Bacterial ring rot of potato – the facts

(Clavibacter michiganensis subsp. sepedonicus)

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1. Introduction
1.1. Bacterial ring rot of potato is caused by the bacterium Clavibacter michiganensis subsp. sepedonicus (Spieckermann & Kotthoff, 1914) Davis et al., 1984 (Cms).
1.4. Direct losses result from yield reduction due to wilt, stunting and tuber rotting in field and store. Statutory control measures include destruction of infected and associated potato crops and restrictions on further cropping. Indirect losses also include disinfection and disposal costs and effects on export trade.
1.5. The pathogen has been spreading throughout Europe in recent years and the first UK case of ring rot recently occurred in a Welsh seed farm in 2003.

2. The pathogen
2.1. Cms is a Gram-positive, non-spore-forming, rod-shaped bacterium, 0.5 – 1.0 µm in size.
2.2. Isolates of the pathogen are relatively homogeneous. The narrow host range and the biotrophic character of Cms are probably related to the low diversity of this pathogen. The risks that variant strains could escape detection or may adapt in their biology and ecology are therefore considered low.
2.3. Slow growth of Cms hampers isolation methods. The bacterium does not compete well with other bacteria, a fact which often hinders its detection.
2.4. It is difficult to produce specific high-titre antibodies against Gram-positive bacteria and antisera against Cms often lack specificity.
2.5. Cells and colonies often vary in morphology, requiring high levels of experience for their recognition.
2.6. Ring rot, particularly in its latent form, is renowned to be difficult to diagnose, especially within the time frames appropriate for statutory actions.
2.7. Cms cannot resist high temperatures and is susceptible to a wide range of disinfectants, providing opportunities for its control on surfaces and equipment.

3. Symptoms
3.1. Cms can latently infect and colonise potato plants and tubers without causing any symptoms and can remain undetected over several generations. Conditions that favour infection and colonisation of potato by the pathogen may differ from those, which favour symptom expression.
3.2. Disease expression varies widely with potato cultivar and environmental conditions. Symptoms are likely to develop most rapidly at temperatures between 18 and 25 ºC but optimum temperatures may vary between potato cultivars. High soil moisture and low light intensity have also been reported to increase the rate of symptom development.
3.3. Yield loss may be significant, even in the absence of typical ring rot symptoms, due to stunting of normal growth, which usually remains unnoticed.
3.4. Initial tuber symptoms can only be observed by cutting the tuber transversely near the heel end. The vascular tissues become glassy and darken, starting from the stolon.

3.5. A rot develops in the vascular bundles and a creamy-yellow, cheesy, bacterial slime can be expressed on squeezing the cut tuber.

3.6. Rotting extends around the vascular ring and eventually spreads into the cortex tissues where it can cause internal hollowing and externally visible dark blotches under the periderm which eventually cracks.

3.7. Secondary rotting by other bacteria and fungi are common in the latter stages, finally resulting in either complete breakdown or mummification of the tubers.

3.8. Wilting symptoms in the haulm usually appear late in the season, if at all. Wilt develops slowly and is initially limited to the leaf margins which soften and curl upwards.

3.9. Affected leaves develop light green to pale yellow areas between the veins and eventually become necrotic and desiccate.

3.10. In extreme cases the whole plant wilts and/or desiccates and is killed. Partial wilting is more common, allowing affected plants to survive until harvest, and is difficult to detect amongst the naturally senescing crop.

4. Geographic Distribution

4.1. Ring rot has been reported from some 31 countries distributed over 5 different continents.

4.2. Although reported for the first time in Germany in 1906, it was found to be widespread across North America in 1940.

4.3. It has been regarded as a disease of cool northern regions of America, Canada, China and Northern Europe and Russia but is currently spreading within Europe, including some southern areas (Crete, Cyprus and Spain).

4.4. Within Europe, it has been widely reported throughout Scandinavia, the Baltic countries and Poland. Within the EU, there have also been reports from Germany (from 7 Länder), and occasional outbreaks have been reported in Austria, Belgium, France, Greece (Crete), the Netherlands and Spain. The disease is reasonably well under control in seed potato production in Denmark, Finland and Sweden, but still occurs in ware production. There have been no findings in seed potatoes in Greece and France for several years and German outbreaks in seed and ware have been decreasing since 2000. The first ever UK field occurrence of ring rot was detected in seed potatoes in Wales in 2003. Italy, Ireland, Luxembourg and Portugal are currently considered free from the pathogen.

