LA NIÑA ALERT – THE HEIGHTENED RISK OF LATE BLIGHT

Plant diseases can change the world. The English took to drinking tea when their coffee plantations in Ceylon (now Sri Lanka) were wiped out by coffee rust in 1869. Both the Salem witch trials and – more arguably – the French Revolution have been blamed on ergot infection of rye, the toxin of which causes muscle spasms, mania and hallucinations. By Jenny Ekman

However, there is surely no plant disease which has had as big an impact on human history as late blight – *Phytophthora infestans*. We are all familiar with the terrible story of the Irish famine (1845-1849). The disease caused at least 1.5 million deaths from hunger and more than a million permanent emigrations, spreading the Irish people around the globe. The population of Ireland has never recovered.

Late blight remains one of the most destructive and costly plant diseases around the world today. It can cause complete crop loss within weeks, as well as have significant effects on quality and yield on surviving crops.

According to Agriculture Victoria senior research scientist Dr Rudolf (Dolf) de Boer, more pesticides are used worldwide for control of late blight on potatoes than for any other plant disease. There are also major issues with fungicide resistance, making it ever more difficult to control.

WHAT IS *PHYTOPHTHORA INFESTANS?*

P. infestans is not actually a fungus but belongs to a group of organisms known as the oomycetes. These produce mobile, swimming spores called zoospores.

In most of the world there are two forms of *P. infestans*; the A1 and A2 'mating types'. The sexual stage of the life cycle occurs when compatible strains of A1 and A2 mating types both infect a plant. The two strains merge, producing oospores with recombined DNA. These oospores are very robust, potentially surviving many years in the soil without a host.

In Australia we only have the A1 type, a relic of the original Irish late blight pathogen. This means that the lifecycle occurs solely through asexual growth and division. As robust oospores are not produced, the pathogen can only survive in living host material.

This means that *P. infestans* can be more readily controlled through crop rotation here than in many countries. Lack of genetic variability also makes it more difficult for resistance to develop.

However, it does not mean the fungus does not present challenges. There are a number of highly aggressive strains of the A1 (and A2) mating types. For example, an A1 strain present in Papua New Guinea would likely create major issues if introduced here, potentially replacing our existing strain. Similarly, a very aggressive A2 strain has become dominant in Scottish crops. Although it can potentially produce oospores, this strain still mainly spreads through infected materials, possibly due to incompatibility with local A1 types.

HOW DOES IT SPREAD?

The late blight pathogen was introduced to the Australian colonies on infected seed potatoes sometime in the 1840s. However it was not until the early 1900s that major epidemics occurred, the infection spreading from farm to farm on infected seed potatoes. These days the pathogen is most likely to survive in small pockets, most likely as latent infections in old tubers and pre-emergent sprouts.

