Erwinia chrysanthemi
(*Dickeya* spp.)

The Facts

Compiled for the British Potato Council by;
Dr John Elphinstone, Central Science Laboratory
Dr Ian Toth, Scottish Crop Research Institute

Contents

Executive Summary

The pathogen

Erwinia chrysanthemi (Echr) is a complex of different bacteria now reclassified as species of *Dickeya*. While *D. dadantii* and *D. zeae* (formerly Echr biovar 3 or 8) are pathogens of potato in warmer countries, *D. dianthicola* (formerly Echr biovar 1 and 7) appears to be spreading on potatoes in Europe. The revised nomenclature of these pathogens has distinguished them from other soft rot erwiniae (including *P. atrosepticum* and *P. carotovorum).*

Symptoms

Symptoms of soft rot disease on potato tubers are similar whether caused by *Dickeya* or *Pectobacterium* spp. In the field, disease develops following movement of either pathogen from the stem base. Whereas *P. atrosepticum* typically causes blackleg symptoms under cool wet conditions, symptoms due to *Dickeya* spp. have been more commonly observed to occur under warm conditions (when temperatures exceed 25 ºC). The foliar symptoms most commonly associated with *D. dianthicola* in warm dry growing conditions include brown staining of the vascular tissues and occasionally necrosis and hollowing of the stem, which usually remains green until leaf desiccation is complete. Symptoms due to *Dickeya* spp. are also thought to occur later in the season. However, there is some dispute as to whether disease symptoms and timing alone can differentiate the two pathogens. Symptoms caused by *D. dianthicola* under warm dry conditions can be confused with those of other wilting diseases.

Geographic distribution

Dickeya spp. were first reported on potato in Europe in the Netherlands in the 1970s and has since been reported on potato in a number of other European countries. To date all European potato isolates appear to be *D. dianthicola*, although other *Dickeya* spp. have been found on potato in other countries including Australia and Peru. The pathogen has been reported worldwide on many hosts as *Erwinia chrysanthemi*, but the corresponding *Dickeya* spp. has yet to be determined in most cases. Over 40 outbreaks of *D. dianthicola* in England have been officially confirmed by laboratory testing at CSL since 1990, and is probably more widespread in potato crops in England and Wales than official records suggest. Most cases appear to be from imported seed but at least one was from UK-produced seed. To date there have been no findings of *Dickeya* spp. on potatoes in Scotland.

Biology, survival and dissemination of the pathogen

Factors influencing disease development on potato caused by *Dickeya* spp. are generally the same as for *P. atrosepticum*, with the exception of temperature, where a warmer spring and summer favours disease development by *Dickeya* spp. A lower level of inoculum, irrigation from contaminated water courses and more rapid movement through the vascular system of the plant may also favour disease caused by *Dickeya* spp. over *P. atrosepticum.* The most important means of dissemination for potato is movement of latently infected seed tubers. In other host plants, of which there is a wide range, spread over long distances and especially across borders, is mainly via infected vegetative material. *Dickeya* spp. have been identified in water courses in several countries and in one case in Sweden on the riparian weed *Solanum dulcamara*. Occurrence in GB watercourses or *S. dulcamara* is unknown. The high host diversity across the *Dickeya* species suggests that wild host plants could play an important role in survival. In plant-free soil, survival is less than 6 months and, therefore, over-wintering is unlikely.

Assessment of risk and economic loss

Unpublished UK studies found that *D. dianthicola* was highly contagious and aggressive with low tuber inoculum levels leading to high wilting incidence in field plots. However, losses due to potential infections by *Dickeya* spp. other than *D. dianthicola* are not expected to be significant under UK growing conditions, although seed infections may result in significant disease levels if seed is exported to warm climates or our own climate warms.

Control and diagnostics
Dickeya, like *P. atrosepticum,* is regarded as a seed-borne pathogen and is controlled largely through seed classification in line with domestic and EU legislation. In the UK, as in other European countries, the seed potato classification schemes set tolerances for diseases encountered during visual inspections of growing crops and harvested tubers. There is no official post-harvest testing programme, although voluntary testing services provide useful decision support. On-farm control measures for *Dickeya* spp. are currently the same as for *P. atrosepticum*, largely because there is insufficient data available to support alternative *Dickeya*-specific measures. However, where information is available, it suggests that the use of diagnostics, avoiding over-irrigation and controlling secondary hosts may be a way to avoid disease caused by *Dickeya* spp. In other European countries, as in the UK, there are no *Dickeya* spp.-specific control measures and no compulsory testing in operation. Some countries attempt to differentiate *Dickeya* spp. and *P. atrosepticum* based on visual inspection, while others also use diagnostics but on a voluntary basis. There are media-, antibody- and PCR-based diagnostics available for *Dickeya* spp. and in some cases for the soft rot erwiniae as a group. All three method types are used throughout Europe although PCR-based methods appear to be the most reliable. Most countries do not differentiate between these pathogens but consider disease as caused by "soft rot erwiniae" and use control measures accordingly. A major opportunity we have to reduce the risk of introducing *Dickeya* spp. into the UK is by growers joining the new "Safe Haven" Scheme.

Threats
D. dianthicola now appears to be as important on potato as *P. atrosepticum* in several Northern European countries, and experts consider it to be increasing in importance. It is very likely to pose a threat to UK potato production and has already been detected in an English seed crop. The range of wilting symptoms due to *D. dianthicola* estimated in English crops has varied from <1% to 20-30%. Currently, the protected Scottish seed potato regions appear to be clear of this pathogen but for how long remains to be seen. Effective control measures implemented now are our best, and possible only, chance of preventing economic losses caused by this pathogen as it gains a foothold in the UK.

Opportunities
Demonstrating that seed-growing areas in the UK are free from *Dickeya* would provide a competitive advantage for GB seed exports. There may also be added advantages to GB ware potato growers, particularly in warm growing seasons, in having access to *Dickeya*-free seed supplies (e.g. through safe-havens accredited seed stocks).

Recommendations
Follow the best practice guidelines and join the Safe-Haven scheme to safeguard against *Dickeya dianthicola* infections; Confirm species responsible for disease in Europe; assess importance of *Dickeya* spp. compared to *P. atrosepticum* in terms of risk to GB seed and ware; Conduct survey to assess frequency of pathogen introduction to UK and confirm absence in protected seed producing areas; Identify optimal conditions for disease development and survival and potential niches for that survival; assess varietal resistance susceptibility to *Dickeya* spp.

1. Introduction

Erwinia chrysanthemi (Echr) contains a complex of different bacteria, each with distinct pathogenicity on different hosts, which have been recently reclassified as species of the genus *Dickeya* (Samson *et al*., 2005).

A number of *Dickeya* species (spp.) can cause disease on potato, with symptoms ranging from typical blackleg and tuber soft rot to a vascular wilt (known as slow wilt). The most important of these are strains of *D. dadantii* and *D. zeae* (formerly Echr biovar 3), which are pathogens of potato in warm climates, and the more temperate-adapted strains of *D. dianthicola* (formerly Echr biovar 1 and 7), which appear to be spreading on potato in Europe.

D. dianthicola (formerly *Erwinia chrysanthemi* pv. *dianthicola*) is a listed quarantine organism (EPPO A-2) only on *Dianthus* (Council Directive 2000/29/EC) but can also cause disease on potato, tomato, chicory and artichoke, as well as on ornamental plants such as *Dahlia*, Freesia, Hyacinth, Iris, *Kalanchoe* and *Zantedeschia*. Formerly widespread in Europe on *Dianthus*, it has been successfully controlled on this host through strict glasshouse hygiene and certification of planting material.

On potato, control of *D. dianthicola* through seed certification is currently based on visual inspection for blackleg or slow wilt symptoms in the field and soft rot in store.

The first European report of *D. dianthicola* on potato was from the Netherlands in the 1970's. The first case on potatoes in England was reported in 1990. It has since been regularly detected on UK-grown potatoes grown from seed originating in the Netherlands. In a recent DEFRA study (D. E. Stead, unpublished), a selection of *Dickeya* strains previously isolated from potato in England, France, Hungary, Jersey, Netherlands, and Switzerland were all identified as *D. dianthicola* (either Echr biovar 1 or 7). In 2001, it was detected for the first time on seed potatoes (cv. Maris Piper) produced in the UK. The pathogen has never been reported in Scotland.

Erwinia chrysanthemi (Echr) is a complex of different bacteria now reclassified as species of *Dickeya*. While *D. dadantii* and *D. zeae* (formerly Echr biovar 3 or 8) are pathogens of potato in warmer countries, *D. dianthicola* (formerly Echr biovar 1 and 7) appears to be spreading on potatoes in Europe.

2. The pathogen

2.1. Members of the genus *Dickeya* are motile, Gram-negative, non-sporing, straight rods with rounded ends, which occur singly or in pairs. The bacteria vary in size from 0.8-3.2 x 0.5-0.8 µm (average 1.8 x 0.6 µm). There may be 3-14, but more usually 8-11, peritrichous flagellae.

