potato, globally potato production and consumption is growing, particularly in developing countries. As such, *P. infestans* continues to pose the most serious biotic threat to global potato crops, with intensive fungicide programmes rigorously implemented for its control on an annual basis.

Global crop threat

So what is it about this pathogen that makes it such a threat to Irish potato crops? Although late blight caused by *P. infestans* is often referred to as a fungal disease, it is actually an oomycete, more closely related to brown algae than to true fungi. The differences between oomycetes and fungi may be subtle but can have enormous consequences: for potato growers this is probably most evident in the different chemicals used to control them. Most fungicides used to control cereal diseases provide no control against late blight and those used for late blight have little or no effect on cereal diseases. The arrival of late blight in Europe and its resulting devastation also led to the development of the science of plant pathology with the discovery that microbes can cause the infection and are not only a symptom of disease. These findings form the cornerstone of plant disease control programmes, with manipulation of one or more of the components of the disease triangle – pathogen, environment or host potato crop – providing control. Over the past 175 years *P. infestans* has demonstrated that it can also readily adapt to overcome any changes imposed, whether directly or indirectly, in the form of the host or environment. As production systems in Ireland and throughout Europe have changed, the economic consequences associated with even minor outbreaks of the disease have increased. This has led to increased monitoring of the pathogen for changes that may indicate potential changes in disease development and subsequent control measures.

New genotypes

The first method readily utilised to monitor potential changes in the pathogen is an assessment of the structure of the population based on its mating type. *P. infestans* can reproduce both clonally or by hybridisation between different mating types designated A1 and A2, which produces long-lived oospores, hence altering the pathogen lifecycle. For this to happen, a co-infection must occur on the same leaf or tuber. As *P. infestans* is not native to Ireland it has until now been dominated by a small number of clonal genotypes that overwinter in infected tubers and spread from volunteer plants and cull piles. Initially, population monitoring suggested that a single clonal genotype designated US-1, which was A1, dominated the *P. infestans* population in Ireland and globally from shortly after the famine until the late 1970s/early 1980s. At this time a second major migration of *P. infestans* from South America is believed to have occurred, followed by potentially another in the late 1990s/early

When weather conditions are conducive to the spread of late blight, the speed at which the disease can spread and devastate potato crops if left unprotected is astonishing, as is evident from the above pictures from the Teagasc late blight trials in 2009 (pictures taken two weeks apart).

2000s, both indicated by the increased detection of the A2 mating type in field populations. The first A2 strain was confirmed in Ireland in 1987 but had little impact on disease control or oospore formation. Analysis of the 1996 population structure suggested that the original US-1 genotype had been completely displaced but the population was still clonal with a few genotypes dominating, which were mainly A1 and sensitive to the phenylamide fungicides. The arrival of the A2 genotype Blue-13 or EU_13_A2 in the mid 2000s, which was more aggressive and resistant to the phenylamide fungicides, had a major impact on all aspects of late blight, from disease control to monitoring. Blue 13 quickly established and with other A1 genotypes present in the population, mating type determination alone was no longer useful to determine whether *P. infestans* was sexually active in Ireland. With the advent of molecular techniques, including simple sequence repeat markers (SSRs), it has been possible to monitor *P. infestans* in more detail, down to specific strains. Through continued monitoring of populations, it is believed that although both mating types can now readily be identified in the same field, and in some instances on the same plant, there is no evidence of genotypes resulting from sexual combination. For the continued management of the disease this is of importance as oospores, in addition to increasing genetic diversity in the population, are long lived and can survive in soil in the absence of the host and initiate late blight epidemics following planting.

Fungicide resistance

The arrival of Blue-13 presented an additional hurdle for the control of late blight. The genotype combined increased aggressiveness with resistance to the phenylamide fungicides and no fitness penalty, which had traditionally maintained resistant genotypes at a low level in the population. Furthermore, Blue-13 was more aggressive on previously resistant varieties, which changed variety resistance ratings. Throughout Europe strains of this genotype rapidly dominated the *P. infestans* population, resulting in increased intensity of fungicide applications. Unfortunately the emergence of Blue-13 has marked the emergence of a continual wave of increasingly aggressive genotypes, each displacing the other. Contemporary Irish populations are now composed of up to five major genotypes that we know of, each with their own unique advantage. For instance, in 2019 the genotype EU_37_A2 was first detected in the Republic of Ireland. This genotype displays resistance to the fungicide fluazinam, which until this point was heavily relied upon by Irish growers. As it remains difficult to predict what strains of *P. infestans* may be present in any given field prior to the outbreak of the disease, Irish growers have adjusted control programmes by increasing the intensity of fungicide applications to ensure control of all potential genotypes. Even with this increased vigilance, given the variability of the Irish weather, the risks posed by late blight to Irish potato production continue to be immense, highlighting the need to devise fully integrated disease control strategies, including pathogen monitoring, host resistance deployment and accurate forecasting.

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