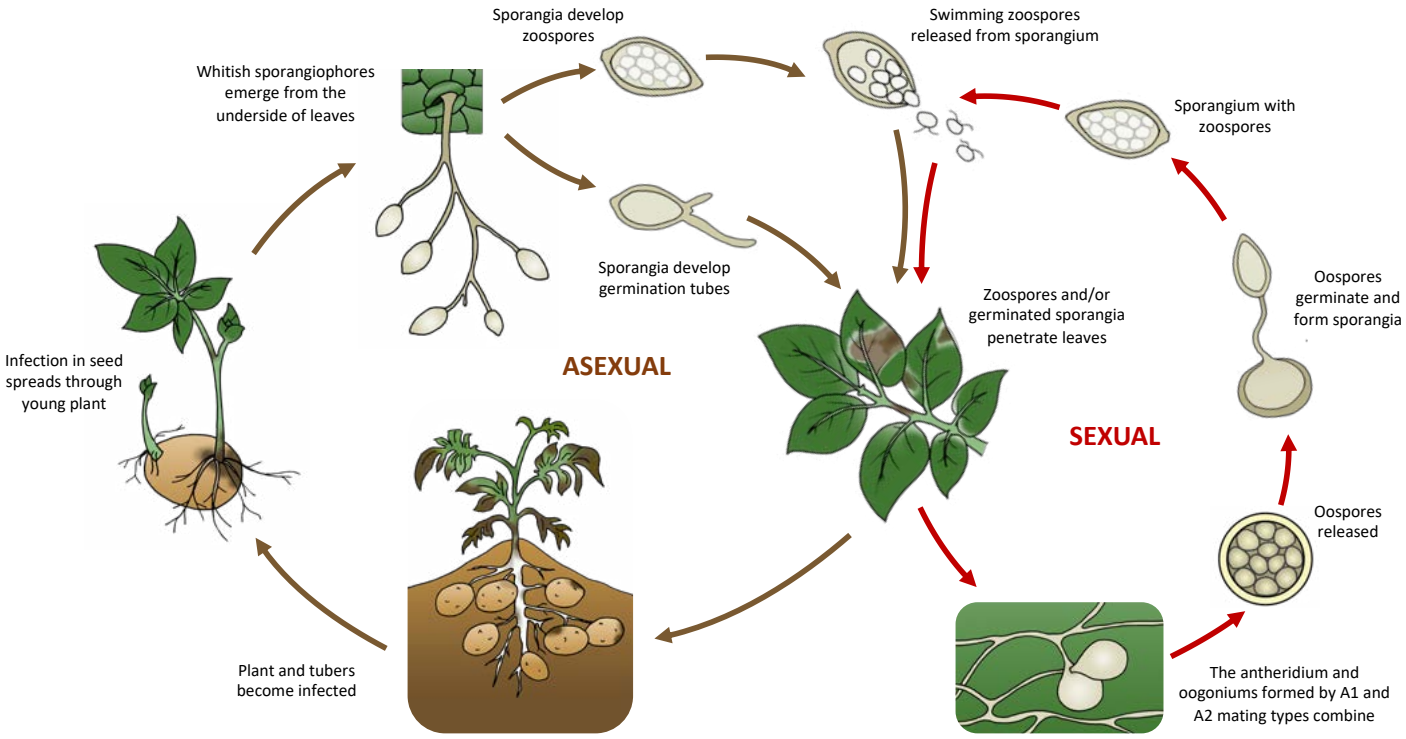

Figure 1. P. infestans lifecycle. Note that only asexual reproduction occurs in Australia as the A2 type is not present.

After emergence, the pathogen follows the growth of the plant from the tuber to the young stems. Here it spreads onto the new foliage and sporulates. What look like upside-down trees of whitish sporangia emerge from leaf lesions and through the leaf stomata. The sporangia are then spread by wind, or in rain or irrigation water to neighbouring plants, fanning out into the crop.

While sporangia can germinate directly, they can also release swimming zoospores. The zoospores actively move through water films on the leaves down the stems, to be further spread by wind and rain.

Sporangia can also be washed down into the soil to infect tubers.

Modern farming practices greatly reduce the risk of spreading the Australian A1 strain in seed. However, spread is still possible, as latent infection of tubers is not detectable through visual inspection.

Typical infection cycles are only 5-7 days. Where free moisture is present, the pathogen can spread rapidly, with devastating effects.

Between potato crops, the pathogen can survive in seed and waste tubers, volunteer potato plants and alternative Solanaceous hosts.

WEATHER DRIVES DISEASE

Late blight generally occurs sporadically, being very much driven by weather conditions. "Typical late blight weather is warm and sultry," states Dr de Boer. "Lengthy periods of warm, still and humid overcast days with cool nights and warm days present the greatest risk of disease."

Dew is the number one issue. "Rain in itself does not pose the risk. Rather, it's the prevailing weather conditions that accompany the rainy periods which are the problem."

In most potato growing regions of Australia, conditions are too dry during spring through to autumn to cause high levels of disease, especially if humidity is low. However, predictions of a wetter than average November suggest that risk may be higher this year.

There is a strong correlation between wet seasons and outbreaks of late blight. "La Niña is the link here," suggests Dr de Boer. "La Niña years are when I get the most phone calls about late blight." Outbreaks occurred between 1998-2000, 2010-2012 and now 2021-2022, all of which were La Niña cycles. While La Niña predominantly affects the north east, it can also bring late blight conducive conditions further south.

WHERE DOES IT COME FROM?