2.2. The complex taxonomy of the *Dickeya* pathogens has necessitated a complete revision of the nomenclature (Samson *et al*., 2005). The new classification now clearly distinguishes them from the other potato soft rot erwiniae, *Erwinia carotovora* subsp. *atroseptica* (now renamed *Pectobacterium atrosepticum*) and *Erwinia carotovora* subsp. *carotovora* (renamed *Pectobacterium carotovorum* subsp. *carotovorum*). Table 1 shows the relationship between new and old pathogen names.

2.3. Although *Dickeya* spp. are mostly considered warm climate pathogens, *D. dianthicola* is able to cause disease on potato in Northern Europe and was first reported on potato in Europe in the Netherlands in the 1970's (Maas-Geesteranus, 1972).

2.4. *D. dianthicola* is a vascular pathogen of potato which invades the xylem. *D. dadantii* and *D. zeae* have been reported (as Echr biovars 3 and 8) to cause blackleg symptoms typical of those caused by *P. atrosepticum*.

The revised nomenclature of these pathogens has distinguished them from other soft rot erwiniae (including *P. atrosepticum* and *P. carotovorum).*

Table 1: Transfer of *Pectobacterium* (*Erwinia*) *chrysanthemi to Dickeya* spp. according to Samson *et al*. (2005).

New name	Old names	Hosts
Dickeya dianthicola	*Erwinia chrysanthemi* biovars 1, 7 and 9 *Erwinia chrysanthemi pv dianthicola* *Pectobacterium chrysanthemi pv dianthicola*	*Dianthus spp.*, potato, tomato, chicory, artichoke, *Dahlia & Kalanchoe.*
Dickeya dadantii	*Erwinia chrysanthemi* biovar 3 (some strains) *Pectobacterium chrysanthemi* biovar 3 (some strains)	*Pelargonium*, pineapple, potato, *Dianthus* spp., *Euphorbia*, *sweet potato*, banana, maize, *Philodendron & Saintpaulia*.
Dickeya zeae	*Erwinia chrysanthemi* biovar 8 and other strains of biovar 3 *Pectobacterium chrysanthemi* biovar 8 and other strains of biovar 3	Maize, potato, pineapple, banana, tobacco, rice, *Brachiaria*, *& Chrysanthemum*
Dickeya chrysanthemi bv. *chrysanthemi*	*Erwinia chrysanthemi* biovar 5 *Erwinia chrysanthemi* pv. *chrysanthemi* *Pectobacterium chrysanthemi pv. chrysanthemi*	*Chrysanthemum spp.*, chicory, tomato & sunflower
Dickeya chrysanthemi bv. *parthenii*	*Erwinia chrysanthemi* biovar 6 *Erwinia chrysanthemi* pv. *parthenii* *Pectobacterium* chrysanthemi pv. *Parthenii*	*Parthenium*, artichoke & *Philodendron*.
Dickeya paradisiaca	*Erwinia chrysanthemi* biovar 4 *Erwinia chrysanthemi* pv. *paradisiaca* *Erwinia paradisiaca* *Brenneria paradisiacal*	Banana & maize
Dickeya dieffenbachiae	*Erwinia chrysanthemi* biovar 2 *Erwinia chrysanthemi* pv. *dieffenbachiae* *Pectobacterium chrysanthemi pv. dieffenbachiae*	*Dieffenbachia*, tomato & banana

3. Symptoms

3.1. Distinction from symptoms caused by Pectobacterium atrosepticum and P. carotovorum.
Symptoms of soft rot on potato tubers, as described by Powelson and Franc (2001), are similar whether caused by *Dickeya* or *Pectobacterium* spp. Tuber soft rot ranges from a slight vascular discolouration to complete decay. Lesions commonly first develop in lenticels, at the site of stolon attachment or in wounds. Affected tuber tissue is cream- to tan-coloured and is soft and granular. Brown to black pigments often develop at the margins of decayed tissue (Fig. 1). Circumstantial evidence from growers in the UK suggests that *Dickeya* spp. may be less of a problem than *P. atrosepticum* on the stored crop (Crowhurst 2006).

The foliar symptoms most commonly associated with *D. dianthicola* occur in warm dry growing conditions, as described by Lumb *et al*. (1986) in Israel. The first symptom is a wilt of the top leaves with subsequent desiccation around the margins and eventually of the entire leaves (Fig. 2). These symptoms eventually spread to the lower leaves and, in extreme cases, the whole plant or stem dries out. Often only one stem per plant is affected. Symptom development is usually associated with soft rotting of the mother tuber but the soft rot symptoms do not extend up the stolon or stem, either externally or internally, as observed with blackleg caused by *P. atrosepticum* (Fig. 3). Vascular tissues stain brown from the stem base, progressing upwards and occasionally resulting in necrosis and hollowing of the stem (Fig. 4). Externally, the stems usually remain green until leaf desiccation is complete. Under Israeli conditions, symptoms usually first appear when the air temperature exceeds 25 ºC. In contrast, the first occurrence of blackleg symptoms caused by *P. atrosepticum* is usually earlier in the season when air temperatures are below 25 ºC. Some cultivars (e.g. Pentland Crown and Maris Bard) were seen to express leaf desiccation symptoms more readily than others (e.g. Desiree and Spunta). While some growers in the UK who have seen the disease over time are able to spot subtle differences in appearance and timing of foliar symptoms (Crowhurst 2006), researchers in the Netherlands suggest that such predictions can be unreliable as similar symptoms may also be seen with *P. atrosepticum.*

Other *Dickeya* spp. (*D. dadantii* and *D. zeae*), generally found on potato in warmer, humid tropical and sub-tropical environments, are able to cause symptoms which are indistinguishable from those of blackleg disease caused by *P. atrosepticum* in cooler environments (DeLindo and French, 1981; Cother, 1980). Plants affected by these organisms show wilting, stunting and chlorosis and a brown to black soft rot of the stem base extends upwards from the rotting mother tuber, with eventual total collapse of the plant. When disease occurs before or just after emergence, missing hills (blanking) are observed in the crop. Observations made in the Netherlands suggest that the same symptoms may be caused by *D. dianthicola* under warm, wet growing conditions (J M van der Wolf and E. de Haan, personal communication).

3.2. Distinction from other diseases
Whilst typical blackleg and soft rot are easily distinguishable from other diseases on the basis of visual inspection, symptoms caused by *D. dianthicola* under warm dry conditions may be confused with those of other wilting diseases. In Israel, symptoms caused by *Dickeya* spp. were indistinguishable from those of wilt caused by *Verticillium dahliae* or those due to natural plant senescence (Lumb et al. 1986). These symptoms could also be confused with those of other bacterial diseases of potato including brown rot (caused by *Ralstonia solanacearum*) and ring rot (caused by *Clavibacter michiganensis* subsp. *sepedonicus*).

Fig. 1 Disease symptoms in Israel (which may differ from those in the UK) showing soft rot of daughter tubers (photographs courtesy of L. Tsror, Gilat Research Centre, Israel)

Fig. 2 Disease symptoms in Israel (which may differ from those in the UK) showing initial wilt in upper leaves followed by wilt and desiccation in the lower leaves (photographs courtesy of L. Tsror, Gilat Research Centre, Israel)

Fig. 3 *D. dianthicola*: rotting mother tuber©

Fig. 4 *D. dianthicola*: internal stem symptoms©

Symptoms of soft rot disease on potato tubers are similar whether caused by *Dickeya* or *Pectobacterium* spp. In the field, disease develops following movement of either pathogen from the stem base. Whereas *P. atrosepticum* typically causes blackleg symptoms under cool wet conditions, symptoms due to *Dickeya* spp. have been more commonly observed to occur under warm conditions (when temperatures exceed 25 ºC). The foliar symptoms most commonly associated with *D. dianthicola* in warm dry growing conditions include brown staining of the vascular tissues and occasionally necrosis and hollowing of the stem, which usually remains green until leaf desiccation is complete. Symptoms due to *Dickeya* spp. are also thought to occur later in the season. However, there is some dispute as to whether disease symptoms and timing alone can differentiate the two pathogens. Symptoms caused by *D. dianthicola* under warm dry conditions can be confused with those of other wilting diseases.

4. Geographic Distribution

4.1 Presence in GB

Over 40 findings of *D. dianthicola* in seed and ware crops grown in at least 15 English counties have been officially confirmed by laboratory testing at CSL since 1990 (Table 2). The pathogen is probably more widely distributed in potato crops in England and Wales, and in other countries, than the official records suggest. The English findings are based on official seed inspections and random surveys or, since 2001, on voluntary submission of samples by growers (for which details of variety and origin are not always supplied). There have been no findings of *Dickeya* spp. on potatoes in Scotland. All outbreaks in England were initially found in crops grown from seed imported from the Netherlands. However, in 2001 it was first confirmed that the pathogen had infected a seed crop of Maris Piper grown from UK-produced seed, indicating that the pathogen had spread and caused primary infection under English conditions and can thus pose a risk to English seed production. A recent article published in Potato Review magazine suggests that growers in the UK are aware of the arrival of *Dickeya* spp. into the UK and are concerned about ways to combat subsequent disease. While they suggest that the import of Dutch seed is the most likely source of the pathogen, there are growing concerns that global warming may lead to more local problems in the future (Crowhurst 2006).

