Research Review

The use of plant resistance to manage potato pests and diseases

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1. The use of plant resistance to manage potato pests and diseases

1.1. Introduction

This survey of literature reviews the use of host resistance in the control of potato diseases and pests. All potato diseases and pests found in, and posing a threat to, the UK are reviewed. Information is drawn from world scientific literature and other information sources. The review is written for growers, agronomists and breeders and every attempt has been made to limit scientific jargon. For breeders, it is hoped that the review may influence selection priority for new varieties which will result in a wider availability of host plant resistance to many pests and diseases. Many breeders only concentrate on potato blight, potato cyst nematode (PCN), common scab and blackleg.

Host resistance is the most cost effective and environmentally benign way to control diseases and pests. For some diseases, resistance is either complete or non-existent. With other diseases, and for pests, resistance varies in its expression and the control of diseases or pests varies accordingly. In addition to resistance, there is also the phenomenon of tolerance, which is the ability to endure infection by disease or pest attack without showing severe effects (that is severe disease or pest symptoms or substantial reduction in yield or quality). At the extreme of tolerance, infected tubers may carry disease symptomlessly and this results in difficulties in control (e.g. spraing, brown rot and ring rot). At the other end of the tolerance spectrum, a tolerant variety may develop as much disease or succumb to similar pest damage as another variety but loss in yield or quality is much less. One example where tolerance occurs is in relation to potato cyst nematodes. Some varieties exhibiting vigorous growth may not be as affected by attack by cyst nematodes as a less vigorous variety and thus can be considered more tolerant.

The use of host resistance alone is considered in this review but it is also considered in conjunction with other control measures. These other control measures may be important in optimising the effect of host resistance but the reverse may also true - that other control measures can be enhanced where host resistance is improved.

Whilst host resistance is a cornerstone of integrated control the selection of varieties for production is frequently dictated by quality characters for specific market outlets and not by resistance to diseases or pests. Thus, for many growers, the option to select varieties for control of pests and diseases may not be possible. However, there are opportunities for utilising resistance in modern production such as targeting susceptible varieties for fields where risk of a disease or pest is the lowest.

1.2 Breeding for pest and disease resistance

Whilst every effort is made to breed varieties with resistance to pests and diseases, it is not possible to ensure resistance against all the main pest and disease problems. Diseases such as potato blight and pests such as PCN are high priorities for breeding but often it is physical, nutritional or cooking characteristics that take priority in a breeding programme.

When a breeding company wants to introduce a new variety into GB it has to first gain acceptance onto the European Catalogue of varieties. To achieve this it has to go through National List testing. This can be done in any country of the EU as there is a standardised procedure for testing. Most varieties bred in GB tend to go through National List testing in this country. National List testing is carried out by SASA in Edinburgh and besides the standard tests for distinctiveness, uniformity and stability (DUS) and variety conformity (VCU) there are also tests to establish resistance to various pests and diseases.

To complement the National List pest and disease resistance tests, the BPC pay for a further range of tests to be carried out in their IVT (Independent Variety Trials) project. This latter testing is carried out by SASA, SAC and SCRI in conjunction with BioSS who perform the statistical analyses. The range of tests carried out on varieties under both the NL and IVT testing is shown in Table 1.1 below. (Tests carried out in the IVT project are indicated by *)

TABLE 1.1 LIST OF RESISTANCE TESTS CARRIED OUT FOR NL AND IVT

Foliage late blight	**Tuber late blight**	**Blackleg**	**Field foliage late blight ***
Common scab	**Powdery scab**	**Internal damage**	**Skin spot ***
Dry rot - *Fusarium coeruleum*	**Dry Rot - *Fusarium sulphureum***	**External damage**	**Black scurf ***
***Globodera rostochiensis* Ro 1**	***Globodera pallida* Pa 1 & 2/3**	**Black dot ***	**Silver scurf ***

1.3 Determination of variety resistance ratings for potato pests and diseases

In the IVT testing programme, resistance of a variety to a range of diseases is evaluated in a series of standardised tests which each include a set of standard reference varieties whose reactions are known. For each disease or pest, the resistance rating of a candidate variety is determined by comparing the amount of disease or pest damage developing on the candidate variety with that on the reference varieties over at least two years of testing.

Disease or pest resistance ratings are recorded on a 1 to 9 scale where 1 is highly susceptible and 9 very resistant. Thus the higher the value, the more resistant a variety is to a disease or pest. Typically, varieties with a score of 1, 2 or 3 are considered highly susceptible. Those with a score 4 or 5 are susceptible. Those with a score 6 or 7 are moderately resistant and those with scores 8 or 9 highly resistant. However, in the case of PCN, full resistance or susceptibility, as measured by multiplication of cysts on roots, is indicated by R or S in brackets after the rating.

In carrying out tests to determine variety resistance rating, it is not possible to use reference varieties exhibiting the complete spectrum of resistance from highly susceptible to completely resistant, except for a few pests and diseases such as PCN Ro1. For most diseases and pests, all varieties can be affected to a greater or lesser extent; a high resistance score should not be taken as indicating that a disease or pest will be absent but that there is less risk of the disease or pest developing on these varieties. This applies, in particular, to late blight, *Rhizoctonia solani* (the cause of stem canker and black scurf), silver scurf and black dot. In consequence, the need for other control measures such as fungicide application should be evaluated, based on other factors such as the level of inoculum likely to be present and whether environmental conditions favour the pathogen.

It should be noted that pests and diseases are able to evolve and develop strains that can overcome the resistance of a variety. The standardised tests carried out to determine potato disease resistance ratings in the IVT programme use strains or isolates of pathogens that reflect the populations found in the UK. The tests cannot predict the development of resistance breaking strains and when using variety resistance as a control measure this fact should be borne in mind.

1.4 What do resistance ratings mean in practice?

The use of resistance ratings in practice depends on the pest or disease concerned. Of course, the higher the rating the less risk there is but even where a rating is high care still needs to be taken. A rating of 9 does not mean a variety is immune to the pest or pathogen, neither does a high resistance rating mean that a variety will not be infected by a specific disease or succumb to pest attack. A high resistance rating simply means that compared to reference varieties of known provenance it is likely to be less affected by a specific pest or disease.

It is apparent for several diseases, e.g. erwinia soft rot, tuber blight and gangrene, that the relative resistances of varieties depends on the method used to screen for resistance and which resistance mechanism(s) it assesses (Wellving, 1975; Bain *et al*., 1988). Swiezynski *et al*. (2001) reported that in a parallel screen of variety resistance to tuber blight involving 5 countries the relative resistances ascribed to individual varieties differed by up to 8 grades (on a 1 to 9 scale) between some countries.
Where there is no on-farm information on the susceptibility of varieties to a disease in practice then there will always be some doubt concerning the accuracy of official resistance scores. Frequently variety resistance screening tests are simplistic because they were devised by breeders who are obliged to screen very large numbers of potato genotypes and therefore require straight forward and rapid screening methods.

There are certain circumstances where even a resistant variety can be substantially attacked. Where:

- High disease or pest pressure occurs
- Ideal environmental conditions tip the balance in favour of a specific pest or disease
- The pest or pathogen has changed genetically (e.g. mutated) to overcome the resistance

Additionally, there are other key factors that need to be borne in mind when utilising host resistance. These are:

- The market place
- The achievement of quality
- The degree of effectiveness of host resistance

These circumstances and factors are discussed in section 3.

1.5 Summary of current potential and limitations in the use of plant resistance

Potential

1. To select varieties which generally offer low risk for a range of pest or disease problems
2. To use a variety or varieties to minimise specific pest or disease risks
3. To use host resistance to enhance the effectiveness of other control measures
4. To provide a degree of insurance that pest and disease attack will not be severe. In turn this forms the backbone for a risk analysis
5. Plant resistance being inherent in the variety grown is cost neutral (that is, disease resistance is part of the price)

Limitations

1. Varieties do not have a full spectrum of good resistances – most have weaknesses
2. Choice of variety is often outside the grower's control – the market normally dictates variety choice based largely on characteristics other than pest or disease resistance
3. Quality requirements sometimes mean even a little disease or pest damage is unacceptable and use of host resistance to manage pest or disease alone is limited
4. There is limited good resistance available for some pest and disease problems (e.g. powdery scab and slugs)
5. Under high disease/pest pressure or high favourable environmental conditions for the pest or disease, varieties with even high resistance may require additional control measures
6. Some diseases do not necessarily express symptoms (e.g. certain viruses such as potato mop top virus (PMTV) and tobacco rattle virus (TRV))
7. Plant resistance may mask the presence of certain undesirable pests or diseases (notably quarantine organisms such as brown and ring rot)
8. Resistance may not be durable

1.6 Practical implications of host resistance for the seed potato industry

Seed growers only grow varieties that are likely to have a future market. Thus the market place dictates which varieties they grow. However, unlike pre-pack or processing potato growers they have to minimise pests and diseases over a number of generations. Thus, pest and disease control must be sustained across the generations and must be contained (for the majority of pests and diseases) below certain thresholds required for certification, and perhaps additional standards higher than the certification ones for specific markets – including export.

As much as other sectors of the industry, seed growers must monitor disease and pest health through the multiplication cycle in order to prevent a build up to unacceptable levels. A high level of host resistance will provide re-assurance that specific pests and diseases will be contained

Seed producers, by the fact that they are not generally producing ware for consumption, and are not restricted by protocols of production often use more agrochemicals for storage diseases during the years of multiplication. These should be used judiciously where plant resistance is limited.

Seed producers have an advantage in that land must be certified free of potato cyst nematode and they are forced to operate a wide rotation. Thus for certain soil-borne pests and diseases the absence of a high level of resistance in a variety should be less of a problem. However, persistent soil-borne

diseases such as common scab, powdery scab and spraing (PMTV & TRV) require careful management. The use of resistant varieties in situations of risk is worthwhile.

1.7. Practical implications of host resistance for the pre-pack potato industry

Standards for tuber quality, including freedom from surface and internal disease symptoms and pest damage, are particularly high. Thus significant attention is needed to limit pests and diseases. This requires a holistic approach in which risks from pests and diseases are considered carefully. Pest and disease resistance plays a large part in the strategy of disease and pest control. However, because even relatively low levels of blemish can detract from quality, attention is needed to factors that affect utilisation of resistance such as inoculum pressure and favourable environmental conditions.

Pre-pack producers mostly operate under strict protocols. In these protocols there is a preference to avoid or minimise pesticide use. Thus the optimisation of plant resistance is important. This is particularly so for soil-borne diseases and storage diseases. For example, assessing levels of black dot soil contamination should permit either justification of the use of in-furrow Amistar application for susceptible varieties, or the targeting of resistant varieties to higher risk fields. Similarly, treatment of tubers with fungicides at harvest is discouraged in protocols and the risk of development of storage diseases may be higher unless environmental conditions are manipulated to reduce disease risk. Thus, on the rare occasions when into-store fungicide treatments are considered for pre-pack crops, control measures may be targeted at those varieties where the resistance is lowest for the risk diseases. An example, could be where through late lifting skin spot is a high risk in a very susceptible variety (e.g. King Edward).

For any pre-pack outlet, there is a limited range of varieties that will be grown and this restriction in choice can have an impact on the utilisation of plant resistance. However, although the choice is limited, there should be sufficient flexibility to utilise specific host resistance to minimise specific disease or pest risks, perhaps by targeting varieties to specific fields.

1.8. Practical implications of host resistance for the processing potato industry

As with pre-pack potatoes, quality criteria for processing are strict. However, for processing where the skin is removed, skin blemish issues are less critical. Nonetheless, pest and disease control is important to ensure that they do not affect processing quality or increase the degree of peeling. Thus whilst levels of surface diseases can be tolerated, where they cause deep lesions, such as pitted common scab or skin spot lesions exacerbated by CIPC, losses through peeling can be increased. Attention is thus required to limiting surface diseases even though they are of less importance generally.

With processing, perhaps even more than pre-pack, internal tuber defects must be avoided. Some diseases, symptomless in the tuber can have an effect on processing. Infection of certain viruses (e.g. PVY^{VN}) has been shown to result in sugar spotting in the final cooked product (e.g. crisps). Some viruses result in internal symptoms (e.g. PMTV and TRV) and these also should be avoided. Where host resistance is high to viruses the risk is concomitantly lower. However, the integration of testing fields for presence of TRV or PMTV with disease resistance rating is valuable.

Choice of variety for processing tends to be even more limited than for pre-pack and the options to utilise plant resistance by targeting varieties to specific fields correspondingly more limited. Thus a strong appreciation of variety strengths and weakness in disease and pest resistance are key to reducing pest and disease impact.

Processing crops are subject to protocols and whilst not quite as strict as pre-pack protocols, similar limitations and consequences for utilisation of plant resistance apply.

2. Basic concepts of host resistance

When plants are exposed to a pest or disease, resistance is the rule and infection the exception. For example, virtually all other crops and weeds grow unaffected in, or close to, a potato crop severely infected with potato blight. This demonstrates the idea of non-host resistance or species-specific resistance. Host resistance occurs when a variety (or cultivar) of a species normally susceptible to a pest or disease demonstrates resistance. Non-host resistance refers to interspecific variability in resistance and host resistance to intraspecific variability in resistance. The validity and permanence of non-host resistance has been questioned. For example, tree tomatoes, *Solanum betaceum*, appeared to be unaffected by potato blight, *Phytophthora infestans*, until the 1980's when they started to suffer severely (Turkensteen & Flier, 2003).

Non-host resistance is poorly understood: most current theories suggest a mixture of specific resistance and non-specific defence responses (Huitema *et al.*, 2003). Understanding the mechanisms involved could lead to practical progress in developing host plant resistance but some authors question whether introducing resistance genes from non-host species to closely related host species may provide a "bridge" for the target pathogen to the non-host (Turkensteen & Flier, 2003).

The scientific literature on host resistance is extensive with numerous monographs, papers and conference proceedings on the subject (for example, Fraser, 1985). Many are extremely technical, particularly in relation to the genetics and biochemistry of host resistance. Failure to standardise terminology can add to the difficulties of interpreting this work, for example production of volatile repellents to pests is sometimes considered an avoidance mechanism and sometimes resistance. Terms such as "minor gene resistance", "polygenic resistance" and "quantitative resistance" are sometimes used interchangeably or with subtle differences in meaning.

2.1 Types of resistance

Most plants have ways of either avoiding or lessening the impact of their potential pests and pathogens. Avoidance, resistance and tolerance are the main mechanisms employed. A crop may avoid contact with pest or pathogen. If contact with a pest or pathogen is unavoidable a resistant crop will slow development of the pest or pathogen. A tolerant crop suffers only minor damage despite being infected by a pathogen or grazed by a pest.

Avoidance mechanisms involve escape in time or space. They may be incompatible with economic requirements to produce potatoes at certain times of year and in areas where potatoes are needed to maximise profitability of a farm unit. Production of seed potato crops in areas with relatively low aphid populations, such as Scotland, can be considered an avoidance mechanism. Some authors (e.g. Ribeiro Do Vale *et al.*, 2001) consider mechanisms such as volatile repellents, mimicry, hairs and thorns to be avoidance mechanisms.

Resistance usually has a chemical basis. Chemicals produced by the host plant slow or stop development of the pest or pathogen. The chemicals involved may already exist in the plant or they may be produced in response to invasion.

Tolerance is poorly understood and may be confounded with some forms of resistance (Ribeiro Do Vale *et al.*, 2001).

Resistance is the most important mechanism by which crop plants lessen the impact of pests and pathogens.

2.2 Genetics of resistance

Most published research papers on host resistance describe resistance controlled by major genes, usually inherited dominantly. Major gene resistance is effected by one or more genes (R genes) with large effects. The resistance produced is qualitative: i.e. susceptible and resistant genotypes can easily be separated (similar to Mendel's crosses of purple and white flowered peas which produced only purple and white flowered offspring without intermediate colours). Unfortunately, major gene resistance (otherwise known as **vertical resistance)** is often temporary: new strains of a pathogen can quickly evolve to overcome resistance specified by a single gene: all varieties relying on that gene for resistance are then susceptible. A series of R genes for potato blight, *Phytophthora infestans*, were introduced into potato varieties from the 1920's: all were quickly neutralised by new strains of the pathogen (Turkensteen & Flier, 2003).

For each major resistance gene (R gene) in the host there is a corresponding avirulence gene (A_{vr} gene) in the pathogen. This is the gene-for-gene concept (Flor, 1971). When challenged by the avirulence gene in the pathogen, the resistance gene will be effective and prevent infection. Normally the resistance gene and avirulence gene are inherited in a dominant manner. Where aviruence in the pathogen or resistance gene in the host is absent, disease will develop. Major gene resistance can be described, therefore, as specific to a strain (race) of the pathogen.

Where resistance is governed by a number of minor genes (polygenic), each with small effects, the resistance is quantitative: host genotypes show a continuous range of variation from extremely susceptible to fairly resistant (the hypothetical situation where crossing white and purple flowered peas produces peas with flowers in varying shades of purple). This is also known as **horizontal resistance** and functions equally against all strains of a pathogen. It is probably more common than published research indicates, due to the difficulties of research into these traits (Ribeiro Do Vale *et al.*, 2001). It is durable and is unlikely to be overcome by a simple mutation or change of a pathogen.

2.3 Mechanisms of resistance

When a pathogen successfully invades a plant it may then go on to infect that plant or it may induce an incompatible response with the plant resisting infection. The resistance response may be localised at the site of infection or it may be systemic, affecting remote and as yet uninfected parts of the plant (systemic acquired resistance).

Localised resistance responses include cross-linking and strengthening of cell walls, synthesis of antimicrobial compounds such as phytoalexins and induction of pathogenesis related proteins such as chitinases and glucanases. Localised resistance to a pathogen is often associated with a hypersensitive response, an induced cell death at the infection site. Hypersensitive responses trap the pathogen in dead cells and stop it spreading from the original site of infection.

Infection may also induce similar defensive changes in parts of the plant away from the site of infection. These changes are particularly interesting because they are regarded as non-specific and long lasting (Heil & Bostock, 2002). Several lines of evidence suggest salicylic acid is a key component in these systemic acquired resistance responses. Levels of salicylic acid increase locally

and systemically after infection and it is also found in the vascular tissues. Mechanisms of systemic acquired resistance are complex but lead to production of pathogenesis related proteins and stimulation of mechanisms to inhibit virus replication or movement.

Insect feeding and wounding also causes local defence reactions and systemic responses through the plant. Responses include production of anti-nutritive and toxic compounds (for example, alkaloids and terpenoids), formation of physical barriers and indirect production of repellent compounds and compounds which attract predatory and parasitic insects. The part played by jasmonic acid in responses to insect feeding is thought similar to that of salicylic acid in responses to pathogens.

The salicylic acid and jasmonic pathways are not independent: there may be synergistic or antagonistic effects.

2.4 Enhancing resistance: the use of elicitors

Use of agents which mimic natural inducers, or elicitors, of plant defence responses has been shown to give good control of some crop diseases (Reglinski *et al.*, 1994 a,b). These inducers or elicitors, themselves, in the absence of living pathogens initiate plant defence responses.

Induced resistance may allow pesticides to be used less frequently and/or at reduced rates. At present, however, the efficacy of these techniques may be inadequate for commercial use. Different varieties may vary in their potential for inducible resistance suggesting prospects of breeding varieties more responsive to elicitors in the future (Walters *et al.*, 2005). This offers a possible alternative mechanism for exploitation of host resistance in potato crops. Navarre *et al.* (2003) have reported studies of systemic acquired resistance in potato but little other work appears to have been undertaken on this crop.

2.5 GM technology and host resistance

The ancestors of cultivated potatoes developed in nature where those unable to survive the attack of pests and pathogenic organisms were replaced by the mechanism of "survival of the fittest". Plant breeders, therefore, have been able to successfully source genes for resistance to many pests and diseases from the wild ancestors of potatoes. Unfortunately, in many cases there are no sources of resistance available or available resistance is tightly linked to undesirable traits.

Development of genetic modification technology means genes from non-related plant species, or even non-plant sources, can be incorporated into potatoes. For example, genes may be transferred from a bacterium controlling the production of proteins toxic to insect pests to provide resistance to insects (for example, *Bacillus thuringiensis* delta-endotoxin as produced by the CryIIIA(b) gene – registered by Monsanto) Such transformed plants present a new situation, leading to concerns about environmental impacts and safety for those consuming the crop.

The technical possibilities of these technologies, coupled to an increasingly sophisticated understanding of the natural processes of host plant resistance, provide considerable future opportunities, always provided concerns about potentially harmful consequences of this technology can be addressed to the satisfaction of society.

3. Key factors in utilising host resistance

3.1 The market place

Key criteria for variety choice for a specific market are usually quality or cooking/processing qualities. Only in exceptional circumstances are varieties selected on the basis of pest or disease resistance – and then these may be only secondary considerations. Thus for example, a crisp manufacturer will require a variety that produces crisps of the correct fry colour consistently, even if such variety has one or more pest or disease weaknesses. Similarly, a pre-pack company will require tubers of a uniform size free from blemish or disfigurement of specific varieties for supplying a supermarket. There may be a range of varieties that are supplied to a specific market but the choice is usually limited. Where this is the situation, the opportunity to utilise plant resistance to pests or diseases may be limited. However, even where a choice is unavailable, if several fields are available to grow the crop, it may be possible to target the variety to a field where the risk is lower for specific pests or diseases that a variety has weaknesses to.

Conversely, most varieties have some strengths in relation to pest and disease resistance which, with careful planning, can be used to lessen risks in certain situations. For example, where a field is known to be contaminated by TRV and the risk of spraing is high, a variety that has weaknesses in other respects but exhibits resistance to spraing may be targeted to that field. An example might be using the variety King Edward in a TRV contaminated field because it has a resistance rating of 6. Its susceptibility to potato blight and skin spot can be coped with irrespective of the field location. However, if this option was taken other agronomic criteria would need to be considered to ensure the end market specification was met - such as whether the soil type was appropriate for the skin finish required.

3.2 The achievement of quality

Quality standards for all sectors of the potato industry (seed, pre-pack and processing) have risen substantially in the last two decades. Indeed, GB has among the highest quality standards in the world. Thus to achieve the high standards required, even low levels of pest and disease attack may be unacceptable. Whilst high resistance ratings do provide some reassurance that risk of disease and pest attack will be lower, if the standards for certain pests and diseases are very high, the use of variety resistance alone may be insufficient. In these circumstances other control measures, applied in an integrated way, will be required.

For example, the blemish disease black dot can diminish the value of even a resistant variety if other control measures are not taken. Thus, whilst a resistant variety may be planted in a field where a risk of black dot is evident, in order to minimise disease occurrence, harvest must be carried out timeously otherwise the benefit of the resistance can be lost. Similarly, even where a variety is considered moderately resistant to slug damage, where the crop is destined for the fresh pre-pack market, the additional use of slug pellets is likely to be required.

3.3 The degree of effectiveness of host resistance

As described in section 1, for certain diseases and pests, the resistance rating is an expression of relative susceptibility and even varieties determined as having high resistance may still be attacked. Where diseases and pests may have a significant effect on marketability and where host resistance

is not necessarily fully effective, other control measures may be required. This can be illustrated with reference to *Rhizoctonia solani*. Even resistant varieties can be affected by the pathogen, not necessarily by producing black scurf on daughter tubers but by stem and stolon canker and the effect on emergence and tuber size distribution and through disfigurement of tubers as they develop. The judicious use of seed tuber fungicide treatments and soil-applied fungicide treatments might also be needed to achieve market, even where a variety described as highly resistant is used.

3.4 The risk of resistance being overcome

The potential of pests and diseases to evolve in order to overcome host resistance (and efficacy of agrochemicals) is well understood and should be considered when planning control strategies. However, examples of potato pests and diseases overcoming host resistance are relatively rare. The most well known example is that of potato blight and its ability to evolve to overcome major gene resistance. This has occurred despite the attempt to stack major genes together and has resulted in increased effort to breed varieties with polygenic or field resistance.

Potato blight is polycyclic pathogen (that is, it goes through several cycles of reproduction in a season) and within a season the opportunity for genetic changes in the pathogen is high. Most other pests and diseases are monocyclic or have limited cycles annually and thus the opportunity to change is much less limited than for potato blight.

There are some good examples of host resistance proving effective for many years. Most notable is that of resistance to wart disease which was introduced into varieties around a century ago, at a time when wart was a major problem. To date, this resistance is still fully effective.

Complacency that plant resistance will remain effective should not be held for whilst plant resistance to most of the pathogens and pests discussed in this review appears stable, it is known that for some diseases and pests, development of resistance to agrochemicals has occurred. Examples of changes in sensitivity to agrochemicals include:

- Resistance to thiabendazole in *Helminthosporium solani* and *Polyscytalum pustulans*
- Resistance to thiabendazole in *Fusarium sulphureum*
- Resistance to pyrethroids and pirimicarb in aphids
- Resistance to acylalanines in *Phytophthora infestans* and *P. erythroseptica*

3.5 An overview of strategies for the use of plant resistance

Potentially, plant resistance should be the first line of defence for control of pests and diseases. However, when utilising plant resistance, a range of factors need to be taken into consideration. The first four factors are described above in sections 3.1 to 3.4. Thus, for each stock to be grown a grower or agronomist will need to consider the market for which the crop is being grown and assess the quality and other criteria which the crop will be required to meet. In doing this pest and disease risks should be evaluated and the potential to which the plant resistance will reduce the risk. The value of the disease or pest resistance rating should be considered in this process. Where resistance ratings are sufficiently low that host resistance alone may not provide sufficient control, other control measures will need to be considered. Similarly, where quality criteria are particularly high and even low levels of damage threaten the ability tto achieve market criteria, the inclusion of other control measures will be required.

In addition to the factors listed above, considerations of disease or pest pressure, favourable environmental conditions and practical factors require to be taken into account. As explained in section 1, where inoculum levels are particularly high for a particular pathogen or pest, even highly resistant varieties may succumb to attack. In these circumstances, other control measures will be required. Judging the inoculum level is sometimes difficult. However, there are now available a range of tuber and soil tests which can provide quantification of inoculum. Table 3.1 provides a summary of those pests and pathogens for which tests or monitoring are available. It also provides an assessment of the value of host resistance in strategies for their use in control of pests and diseases.

The measurement of inoculum may not require a diagnostic test or results of monitoring, it may simply require visual assessment (e.g. of disease on seed) or judgement based on past or present knowledge. Thus, for example, if a field has a history of a soil-borne disease, care will be required in selecting an appropriate variety or applying extra control measures.

A further factor to be considered is the favourability of environmental factors for the disease. If conditions are highly conducive for a pest or disease, irrespective of host resistance further control measures may be required. This may apply in the store or in the field. Thus, for example, if conditions are wet prior to harvesting a seed crop of a skin spot susceptible variety, fungicide use may be required to reduce development of the disease.

Finally, as part of a strategy for utilising host resistance, the grower or agronomist needs to consider practicalities. These in themselves may over-ride the utilisation of host resistance alone and encourage the use of other control measures. An example of this is planning blight programmes. As much as a grower may wish to utilise host resistance to reduce blight fungicide applications, if there is a large acreage to treat, it is unlikely that individual fields will be treated separately. Thus a blight programme will be established to cater for the highest risk and will be applied across all fields irrespective of variety resistance available.

In order to illustrate some of these points relating to the strategies of using host resistance, Appendix 1 provides illustrations of how knowledge of host resistance might be used in practice by growers and agronomists.

TABLE 3.1 AN ASSESSMENT OF THE VALUE OF HOST RESISTANCE IN STRATEGIES FOR THEIR USE IN CONTROL OF PESTS AND DISEASES

Pest or pathogen	Value of host resistance			Tests measuring inoculum
	High	Medium	Low	
Potato leaf roll virus	+			Tuber test – incidence
Potato mop top virus	+			Tuber test – incidence Soil test - +/-
Potato virus X	+			Tuber test – incidence
Potato virus Y	+			Tuber test – incidence
Tobacco rattle virus	+			Tuber test – incidence Soil test - +/-
Other viruses	+			Tuber test – incidence
Blackleg and soft rot	+			Tuber test – quantitative
Brown rot			+	Tuber test - +/-
Common scab		+		-
Ring rot			+	Tuber test - +/-
Black dot		+		Soil test – quantitative* Tuber eye plug test - quantitative
Black scurf			+	Soil test – quantitative* Tuber eye plug test - quantitative
Dry rot	+			Tuber test – risk**
Gangrene	+			Tuber test – risk**
Pink rot	?			-
Potato blight		+		-
Powdery scab	+			Soil test – quantitative* Tuber eye plug test - quantitative
Silver scurf		+		Tuber eye plug test - quantitative
Skin spot	+			Tuber eye plug test - quantitative
Wart disease	+			-
Watery wound rot	?			-
Potato cyst nematodes		+		Soil test – quantitative
Wireworm			+	Various tests – risk
Aphids			+	Aphid trapping
Slugs			+	-

Notes: Tubers can be visually examined for most diseases over and above the tests listed

* Soil tests not yet commercially available

** Tests not validated and of limited availability

4. Overview of existing knowledge on plant resistance to potato pests and diseases

4.1 Viruses

The European Cultivated Potato Database (www.europotato.org/menu.php) has been used to provide an overview of the resistance of potato varieties to the main viruses discussed below. Ratings for a number of potato variety characteristics, including virus resistance, are collated from up to 18 sources and presented on a searchable database. Table 4.1 shows basic data on virus resistance derived from this database in June 2006.

TABLE 4.1 RESISTANCE RATINGS TO POTATO VIRUSES ON THE EUROPEAN CULTIVATED POTATO DATABASE.

	PLRV	***PMTV***	***PVX***	***PVY***	***TRV***
Number of records	2330	51	1491	2293	296
1. % Very low	1.4	5.9	2.5	2.5	3.0
2. % Very low to low	0.9	0.0	1.4	3.2	2.0
3. % Low	12.9	17.6	25.0	12.2	12.5
4. % Low to medium	13.9	7.8	4.0	9.0	5.5
5. % Medium	25.8	9.8	10.5	15.8	9.5
6. % Medium to high	19.3	3.9	12.8	16.7	12.5
7. % High	18.2	7.8	23.5	19.5	25.7
8. % High to very high	4.8	19.6	4.6	10.5	24.7
9. % Very high	2.7	27.5	15.6	10.6	4.5
Mean Score	5.33	6.21	5.67	5.77	6.08
Median score	5	7	6	6	7

Care is needed when utilising the data in this and subsequent summaries of results from the Eurpean Cultivated Potato Database. Different testing methods may give different results for individual varieties. It may be more appropriate to classify varieties into three broad groups, resistant (7-9), moderately resistant (4-6) and susceptible (1-3) to iron out testing differences and examine trends but the presentation of all 9 categories will permit the reader to do this.

The relatively large number of ratings of "Low" resistance compared to neighbouring categories (for all viruses except PLRV) appears anomalous and suggests some ratings may be subjective. Some discrepancies in the ratings of varieties, even very important ones, are discussed below but the large amount of data included suggests much interest in this subject. Hopefully, in view of the very considerable work involved, these ratings are put to frequent and practical use, and do not only provide material for literature reviews.

Except for PLRV over 40% of ratings are of high resistance or better. For PMTV and TRV over 50% of resistance ratings are high or better. This suggests there is little need to use susceptible varieties unless they have other characteristics sought in the market. The narrow range of varieties (mostly Scandinavian) rated for PMTV means these results should be treated cautiously.

Organic growers are usually particularly keen to use varieties with good host resistance to diseases. The important varietal characters organic potato growers should consider are "rapid establishment, good ground cover, early bulking yield potential and a good resistance to pests and diseases,

especially late blight" (Anon., 2003). A comparison of virus resistance ratings of varieties recommended for organic cultivation (Anon., 2003) with resistance ratings of the most commonly grown varieties and the wider range of varieties listed on the BPC website (www.potato.org.uk) is given in Table 4.2.

TABLE 4.2 MEAN VIRUS RESISTANCE RATINGS FOR THE MOST COMMONLY GROWN VARIETIES* , VARIETIES RECOMMENDED FOR ORGANIC CULTIVATION AND A BROADER RANGE OF SECOND EARLY AND MAINCROP POTATO VARIETIES.

	PLRV	***PVY***	***Spraing***
All varieties listed by BPC	5.09	4.76	4.24
Six most widely grown varieties	4.66	3.33	4.83
Varieties recommended for organic growers	5.04	5.04	4.66

*Source BPC: Estima, Lady Rosetta, Maris Peer, Maris Piper, Pentland Dell, Saturna
Ratings on a 1 to 9 scale where 9 = high resistance.