Changing weather conditions means there are big gaps between outbreaks of late blight. But we know that the A1 strain can only survive on live materials – where does it go?

According to Dr de Boer "It most likely survives in pockets of very protected areas which have favourable microclimates. These allow the fungus to regularly complete its lifecycle during drier seasons. It may be present on old tubers, self-sown potatoes or other hosts such as kangaroo apples. But that's a big question mark".

Prof. Steven Johnson agrees. In his experience, epidemics don't start from

volunteer potatoes, but these plants will let an epidemic continue. "Late blight likes a young lunch, it prefers very actively growing tissue. Self-sown potatoes tend to be a little slower, so they get infected later in the season." While some mystery remains about where infection comes from, certainly the importance of using clean seed can't be overstated.

Because of the sporadic nature of conducive weather conditions, the pathogen experiences "boom" and "bust" cycles in the Australian environment. In a conducive season it will take some time for the population to build up from a very low base and the disease may not be so obvious. However, if followed by a second favourable year, the disease is likely to be much more serious. This resulted, for example, in a higher disease incidence the 2021/22 La Niña cycle compared with the 2019/20 cycle.

LATE BLIGHT SYMPTOMS

In the early stages of infection, leaves develop pale, grey green lesions. These expand rapidly, turning brown to black with pale margins. Under very wet conditions the lesions become black and slimy. Necrotic lesions can also be found on the stems (Figure 2).

Under humid conditions white fuzz develops on the infected stems and undersides of the leaves (Figure 3). These contain the wind and rain borne sporangia, which spread to neighbouring plants. Once this occurs plants generally collapse, with the infected zone clearly visible as a patch of dead and dying plants.

The spores produced on the upper parts of the plant eventually wash down into the soil and infect the tubers. Initially, the tubers develop a tan-brown reddish or purplish rot just under the skin. Irregularly shaped, sunken areas develop, turning into wet and slimy lesions. Bacterial infections often then attack the tubers, causing complete collapse.

Figure 2. As the disease develops, pale grey lesions on the leaves expand and turn brown (a). Under very humid conditions (such as in the highlands of Java) these lesions can become black and slimy (b). Necrotic areas spread through the stems (c). Eventually the plant dies, as shown on this 4 week old, untreated 'Sequioa' plant (d). - Images: R. de Boer.

Figure 3. Under humid conditions, whitish sporangia containing zoospores emerge on leaf undersides, mainly around the active margins of the leaf lesions - Images: R. de Boer

LATE BLIGHT IN AUSTRALIA

The epidemics of the early 1900s saw outbreaks occur across all Australian potato growing districts, from the Atherton tablelands, across to Perth and down to Tasmania. Fortunately for WA it has not been recorded since. In fact, since the 1980s control has improved around Australia, with outbreaks now concentrated in pockets of NSW, Victoria, South Australia and Tasmania.

After the 1909 outbreak plant pathologists looked at where else it could be found. They found the disease widely on potatoes, tomatoes and the weed kangaroo apple (*Solanum aviculare*) – which was frequently growing around and within potato production areas. Curiously, black nightshade plants (*Solanum nigrum*) growing in late blight affected crops were immune.

"It is important to note that the 1909-11 epidemic occurred with a La Niña weather event, just like the La Niña we are having at the moment," states Dr de Boer.

KEEPING A2, AND NEW STRAINS OF A1, OUT

Not having the A2 strain in Australia is definitely a major advantage.

The A2 strain initially came out of Mexico, spreading first to the United States and Europe, but eventually – thanks to the trade in seed potatoes – to many other potato producing countries around the world. This means there is much wider genetic variability in pathogen populations in most countries outside Australia.

Dr DeBoer has worked extensively on identification of *P. infestans* strains. "These new strains are far more aggressive than the old clonal strains we had before." He explains "they have a shorter lifecycle; instead of having a turnaround of 4 to 7 days, this is reduced to 3 to 5 days or even quicker. Many are metalaxyl resistant, and some A2 strains are also 'resistance busting'; there's a strain kicking around the UK which has overcome resistance bred into new varieties. Wider genetics mean that these new strains are also more adaptable to wider temperature regimes, lower moisture and so on."