Table 2: Affected varieties in officially confirmed outbreaks of *Dickeya dianthicola* (*Erwinia chrysanthemi* pv. *dianthicola*) on potatoes in England.

Variety	No. positive samples detected
Sante	7
Morfona	5
Estima	3
Markies	3
Ostara	3
Accord	2
Saturna	2
Ausonia	1
Fambo	1
Lady Rosetta	1
Maris Piper	1
Nadine	1
Rembrandt	1
Xantia	1
Unknown	14
Total:	**46**

4.2 Europe

Dickeya dianthicola has been known to occur for some time in several European countries on *Dianthus spp.* (Fig. 5). Bradbury (1986) recorded the presence of this pathogen in Denmark, England, France, Germany, Italy, the Netherlands, Norway, Poland, Romania, Sweden and Greece. *Erwinia chrysanthemi* was first found to be infecting potatoes in the Netherlands in the 1970's (Maas-Geesteranus, 1972) and has since been reported on potato in a number of other European countries including England, France, Hungary, Jersey and Switzerland. In a recent DEFRA study (D. E. Stead, unpublished), a selection of strains previously isolated from potato in these countries were all identified as *D. dianthicola*. *Erwinia chrysanthemi* has also recently been reported on potato in Finland (Laurila *et al*., 2006) but the *Dickeya* sp. was not identified. *E. chrysanthemi* (probably *D. dianthicola*) was reported to be the most frequent bacterial pathogen on seed *potato* in western Switzerland followed by *P. atrosepticum* (Cazelles and Schwarzel, 1992).

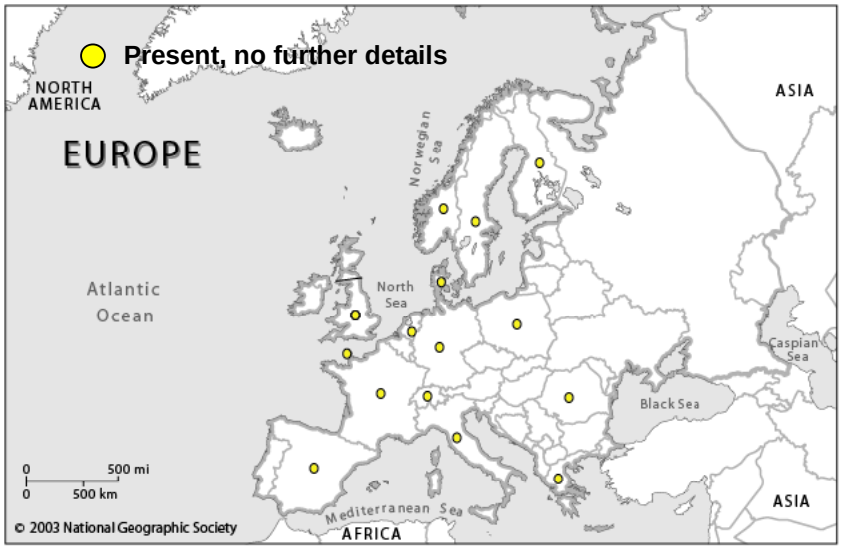

Fig. 5: Distribution of *Dickeya dianthicola* on all hosts in Europe (updated from CAB International, 2005).

4.3 Overseas

There are reports worldwide of *Erwinia chrysanthemi* on many hosts (including potato) but in most cases the corresponding *Dickeya* spp. have yet to be identified. Outside Europe, *Dickeya dianthicola* has so far been reported on ornamental hosts only in Colombia, Japan, New Zealand and the USA (New York, Pennsylvania and Texas) (Bradbury 1986). To date, no potato isolates from outside of Europe have been confirmed as *D. dianthicola*. Further testing is required to confirm whether *Erwinia chrysanthemi* reported on crops grown from European seed in Israel, North Afriica, Cuba and elsewhere can also be classified as *D. dianthicola*.

Not all *Erwinia chrysanthemi* reported on potato around the world corresponds to *D. dianthicola*. For example, *E. chrysanthemi* biovar 3 (*D. dadantii* or *D.* zeae) has been reported to cause premature rotting of developing tubers at temperatures above 25 °C in Australia (Cother, 1980; Cother *et al*., 1992). *Erwinia chrysanthemi* biovar 3 was also shown to induce typical blackleg symptoms on potato in Peru (de Lindo et al., 1978).

Dickeya spp. were first reported on potato in Europe in the Netherlands in the 1970s and has since been reported on potato in a number of other European countries. To date all European potato isolates appear to be *D. dianthicola*, although other *Dickeya* spp. have been found on potato in other countries including Australia and Peru. The pathogen has been reported worldwide on many hosts as *Erwinia chrysanthemi*, but the corresponding *Dickeya* spp. has yet to be determined in most cases. Over 40 outbreaks of *D. dianthicola* in England have been officially confirmed by laboratory testing at CSL since 1990, and is probably more widespread in potato crops in England and Wales than official records suggest. Most cases appear to be from imported seed but at least one was from UK-produced seed. To date there have been no findings of *Dickeya* spp. on potatoes in Scotland.

5. Biology, survival and dissemination of the pathogen

5.1. Factors influencing disease development

Factors influencing disease development on potato caused by *Dickeya* species are, on the whole, similar to those for *P. atrosepticum* and these are outlined in the BPC grower's guide "Managing the risk of blackleg and soft rots". Such factors include varietal susceptibility, damage and lack of cleanliness at grading, poor soil drainage, presence and level of the pathogen on seed, use of sprouted seed, over-irrigation, wet spring weather, damage at harvest, and lack of adequate ventilation at storage. However, information on important differences between *Dickeya* spp.- and *P. atrosepticum*-induced disease that may allow different targeted control measures are largely unknown. Factors that do or may affect disease development differently between the two pathogens are outlined below:

- **Temperature** is perhaps the most important factor in determining whether disease in any one season will be predominantly caused by *Dickeya spp.* or *P. atrosepticum*, and there is both published and circumstantial evidence to support this. Lumb et al. (1986) found that in Israel symptoms caused by *Dickeya* spp. tended to develop when temperatures exceeded 25ºC. Perombelon et al. (1986) showed that temperature plays a vital role in determining which pathogen predominates in causing disease symptoms during and between growing seasons. Similarly, investigations in the Netherlands suggest a correlation between *Dickeya* spp.- or *P. atrosepticum*-related diseases in the field depending on whether there has been a hot or cooler spring / summer, respectively.
- The **level of pathogen inoculum** may be an important factor in disease development but there are few data to support this possibility. However, unpublished data has indicated that lower levels of *Dickeya* spp. are more likely to lead to disease than *P. atrosepticum*.

- **Over-irrigation** is important in disease development for both pathogens but current evidence suggests that *Dickeya* spp. may be more readily isolated from water courses than *P. atrosepticum* (see 5.2 below). This could potentially lead to spread as well as multiplication of *Dickeya* spp. on tubers in the field.
- Rate of **movement through vascular system** of the plant by Dickeya spp. may be higher than for *P. atrosepticum* and, as a result, lead to more rapid disease development. However, there is only circumstantial evidence for this. Recent unpublished evidence has shown that there is no obvious difference in the rate of mother tuber breakdown between the two pathogens.
- **Susceptibility of varieties** to the erwiniae is a major factor in disease development. However, there is currently no data on potential differences in varietal susceptibility between the two pathogens. Varietal resistance / susceptibility ratings for *P. atrosepticum* are often used to assess potential resistance to *Dickeya* species, although the reliability of such an approach is so far undetermined.

5.2. Dissemination

In Solanum tuberosum

The most important means of dissemination for potato is the movement of latently infected seed tubers. The pathogen can be carried on the tuber surface and in lenticels (as for *Pectobacterium* spp.) but is most likely found in the tuber vascular system which it enters systematically via the stolon from the infected mother plant.

In other host plants

Over long distances, and especially across national borders, the pathogen is spread mainly by infected vegetative propagating material. In addition to potato, *D. dianthicola* can also infect the crop plants tomato, chicory and artichoke. The pathogen can remain latent in ornamental stock plants and can thus be spread in cuttings from them. Ornamental hosts of *D. dianthicola* include *Dianthus*, *Dahlia* and *Kalanchoe*, although the strain which affects *Kalanchoë* (formerly *E. chrysanthemi* biovar 9) has not been recorded on potato. In the Netherlands, an increased incidence of *Dickeya* spp. causing soft rot of flower bulbs (*Dahlia*, *Freesia, Iris*, *Muscari*, and particularly *Hyacinth*) was recently reported (van Doorn *et al*., 2006), although it was not specified whether or not the causal agent was *D. dianthicola*.