Despite the apparent availability of large numbers of varieties with good host resistance to virus diseases the results suggest there is currently little attempt to select varieties with good virus resistance, even by organic growers. Purchase of classified seed is the main control method for virus diseases advocated to organic growers (Anon., 2003).

4.1.1 PLRV

4.1.1.1 Introduction to PLRV

The consequences of Potato Leaf Roll Virus (PLRV) infection depend on variety, the strain of the virus, environmental conditions and time of infection. Plants may be stunted and the number and size of daughter tubers is reduced, so ware yields are smaller. In some varieties infection may also cause internal net necrosis making the potatoes unmarketable.

Potato leaf roll virus (PLRV) occurs in ware potato crops as a result of planting infected seed (secondary infection) or by the infection of healthy plants by aphids carrying the virus (primary infection). When healthy plants are infected in the field, symptoms (inward and upwards rolling of the youngest leaves) may not be seen that year unless infection occurs early in the season. In contrast, when infected tubers are planted symptoms appear about four weeks after emergence. Loss of ware yield due to primary infection is often slight (unless infection occurs early in the season) and much less than yield loss due to secondary infection. PLRV, and the resistance of varieties to it, is, therefore, of less concern to ware potato growers (provided they purchase healthy seed) than seed potato growers whose product can only contain insignificant numbers of tubers infected by PLRV.

PLRV is transmitted by aphids. The peach potato aphid (*Myzus persicae*) is the most important but other aphids such as *Macrosiphon euphorbiae* can also transmit PLRV (Robert, 1999). PLRV is persistent in the aphid vector. Aphids become infective after feeding on an infected plant for around two hours but cannot transmit infection for around a further day. They remain viruliferous for life. The persistent nature of the virus in the vector means PLRV can be carried long distances by viruluferous aphids carried on wind currents. Applications of aphicide are used to control the aphid vectors of PLRV. This can prevent spread of the virus within a crop but may not prevent some plants being infected by aphids blown into the crop over long distances. However, control of PLRV by control of the aphid vector is easier than control of non-persistent viruses such as PVY (Harrison, 1999)

4.1.1.2 Disease resistance ratings for PLRV

Tables of the relative resistance of potato varieties to PLRV are available in many published booklets and on internet sites. These are too numerous to review in detail. The European Cultivated Potato Database collates 2330 assessments of the resistance of potato varieties to PLRV from many European countries. Using essentially a 1-9 scale, the average resistance rating is 5.33 (Table 4.1), numerically very similar to the BPC (Table 4.2) and representing slightly above medium resistance. The distribution is fairly normal about the mean: much more so than for the other viruses in Table 4.1. This may indicate quantitative resistance controlled by many minor genes.

There is a low percentage of records of very high resistance (Table 4.1). Some Dutch lists of potato varieties (e.g. www.potato.nl) classify some varieties as resistant on the basis of possession of major gene resistance: no varieties are given this rating for PLRV.

Of the six most widely grown varieties the different ratings for Maris Peer, Maris Piper, Pentland Dell and Saturna are reasonably consistent, but the ratings for Estima vary from low (France) to medium/high (Netherlands) and for Lady Rosetta from very low (Spain) to medium/high (Netherlands). Tables from the Plant Breeding and Acclimatisation Institute, Poland (www.ihar.edu.pl/gene_bank/potato/new/potato.html) are particularly useful in allowing comparison of the highest and lowest resistance ratings from eleven different assessors. The widest differences in scoring are less for PLRV than PVY or spraing but 13 varieties have extreme scores differing by three units (1 – 9 scale): Adora, Agria, Bintje, Charlotte, Duke of York, Estima, Maris Piper, Nicola, Obelix, Ostara, Premiere, Saskia and Spunta. It is questionable whether the different assessments represent real differences in resistance to PLRV in different environments or inconsistencies in assessment.

4.1.1.3 The value of resistance to PLRV

The two possible approaches to preventing infection of field potato crops by PLRV using host resistance techniques are to select resistance to the PLRV virus or to the aphid vectors. Mndolwa *et al.* (1984*)* evaluated potato varieties and breeding selections for resistance to *M. persicae* and PLRV. They concluded that resistance to *M. persicae* colonisation was not directly related to resistance to PLRV infection and that breeding for resistance to PLRV was a more promising method of PLRV control than breeding for resistance to aphid colonisation. Other work suggests host resistance to aphids may have an important role in PLRV control: Rizvi and Raman (1983) working with two accessions of the wild potato species *Solanum berthaultii* showed 84% spread of PLRV in an accession with one aphid resistance mechanism (A hairs) but only 22% spread in an accession with two aphid resistance mechanisms (A and B hairs).

Planting resistant varieties is not an important method for controlling PLRV (Barker & Waterhouse, 1999; Hill, 1990; Slack, 2001a). Barker & Waterhouse (1999) and Foxe (1992) attribute the failure to develop varieties with good resistance to PLRV to a difficulty in identifying strong sources of resistance, lack of knowledge about the genetics of available sources of resistance and long winded screening and selection procedures. A small number of varieties react to PLRV infection by developing a systematic necrosis which is controlled by a single major gene and modifying minor genes (Ross, 1986; and discussion in Foxe, 1992; and Barker & Waterhouse, 1999). Most forms of resistance to PLRV in commercial potato varieties, however, are controlled by several genes (polygenic) (references in Foxe, 1992; Solomon- Blackburn & Barker, 1999b). Additionally, high resistance to PLRV is often inversely correlated with culinary quality (Bagnall, 1977a; Davidson 1980). PLRV resistant seedlings tend to be “horticulturally substandard” (Bagnall & Tai, 1986a). Consequently, it is perhaps not surprising that only 2.7% of assessments on the European Cultivated

Potato Database classify varieties as having "very high" resistance to PLRV (Table 4.1) – a much lower percentage than for the other major viruses reviewed here. The mean and median resistance scores on the database are also lower than for the other viruses discussed here (Table 4.1).

Nevertheless, Robert *et al.* (2000) see value in the available resistance levels: citing reports that the incidence of viruses is less in virus-resistant varieties than susceptible ones in epidemic years or when grown in places with large aphid populations. For example, Howell (1977) reported that in 1976 (an epidemic year for PLRV) only 1.5% of Scottish seed crops of the PLRV resistant variety Pentland Crown were rejected because of PLRV but more than 10% of King Edward and Desiree and 24.4% of the highly susceptible Maris Piper were rejected. This observation is particularly interesting: resistance ratings listed by the BPC do not fully reflect these differences (Pentland Crown 7; King Edward 5; Desiree 4; Maris Piper 4) and Maris Piper is rated medium resistance (Germany) and medium to high resistance (Scotland) to PLRV by sources on the European Cultivated Potato Database. Barker and Harrison (1985) and Marshall *et al.* (1988) trialled some of these varieties and concluded that Pentland Crown had high resistance and King Edward and Maris Piper low resistance. The effect of a possible climatically mediated cycle in the incidence of PLRV was also put forward by Bagnall (1977b) following a PLRV epidemic in North East Canada in 1972-75. Such cycles could be "dampened down" by use of PLRV resistant varieties. Barker and Harrison (1985) concluded that the effectiveness of resistance depends on the infection pressure which in turn depends on the abundance of virus sources and the activity of vector aphids.

Bantarri *et al.* (1993) give a North American example of the value of PLRV resistance, claiming use of varieties such as Kennebec and Katahdin, which have generalised resistance to PLRV, has reduced the severity of this virus in several N. American production areas. Although varieties with superior resistance such as Abnaki and Penobscot are available these have not been widely grown because of inferior market characters. Bagnall (1977b) considers there has been a slight shift towards varieties with some resistance to PLRV, but that several important varieties have resisted the trend such as Russet Burbank in N. America and Bintje in Europe. Howell (1977) identified a similar trend in Scotland: in the 1976 PLRV leaf roll "epidemic" 11% of the certified seed area was lost, but if the varieties certified had remained the same as in 1965 the loss would have been over 13%.

Bagnall and Tai (1986a) classified 36 potato varieties into groups based on their resistance to PLRV. Group B was taken as representative of a New Brunswick commercial variety "susceptible" to PLRV. Using this as a standard group A varieties were considered 157% susceptible, group C 57% susceptible and group D only 6% susceptible. Using more resistant varieties would not only reduce current season spread but would lower the disease reservoir reducing disease incidence in future years. If only group D varieties were grown PLRV could be virtually eliminated.

The most significant cost of PLRV, however, is that of the strict regimes necessary to ensure production of seed potatoes largely free of PLRV (Solomon –Blackburn & Barker, 2001b). This includes the procedures of seed potato production (such as control of virus vectors and roguing) and the certification procedures (field inspections etc) necessary to assure the quality of the seed produced. The development of potato varieties with PLRV resistance could therefore reduce the barriers to seed potato production, increase the potential geographic production areas and may not be viewed favourably by existing seed growers. Additional benefits would also accrue through reductions in insecticide use for aphid control. A further conflict between the interests of ware and seed potato growers may arise with varieties carrying low-level resistance to PLRV: this may be of benefit to ware growers, but could increase the difficulty of roguing for seed producers (Barker & Harrison, 1985). Resistance to PLRV may benefit seed growers by preventing the losses they suffer through failing to get crops certified in epidemic years (described by Howell, 1977) or reductions in yield when crops have to be burnt down early to prevent infection by viruliferous aphids. If isolated

production areas, periodic re-introduction of high grade virus-tested stock and rigorous roguing and certification procedures remain necessary for the control of other viruses then PLRV resistance could contribute to yield stability for seed potato producers.

Some caution is needed in interpreting trials on PLRV transmission and host resistance as the sources of viruliferous aphids differ in different countries. In the UK groundkeepers are potentially important sources of PLRV but not in countries where winters are sufficiently severe to kill them.

4.1.1.4 Problems in the adoption of resistance to PLRV

Jayasinghe *et al.* (1989) suggested that potato clones with good resistance to PLRV were less resistant if already infected by PVX or PVY. Subsequently, Brandolini *et al.* (1992) suggested varieties bred for PLRV resistance should also possess extreme resistance to PVY and PVX. Although the significance of this is not fully understood (discussion in Barker & Waterhouse, 1999) there appears a possibility that varieties resistant to some viruses will behave in unexpected ways when infected by other viruses (Valkonen, 1994). Ideally, varieties would be developed with resistance to a broad range of viruses. Varieties with resistance to PLRV but which develop obvious symptoms when infected might be favoured by seed growers.

Resistance, such as that to PLRV, controlled by many genes is unlikely to be broken down by mutation of the pathogen.

4.1.1.5 Integration of host resistance and other control methods

Difonzo *et al.* (1995) working in the United States studied the action thresholds for *M. persicae* necessary to prevent transmission of PLRV on susceptible and resistant varieties. They concluded that lower action thresholds could be used on resistant than susceptible varieties. This work may not be directly applicable to the UK where potato groundkeepers are a potential source of PLRV inoculum and aphids may fly into a crop already carrying infection.

In the absence of aphid control by insecticides the observed resistance of susceptible and resistant varieties will vary according to the population of viruliferous aphids. Where infection pressure is light it will be difficult to distinguish between moderate and high resistance and if infection pressure is heavy it will be difficult to distinguish between susceptible and moderately resistant varieties. Immune varieties, however, will remain free of the virus (Bagnall, 1977b).

In the UK at present, PLRV is of much less concern that PVY. The low inherent levels of virus in seed stocks, roguing during seed production and the use of insecticides have eased problems with this virus.

4.1.1.6 Genetic modification

The production of potato varieties resistant to PLRV is particularly attractive because of the worldwide importance of the disease (Solomon – Blackburn and Barker, 2001b) and because of the scarcity of alternative mechanisms such as the R_x and R_y genes for PVX and PVY.

In the United States, Russet Burbank Potato and PLRV may be the most important crop/virus combination (Kaniewski & Thomas, 2004). Monsanto have produced Russet Burbank NewLeaf®Plus Potato lines resistant to Colorado Potato Beetle (CPB) and infection by PLRV. Apart from their lack of susceptibility to CPB and PLRV these clones behave normally and have unchanged susceptibilty to other pests and diseases.(www.agbios.com/dbase.php). When grown

commercially the NewLeafPlus clone produced healthy potato crops free of net necrosis and with a markedly reduced or zero need for insecticide treatment. In the Idaho and Basin growing areas the financial benefit to the grower through the absence of net necrosis (caused by PLRV) was estimated at $86 –102/acre. Additional benefits accrued from the savings in insecticide due to Colorado Potato Beetle resistance (potentially "equivalent to 30,000 spray plane sorties") (Kaniewski & Thomas, 2004)

The use of genetically modified potatoes in the United States demonstrates the potential value of PLRV resistance to ware potato growers, however, the consequences for seed growers appear more uncertain.

4.1.2 PMTV

4.1.2.1 Introduction to PMTV

Potato Mop Top Virus (PMTV) is important as one cause of spraing symptoms. Symptoms may be present in the tubers at the time of harvest in the year of primary infection, however, no symptoms develop on the haulm in the year of infection. When tubers from infected plants are grown some may show secondary symptoms: mop top of the haulm, chevrons in the foliage (mediated by low light levels and moderate temperature) and malformed tubers. The movement of PTMV in potato is relatively poor and the virus may not invade every stem on an individual plant. In addition, a proportion of tubers formed from an infected plant are virus free. Furthermore, the virus may be passed to only 20% of plants grown from infected tubers (Barker *et al*., 2000).

PMTV is transmitted by the powdery scab fungus *Spongospora subterranea*, and resting spores from lesions on seed tubers may be viruliferous. Resting spores formed on the roots of PMTV infected plants can carry the virus for up to 18 years (Weingartner, 2001).

4.1.2.2 Disease resistance ratings for PMTV

Disease resistance ratings for PMTV are much rarer than for the other important potato viruses, possibly because of the difficulties in testing. Some sources, such as BPC, NIAB and the Polish Plant Breeding and Acclimatisation Institute, give resistance ratings to "spraing" which may be caused by PMTV or TRV. The European Cultivated Potato Database has only 51 entries (mostly Scandinavian) for resistance to PMTV (Table 4.1) with none of the six most widely grown varieties included.

Some further data is available from the scientific literature, for example, Germundsson *et al.,* (2002) report Saturna unacceptably sensitive; Appell and Desiree more resistant; and Bintje tolerant with symptomless infected tubers. Unusually, whilst Saturna appears susceptible and exhibits severe PTMV spraing in Scandanavia, in the UK symptoms rarely occur despite powdery scab being relatively common. Sandgren (1995) states "to avoid an outbreak of spraing in a PMTV infected field only Bintje can be recommended among the Swedish varieties".

4.1.2.3 The value of resistance to PMTV

Spraing symptoms can appear following primary infection by PMTV so resistant cutivars would allow ware potato growers to plant fields harbouring viruliferous resting spores of powdery scab.

New sites can be infected by planting tubers carrying viruliferous lesions of powdery scab (Jones and Harrison, 1969).

According to Weingartner (2001) "There is presently no good source of resistance or tolerance (to PMTV) in commercially acceptable potato varieties" and Germundsson *et al.*, (2002) state "sufficiently effective natural resistance to PMTV is not yet available in potato varieties". In view of this observation it is surprising that the mean resistance score for PMTV on the European Cultivated Potato Database is the highest of the viruses reviewed here, as is the proportion of ratings in the very high resistance rating. This is probably an artefact arising from the small number of observations for this virus: nearly all listed varieties are of Scandinavian origin with assessment by the Nordic gene-bank in Sweden.

The value of a moderate level of resistance varies from year to year as weather conditions affect the ability of the vector to infect the host. Resistance may also vary with location: in Sweden the varieties Lady Rosetta and Hulda are just as sensitive to PMTV as Saturna but in Denmark they appear more tolerant of PMTV (Sandgren, 1995)

4.1.2.4 Genetic modification

The shortage of naturally occurring sources of resistance to PMTV has led to interest in the use of genetic modification, particularly in Scandinavia where PMTV causes significant losses (Germundsson *et al.*, 2002). Work has also been undertaken at SCRI (Barker *et al.*, 1998).

4.1.3 PVX

4.1.3.1 Introduction to PVX

Potato Virus X (PVX) differs from the two viruses discussed above in being mainly transmitted mechanically, rather than by a vector. It is spread by contact of healthy and infected leaves or by contaminated machinery, people or large animals moving through a crop. Symptoms are the same whether the infection arises through planting infected seed or through infection in the field. There are many strains which produce symptoms of varying severity. However, the virus is sufficiently homogeneous that polyclonal antibodies can detect all strains (Slack, 2001b). Typical symptoms are mild mosaic and yield loses may be up to 15 – 20% (Slack, 2001b). Mixed infections with other viruses, such as PVA or PVY, may result in higher yield losses. The occurrence of the disease has been reduced greatly in the last few decades by the action of seed certification schemes. The infection of many varieties by many strains produces no symptoms and infection remains latent. PVX can infect a number of weed species and these may provide a source of inoculum, together with potato groundkeepers.

4.1.3.2 Disease resistance ratings for PVX

Many of the disease resistance tables for potato varieties do not include PVX (for example BPC, NIAB and the Plant Breeding and Acclimatisation Institute, Poland). Nevertheless, there are 1471 ratings of varieties for resistance to PVX on the European Cultivated Potato Database. These have an erratic distribution (Table 4.1) compared to the more normal distribution of ratings for the other viruses.

Considering the assessments for the most commonly grown varieties, Maris Peer, Maris Piper, Lady Rosetta and Pentland Dell are all reasonably consistent but Estima varies from Low (Scotland) to

Medium/High (Netherlands) and Saturna from Medium/High (Germany and Hungary) to Very High (France).

Dutch sources (www.potato.nl) list 12 varieties (out of a total of 104) as having resistance to PVX. These varieties have 33 ratings for PVX resistance on the European Cultivated Potato Database: 76% are very high resistance and a further 18% are high or high to very high. However, two varieties (Minerva and Timate) have low resistance ratings from Germany. These anomalous results appear puzzling, particularly in the absence of climatic effects on a vector.

4.1.3.3 The value of resistance to PVX

Some varieties have extreme resistance, determined by a single dominant gene R_x (Slack, 2001b). The varieties Alcmaria, Astarte, Barbara, Cara, Saco, Saphir and Serrata Inta all possess at least one R_x gene to PVX (www.scri.sari.ac.uk/TiPP). All are rated with at least high resistance to PVX on the European Cultivated Potato Database except Cara and Saco which also have low ratings (in addition to high or better) and Serrata Inta which is not rated. The R_{xadg} gene in Cara is considered by most sources to confer resistance to all four strains of PVX (Solomon-Blackburn & Barker, 2001a). Additionally, some PVX strains induce a hypersensitive reaction in some varieties: infected cells die quickly and the virus is unable to spread further. The varieties King Edward, Epicure and Pentland Dell are hypersensitive to common strains of PVX (Brenchley & Wilcox, 1979). Whilst these varieties are usually rated at least high on the European Cultivated Potato Database, Epicure has one rating of Low (Germany). The varieties Arran Victory and Craigs Defiance also possess hypersensitive resistance to PVX (www.scri.sari.ac.uk/TiPP). Arran Victory (resistance to strain group 2) is not listed on the European Cultivated Potato Database but Craigs Defiance is rated very high resistance. This suggests field experience generally correlates well with the genetics of a variety – but with a small number of discrepancies.

Davidson (1980) considered good control of PVX had been achieved by seed certification but also "the abundance of varieties with resistance to PVX".

4.1.3.4 Problems in the adoption of resistance to PVX

The Rx gene is highly specific to the pathogen and potentially could be overcome by a simple mutation of the pathogen (Ghislain, 2000). Major gene resistance in potato has proven quite durable (Solomon-Blackburn & Barker, 2001b), in some cases it has already withstood 50 years of field exposure (Bagnall, 1977b). Nevertheless, a South American strain of PVX that can overcome all known resistance sources is known. That it has not become more widespread suggests it has other weaknesses and less fit to survive (Foxe, 1992; Hill, 1990).

4.1.3.5 Integration of host resistance and other control measures

Resistance to PVX can only be achieved through resistance to the virus itself and not through resistance to a vector. Use of PVX free seed is an effective control for ware growers. Resistance to PVX would mainly assist the production of healthy planting stocks (Slack, 2001b).

4.1.3.6 Genetic modification

Genetic modification for resistance to PVX has received less attention than other viruses which are less easily controlled by other means (Ghislain, 2000; Kaniewski & Thomas, 2004). Nevertheless, genetically modified varieties for PVX resistance may be of interest in developing countries, such

as Mexico, where seed certification schemes are poorly developed (Kanieswki & Thomas, 2004; Qaim 1998).

4.1.4 PVY

4.1.4.1 Introduction to PVY

Potato Virus Y (PVY) causes particularly variable symptoms, depending on the virus strain and variety infected. "Textbook" symptoms of primary infection by the common strain PVY ^{o} are "leaf drop streak". Black necrotic stripes appear on the lower leaves which eventually drop off. The effect on yield depends on when infection occurs, the strain of virus involved, the potato variety and environmental conditions. Losses may reach 50% but are usually smaller (Hill, 1990). Secondary infection causes symptoms described as severe mosaic – plants are dwarfed with small, crinkled and twisted leaves. Affected crops give very poor yields. The PVY^{n} strain causes milder symptoms. It can infect tobacco, so is important if seed potatoes are to be exported to tobacco growing areas.

PVY ^{o} and PVY^{n} are transmitted non-persistently by a range of aphid species but particularly *Myzus persicae.* Use of aphid host resistance is less likely to be effective for a non-persistent virus such as PVY than a persistent virus such as PLRV. Indeed frequent probing and movement by aphids on a distasteful resistant host may spread a non-persistent virus at a faster rate than on an aphid susceptible host (Valkonen, 1994). Moreno & Mosquera (1983) demonstrated more rapid spread of PVY on an aphid resistant variety than three susceptible ones, but PLRV spread more rapidly on the aphid susceptible varieties.

4.1.4.2 Disease resistance ratings for PVY

Disease resistance ratings to PVY accorded to different potato varieties by different authorities appear more variable than for the other major viruses discussed here. The tables of the Polish Plant Breeding and Acclimatisation Institute have ten varieties where the highest and lowest ratings given by different authorities vary by five or more units (1-9 scale). Even the ratings of commonly grown varieties can vary widely. On the European Cultivated Potato Database (resistance to PVY – strain not specified) the ratings for Estima vary from low resistance (France) to high resistance (Netherlands), for Pentland Dell from very low (Scotland) to high (Netherlands) and for Saturna from low/medium (Germany) to high/very high (Scotland).

The difference in resistance ratings could be due to the occurrence of different strains of the disease or different effects of environment on disease development or aphid activity. Both explanations seem doubtful given the scale of reported differences within Northern Europe and the accuracy and usefulness of published resistance ratings for PVY may be questionable.

In contrast, Bagnall & Tai (1986b), found good agreement between assessments of PVY resistance in America and in Holland, despite the use of different strains of PVY (PVY^{o} in America and PVY^{n} in Holland)

In theinformation listed by the BPC (www.potato.org.uk) the mean score of the six most widely grown varieties is 3.33 compared to a mean of 4.76 for all varieties (Table 4.2). The most widely grown varieties are therefore more susceptible to PVY than average suggesting this is not an important consideration in variety selection.

4.1.4.3 The value of resistance to PVY

Comprehensive (effective against several strains of the virus) extreme resistance genes to PVY are available (Foxe, 1992; Solomon- Blackburn & Barker, 2001b) but are included in relatively few varieties. To date no UK-bred varieties are known to include an R_y gene (Solomon- Blackburn & Barker, 2001b) but the gene has been included in varieties from Germany, Holland, Hungary and Poland where breeders afford resistance to PVY a higher priority. Foxe (1992) points out that immunity is inherited as a single dominant gene and considers it "relatively simple to breed for", however, Solomon-Blackburn and Barker (2001b) point out a number of difficulties including an association of the resistance genes with wild characteristics, male sterility and difficulties in selection when hypersensitive resistance genes are also present. Inclusion of the R_y gene in continental varieties but not UK varieties suggests this character is not sought by UK growers. Of the potato varieties listed at www.potato.nl only one (Sante) has claimed resistance to PVY^n strain.

The varieties Barbara, Corine, Fanal, Pirola and Szignal all possess extreme resistance (R_y gene) to PVY (www.scri.sari.ac.uk/TiPP) as do Roeslau and Sante (Robert *et al.*, 2000) and all are classified as having at least high resistance to PVY on the European Cultivated Potato Database. The varieties Cara, Desiree and Pentland Crown all possess hypersensitive resistance genes to PVY (N_y gene) (www.scri.sari.ac.uk/TiPP). All have at least high ratings for PVY resistance on the European Cultivated Potato Database: Desiree also has a medium/high rating. The N_y genes are variable in expression and appear dependent on the virus strain that dominates (Foxe, 1992). The varieties Epicure, King Edward, Maris Piper and Pentland Dell have hypersensitive resistance to PVY strain C (N_c gene) (www.scri.sari.ac.uk/TiPP). These varieties have much lower PVY resistance ratings on the European Cultivated Potato Database: King Edward and Epicure are low resistance, Maris Piper medium or low and Pentland Dell very low/low, medium or high. The C strain of PVY is now rare and is not effectively transmitted on its own (Hill, 1990). Where different genes confer resistance to different strains of a virus it is therefore necessary to ensure appropriate ones are chosen. One recent UK-bred variety with a high resistance to virus Y is Isle of Jura (rated 9).

Bagnall and Tai (1986b) classified potato varieties into five groups based on their susceptibility to PVY. They estimated circumstances leading to 100 PVY infected plants in group A would lead to 73.3 infected plants in group B, 25.6 in group C, 12.5 in group D and only 1.4 in group E. If A and B group varieties were withdrawn the virus would spread sufficiently to infect C and D group varieties only in years of high aphid activity and would not infect group E varieties at all. This is consistent with the review of Ribeiro Do Vale *et al.* (2001) which found that quantitative resistance reduces the incidence of diseased plants in a single season and also over several seasons.

4.1.3.4 Problems in the adoption of resistance to PVY

Similarly to the R_x gene for PVX (section 4.1.3.4) the R_y gene could potentially be overcome by pathogen mutation although there are no records of this to date. Davidson (1980) considered the introduction of new strains was the most likely cause of resistance breaking down, giving the introduction of PVY^n from South America as an example.

4.1.3.5 Genetic modification.

Russet Norkotah and Shepody are important potato varieties in the Pacific Northwest of America. They are susceptible to PVY but express only slight foliar symptoms making production of healthy seed difficult. Consequently, genetic engineering of these varieties for PVY resistance was undertaken at Oregon State University. One Russet Norkotah and two Shepody clones have been

identified with high resistance to PVY and similar yields and quality to standard clones of the same varieties (Rykbost *et al*.,1998)

Monsanto have also produced two clones of Shepody resistant to PVY (strain PVY^{o}) and Colorado Potato Beetle (CPB) and one clone of Russet Burbank with similar resistance. These lines have been shown to grow normally and to have unaltered resistance to pests and diseases other than PVY and CPB (www.agbios.com/dbase.php).

4.1.5 TRV

4.1.5.1 Introduction to TRV

Tobacco Rattle Virus (TRV) is transmitted by trichodorid nematodes and causes the symptoms known as "spraing" in tubers of some varieties. "Stem mottle" symptoms may also develop after secondary infection. Precise symptoms vary widely with the virus strain and potato variety. The virus is persistent in the vector nematodes: they can also infect a range of weed species which act as a reservoir for the virus.

4.1.5.2 Disease resistance ratings for TRV

Resistance rating values may not be wholly reliable. Many sources, for example BPC, NIAB and Polish Plant Breeding Institute and Acclimatisation give resistance ratings to "spraing", which may be caused by PMTV as well as TRV. Differences in the ratings for individual varieties listed by the Polish Plant Breeding and Acclimatisation Institute differ less than those for PVY, but more than those for PLRV: three varieties (Colmo, Desiree and Kondor) have ratings differing by five or more units. The European Cultivated Potato Database has 296 ratings for resistance to TRV. There are some large differences in the resistance ratings for well known varieties: Maris Piper has medium (Germany) and high (Scotland) ratings and Estima low (Germany) or high (Scotland) ratings.

In general, resistance to spraing does not appear a major consideration when selecting a potato variety. The mean resistance score for all varieties listed by the BPC (www.potato.org.uk) is 4.2. The mean score of the six most widely grown varieties is slightly higher (Table 4.2) at 4.8 (more resistant) but the mean includes a very wide variation. Pentland Dell is only rated 1 (highly susceptible) whereas Lady Rosetta scores 8.

In the UK, varieties fall into three groups:
Resistant: varieties show no symptoms and virus cannot be recovered or detected (ratings 7-9)
Sensitive: varieties exhibit spraing symptoms but virus particles are difficult to detect. Only limited transmission occurs to progeny tubers. Nematodes feeding on roots so not acquire the virus (ratings 1-3)
Susceptible: varieties show few symptoms in the tuber flesh but can be systemically infected and nematodes are able to acquire the virus when feeding (ratings 4-6)

4.1.5.3 The value of resistance to TRV

TRV produces symptoms in the year of infection and is present in many fields. Evans *et al.*, (2006) found the virus in 30% of tested fields in Scotland. Varieties which are immune to infection, or which do not develop economically damaging symptoms, would therefore be beneficial. An increase in the number of Trichodorid virus-vectors found during routine soil testing (Evans, 2006) may suggest an increasing likelihood of infection by TRV in potato fields.

Resistance to TRV in potatoes appears to be controlled by a single major gene (N_t) and modifying major genes (Foxe, 1992)

4.1.5.4 Problems in the adoption of resistance to TRV

Trichodorid nematodes move only short distances so TRV populations in different areas are isolated and as a result different strains have developed (Robinson & Dale, 2004) According to Maas (1975) growing resistant varieties is of little benefit in avoiding spraing because of these regionally different strains of TRV. However, Robinson and Dale (2004), assessed spraing symptoms caused by TRV in 15 potato varieties at three sites and found no evidence of differences attributable to the virus isolates at the different sites. They suggest the durable resistance of variety Record should be further investigated as a source of resistance for breeding TRV resistant varieties.

TRV in potato varieties which develop spraing symptoms tends not to be systemic and is transmitted to a very limited proportion of daughter tubers. In contrast TRV infection of potato varieties which are susceptible but do not usually develop spraing symptoms, such as Wilja, is systemic and transmitted to all daughter tubers. In these varieties the virus can persist through many generations (Xenophontos *et al.,* 1998). Planting TRV infected susceptible varieties is therefore an important means of spreading TRV to uninfected ground. This is not a problem with varieties such as Record which are resistant to infection by TRV (www.scri.sari.ac.uk/TiPP).

4.1.5.5 Integration of host resistance and other control measures

The main use of nematicides (apart from potato cyst nematode control) on potatoes is to control TRV vectors, such as in Scotland (Evans *et al.,* 2006). The vectors may cause damage by direct feeding and where numbers are high this alone may justify nematicide treatment. Where direct feeding damage is not of concern measurement of TRV levels in the virus vector population will indicate when nematicide treatment to prevent TRV induced spraing is necessary (Evans *et al.,* 2006). The degree of host resistance to TRV in the variety grown will be an additional factor affecting the need for nematicide treatment. Given that control of spraing using nematicides is incomplete and the potential concern by supermarkets over residues, the use of host resistance to limit infection will become more important.

4.1.6 Other viruses

In addition to the major viruses discussed above numerous others have been reported in potato. Plant Viruses Online (//image.fs.uidaho.edu/vide/refs.htm) lists 54 viruses to which *Solanum tuberosum* is susceptible. Fortunately, most are only of academic interest.

In addition to those described above the European Cultivated Potato Database gives resistance ratings for potato viruses A, B, C, M and S. These are summarised in Table 4.3. Nomenclature of these viruses can be inconsistent: some are often considered strain groups of other viruses (Valkonen *et al*., 1996).

TABLE 4.3 RESISTANCE RATINGS TO POTATO VIRUSES ON THE EUROPEAN CULTIVATED POTATO DATABASE.