4.5. Of the two main seed potato production areas in the EU, the disease has never been found in Scotland and has been found only in a small number of cases in the Netherlands.

4.6. The true scale of the problem in China and Eastern Europe is largely unknown. Belarus, The Czech Republic, Estonia, Latvia, Lithuania, Poland The Russian Federation and The Ukraine all have reports. Romania, Slovakia, Slovenia and Hungary are currently considered free from the pathogen.

4.7. The disease is widespread in some countries joining the EU. For example, 17.7%, 6% and 5.7% of seed crops for certification in Poland failed because of ring rot in the years 1999 – 2001. Moreover, the certification scheme supplies a very low percentage of seed potatoes planted there. In the Czech Republic, 197 farms were quarantined because of ring rot in 2001, a large proportion of which had also been affected in previous seasons. The Commission is monitoring progress on
compliance with ring rot and other phytosanitary measures by accession countries to ensure that the necessary measures are fully implemented.

4.8. In the last 10 years there has been an increase in the number of cases of ring rot intercepted on entry into EU Member States. In GB, these have originated from Cyprus, France, Germany, the Ukraine and the USA. Those from Cyprus and Germany were in ware potatoes in 2001 and from the USA in illegally imported ware potatoes in 2002.

4.9. As a result of increased interceptions in ware potatoes originating in Germany, statutory measures (SI 2001 No. 3194) were introduced in 2001 to ensure that all arrivals in the UK of potatoes from Germany are notified in advance to the Plant Health Service to allow more vigilant ring rot monitoring.

5. Biology, survival and dissemination of the pathogen
5.1. Infected seed potatoes are the main source of Cms.
5.2. The pathogen can latently infect and colonise seed potatoes without expression of symptoms and in this way can survive undetected in high populations over several generations of seed multiplication.
5.3. Some potatoes cultivars are less pre-disposed to symptom development following infection than others and their response also varies with environmental conditions.
5.4. The bacterium spreads readily from diseased tissue by direct contact between tubers during handling or indirectly through contamination of surfaces or packing materials in stores, on planting and harvesting machinery, conveyors and graders.
5.5. The bacterium has been shown to survive for up to 2 years in tuber debris smeared on many surfaces and materials, particularly at low humidity and low temperature.
5.6. The risk of spread of infection during handling is increased if the tubers or sprouts are damaged, thus facilitating vascular infections through wounds. Hence, within an infected crop, the pathogen is rapidly spread by cutting seed tubers prior to planting.
5.7. The bacterium can survive for up to one year in some soils under cool, dry conditions. However, most studies indicate that plant-to-plant dissemination in the field plays little or no role in ring rot epidemiology. There is some indication that spread of the pathogen through waterlogged soils can lead to secondary disease development in the field.
5.8. The only known natural host of Cms in Europe is potato.
5.9. Reports from the USA of infection and seed transmission in sugar beet have not been reproducible under European conditions or with commonly grown varieties.
5.10. Infected volunteer potatoes (groundkeepers) can maintain infections over several generations. Spread is most likely to occur through contact with infected volunteers during harvesting and handling of a subsequent crop.
5.11. The bacterium is relatively short-lived in water, surviving up to 35-52 days in sterile tap or de-ionised water and for less time in non-sterile surface water. Spread of Cms through surface irrigation water is unlikely to be as important as for the potato brown rot bacterium (Ralstonia solanacearum) because of the lack of aquatic secondary host in which the pathogen can establish and multiply.
5.12. Transmission between ware potato lots has been demonstrated when healthy tubers were washed up to 48 hours after the same water was used to wash an infected lot.
5.13. There are some reports demonstrating insect transmission under experimental conditions by Colorado beetles (Leptinotarsa decemlineata), green peach aphids (Myzus persicae), grasshoppers (Melanopulus differenialis) and black blister beetles (Epicauta pennsylvanica). No data are available on the persistence of Cms
in insects and the relevance of insect transmission in ring rot epidemiology is unknown although expected to be of low risk.