Other *Dickeya* spp. have most often been reported in warm climates or under glasshouse conditions. It remains unclear whether these pathogens have spread from ornamental plants and glasshouse crops to potato under European conditions. Reports of the isolation from potato of *D. chrysanthemi* bv*. chrysanthemi* (formerly *E. chrysanthemi* biovar 5) in the Netherlands and Spain (Janse and Ruissen, 1988; Palacio-Bielsa *et al*., 2006) and *D. chrysanthemi* bv*. parthenii* (*E. chrysanthemi* biovar 6) in Spain (Palacio-Bielsa *et al*., 2006) require further substantiation, particularly in light of the fact that the biovar identification method was found to lack reproducibility during studies at CSL, and more reliable molecular identification methods are now available. Natural hosts of *D. chrysanthemi* biovar *chrysanthemi* are known to include *Chrysanthemum*, chicory, tomato and sunflower, whereas those of *D. chrysanthemi* biovar *parthenii* include *Parthenium*, artichoke and *Philodendron*.

Infection of potato by strains of *D. dadantii* and *D. zeae* (formerly *E. chrysanthemi* biovar 3 and 8) has been observed in warmer areas of the world such as Peru (De Lindo *et al*., 1978) and Australia (Cother *et al*., 1992). If substantiated, the finding in Spain of one biovar 3 potato isolate amongst 13 isolates tested (Palacio-Bielsa *et al*., 2006) may be the first record of one of these species causing disease on potato in Europe. These *Dickeya* spp. have a wide host range including *Aloe vera*, banana, *Brachiaria*, *Chrysanthemum*, *Dianthus*, *Euphorbia*, maize, *Pelargonium*, *Philodendron*, pineaple, potato, *Saintpaulia*, sweet potato, tobacco and rice.

Other *Dickeya* spp. have not been reported as naturally infecting potato. Natural hosts of *D. paradisiaca* (formerly *E. chrysanthemi* biovar 4) include banana and maize, and the natural host range of *D. dieffenbachia* (formerly *E. chrysanthemi* biovar 2) includes *Dieffenbachia*, banana and tomato.

Further identification of *Dickeya* spp. is required among isolates previously reported as *Erwinia chrysanthemi* from a number of other natural hosts worldwide (Bradbury, 1986), including: *Allium fistulosum*, *Brassica chinensis*, *Capsicum*, cardamoms, carrot, celery, *Colocasia esculenta*, Poaceae (such as *Panicum maximum* and *Pennisetum purpureum*), *Hyacinthus* sp., *Leucanthemum maximum*, lucerne, onion, radish, *Sedum spectabile*, sugarcane, sorghum, tulip and glasshouse ornamentals such as *Aechmea fasciata*, *Aglaonema pictum*, *Anemone* spp., *Begonia intermedia* cv. Bertinii, *Cyclamen* sp., *Dracaena marginata*, *Opuntia* sp., *Phalaenopsis* sp., *Polyscias filicifolia*, *Rhynchostylis gigantea* and *Syngonium podophyllum*.

In water courses

Olsson (1985) reported the isolation in 1976 of *Erwinia chrysanthemi* from *Solanum dulcamara* growing in a watercourse used for irrigation in Sweden. In 1983 the pathogen was isolated directly from water samples obtained from the same source. The bacteria were shown to infect potatoes and to be transmitted to subsequent generations. *Dickeya* spp. have also been isolated from irrigation water sources in the Netherlands (Van Vuurde and De Vries, 1992), although the frequency and distribution of infested watercourses is unknown. A similar observation has recently been made in Finland (Laurila *et al*., 2006) where 27% of soft rotting bacteria isolated from blackleg potato stems, 7% from rotting tubers and 100% from river water samples were found to be *Dickeya* spp. Further investigation is required to determine whether the *Dickeya* spp. found in watercourses is the same as that consistently isolated from potato in Northern Europe (*D. dianthicola*).

In NSW, Australia, different *Dickeya* spp. were isolated from the headwaters of the Murrumbidgee River and the source of the Murray River only 140 km away (Cother *et al*., 1992). *Dickeya chrysanthemi* biovar *chrysanthemi* (*E. chrysanthemi* biovar 5) was found consistently in the upper reaches of the Murrumbidgee River but was not detected in the Murray River where the majority of strains isolated were characterized biochemically as biovar 3. In both cases, biochemical, fatty acid and DNA characterization methods showed isolates from potatoes were identical to those from the river water with which they were irrigated, supporting the hypothesis that the *Dickeya* spp. were natural components of the aquatic microflora and spread to potatoes via irrigation water. In Florida, high populations of *Dickeya* spp. able to infect *Dieffenbachia* have also been detected in irrigation ponds containing recycled water (Norman *et al*., 2003).

5.3. Survival

Survival studies reported in the scientific literature often do not specify the *Dickeya* spp. involved and the results are thus difficult to extrapolate to UK or European conditions. It appears unlikely that the pathogen can over-winter freely in soils. Studies in Italy showed that *Dickeya* isolates from *Dianthus* could not survive in plant-free soil for more than 6 months (Garibaldi, 1972). Recent studies in the Netherlands (J.M. van der Wolf, personal communication), suggested that *Dickeya* isolates from potato or hyacinth could not survive for more than 7 days when added to different soils at 6 ºC and 50% field moisture capacity (compared with 60 days or more for *P. carotovorum* isolates). Nevertheless, potato crops multiplied once in the field from pathogen-free mini-tubers were observed to have 20-56% infection by *Dickeya* spp. in the harvested tubers. Confirmation of such high primary infection rates suggests that the pathogen can (a) survive in the potato-growing environment (e.g. on plant debris or on alternative hosts, either other crops or weed spp.), and/or (b) is transmitted from outside of the cropping environment (e.g. via irrigation water, aerosols or insects).

Apart from the short report of infection of *Solanum dulcamara* in Sweden (Olsson, 1985), there is very little information on the potential for survival of *D. dianthicola* and other *Dickeya* spp. in weed hosts. However, given the high host diversity across and sometimes within the *Dickeya* spp., it is highly likely that wild host plants could play a role in survival.

Factors influencing disease development on potato caused by *Dickeya* spp. are generally the same as for *P. atrosepticum*, with the exception of temperature, where a warmer spring and summer favours disease development by *Dickeya* spp. A lower level of inoculum, irrigation from contaminated water courses and more rapid movement through the vascular system of the plant may also favour disease caused by *Dickeya* spp. over *P. atrosepticum.* The most important means of dissemination for potato is movement of latently infected seed tubers. In other host plants, of which there is a wide range, spread over long distances and especially across borders, is mainly via infected vegetative material. *Dickeya* spp. have been identified in water courses in several countries and in one case in Sweden on the riparian weed *Solanum dulcamara*. Occurrence in GB watercourses or *S. dulcamara* is unknown. The high host diversity across the *Dickeya* species suggests that wild host plants could play an important role in survival. In plant-free soil, survival is less than 6 months and, therefore, over-wintering is unlikely.

6. Assessment of Risk and Economic Loss

6.1. Quarantine status

Dickeya dianthicola and *D. chrysanthemi* bv. *chrysanthemi* (as *E. chrysanthemi* pvs. *dianthicola* and *chrysanthemi*) are listed as A2 quarantine pests of *Dianthus* and *Chrysanthemum* spp. by EPPO (OEPP/EPPO, 1982 and 1988). However, *Dickeya* spp. are already distributed in the EPPO region and *Dianthus* and *Chrysanthemum* can both be infected by *Dickeya* spp. other than *D. dianthicola* or *D. chrysanthemi* bv. *chrysanthemi*. It is therefore proposed that the pathogens will be deleted from the EPPO A2 list since the risk can be adequately covered by national nuclear-stock certification schemes for the crops concerned. As a phytosanitary measure, EPPO recommends (OEPP/EPPO, 1990) that plants for planting of carnations or chrysanthemums should come from mother plants free from the bacteria originating from nuclear stock certification schemes.

6.2. Potential GB economic impact

In initial studies at CSL (D.E. Stead, unpublished), *D. dianthicola* was found to be highly contagious and aggressive when tubers were inoculated by injection into the stolon end with low inoculum levels, leading to high wilting incidence in field plots. Of 20 cultivars tested over 3 years, all were found to be susceptible to infection by *D. dianthicola* (biovar 7). The data on cultivar behaviour varied between seasons but some general trends were apparent. Maris Piper was the least susceptible cultivar to wilting, whereas, cvs. Morene, Sante, Premiere, Rubina, Obelix, Romano, Record, Wilja, Estima, Rocket and Cara all showed high levels of susceptibility. With respect to tuber soft rot, cvs. Morene and Rocket were least susceptible although they were highly susceptible to wilting. Official inspections by the DEFRA Plant Health and Seeds Inspectorate have identified commercial ware and seed crops in England with a range of wilting symptoms from <1% to 20-30%. Losses due to potential infections by *Dickeya* spp. other than *D. dianthicola* are not expected to be significant under UK growing conditions, although seed infections may result in significant disease levels if seed is exported to warm climates or our own climate warms.