	A	***B***	***C***	***M***	***S***
Number of records	1586	144	135	716	219
1. % Very low	0.9	0	0	3.8	23.3
2. % Very low to low	0.8	0	0	1.8	6.4
3. % Low	12.5	36.7	31.1	18.9	27.4
4. % Low to medium	3.1	0	0.7	7.8	4.6
5. % Medium	8.9	3.5	3.0	20.5	19.6
6. % Medium to high	10.5	47.9	60.7	16.3	2.7
7. % High	24.5	13.9	4.4	27.4	13.2
8. % High to very high	10.3	0	0	1.0	0.4
9. % Very high	28.5	0	0	2.5	2.3
Mean Score	6.70	5.06	5.07	5.18	3.68
Median score	7	6	6	5	3

Apart from PVA the mean and median resistance scores for these viruses are lower than for the major viruses (Table 4.1). In particular, with the exception of PVA the proportion of records of high resistance or better is much lower than for the major viruses in Table 4.1. This illustrates the importance of relating host resistance to the occurrence of the disease and its economic effect: the above viruses occur rarely or have little effect on yield. For example, PVS where Table 4.3 suggests a worrying lack of available resistance is rarely of economic significance, gives normally undetectable symptoms and has comparatively little effect on yield (Hill, 1990). Similar considerations apply to the adoption of host resistance to viruses where this is strain specific, for example PVY – section 4.1.4.3.

The Seed Potatoes (England) Regulations 2006 additionally have requirements for Tobacco Veinal Necrosis and the Scottish Agricultural Science Agency also offer testing for Tomato Black Ring Virus.

In view of the limited importance of these viruses, the complexity and limited availability (many papers in difficult to locate sources) of the literature they are not reviewed in detail here. Further information and references on these less important viruses is available in standard works such as Brenchley and Wilcox (1979); Hill (1990); Stevenson *et al.* (2001); and Valkonen (1994). The review of Solomon-Blackburn and Barker (1999b) includes information on PVA, PVV, PVM and PVS.

4.1.7 Conclusions on viruses

In general, virus diseases of potatoes have been controlled by seed certification schemes, rather than the introduction of host resistance. This has generally been highly successful but such schemes are expensive to operate. Host resistance to potato viruses may be of particular value in less developed countries where the infrastructure may not exist for effective seed certification schemes. Major resistance genes are available for viruses such as PVX and PVY and appear to be much more durable than resistance genes for fungal pathogens. Introduction of varieties with improved quantitative resistance to viruses should reduce the incidence of those viruses over a number of growing seasons (Ribeiro Do Vale *et al.*, 2001). It will also slow the rate of epidemic build up and

possibly allow more time for the use of other control measures such as chemical control of aphid vectors. An improvement in yield stability would be a significant benefit.

Tables and databases on the characteristics of potato varieties suggest good availability of worthwhile resistance to most potato viruses. Resistant varieties are often not widely grown: presumably because they lack other characters essential in the marketplace. There is reasonable agreement between data on resistance of potato varieties to virus from different sources but with sufficient discrepancies, even between major varieties, to be worth investigating.

Growing existing, or newly introduced, varieties with improved virus resistance would provide benefits to ware potato growers at little, or no, extra cost. If potato varieties with market acceptability and wide ranging effective resistance to viruses become available the effect on seed potato growers is less clear. In the extreme case ware potato growers may simply retain their small tubers and use them to establish next year's crop. More virus resistance may also permit production in wider geographic areas. A parallel exists with the daffodil crop (grown in a similar way to potatoes) where virus diseases are relatively unimportant, and the crop is a poor host to virus-vector aphids. Daffodil growers merely grade out their small bulbs after lifting and use them to establish the following crop. Roguing is carried out to maintain the health of stocks and a certification scheme is available in Scotland but there is not the same separation of propagation and ware stocks as with potato. Although benefits from virus-tested daffodil bulb stocks have been demonstrated (Sutton *et al.*, 1988) they have been insufficient to cause their adoption into commerce.

4.2 Bacterial diseases

The European Cultivated Potato Database (www.europotato.org/menu.php) has been used to provide an overview of the resistance of potato cultivars to the main bacterial diseases discussed below. Ratings for a number of potato cultivar characteristics, including disease resistance, are collated from many European sources and presented on a searchable database. Table 4.4 shows basic data on resistance to bacterial diseases derived from this database in October 2006.

TABLE 4.4 RESISTANCE RATINGS TO BACTERIAL DISEASES ON THE EUROPEAN CULTIVATED POTATO DATABASE.

	Bacterial soft rot	***Blackleg***	***Common scab***	***Ring rot***
Number of records	275	748	2364	17
1. % Very low	0.7	0.7	0.9	0
2. % Very low to low	0	0.4	1.1	0
3. % Low	8.7	5.5	11.4	58.8
4. % Low to medium	8.7	6.0	13.1	0
5. % Medium	26.5	15.4	25.9	11.8
6. % Medium to high	15.3	18.0	24.0	0
7. % High	35.6	47.6	19.6	23.5
8. % High to very high	3.6	5.2	3.0	0
9. % Very high	0.7	1.2	1.0	5.9
Mean Score	5.71	6.13	5.34	4.53
Median score	6	7	5	3

The ratings for bacterial soft rot and blackleg follow a similar distribution: the causal organism of both is given as *Erwinia spp*. on the database so this seems unsurprising. Ratings for common scab have a fairly normal distribution with a mean slightly above medium. There are few records available for ring rot. For all the above diseases at least 20% of records are for high or better resistance suggesting that suitable sources of resistance are available.

4.2.1 Blackleg

4.2.1.1 Interpreting published disease and pest resistance

Three erwinias, *Erwinia carotovora* subsp. *atroseptica* (*Eca*), *E. carotovora* subsp. *carotovora* (*Ecc*) and *E. chrysanthemi* (*Echr*) are pathogenic on potato plants (Perombelon & Kelman, 1980). All three can rot seed tubers or pieces, causing non-emergence (Molina & Harrison, 1977; de Lindo *et al.*, 1978; Cother, 1980; Perombelon *et al.*, 1987) but blackleg is caused by the bacterial species *Eca* and *Echr* in cool and hot climates respectively (Perombelon, 1992). *Ecc* does not cause blackleg because it apparently fails to compete with the other erwinias and other pectolytic bacteria on seed tubers (Perombelon, 1992). Blackleg is a stem rot, or plant wilting and desiccation, that originates at the point of attachment to the seed tuber; aerial stem rot, which is caused mainly by *Ecc*, originates from infections of stems above ground level (Perombelon & Kelman, 1987). Most of the information available in the literature on blackleg refers to *Eca* blackleg and not that caused by *Echr*. In this section of the review, where information or comments refer to *Echr* this will be clearly stated.

4.2.1.1.1 Background to resistance breeding

Many test methods for assessing resistance to blackleg have been developed over the years. Most involve testing in the field. One main difference between tests is whether or not it is the seed tuber that is challenged by the pathogen (Hossain & Logan, 1983; Lapwood & Gans, 1984) or whether the stems are inoculated directly (Hossain & Logan, 1983; Lapwood & Read, 1986). For tests involving tuber inoculation there are many ways of inoculating the seed tubers. For example, Maas Geesteranus & Vruggink (1976) and Logan & Copeland (1979) dipped undamaged tubers in inoculum. Other tests involved damaging tubers (Hossain & Logan, 1983; Logan, 1969; Wastie, 1984). In other tests inoculum was introduced into tubers by vacuum infiltration (Perombelon & Lowe, 1979) to simulate infection of lenticels. Some of these methods were compared in a multi-site experiment conducted in two consecutive years using *Eca* (Gans *et al*., 1991). The main comparison was between inoculation of seed tubers using a needle compared with a jet-injector. Overall, the results for both methods were similar. Very significantly, the authors obtained 8 years of data (1979-1986) on the incidence of blackleg recorded in seed crops entered in the Scottish Seed Classification Scheme and therefore they could make an all-too-rare comparison of variety resistance ratings in practice and as determined by testing methods. The needle-inoculation method correlated well with the SSCS results ($r=0.85$, $P<0.01$) whereas the corresponding correlation coefficient for the jet inoculation method was not significant.

Allefs *et al.* (1996) developed an inoculation method that assesses the resistance to spread of *Erwinia* rots from seed tubers into the sprouts. They termed this component of resistance "stem base resistance". When 12 varieties were screened differences in stem base resistance were clearer for *Echr* than *Eca*. Allefs *et al.* (1996) recommended that for blackleg resistance screening was not carried out in the field, this component of resistance should be concentrated on rather than the resistance of stem tissue or of mother tubers.

There are few reports of the relative resistance ranking orders of varieties for *Eca* and *Echr* blackleg. However, Allefs *et al.* (1995) reported that ranking orders were consistent over locations and years when seed tubers were vacuum infiltrated with the bacterial pathogens.

Reports of the relationship between variety resistance to blackleg and tuber soft rot are not consistent. Zimnoch-Guzowska *et al.* (1999) stated that it should be possible to breed for genotypes resistant to blackleg and tuber soft rot. They observed a positive relationship between tuber and stem resistance to *Erwinia* species. However, Carputo *et al.* (1997) found that correlations between blackleg and tuber soft rot were never significant. Allefs *et al*. (1995) found that blackleg resistance in the field was not related to the tissue resistance of tubers in the laboratory, and stated that seed tuber resistance changed substantially within weeks of planting.

4.2.1.1.2 What resistance is available?

In the last 10 years many potato genotypes with enhanced resistance to blackleg have been identified or produced, using either wild or cultivated *Solanum* spp. or through genetic modification. Bradshaw *et al*. (2006), writing about potato breeding in general, stated that only a very small amount of the available biodiversity in wild and cultivated species of potato has so far been used by breeders and that new technology will allow much greater use of it in future.

Hybrids between *Solanum tuberosum* and four wild species of *Solanum* were screened for resistance to blackleg (*Eca*). Hybrids of *S. tuberosum* with *S. multidissectum* or *S. tarijense* gave the highest number of genotypes resistant to both blackleg and tuber soft rot. Some clones derived from

S. tuberosum x *S. tarijense* crosses were susceptible (Carputo *et al.,* 1997). Bains *et al.* (1999) screened hundreds of accessions of six wild species of *Solanum* for stem resistance to *Eca*. A high percentage of the accessions of *S. boliviense*, *S. chacoense* and *S. sancta-rosae* were resistant or highly resistant. In total 65 resistant or highly resistant clones of wild *Solanum* species were identified. In addition, the study identified three clones that were resistant to both stem rot and tuber rot. Stem bases of interspecific hybrids and somatic hybrids of *S. tuberosum* and *S. brevidens* were challenged with *Eca* to assess blackleg resistance (Zimnoch-Guzowska *et al.,* 1999). In two years of evaluation three diploid hybrids and a derivative of one of the somatic hybrids did not develop any blackleg symptoms. Zimnoch-Guzowska *et al.* (2000) reported that new sources of resistance to *Eca* have been selected in genotypes originating from crosses of *Solanum tuberosum* with the wild species *Solanum chacoense* and *Solanum yungasense*. Lees *et al.* (2000) assessed long-day-adapted *Solanum phureja* clones for resistance to blackleg (*Eca*). Many of the clones were as resistant to blackleg as the most resistant control variety, Ailsa, which was rated 7 (on a 1 to 9 scale) (Anon., 2002). Chen *et al.* (2003) identified accessions of *S. circafolium* and *S. bulbocastanum* that had significantly higher resistance to blackleg (*Eca*) than the existing varieties or wild *Solanum* spp. tested. However these accessions were more susceptible to late blight.

Desiree plants were genetically modified to include the chicken lysozyme gene. Approximately one fifth of the modified clones had increased resistance to *Eca* blackleg (Serrano *et al.,* 2000). Arce *et al.* (1999) reported that transforming potato clones with either the gene encoding the acidic attacin protein or the cecropin analog peptide SB-37 resulted in about one fifth of the clones having increased resistance to *Eca*.

4.2.1.2 Practical use of host resistance

The evidence from the blackleg (*Eca*) resistance ratings of the most popular 31 varieties in GB is that varieties are not selected because of their resistance to blackleg (Table 4.5). At present too many of the potato crops have a rating of 5 or below. There is considerable scope to make much greater use of variety resistance to control blackleg. Zimnoch-Guzowska *et al.* (2000) also stated that the level of genetic resistance currently present in varieties is insufficient to protect the crop.

TABLE 4.5 THE PERCENTAGE CROP AREA (2004) OF POTATO VARIETIES WITH EACH RESISTANCE RATING FOR BLACKLEG

	Blackleg resistance rating									
	1	2	3	4	5	6	7	8	9	None
% of crop area for 31 varieties[1]	1.9	13.8	12.5	20.3	31.0	7.0	0	3.8	1.5	8.2

4.2.1.2.1 Use of resistance as a sole method of pest and disease control

Varietal resistance is infrequently used alone (see 4.2.1.2.4)

4.2.1.2.2 Assessing risks when utilising host plant resistance

The risk of blackleg developing in a crop can be determined from a blackleg risk assessment. This uses one of several diagnostic tests to determine the number of *Eca* cells on the seed tubers at, or close to, the time of planting. The information on the extent of seed tuber contamination by *Eca* is used firstly to determine the market for a seed stock. The information can also be used in conjunction with information on variety resistance and field information, e.g. how free-draining the field is, to select fields for particular seeds stocks.

There are reports of interactions between the blackleg pathogen and some other potato pathogens. The interactions have generally not been studied in sufficient detail to indicate whether the blackleg resistance ranking order of varieties is significantly different where variety resistance to the second pathogen alters the incidence or severity of the second disease. However, it is possible that variety resistance to other pathogens could substantially affect blackleg development. Kelly *et al.* (unpublished) found in 2 years that seed tubers, naturally infected with *Eca*, when inoculated with *P. infestans* prior to planting produced plots with significant incidences of blackleg whereas none developed in plots grown from non-inoculated seed tubers of the same stock. Plants produced from gangrene infected seed were more prone to infection by *Eca* (Griffith *et al.*, 1974). Davis *et al.* (1983) reported that *Fusarium* spp. in combination with *Eca* on seed pieces increased seed piece decay and also the incidence of blackleg. An earlier report (Boyd, 1972) stated that blighted tubers (and also those with dry rot or pink rot) were more likely to develop secondary bacterial soft rot. Sicilia *et al*. (2002) confirmed the predisposition to soft rotting of tubers infected with blight. They observed that *P. infestans* promoted tuber rotting by *Eca* much more than *Fusarium coeruleum* or *Phoma foveata*. The impact of any second pathogen on blackleg development will be determined to a large extent by its incidence and severity of infection.

4.2.1.2.3 Relationship of inoculum level and environment to effectiveness of host plant resistance

The incidence and severity of blackleg is affected by soil moisture content (Perombelon *et al.* 1989) temperature (Aleck & Harrison, 1978; French & de Lindo, 1979; Molina & Harrison, 1980; Perombelon *et al.*, 1987) and soil nutrient status (Graham & Harper, 1966) as well as variety resistance and the synergistic or antagonistic effects of other pathogens and micro-organisms (Perombelon *et al.*, 1989). However, the number of viable cells of *Eca* contaminating the seed tubers at planting is of primary importance, as first proposed in the 1970s (Perombelon, 1973; Maas Geesteranus & Vruggink, 1976; Aleck & Harrison, 1978; Perombelon *et al.*, 1989; Bain *et al.*, 1990). Bain *et al.* (1990) observed that for the blackleg susceptible varieties Desiree, Maris Bard, Spunta and Estima, blackleg incidence in two countries in 2 years was significantly correlated with the number of *Eca* on the seed tubers. This was not the case for the more resistant variety Pentland Crown. For this variety the incidence of blackleg was too low to test the relationship. However, it is interesting to note that at one site in one year the relationship between seed contamination and blackleg was very similar for P. Crown and the more susceptible varieties. This suggests that variety resistance can occasionally not be effective if environmental conditions strongly favour development of the pathogen.

4.2.1.2.4 Integration of plant resistance with other control measures

Varieties that are highly resistant to blackleg are not yet widely available. Therefore, at present, the control of blackleg combines variety resistance with cultural control methods to avoid tuber contamination (e.g. use of seed of limited field generations, use of diagnostic tests to identify seed stocks with no, or very few, *Eca* or *Echr*, early desiccation and harvest, and decontamination of equipment between seed stocks) and reducing the numbers of erwinias on the seed after harvest (e.g. positive ventilation of seed stocks, avoidance of condensation on tuber surfaces and refrigerated storage of seed). Seed stocks with high numbers on blackleg bacteria should not be grown in fields likely to have prolonged periods with a high soil moisture content.
In spite of considerable research effort over many decades, control of erwinias on seed tubers using bactericides has not been possible because of the difficulty of accessing the erwinias in tuber lenticels and healed wounds. Erwinias can survive potato storage in a latent state in the lenticels of tubers and also in healed wounds (Perombelon, 1992).

4.2.2 Brown rot (*Ralstonia solanacearum*; synonyms: *Pseudomonas solanacearum, Burkholderia solanacearum*)

Strains of *R. solanacearum* have been informally grouped into 5 races based primarily on host affected (Buddenhagen, 1986) or into 5 biovars based on the catabolism of certain sugars and sugar alcohols (Haywood *et al*., 1990). The common strain in Europe is race 3 which is a variant of biovar 2, known as biovar 2A (Stead *et al*., 1996). This strain has a narrower host range including potatoes, woody nightshade (*Solanum dulcamara*) and black nightshade (*Solanum nigrum*) and is adapted to lower temperatures. It is responsible for brown rot outbreaks in Europe and North Africa.

In GB, where *R. solanacearum* is a quarantine organism (http://www.defra.gov.uk/planth/checklst/solanace.htm), consideration of resistance in potato varieties is of less relevance than preventing its introduction. Plant health orders (e.g. http://www.defra.gov.uk/planth/phnews/legi.htm) specifically restrict the import of seed and ware into GB carrying the pathogen and statutory notification requirements exist (currently for Germany, The Netherlands and Poland). From these countries imported seed tubers require to be tested for presence of the organism and are also tested in GB before planting. Because of its high health status Scotland operates a voluntary notification arrangement for all seed potato imports from countries other than those where statutory notification exists (http://www.scotland.gov.uk/Topics/Agriculture/plant/17937/newpotatobrownrot/notificationbrownrot).

Because of its absence in the UK there is no information available on the relative susceptibility of commonly grown GB varieties to brown rot. Differential resistance (and tolerance) to *R. solanacearum* has been identified in varieties of *Solanum tuberosum* (e.g. Weingartner & Shumaker, 1987; Fahmy & Mohamed, 1990) and evaluation of host resistance is carried out where the pathogen is endemic (e.g. USA)

In countries where the organism is endemic host resistance can play a major part in restricting the development of the organism during production. However, in these countries host resistance alone cannot ensure eradication of the disease.

Whilst symptoms may be suppressed, a large population of the pathogen may build up on resistant varieties and they may transmit the pathogen. In a field trial in Germany, inoculated potato plants showed a high degree of latent infection in stems and tubers (Abdel-Kader *et al*., 2000). Cultivation of the progeny resulted in a decrease in infection. Maciel *et al.* (2004) found no relationship between the population density of the pathogen and resistance of 7 varieties in Brazil. This has implications for the spread of disease. In the UK, if infected seed of a resistant or tolerant variety is imported and planted it is possible for the pathogen to reach high levels without symptom expression. The disease may thus go un-noticed with implications for further spread. Additionally, artificial fertilisers may decrease disease incidence (Fahmy & Mohamed 1990) thus masking the presence of the pathogen. *Solanum phureja* is more resistant than *S. tuberosum* but has been shown to transmit the pathogen.

In the UK, current control measures, governed by national and European Union plant health legislation, rely on i) accurate detection and reporting of brown rot outbreaks and distribution of the pathogen ii) prevention of importation of potatoes from known infected areas and iii) restrictions on use of infested land and irrigation water for potato production (Stead *et al*. 1996). Avoidance and control of bacterial plant diseases when using pathogen tested (certified) planting material has been reviewed by Janse and Wenneker (2002).

4.2.3 Common scab (*Streptomyces scabiei*; synonym. *Streptomyces scabies*)

There are 10 validly named pathogenic *Streptomyces* species for which pathogenicity has been clearly demonstrated in the recent literature. *S. scabiei* is perhaps the most widespread and common. As species other than *S. scabiei* are not recorded in GB, this review is confined to this species.

4.2.3.1 Interpreting published disease and pest resistance

Variety resistance is an important tool in controlling common scab of potatoes and tables of variety resistance are published, for example by the National Institute of Agricultural Botany (NIAB) (Anon., 2004). Mishra and Srivastava (2001) working in India consider that satisfactory and eco-friendly control cannot be achieved without genetic resistance, which is not only cheaper but also stable and thus acceptable to farmers. Rich (1983) is unequivocal: "The most effective method of scab control is to plant resistant cultivars".

Numerous varieties with different levels of resistance have been identified worldwide through useful field screening programs. Both durable horizontal resistance (Gergely *et al*., 2003) and simply inherited sources of resistance (Murphy *et al*., 1995) appear to be available. *Solanum phureja* has been identified as a useful source of scab resistance in the production of *S. tuberosum* varieties (Maine *et al*., 1993) although resistance levels tend to decrease on backcrossing with *S. tuberosum*.

Although many new potato varieties come with resistance to common scab, no commercially important potato varieties are immune to infection and the level of resistance is usually not high. One early study of resistance levels amongst 684 potato varieties from a world collection indicated that none was immune and only 1.3% were strongly resistant. The remainder could be allocated to seven categories of lower resistance, and almost 75% of the total showed intermediate levels of resistance (Zadina *et al*., 1975).

In multi-location trials on 23 potato clones in the USA (Haynes *et al*., 1997) significant environment, genotype and genotype x environment effects on heritability of resistance to common scab were observed. It was concluded that scab resistance was unstable whereas scab susceptibility was stable across environments and that new sources of scab resistant germplasm would be required to obtain genotypes with high levels of scab resistance that are stable across environments. The fact that even resistant varieties will become infected given high inoculum levels or favourable environments, means that currently available resistant varieties will best be used as part of an integrated control system.

The value of common scab resistance in different varieties may vary with a number of variables including *Streptomyces* species or isolate, soil moisture content and soil pH (Haynes *et al.*, 1997). Mishra and Srivastava (2001) found good correlation between the resistances of 27 potato varieties to common scab over two years. Hooker (1981) reports that physiological specialisation of subculture isolates of *S. scabies* has been demonstrated in glasshouse trials. However, selective pathogenicity by biotypes in the field is of little importance and relative resistance of potato varieties remains relatively constant over a wide range of natural soil populations.

The limits of currently available sources of genetic resistance to scab are demonstrated by the French fry processing industry in Tasmania where incidence of common scab has steadily increased since the late 1980's. This is despite Russet Burbank, which is recognised as possessing moderate

resistance dominating production (more than 80%) (Wilson, 2001a). Trials by Wilson (2001a) have demonstrated differences in the resistances of clones of Russet Burbank to common scab, although differences between varieties were greater than differences between the Russet Burbank clones.

Future development of higher levels of resistance will largely depend on an increased understanding of the host-pathogen interaction. Recent research on the mechanisms and genetic control of pathogenicity in the scab-forming streptomycetes is relevant in this respect. Pathogenicity of *Streptomyces* spp. on potato is largely dependent on the ability of strains to produce at least one of two major phytotoxins, thaxtomins A and B (Lawrence *et al*., 1990; King *et al*., 1991). Interestingly, glucosylation appears to be a mechanism of thaxtomin A detoxification and is related to scab resistance and susceptibility in potato (Acuna *et al*., 2001). Therefore, there may be useful marker genes in potato related to an ability to glucosylate the toxins which will aid in effective selection of resistant genotypes. In a more direct approach, scientists at the Tasmanian Institute of Agricultural Research are aiming to develop extreme resistance to common scab within existing potato varieties through exposure of cell lines to the toxins and regeneration of potato plants from those which survive the treatment (Wilson, 2001a; Wilson 2001b). Goto (1981) concluded that the concentration of reducing sugars in peel extracts is positively correlated with scab severity and may therefore be a useful indicator during screening of new breeding material for resistance.

The ability to cause scab symptoms is believed to be inherited between *Streptomyces* spp. by horizontal transfer of a pathogenicity island containing the gene nec1 (which is involved in pathogenicity and physically linked to the thaxtomin A biosynthetic genes) and a transposase pseudogene ORFtnp (Bukhalid *et al*., 1998; Healy *et al*., 1999). Resistance in potato genotypes to thaxtomin activity may therefore be expected to be broad-based across all scab-forming streptomycete strains. However, phytotoxin production was found to vary differently between scab-forming isolates of *Streptomyces scabiei* and *acidiscabiei* in response to changes in pH, temperature and calcium or phosphate availability (Natsume *et al*., 2001). It may therefore be important to ensure stability of resistance in potato across the range of variability within the pathogenic streptomycetes.

4.2.3.2 Use of resistance as a sole method of disease control

As no variety is immune to common scab, the reliance on varietal resistance alone depends on the market outlet. For sectors of the processing industry where peel is removed before processing, common scab can be tolerated provided the lesions do not penetrate deeply. Thus, in these processing sectors, where varieties are resistant, there is a general acceptance that varietal resistance alone is sufficient.

By contrast, where skin finish is critical to achieving the market standards, such as in the washed pre-pack sector and certain parts of the processing sector, varietal resistance as the sole method of disease control represents a high risk. Achieving acceptable levels of skin finish largely depends on weather conditions during the critical phase of tuber initiation being wet and not conducive to infection. Thus in the sectors of the industry where skin finish is critical, irrigation is considered vital to limit common scab. This applies even to varieties with a relatively high resistance rating (7, 8 or 9) as the market standards are so high.

4.2.3.3 Assessing risks when utilising host plant resistance

The risk will depend on the market outlet and knowledge of the history of the field. Some fields are known to exhibit suppressiveness and develop little common scab even in dry seasons where no irrigation is applied. The nature of this suppressiveness is largely unstudied although recent research in Japan has suggested it is related to the concentration of water soluble aluminium

(Mizuno *et al.*, 1998; Mizuno *et al.*, 2003). The level of aluminium availability is much higher in soils with low pH's (5 or less) and this concurs with experience that common scab is less in acid soils. However, whether the level of water soluble aluminium explains suppressiveness in soils of higher pH's in the UK is largely unknown. The influence of pH on common scab means that soil pH can be taken into account when assessing risk. Where soil pH is low (but not as low as 5.0) artificial and temporary reduction of soil pH has been used to reduce the risk of common scab.

4.2.3.4 Relationship of inoculum level and environment to effectiveness of host plant resistance

Studies on the relationship of inoculum level to common scab development are limited. They are discussed in a recent review (Stead & Wale, 2004; Wale & Sutton, 2004) and the conclusion drawn that soil-borne inoculum was more important than tuber-borne inoculum. This will depend on several factors including the relative levels of inoculum on tubers and in the soil and the level of antagonists to the pathogen. A recent study using pasteurised soil demonstrated that where soil inoculum was low or insignificant, tuber-borne inoculum would be important (Wang & Lazarovits, 2005).

The effect of environment, and particularly soil moisture on the development of common scab was ably demonstrated over 30 years ago and has been the foundation of irrigation scheduling ever since (Lapwood *et al*., 1973). The role of antagonists in suppressing the common scab pathogen has received scant study to date.

4.2.3.5 Integration of plant resistance with other control measures

A recent review (Stead & Wale, 2004; Wale & Sutton, 2004) investigated non-water control measures for the control of common scab. It was concluded that no method was as consistent as modifying soil moisture content at critical times in reducing common scab. In addition, besides varietal resistance, other non-water control measures tended to be inconsistent. As indicated above, some non-water control measures have indirect effects on common scab. Thus treatments that reduce soil pH (e.g. fertilisers such as ammonium sulphate, high levels of pig slurry or additives such as sulphur) may have an effect on common scab. A recent study has suggested that sulphate fertilisers may also act by increasing the biodiversity of organisms in the root zone thus encouraging antagonists to the pathogen (Sturtz *et al*., 2004).

Thus, there may be non-water control measures that can be used in addition to varietal resistance or irrigation that may enhance control of common scab. However, their use in an integrated programme has been little studied.

Studies on control of common scab have been hampered by a restricted ability to measure the pathogen population and their antagonists and understand how water and non-water control measures work. In consequence, identifying how resistance can best be utilised, for example by improvements in irrigation scheduling to reduce water use further, have not been carried out.

4.2.4. Ring rot (*Clavibacter michiganensis* subsp. *sepedonicus* (Spieck. *et* Kotth.) Davis *et al*.; synonym *Corynebacterium sepedonicum*)

In GB, where *Clavibacter michiganensis subsp. sepedonicus* is a quarantine organism (http://www.defra.gov.uk/planth/checklst/solanace.htm), consideration of resistance in potato varieties is of less relevance than preventing its introduction. Plant health orders (e.g.

http://www.defra.gov.uk/planth/phnews/legi.htm) specifically restrict the import of seed and ware into GB carrying the pathogen and statutory notification requirements exist. From these countries imported seed tubers require to be tested for presence of the organism and are also tested in GB before planting. Because of its high health status Scotland operates a voluntary notification arrangement for all seed potato imports from countries other than those where statutory notification exists
(http://www.scotland.gov.uk/Topics/Agriculture/plant/17937/newpotatobrownrot/notificationbrownrot).

Because of its absence in the UK there is no information available on the relative susceptibility of commonly grown GB varieties to ring rot. In any case it is considered that resistance of any substance to *Clavibacter michiganensis subsp. sepedonicus* is not available in cultivated varieties of *Solanum tuberosum* (Janse & Wenneker, 2002) and cannot be practically used to control the disease. Despite this breeding and screening to select resistance is commonly reported in the scientific literature and immunity has been identified in some accessions of the tetraploid *Solanum acaule* (Kriel *et al.*, 1995).

In countries where the organism is established, the use of zero tolerance for disease in seed potatoes plays the most important role in reducing the impact of disease (De Boer & Slack, 1984). Whilst host resistance is not a feature of disease control, the differential appearance of symptoms in stocks grown from infected or contaminated seed can result in establishment of the pathogen before its occurrence is recognised. The latency of symptom development adds weight to the need for effective testing of all seed entering a ring rot free country. Symptom expression is affected by the strain of *Clavibacter michiganensis subsp. sepedonicus* (Bishop & Slack, 1982a), inoculum concentration (Bishop & Slack, 1982a; Nelson, 1982; Starr, 1947), variety (Bishop & Slack, 1982a; Bond & Covell, 1950; De Boer & Slack, 1984; Manzer & Mckenzie, 1988; Sletton, 1985) and environmental conditions notably temperature (Bishop & Slack, 1982b; LaChance & Genereux, 1963; Logsdon, 1967; Nelson & Kozub, 1983 & 1987; Sherf 1944).

A series of detailed experiments in the USA by Westra & Slack (1994) and Westra *et al*. (1994) showed that inoculum dose was positively correlated with expression of foliar and external tuber symptoms and affected by location. However, the relative effect of variety on the relationship of inoculum dose and disease expression was constant across a range of locations, variety affecting the time of onset of symptoms. The effect of variety was not consistently related to maturity class (Westra *et al*., 1994). The impact of variety on development of symptoms is unknown for GB varieties. In Canada, disease symptom development was examined in 108 potato varieties inoculated uniformly with the *C. michiganense subsp. Sepedonicus*. Considerable variation in the incidence of foliar and severity of tuber symptoms was observed in all varieties, from no foliar or tuber symptoms to complete necrosis of the haulm or complete breakdown of tubers (Kawchuk *et al*., 1998). In an earlier test on 156 accessions from a collection selected as highly resistant to ring rot based on symptom expression, 57 yielded plants with 100% plants producing symptoms. The remaining 99 accessions yielded some plants which failed to show symptoms but even with these detectable numbers of *C. michiganense subsp. sepedonicus* were recorded (Kurowski & Manzer, 1992).

In the UK, current control measures, governed by national and European Union plant health legislation, rely on i) accurate detection and reporting of ring rot outbreaks and distribution of the pathogen ii) prevention of importation of potatoes from known infected areas and iii) strict hygiene measures (Stead *et al*., 1996). Avoidance and control of bacterial plant diseases when using pathogen tested (certified) planting material has been reviewed by Janse and Wenneker (2002).

4.3. Fungal and fungal-like diseases

The European Cultivated Potato Database (www.europotato.org/menu.php) has been used to provide an overview of the resistance of potato cultivars to the fungal and fungal like diseases discussed below. Ratings for a number of potato cultivar characteristics, including disease resistance, are collated from many European sources and presented on a searchable database. Table 4.6 shows basic data on resistance to fungal diseases (other than potato blight) derived from this database in October 2006.