6. **Assessment of Risk and Economic Loss**

6.1. Ring rot is highly contagious and persistent and there are few, if any, examples where it has been successfully eradicated after introduction.

6.2. The direct economic impact of ring rot may be only moderate, especially with modern production systems and statutory controls in place within the EU. However, ring rot would constitute a major constraint on seed production in areas where it does not occur, with considerable indirect effects on trade.

6.3. An independent economic evaluation of the Defra Plant health Programme in 2000 concluded a benefit:cost ratio of the current exclusion policy of 29.8:1 over a 30 year time horizon, with a net social benefit of £88.2 million. This was based on the estimated impact of ring rot in England on sales of seed potatoes from Scotland, Wales and N. Ireland. Public costs of the current exclusion policy were estimated at £222,000 per year and the potential costs on establishment of the disease at £10.68 million per year.

6.4. Direct yield losses of over 50% have been estimated from field trials in Norway and the USA. In commercial practice, such heavy direct losses are not usually observed. Nevertheless, due to the zero tolerance policy for this disease, a single infected tuber can lead to destruction of entire affected and associated crops, with additional indirect losses resulting from disposal and disinfection costs and negative effects on domestic trade and export, particularly for seed potatoes.

6.5. High losses arising from rejection during seed certification have been recorded in the USA and Canada since the 1940’s, reaching up to 60% of the total rejected seed area in the USA in 1978. High direct losses are often related to crops in which seed was cut before planting, a practice, which effectively spreads the pathogen and increases infection rates through the crop.

6.6. The actual cost of ring rot can be estimated from specific insurance payments. The Dutch potato insurance company Potatopol paid out an estimated €0.46 million between May 2002 and May 2003 for only 6 claims for losses related to ring rot outbreaks.

7. **Statutory ring rot control**

7.1. There are no effective methods currently available for chemical or biological control of ring rot. Nor are there any currently available potato cultivars with immunity or useful levels of resistance to the disease.

7.2. Complete avoidance of the pathogen is the best policy for control. This involves ensuring that seed potatoes have been certified under zero tolerance schemes.

7.3. Since ring rot often occurs at low levels and in a latent form, phytosanitary measures aimed only at visual inspections or testing of samples from potato consignments are not sufficient to guarantee freedom from ring rot. Measures have to be aimed at whole production systems including all multiplication generations and their places or areas of production during the production of healthy seed stocks.

7.4. Current EC legislation (Directive 2000/29/EC) which is implemented by the Plant Health (Great Britain) (Amendment) Order 1995 effectively prohibits the import of seed potatoes from outside the EU, other than from Switzerland, and restricts the movement of ware potatoes into the EU from third countries where ring rot is known to occur.
7.5. Directive 93/85/EEC deals more specifically with demarcation and containment of ring rot wherever it has been found within the EU and requires additional measures to be taken towards its eradication. Annual random surveys of domestic and imported seed and ware potatoes, and testing with standardised methods, are required in support of the requirements of the Directive.

7.6. Actions taken under the Directive following a finding of ring rot include; (a) holding of movement and prohibition of planting of infected and associated (“probably-contaminated”) potatoes, which are potatoes which have tested free but remain subject to restrictions (b) trace-back of the origin of infected potatoes, (c) investigation and demarcation of the limits of potential spread of the bacterium, and (d) holding and investigation of all clonally-related potato stocks elsewhere.

7.7. Cropping restrictions on affected places of production during the 3-4 years following an outbreak include the prohibition of planting potatoes on infected fields and elimination of all volunteer potatoes for a minimum two year period. On lifting of the restrictions, only classified seed potatoes can be planted for the first crop, for ware production only, and the produce must be tested after harvest. Restrictions also apply to fields other than the outbreak fields on the affected and associated places of production.

7.8. Controlled disposal of infected and probably contaminated potatoes is required. Approved methods for infected material include incineration, feeding to animals after boiling, deep burial in a licensed landfill site or industrial processing at a site with approved waste disposal and disinfection facilities. For probably contaminated potatoes, the additional options of packing and sale for consumption and animal feeding under controlled conditions are available. Contaminated equipment, premises, machinery, packaging etc must either be destroyed or cleansed and disinfected.