6.3. Economic impact to overseas markets

The organism first described as *Erwinia chrysanthemi* specifically occurring on potato crops grown in Israel from seed imported from the Netherlands (Lumb *et al*., 1986) is assumed to be *D. dianthicola*. However, it has yet to be confirmed whether this is the only *Dickeya* sp. causing disease on potato in Israel. Tsror *et al*. (2006) described recent increases in disease incidence in Israel arising from suspected infected seed imported from the Netherlands. In spring 2005, a severe outbreak of the disease was observed, in more than 200 ha (different locations), affecting various cultivars (including Mondial, Desirée, Lady Crystal, Sapphire and Quincy). Disease incidence ranged from 5 to 30% (8.2% in average). In addition to foliar wilting symptoms, rotted progeny tubers were also observed in the field. The highest wilt incidence (30%) was observed in the cv. Sapphire. When visually healthy potatoes from this crop were replanted, a further wilt incidence of 10-15% was observed in the following season. When healthy seed potatoes (cv. Desiree and Vivaldi) were planted in two locations following the original diseased crop, no transmission of the disease to the healthy crop was observed and the pathogen was not detected on the progeny tubers. It was therefore concluded that the pathogen is not significantly soil-borne. In spring 2006, the disease was again observed in more than 260 ha (Sharon and Negev regions) in various cultivars (Mondial, Desirée, Rodeo, Quincy, Nicola), with disease incidence (surveys conducted by the Israeli Plant Protection Services) that ranged from 2 to 30% (10% in average). Seed tubers were sampled from commercial lots originating from Holland, France and Scotland, and tested for latent infection. Out of 36 Dutch lots, 24 were *E. chrysanthemi*-positive, whereas 6 Scottish lots and 1 French lot were *E. chrysanthemi*-negative.

Unpublished UK studies found that *D. dianthicola* was highly contagious and aggressive with low tuber inoculum levels leading to high wilting incidence in field plots. However, losses due to potential infections by *Dickeya* spp. other than *D. dianthicola* are not expected to be significant under UK growing conditions, although seed infections may result in significant disease levels if seed is exported to warm climates or our own climate warms.

7. Control

7.1. Statutory (Certification)

Since *Pectobacterium atrosepticum* and *Dickeya* spp. are both regarded as seed-borne pathogens, their control is largely brought about through seed classification in line with domestic and EU legislation. Most Scottish seed potatoes are derived initially from pathogen-tested microplants held in SASA's nuclear stock unit. As a protected EU “high grade seed region”, Scotland produces only pre-basic and basic seed over a limited number of field generations (usually 3-5). In England and Wales, approved stocks of eligible varieties produced in other EU Member States can enter the classification scheme.

In the UK, as in other European countries, the seed potato classification schemes set tolerances for the levels of soft rot and blackleg diseases encountered during two visual inspections of growing crops and a single visual inspection of harvested tubers after grading and dressing and prior to marketing. For pre-basic seed there is a nil tolerance for blackleg and soft rot diseases caused by these pathogens in both field and tuber inspections. Field inspection tolerances for the incidence of blackleg are set at 0.25% (1 in 400 plants) for super elite (SE) grade, 0.5% for elite grade, 1.0% for A grade and 2.0% for CC grade. Similar tolerances for equivalent grades are set in the classification schemes for England and Wales as in the Scottish scheme. Rogueing of blackleg plants is not allowed prior to the first inspection of pre-basic seed stocks but is allowed between the first and second inspections. For the other grades rogueing of diseased plants is allowed prior to both inspections but a certificate/approval may be refused if

rogueing appears to have been excessive. In tuber inspections, the maximum tolerance for soft rots caused by these bacteria is 1% in England and Wales and 0.5% in Scotland.

Records of the levels of blackleg and soft rot encountered at inspection provide some guidance on general seed health across stocks and generations but do not accurately reflect the status of latent infections, the pathogen species present or the general risk of blackleg or soft rot disease development in subsequent generations (which is highly dependent on environmental conditions). There are no official post-harvest testing programmes for *Dickeya* or *Pectobacterium* spp. although voluntary testing services offered in some countries (including the UK and the Netherlands) can provide useful decision support for growers and store managers.

7.2. On-farm

On-farm control measures for *Dickeya* species are the same as for *P. atrosepticum*, largely because there is insufficient data available to support alternative *Dickeya*-specific measures. Currently, therefore, control measures effective against *P. atrosepticum* are considered to be effective against *Dickeya spp*. These include, varietal resistance, ensuring a clean grading line, avoiding poorly drained fields, avoiding short rotations, using diagnostics to test seed stocks for the presence of the pathogen, avoiding de-sprouting at planting, avoiding over-irrigation, harvesting crops early, minimising damage at harvest and adequate ventilation during cold storage. These are all outlined in more detail in the BPC grower's guide "Managing the risk of blackleg and soft rot". However, where information is available, it suggests that both the use of diagnostics and avoiding over-irrigation could be especially useful as a method of reducing disease caused by *Dickeya* spp. BPC have been supporting the validation of molecular diagnostics which differentiate between the different soft rot bacteria (formerly erwinias) and these can effectively be used to identify the presence of *Dickeya* spp. Such diagnostics can be used to avoid planting of *Dickeya*-infected stocks or to help predict likely problems in different climatic conditions at home (e.g. between north and south UK) or when exporting. It is thus prudent to use diagnostics for identification of the causal agent where possible rather than simply reporting the presence and level of "soft rot erwinias". Evidence is growing that *Dickeya* spp. are present in waterways of some countries and that the use of these as a source of irrigation could spread the pathogen to potato fields. This does not appear to be the case with *P. atrosepticum*. However, there is some evidence that alternative hosts may also be important in the spread of disease caused by both pathogens and monitoring / avoiding these hosts could help to reduce the incidence of disease (Toth et al. 2006). There is further laboratory evidence that *Dickeya* spp are more susceptible to cool temperatures (4-10 ºC) than *P. atrosepticum*, suggesting that cold storage may offer a simple means of reducing *Dickeya* numbers on stored seed. However, this needs further substantiation. This is supported in circumstantial evidence from Israel, where extending cold storage of tubers that initially showed signs of disease in the field showed reduced disease when later planted. A major opportunity we have to reduce the risk of introducing *Dickeya* spp. into the UK is by joining the new "Safe Haven" Scheme, which was originally introduced to protect UK growers from ring rot (*Clavibacter michiganensis* subsp. *sepedonicus*).

7.3. Specific approaches and control measures in other countries.

Plant protection agencies from at least ten other European countries, including Germany, Denmark, Spain, Slovenia, the Netherlands, Poland, Sweden, Norway, Israel and Finland are aware of either the presence of *Dickeya* spp. in their potato production or the potential for its arrival. In all cases, there are no *Dickeya* spp.-specific control measures and no compulsory testing in operation. Some countries attempt to differentiate *Dickeya* spp. and *P. atrosepticum* based on visual inspection, while most also use diagnostics but intermittently and on a voluntary basis. However, most do not differentiate between these pathogens and consider disease problems as caused by "soft rot erwiniae" and use control measures accordingly. Diagnostic methods employed include CVP with colony PCR (Spain), ELISA (Denmark, the Netherlands and Sweden), immunofluorescence test (Slovenia), ELISA and PCR (Poland), IFAS and PCR (Germany) and non-quantitative PCR (Finland) (see section on diagnostics). Following the

recent discovery of *Dickeya* spp. on potato seed stocks in Israel, thought to have arrived on imported seed, this country has undertaken a thorough investigation of the most appropriate methods of testing and these are reported in detail below.

Israel has introduced a number of detection, identification and differentiation methods to combat current and potential future disease issues caused by *Dickeya* spp. These methods include; isolation on CVP; ELISA; PCR; biochemical testing; Koch Postulates; and erythromycin sensitivity (see section on diagnostics). Two PCR-based methods were tested (Toth et al. 2001 and Nasar et al 1996). Toth et al. (2001) primers (G1/L1) are used to detect the soft rot erwiniae as a group and then to identify them as *P. carotovorum*, *P. atrosepticum* or *Dickeya* spp., while ADE1/2 are used as specific primers for detection of *Dickeya* spp. only. Based on the use of both methods ADE1/2 primers were, not surprisingly, found to be more sensitive than G1/L1 when used directly for detection of *Dickeya* spp.

An ELISA-based method was used to test a number of *Dickeya* spp. reference strains including those from Israel, the Netherlands and the Scottish-based company Adgen, as well as *P. atrosepticum* SCRI1043 and *P. carotovorum* SCRI193 (SCRI reference strains). Following investigation, it was concluded that PCR using ADE1/2 was more sensitive than ELISA and primers G1/L1 PCR for the detection of *Dickeya* spp. A protocol to detect *Dickeya* spp. in seed tubers has been developed in Israel and is expected to be published in 2007.