TABLE 4.6 RESISTANCE RATINGS TO FUNGAL DISEASES OF POTATOES (OTHER THAN POTATO BLIGHT)ON THE EUROPEAN CULTIVATED POTATO DATABASE.

	Dry rot	***Gangrene***	***Powdery scab***
Number of records	696	464	454
1. % Very low	0.7	3.7	3.5
2. % Very low to low	1.9	4.3	1.1
3. % Low	10.5	20.0	9.0
4. % Low to medium	15.9	15.7	12.3
5. % Medium	26.7	25.2	27.3
6. % Medium to high	23.7	14.2	24.2
7. % High	15.8	11.0	16.1
8. % High to very high	3.9	3.2	4.6
9. % Very high	0.9	2.6	1.8
Mean Score	5.25	4.73	5.30
Median score	5	5	5

The resistance ratings for all these diseases follow a fairly normal distribution with the mean around medium resistance. Between 15% and 25% of assessments suggest high resistance or better to the appropriate disease.

The European Cultivated Potato Database includes separate assessments for resistance to potato blight on foliage and tubers. The assessments are further broken down according to the method of assessment (unspecified, laboratory, artificial inoculation in field and natural inoculation in field). The distribution of these assessments is shown in tables 4.6 and 4.7.

TABLE 4.6 RESISTANCE RATINGS TO POTATO BLIGHT ON FOLIAGE ON THE EUROPEAN CULTIVATED POTATO DATABASE WITH DIFFERENT ASSESSMENT METHODS.

	Method unspecified	***Laboratory test***	***Artificial inoculum field***	***Natural inoculum field***
Number of records	3099	591	146	49
1. % Very low	1.0	0	0	2.0
2. % Very low to low	1.6	5.4	9.6	0
3. % Low	18.4	23.7	11.6	4.1
4. % Low to medium	12.6	22.2	19.2	6.1
5. % Medium	36.1	18.8	28.8	26.5
6. % Medium to high	12.4	15.1	10.3	10.2
7. % High	15.9	11.5	13.7	30.6
8. % High to very high	1.1	2.9	5.5	12.2
9. % Very high	0.9	0.5	1.4	8.2
Mean Score	4.93	4.63	4.88	6.18
Median score	5	4	5	7

TABLE 4.7 RESISTANCE RATINGS TO POTATO BLIGHT ON TUBERS ON THE EUROPEAN CULTIVATED POTATO DATABASE WITH DIFFERENT ASSESSMENT METHODS.

	Method unspecified	***Laboratory test***	***Artificial inoculum field***	***Natural inoculum field***
Number of records	2645	581	146	51
1. % Very low	1.1	0.5	0	2.0
2. % Very low to low	1.4	9.1	0	15.7
3. % Low	7.4	18.6	2.7	13.7
4. % Low to medium	6.7	12.4	2.7	17.6
5. % Medium	35.5	15.3	15.1	2.0
6. % Medium to high	11.3	13.8	18.5	5.9
7. % High	29.3	14.3	28.8	3.9
8. % High to very high	4.9	13.1	24.7	29.4
9. % Very high	2.3	2.9	7.5	9.8
Mean Score	5.64	5.14	6.72	5.41
Median score	5	5	7	5

For resistance to foliage blight the results of the unspecified, laboratory and artificial inoculation in the field methods are similar. However, the assessments based on natural inoculation in the field generally suggest higher levels of resistance. It is not clear if this is meaningful, or simply relates to the relatively small number of assessments carried out by this technique.

For resistance to tuber blight the results differ more widely depending on the method of assessment. The unspecified results give most assessments between medium (5) and high (7); laboratory testing gave a fairly even distribution of results between low (3) and high to very high (8); artificial inoculation in the field suggested a higher level of resistance with most observations either of high (7) or high to very high (8) resistance. There were relatively few observations based on natural inoculation in the field but these were clustered between very low to low (2) and low to medium (4) and at high to very high (8).

Regardless of the assessment technique at least 30% of assessments for resistance to tuber blight were of high or better resistance.

4.3.1. Black dot (*Colletotrichum coccodes*)

Until recently, black dot, caused by *Colletotrichum coccodes*, was considered to be a disease of little importance. The growth in demand for washed fresh produce has meant that blemish diseases are now a serious problem. One reason, for black dot being overlooked in the past is that its symptoms are similar to those of silver scurf so the degree of infection may have been underestimated (Errampalli *et al*., 2001).

Black dot is both a seed and soil-borne pathogen, but in the UK the soil-borne inoculum is more important in terms of causing disease (Wale *et al*., unpulished). Initial infection by *C. coccodes* occurs on developing tubers in the field. Once in store, further symptoms of black dot can occur (Peters *et al*., 2005).

4.3.1.1 Interpreting published disease and pest resistance

As the interest in black dot is recent, little work has been done on breeding for resistance. The work that has been done has focused on identifying differences in susceptibility to black dot, in field experiments (Read, 1991) and development of methods for screening for resistance (Hilton *et al*., 1999; Gans *et al*., 2002). Testing for resistance has developed using assays on growing plants (Hilton *et al*., 1999; Gans *et al*., 2002; Thomas *et al*., 2005).

Early information on resistance to black dot relied on data obtained from field experiments. In the UK differences between 15 varieties to susceptibility to black dot were observed where healthy seed was planted at sites where potatoes had previously grown (Read, 1991). To ensure symptoms developed these workers added additional inoculum to the soil in some experiments. Desiree and Maris Piper were the most susceptible, whilst Romano and Cara were the most resistant. In Israel, field experiments showed that Cara and Nicola were less susceptible than Alpha, Desiree and Agria (Tsror *et al*., 1999)

Further, information on variety resistance has been obtained from surveys on the incidence of black dot in the national crop in England and Wales (Read *et al*. 1995) with Cara being the least affected variety and Maris Piper being the most affected. In the USA, black dot symptoms were more common on tubers from thin-skinned, than russett-type varieties (Hunger & McIntyre 1979). However, as these types are not popular for table consumption in the UK so this source of resistance is unlikely to prove a useful control option.

In order to test a larger number of varieties cheaply without having to perform expensive field trials simple assays had to be developed (Hilton *et al*., 1999; Gans *et al*., 2002). These tests involve planting healthy seed tubers into soil amended with inoculum of *C. coccodes*. Pots are placed in a glasshouse or growth cabinet and grown to maturity, harvested and then assessed for disease. These tests used either used inoculum of *C. coccodes* grown on sand-corn meal media (Hilton *et al*., 1999) or microsclerotia (Gans *et al*., 2002) added to the compost at the start of the test. These simple tests have demonstrated differences in susceptibility between varieties. In general these tests have proved reliable with ranking of varieties between tests being consistent. However, in some experiments ranking of varieties between tests has been inconsistent (Thomas *et al*., 2005). For recent independent variety testing in the UK, testing of resistance to black dot now uses a standard

inoculum concentration. This has resulted in variation between replicate pots being reduced (Carnegie *et al*., 2006).

Although these tests give good differentiation between varieties much of the variation is due to physiological maturity. As with testing for resistance to silver scurf in a growing crop, earlier maturing varieties tend to be more susceptible than maincrop types (Read, 1991). In a study, on initial symptom development of black dot on roots, stems and tubers more symptoms developed on the second early variety, Bintje compared with the maincrop variety Roseval (Andrivion *et al*., 1998). These authors believed this was due to the more vigorous growth of Roseval which enabled stems and stolons of plant parts to grow away from the source of inoculum.

4.3.1.2 Use of resistance as a sole method of pest and disease control

Although no immunity exists some good levels of resistance are available among commercial varieties. Among commercial varieties in the UK, Cabaret and Celia are rated with 8 and Saxon 7. The majority of varieties are more susceptible with Estima scoring 5, Maris Piper 4 and Lady Christl, Isle of Jura and Hunter rated at 2 (Anon. 2005). Although some good resistance does exist, the lack of immunity means that black dot can still occur on resistant varieties if favourable conditions exist. Therefore resistance is best utilised in conjunction with other control measures. However, the majority of UK pre-pack varieties do not yet have resistance rating for black dot.

To use resistance as the sole method of control the level of resistance to black dot would have to be improved. Among some of the new varieties tested as part of the IVT testing in 2005, both Annabelle and Vivaldi were more resistant than the resistant control Saxon (Carnegie *et al*., 2006). It has also been shown that resistance to black dot is a heritable character and that by crossing commercial varieties more resistant progeny can be produced (Hilton *et al*., 1999).

4.3.1.3 Assessing risks when utilising host plant resistance

Where a resistant variety is planted at a site with a high concentration of soil-borne inoculum black dot can still develop. Further, disease will be encouraged if wet conditions exist during the growing season (Wale *et al*., unpublished)

4.3.1.4 Relationship of inoculum level and environment to effectiveness of host plant resistance

Although seed infected with *C. coccodes* can cause black dot on daughter tubers, it is soil-borne inoculum which is more important in terms of causing disease. In field experiments, where seed tubers with different amounts of black dot were planted at a site not contaminated with *C. coccodes* little effect was observed on incidence and severity of disease on daughter tubers at harvest. In contrast, a strong relationship was found in controlled environment studies and a crop monitoring exercise between degree of soil contamination and disease on daughter tubers (Wale *et al*., unpublished). In 2004 and 2005 using data from 4 field trials the degree of soil-borne inoculum measured using real-time PCR (Cullen *et al*., 2002) was compared to the incidence of black dot on daughter tubers at harvest. A significant association was observed in both years ($R^2 = 0.81$ – 2004 and $R^2 = 0.82$ – 2005) although the angle of slope differed between years and it is believed that this was due to the more favourable for disease development in 2004. Although little disease developed on the more resistant variety Sante where little inoculum existed an incidence of 70% black dot on daughter tubers were observed where 100 pg DNA / g soil were found in 2004. In 2005, although higher levels of inoculum were found less disease occurred due to the drier soil conditions.

In general, it is believed that black dot is a disease favoured by warm and damp conditions (Lees & Hilton, 2003). In studies performed under controlled environmental conditions disease incidence was greatest at harvest when plants were grown at 22°C than at 18°C. Further disease developed when the soil was kept damp instead of dry (Wale *et al*., unpublished)

4.3.1.5 Integration of plant resistance with other control measures

As the predominant source of inoculum in the UK is soil-borne a knowledge of which fields are at risk from the disease is vital. A real-time PCR assay has been developed (Cullen *et al*., 2002) and subsequently modified (Wale *et al*., unpublished) which has enabled *C. coccodes* inoculum to be quantified. Using this method on soil prior to planting, 45 and 34 fields in 2004 and 2005, respectively were assessed for contamination and related to the incidence of black dot at harvest. In both years where the results of the soil test were greater than 75 pg DNA / g soil then the incidence of black dot was moderate to high, with the risk of black dot occurring being lower below this figure. Although this is only an arbitrary figure at present the concept of setting a threshold figure would greatly assist with deciding which varieties to grow in a particular field. Thus at a site where there is a high risk of black dot developing a more resistant variety could be grown, and conversely at a low risk site a more susceptible variety could be grown, although care must be taken not to re-introduce the pathogen on the seed.

Integrating control measures provides the best opportunity of controlling this disease where the soil is contaminated. In a series of field experiments in England and Scotland in 2004 and 2005 variety choice, azoxystrobin application in furrow at planting, early harvesting and reducing irrigation all reduced both the incidence and severity of black dot. In 2004, variety choice caused the largest reduction in disease, compared with the other measures, when the resistant variety Saxon and was compared with the susceptible variety Maris Piper were grown. Disease reduction was less in 2004 when the intermediate variety Sante was compared with Maris Piper (Wale *et al*. unpublished).

4.3.1.6 Avoidance of resistance breaking races developing in populations of potato pests and diseases

No reports of isolates of *C. coccodes* overcoming host resistance have been reported. As only partial resistance exists to black dot it is unlikely that resistant isolates will occur.

4.3.2 *Rhizoctonia solani* (Perfect stage: *Thanetophorus cucumeris*)

Infection by *Rhizoctonia solani* causes a number of diseases of potato including black scurf on the surface of tubers and stem and stolon canker in the growing crop. Black scurf is characterised by the formation of irregular black sclerotia which can range in size from 0.5 to 5 mm in size. The presence of black scurf on fresh pre-pack tubers reduces quality and can result in crops being rejected. Similarly, black scurf can affect certification and marketability of seed potatoes.

Stem canker develops prior to emergence and is evident as dark brown elongated lesions on stems. Stolon canker can occur throughout crop growth and in severe cases stolon pruning can occur. This can result in an uneven tuber size distribution. The fungus is also capable of causing malformation of tubers. *R. solani* can be both seed- and soil-borne.

The species *R. solani* is complicated by the existence of several intraspecific sub groups, which are often related to host plants. The main criterion to delineate these groups is hyphal anastomosis (fusion) (Parmeter *et al*., 1969). Although a number of anastomosis groups (AG's) affect potatoes

AG-3 is believed to be the most important. In a survey in the UK, AG-3 was identified as the predominant group (92%) in potato crops, followed by AG 2-1 (7%) and AG-5 (less than 1%) (Woodhall, 2005). When assessing resistance to stem canker and black scurf workers have focussed on AG-3.

4.3.2.1 Interpreting published disease and pest resistance

Field experiments evaluating resistance to *R. solani* have been performed either at sites naturally contaminated, amended with *R. solani* or where seed has been inoculated.

At a naturally contaminated site, severity of stem and stolon infection was found to be more severe in two early varieties than two late ones (Scholte, 1989). They believed this was due to late maturing varieties emerging much later and hence escaping *R. solani* attack. In field experiments where soils were naturally contaminated with *R. solani* in Ireland (Dowley, 1972) and in Scotland (Parker, 1986) significant differences in disease severity were observed among forty and sixteen varieties respectively with early varieties again showing more disease. Gilligan *et al*. (1996) also working in soil naturally contaminated with *R. solani* found rapid emergence enabled plants to avoid infection. In later experiments these workers pre-sprouted seed tubers, so that differences in date of emergence were small and found significant differences between varieties not related to physiological maturity

When seed tubers of 10 varieties were planted in soil amended with *R. solani* no significant differences in stem lesions or shoot pruning were observed (Chand & Logan, 1982).

Over two years, 1983 and 1984, Hide *et al*. (1989) inoculated seed of 12 varieties with *R. solani*. Mean disease scores of stem and stolon infection were greater for five early than seven maincrop varieties. However, within each of these groups significant differences between varieties were observed with Maris Peer showing a consistently lower mean disease score than early varieties; whilst Record showed consistently lower mean disease than other maincrop varieties. From these results it was concluded that although physiological maturity does affect severity of *R. solani* there is a significant genetic component. In the USA, artificially inoculated field experiments have also revealed differences in susceptibility to black scurf with russett skinned varieties showing less disease than white skinned types (Leach & Webb, 1993). For example, incidence of malformed or with sclerotia was an average of 13.7 on seven table varieties and 10.1 on eight russett genotypes.

Although varietal differences for both stem canker and black scurf have been established the relationship between the two diseases is inconsistent. When seed of 10 varieties was planted in a field experiment in soil amended with *R. solani* all but Desiree and Ulster Sceptre showed a significant correlation between stem canker severity and incidence of black scurf (Chand & Logan, 1982). A more extensive investigation found that when seed of 12 varieties was inoculated with *R. solani* and planted in the field, those varieties with most stem canker did not have most black scurf (Hide *et al*., 1989). It can be concluded that the mechanisms of resistance to *R. solani* on the stem and tuber are different and should be assessed separately.

Although differences in susceptibility to Rhizoctonia diseases in potato have been observed in naturally and artificially inoculated field experiments such methods are time consuming when testing a large number of varieties or breeding lines. No rapid tests have been described for assessing stem canker, although Kyritsis & Wale (2002a) describe an experiment performed in a growth cabinet where soil was amended with *R. solani* and differences in stem canker incidence and severity were detected between 7 varieties when plants were assessed soon after emergence. Such a test could be used to screen varieties. There has been no evaluation of varietal resistance to other

disease symptoms resulting from infection by *R. solani* (elephant hide, tuber distortion, dry core etc.).

More experiments have been done to screen for resistance to black scurf *in vitro*. These tests, have involved inoculating a sand and cornmeal (Hilton *et al*., 1999) or a vermiculite and cornmeal (Gans *et al*., 2002) mixture with isolates of *R. solani* and incubating for 6 weeks. The inoculum is then added to compost into which healthy seed tubers of test varieties are planted. Plants are then grown to maturity and black scurf is assessed on daughter tubers after harvest. These have produced significant differences in severity between varieties but results have not always proved repeatable (Hilton *et al*., 1999). In independent variety trials in the UK ranking of varieties between tests was consistent between 1999-2001, but inconsistent between 2002-2004 (Thomas *et al*., 2005). In IVT testing in 2005-2006 the concentration of inoculum added to the compost has been standardised and it is hoped that this will improve consistency between tests (Carnegie *et al*., 2006).

4.3.2.2 Use of resistance as a sole method of pest and disease control

Some useful resistance is available among commercially grown GB varieties with a number having good resistance (ratings of 7 and above) (Anon. 2005). However, there is a wide variation in resistance to black scurf from the highly susceptible variety, Rembrandt (rated 1) several varieties with good resistance (Lady Christl, Lady Felica). The majority of varieties have intermediate resistance with King Edward and Maris Piper scoring 6 and Nadine 7 (Anon. 2005). In contrast, the variety Sante which is widely used in organic production is very susceptible (rating of 3).

However, all varieties can be infected to some degree and the testing methodology only describes relative resistance to black scurf. Thus to rely entirely on host resistance is not advisable. However, using this resistance as part of an integrated programme of control would be valuable.

No independent variety testing for stem canker is performed at present and as it cannot be simply be related to black scurf it is not possible to use black scurf resistance rating to judge reaction of commercial varieties to risk of stem canker. However, among varieties that have been examined in the past all can be considered to be susceptible. When seven varieties were compared for their susceptibility to stem canker under controlled environmental conditions only marginal differences were seen with Pentland Dell being most resistant to stem canker (incidence of 48%) and Estima being most susceptible (incidence of 59%) (Kyritsis & Wale, 2002a). Other reports also suggest that differences between varieties in susceptibility to stem canker are limited (Hide *et al*., 1989). It is suggested that the value of using resistance to stem canker is limiting.

4.3.2.3 Assessing risks when utilising host plant resistance

When discussing resistance to stem canker and black scurf in the UK most workers have focussed on AG-3. However, a number of other AGs have been associated with potato plants including AG 1 (Chang & Tu, 1980), AG 2 (Chand & Logan, 1983), AG 4 (Anguiz & Martin, 1989), AG 5 (Carling & Leiner, 1990) and AG 8 (Balali *et al*., 1995). In general AG-3 is more pathogenic on potatoes than other AG's. Balalili *et al*. (1995) showed that although AG 3, AG 4, AG 5 and AG8 all cause stem and root cankers, only AG 3 and AG5 can cause black scurf.

At present there is no indication how isolates from these other AG's perform against varieties with good host resistance to AG 3.

4.3.2.4 Relationship of inoculum level and environment to effectiveness of host plant resistance

The ability for disease to occur is dependent on availability of inoculum, whether that be seed or soil, the status of the crop, the susceptibility of the host and a set of suitable environmental conditions for infection and disease development.

Both seed and soil-borne inocula can be important. Seed-borne inoculum is probably the more important of the two, but with shorter rotations fields have become increasingly contaminated and soil-borne inoculum has increased in importance. Due to the proximity of the fungus on seed potatoes to the growing stem, early symptoms of canker are most likely to come from this source (Adams *et al*., 1980). Selecting seed stocks with as little *R. solani* inoculum as possible and treating seed with a fungicide application will reduce serious damage. In contrast, soil-borne inoculum must grow towards the developing plant to cause infection and hence has been associated with later infection. Increasing soil contamination will increase the likelihood of all plant parts becoming infected.

Resistance to infection is also dependent on the status of the crop. Stems and stolons are most susceptible to pruning before emergence (Hide *et al*. 1985). Therefore planting chitted seed or early varieties in al highly contaminated sites to encourage rapid emergence will diminish to likelihood of stem pruning or canker.

R. solani AG-3 can infect potato plants in a wide range of environmental conditions. Infection can occur at soil temperatures from 9-27^{o}C, with an optimum of 10-15^{o}C (Kyritsis & Wale, 2002b) and moisture content of 40%WHC (Kyritsis & Wale, 2002b). In contrast, other AG's have more specific requirements and it is believed this is one reason why AG-3 predominates in the UK.

4.3.2.5 Integration of plant resistance with other control measures

In the control of Rhizoctonia it is important to consider both soil- and seed-borne phases of disease. Visual assessment of seed can be made to judge whether seed should be treated with a specific Rhizoctonia fungicide treatment. Where high-grade seed is required for 'virgin' sites or specific export markets eye-plug tests can be performed to identify if any microscopic infection exists (Lapwood *et al*., 1979).

Quantifying inoculum in soil is far harder and in the past this relied on knowledge of the rotation and past disease experience. However, a new bioassay (Kyritsis, 2003) and a real time PCR assay (Lees *et al*., 2002) have enabled inoculum of *R. solani* in soil to be quantified. Using these tests it is possible to highlight which soils have high levels of contamination. Whilst selecting more resistant varieties for growing in these fields, many growers are now using azoxystrobin (Amistar) as an in-furrow treatment to control soil-borne Rhizoctonia (Wale *et al*., 2004). Thus decisions on whether to use this product should take into consideration the degree of soil-borne contamination and the resistance of the variety to be grown in that field.

4.3.2.6 Avoidance of resistance breaking races developing in populations of potato pests and diseases

No reports of isolates of *R. solani* overcoming host resistance have been reported.

4.3.3 Dry rot (*Fusarium coeruleum*, *F. sulphureum*, *F avenaceum*, *F. culmorum*)

4.3.3.1 Interpreting published disease and pest resistance

Dry rot can be caused by a number of *Fusarium* species. In a survey in the UK, *Fusarium coeruleum* was the most commonly isolated species (49%), followed by *F. avenaceum* and *F. culmorum* and then *F. sulphureum* (about 13%) (Peters & Lees, 2004). Inoculum of Fusarium can survive in soil or in dust found in store. However, infection can only occur following wounding of tubers, which can occur either at harvest or grading. In storage susceptibility to infection is lowest at harvest but increases with time (Boyd, 1952a).

Resistance testing takes the form of making a standard wound to a specific depth in a tuber and inoculating with a spore suspension (Jellis & Starling, 1983) or mycelium grown in sand and cornmeal (Wastie & Bradshaw, 1993) of the relevant *Fusarium* species. After 4-6 weeks of incubation tubers are cut in half through the wound and the degree of infection assessed (Boyd, 1952b). Two types of resistance can be recorded; resistance to penetration, which is recorded as the incidence of infection, whilst resistance to colonisation is measured as width and depth of necrosis.

When testing varieties for resistance, factors which affect test results include incubation temperature (Boyd, 1952b), inoculation method (Boyd, 1952c) and the time of year (Boyd, 1952a). As a consequence it has been suggested that the tests should be repeated over 2 years (Wastie *et al*., 1989). Such tests have shown differences in susceptibility and good correlation between tests (Jellis, 1975; Jellis & Starling, 1983; Wastie *et al*., 1989).

4.3.3.2 Use of resistance as a sole method of pest and disease control

At present no variety has been identified with good resistance to all *Fusarium* species. As resistance to dry rot is *Fusarium* species dependent, although a variety may have good resistance to one pathogen it may be susceptible to another. Relying on host resistance is therefore difficult especially when it cannot be certain which species is prevalent.

Good resistance to individual *Fusarium* species does exist. Differences in resistance to *F. coeruleum* and *F. sulphureum* have been observed both in term of penetration and colonisation (Wastie *et al*., 1989). Arran Banner and Pentland Ivory were among the most resistant varieties to *F. coeruleum*, whilst Catriona and Dunbar Standard were among the most susceptible. For *F. sulphureum*, Desiree and Maris Piper were the most resistant and Arran Pilot and Doon Star were most susceptible. In the NIAB list of varieties, ratings for both *F. coeruleum* and *F. sulphureum* are provided (Anon, 2005) although results are not available for all varieties. Varieties with good resistance to *F. coeruleum* include Asterix (8) and Tay (7), with more susceptible ones being Estima and Maris Piper (3), Nectar (2) and Savanna (1). Varieties with good resistance to *F. sulphureum* include, Romano, Sante, Saxon and Marfona (7) and Tay (8). The susceptible varieties include Nadine and Cara (2).

In future breeding for resistance could improve variety resistance against specific species (Wastie & Bradshaw, 1993; Lees & Bradshaw, 2001). Other workers, have identified common sources of resistance to both *F. coeruleum* and *F. sulphureum* (Leach & Webb, 1981).

4.3.3.3 Assessing risks when utilising host plant resistance

As the susceptibility to each *Fusarium* species differs between varieties it is important to know which species might be present. In a survey of species causing dry rot in GB, the pathogenicity of isolates of *F. coeruleum, F. sulphureum, F. culmorum* and *F. avenaceum* was determined. The isolates were tested on tubers of 10 varieties. Results showed that differences were not consistent between species. In general, the least common of the four species, *F. sulphureum,* was the most aggressive (Peters & Lees, 2004). *F. coeruleum* was the most common species GB wide, with *F. culmorum, F. avenaceum* and *F. sulphureum* being more common in England.

TABLE 4.8 ASSESSMENT OF VARIETY SUSCEPTIBILITY TO FOUR SPECIES OF *FUSARIUM*. ROT VOLUME (CM^3) INDUCED BY INOCULATING TUBERS WITH CONIDIA OF *FUSARIUM* SPP. DATA ARE THE MEANS ACROSS FOUR ISOLATES FOR EACH SPECIES (PETERS & LEES , 2004).

	Fusarium species			
Variety*	*F. avenaceum*	*F. coeruleum*	*F. culmorum*	*F. sulphureum*
Russett Burbank	0.1	5.0	3.2	67.8
Hermes	15.6	2.4	11.9	29.2
Marfona	2.9	13.9	0	37.9
Estima	16.4	5.5	6.1	26.3
Desiree	11.0	6.8	0.1	12.6
Cara	4.5	7.7	0	16.0
Maris Piper	3.1	13.1	0.1	11.6
Lady Rosetta	4.3	0.1	2.0	17.3
Sante	11.2	2.8	0	4.2
Saturna	1.3	3.9	1.2	7.1
LSD	5.4	3.3	11.5	16.5

* The variety order is ranked by total dry rot

4.3.3.4 Relationship of inoculum level and environment to effectiveness of host plant resistance

In storage, infection occurs when inoculum is present at sufficient levels and if the right environmental conditions exist. For *F. coeruleum* the frequency of infection was found to increase when duration of surface wetness was raised from 3 to 9 hours and temperature increased from 5 to 10^{o}C. However, reducing infection by *F. coeruleum* by preventing condensation only occurs when inoculum levels are low (Hilton & Blackwood, 2001). The effectiveness of host resistance under high inoculum levels and under conditions that favour disease is not known.

4.3.3.5 Integration of plant resistance with other control measures

In order to optimise host resistance to dry rot, it is important to know which *Fusarium* species are present. Identification of *Fusarium spp.* can be made by either isolating the fungus and growing on media and identifying the subsequent culture (Booth, 1971) or by PCR diagnostics (Cullen *et al*., 2005). However, by the time symptoms have developed on tubers it is too late to take preventative action and as the make-up of species may change between years it is difficult to predict which is present in store. Trying to select a variety resistant to the relevant pathogen is therefore difficult. In future, use of diagnostic tools to predict which are the predominant *Fusarium* species present in a particular field may assist in selecting varieties to be grown at a particular site (Cullen *et al*., 2005).

Control options other than host resistance are very important. Infection can be reduced by preventing damage, rapid drying after harvest and ensuring that rapid wound healing occurs by dry

curing. As some damage is always likely to occur, keeping stores clean to reduce inoculum (Clayton *et al*., 2001) and keeping conditions dry and cool will reduce risk of disease development. Into store fungicide treatments including imazalil (Carnegie *et al*., 2001) and thiabendazole (Carnegie *et al*., 1990) also provide good control options. Although thiabendazole is effective against most species, all isolates of *F. sulphureum* are considered insensitive to this compound.

4.3.3.6 Avoidance of resistance breaking races developing in populations of potato pests and diseases

No reports of isolates of *Fusarium* overcoming host resistance have been reported. However, as more than one *Fusarium* species may be present in storage, variety choice and use of thiabendazole are likely to encourage specific species.

4.3.4 Gangrene (*Phoma exigua* var. *foveata*)

4.3.4.1 Interpreting published disease and pest resistance

A considerable amount of research into potato gangrene was carried out in the 1970s and 1980s. The disease is now considered to be of much less significance. The research conducted on this disease has been much less in the last 20 years.

4.3.4.2 Background to resistance breeding

As with some other potato diseases, there is an issue with the methods used to screen potato varieties for resistance to gangrene. The first main problem is the relative lack of information on the relative incidences of gangrene on varieties in commercial practice. Such information would help enormously by providing a benchmark against which to assess the usefulness of the many different screening methods that have been devised by breeders. The second problem concerns how close conditions in the tests are to those the crop experiences when at risk of infection. Screening tests are likely to be more relevant where breeders are regularly updated by agronomists on current appropriate aspects of current potato production. For example, what is the most appropriate temperature when tubers are challenged by the pathogen and during subsequent storage?

The method used to inoculate tubers with *P. exigua* var. *foveata* affected the variety resistance ranking order (Wellving, 1976). The best inoculation method is the one that accurately predicts the relative resistances of varieties in practice. Bain (1985) concluded that an appropriate inoculation method in screening for gangrene resistance was that which consisted of damaging artificially-contaminated tubers on a commercial grader since it incorporated an assessment of variety resistance to tuber damage in addition to assessing the resistance of tuber tissue to infection. Greater differences in resistance were evident among varieties when their damage resistance was assessed in the inoculation method. Wellving (1976) and Jellis (1982) also found that when the inoculation procedure assessed damage resistance varieties were more easily discriminated.

One drawback with the grader damage inoculation method is that a large number of tubers per variety is required. Therefore, this inoculation method could only be used late on in the breeding programme when the number of clones to be screened has been reduced and more tubers of each clone are available. This means that, for the early screening of varieties, methods that only assess tissue resistance to infection will have to be used (Langton, 1971b; Wiersema, 1977). Bain *et al.* (1987) found that point inoculation methods could give misleading results. For example, when assessed using a point inoculation method the variety Record was frequently classed as a

susceptible variety whereas due to this variety's good damage resistance it is likely to be fairly resistant to gangrene in practice.

The physiological, or biochemical processes of the tuber responsible for variety differences in tissue resistance to *P. exigua* var. *foveata* are unknown.

4.3.4.3 What resistance is available?

No potato variety has tubers that are completely resistant to gangrene. However, varieties differ markedly in their degree of susceptibility. There are reports that gangrene incidence tends to be higher in early than in maincrop varieties, but Malcolmson (1958) found that susceptibility was not correlated with earliness in tests on 39 varieties.

Varieties differ in the resistance of their tuber tissue to infection and colonisation by *P. exigua* var. *foveata* (Malcolmson, 1958; Jellis, 1975b; Pietkiewicz & Jellis, 1975; Wellving, 1976). Wellving found that resistance to infection was closely related to resistance to colonisation in varieties. Walker and Wade (1976) demonstrated that there were three phases of tissue resistance following infection by *P. exigua* var. *foveata*: the retardation of lesion development, its arrest and lesion rejection. It was found that tuber incubation temperature influenced which of the phases operated. At 2 ^{o}C none operated whereas at 6^{o}C only lesion retardation occurred but at 10^{o}C lesion arrest followed lesion retardation.

4.3.4.4 Practical use of host resistance

Bain (1985) reported that selection for tuber tissue resistance may not necessarily be reflected in a large reduction in gangrene incidence in practice. He found that, over a wide range of inoculum densities, the level of tissue resistance in different varieties had only a moderate effect on the incidence of gangrene at 4^{o}C. However, two of the varieties used in the experiment, namely Pentland Crown and Maris Piper, are generally accepted to differ greatly in resistance: Pentland Crown is reported to be susceptible and M. Piper recognised to be much more resistant (Logan & Woodward, 1971; Anon. 1980; Anon. 1981).