7.9. For detecting latent infections in potato tuber samples, laboratory testing is required. Official screening methods involve an immunofluorescence test whereby $Cms$ cells are stained with a specific antibody bound to a fluorescent marker and observed microscopically. A second screening test provides rapid confirmation of a positive IF result and involves a polymerase chain reaction (PCR) test for sensitive detection of DNA sequences in $Cms$. Final confirmation of a positive test requires isolation and identification of the pathogen, including injection of a sample extract or suspect isolate of the bacterium into eggplant seedlings and the development of typical symptoms.

7.10. The standard sample size for tuber testing is 200 tubers per 25 t lot of potatoes. This gives a theoretical probability of 95% of detecting $Cms$ in a lot with an infection level of 1.5%. To obtain 95% confidence of detection for infections levels of 1.0, 0.5, 0.1 and 0.05%, sample sizes of 300, 600, 3000 and 4600 tubers are required respectively.

8. Best practice for ring rot avoidance

8.1. Use only classified seed potatoes produced under a system with zero tolerance for ring rot. All classified seed potatoes produced in the EC must have been derived from material found free from this disease.

8.2. Spread of ring rot is most common via clonal multiplication links. Check seed traceability, both up (previous generations) and sideways (sister stocks).

8.3. Avoid cutting seed potatoes prior to planting. Cutting is an ideal means of spreading ring rot within and between potato stocks.
8.4. Be aware of the symptoms of ring rot and check thoroughly for typical foliar symptoms or stunting (particularly late in the season) and harvested tubers for signs of the disease. Contact your local Defra PHSI or SEERAD inspector immediately should you suspect the disease.

8.5. Laboratory testing of seed tuber samples for latent infections is an essential component of regulatory control. However, it is important to remember that potatoes cannot be guaranteed free from infection by the ring rot bacterium on the basis of visual inspections or testing of samples.

8.6. Intensive and repeated surveillance and testing over a number of years can increase confidence in the ring rot-free status of a particular area of production. This is heavily dependent on the combined vigilance of both the whole potato industry and plant health authorities.

8.7. Potato groundkeepers are a key factor in the long-term survival of the disease. Their control removes an important source of disease inoculum.

8.8. Regularly clean and disinfect all machinery, equipment, containers, vehicles and storage facilities used during potato production.

8.9. The thorough washing off of all soil and debris should precede disinfection of machinery and equipment. This is because organic material and soil can significantly reduce the effectiveness of disinfectants. Steam cleaning is particularly effective but will not be appropriate for all situations.

8.10. There are numerous disinfectants suitable for use in the varying circumstances under which ring rot could be found and hot water can also be an effective treatment for certain items. A minimum temperature of 82 °C maintained for 5 minutes is required for complete inactivation of the bacterium. Try to maintain at least 10 minutes contact between disinfectant and surface during treatment. The PHSI/SEERAD will provide further disinfection advice to growers on a case-by-case basis.

8.11. Observance of general on-farm hygiene measures in respect of cleaning and disinfection of boots, leggings, knives etc. is important at all times.

8.12. Avoid sharing machinery or ensure adequate cleaning and disinfection between places of production or between different stocks of potatoes (particularly if moving between ware and seed stocks).

8.13. Similarly, the use of the same equipment and machinery to grade/process ware and seed potatoes poses a very high risk of cross-infection, particularly when the potatoes originate from different growers.

8.14. Cross infection from stock to stock can occur, most commonly, via superficial damage to tubers through direct physical contact with infected tubers or through contact with contaminated machinery or wash water. Ensure careful handling and grading to minimise damage. Handle seed tubers before sprouts are too long and more prone to damage.

8.15. The movement of machinery through a crop with infection in the haulm could also potentially spread infection, via ooze or infected sap entering wounded stems.

8.16. Discarded potatoes and potato processing waste could harbor the pathogen. Dispose of all potato waste in accordance with the Defra Code of Practice for the Management of Agricultural and Horticultural Waste.

8.17. There is clear evidence that contaminated wash water from infected ware tuber lots can transmit the pathogen to subsequent lots washed in the same water during packing. Disinfect and change the water between lots from different origins.
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