When laboratory tests were compared with the incidence of disease in the field, 26 lots (50%) from a total of 52 showed a correlation between zero presence of *Dickeya* spp. in the lab and no disease in the field, while 16 (30%) lots showed a correlation between the presence of *Dickeya* spp. in the lab and the presence of disease in the field. 10 lots (20%) showed results that did not correlate between the lab and the field, although in 1 case a low level of disease not unexpectedly gave a negative result in the lab. In 7 of these 10 lots, *Dickeya* spp. were detected in the lab but no disease ensued, suggesting that tubers may carry *Dickeya* spp. without it resulting in visible disease symptoms. Thus, in general, a clear relationship exists between positive diagnostic tests and the presence of disease. It is expected that zero tolerance for *Dickeya* spp. will be proposed in Israel in the near future, and PCR is likely to be the method of choice for diagnostic testing.

7.4. Best practice guide

See BPC guide “Managing the risk of blackleg and soft rot”.

Dickeya, like *P. atrosepticum,* is regarded as a seed-borne pathogen and is controlled largely through seed classification in line with domestic and EU legislation. In the UK, as in other European countries, the seed potato classification schemes set tolerances for diseases encountered during visual inspections of growing crops and harvested tubers. There is no official post-harvest testing programme, although voluntary testing services provide useful decision support. On-farm control measures for *Dickeya* spp. are currently the same as for *P. atrosepticum*, largely because there is insufficient data available to support alternative *Dickeya*-specific measures. However, where information is available, it suggests that the use of diagnostics, avoiding over-irrigation and controlling secondary hosts may be a way to avoid disease caused by *Dickeya* spp. In other European countries, as in the UK, there are no *Dickeya* spp.-specific control measures and no compulsory testing in operation. Some countries attempt to differentiate *Dickeya* spp. and *P. atrosepticum* based on visual inspection, while others also use diagnostics but on a voluntary basis. Most countries do not differentiate between these pathogens but consider disease as caused by “soft rot erwiniae” and use control measures accordingly. A major opportunity we have to reduce the risk of introducing *Dickeya* spp. into the UK is by growers joining the new “Safe Haven” Scheme.

8. Diagnostic methods

- Soft rots, blackleg and other wilt diseases can be caused by various *Dickeya* and *Pectobacterium* spp. The identity of the causal bacterium must therefore be checked. Latent infections can be detected in cuttings or tubers.

- *D. dianthicola* has been observed to form two distinct colony types (mucoid and spreading types) when isolated from the same diseased tissue (D.E. Stead, unpublished). One colony type produces little or no pectic enzymes on pectate media. It is therefore advised to use both pectate-based and non-pectate media (potato dextrose agar or 5% sucrose nutrient agar) for isolation.

- *D. dianthicola* does not consistently grow on selective CVP medium (Cuppels and Kelman, 1974) which has traditionally been used for isolation of the other soft rot bacteria (*P. atrosepticum* and *P. carotovorum* subsp. *carotovorum*) from potato stems and tubers. It is thus possible that *D. dianthicola* has escaped detection in the past.

- Tolerances to temperatures and erythromycin have been proposed for direct differential isolation of different soft rot pathogens on potato (Perombelon and Hyman, 1986). However, the ability of *D. dianthicola* to grow at lower temperatures prevent it from being consistently differentiated from the other blackleg and soft rot causing *Pectobacterium* spp. (Janse and Spit, 1989).

- A differential medium based on the characteristic production of blue-pigmented indigoidine by *Dickeya* spp. has recently been shown to differentiate *Dickeya* from other soft rot *Pectobacterium* spp. (Lee and Yu, 2006) but has not yet been tested in the UK.

- On Potato Dextrose Agar (PDA), young colonies of *D. dianthicola* are either circular, convex, smooth and entire, or sculptured with irregular margins, depending on the moisture content of the growth medium. After 4-5 days, colonies resemble a fried egg, with a pinkish, round, raised centre and lobed periphery, which later becomes feathery or almost coralloid (Lelliott and Stead, 1987).

- Inoculated artificially into aubergines, *D. dianthicola* from potatoes can cause symptoms resembling those caused by the ring rot bacterium *Clavibacter michiganensis* subsp. *sepedonicus* (Persson and Janse, 1988).

- Antisera and ELISA kits are commercially available to detect *Dickeya* spp.. The antibodies are generally directed against O-serogroup 1, recognizing only 68% of the strains (Samson *et al*., 1990). Commercial ELISA tests are available (Sanofi Diagnostics Pasteur, 1998) which can give false positive results and also have limitations regarding the sensitivity of detection.

- A monoclonal antibody (6A6) to a fimbrial antigen detected all *D. dianthicola* isolates tested and some other *Dickeya* spp. in a triple antibody sandwich (TAS) ELISA (Singh *et al*., 2000). However, sensitivity was limited to 10^7 cfu/ml, compared to a sensitivity of 10^3 cfu/ml for a PCR test using published primers directed to the pectate lyase gene (Nassar et al. 1996).

- An enrichment ELISA procedure is used in the Netherlands for routine detection of *Dickeya* spp. in voluntary testing offered commercially by NAK. Advantages include cost:efficiency and NAK report a correlation of 95% between this method and PCR testing (Nassar et al. 1996) (van den Bovenkamp personal communication).

- A new immunoassay based on Luminex xMAP® technology has been proposed as an alternative to ELISA (van der Wolf *et al*., 2006) for simultaneous detection of *P. atrosepticum* and *D. dianthicola*. Pre-enrichment in semi-sective polypectate broth is still required to achieve the necessary detection sensitivity.

- PCR assays are available for specific detection and identification of *Dickeya* spp. The most widely-used for detection of all *Dickeya* spp. are the ADE primers (ADE1/ADE2) from the pectate lyase (*pel*) gene (Nassar et al., 1996). However, PCR methods developed by Toth et al. (1999, 2001) allow for the detection of the "soft rot erwiniae" as a single group, together with differentiation of the individual pathogens. Other conventional PCR assays are also available (Smid *et al*., 1995; van der Wolf *et al*., 1995). Automated real-time PCR assays have been developed and are currently under validation at CSL in the UK (J.G. Elphinstone, unpublished) and PRI in the Netherlands (J.M. van der Wolf, unpublished).

- The various *Dickeya* spp. can be routinely identified according to either their fatty acid methyl ester (FAME) profiles or repetitive (REP) PCR product polymorphisms using enterobacterial repetitive intergenic consensus (ERIC) primers (D E Stead unpublished). *D. dianthicola* isolates (including biovars 1, 7 and 9) form a unique profile with either method. Isolates from potato in the Netherlands, originally identified as biovar 5, also grouped within the typical *D. dianthicola* profiles.

- Ribotyping has also been used successfully to type strains within species of *Dickeya* (Nassar et al. 1994).

There are media-, antibody- and PCR-based diagnostics available for *Dickeya* spp. and in some cases for the soft rot erwiniae as a group. All three method types are used throughout Europe although PCR-based methods appear to be the most reliable.

9. Knowledge gaps

A number of questions remain:

9.1. Which Dickeya spp. are currently spreading in Europe?

There is a need for **accurate identification**, using 16-23S rRNA gene sequencing (Samson *et al*., 2005) to identify *Dickeya* spp., amongst collections of isolates (such as those held at CSL and SCRI) which were previously identified as *Erwinia chrysanthemi* from potato and other hosts in the UK, around Europe and elsewhere worldwide. This will establish whether or not *Dickeya dianthicola* is the only or the main spp. of threat to European potato production.

9.2. What are the key biological differences between the various Dickeya spp. and the Pectobacterium spp.? Do Dickeya spp. present additional risks to the UK potato industry in relation to those already presented by P. atrosepticum and P. carotovorum subsp. carotovorum? What are the optimal temperatures for disease caused by Dickeya spp. on potato in the UK?

Comparative data is needed on **environmental effects** (particularly of temperature, inoculum level and aggressiveness) on potential potato disease incidence and severity caused by *D. dianthicola* and any other relevant *Dickeya* spp. in comparison with *P. atrosepticum* and *P. carotovorum* subsp. *carotovorum*. This will allow more accurate assessment of the risks associated with introduction and establishment of *Dickeya* spp. and in particular, the potential for *D. dianthicola* to infect and cause disease under prevailing or future environmental conditions found in Scotland and elsewhere in the UK. How much would our climate need to change before disease caused by *Dickeya* spp. (possibly already present in the environment) became a

problem? Does cold storage affect survival of these strains and, if so, would storage offer a means of control?

9.3. Are there specific control measures relevant to D. dianthicola and any other Dickeya spp.?
Differences in biology encountered between the various *Dickeya* and *Pectobacterium* spp. may justify the introduction of additional specific control measures or classification tolerances.

9.4. What risk is posed by Dickeya spp. found in watercourses?
Dickeya sp. have been **detected in watercourses** in Northern Europe. The predominant spp. need to be identified. Their presence or potential for entry and survival in UK watercourses requires investigation. The risks to seed stocks from populations in water should be determined and, if justified, control methods validated.