It could be argued that although variety tissue resistance level appears to have little practical influence on gangrene incidence at 4^{o}C it may do at higher incubation temperatures. However, Bain (1985) found that at 10^{o}C lesion arrest occurred in all varieties irrespective of their tissue resistance level. It is surprising, therefore, that an incubation temperature of 10^{o}C has been used by breeders when screening clones for gangrene resistance (Jellis, 1975b) and that variety differences are reported to be greater at 10^{o}C than 5^{o}C (Langton, 1971a). The results obtained by these authors probably arose because the pathogen was favoured by the inoculation techniques used: tubers were inoculated with actively growing mycelium whereas Bain (1985) used pycnospore suspensions.

At 4^{o}C, the temperature at which gangrene development is widely accepted to be greatest (Malcolmson, 1958; Malcolmson & Gray, 1968; Langton, 1972; Adams & Griffith, 1983), lesion development was not arrested in varieties with a high level of tissue resistance: the difference between varieties with high and low tissue resistance at 4^{o}C was simply that in the more resistant varieties lesion development was slower. Thus over a normal storage period lesions that detract from the commercial value of the crop could well have appeared in resistant varieties. The above findings suggest that gangrene could be controlled more effectively through the use of resistant varieties if the methods employed to screen for resistance placed less emphasis on tissue resistance and more on other resistance components, e.g. resistance to damage. The greater use of resistance screening methods which assess variety resistance to damage, such as that developed by Jellis (1982), is desirable.

4.3.4.5 Relationship of inoculum level and environment to effectiveness of host plant resistance

Inoculum level

Bain (1985) concluded that when varieties are being screened for resistance to gangrene the variety ranking order is likely to be the same irrespective of the level of inoculum with which the tubers are inoculated. The extent to which varieties can be discriminated will however be greatly influenced by the inoculum density.

Bain *et al.* (1988) found that the higher the level of inoculum on tubers the greater the development of gangrene in storage given suitable conditions for its development. However, the response to increased inoculum density was shown to be markedly affected by variety and the amount of tuber damage. The increase in disease incidence resulting from raising the inoculum density was greater for damaged compared with undamaged tubers.

There is not enough information to determine whether variety differences in the extent to which seed-borne inoculum is transmitted to the progeny tubers are large enough to affect the variety ranking order based on tuber resistance to damage and infection by the gangrene pathogen (see below). However, this aspect of variety resistance to gangrene has generally not been assessed, most probably because the benefit is unlikely to be worth the extra effort involved. The amount of gangrene inoculum on the progeny tubers can be more easily reduced using other control methods, e.g. the use of limited generation seed and fungicide seed treatments.

In a very limited study, Bain (1985) found that potato varieties differed in the extent to which they transmitted *P. exigua var. foveata* from seed to progeny tubers in the field, although this occurred with only one of the two isolates used in comparing Maris Piper and Pentland Crown. Adams (1980b) found that the degree of transmission of *P. exigua* var. *foveata* from seed to progeny tubers depended on variety. In his experiments, to examine the role of seed-tuber and stem inoculum in gangrene development, the two varieties Pentland Crown and Ulster Sceptre were used. It was found that the contamination of Ulster Sceptre progeny tubers was greater than that of Pentland Crown. Ulster Sceptre is a first early whereas Pentland Crown is a maincrop variety, therefore the difference in progeny tuber contamination levels for these two varieties may have been due to the earlier senescence of, and hence greater build-up of *P. exigua*. var. *foveata*, on Ulster Sceptre. The results of Bain (1985) show that varieties of a similar maturity class also differ in the degree of inoculum transmission to progeny tubers, but that this may be again related to the rate of haulm senescence: Maris Piper showing a higher degree of haulm desiccation when sampled than Pentland Crown. The extent of haulm senescence appeared to be more important than seed infection in relation to transmission.

There is evidence that variation in tuber resistance among varieties is of little significance as far as the transmission of inoculum from the seed to progeny tubers is concerned. It was found the level of progeny tuber contamination was generally greater when the seed had only been contaminated with *P. exigua var. foveata* rather than having had gangrene lesions at the time of planting (Adams, 1980b; Bain, 1985).

Wellving (1976) reported considerable variation among varieties in tissue resistance to infection. He found that, following point inoculation and incubation at 4^{o}C, the level of infection in the most susceptible variety used was many times greater than that in the most resistant and exceeded the range found by Bain (1985).

Tuber damage

As *P. exigua var. foveata* is primarily a wound pathogen the resistance of a variety to gangrene is greatly influenced by the resistance of its tubers to damage. Since different types of wound differ in their susceptibility to infection (Adams, 1980a), it is the level of resistance to the wound types which particularly predispose tubers to gangrene which has the greatest influence on resistance. Varieties differ greatly in their susceptibility to damage (Blight & Hamilton, 1974) but the damage resistance ranking order of varieties varies with damage type (Jellis & Haslam, 1980). Wounds which incorporate crushing of the tuber tissue are more susceptible to infection by *P. exigua var. foveata* than cuts, splits or scuffs (Griffith, 1970; Bak Henriksen, 1975; Jellis & Howard, 1975; Adams, 1980a). There are two components of resistance to tuber damage, namely resistance to wound number and resistance to wound severity. Differences in wound size are important, since severe wounds are more susceptible to gangrene than slight wounds of the same type. Adams (1980a) confirmed that it is a genuine difference in susceptibility and that larger wounds do not simply trap more inoculum. Furthermore, deeper wounds which expose the tuber's more susceptible medullary tissue are more likely to become infected than more superficial wounds which only penetrate the more resistant cortical tissue (Pietkiewicz & Jellis, 1975).

Although the basis of variety differences in susceptibility to damage is not fully established, variety susceptibilities have been linked with specific characters that are genetically determined. Varieties with large tubers are generally more susceptible to damage (Blight & Hamilton, 1974). However, Jellis & Haslam (1980) found that varietal differences in susceptibility to wounds which exposed the medulla were not accounted for by tuber size alone. Tuber shape also affects susceptibility to damage. Hughes (1980) stated that tubers with small radii of curvature tend to damage more easily than those with large radii of curvature. Blight & Hamilton (1974) found that, although low skin hardness is generally associated with high damage, the susceptibility of the tuber to mechanical damage appears to be affected by other characters. Umaerus (1975) from a study with 54 clones, reported that those with tubers of high elasticity, as measured by the rebound pendulum, were more resistant to shatter cracks and splits than those with less elastic tissues. Wellving (1976) also found that varieties with tuber tissue of low elasticity more often sustained wounds severe enough to allow infection by *P. exigua* var. *foveata* than did varieties with a higher elasticity.

Since the susceptibility of tubers to gangrene is related to their susceptibility to damage, as well as to infection, then factors that influence resistance to injury need to be considered in addition to those which affect tissue resistance to infection. Some of the factors identified as affecting resistance are the availability of nutrients (nitrogen and magnesium) to the growing crop, tuber age, tuber temperature, relative humidity and site of infection on the tuber (Bain 1985).

Other factors

Jellis (1978) found that the relative susceptibility of varieties to infection of the medulla tissue by *P. exigua* var. *foveata* was affected by the site at which the tubers were grown. Wellving (1976) demonstrated that the resistance ranking order of varieties was greatly influenced by the method used to inoculate the tubers, since different resistance characteristics were assessed by the various methods used. Pietkiewicz & Jellis (1975) observed that the ranking order of varieties could be different for cortical inoculations compared with inoculations of the medulla.

The evidence is conflicting as to whether the resistance ranking order of varieties is affected by the temperature at which the inoculated tubers are incubated. Wellving (1976) observed no difference in ranking order at 3^{o}C and 10^{o}C. However, Seppanen (1982) observed at differences at 6^{o}C, 12^{o}C and 18^{o}C.

4.3.4.2.5 Integration of plant resistance with other control measures

Adams (1980a) suggested that priority should be given to controlling gangrene by reducing the incidence of tuber damage, through the breeding of damage-resistant varieties and better husbandry, rather than by reducing the level of daughter tuber contamination. However, it is unlikely that gangrene could be effectively controlled by minimising tuber damage alone since damage is unlikely to be completely eliminated either through breeding of resistant varieties or by better production methods (although destoning has had a very positive impact). The best approach would be to minimise damage through breeding and better husbandry coupled with the use of fungicides and cultural control methods (e.g. limited generation seed, rotation, prompt desiccation and early harvest) to reduce the amount of inoculum on progeny tubers at harvest time.

4.3.4.6 Avoidance of resistance breaking races developing in populations of potato pests and diseases

There are reports that the relative susceptibilities of varieties can vary with different isolates of *P. exigua* var. *foveata*. Jellis (1978) found that, with one isolate, the normally resistant variety Maris Piper was sometimes more susceptible than the generally susceptible Pentland Crown. Rogers and Killick (1974) also reported a variety x isolate interaction. Of three varieties tested, Pentland Falcon was the most susceptible to three isolates but the most resistant to one other. This interaction occurred only for gangrene lesion length, not breadth or depth. In studies on the infection of potato varieties with different field isolates of the gangrene pathogen, Bain *et al.* (1987) found that variety x isolate interactions, were not substantial enough to affect variety ranking order. However, variety rank was markedly affected by pathogen isolate when both field and culture collection isolates were compared. This suggests that the complications of variety x isolate interactions in screening tests can be avoided by the use of recent field isolates of high pathogenicity.

4.3.5. Pink Rot (*Phytophthora erythroseptica*)

4.3.5.1 Interpreting published disease and pest resistance

Pink rot is believed be an increasingly common disease of potato. Although no survey on its incidence or severity hase been carried out, it is a disease that is being reported more frequently by agronomists. The disease is named after the pink colour that develops in infected tubers when cut and exposed to air for 20 to 30 minutes. Often infected tissue has a distinct smell of vinegar. Knowledge of resistance to this disease is restricted to a number of papers written in Scotland (Lennard 1980), USA (Salas *et al*. 2003) and Canada (Peters *et al*. 2004; Peters & Sturz 2001).

As a soil-borne disease one method of assessing disease resistance in varieties is to have plots of different varieties at a site with a history of pink rot. This method was used to assess field resistance to pink rot in Scotland and at harvest, tubers were dug and incidence of infected plants and tubers determined (Lennard 1980). Differences in susceptibility were found between a number of varieties over a three year period. Among the varieties tested, Home Guard developed most infection, in the two years it was tested and Record and Stormont Enterprise developed little pink rot. There was some inconsistency in the response of varieties between years with King Edward showing little disease in 1975 and only moderate infection the following year. Results from these tests are dependent on time of harvest as disease becomes increasingly frequent with later harvests (Lennard 1980).

Host resistance testing can also be performed in storage. Early tests involved taking daughter tubers, removing a circular core and inserting a plug of mycelium into the wound. Tubers were incubated under warm damp conditions for one week and cut longitudinally and the depth of lesion measured (Lennard 1980). The results from these post-harvest tests corresponded well with those found in field tests. In USA and Canada, daughter tubers have been inoculated by placing drops of spore suspensions onto the eyes (Peters *et al*. 2004; Salas *et al*. 2003). This second method of post-harvest testing also resulted in significant differences in susceptibility. Differences between varieties in depth of symptoms (colonisation) have also been observed (Salas *et al*. 2003). The relationship between incidence and severity was weak so that some varieties were more resistant to infection but not colonisation of tissue and vice versa.

4.3.5.2. Practical use of host resistance

Although differences in susceptibility to pink rot have been shown no variety with a high degree of resistance has been found. In the UK, resistance to pink rot on the recommended list of potato varieties is not known (Anon. 2005).

4.3.5.3 Assessing risks when utilising host plant resistance

As no variety has been identified with high resistance it is likely that where there is a high risk of infection even the more resistant varieties will be at risk. Variety resistance must therefore be used in-conjunction with other control measures.

4.3.5.4 Relationship of inoculum level and environment to effectiveness of host plant resistance

Little is known about how pink rot develops in the field. It is a disease associated with warm damp soil conditions. Infection occurs through stolons but infection may also occur through wounds created at harvest or grading. The later crops are harvested the greater the risk of pink rot developing (Lennard, 1980). How inoculum levels and environmental conditions interact with field resistance is not known.

Further disease can develop in storage. In an experiment looking at effect of wounding, temperature and inoculum on development of pink rot in store, severe wounding, temperatures of 15-25^{o}C and high inoculum densities all encouraged infection. Infections in unwounded tubers occurred at 15^{o}C whilst on wounded tubers infection occurred at 10^{o}C. Incidence of infection was high where all factors were favourable but reduced where only one factor was present (Salas *et al*., 2000). It is not known how storage conditions or inoculum level in storage may affect host resistance.

4.3.5.5 Integration of plant resistance with other control measures

Where fields are contaminated with *P. erythroseptica*, avoiding susceptible varieties would be advisable but differences in resistance in current varieties is unknown. This should be done in-conjunction with early harvest of the crop (Lennard, 1980). At harvest and grading care must be taken to avoid damage in order to avoid further infection. In storage, varieties which are susceptible to colonisation by the fungus may be at risk of rotting. Special care must be taken with these stocks to ensure diseased tubers are removed at grading to avoid spread to non-infected tubers.

In other countries, including USA and Australia, mefenoxam and metalaxyl have been used in-furrow at planting to effectively control pink rot (Wicks *et al*., 2000). However, mefenoxam and metalaxyl strains of *P. erythroseptica* now exist in USA (Taylor *et al*., 2002) and Canada (Peters *et a*l., 2001).

4.3.5.6. Avoidance of resistance breaking races developing in populations of potato pests and diseases

No reports of host resistance being overcome by *P. erythroseptica* have been reported.

4.3.6 Potato Blight (*Phytophthora infestans*)

4.3.6.1 Background to resistance breeding

There are two types of resistance, i.e. race specific resistance (conferred by R-genes) and race non-specific resistance. This distinction has been recognised for decades. Consequently there are many different terms for the two types of resistance (Table 4.9).

TABLE 4.9 TERMS USED FOR DESCRIBING TYPES OF RESISTANCE TO FOLIAR LATE BLIGHT IN POTATO

Resistance type 1	**Resistance type 2**
Race specific	Race non-specific
Vertical	Horizontal
Immunity	Field resistance
Qualitative	Quantitative
Complete	Partial
Monogenic	Polygenic
R-gene or major gene	Minor gene
Specific (compatible versus incompatible)	General (rate reducing)
R-gene based	Maturity related

Extracted from Allefs *et al.* (2005)

Resistance genes (R genes) have been identified in many wild *Solanum* species. In the early 20th century genes from *Solanum demissum* were introduced into *Solanum tuberosum* to produce more resistant varieties. This strategy was not successful because the R-genes were overcome when the pathogen developed races that could overcome the R-genes. R-gene resistance was abandoned in the 1970s. At that time there were 11 ex-demissum R-genes (R1 to R11). Failure of the R gene breeding system coincided with development of better blight fungicides. This development reduced the pressure on breeders to produce varieties with high resistance levels. After the R gene system breeders switched to using race non-specific resistance because it was considered that this would be more durable (Wastie, 1991). The problem with race-non-specific resistance is that it is strongly linked to foliage maturity. Potato genotypes that are more resistant tend to be later maturing.

Allefs *et al*. (2005) were highly critical of potato breeders' attempts to add significantly to late blight disease management. They presented two main facts as evidence for their criticism. Firstly, the cost of fungicides required for an optimal spray programme for a susceptible variety is estimated at 1.7 billion Euros per year for the top 38 potato producing countries in the world. Secondly, in The Netherlands the average yield loss for the most important varieties when grown organically, with no fungicide protection, between 2002 and 2004 was 45 percent.

There is clear evidence from some European countries that epidemics are starting earlier. For example, the start of the blight epidemic at Jogeva in Estonia has been recorded since 1922. There is a very clear trend that the epidemic starts much earlier now than decades ago. In the 1920s the

epidemic generally started in mid-August whereas now it generally starts in mid- to late-July. The reason for this is not clear. However, whatever the cause, crops appear to be more at risk earlier in the growing season. In the absence of weather favourable for medium to long distance spread of *P. infestans*, earlier initiated epidemics are most likely due to either soil-borne oospores, infected seed or infected groundkeepers. If outbreaks are earlier in relation to crop development then presumably early varieties will require as good resistance to blight as later maturing varieties. This was not the case previously when early maturing, rapid-bulking varieties could be more susceptible.

It is interesting to compare the average resistance ratings for varieties in 1988 and 2004 (Table 4.10). Wastie (1991) presented the average resistance ratings for the 23 recommended varieties in the UK in 1988. The comparison with the 31 most widely grown varieties in 2004 confirms that the industry is certainly not making more use of variety resistance in the management of potato late blight. Of course there are exceptions. For example, the first early variety Orla with resistance ratings for foliar and tuber blight of 8 and 8, respectively is available to the pre-pack and general ware market.

TABLE 4.10. AVERAGE RESISTANCE RATINGS IN 1988 AND 2004

	First early	Second early	Maincrop
1988			
Number of varieties	6	5	12
Foliage blight	3.8	5.4	4.8
Tuber blight	3.2	5.6	4.6
2004			
Number of varieties	3	11	17
Foliage blight	4.3	3.5	4.5
Tuber blight	4.7	4.0	4.5

1988 data from Wastie (1991)

Lammers *et al.* (1998) found that in field trials there was no correlation between leaf blight and stem blight assessed separately on six cultivars. In general, stem blight is not assessed separately in cultivar screening tests. However, cultivar resistance to stem blight could be more important where oospores or infected seed tubers are the primary source of inoculum and stem infection is prevalent in disease development.

4.3.6.2 What resistance is available?

Breeding lines and varieties with much greater resistance than that generally utilised in the industry today are available. They have been produced by a variety of breeding techniques.

In response to new populations of *P. infestans* spreading from Mexico into Europe and North America, SCRI breeders have carried out fresh screenings of germplasm collections and identified a large number of resistant accessions (Bradshaw & Birch, 2006). Currently these are being held in reserve.

It was clear in the late 1960s that R genes would not provide durable resistance therefore the strategy at SCRI for producing varieties with greater resistance has been to combine R genes with

race non-specific resistance. This approach was taken because defeated R-genes, or genes for field resistance linked to the R-gene, do contribute to variety resistance (Stewart *et al*. 2003).

Other research groups are screening for R genes from *Solanum* species other than *S. demissum*. Trognitz (1998) reported an unknown R gene, R-T, in *S. tuberosum ssp. andigena* clone TPS67. This was the first report of an R gene from this subspecies. It is considered that novel R genes could contribute substantially to late blight management (Allefs *et al*. 2005). In 2004 the first potato cultivar, called Biogold, that had resistance derived from *Solanum bulbocastanum* was released (Bradshaw *et al.*, 2006).

A conventional breeding programme in Hungary has produced several clones showing high levels of rate-limiting, isolate non-specific resistance (Shaw & Kiezebrink, 2005). The clones are particularly suited to low-input systems of potato production because in addition to their resistance to potato blight they also have good resistance to virus, slugs and wireworm in addition to weed suppression and long dormancy. It is stated that the source of resistance was eight *Solanum* species. The blight resistant varieties were selected from the very large numbers of genotypes screened for foliar and tuber resistance. Many of these 'Sarpo' clones have a higher level of blight resistance than any of the reference varieties, e.g. Cara, Valor, Stirling. The Sarpo varieties are not immune to blight. When infected, Sarpo clones have a low infection efficiency, slow lesion growth and limited sporulation. Sarpo clones tend to have a high dry matter. Two varieties Sarpo Mira and Axona are on the National Lists in the UK and Denmark.

Genetic modification techniques have been used to produce more resistant varieties. Filippov *et al.* (2006) reported that some clones of Russian potato varieties that were transformed with the thaumatin II gene were significantly more resistant to foliar and tuber blight compared with the non-transgenic plants of these varieties. Genetically modified plants (normally fully susceptible) containing the RB gene from the wild diploid species, *Solanum bulbocastanum*, had broad-spectrum resistance to late blight (Staples, 2004). A modified, small, naturally occurring cationic peptide, temporin A, was expressed in potato plants and gave strong resistance to late blight, pink rot and *Erwinia carotovora* (Osusky *et al*. 2004).

Variety resistance to blight obviously contributes to blight control but it is also considerably underused as a control measure. For it to be used more it needs to be present in varieties with the agronomic and quality characteristics currently sought by potato buyers. In addition, for it to complement fungicide control then extensive, robust information on the necessary fungicide inputs for different levels of variety resistance is required. This work has started in The Netherlands. What is also required are reliable and up to date resistance ratings for varieties.

There are contradictory reports regarding the genetic link between foliar and tuber blight resistance.

Evidence for*:*

- Stewart *et al.* (1994) reported that in a screen of 50 clones of each of 5 progenies from crosses where one parent was resistant to foliage and tuber blight and the other susceptible, foliage and tuber resistance were correlated.

Evidence against*:*

- Collins *et al*. (1999) stated that in contrast with previous findings, a negative correlation was found between foliage and tuber blight resistance.
- Wastie (1991) stated that resistance to *P. infestans* in tubers does not correlate with resistance in the foliage.

Mixed evidence*:*

- In a comparison of foliage and tuber blight resistance in four mapping populations of potato, Park *et al.* (2005) found that tuber blight resistance was inherited independently from foliage blight resistance in one population but in two others tuber and foliage blight correlated significantly.

Most evidence suggests that non race-specific foliar resistance and foliage maturity are genetically linked.

Evidence for:

- The most important locus for foliage resistance and foliage maturity was found on chromosome 5 near marker GP21: the allele of marker GP21 that is associated with resistance to late blight is also associated with late foliage maturity (Visker *et al.* (2005).
- Field resistance, earliness and vigour were examined in a diploid segregating potato population grown in replicated trials over three consecutive growing seasons by Collins *et al.* (1999). There was co-localisation of QTL for resistance, vigour and earliness.
- Allefs *et al.* (2005) stated that under long day conditions race non-specific resistance is strongly correlated with foliar maturity. These authors stated their belief that varieties with better scores for foliar resistance require more fungicides in ware potato production than early maturing, more susceptible varieties. This is because little account is taken of cultivar resistance rating but the longer growing season for later maturing varieties results in a greater number of fungicide applications.

Evidence against*:*

- None

Mixed evidence*:*

- Visker *et al.* (2004) reported that of six progenies with race non-specific resistance, i.e. free of the 11 known R genes, for five there was a significant correlation between resistance to foliage blight and foliage maturity. The results suggest that it should be possible to have some selection for late blight resistance without affecting foliage maturity type.

4.3.6. Practical use of host resistance

How extensively is variety resistance used? A comparison of the areas of crop planted with varieties with ratings across the range (1 to 9) in Great Britain for 1996 and 2004 (Tables 4.11 and 4.12) suggests that this is not the case. The area of varieties more susceptible to foliar blight (resistance ratings of 2 to 4) has increased considerably over that 8-year period. The situation for tuber blight resistance is not much better. Admittedly, the acreage of varieties with a rating of 1 or 2 has declined considerably. However, the crop area of rating 6 or 7 has declined even more. There has been an increase in the popularity of varieties with ratings of only 3 or 4. At present, 23 of the top 31 varieties have a foliar resistance rating of 4 or less. The corresponding number for tuber blight is better at 15 of the top 31. With so many varieties of similar resistance there is little incentive for growers to match fungicide inputs to crop resistance.

TABLE 4.11 AREAS OF VARIETIES WITH DIFFERENT RESISTANCE RATINGS FOR FOLIAR BLIGHT GROWN IN GB IN 1996 AND 2004

Variety resistance rating	1996 areas (ha)	2004 areas (ha)	Change in hectares	% change
2 (susceptible)	2563	5178	+2615	+102 %
3	10659	15519	+4840	+45 %
4	74481	77377	+2896	+4 %
5	8676	868	-7808	-90 %
6	31058	15389	-15669	-50 %
7 (resistant)	5489	3409	-2080	-38 %

TABLE 4.12 AREAS OF VARIETIES WITH DIFFERENT RESISTANCE RATINGS FOR TUBER BLIGHT GROWN IN GB IN 1996 AND 2004

Variety resistance Rating	1996 areas (ha)	2004 areas (ha)	Change in hectares	% change
1 (susceptible)	4289	3024	-1265	-29%
2	3114	622	-2492	-80%
3	3955	4843	+888	+22%
4	27786	35410	+7624	+27%
5	73355	68822	-4533	-6%
6	11131	3277	-7854	-71%
7 (resistant)	9296	1742	-7554	-81%

Potato breeders continue to improve the resistance of breeding lines and varieties (some very resistant varieties have been made available in recent years, most notably the Sarpo varieties) but as we have seen the more resistant varieties are not being utilised much, presumably, and understandably, because the combination of characteristics offered by these varieties does not meet what the market requires. The failure to make use of better resistance was obviously not a deliberate decision but probably reflects changes in the market for potatoes, e.g. the move away from late maturing varieties in the pre-pack sector due to poor skin finish or the significant growth in demand for salad potatoes. Salad crops are in the ground for a very short time compared with general ware and baker crops and therefore a high rating for blight resistance is less critical. The comparison of years 1996 and 2004 did not take account of the time period that different crop types are in the ground.

Swiezynski *et al.* (2001a) note that in Europe many varieties and advanced breeders selections have been obtained with tuber resistance to *P. infestans*, but in the last decades no progress is noted in the mean level of this resistance in varieties.

4.3.6.4 Use of resistance as a sole method of pest and disease control

There have been several studies examining whether mixtures of varieties with different levels of resistance offer better protection of susceptible genotypes compared with pure stands. Based on 3 years of field trials with natural epidemics Andrivon *et al.* (2003) concluded that the resistance of

host varieties is either not effective enough, or is too easily overcome by the pathogen, to be used alone. The authors did observe that the severity of late blight in a susceptible variety growing in rows alternating with partially resistant varieties was significantly lower than in unmixed plots of the susceptible variety alone. There was a significant yield increase for the susceptible variety in mixture compared with pure stands. Phillips *et al.* (2005) reached a similar conclusion after four experiments over 3 years. These scientists found that mixtures of potato varieties do not offer any improvement on the average of the disease resistance of the component varieties.

Initially the greatest use of variety resistance to blight is likely to be by the organic sector of the industry. Varieties with considerably better resistance than most conventional ones have been identified and trialed across Europe (Speiser *et al.*, 2006), for example, Sarpo Axona, Eve Balfour, Lady Balfour, Sarpo Mira and Sarpo Tominia in the UK. With these varieties the effectiveness of the resistance is not in question, it is whether their other characteristics meet market requirements.

4.3.6.5 Integration of plant resistance with other control measures

At a potato blight conference in Dublin in 1995 there were several papers demonstrating the value of using variety resistance to blight to reduce fungicide inputs. The principle of substituting variety resistance for some fungicide input was established long before the Dublin conference. Since 1995 there have been several reports of satisfactory foliar blight control on more resistant varieties with reduced fungicide inputs. For example Kirk *et al.* (2005) in 3 years of field experiments in the US demonstrated that varieties with greater resistance to late blight could be managed with reduced fungicide rates and/or longer application intervals. In The Netherlands current commercial decision support systems (DSSs) take account of variety resistance when making fungicide recommendations. However, Dutch researchers are improving the DSSs by taking even more account of foliar resistance to blight. The aim is to reduce the fungicide inputs required to control blight for both environmental and economic reasons. Three different systems have been evaluated, i.e. modified versions of the two commercial systems 'Plant Plus' and 'Prophy', plus a Wageningen University Research system. In the research carried out so far (Wander *et al.*, 2006) for two more resistant varieties, Seresta (foliar resistance rating 7) and Aziza (foliar resistance rating of 8), the dose of fluazinam could be reduced considerably, except at the end of the growing season.

There have been many studies demonstrating the principle that variety resistance can replace some fungicide input with blight control remaining acceptable. For example, Clayton *et al.* (1995) found that AUDPCs (AUDPC is the abbreviation for "area under the disease progress curve". The AUDPC is a measure of disease control throughout the epidemic. The larger the area the less good the control) offered more reliable indications of required fungicide doses than final disease ratings or apparent infection rates. The epidemics were controlled using 20-80% of the recommended rate of mancozeb on plots of Brodick, Cara and Torridon compared with full rates on Maris Piper. Also, in 3 years of field experiments using fluazinam, Kirk *et al.* (2005) demonstrated that potato varieties with greater resistance to late blight can be managed with reduced fungicide rates and/or longer application intervals. It is vital that these studies evaluate the impact of such an approach on the incidence of tuber infection. Where fungicide inputs are tailored more closely to variety resistance then tuber resistance also needs to be considered. A harvested crop with no tuber blight is unlikely if fungicide inputs are matched to a variety's foliar resistance rating without also considering the tuber resistance rating. Naerstad (2002) showed that exploiting high foliage resistance to reduce fungicide input was risky when field resistance to tuber blight was low. In contrast when resistance to tuber blight was high, use could be made of medium to high foliage resistance to reduce fungicide inputs. Toxopeus (1958) stated that a slow spreading and sporulating infection on a resistant variety may produce inoculum over a much longer period than would a more rapidly spreading and heavily sporulating infection on a susceptible genotype

The resistance of leaves at different positions in the crop canopy varies significantly (Visker *et al*., 2003). Unfortunately, leaves at the top of the plant are considerably more resistant than those lower in the canopy, meaning that the most resistant leaves intercept most of the fungicide spray when sprayed conventionally. Visker's work examined five varieties with race non-specific resistance in field and controlled environment experiments. Apical leaves were far more resistant to late blight than basal leaves. Plant and leaf age had only minor effects. The resistance of a specific leaf was similar during its lifetime.

The Dutch are the furthest ahead in incorporating variety resistance into DSSs. Their systems are most likely to be the most appropriate for GB. Duvauchelle *et al.* (2006) in France are also developing a DSS to take account of varietal resistance. The system is called DSS MILPV. Grunwald *et al.* (2002b) found that the DSS SimCast recommended too many fungicide sprays for Mexican varieties with high levels of field resistance. A modified SimCast that took account of variety resistance, evaluated in 1999 and 2000, resulted in good disease control on varieties with a range of resistances. More resistant varieties required fewer fungicide applications. No account is made of the mechanism of host resistance in developing DSS's but such knowledge could allow improvement in the forecasting schemes.

The organic sector of the potato industry has shown greater interest in the new more resistant varieties than the non-organic sector. Blight control in organic systems using these more resistant varieties and copper fungicides has been evaluated (Speiser *et al.,* 2006). The following varieties were trialed in four countries. Appell, Derby, Innovator and Naturella (in Switzerland), Derby, Eden , Escort and Naturella (in France), Sarpo Axona, Eve Balfour, Lady Balfour, Sarpo Mira and Sarpo Tominia (in the UK) and N89-1756 and N92-15138 (in Norway). The varieties were far less susceptible to foliar and tuber blight than most of the varieties currently grown. However, it was commented on that they might not meet market requirements.

Allefs *et al.* (2005) have proposed a novel blight management strategy using new R genes. These scientists consider that race non-specific resistance, which is related to foliage maturity, will not make a substantial contribution to integrated control of potato late blight under long day conditions. They believe that the use of as yet unknown R-genes could be more effective in the long term. They propose that a system of real-time monitoring of virulence factors be set up which would allow warnings to be used for protective spraying of only those varieties with the R-genes for which a matching virulence is detected. This is an interesting concept but may prove difficult operate in practice.

4.3.6.6 Avoidance of resistance breaking races developing in populations of potato pests and diseases

Wastie (1991) stated that the requirement for stable resistance must eventually preclude the use of R genes. Swiezynski *et al.* (2000) reported that all 11 virulence factors were found in both the old and new populations of *P. infestans*.

The evidence for race non-specific resistance being stable is mixed.

Evidence indicating that resistance is stable:

- In an international trial series the stability of resistance of seven and fourteen host genotypes was evaluated in seven and eight different locations worldwide. Overall, resistance to *P. infestans* was robust; resistant genotypes were consistently resistant in all locations and trials. Specific genotypes were stable across sites for resistance (Forbes *et al.* 2005).
- Grunwald *et al.* (2002a) reported that field resistances to potato late blight of 12 Mexican varieties released between 1965 and 1999 were durable. At least two of the varieties, i.e.

"Sangema" and "Tollocan" have been grown on at least 4 to 5% of the acreage and over long periods of time without loss of field resistance.