It is clearly critically important to keep A2, and new strains of A1, out of Australia. It is also important to regularly identify strains present in Australia to identify if incursion has occurred.

From 1998 – 2001 Dr de Boer led a HIA funded project examining the strains then present in Australia. "We got a lot of help from industry – chemical companies, agronomists and growers all collected samples. The pathogen was tested for mating type, metalaxyl resistance and DNA fingerprint analysis."

"All our samples were a single clone of A1 mating type (designated AU-1) and very sensitive to metalaxyl. It's the same genotype as the strain that caused the Irish famine (FAM-1). This was displaced by new strains elsewhere around the world by the 1950s, so is essentially a relic found only in Australia and New Zealand."

What this means is that there have been no new introductions of *P. infestans* to Australia in more than 100 years. This is undoubtedly due to the quarantine systems that were established in Australia in the early 1900s, including mandatory testing of seed since 1913. There have been no imports of unprocessed potatoes for several decades.

However, risk remains. The A2 strain is present in Indonesia, and various A1 strains are present in Thailand, PNG and East Timor. There is always the possibility of someone bringing back some oospores on their boots or a souvenir tuber in their bag.

CONTROLLING LATE BLIGHT

Prevention is clearly the best method of control. This means using only certified seed, ensuring there are no volunteers left over from previous crops, and removing Solanaceous weeds, especially kangaroo apple.

Minimising any initial source of infection will delay spread during cropping. However, even a tiny amount of inoculum can spread exponentially under wet conditions, infecting the entire crop. When risk is high, they key questions are therefore:

- When to spray
- What to spray
- When to salvage

When to spray is often based on prediction modelling. According to Prof. Steven Johnson from the University of Maine, "We don't control late blight, we manage it. Going for zero tolerance is expensive and can be an unreasonable approach."

According to Prof. Johnson, the most important period is during early crop development. "If late blight gets a foothold during early growth, then you just can't keep up with it, as it develops exponentially early in the crop cycle." Once a large amount of inoculum is present in the field, no amount of fungicide will be able to hold it back.

This means that the intervals for subsequent applications should be weather driven. "So not every Friday night," says Prof. Johnson; "Calendar based spray schedules are not environmentally or economically sound."

Figure 4. Timing sprays appropriately is essential. The healthy crop in the foreground has had just one more fungicide application than the late blight affected crop to the left (Image: S.B. Johnson).

Figure 6. It is essential to cover the entire crop when applying fungicides. In this field the grower has left the edge of the crop unsprayed; this has allowed development of late blight, which can then spread to the remainder of the crop (Image: S.B. Johnson).

What to spray includes choices of;

- Contact (protectant) fungicides, which don't move into the plant tissue e.g. chlorothalonil, mancozeb, fluazinam
- Translaminar fungicides, which move into the plant tissue but don't move within the plant vessels e.g. dimethomorph (Acrobat® SC)
  - Move slowly through the plant leaf from top to bottom
  - These have longer lasting effects than contact fungicides
- Systemic fungicides, which move into the plant tissue and around the entire plant
  - Most are upwardly mobile, so can protect new foliage as it emerges (e.g. Infinito®)
  - Some are also downwardly mobile (e.g. Ridomil Gold®)

Prof. Johnson suggests using either full or partial rates at different points in the season and emphasises the importance of putting the fungicide where and when it is needed. "You've got to replace eroded material," he says. "That could mean that just one extra application has a big impact on rates of disease." (Figure 4).

This is especially important when the plant is rapidly increasing its leaf area (Figure 5). "Early in the season the plant can double in size every three or four days. If you're putting on a protectant, the plant is going to outgrow it very quickly. This is when it's best to use a systemic, or at least one of the translaminar products, that can move with the new growth."

Getting good coverage is essential, so applicators need to understand their equipment. This means considering boom height, nozzle type and pattern, forward speed and using adequate water volume to fully cover the crop.

"I've seen crops where the grower didn't want to run the extra half pass with the boom spray to get right to the edge of the field, so those plants

got late blight. This then became a spreader row for the rest of the field," comments Prof. Johnson (Figure 6).