9.5. What is the potential host range of D. dianthicola and any other Dickeya spp. under risk of introduction and what are the major target hosts?
Of particular concern is whether or not there are **alternative crop or weed spp.** that can harbour the pathogens. What is the relevance of the findings of *D. dianthicola* on flower bulbs in the Netherlands?

9.6. Would post harvest testing provide additional security against the introduction of Dickeya spp. and should they be differentiated from other soft rot erwiniae?
For example, would **testing of all stocks** prior to introduction to seed classification schemes be justified? Is visual inspection alone adequate? Are current diagnostic methods adequate for detection of all soft rot and blackleg pathogens.

9.7 Are there differences in the resistance / susceptibility of potato cultivars to P. atrosepticum and Dickeya spp.?
Do differences in **resistance and susceptibility of cultivars** to *P. atrosepticum* necessarily mean the same for *Dickeya* spp.? Does the cultivar *S. phureja*, currently used in the breeding program at SCRI, offer the means to develop cultivars that are resistant to *Dickeya* spp. as well as *P. atrosepticum?*

10. Threats, opportunities and recommendations

10.1. Threat to the GB potato industry
In the light of the recently improved understanding of the taxonomy of the *Dickeya* spp., a number of fundamental questions have been raised regarding (a) the biology of the pathogen (*D. dianthicola*) which appears to be spreading in Europe and (b) the risks it poses to the UK potato industry (see 9. above). The level of threat posed by *D. dianthicola*, in addition to that already presented by *Pectobacterium* spp., will depend upon:

- The relative aggressiveness of *D. dianthicola* on potato under UK conditions.
- Its current distribution within the UK, especially within seed potato production.
- The mode and frequency of it's introduction to the UK.
- Its ability to establish in the environment (e.g. on alternative hosts).
- The mode and likelihood of spread within and between crops (including the importance of spread through irrigation water).

D. dianthicola now appears to be as important on potato as *P. atrosepticum* in several Northern European countries (including the Netherlands and Switzerland), and experts in at least 10 European countries consider it to be increasing in importance. It is therefore very likely that this pathogen also poses an imminent threat to UK potato production. It is known, since 1990, to be

entering the UK on seed produced in the Netherlands and has also already been detected in an English seed crop. The incidence of wilting due to *D. dianthicola* estimated in English crops has varied from <1% to 20-30%. Whether the same threat extends to the protected Scottish seed potato region will remain unclear until the ability of the pathogen to survive and cause disease under Scottish conditions is established.

Anecdotal evidence from the Netherlands suggests that blackleg due to *P. atrosepticum* is more commonly observed in cool wet seasons, whereas, *D. dianthicola* is more frequently observed in warm wet seasons. It is therefore likely that the introduction of *D. dianthicola* would extend the range of environmental conditions over which blackleg could occur. Furthermore, it is possible that *Dickeya*, rather than *Pectobacterium*, could increase in importance in response to global warming.

Experiments in the Netherlands and Cuba (Van der Wolf, personal communication) have shown that the same seed stock infected with *D. dianthicola* gave rise to significantly more disease when planted in Cuba rather than in the Netherlands. These results indicate that the optimum temperature for *D. dianthicola* is probably higher than those routinely experienced during potato production in Northern Europe. However, they also highlight the risks involved in exporting seed potato with *Dickeya* infections to warmer climates. Unlike *Pectobacterium*, the damage to our reputation for seed exports would probably contribute more to the overall expected economic losses resulting from *Dickeya* infections than to direct crop losses at home.

10.2. Opportunities to the GB potato industry

Demonstration that seed-growing areas in the UK are free from *Dickeya* would provide a competitive advantage for GB seed exports. European seed importers are aware of the increase in disease caused by *Dickeya* in stocks originating in affected countries. Countries in continental Europe, including those in Eastern Europe (e.g. Hungary), may be experiencing particularly high losses due to the presence of *Dickeya* in seed imports combined with warmer growing seasons (J. Németh, personal communication). There may also be added advantages to GB ware potato growers, particularly in warm growing seasons, in having access to *Dickeya*-free seed supplies (e.g. through safe-havens accredited seed stocks).

10.3 Recommendations

a) Encourage growers to follow the best practice guidelines in 7.4 to safeguard against *Dickeya dianthicola* infections and join the safe-havens scheme. Implement suitable control measures as soon as possible.

b) Confirm that *Dickeya dianthicola* is the primary species of *Dickeya* causing disease of potato in Europe and determine whether other *Dickeya* spp. are also involved.

c) Conduct biological investigation to assess the relative importance of *Dickeya* and *Pectobacterium* spp. in terms of risk to GB seed and ware production under UK growing conditions.

d) Conduct surveys to accurately assess the frequency of introduction of *Dickeya* spp. on seed originating outside of GB and confirm its absence from protected seed production areas within the UK.

e) Identify optimal temperatures for disease development and potential niches (plant water and soil) for current / future survival.

f) Determine whether information on varietal susceptibility to *P. atrosepticum* is applicable to *Dickeya* spp.

11. References

1. Bradbury JF, 1986. Guide to plant pathogenic bacteria. Wallingford, UK: CAB International.
2. CAB International, 2005. Crop Protection Compendium. Wallingford, UK: CAB International.
3. Cazelles O, Schwarzel R, 1992. Survey of bacterial diseases caused by *Erwinia* in seed potato fields in western Switzerland. Revue Suisse d'Agriculture, 24:215-218.
4. Cother EJ, 1980. Bacterial seed tuber decay in irrigated sandy soils of New South Wales. Potato Research, 23:75-84.
5. Cother EJ, Bradley JK, Gillings MR, Fahy PC, 1992. Characterization of *Erwinia chrysanthemi* biovars in alpine water sources by biochemical properties, GLC fatty acid analyses and genomic DNA fingerprinting. Journal of Applied Bacteriology, 73: 99-107.
6. Crowhurst R, 2006. Warm summers could favour wilt disease. Potato Review, March 2006.
7. Cuppels D, Kelman A, 1974. Evaluation of selective media for isolation of soft-rot bacteria from soil and plant tissue Phytopathology, 64: 468-475.
8. DeLindo L, French ER, Kelman A, 1978. *Erwinia* spp. pathogenic to potatoes in Peru. American Potato Journal, 55: 383 (abstract).
9. DeLindo L, and French ER, 1981. *Erwinia* species attacking potato in the humid tropics of Peru. Fitopatologia, 16: 69-74.
10. Garibaldi A, 1972. Research on carnation bacterial wilt diseases. IV. Survival in soil of *Erwinia chrysanthemi*, agent of slow wilt. P. 29-32. In: Actas do III Congresso da Uniao Fitopatologica Mediterranea, Oerias, Portugal.
11. Janse JD, Ruissen MA, 1988. Characterization and classification of *Erwinia chrysanthemi* strains from several hosts in the Netherlands. Phytopathology, 78: 800-808.
12. Janse JD, Spit BE, 1989. A note on the limitations of identifying soft rot erwinias by temperature tolerances and sensitivity to erythromycin on a pectate medium. Journal of Phytopathology, 125: 265-268
13. Laurila J, Joutsjoki T, Ahola V, Hannukkala A, Pirhonnen M, 2006. Characterisation of erwinias causing blackleg and soft rot in Finland by sequencing and virulence tests. Abstracts from the 8th Conference of the European Foundation for Plant Pathology and the British Society for Plant Pathology Presidential Meeting 2006, 13th-17th August 2006. KVL, Frederiksberg, Denmark. p. 73.
14. Lelliott RA, Stead DE, 1987. Methods for the diagnosis of bacterial diseases of plants. Methods in Plant Pathology Volume 2 (Ed. T.F. Preece). Blackwell, UK. 216 pp.
15. Lee Y-A, Yu C-P, 2006. A differential medium for the isolation and rapid identification of a plant soft rot pathogen, *Erwinia chrysanthemi*. Journal of Microbiological Methods, 64: 200-206.
16. Lumb VM, Perombelon MCM, Zutra D, 1986. Studies of a wilt disease of the potato plant in Israel caused by *Erwinia chrysanthemi*. Plant Pathology, 35:196-202.
17. Maas Geesteranus HP, 1972. Natrot en zwartbenigheid bij aardappelen. Bedrijfsontwikkeling, 3; 941-945.
18. Nassar A, Darrasse A, Lemattre M, Kotoujansky A, Dervin C, Vedel R, Bertheau Y, 1996. Characterization of *Erwinia chrysanthemi* by pectinolytic isozyme polymorphism and restriction fragment length polymorphism analysis of PCR-amplified fragments of pel genes. Applied and Environmental Microbiology, 62: 2228-2235.

19. Nassar A, Bertheau Y, Dervin C, Narcy JP, Lemattre M, 1994. Ribotyping of *Erwinia chrysanthemi* strains in relation to their pathogenic and geographic distribution. Applied and Environmental Microbiology, 60: 3781-3789.