- In an evaluation of 16 potato clones in 8 locations in the United States in 1996 there was no genotype x environment interaction on the ranking of the clones in the different locations (Haynes *et al.* 1998).
- At two locations in the united States in 1993 and 1994 variety rankings, based on AUDPC, in response to infection by new immigrant isolates of *P. infestans* were nearly identical to rankings obtained previously with isolates prevalent prior to 1990 (Inglis *et al.* 1996).
- Colon *et al.* (1995) field tested 22 R gene-free varieties introduced between 1900 and 1954 and concluded that resistance was durable because 1) disease assessments (AUDPCs) closely correlated to resistance ratings given between 1929 and 1954 and 2) the stability over time since 1929 of the ratings in the Dutch Descriptive List of Varieties of Field Crops. The most resistant varieties were Robijn, Populair, Pimpernal, Libertas and Surprise. These five varieties were among the latest maturing of the 22.

Evidence indicating that resistance may not be stable:

- In studies in two consecutive years local populations of *P. infestans* in France were adapted for virulence and aggressiveness to host resistance (Montarry *et al.* 2006).
- In an international field trial over 4 years there were four standard varieties, i.e. Sava, Oleva, Danva and Kuras. The resistance of the first three varieties to foliage blight was stable but that of Kuras was not. 1 to 9 scores were estimated and these were similar to, or slightly lower than, those from official variety lists or from the European Cultivated Potato Database (Hansen *et al.* 2005)
- Flier *et al.* (2003) identified differential variety by isolate interactions for both foliage and tuber blight resistance, i.e. changed variety resistance ranking orders for different pathogen genotypes. In general, the severity of late blight epidemics in field plots was not closely related to the foliage blight resistance ratings in the National List of Recommended Potato Varieties. There was no significant correlation between variety resistance to tuber blight (field protocol) and tuber blight ratings in the National List. There was no relation between tuber blight resistances assessed in the field and in the laboratory. The presence of differential interaction, independent of R-gene-based resistance, indicates some adaptation of *P. infestans* to race non-specific resistance. This has consequences for the stability and durability of race non-specific resistance to late blight.
- When tuber blight incidence was screened in 2 successive years there was a highly significant variety x year interaction. Variety ranking orders were not the same for different aggressive isolates of P. infestans (Flier *et al.* 2001).
- Stewart *et al.* (1996) reported that when the tuber resistance of 10 varieties was tested twice in 2 years there was good agreement between the mean of field and glasshouse-grown tubers. For the field tests, but not for the glasshouse tests, there were variety x harvest date and variety x year interactions.
- There has recently been concern in The Netherlands that official resistance ratings have not been appropriate for decision support systems. For example, in the research to improve DSSs it was found that the variety Karnico had a much lower level of foliar resistance than the official resistance rating of 8 (Wander *et al.*, 2006). Resistance ratings are not set in tablets of stone; they can change with changes in the pathogen population. For example, Carlisle *et al.* (2002) found highly significant variation for disease parameters among a wide range of isolates of the blight pathogen and the variation was greater on the varieties with higher levels of resistance. There was a significant interaction between isolate and variety for all parameters assessed indicating that variety resistance ranking order can depend on the pathogen population.

At present, in some years it can be very difficult to keep crops blight-free. This was the case in 2004 because of periods of severe high risk coupled with difficult spraying conditions. There is growing evidence that the pathogen population in Europe has undergone rapid change, and continues to do so. This is likely to make blight control more difficult, not easier, in future. It is difficult to envisage satisfactory control of blight in future without making greater use of variety resistance. This is especially the case if fungicide inputs are to be optimised/reduced for environmental reasons.

4.3.7. Powdery scab (*Spongospora subterranea* f.sp. *subterranea*)

4.3.7.1 Interpreting published disease and pest resistance

The methods of disease resistance testing used in the UK to develop resistance ratings require maintaining wet conditions suitable for infection over the period of tuber initiation in a uniform and heavily contaminated field soil or artificially contaminated beds. Test varieties are compared to standard varieties of known resistance/susceptibility. Currently, the standard varieties and their resistance ratings are Accent (2), Estima (3), Cara (3), Pentland Crown (4) and Sante (8).

However, because of differences in physiological age and inherent varietal differences in crop development, different varieties may emerge at different times and/or develop tubers at different times. It can be difficult to achieve a uniform comparison of susceptibility to powdery scab as small differences in environmental conditions over the critical period can lead to differences in disease development or result in increased variability. Artificially contaminated beds often produce more uniform results than in the field (Wastie *et al*., 1988).

Measuring root infection for rapid screening of varieties for tuber susceptibility has produced mixed results. Falloon *et al*. (2003) using glasshouse and field studies found the method to be effective. In their screen of varieties, all those tested developed zoosporangia and root galls and root infection was usually related to tuber infection. On the other hand, Hughes (1980) whilst finding a positive correlation between root infection and tuber infection suggested a few abnormal results made the technique less valuable. Falloon *et al*. (2003) identified one exception (the variety Swift) where high levels of root infection did not result in high levels of tuber infection. Similar notable exceptions between root and tuber infection have been found with Russet Burbank (noted in New Zealand, GB, the USA and elsewhere – for example Eraslam & Turham 1989) and in Pentland Dell (reported in Harrison, 1997).

A laboratory bioassay (Merz *et al.*, 2004) using tissue cultured plantlets and cystosori as inoculum was found valuable as an initial screen. Differences in relative trends of susceptibility between root infection and tuber infection of five selected varieties were found when using naturally infested soil instead of prepared inoculum in the bioassays. This method is not suggested as an alternative to conventional screening, rather it makes the process more efficient.

Screening for resistance under field and glasshouse conditions has given broadly similar results (De Boer, 1991) although the level of disease developing was higher in the glasshouse.

In GB, resistance ratings to powdery scab are given on the standard 1- 9 scale with 1 as highly susceptible. Whilst there is variability in the tests as suggested above, the ratings do provide a valuable guide to risk of powdery scab developing. However, because little is known about variability in populations of *S. subterranea* or whether races or strains exist, as with all disease resistance testing, some caution is required.

4.3.7.2 Use of resistance as a sole method of pest and disease control

Whilst most references to control of powdery scab refer to the need for integrated control (e.g. Burgess & Wale, 1994; Zambolim *et al.*, 1995; Wale *et al*., 2004; Genet *et al.*, 2005), there is acceptance that host resistance is by far the most important single control measure (Wale *et al*., 2004; Merz *et al.*, 2004). This is because other individual control measures only contribute in a minor way to overall control.

However, in GB the spectrum of varietal resistance is poor, with 75% of varieties susceptible or moderately susceptible to powdery scab (Wale, 2002). In addition, the choice of variety grown, particularly in the pre-pack sector, may be dictated by the market.

Extensive evaluations of powdery scab resistance have taken place in countries across the world and in most countries the proportion of susceptible varieties is often high. Thus in New Zealand Falloon *et al*. (2003) when testing 99 varieties and 13 breeding lines found 52% to be moderately or very susceptible. In Peru, Torres *et al*. (1995) when testing 467 varieties and advanced clones found around two thirds were susceptible. In India, Bhattacharya *et al*. (1985) found the majority of 513 varieties or lines were susceptible or moderately susceptible. The predominance of susceptible varieties confirms that historically there has been no specific breeding for resistance.

4.3.7.3 Assessing risks when utilising host plant resistance

The most effective use of host resistance is to target varieties according to risk. The risk of disease developing will depend on the level of inoculum but particularly the occurrence of environmental conditions favourable for infection. Soil-borne inoculum is the most important source of the pathogen and, currently, judging the level of inoculum and interpreting the result is difficult. Some use of previous field history has proved useful but a survey in Northern Scotland suggested that where potatoes had been grown previously the risk was high (Burgess & Wale, 1996). Quantification techniques for soil-borne inoculum are being developed, including PCR assays (van der Graaf *et al*., 2003), although none are yet commercially available. Once field soil can be tested for contamination by *S. subterranea*, more objectivity in field selection and selection of varieties to grow in them can be made. However, environmental conditions are considered the major driver in disease development rather than level of inoculum (Wale *et al*., 2005) and knowledge of soil-borne inoculum alone is not sufficient to minimise risk.

4.3.7.4 Relationship of inoculum level and environment to effectiveness of host plant resistance

Studies on the relationship between inoculum level and disease development are difficult where inoculum cannot be quantified. Thus there are few unequivocal studies suggesting a clear relationship. Wale and Clayton (2002) showed there was no clear relationship between seed-borne inoculum and disease developing on a progeny crop. The same is believed to be true for soil-borne inoculum as environmental conditions are a major driver in disease development rather than level of inoculum (Wale *et al*., 2005). Where environmental conditions are less ideal (e.g. where temperatures are sub-optimal or free water in the soil matrix is limited) inoculum level is likely to take on greater importance.

4.3.7.5 Integration of plant resistance with other control measures

Although variety resistance is the most effective single control measure, integration of control measures is considered essential to optimise control. However, there are few studies that

demonstrate how such integration improves control more than the additive effect of the individual control measures. One study that does demonstrate this was reported by Burgess and Wale (1994) In a series of field experiments, the use of zinc seed and soil treatments were more effective when used in conjunction with moderately resistant varieties than susceptible varieties.

4.3.7.6 Avoidance of resistance breaking races developing in populations of potato pests and diseases

Little is known about the structure of populations of powdery scab. No races have been identified, or demonstrated although their existence is suspected. In a recent research project (Wale *et al*., 2004) genetic variation within *S. subterranea* from different sources in Scotland was investigated using the AFLP technique. The results were difficult to interpret since no methods were found to obtain enough clean DNA of the pathogen. In consequence, the mutability of *S. subterranea* and potential to overcome resistance is unknown. However, whilst in general resistance ratings appear to change little over years, on occasions the resistance rating has changed substantially. For example the variety Nadine was initially rated as resistant (8 – Anon. 1992) but was subsequently rated susceptible (3 – Anon. 1997). This fall in rating may have been a consequence of the method of disease resistance testing. Merz *et al*. (2004) when evaluating a bioassay to screen varietal resistance tested inoculum from different countries and noted a genotype x pathogen interaction. That such an interaction occurs is exemplified by the results of screening under Andean conditions (Torres *et al*., 1995) where Pentland Crown was considered resistant. In the UK this is a highly susceptible variety.

Breeding for resistance to powdery scab has now been incorporated into breeding programmes where the disease has proved to be a major importance (e.g. New Zealand - Genet *et al*., 2005). It has been shown that resistance is heritable but because it appears to be under polygenic control (Wastie, 1991), breeding for resistance is not straightforward. However, Falloon (1997) in a summary of control measures for powdery scab concluded that it may be possible to breed resistant varieties when one parent is resistant. When testing individual seedlings of 19 progenies involving 11 parental varieties, Wastie (1991) found a significant correlation between the mean resistance of a progeny and the phenotypic resistance of its parents.

Mechanisms of resistance are not known but there is some evidence that the mechanism of resistance can be expressed in both roots and tubers. Jellis *et al*. (1987) and Thomson *et al*. (1987) believed there were differences in resistance of root hairs to primary infection and the development of zoosporangia in root hairs. Mechanisms for resistance in tubers have been suggested as related to skin thickness (Wild, 1929) and ability to form a cork layer beneath lesions (Bonde, 1955) but the evidence to support these contentions is limited.

4.3.8. Silver Scurf (*Helminthosporium solani*)

Silver scurf, caused by the fungus *Helminthosporium solani*, is a common disease of potatoes (Read *et al*., 1995) which causes metallic silvery lesions on the surface of tubers (Errampalli *et al*., 2001). These symptoms are mainly cosmetic and they reduce the quality for the fresh-packed market where good appearance is vital. In the processing sector silver scurf can cause excessive water loss leading to shrivelling and shrinkage of tubers (Errampalli *et al*., 2001). In the past, little effort has been made in improving resistance to this disease. The work that has been performed has focussed on developing methods for screening commercial varieties (Mérida *et al*., 1994; Hilton *et al*., 2000; Thomas *et al*., 2005) and among wild species (Rodriguez *et al*., 1995).

The disease epidemiology of silver scurf falls into two distinct phases, field (growing crop) and storage. Infection by *H. solani* initially occurs through infected seed, with conidia infecting newly formed daughter tubers (Jellis & Taylor 1977). This phase provides the initial inoculum. Once in-store, secondary infection takes place (Rodriguez *et al*. 1996) and if condensation events occur then the fungus can sporulate encouraging further infection (Hardy *et al.* 1997). As the conidia are air-borne then infection can spread through out the store.

4.3.8.1. Interpreting published disease and pest resistance

When screening varieties for resistance, testing falls into two categories representing the two phases of the disease cycle. They include tests that occur on the growing crop and assays performed under storage conditions.

Testing under field conditions involves planting seed naturally (Mérida *et al*. 1994) or artificially (Gans *et al*., 2002) infected with *H. solani* into soil. Other tests have used seed tubers planted into soil amended with inoculum of *H. solani* (Mérida *et al*., 1994; Thomas *et al*., 2005). In tests of varieties for GB independent variety trials, healthy seed tubers are dipped in spore suspensions which are then planted into compost and grown in pots in a glasshouse. Symptoms of silver scurf are then assessed on daughter tubers after harvest (Gans *et al*., 2002). Similar methodology is being used in the present independent variety testing, except that after harvest daughter tubers are stored at 12-15^{o}C and high humidity until symptoms develop (Carnegie *et al*., 2006). These methods have been used effectively to detect differences in susceptibility. However, ranking of varieties in order of susceptibility has not always proved repeatable between tests (Gans *et al*., 2002). One problem associated with this sort of testing is that many of the differences in susceptibility can be related to physiological maturity of the variety, with earlier maturing varieties being more susceptible than main crop varieties. When daughter tubers of different varieties are harvested at the same time, because tubers develop quicker in earlier varieties, they have a longer period of time for infection to occur. Despite this, Mérida *et al*. (1994) showed differences in scores between varieties in individual maturity categories suggesting that genetic resistance, independent of physiological maturity does exist.

In storage, testing involves placing tubers in a store and relying on natural infection to occur (Peters & Saunders, 2005) or dipping daughter tubers in a spore suspension of *H. solani* and incubating in humid conditions (Mérida *et al*., 1994; Hilton *et al*., 2000). These tests have also detected differences in susceptibility between varieties. Ranking variety order between tests has also proved possible (Hilton *et al*., 2000) suggesting results from such tests are reliable. When results from such a test on 6 varieties in the USA were compared to maturity of the variety no relationship was found (Mérida *et al*., 1994). These workers speculated that storage assays could be a more reliable way of screening for resistance as factors like physiological maturity can be eliminated.

4.3.8.2 Use of resistance as a sole method of pest and disease control

When using resistance as the sole method of control, high levels of resistance are needed for silver scurf as the tubers are susceptible to infection from tuber initiation through to the end of storage. Among published data there is little evidence that high levels of resistance exist and no reports of complete immunity have been made. In GB, there are no varieties described in the recommended list which have a high resistance rating (Anon. 2005). Ratings vary from 2 for Lady Christl to 7 for Cara (where 1 = very susceptible and 9 = highly resistant). Most varieties including Maris Piper (4) and King Edward (3) are considered susceptible to the disease. Not all GB varieties have resistance ratings for silver scurf. Other work in the UK (Hilton *et al*., 2000) and the USA (Mérida *et al*., 1994) have confirmed a lack of useful resistance among commercially grown varieties.

To be able to use resistance to silver scurf, varieties must be improved through breeding. Good potential sources of resistance to silver scurf do exist among tuber-bearing *Solanum* species (Rodriguez *et al*. 1995). When 212 accessions were screened, a number from species including *Solanum chacoense, S. acaule, S. stoloniferum, S. oxycarpum* and *S. hondelmanni* consistently exhibited low sporulation of the pathogen in laboratory tests.

4.3.8.3 Assessing risks when utilising host plant resistance and relationship of inoculum level and environment to effectiveness of host plant resistance

Factors affecting the development of silver scurf include inoculum level and environmental conditions. In the field, the seed tuber is the main source of inoculum (Jellis & Taylor, 1977) and even a low percentage of silver scurf (<5%) can result in severe infection (Firman & Allen, 1995). More silver scurf developed on daughter tubers grown from seed with newly formed lesions than from tubers where the whole of the periderm was colonised with *H. solani* (Jellis & Taylor, 1977). It is believed that as lesions age sporulation is reduced. How variety resistance and inoculum level on seed interact is not known, but it is possible that where highly susceptible varieties are grown and the majority of tubers are covered in lesions then less symptoms of silver scurf may develop on daughter tubers at harvest.

In storage, high relative humidities and temperatures provide an ideal climate for silver scurf to infect and develop. In order for resistance to be effective in storage, conditions must be regulated. When developing a test for assessing silver scurf under storage conditions, the effect of inoculum and environmental conditions were examined to optimise differences between varieties (Hilton *et al*., 2000). Where tubers were inoculated with a high concentration of spores (10^5 spores per ml^{-1}) no differences in disease score was observed between the more resistant variety Shelagh and the susceptible variety Shula. When the inoculum concentration was lowered (10^4 spores per ml^{-1}) differences between the two varieties were found. It was speculated that resistance could be overridden under high inoculum pressure. Similarly, favourable environmental conditions may also override resistance. Following inoculation a high relative humidity (RH 95%) is needed for infection to occur, but if humidity was maintained at this level for over a month then no differences between varieties were observed (Hilton *et al*., 2000). It was concluded that resistance testing should be conducted by inoculating tubers with a conidial suspension of 10^4 spores per ml^{-1} and then incubated at a temperature of 15C and 95%RH for 1 month followed by 2 months at 85%. In a practical situation this means that resistant varieties will only be effective in storage where inoculum levels are low and environmental conditions do not favour fungal development

4.3.8.2.4 Integration of plant resistance with other control measures

As levels of resistance among commercial varieties are generally low it is vital that available host resistance is used only as part of an integrated approach to controlling silver scurf. Use of host resistance must involve reducing inoculum and maintaining environmental conditions which are not conducive to disease development.

As the main source of initial inoculum is the seed tuber (Jellis & Taylor, 1977) chemical treatment of seed is advisable where silver scurf is found. Fungicide treatments are widely discussed by Errampalli *et al.* (2001). Much of the seed in conventional crops in the UK is treated with imazalil or thiabendazole, which have proved effective in controlling silver scurf on daughter tubers (Tsror & Perelz-Alon, 2002; Carnegie *et al*., 1998). In ware production, into-store treatments also provide good control. To advise UK growers on whether to use a seed or in-store fungicide treatment to control various storage diseases, including silver scurf, a decision tree has been developed (Wale, 2005). It suggests that where variety resistance is good (7-9) then no fungicide need be applied.

However, as only one GB variety meets this criterion this option is limited. It recommends that varieties with a resistance rating of less than 7 should be treated with fungicide.

In storage, to prevent resistant varieties developing silver scurf, residual inoculum must be minimised. As spores of *H. solani* can survive on dust particles, good store hygiene must be maintained through regular cleaning (Clayton *et al*., 2001). If store conditions favour silver scurf development then resistance will again prove ineffective. Holding tubers in cool conditions to prevent condensation events (Hardy *et al*., 1997) will ensure better control.

4.3.8.2.5 Avoidance of resistance breaking races developing in populations of potato pests and diseases

No reports of host resistance being overcome by specific isolates *H. solani* have been reported. As there is only partial resistance to silver scurf it is unlikely that resistance would be overcome. Resistant isolates to thiabendazole have been reported (Hide *et al*., 1988).

4.3.9. Skin spot (*Polyscytalum pustulans*)

4.3.9.1. Interpreting published disease and pest resistance

Potato varieties differ appreciably in their susceptibility to skin spot (Anon., 2005). Historically, the assessment of varietal susceptibility by research workers has sometimes produced inconsistent results depending on the method used (e.g. Nagdy & Boyd, 1965; Bannon, 1975). A relationship was identified between the level of stem base infection and tuber eye infection (Bannon, 1975). Carnegie & Cameron (1983) found that in the field susceptibility to stem base infection was related to the percentage of eyes infected in progeny tubers. Hide and Adams (1980), found that stem lesions and tuber eye infection was related to the level of infection on seed tubers for a susceptible variety. There are complicating physiological factors that may influence the extent of disease development such as stolon length. However, the standardised methodology now used to compare the resistance of varieties in the UK uses pots to grow potatoes in thereby restricting stolon development and incorporates inoculum uniformly into the growing medium. With at least five control varieties and three years of testing, the methodology is believed to be robust enough to produce reliable results.

Unfortunately, of the 97 varieties listed in the NIAB pocket guide 2005 (Anon. 2005), only 33 have ratings determined for them. Several older varieties which are known to be susceptible do not have ratings (e.g. Cultra, Desiree, Record, Kerrs Pink). Of the 33 varieties tested, 7 are rated susceptible (score of 1, 2 or 3) and 9 are resistant (rated 7, 8 or 9). In practice, only those varieties rated 1, 2 or 3 are likely to warrant concern in most growing conditions, although those rated 4 may require some careful attention.

4.3.9.2. Use of resistance as a sole method of pest and disease control

There is scant information in the literature indicating whether resistance to skin spot alone is sufficient to reduce risk in production. However, commercially only a few susceptible varieties, particularly King Edward, Pentland Squire and Sante (and more recently Lady Christl and Lady Balfour) have caused concern in relation to this disease. Whereas, skin spot is detected on some mainstream varieties during seed surveys (SAC commercial results) such as Maris Peer and Maris Piper, the disease is seldom of any significance in the ware crop.

4.3.9.3 Assessing risks when utilising host plant resistance and relationship of inoculum level and environment to effectiveness of host plant resistance

It has been established that soil type affects the impact of *P. pustulans* on emergence and blanking; the effect being worse in heavier soils (McGee *et al*., 1972). In a susceptible variety, King Edward, transmission to the stem base, stolon and progeny tubers depended on the level of seed infection. Dashwood *et al.* (1992) also found a relation between incidence of skin spot on tubers and seed tuber-borne inoculum. However, in heavier soils even a minimum of inoculum caused stolon and tuber infection, whereas transmission by severely infected seed was greatly reduced in light soils (McGee *et al*., 1972).

Many stocks of seed sold for ware production are washed and inspected prior to sale. This is carried out routinely by the larger vertically integrated potato companies. Experienced staff carrying out this operation will usually identify skin spot even at low levels. However, individual seed growers during multiplication may not have the experience to identify skin spot and, at low levels, infection may be missed. For seed production of early or second early varieties, and even many maincrop varieties, where stocks are lifted early, the risk of transmission from year to year may be limited. But with susceptible late bulking maincrop varieties (such as King Edward, Cara, Kerrs Pink, Cultra) which are lifted later, a build up of infection on seed is important to detect.

4.3.9.4 Integration of plant resistance with other control measures

In series of experiments Hide (1991) found that seed tuber treatments reduced stem base infection by skin spot. Similarly, Carnegie & Cameron (1991) found that fungicide treatment of seed tubers soon after harvest reduced the incidence of skin spot.

Because post-harvest treatment of ware with fungicide is restricted by protocols, because skin spot may be exacerbated by CIPC treatment and because irrigation can increase the level of skin spot (Hide, 1987), ware growers are increasingly demanding relative freedom from skin spot (well below the thresholds for certification) on seed. Thus to remove risk, seed growers require to take more care during seed multiplication to ensure the seed stocks to be sold for ware production are free from disease. To achieve this, inspection of seed samples from stocks, particularly of susceptible varieties, should be made and tuber treatments applied where required. In practice, these tuber treatments will either be prophylactic (applied into store or 2-aminobutane treatment early in storage – well before symptom expression) or at grading to reduce the risk of spread from mother to daughter tubers.

Practicing wide rotations to allow inoculum of *P. pustulans* to decline in soil after a potato crop will reduce infection (Carnegie & Cameron, 1990).

Seed growers should practice good hygiene by avoiding build up and spread of dust in store to reduce contamination of seed stocks (Carnegie & Cameron, 1987).

4.3.9.5 Avoidance of resistance breaking races developing in populations of potato pests and diseases

Although there is no published literature on races of *P. pustulans*, to date there has been little indication from experience that strains have developed which can overcome host resistance.

4.3.10. Wart disease (*Synchytrium endobioticum*)

Wart has been reported in most potato growing regions of the world. The disease, is caused by the soil-borne fungus *Synchytrium endobioticum*, and is characterised by wart-like galls produced on several plant parts, including stem bases, stolon buds and tuber eyes. Once contaminated, inoculum in the soil can remain viable for more than 30 years. It is controlled by statutory measures and use of immune varieties. The statutory measures include scheduling contaminated fields. In the early part of last century wart disease was very important and damaging disease in northern Britain. It has now declined in importance and outbreaks are now extremely rare.

4.3.10.1 Interpreting published disease and pest resistance

Field testing of varieties can only distinguish between susceptible and resistant varieties. Laboratory testing can further distinguish between resistance responses. The main method for testing in the laboratory is the Glynne-Lemmerzahl method (Glynne, 1925; Lemmerzahl, 1930) where a ring of warm petroleum is applied to the germinating eyes of a seed tuber. Pieces of wart tissue with abundant sporangia are then added inside the ring. After 2 days incubation the wart tissue is removed and the seed tubers is planted in a pot containing compost. Symptoms are recorded after 18 days. Modifications of this method have also been used for testing resistance (Sharma & Cammack, 1976). Varieties can be grouped into resistant (RG1) and less resistant (RG2) (Noble & Glynne, 1970). Among less resistant varieties development of summer and winter sporangia, the means by which the disease can spread, can develop even though wart symptoms do no develop (Pratt, 1974).

4.3.10.2 Assessing risks when utilising host plant resistance

Most varieties grown in the UK and Europe are immune to this disease, although some of the older varieties such as King Edward are susceptible. Simple testing using the Glynne-Lemmerzahl method mean that susceptible genotypes can be removed from breeding programmes ensuring all new varieties are resistant.

4.3.10.3 Assessing risks when utilising host plant resistance

A number of strains of *S. endobioticum* exist. In the UK, only one strain has been found. However, new strains of the fungus capable of attacking potato varieties now exist in Europe (Anon, 1998)

4.3.10.4 Relationship of inoculum level and environment to effectiveness of host plant resistance

The disease is spread on infected seed tubers or contaminated soil on machinery. It is a disease favoured by cool, wet soil conditions. Most of the outbreaks have occurred in the Midlands or North West England (Anon. 1998).

4.3.10.5 Integration of plant resistance with other control measures

Where outbreaks of wart disease control is managed through official measures. In infected areas no potatoes may be grown and in the surrounding buffer zone only immune varieties can be grown. On prohibited ground potato production can only resume after the pathogen is no longer found to be present. Two fields were recently passed for potato production having been de-scheduled since 1927 and 1966 (Reed, 2004)

4.3.11. Watery Wound Rot (*Pythium ultimum, P. debaryanum* and *P. splenens*)

4.3.11.1. Interpreting published disease and pest resistance

Watery wound rot is an overlooked disease. In the literature it is also referred to as Leak (Priou *et al*., 1997) or shell rot (Blodgett & Ray, 1945). Watery wound rot is a disease of the tubers which causes the flesh to become brown to black with a wet, pulpy texture. Cavities often develop on the inside of the tuber, with the outer flesh remaining firm. This disease is often incorrectly identified as bacterial soft rot and as a consequence its incidence may have been under-estimated. The fungi that cause this disease are common in soil but can only infect through damaged tissue. Infection is most common after harvest particularly when warm and wet (Turkensteen, 2005).

In terms of assessing resistance to watery wound rot only two papers have been published; one from Tunisia, which looks at resistance to *P. aphanidermatum* the pathogen in tropical areas (Priou *et al*., 1997) and one from USA (Salas *et al*., 2003). In both studies tubers were given a standard wound and then inoculated with either oospores (Priou *et al*. 1997) or mycelial plugs (Salas *et al*., 2003). After a period of incubation the depth and width of necrosis was measured. Significant differences between varieties were observed with correlations between different tests (Priou *et al*., 1997). Differences in susceptibility to *P. ultimum* were also observed (Salas *et al*., 2003).

There are no resistance ratings to this disease known for varieties grown in GB.

4.3.11.2 Use of resistance as a sole method of pest and disease control

All varieties tested with *P. aphanidermatum* were susceptible to different degrees (Priou *et al*., 1997). These authors put the varieties into groupings of moderately susceptible, susceptible and highly susceptible. Among the varieties grown in the UK, Desiree and Spunta were susceptible whilst Nicola was highly susceptible.

Similarly, all varieties tested for resistance to *P. ultimum* were susceptible and differences between varieties were limited. Among russett and white varieties significant differences in disease severity were observed but no differences between red varieties were observed. Among the varieties grown in the UK, Kennebec and Atlantic were more susceptible than Shepody and Yukon Gold.

No study, on resistance to *P. ultimum* on varieties grown in the UK has been performed. The value of resistant varieties as a control measure is therefore not known.

4.3.11.3 Assessing risks when utilising host plant resistance, relationship of inoculum level and environment to effectiveness of host plant resistance and integration of plant resistance with other control measures

Control options for this soil-borne pathogen include, crop rotation, planting in a well drained field and avoiding excess irrigation particularly late in the season. Avoiding damage to tubers at harvest will prevent entry points for infection.

4.3.11.4 Avoidance of resistance breaking races developing in populations of potato pests and diseases

No data available

4.4 Pests

Ratings for the resistance of potato varieties to common pests on the European Cultivated Potato Database (www.europotato.org/menu.php) were reviewed. Ratings for a number of potato cultivar characteristics, including pest resistance, are collated from many European sources and presented on a searchable database. There were no ratings available for slugs or wireworm and only one for aphids (the variety Chernigovskii). Ratings are available for five pathotypes of *Globodera rostochiensis* and three pathotypes of *Globodera pallida.* A summary of the ratings for the three pathotypes of *G. pallida* and race 1 of *Globodera rostochiensis* derived from this database in October 2006 is presented in Table 4.13. Races 2 to 5 of *Globodera rostochiensis* are unimportant in the UK and have many fewer records than race 1 (up to 548 for race 5) so have not been included.

TABLE 4.13 RESISTANCE RATINGS TO *GLOBODERA SPP* ON THE EUROPEAN CULTIVATED POTATO DATABASE.

	***G. pallida* race 1**	***G. pallida* race 2**	***G. pallida* race 3**	***G. rostochiensis* race 1**
Number of records	270	343	78	2353
1. % Very low	0.4	0.6	0	0.2
2. % Very low to low	8.9	14.9	16.7	5.2
3. % Low	77.8	53.3	23.1	56.5
4. % Low to medium	4.1	2.0	3.8	0.1
5. % Medium	1.1	2.3	6.4	0.5
6. % Medium to high	6.3	5.2	7.7	0.4
7. % High	0.4	7.3	26.9	25.2
8. % High to very high	1.1	13.1	15.4	5.4
9. % Very high	0	1.2	0	6.5
Mean Score	3.23	4.08	5.08	4.63
Median score	3	3	5	3

There appears to be a lack of host resistance to *G. pallida* race 1 with only 1.5% of records indicating high or better resistance. For all the other pathotypes at least 20% of records indicate high or better resistance. However, for all pathotypes except *G. pallida* race 3 the majority of records indicate low resistance.

4.4.1 Potato cyst nematodes (*Globodera rostochiensis* and *Globodera pallida*)

Often there is confusion between the use of 'resistance' and 'tolerance' of potato varieties to PCN. 'Resistant' varieties of potato are those that limit or prevent PCN from completing its life cycle, whist 'tolerant' varieties are those that can grow and withstand the damage inflicted by PCN (Dale & de Scurrah, 1998).

PCN resistance is now an everyday component of integrated management of PCN, through the use of varieties that are fully resistant to *G. rostochiensis,* and with some varieties that are partially resistant to *G. pallida,* mainly through reducing multiplication rates.

Resistance genes have been identified from a variety of Solanaceous species, and potato breeding specifically for PCN resistance has been underway since the 1940's (Ellenby, 1948). Matters are complicated by each PCN species having several pathotypes (Fleming & Powers, 1998), and some pathotypes within a species being able to 'break' resistance in varieties that were thought to be resistant to a particular species of PCN. There are at least 5 pathotypes of *G. rostochiensis*, and at least 7 pathotypes of *G. pallida* (Canto Saenz & de Scurrah, 1977; Franco & Gonzalez, 1990). Because of this genetic diversity within the two PCN species, finding genes that confer resistance against every pathotype has proved difficult, and pathotyping of PCN populations needs to go hand in hand with potato breeding programmes so that resistance to as many PCN pathotypes as possible is achieved for commercially grown potatoes (Fleming & Powers, 1998).