Salvaging involves regular checks for areas of late blight within the crop. For example, if seed introduces disease to one area of the field, it may be better to kill those plants to prevent spread, protecting the rest of the crop (Figure 7). Prof. Johnson suggests an area around 10x the size of the initial outbreak, to be sure to get good control.

"Late season finds are important as well. We don't get a lot of tuber infection when conditions are warm, so in this case you might want to go for an early kill rather than leaving the crop to full term," suggests Prof. Johnson.

CONCLUSIONS

Late blight is something growers are likely going to have to deal with this season. As Prof. Johnson says "I'm pretty darn sure that you're going to have late blight this year, and it's going to come early. Certainly, in the Ballarat area, and east and west of Melbourne, you're starting out with a higher initial level of inoculum, and that's what is likely to drive any epidemic hard".

In central northern Tasmania late blight was a major issue last season, with infection occurring relatively early in the season. There are reports of yield losses up to 30%. There is a high risk that the pathogen has overwintered in / on other host plants or volunteer potatoes (Figure 8).

Dr Nigel Crump from AuSPICA also sees an increased risk of late blight this year. "Generally in Victoria we see late blight fairly late in the season, as inoculum builds up. It's a community disease, rather than individual paddocks, and a community approach is important for control."

Early detection is essential, so checking the crop regularly when weather conditions are right for infection is critical. If late blight starts to get away, it's virtually unstoppable.

Testing services are available, so sending in samples to confirm presence of late blight is highly recommended. These will also help to confirm that we still only have the A1, metalaxyl-sensitive strain present.

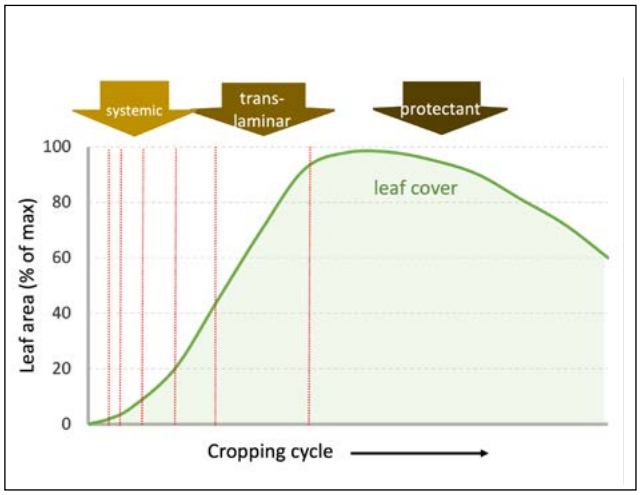

Figure 5. The red lines indicate doubling of the plant canopy area. If forecasting indicates risk of disease is high, systemic or translaminar fungicides should be applied when leaf area is increasing rapidly, and protectants once new growth has slowed or ceased. Derived from data presented by Prof. Stephen Johnson.

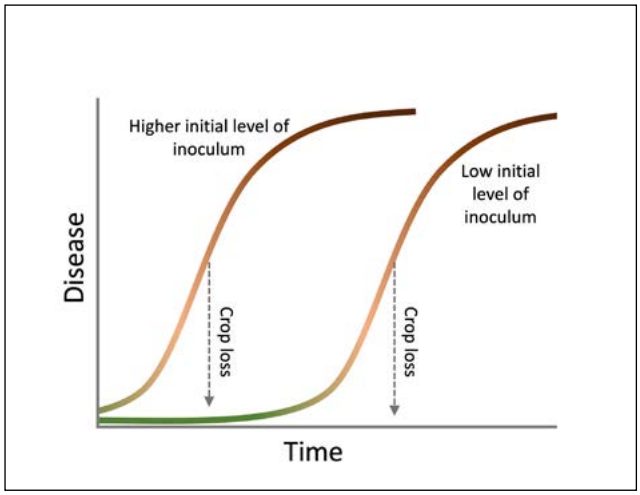

Figure 8. Even a small increase in the initial level of inoculum can see late blight become a major issue much earlier in the season.

Figure 7. If an outbreak of late blight occurs within a field, the best option may be to kill those plants, rather than risk the disease spreading into the remainder of the crop. (Images: S.B. Johnson).