20. Norman DJ, Yuen JMF, Resendiz R, Boswell J, 2003. Characterization of *Erwinia* populations from nursery retention ponds and lakes infecting ornamental plants in Florida. Plant Disease. American Phytopathological Society (APS Press), St. Paul, USA: 87: 193-196.

21. OEPP/EPPO, 1982. Data sheets on quarantine organisms No. 53, *Erwinia chrysanthemi*. Bulletin OEPP/EPPO Bulletin, 12(1).

22. OEPP/EPPO, 1988. A1 and A2 lists of quarantine pests. Specific quarantine requirements. EPPO Publications Series B No. 92.

23. OEPP/EPPO, 1990. Specific quarantine requirements. EPPO Technical Documents, No. 1008. Paris, France: EPPO.

24. Olsson K, 1985. Detection of *Erwinia* spp. in some Swedish streams. In, Report of the International Conference on Potato Blackleg Disease, Royal Society of Edinburgh, 26-29 June 1984 (D.C. Graham and M.D. Harrison eds.) Potato Marketing Board, Oxford. pp. 45-46.

25. Palacio-Bielsa A, Cambra MA, Lopez MM, 2006. Characterisation of potato isolates of *Dickeya chrysanthemi* in Spain by a microtitre system for biovar determination, Annals of Applied Biology, 148: 157-164.

26. Perombelon MCM, Hyman LJ, 1986. A rapid method for identifying and quantifying soft rot erwinias directly from plant material based on their temperature tolerance and sensitivity to erythromycin. Journal of Applied Bacteriology, 60: 61-66.

27. Persson P, Janse JD, 1988. Ring rot-like symptoms in *Solanum melongena* caused by *Erwinia chrysanthemi* (potato strain) after antifungal inoculation. Bulletin OEPP, 18:575-578.

28. Powelson ML, Franc GD, 2001. Blackleg, aerial stem rot and tuber soft rot. In: Compendium of potato diseases second edition. W.R. Stevenson, R. Loria, G.D. Franc and D.P. Weingartner (eds.) APS Press, St Paul, Minnesota, USA. pp. 10-11.

29. Samson R, Legendre JB, Christen R, Fischer-Le Saux M, Achouak W, Gardan L, 2005. Transfer of *Pectobacterium chrysanthemi* (Burkholder et al. 1953) Brenner et al. 1973 and *Brenneria paradisiaca* to the genus *Dickeya* gen. nov as *Dickeya chrysanthemi* comb. nov and *Dickeya paradisiaca* comb. nov and delineation of four novel species, *Dickeya dadantii* sp nov., *Dickeya dianthicola* sp nov., *Dickeya dieffenbachiae* sp nov and *Dickeya zeae* sp nov. International Journal of Systematic and Evolutionary Microbiology 55: 1415-1427.

30. Samson R, Ngwira N, Rivera N, 1990. Biochemical and serological diversity of *Erwinia chrysanthemi*. Proceedings of the seventh International Conference on Plant Pathogenic Bacteria, Budapest, Hungary. Plant Pathogenic Bacteria, Z. Klement ed: 895-900.

31. Sanofi Diagnostics Pasteur, 1998. Phytodiagnostics. World Wide Web page at http://www.biomax.hr/Phyto/Elisa/Elisa_ph.html.

32. Singh U, Trevors CM, Boer SH, de Janse JD, 2000. Fimbrial-specific monoclonal antibody-based ELISA for European potato strains of *Erwinia chrysanthemi* and comparison to PCR. Plant Disease, 84: 443-448.

33. Smid EJ, Jansen AHJ, Gorris LGM, 1995. Detection of *Erwinia carotovora* subsp. *atroseptica* and *Erwinia chrysanthemi* in potato tubers using polymerase chain reaction. Plant Pathology, 44: 1058-1069.

34. Tanii A, Baba T, 1971. Bacterial plant diseases in Hokkaido. II. Bacterial stem rot of potato plant caused by *Erwinia chrysanthemi* Burkholder *et al*. (*Pectobacterium cartovorum* var. *chrysanthemi*). Bulletin of the Hokkaido Prefectural Agricultural Experiment Station No. 24: 1-10.

35. Toth IK, Avrova AO, and Hyman LJ, 2001. Rapid identification and differentiation of the soft rot erwinias using 16S-23S intergenic transcribed spacer (ITS)- PCR and RFLP analyses. Applied and Environmental Microbiology, 67: 4070-4076.

36. Toth IK, Hyman LJ, Moleleki L, Ravensdale M, Robert C, Liu H, Humphries S, Hedley P, Gilroy E, Pritchard L and Birch PRJ, 2006. What has genomics ever done for us? – A study of *Erwinia* and blackleg disease. Proceedings of Crop Protection in Northern Britain. Dundee. February 2006.

37. Toth IK, Hyman LJ, Wood JR, 1999. A one step PCR-based method for the detection of economically important soft rot *Erwinia* species on micropropagated potato plants. Journal of Applied Microbiology. 87: 158-166.

38. Tsror L, Erlich O, Lebiush S, Zig U, van de Haar JJ, 2006. Recent outbreak of *Erwinia chrysanthemi* in Israel – epidemiology and monitoring in seed tubers. Proceedings of the 11th International Conference on Plant Pathogenic Bacteria, 10th-14th July 2006, Edinburgh, Scotland. P70.

39. van der Wolf JM, Beckhoven JRCM, de Vries PM, de Raaijmakers JM, Bakker PAHM, Bertheau Y, van Vuurde JWL,. 1995. Polymerase chain reaction for verification of fluorescent colonies of *Erwinia chrysanthemi* and *Pseudomonas putida* WCS358 in immunofluorescence colony staining. Journal of Applied Bacteriology, 79: 569-577.

40. van der Wolf JM, Peters J, Sledz W, Bergervoet and van Doorn J, 2006. An enrichment-Luminex immunoassay for detection of *Erwinia carotovora* subsp. *atroseptica* and *E. chrysanthemi*. Proceedings of the 11th International Conference on Plant Pathogenic Bacteria, 10th-14th July 2006, Edinburgh, Scotland. p117.

41. van Doorn J, Hollinger TC, Vreeburg PJM, Speksnijder AGCL, Jafra S van der Wolf JM, 2006. Soft rot in flower bulbs, caused by pectinolytic *Erwinia* spp., as an ermerging disease. Proceedings of the 11th International Conference on Plant Pathogenic Bacteria, 10th-14th July 2006, Edinburgh, Scotland. J.G. Elphinstone, S. Weller, R. Thwaites, N. Parkinson, D.E. Stead and G Saddler (eds.) SASA, Edinburgh. Abstract CP-14, p.141.

42. van Vuurde JWL, de Vries PM 1992. Detectie van pathogene *Erwinia* spp. van aardappel in oppervlaktewater in de Periode 1988-1991. IPO-DLO report No. 92-01. 9 pp.

12. Glossary of terms

Aquatic microflora	Microorganisms naturally present in water.
Biovar	A strain that differs physiologically and/or biochemically from other strains in a particular species.
CVP	Crystal violet pectate – An agar-based media specifically designed for the selective growth of *Dickeya* and *Pectobacterium* spp.
Diagnostics	To check for the presence and identity of a pathogen.
***Dickeya* spp.**	Previously termed *Erwinia chrysanthemi,* this group of pathogens is now divided into 6 species, including *D. dianthicola*, *D. didantii* and *D. zeae*.
Dissemination	Spread of a pathogen.
ELISA	Enzyme-linked immunosorbant assay – an antibody-based method using colorimetric detection.
Erythromycin	Name of an antibiotic.
Erwinia chrysanthemi	Recently renames to *Dickeya* species (spp.).
Koch's postulates	Criteria proposed by a microbiologist called Koch for proving the pathogenicity of an organism. The suspected causal organism must be constantly associated with the disease; it must be isolated and grown in pure culture, and when inoculated into a healthy plant it must reproduce the original disease.
Latent	The presence of a pathogen in a host (plant) but not replicating or causing disease symptoms.
Luminex	Antibody-based diagnostic method.
Motile	The ability of an organism to move through self-propulsion.
Non-sporing	Does not form spores that are used to aid survival in some microorganisms.
PCR	Polymerase chain reaction – a method for amplifying specific pieces of DNA.
Peritrichous flagellae	The flagella (swimming organs) are distributed evenly around the cell surface.
Quarentine organism	Restricted entry into a country where pre or post entry testing is required.
Riparian	Inhabiting or situated on the banks of a river.
Solanum dulcamara	Commonly called Bittersweet it is a weed of many habitats including waterways.
Straight rods	Shape of bacterial cells.
Systemically	Spread throughout the host plant.
Varietal resistance	Resistance of a variety to disease usually on a scale from 1 (highly susceptible) to 9 (highly resistant).
Vascular pathogen	A pathogen that uses the plant's veins to transport itself throughout the plant.
Verticillium dahliae	Soil and seed-borne fungal pathogen causing vascular wilt disease of potato.
Xylem	Part of the vascular system that moves water and minerals around the plant.