One of the major sources of PCN resistance was derived from *Solanum tuberosum* ssp. *andigena* (Toxopeus & Huijsman 1953), and is referred to as the H_1 resistance gene (Kort el 1977; Mulder & Van der Wal 1997). Subsequently the potato varieties Maris Piper and Saturna were produced by plant breeders and were the first commercially available varieties with PCN resistance. Since the mid 1960's over 50 potato varieties have been bred for commercial sale which contain the H_1 gene. Further sources of *G. rostochiensis* resistance have been identified in *S. kurtzianum, S. spegazzini* and *S. vernei* (Momeni *et al*., 1969; Rouselle-Bourgeois & Mugniery, 1995; Jacobs *et al*., 1996).

The widespread planting of H_1 resistant varieties led to selection for *G. pallida* populations ahead of *G. rostochiensis* in land routinely used for potato production (Dale & de Scurrah, 1998). Polygenic resistance to *G. pallida* and *G. rostochiensis* had been discovered in *S. vernei* in the 1950's (Mai & Petersen, 1952). Commercial varieties incorporating *S. vernei* derived resistance to both PCN species were available from the mid-1970s, but only a handful of other varieties such as Valor and Sante have been made commercially available since then (Dale & de Scurrah, 1998). Other sources of resistance have been identified, such as the H_3 gene in *S. tuberosum* ssp. *andigena* (Dale & Phillips, 1982), and a range of other *Solanum* species (Hawkes, 1994). There is some hope that varieties containing the H_3 resistance gene will become acceptable in the near future, with Vales Everest showing some promise in the processing potato sector.

The natural decline of PCN through spontaneous cyst hatch in the absence of potatoes has been used as a means of managing PCN populations. However, the rates of decline vary between the two species of PCN, and soil type and other environmental variables can influence the amount of spontaneous hatch that occurs. The development of the BPC "Integrated control of potato cyst nematode (PCN) *Globodera pallida*, the white potato cyst nematode" CD-ROM in 2005, brings together the interactions between resistance, rotations and nematicide so that growers can evaluate for themselves the best management for minimising yield loss and reducing the build up of *G. pallida* populations in individual fields.

4.4.1.1 Interpreting published PCN resistance ratings

There is often some confusion regarding PCN resistance ratings within the UK. In the ratings published by the BPC the ratings are only given for resistance to *G. rostochiensis* (Ro1), and are given as 'r' for resistant or 's' for susceptible. Consequently it can be unclear whether there any varieties that may also have resistance to *G. pallida*. For example, the varieties Nadine, Sante and Valor have resistance to *G. pallida*, but this is not explicitly stated in the ratings.

4.4.1.2 Practical use of PCN resistance

Use of PCN resistant varieties is now a standard course of management for most potato growers in the UK and Europe. However, there is still some misunderstanding regarding the often grey area between 'resistance' and 'tolerance'. If there is a high enough PCN population in the soil, damage

will be caused to the crop as the resistance mechanism in most varieties is in preventing multiplication through the production of cysts. Nematode feeding will still happen so there can be significant yield loss (Whitehead, 1998). There is also the problem of low rates of *G. pallida* multiplication in 'resistant' varieties, which can lead to more *G. pallida* cysts after cropping than there was beforehand. Yield of the current crop may well be protected but problems for future cropping can be caused through this multiplication of PCN. Consequently it is recommended that where *G. pallida* is present, and a resistant variety is to be grown, a nematicide treatment should also be used so that the combination of potato resistance and nematicide will lead to a significant reduction in *G. pallida* population in that field, safeguarding future cropping.

Ideally varieties with resistance to both *G. pallida* and *G. rostochiensis* should be favoured, however, there needs to be a significant shift in the demand for these varieties from the consumer and processors for this approach to be embraced by potato growers in the UK. The problem (particularly in England) of prime potato land being primarily infested with *G. pallida* means that nematicides and soil fumigation are being over utilised to manage this problem. Better choice of cultivars with *G. pallida* resistance coupled with targeted nematicide use and longer rotations can provide a long term solution to this problem.

Some potato varieties show different levels of tolerance to PCN (primarily *G. pallida*), and trials (Keer, 2006) have demonstrated differences between cultivars in terms of their tolerance to PCN damage. Tolerance refers to the ability of the crop to yield well despite the feeding activity of the *G. pallida* nematodes, and is often seen in varieties which produce extensive and vigorous roots. However growing tolerant cultivars such as Cara and Maris Piper whilst not suffering yield loss at low *G. pallida* populations, does lead to a significant increase in the PCN population post-harvest and could make that field unsuitable for potatoes for many years afterwards. Growing tolerant varieties which also have some degree of resistance to *G. pallida* through reducing multiplication (e.g. Vales Everest) could allow field management of PCN without the need for nematicide use (Keer, 2006).

It is essential that the species of PCN present in a field is determined, as this information is needed to allow the correct choice of variety to be undertaken. If *G. rostochiensis* is the only species of PCN present, then there is a wide choice of varieties that can be chosen from that will allow a crop to be grown without the need for a nematicide (unless PCN populations are high) without any threat from PCN multiplication. If *G. pallida* is present, then a smaller pool of resistant cultivars is available, and nematicide use may also be desirable to prevent any significant multiplication during the growing of the crop. There is a possibility that both species of PCN may be present in the same field: for example, 1 in 4 fields positive for PCN in Scotland had both species present (Evans, 1999), and in this case choice of a variety with dual resistance is preferable coupled with nematicide use.

4.4.2 Wireworm (Larvae of Click Beetles including *Agriotes* spp., *Athous* spp., *Conoderus* spp., *Ctenicera* spp., *Limonius* spp., *Melanotus* spp.)

There are several species of wireworm in the UK, but they all cause the same damage to potato tubers. Parker (2002) and Parker and Howard (1999, 2001) reviewed the current status of wireworm in the UK and noted the change in the traditional perception of wireworm as being a problem after grass to the so called 'arable' wireworm problem, where wireworms are being seen in fields that have not been in grass for many years, but may have been in set-aside (Hancock *et al*., 1992; Parker & Howard, 1999). Several approaches to assessing the risk of wireworm have been developed in recent years, through the use of baiting traps (Parker 1994, 1996) and pheromone traps (Parker, 2004) and these have been utilised by potato growers in the UK.

There has been some research into resistance of potatoes to wireworm damage (Jonasson & Olsson, 1994; Olsson & Jonasson, 1995; Kwon *et al*., 1999; Parker & Howard, 2000), and there is evidence that some potato varieties are more resistant to wireworm damage than others. Resistance is linked to glycoalkaloid and sugar levels in the tuber (Jonasson & Olsson, 1994; Olsson & Jonasson, 1995), however high glycoalkaloid levels are undesirable in tubers because of their potential human toxicity.

Recent trials in the UK with commercially available potato varieties have confirmed that some varieties are more resistant to wireworm damage than others (Bayer Crop Science, 2006; Parker & Howard, 2000), but the most resistant variety (Pentland Dell) still had 27% of tubers damaged with a mean of 1.8 holes per tuber, which is commercially unacceptable (Parker & Howard, 2000). The differences between varieties is in the relative amount of damage, as all varieties will be damaged if wireworm is present, it is the magnitude of damage that varies. In all cases, the earlier the potatoes can be harvested, the less time there is for wireworms to cause damage.

4.4.2.1 Interpreting published wireworm resistance ratings

There are no published potato variety resistance ratings to wireworm, only trials and laboratory data that indicate which varieties are more susceptible than others. For example, Parker and Howard (2000) found that Pentland Dell and Maris Piper were less susceptible to wireworm damage than Marfona, King Edward, Estima, Charlotte and Cara. Trials carried out for Bayer CropScience in 2005 indicated that Pentland Dell, Maris Piper and King Edward were some of the least damaged cultivars, whilst Maris Peer and Marfona were the most susceptible to wireworm damage (Bayer CropScience, 2006).

4.4.2.2 Practical use of wireworm resistance

As the commercial tolerance for wireworm damage to tubers is virtually zero, it is not possible to rely on wireworm resistance alone, especially as resistance is linked to glycoalkaloid content in the tubers which itself is variable and dependent on season and soil nitrogen (Love *et al*., 1994). The use of wireworm resistance/susceptibility needs to be linked with insecticide use, the risk of wireworm damage (assessed via pheromone traps, bait traps, previous cropping) and early lifting, as the longer the tubers are in the ground, the greater the damage.

4.4.3 Aphids

Whilst many aphid species may be found on potatoes, a few of these may not be breeding on the crop, simply landing on the foliage, feeding briefly, then flying off to a suitable host-plant for breeding. However, this may be a source of infection by non-persistent viruses such as PVY. Most aphid species will fly into the crop from May onwards, and then begin to deposit live young, forming groups of aphid colonies usually on the undersides of leaves.

The peach-potato aphid (*Myzus persicae*) is the most important aphid species affecting potatoes, as it is an effective vector of viruses and can also cause direct feeding damage if numbers are allowed to build up. The potato aphid (*Macrosiphon euphorbiae*) is usually the first species to appear on potato crops, overwintering on weeds and roses. Whilst it is not as effective a virus vector as the peach-potato aphid, its numbers can build up rapidly on the upper leaves of plants and cause 'false top roll' of the leaves. The glasshouse and potato aphid (*Aulacorthum solani*) may also be found on potato crops. Numbers rarely rise to damaging levels, but it can be a vector of PLRV and PVY viruses.

Whilst these are the most common aphids found on potato crops, other species may occasionally be seen, and the probing of winged aphids that do not breed on potato can transmit non-persistent viruses.

Heavy infestations of aphids can significantly damage the haulm and reduce yield of potatoes. Most damage occurs from July onwards when the tubers are bulking.

4.4.3.1 Interpreting published aphid resistance ratings

There are no resistance ratings published for aphid resistance in potatoes despite their being research into identifying resistance genes and aphid-resistant breeding lines (e.g. Le Roux *et al*., 2004; Pelletier & Clark, 2004; Flanders *et al*., 1997).

Genes and breeding lines have been identified that confer resistance to *M. persicae* and/or *M. euphorbiae* in other solanaceous plants such as aubergines and tomato (Li *et al*., 2006; Kohler & St Clair, 2005; Cooper *et al*., 2004). Aphid resistance has been found in lines of the non-tuber bearing species *Solanum etuberosum* (Novy *et al*., 2002) and use of this germplasm is being looked at to introduce aphid resistance into potato breeding lines.

The resistance mechanisms that have been identified include the salicylic acid signalling pathway in *Mi*-1 mediated aphid resistance in tomato (Li *et al*., 2006, Cooper *et al*., 2004), feeding deterrency (Cooper *et al*., 2004; Kaloshian *et al*., 2000), presence of glandular leaf trichomes or dense hairs on the leaf (Ashouri *et al*., 2001; Musetti & Neal, 1997; Flanders *et al*., 1992; Tingey, 1991)

Much of the research into aphid resistance in potatoes has centred on transgenic potatoes expressing plant protease inhibitors such as oryzacystatin I (Azzouz *et al*., 2005; Rahbe *et al*., 2003), chitinase (Saquez *et al*., 2005), snowdrop lectin (Birch *et al*. ,1999) and jackbean lectin (Gatehouse *et al*., 1999). Transgenic potatoes expressing these traits have reduced fecundity and feeding in several aphid pests of potato.

4.4.3.2 Practical use of aphid resistance

As the most consistent results of aphid resistance have been obtained from transgenic potato plants, their adoption on a practical scale is dependent on the relaxing of government regulations and public opinion on the planting of transgenic crops in the UK. The mechanisms of aphid resistance do not prevent aphids feeding on the leaves; they affect fecundity and reduce (but not eliminate) feeding activity, consequently virus transmission can still occur.

On a worldwide scale resistance to Colorado potato beetle and the aphid-borne viruses themselves are considered a greater priority than a general resistance to aphids alone.

4.4.3.3 Use of resistance as a sole method of aphid control

It is unlikely that aphid resistance would be utilised as a sole method for managing aphids, as the transmission of viruses is still possible using the current aphid resistance mechanisms present in potatoes outlined above, which would make resistant crops impractical for growing seed. Resistant crops could have a role to play in ware potato production in the future.

4.4.4 Slugs

Slugs are a perennial problem in potatoes and there are interspecific differences in terms of the damage that they cause to potato tubers. For example, the grey field slug (*Deroceras reticulatum*) prefers feeding on damaged tubers, whereas keeled slugs (*Tandonia* spp.) quite readily feed on undamaged tubers.

It has been known for many years that there are variety differences in susceptibility to slug damage, and this is reflected in the ratings for slug resistance given in variety lists (Anon. 2005). Resistance can be split into low, intermediate and high, but even varieties rated as having high resistance to slugs can suffer damage, particularly at high slug populations. Also the longer potatoes are in the ground, the greater the damage that can be inflicted by slugs.

Slug resistance in potatoes is thought to be due to the enzymatic oxidation of phenolic compounds in the tuber in response to slug feeding. The activity of the phenolase enzyme was higher in varieties such as Pentland Dell that show some resistance to slug damage. The phenolic compounds present in the tubers are broken down by phenolase to quinones, which reduce slug feeding, growth and survival. Note that it is not the total amount of phenolic compounds in tubers that confers resistance, but the higher activity of the phenolase enzyme that is the important criteria for slug resistance in tubers (Johnston, 1989; Johnston & Pearce, 1994).
The glycoalkaloids present in potato tubers that confer some resistance to wireworm (Jonasson & Olsson, 1994) and snails (Smith *et al*., 2001) do not appear to have an impact on slugs (Johnston & Pearce, 1994).

4.4.4.1 Interpreting published slug resistance ratings

Slug resistance ratings are measured on a scale of 1 (low resistance) to 9 (high resistance), however, there are no potato varieties rated above 5. Varieties commonly referred to having high resistance to slugs such as Pentland Dell, Lady Rosetta and Romano only have BPC ratings of 5; ‘low’ resistance varieties have ratings of 1 (e.g. Maris Piper, Marfona, Claret), and varieties classed as having ‘intermediate’ resistance have ratings of 2-4 (e.g. Cara, Desiree, King Edward).

4.4.4.2 Practical use of slug resistance

Slug management in potatoes requires an integrated approach where the resistance rating of the variety has a role to play, but needs to be considered in the context of other options such as the use and timing of molluscicides (slug pellets), early lifting, irrigation and slug species and populations. Choice of a variety which has some resistance to slugs such as Pentland Dell may well buy some time when it comes to lifting of the crop, but reliance on variety resistance alone is not an option. Molluscicide use is essential if slugs are present in a crop, and slug populations and activity can be monitored through the use of slug trapping. Trials at SAC have demonstrated that the key timing for application of molluscicides is just before the crop canopy meets between the rows. If this timing is missed, slug damage will occur even if molluscides are applied several times after this point. They key is to get the molluscicide slug pellets at the base of the plant just before the crop canopies meet. The increase in humidity below the crop canopy brings slugs up to the surface and their increased activity significantly increases the chance of them feeding on the molluscicide. Further molluscicide applications after this point do reduce the amount of slug damage to tubers, but missing the crop canopy application leads to only a small reduction in damage.

5. Gaps in current knowledge

5.1 Completing the disease and pest resistance ratings for GB varieties

The current pocket guide to varieties of potatoes (Anon. 2005) is incomplete for information on pest and disease resistance ratings. This is particularly true for the six pathogens:

- Black dot
- Black scurf
- Skin spot
- Silver scurf
- Dry rot (*Fusarium coeruleum* and *F. sulphureum*)

Determination of the resistance ratings for these six pathogens on relevant varieties is required. The ratings for black dot and silver scurf are only required for pre-pack varieties, whilst the ratings for the other diseases are probably required only for the top 50 GB varieties.

In addition, the pocket guide provides resistant ratings for 'spraing' which relate to Tobacco Rattle Virus only. There are no ratings available for Potato Mop Top Virus. Ratings for TRV are provided on the standard 1-9 scale and relate to symptom expression in field trials. Work at SCRI has indicated that varieties can be grouped into three:
Resistant – They show no symptoms and virus is not present. These are generally rated 7 to 9 in current variety lists.
Sensitive – They show typical spraing symptoms but virus is rarely found. These are generally rated 1 to 2 in current variety lists.
Susceptible – They show few if any tuber symptoms but virus particles can be detected. These are generally rated 5 to 7 in current variety lists.
Knowing which group a variety falls into is crucial to determine the risk of spread to daughter tubers and which can introduce virus into new locations. SCRI have tested a limited range of varieties but knowledge of the group into which other varieties fall is required particularly for those which may be exported as seed.

In a similar way, resistance ratings for PTMV are required

There are no ratings for resistance to stem canker caused by *Rhizoctonia solani*. Differences in susceptibility among varieties have been recorded in laboratory tests (Kyritsis & Wale, 2002a) and this information would be valuable in assessing risks after planting.

Disease and pest resistance scores are sometimes viewed with suspicion by the potato industry if the ratings do not match personal experience. Disease and pest resistance testing is carried out uniformly at one inoculum level and under the same set of environmental conditions for control and test varieties. Field experience usually relates to a disease or pest occurrence in a range of fields and thus across a range of conditions. None the less, there would be merit in relating disease resistance results to field experience, perhaps by using a grower or agronomist panel with extensive field and QC experience.

5.2 Standardisation of disease resistance testing

The European potato database provides information on disease and pest resistance ratings for varieties on the Common Catalogue. The ratings are provided from a diverse range of sources

including breeders and national governments. Some discrepancy in the ratings are reported in this review. It is apparent for some diseases (e.g. erwinia soft rot, tuber blight and gangrene) that the relative resistances of cutlivars depends on the method used to screen for resistance and which resistance mechanism(s) it assesses. Thus Swiezynski *et al*. (2001b) reported that in a parallel screen of variety resistance to tuber blight involving 5 countries the relative resistances ascribed to individual varieties differed by up to 8 grades (on a 1 to 9 scale) between some countries. Discrepancies in testing methods may be a result of predominance of different strains in different countries, differences in isolates or inoculum levels used in tests, differing testing procedures or subjectivity in the method of assessment.

An attempt to standardise testing procedures would reduce variability across Europe in resistance tests, although it may not stop individual countries continuing to carry out their own tests.

Screening tests used by breeders should be scrutinised by a panel of agronomists to check to what extent the parameters used in the tests, e.g. incubation temperature, reflect current potato production practices.

If the *P. infestans* population in GB changes sufficiently and oospores become significant in the epidemiology of late blight then consideration should be given to amending the methods of screening cultivars for foliar blight resistance to take account of this new source of inoculum, especially at what growth stage and where it initiates infection and also its variability compared with the single strains used currently.

5.3 Determination of strains of pathogens

For a number of pathogens, little is known about the population make-up of the pathogen. Without an understanding of populations of pathogens, it is not possible to carry out disease resistance testing effectively or interpret results of tests effectively. A case in point is that of powdery scab where differences and changes in variety resistance have been recorded. These may be due to changes in the population structure or other factors such as inoculum pressure. The species and strains of common scab are similarly poorly understood. The reaction of a single strain with different varieties can result in differing symptoms. Thus for some diseases there is a need to understand population structure.

5.4 The interaction of inoculum level and resistance

The interaction of inoculum level and resistance rating is poorly understood for many diseases, yet is important for understanding how effective resistance is and how it can be utilised practically. The BPC-SEERAD joint funded black dot project (2004-2007) has demonstrated how knowledge of inoculum can make a substantial difference to utilisation of resistance. Now that diagnostic techniques are available to measure inoculum, further investigation of this relationship is required for a number of key diseases, including common and powdery scab, dry rot and black scurf.

5.5 Understanding the impact of host physiology on disease and pest development

In order to optimise the use of variety resistance, it is important to gain an understanding of the relationship between the physiological status of the host and expression of resistance. The specific aspects of study required depend on the pathogen or pest under consideration but some specific examples include:

- Understanding the link between host maturity and tuber susceptibility to blemish diseases. How does the mechanism of resistance change over the life of the crop, does it vary differently between stems, stolons and tubers?
- Do the chemical and physiological processes involved in skin set alter a variety's susceptibility or resistance to infection?
- Is there a relationship between speed of tuber development at tuber initiation and resistance/susceptibility to infection by common or powdery scab?
- What is the explanation for a strong relationship between maturity and blight resistance?
- When is resistance operating during the growth of a crop, what periods in growth is the crop at risk from infection and what is the effect of resistance on disease development?
- Is there a period when crops are more susceptible to virus transmission and is this affected by differences in resistance?

5.6 Interaction of host nutrition and resistance

Whilst fertiliser rates are prescribed in documents such as RB209, a range of fertiliser rates may be used depending on market (seed, punnet, general ware, baker, crisping etc). The impact of fertiliser, especially nitrogen, on effectiveness of host resistance is poorly understood. Increased nitrogen may increase susceptibility to blight but has been used to reduce blackleg expression. Where appropriate, R&D on specific pests and diseases should incorporate studies of the effects of nutrition on host resistance.

5.7 Integration of control measures

There are few clear examples in the literature how control measures, including plant resistance, can be effectively combined. One exception is the control of potato blight in countries such as The Netherlands in which sophisticated decision suport systems are available to integrate information on variety resistance with other relevant potato crop information to tailor fungicide recommendations. The benefits of such systems to GB growers should be evaluated. With more emphasis on integration in an holistic way, better use can be made of resistance. Thus research into integration of control measures is recommended.

5.8 Mechanisms of resistance

In the literature, there is often scant information on the mechanisms of resistance to pests and diseases. Understanding the mechanisms of resistance is secondary to having the resistance available but where resistance mechanisms are known, it is possible to understand the risks of that resistance will be overcome and thus the durability of resistance. This is particularly true for diseases such as powdery scab where tuber resistance to infection is polygenic but the precise mechanism is not known.

5.9 Damage thresholds for free living nematodes in relation to host resistance and transmission of TRV

The interaction of strains of TRV, weed hosts, population dynamics of vector nematodes and host resistance is poorly understood. Controlled studies in this area will help to unravel the complexities of the disease and how to best use host resistance.

5.10 Understanding partial resistance to *Globodera pallida*

How resistance to *G. pallida* operates in suppression of the nematode and what impact it has on decline rates is important to understand in commercial production. The differences between varieties in their partial resistance and effect on population decline need to be incorporated into Decision Support Systems to improve integrated control.

5.11 Modelling implications of adoption of varieties with good host resistance

If varieties with good host resistance to viruses are adopted what difference does this make to the epidemiology as volunteer plants become increasingly poor sources of virus? Also what changes to seed certification programmes could be made as a result of improved host resistance? Which viruses would make the best targets?

6. Research priorities

The research priorities listed here are a subjective assessment of the gaps in current knowledge (Section 5).

Priority	**Area of research**
1	Completing the disease and pest resistance ratings for GB varieties
2	Integration of control measures
3	The interaction of inoculum level and resistance
4	Understanding partial resistance to *Globodera pallida*
5	Understanding the impact of host physiology on disease and pest development
6	Determination of strains of pathogens
7	Determining damage thresholds for free living nematodes on potatoes in relation to host resistance and transmission of TRV
8	Standardisation of disease resistance testing
9	Interaction of host nutrition and resistance
10	Determining mechanisms of resistance
11	Modelling implications of adoption of varieties with good host resistance

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8. Bibliography and sources of reference

Abdel-Kader D, Seigner L, Zellner M, 2000. Epidemiological field studies on bacterial ring rot (*Clavibacter michiganensis ssp. Sepedonicus*) and brown rot (*Ralstonia solanacearum*) of potato. Gesunde Pflanzen, 52**,** 240-247.

Acuna IA, Strobel GA, Jacobsen BJ, Corsini DL, 2001. Glucosylation as a mechanism of resistance to thaxtomin A in potatoes. Plant Science, 161, 77-88.

Adams MJ, 1980a. The significance of tuber damage and inoculum concentration of *Phoma exigua* var. *foveata* in the development of gangrene in stored potato tubers. Annals of Applied Biology, 95, 31-40.

Adams MJ, 1980b. The role of seed-tuber and stem inoculum in the development of gangrene in potatoes. Annals of Applied Biology, 96, 17-28.

Adams MJ, Hide GA, Lapwood DH, 1980. Relationship between disease level on seed tubers, on crops during growth and in-store potatoes. 5. Seed stocks grown at Rothamsted, 23, 291-302.

Adams MJ, Griffith RL, 1983. The effect of temperature before or after inoculation with *Phoma exigua* var. *foveata* on the development of gangrene in potato tubers. **Plant Pathology, 32,** 325-333.

Aleck JR, Harrison MD, 1978. The influence of inoculum density and environment on the development of potato blackleg. **American Potato Journal, 55,** 479-494.

Allefs JJHM, van Dooijeweert W, de Jong ER, Prummel W, Hoogendoorn J, 1995. The role of the seed tuber in determining partial resistance to potato blackleg caused by *Erwinia* spp. European Journal of Plant Pathology 101, 189-199.

Allefs JJHM, van Dooijeweert W, Prummel W, Keizer LCP, Hoogendoorn J, 1996. Components of partial resistance to potato blackleg caused by pectolytic *Erwinia carotovora* subsp. *atroseptica* and *E. chrysanthemi*. Plant pathology, 45, 486-496.

Allefs JJHM, Muskens MWM, van der Vossen EAG, 2005. Breeding for foliage late blight resistance in the genomics era. In: Potato in progress: science meets practice. Haverkort AJ & Struik PC (Eds). Wageningen Academic Publishers 255-267.

Andrivon, D *et al.* (2003) Development of natural late blight epidemics in pure and mixed plots of potato cultivars with different levels of partial resistance. Plant Pathology 52, 586-594.

Andrivon D, Lucas JM, Ellisseche D, 2003. Development of natural late blight epidemics in pure and mixed plots of potato cultivars with different levels of partial resistance. Plant Pathology 52, 586-594.

Andrivion D, Lucas JM, Guérin C, Jouan B, 1998. Colonization of roots, stolons, tubers and stems of various potato (*Solanum tuberosum*) cultivars by the black dot fungus *Colletotrichum coccodes*. Plant Pathology, 47, 440-445.

Anon., 1980. Potato varieties, 1981. Publication No.72 The Scottish Agricultural Colleges.

Anon., 1981. Classified List of Potato Varieties, England and Wales, 1981/82. National Institute of Agricultural Botany.

Anon., 1992. Potato variety handbook 1993, National Institute Agriculture Botany. 32pp.

Anon., 1997. Potato variety handbook 1998, National Institute Agriculture Botany. 64pp.

Anon., 1998. Potato Wart Disease. http://www.defra.gov.uk/planth/pestnote/pwd.htm

Anon., 2002 Pocket Guide to Varieties of Potatoes 2003. NIAB, Cambridge, 2002.

Anon., 2003. NIAB Organic Vegetable Handbook 2003. Cambridge, UK.

Anon., 2004. Pocket guide to varieties of potatoes 2005. National Institute of Agricultural Botany 218pp.

Anon., 2005. Pocket guide to varieties of potato 2006. National Institute of Agricultural Botany

Anguiz R, Martin C, 1989. Anastomosis groups, pathogenicity and other characteristics of *Rhizoctonia solani i*solated from potatoes in Peru. Plant Disease, 73, 199-201.

Arce P, Moreno M, Gutierrez M, Gebauer M, Dell'Orto P, Torres H, Acuna I, Oliger P, Vebegas A, Jordana X, Kalazich J, Holuigue L, 1999. Enhanced resistance to bacterial infection by *Erwinia carotovora* subsp. *atroseptica* in transonic potato plants expressing the attacin or cecropin SB-37 genes. American Journal of Potato Research, 76, 169-177.

Ashouri A, Michaud D, Cloutier C, 2001. Unexpected effects of different potato resistance factors to the Colorado potato beetle (Coleoptera: Chrysomelidae) on the potato aphid (Homoptera: Aphididae). Environmental Entomology, 30, 524-532.

Azzouz H, Cherqui A, Campan EDM, Rahbe Y, Duport G, Jouanin L, Kaiser L, Giordanengo P, 2005. Effects of plant protease inhibitors, oryzacystatin I and soybean Bowman-Birk inhibitor, on the aphid *Macrosiphum euphorbiae* (Homoptera, Aphididae) and its parasitoid *Aphelinus abdominalis* (Hymenoptera, Aphelinidae). Journal of Insect Physiology, 51, 75-86.

Bagnall RH, 1977a. Potato leafroll virus and potato virus Y – controls or resistance? American Potato Journal, 54, 485-486.

Bagnall RH, 1977b. Resistance to the aphid-borne viruses in the potato. In: Harris KF, Maramorosch K, Aphids as Virus Vectors. New York: Academic Press. 501-526.

Bagnall RH, Tai GCC, 1986a. Potato leafroll virus: evaluation of resistance in potato cultivars. Plant Disease, 70, 621-623.

Bagnall RH, Tai GCC, 1986b. Field Resistance to Potato Virus Y in Potato Assessed by Cluster Analysis. Plant Disease, 70, 301-304

Bain RA, 1985. Factors influencing the resistance of potato cultivars to the gangrene pathogen, *Phoma exigua* Desm. var. *foveata* (Foister) Boerema. PhD thesis, University of Edinburgh, 225 pp.

Bain RA, Lennard JH, Wastie RL, 1987. The influence of cultivar and isolate on the development of gangrene (*Phoma exigua* var. *foveata*) in potato tubers. Annals of Applied Biology. 111, 535-540.

Bain RA, Lennard JH, Wastie RL, 1988. The influence of cultivar and mechanical damage on the relationship between inoculum concentration of *Phoma* exigiua var. *foveata* on potato tubers and gangrene development. Plant Pathology, 37, 265-270.

Bain RA, Perombelon MCM, Tsror L, Nachmias A, 1990. Blackleg development and tuber yield in relation to numbers of *Erwinia carotovora* subsp. *atroseptica* on seed potatoes. Plant Pathology 39, 125-133.

Bain RA, Perombelon MCM (1988) Methods of testing potato cultivars for resistance to soft rot of tubers caused by *Erwinia carotovora* subsp. *atroseptica*. Plant Pathology 37, 431-437.

Bains PS, Bisht VS, Lynch DR, Kawchuk LM, Helgeson JP, 1999. Identification of stem soft rot (*Erwinia carotovora* subspecies *atroseptica*) resistance in potato. American Journal of Potato Research, 76, 137-141.

Bak Henriksen J, 1975. Prevention of gangrene and *Fusarium* dry rot by physical means and with thiabendazole. Proceedings 8th British Insecticide and Fungicide Conference, 603-608.

Balali GR, Neate SM, Scott ES, Whisson EL & Wicks TJ, 1995. Anastomosis group and pathogenicity of isolates from *Rhizoctonia solani* from potato crops in South Australia. Plant Pathology, 44, 1050-1057.

Bannon E, 1975. Susceptibility of potato cultivars to skin spot disease. Potato Research**,** 18**,** 531-538

Banttari EE, Ellis PJ, Khurana SMP, 1993. Management of diseases caused by viruses and virus-like pathogens. In Rowe RC (ed) Potato Health Management. St Pauls, Minnesota: The American Phytopathological Society.

Barker H, Harrison BD, 1985. Restricted multiplication of potato leafroll virus in resistant potato genotypes. Annals of Apllied Biology, 107, 205-212.

Barker H, Reavy B, McGeachy K, 1998. High level of resistance in potato to potato mop top virus induced by transformation with the coat protein gene. European Journal of Plant Pathology, 104, 737-740.

Barker H, Waterhouse PM, 1999. The Development of Resistance to Luteoviruse Mediated by Host Genes and Pathogen-derived Transgenes. In: Smith HG, Barker H, eds The Luteoviridae. Wallingford, UK: CABI Publishing. 169-210.

Barker H, McGeachy KD, Robinson DJ, Ryabov EV, Taliansky ME, 2000. Long distance movement of viral RNA's in the absence of coat protein. Scottish Crop Research Institute Annual Report 1999/2000, pp. 144-146.

Barker H, Woodford JAT, 1992. Spread of potato leafroll virus is decreased from plants of potato clones in which virus accumulation is restricted. Annals of Applied Biology, 121, 345-354.

Bayer CropScience, 2006. http://www.bayercropscience.co.uk/output.aspx?sec=841&con=1251

Bhattacharya SK, Sheo R, Dwivedi R, 1985. Sources of resistance to powdery scab in potatoes. Indian Phytopathology, 38, 174-175.

Bishop AL, Slack SA, 1982a. Effect of cultivar, inoculum dose and strain of *Clavibacter michiganensis subsp. sepedonicus* on symptom development in potatoes. Phytopathology, 72, 1382.

Bishop AL, Slack SA, 1982b. Effect of temperature on the development of ring rot of potato. (Abstract). Phytopathology, 72, 1382.

Blight DP, Hamilton AJ, 1974. Varietal susceptibility to damage in potatoes. Potato Research, 17, 261-270.

Blodgett EC, Ray WW, 1945. American Potato Journal, 22, 250-253.

Bonde R, 1955. The effect of powdery scab on the resistance of potato tubers to late blight rot. Maine Agricultural Experiment Station Bulletin No. 538, 1-11.

Bonde R, Covell M, 1950. Effect of host variety and other factors on pathogenicity of potato ring rot bacteria. Phytopathology, 40, 161-172.

Booth C, 1971. The genus *Fusarium*. Commonwealth Mycological Institute

Boyd AEW, 1952a. Dry-rot disease of the potato. V. Seasonal and local variations in tuber susceptibility. Annals of Applied Biology, 39, 322-329.

Boyd AEW, 1952b. Dry-rot disease of the potato. VII. The effect of storage temperature upon subsequent susceptibility of tubers. Annals of Applied Biology, 39, 351-357.

Boyd AEW, 1952c. Dry rot disease of the potato. IV. Laboratory methods used in assessing variations in tuber susceptibility. Annals of Applied Biology, 39, 322-329.

Boyd AEW, 1972. Potato storage diseases. Review of Plant Pathology, 51, 297-321.

Bradshaw JE, Birch PJR, 2006. Breeding potatoes in Scotland for resistance to late blight. Proceedings Crop Protection Northern Britain, March 2006, Dundee, Scotland. pp249-254.

Bradshaw JE, Bryan GJ, Ramsay G, 2006. Genetic Resources (Including Wild and Cultivated *Solanum* Species) and Progress in their Utilisation in Potato Breeding. Potato Research, 49, 49-65.

Brandolini A, Caligari PDS, Mendoza HA, 1992. Combining resistance to potato leafroll virus (PLRV) with immunity to potato viruses X and Y (PVX and PVY). Euphytica, 61, 37-42.

Brenchley GH, Wilcox HJ, 1979. Potato Diseases. London: Her Majesty's Stationery Office.

Buddenhagen IW, 1986. Bacterial wilt revisited. In: Bacterial wilt disease in Asia and the South pacific. Proceedings of an international workshop, PCARRD, Los Banos, Phillipines, 8-10 Oct 1985. GJ Persley (ed.) ACIAR Proceedings 13, pp 126-143.

Bukhalid RA, Chung S, Loria R, 1998. Nec1, a gene conferring a necrogenic phenotype, is conserved in plant-pathogenic *Streptomyces* spp. and linked to a transposase pseudogene. Molecular Plant-Microbe Interactions, 11, 960-967.

Burgess PJ, Wale SJ, 1994. Development of an integrated control strategy for powdery scab of potatoes. Brighton Crop Protection Conference – Pests and Diseases 1994, 301-306.

Burgess PJ, Wale SJ, 1996. Development of a risk assessment scheme for powdery scab. Proceedings Crop Protection in Northern Britain 1996, 319-324.

Canto Saenz M, De Scurrah MM, 1977. Races of the potato cyst nematode in the Andean region and a new system of classification. Nematologica, 23, 340-349.

Carlisle DJ, Cooke LR, Watson S, Brown AE, 2002. Foliar aggressiveness of Northern Ireland isolates of *Phytophthora infestans* on detached leaflets of three potato cultivars. Plant Pathology, 51, 424-434.

Carling DE & Leiner RH, 1990. Virulence of isolates of *Rhizoctonia solani* AG-3 collected from potato plant organs and soil. Plant Disease, 74, 901-903.

Carnegie S, 2006. Independent variety trials 2005. Annual report to British Potato Council. Project R259.

Carnegie SF, Cameron AM, 1983. Testing potato cultivars for susceptibility to skin spot (*Polyscytalum pustulans*). Potato Research, 26, 69-72.

Carnegie SF, Cameron AM, 1987. Contamination of healthy seed tubers by *Polyscytalum pustulans* and *Phoma exigua var. foveata* in potato stores. Potato Research, 30, 79-88.

Carnegie SF, Cameron AM, 1990. Occurrence of *Polyscytalum pustulans*, *Phoma foveata* and *Fusarium solani* var. *coeruleum* in field soil in Scotland. Plant Pathology, 39, 517-523.

Carnegie SF, Rutheven AD, Lindsay DA, Hall TD, 1990. Effects of fungicide applied to potato tubers at harvest or after grading on fungal storage diseases and plant development. Annals of Applied Biology, 116, 61-72.

Carnegie SF, Cameron AM, 1991. Contamination of seed potato tubers grown from stem cutting or microplant-derived tubers by *Phoma foveata* and *Polyscytalum pustulans* in Scotland: the influence of site and fungicide treatment at harvest. Annals of Applied Biology, 118, 27-38.

Carnegie SF, Cameron AM, Lindsay DA, Sharp E, Nevison IM, 1998. The effect of treating seed potato tubers with benzimadazole and phenylpyrrole fungicides on the control of dry rot and skin blemish diseases. Annals of Applied Biology, 133, 343-363.

Carnegie SF, Cameron AM & Haddon P, 2001. The effect of haulm destruction and harvest on the development of dry rot caused by *Fusarium solani* var. *coeruleum* on potato tubers. Annals of Applied Biology 139, 209-216.

Carputo D, Cardi T, Speggiorin M, Zoina A, Frusciante L, 1997. Resistance to blackleg and tuber soft rot in sexual and somatic interspecific hybrids with different genetic background. American Potato Journal 74, 161-172.

Chand T & Logan C, 1982. Reaction of ten potato cultivars to stem canker and black scurf of potato caused by *Rhizoctonia solani*, Tests of Agrochemicals and Cultivars 3 (Annals of Applied Biology 100 – Supplement), 102-103.

Chand T & Logan C, 1983. Cultural and pathogenic variation in potato isolates of *Rhizoctonia solani* in Northern Ireland. Transactions of British Mycological Society, 81, 585-589.

Chang YC, Tu CC, 1980. Pathogenicity of different anastomosis groups of *Rhizoctonia solani* Kuhn to potatoes. (Abstr.) Journal of Agriculture Research China, 29, 27-33.

Chen Q, Kawchuk LM, Lynch DR, Goettel MS, Fujimoto DK, 2003. Identification of late blight, Colorado potato beetle, and blackleg resistance in three Mexican and two south American wild 2x (1EBN) *Solanum* species. American Journal of Potato Research 80, 9-19.

Clayton RC, Shattock RC, 1995. Reduced fungicide inputs to control *Phytophthora infestans* in potato cultivars with high levels of polygenic resistance. Potato Research, 38, 399-405.

Clayton R, Wale SJ, Blackwood JM & Black S, 2001. Potato store hygiene and disinfection to improve seed and ware quality. British Potato Council Report 62pp.

Collins A, Milbourne D, Ramsay L, Meyer R, Chatot-Balandras C, Oberhagemann P, de Jong W, Gebhardt C, Bonnel E, Waugh R, 1999. QTL for field resistance to late blight in potato are strongly correlated with maturity and vigour. Molecular breeding, 5, 387-398.

Colon LT, Turkensteen LJ, Prummel W, Budding DJ, Hoogendoorn J, 1995. Durable resistance to late blight (*Phytophthora infestans*) in old potato cultivars. European Journal of Plant Pathology, 101, 387-397.

Cooper WC, Jia L, Goggin FL, 2004. Acquired and R-gene-mediated resistance against the potato aphid in tomato. Journal of Chemical Ecology, 30, 2527-2542.

Cother EJ, 1980. Bacterial seed tuber decay in irrigated sandy soils of New South Wales. Potato Research, 23, 75-79.

Cullen DW, Lees AK, Toth IK & Duncan JM, 2002. Detection of *Colletotrichum coccodes* from soil and potato tubers by conventional PCR and real-time quantitative PCR. Plant Pathology, 51, 281-292.

Cullen DW, Toth IK, Pitkin Y, Boonham N, Wlash K, Barker I & Lees AK, 2005. Use of quantitative molecular diagnostic assays to investigate *Fusarium* dry rot in potato stocks and soil. Phytopathology, 95, 1462-1471.

Dale MFB, De Scurrah MM, 1998. Breeding for resistance to the potato cyst nematodes *Globodera rostochiensis* and *Globodera pallida*: strategies, mechanisms and genetic resources. In: Marks RJ and Brodie BB (Eds.) Potato Cyst nematodes. CAB International, Wallingford, pp. 167-195.

Dale MFB, Phillips MS, 1982. An investigation of resistance to the white potato cyst nematode. Journal of Agricultural Science, 99, 325-328.

Dashwood EP, Fox RA, Perry DA, 1992. Effect of inoculum source on root and tuber infection by potato blemish disease fungi. Plant Pathology, 41, 215-223.

Davidson, TMW, 1980. Breeding for resistance to virus disease of the potato (*Solanum tuberosum*) at the Scottish Plant Breeding Station. In: Scottish Plant Breeding Station 59th Annual Report, 100-108.

Davis JR, Sorensen LH, Corsini GS, 1983. Interaction of *Erwinia* spp. and *Fusarium roseum* 'Sambucinum' on the Russet Burbank potato. American Potato Journal, 60, 409-421.

De Boer RF, 1991. Evaluation of potato cultivars in the greenhouse and field for resistance to powdery scab. Australian Journal of Experimental Agriculture, 31, 699-703.

De Boer SH, Slack SA, 1984. Current status and prospects for detecting and controlling bacterial ring rot of potatoes in North America. Plant Disease, 68, 841-844.

De Lindo L, French ER & Kelman A, 1978. *Erwinia* spp. pathogenic to potatoes in Peru. American Potato Journal, 55, 383 (abstract)

Difonzo CD, Ragsdale DW, Radcliffe EB, 1995. Potato leafroll virus spread in differentially resistant potato cultivars under varying aphid densities. American Potato Journal, 72, 119-132.

Dowley LJ, 1972. Varietal susceptibility of potato tubers of *Rhizoctonia solani* in Ireland. Irish Journal of Agricultural Research, 11, 281-285.

Duvauchelle S, Dubois L, Brethenoux C, 2006. French DSS MILPV: Tool for monitoring late blight susceptibility of varieties. Proceedings Ninth Workshop of an European Network for development of an Integrated Control Strategy of potato late blight. Tallinn, Estonia, 19-23 October 2005. PPO Special Report no. 11 105-112.

Ellenby C, 1948. Resistance to the potato root eelworm. Nature, 162, 704.

Errampalli D, Saunders JM & Holley JD, 2001. Emergence of silver scurf (*Helminthosporium solani*) as an economically important disease of potato. Plant Pathology, 50, 141-153.

Eraslam F, Turham G, 1989. Investigations of mycological characteristics of *Spongospora subterranea* (Wallr.) Lageh. In Aegean region and reactions of some potato cultivars and clones. Ege Universitesi Ziraat Fakultesi Dergisi, 26, 313-331.

Evans, K.A. 1999. Potato cyst nematode: why species identification is important. Proceedings Crop Protection in Northern Britain Conference 1999, 243-248.

Evans KA, 2006. Changes in Soil Migratory Nematode Levels over the last decade: Implications for Nematode Management. Proceedings Crop Protection In Northern Britain 2006, 60-74.

Evans KA, Stanley K, Blackwell A. 2006. Tobacco Rattle Virus Prevalence in Scottish Potato Land in 2005. Proceedings Crop Protection In Northern Britain 2006, 285-288.

Fahmy FG, Mohamed MS, 1990. Some factors affecting the incidence of potato brown rot. Assiut Journal of Agricultural Sciences, 21, 221-230.

Falloon RE, 1997. Powdery scab control. Commercial Grower, 52, 16-18.

Falloon RE, Genet RA, Wallace AR, Butler RC, 2003. Susceptibility of potato (*Solanum tuberosum*) cultivars to powdery scab (caused by *Spongospora subterranea f.sp. subterranea*), and relationships between tuber and root infection. Australasian Plant Pathology, 32, 377-385.

Filippov A, Kuznetsova M, Rogozhin A, Spiglazova S, Smetanina T, Belousova M, Kamionskaya A, Skryabin K, Dolgov S, 2006. Increased resistance to late blight in transgenic potato expressing Thaumatin II gene. Proceedings Ninth Workshop of an European Network for development of an Integrated Control Strategy of potato late blight. Tallinn, Estonia, 19-23 October 2005. PPO Special Report no. 11 263-267.

Firman DM & Allen EJ, 1995. Transmission of *Helminthosporium solani* from potato seed tubers and effects of soil conditions, seed inoculum and seed physiology on silver scurf disease. Journal of Agricultural Science, 124, 219-234.

Flanders KL, Hawkes JG; Radcliffe EB, Lauer FI, 1992. Insect resistance in potatoes – evolutionary relationships, morphological and chemical defenses, and ecogeographical associations. Euphytica, 61, 83-111.

Fleming CC, Powers TO, 1998. Potato cyst nematodes: species, pathotypes and virulence concepts. In: Marks RJ and Brodie BB (Eds.) Potato Cyst nematodes. CAB International, Wallingford, pp. 51-57.

Flier WG, Turkensteen LJ, van den Bosch GBM, Vereijken PFG, Mulder A, 2001. Differential interaction of *Phytophthora infestans* on tubers of potato cultivars with different levels of blight resistance. Plant Pathology, 50, 292-301.

Flier WG, van den Bosch GBM, Turkensteen LJ, 2003. Stability of partial resistance in potato cultivars exposed to aggressive strains of *Phytophthora infestans*. Plant Pathology, 52, 326-337.

Flor HH, 1971. Current status of the gene-for-gene concept. Annual Review of Phytopathology, 9, 275-296.

Forbes GA, Chacon MG, Kirk HG, Huarte MA, van Damme M, Distel S, Mackay GR, Stewart HE, Lowe R, Duncan JM, Mayton HS, Fry WE, Andrivon D, Ellisseche D, Pelle R, Platt HW, MacKenzie G, Tarn TR, Colon LT, Budding DJ, Lozoya-Saldana H, Hernandez-Vilchis A, Capezio S, 2005. Stability of resistance to *Phytophthora infestans* in potato: an international evaluation. Plant Pathology, 54, 364-372.

Foxe MJ, 1992. Breeding for viral resistance: conventional methods. Netherlands Journal of Plant Pathology, 98 (Supplement 2), 13-20.

Franco J, Gonzalez A, 1990. A new race of *Globodera pallida* attacking potatoes in Peru. Revue de Nematologie, 13, 181-184.

Fraser RS, (Ed) 1985. Mechanisms of Resistance to Plant Diseases. Advances in Biotechnology, 17.

French ER, de Lindo L, 1979. The erwinias of potato in Peru. In: Developments in Control of Potato Bacterial Diseases. Report of a Planning Conference, pp. 88-93. International Potato Centre, Lima, Peru.

Gans PT, Jellis GJ, Little G, Logan C, Wastie RL, 1991. A comparison of methods to evaluate the susceptibility of potato cultivars to blackleg (caused by *Erwinia carotovora* subsp. *atroseptica*) in the field at different sites. Plant Pathology, 40, 238-248.

Gans PT, Vaughan JE & Thomas JE, 2002. The evaluation of potato cultivar resistance to fungal diseases causing tuber blemishes. Proceedings of Crop Protection in Northern Britain 2002, 263-268.

Gatehouse AMR, Davison GM, Stewart JN, Galehouse LN, Kumar A, Geoghegan IE, Birch ANE, Gatehouse JA, 1999. Concanavalin A inhibits development of tomato moth (*Lacanobia oleracea*) and peach-potato aphid (*Myzus persicae*) when expressed in transgenic potato plants. Molecular Breeding, 5, 153-165.

Genet RA, Falloon RE, Braam W, Wallace AR, Jacobs JME, Baldwin SJ, 2005. Resistance to powdery scab (*Spongospora subterranea)* in potatoes – a key component of integrated disease management. Acta horticulturae, 670, 57-62.

Gergely L, Lonhard M, Proksza P, 2003. Durability of dual resistance of potato varieties to late blight [*Phytophthora infestans* (Mont.) de Bary] and common scab [*Streptomyces scabies* (Thaxt.) Waksman et Henrici]. Acta Phytopathologica et Entomologica Hungarica, 38, 1-6.

Germundsson A, Sandgren M, Barker H, Savekov E, Valkonen JPT, 2002. Initial infection of roots and leaves reveals resistance phenotypes associated with coat protein gene-remediated resistance to potato mop top virus. Journal of General Virology, 83, 1201-1209.

Ghislain M, 2000. Traits and genes: Benefits and Risks. In: Lizarraga L, Hollister A, (Eds). Proceedings of the International Workshop on Transgenic Potatoes for the Benefit of Resource-Poor Farmers in Developing Countries, Manchester UK 5-9 June 2000. Lima, Peru: International Potato Centre.

Gilligan CA, Simons SA & Hide GA, 1996. Inoculum density and spatial pattern of *Rhizoctonia solani* in field plots of *Solanum tuberosum*: effects of cropping frequency. Plant Pathology, 45, 232-244.

Glynne MD, 1925. Infection experiments with wart disease of potatoes *Synchytrium endobioticum* (Schilb.) Perc. Annals of Applied Biology, 12, 34-60.

Goto K, 1981. The relationship between common scab severity and reducing sugar contents in the peel of potato tubers. Potato Research, 24, 171-176.

Graham DC, Harper PC (1966) Effect of inorganic fertilisers on the incidence of potato blackleg disease. European Potato Journal, 9, 141-145.

Griffith RL, 1970. Pathogen, wound type, temperature and gangrene infection. Report Rothamsted Experimental Station, 1969, 165-166.

Griffith RL, Hide GA, Hirst JM, Stedman OJ, 1974. Effects of gangrene (*Phoma exigua*) on potatoes. Annals of Applied Biology, 77, 237-250.

Grunwald NJ, Hinojosa MAC, Covarrubias OR, Pena AR, Niederhauser JS, Fry WE, 2002a. Potato cultivars from the Mexican National Program: Sources and durability of resistance against late blight. Phytopathology, 92, 688-693.

Grunwald NJ, Montes GR, Saldana HL, Covarrubias OAR, Fry WE, 2002b. Potato late blight management in the Toluca Valley: field validation of SimCast modified for cultivars with high field resistance. Plant Disease, 86, 1163-1168.

Hancock M, Ellis S, Green DB, Oakley JN, 1982. The effects of long and short term set-aside on cereal pests. In: Clarke J (Ed.) Set-aside. BCPC Monograph No. 50. pp. 195-200.

Hansen JG, Koppel M, Valskyte A, Turka I, Kapsa J, 2005. Evaluation of foliar resistance in potato to *Phytophthora infestans* based on an international field trial network. Plant Pathology, 54, 169-179

Hardy CE, Burgess PJ, Pringle RT, 1997. The effect of condensation on sporulation of *Helminthosporium solani* on potato tubers infected with silver scurf and held in simulated storage conditions. Potato Research, 40, 169-180.

Harrison BD, 1999. Steps in the development of luteovirology. In: Smith HG, Barker H, eds The Luteoviridae. Wallingford, UK: CABI Publishing. 1–14.

Harrison BD, Cooper JI, 1974. Studies on the nature of the resistance of potato cultivars to tobacco rattle virus. Potato Research, 17, 348-49.

Harrison JG, Searle RJ, Williams NA, 1997. Powdery scab disease of potato – a review. Plant Pathology, 46, 1-25.

Hawkes JG, 1994. Origins of cultivated potatoes and species relationships. In: Bradshaw JE & Mackay GR (Eds.) Potato Genetics. CAB international, Wallingford, pp. 3-42.

Haynes KG, Goth RW, Young RY. 1997. Genotype x environment interactions for resistance to common scab in tetraploid potato. Crop Science, 37, 1163-1167.

Haynes KG, Lambert DH, Christ BJ, Weingartner DP, Douchse DS, Backlund JE, Secor G, Fry W, Stevenson W, 1998, Phenotypic stability of resistance to late blight in potato clones evaluated at eight sites in the United States. American Journal of Potato Research, 75, 211-217.

Haywood AC, El-Nashaar HM, Nydegger U, De Lindo L, 1990. Variation in nitrate metabolism in biovars of *Pseudomonas solanacearum*. Journal of Applied Bacteriology, 69, 269-280.

Healy FG, Bukhalid RA, Loria R, 1999. Characterization of an insertion sequence element associated with genetically diverse plant pathogenic *Streptomyces* spp. Journal of Bacteriology, 181, 1562-1568.

Heil M, Bostock RM, 2002. Induced systemic resistance (ISR) against pathogens in the context of induced plant defences. Annals of Botany, 89, 503-512.

Hide GA, 1987. Effects of irrigation and seed tuber size on yield and infection of potatoes from commercial and healthier seed stocks. Potato Research, 30, 637-649.

Hide GA, Adams MJ, 1980. Relationships between disease levels on seed tubers, on crops during storage and in stored potatoes. 2. skin spot. Potato Research, 23, 215-227.

Hide GA, Read PJ & Sandison JP, 1985. Stem canker (*Rhizoctonia solani*) of maincrop potatoes. I. Development of the disease. Annals of Applied Biology, 100, 105-116.

Hide GA, Read PJ, Firmager JP & Hall SM, 1989. Stem canker (*Rhizoctonia solani*) on five early and seven maincrop potato cultivars. I. Infection of shoots, stolons and tubers. Annals of Applied Biology, 114, 255-265.

Hide GA, Hall SM & Boorer KJ, 1998. Resistance to thiabendazole in isolates of *Helminthosporium solani*, the cause of silver scurf disease of potatoes. Plant Pathology, 37, 377-380.

Hill SA, 1990. Potato viruses. In: Gunn JS, (Ed). Crop Protection Handbook-Potatoes. Farnham, Surrey, UK: British Crop Protection Council.

Hilton AJ, Linton S & Lees AK, 1999. Resistance to potato blemish diseases. Final report for British Potato Council

Hilton AJ, Stewart HE, Linton SL, Nicolson MJ & Lees AK, 2000. Testing of resistance to silver scurf under controlled environmental conditions. Potato Research, 43, 263-272.

Hooker WJ, 1981. Common scab. In: Compendium of Potato Diseases (first edition) St. Paul, Minnesota: The American Phytopathological Society.

Hossain M, Logan C, 1983. A comparison of inoculation methods for determining potato cultivar reaction to blackleg. Annals of Applied Biology, 103, 63-70.

Howell PJ, 1977. Recent trends in the incidence of aphid-borne virus in Scotland. In: Fox RA, ed. Problems of Pest and Disease Control in Northern Britain. Dundee, Scotland: Scottish Horticultural Research Institute. 26-28.

Hughes JC, 1980. Role of tuber properties in determining susceptibility of potatoes to damage. Annals of Applied Biology, 96, 344-345.

Huitema E, Vleeshouwers VGAA, Francis DM, Kamoun S, 2003. Active defence responses associated with non-host resistance of *Arabidiopsis thaliana* to the oomycete pathogen *Phytophthora infestans.* Molecular Plant Pathology, 4, 487-500.

[http://www.oardc.ohio-state.edu/phytophthora/pdfs/MPP03b.pdf]

Inglis DA, Johnson DA, Legard DE, Fry WE, Hamm PB, 1996. Relative resistances of potato clones in response to new and old populations of *Phytophthora infestans*. Plant Disease, 80, 575-578.

Jacobs JME, Vaneck HJ, Horsman K, Arens PFP, Verkerkbakker B, Jacobsen E, Pereira A, Stiekema WJ, 1996. Mapping of resistance to the potato cyst nematode *Globodera rostochiensis* from the wild potato species *Solanum vernei*. Molecular Breeding, 2, 51-60.

Janse JD, Wenneker M, 2002. Possibilities of avoidance and control of bacterial plant diseases when using pathogen-tested (certified) or –treated planting material. Plant Pathology, 51, 523-536.

Jayasinghe V, Chuquillanqui C, Salazar LE, 1989. Modified expression of virus resistance in potato in mixed virus infection. American Potato Journal, 66, 137-144.

Jellis GJ, 1975. Screening of potato clones for resistance to dry rot (*Fusarium solani* var *coeruleum*). Annals of Applied Biology, 81, 417-418

Jellis GJ, 1975b. The susceptibility of potato tuber tissues to infection by *Phoma exigua* var. *foveata*. Potato Research, 18, 116-119.

Jellis GJ, 1978. Determining the susceptibility of potato clones to gangrene (*Phoma exigua* var. *foveata*). Potato Research, 21, 135-143.

Jellis GJ, 1982. Laboratory assessments of the susceptibility of potato tubers to gangrene (*Phoma exigua* var. *foveata*). Plant Pathology, 11, 171-177.

Jellis GJ, Howard HW, 1975. Disease resistance in relation to the quality of seed and ware potatoes. EAPR Triennial Conference Proceedings, 1975, 40.

Jellis GJ & Taylor GS, 1977. The development of silver scurf (*Helminthosporium solani*) disease of potato. Annals of Applied Biology, 86, 19-28.

Jellis GJ, Haslam M, 1980. The incidence and importance of wounds which penetrate the vascular ring. Annals of Applied Biology, 96, 371-374.

Jellis GJ & Starling NC, 1983. Resistance to powdery dry rot (*Fusarium sulphureum*) in potato tubers. Potato Research, 26, 295-301.

Jellis GJ, Phul PS, Starling NC, 1987. Evaluation of potato germplasm for resistance to powdery scab (Spongospora subterranea). Tests of Agrochemicals and Cultivars No.8 (Annals of Applied Biology 110 supplement) 154-155.

Jonasson T, Olsson K, 1994. The influence of glycoalkaloids, chlorogenic acid and sugars on the susceptibility of potato tubers to wireworm. Potato Research, 37, 205-216.

Jones RAC, 1990. Strain Group specific and virus specific hypersensitive reactions to infection with potyviruses in potato cultivars. Annals of Applied Biology, 117, 93-105.

Jones RAC, Harrison BD, 1969. The behavour of potato mop top virus in soil and evidence for its transmission by *Spongospora subterranea.* Annals of Applied Biology, 63, 1-17.

Kaloshian I, Kinsey MG, Williamson VM, Ullman DE, 2000. *Mi*-mediated resistance against the potato aphid *Macrosiphum euphorbiae* (Hemiptera: Aphididae) limits sieve element ingestion. Environmental Entomology, 29, 690-695.

Kaniewski WK, Thomas PE, 2004. The potato story. AgBioForum, 7, 41-46.
[http://www.agbioforum.org]

Kawchuk, L M; Lynch, D R; Kozub, G C; Nelson, G A; Kulcsar, F; Fujimoto, D K (1998) Multi-year evaluation of *Clavibacter michiganensis subsp. sepedonicus* disease symptoms in cultivated potato genotypes. American Journal of Potato Research, **75**, 235-243.

Keer JI, 2006. Assessment of varietal tolerance to potato cyst nematode (PCN) damage. BPC Project Report. Ref. R264.

Kirk WW, Abu-El Samen FM, Muhinyuza JB, Hammerschmidt R, Douches DS, Thill CA, Groza H, Thompson AL, 2005. Evaluation of potato late blight management utilising host plant resistance and reduced rates and frequencies of fungicide applications. Crop Protection, 24, 961-970.

Kohler GR, St Clair DA, 2005. Variation for resistance to aphids (Homoptera: Aphididae) among tomato inbred backcross lines derived from wild Lycopersicon species. Journal of Economic Entomology, 98, 988-995.

Kort J, Ross H, Rumpenhorst HJ, Stone AR, 1977. An international scheme for identifying and classifying pathotypes of potato cyst-nematodes *Globodera rostochiensis* and *G. pallida*. Nematologica, 23, 333-339.

Kriel CJ, Jansky SH, Gudmestad NC, Ronis DH, 1995. Immunity to *Clavibacter michiganense* subsp. s*epedonicus:* screening of exotic Solanum species. Euphytica, 82,125-132.

Kurowski J, Manzer FE, 1992. Re-evaluation of *Solanum* species accessions showing resistance to bacterial ring rot. American Potato Journal,69, 289-297.

Kwon M, Hahm YI, Shin KY, Ahn YJ, 1999. Evaluation of various potato cultivars for resistance to wireworms (Coleoptera: Elateridae). American Journal of Potato Research, 76, 317-319.

LaChance RO, Genereux H, 1963. Effects of fertilisers on symptom expression of ring rot of potatoes. (Abstract). Proceedings Canadian Phytopathological Society, 30, 13.

Langton FA, 1971a. The effects of temperature on the development of gangrene following laboratory inoculation of tubers with *Phoma exigua* var. *foveata*. Physiological Plant Pathology, 1, 477-487.

Lammers JW, Bain RA, Ligertwood GL, Raynor EL (1998). Sporangia production on leaf and stem lesions in relation to cultivar resistance to potato blight (*Phytophthora infestans*). In: Proceedings of the Second Workshop of an European Network for development of an Integrated Control Strategy of potato late blight. Eds. Schepers, HTAM & Bouma E. PAV Special Report no. 3, 85-91.

Langton FA, 1971b. The development of a laboratory test for assessing potato varietal susceptibility to gangrene caused by *Phoma exigua* var. *foveata*. Potato Research, 14, 29-38.

Langton FA, 1972. The reliability of a laboratory test for assessing gangrene susceptibility. Potato Research, l5, 266-268.

Lees AK & Hilton AJ, 2003. Black dot (*Colletotrichum coccodes*): an increasingly important disease of potato. Plant Pathology, 52, 3-12.

Lemmerzahl J, 1930. Neues vereinfachtes Infecktionsverfahren zur Prűfung von Kartoffelsorten auf Krebsfestigkeit. Zűchter, 2, 288-297.

Lennard JH, 1980. Factors influencing the development of potato pink rot (*Phytophthora erythroseptica)*. Plant Pathology, 29, 80-86.

Logsdon CE, 1967. Effect of soil temperature on potato ring rot. American Potato journal, 44, 281-286.

King RR, Lawrence CH, Clark MC, 1991. Correlation of phytotoxin production with pathogenicity of *Streptomyces scabies* isolates from scab infected potato tubers. American Potato Journal, 68, 675-680.

Kyritsis P, 2003. Epidemiology and pathogenesis of mycelial soil-borne *Rhizoctonia solani* AG-3 on potatoes (*Solanum tuberosum*). PhD thesis, Aberdeen University 268 pp.

Kyritsis P, Wale S, 2002a. Effect of mycelial inoculum level and cultivar susceptibility on *Rhizoctonia solani* development on potato stems and seed tubers. Proceedings of BCPC Conference – Pests & Diseases 2002, 761-764.

Kyritsis P, Wale S, 2002b. Effect of soil temperature and moisture, inoculum level and depth of planting on the development of *Rhizoctonia solani* on micropropagated potato seed tubers. Proceedings Crop Protection in Northern Britain 2002, 305-310.

Lapwood DH, Wellings LW, Hawkins JH, 1973. Irrigation as a practical means to control potato common scab (*Streptomyces scabies*): final experiments and conclusions. Plant Pathology, 22, 35-41.

Lapwood DH, Bell F, Harris RI, Hide GA & Adams MJ, 1979. Possibilities of forecasting potato storage diseases from tests on seed tuber and crop samples. Plant Pathology, 28, 181-190.

Lapwood DH, Gans PT, 1984. A method for assessing the field susceptibility of potato cultivars to blackleg (*Erwinia carotovora* subsp. *atroseptica*). Annals of Applied Biology, 104, 315-320.

Lapwood DH, Read PJ, 1986. A comparison of methods of seed tuber inoculation for assessing the susceptibility of potato cultivars to blackleg (*Erwinia carotovora* subsp. *atroseptica*) in the field. Annals of Applied Biology, 109, 287-297.

Lawrence CH, Clark MC, King RR, 1990. Induction of common scab symptoms in aseptically cultured potato tubers by the vivotoxin, thaxtomin. Phytopathology, 80, 606-608.

Le Roux V, Saguez J, Vincent C, Giordanengo P, 2004. Rapid method to screen resistance of potato plants against *Myzus persicae* (Homoptera: Aphididae) in the laboratory. Journal of Economic Entomology, 97, 2079-2082.

Leach SS & Webb RE, 1981. Resistance of selected potato cultivars and clones to *Fusarium* dry rot. Phytopathology, 71, 623-629.

Leach SS & Webb RE, 1993. Evaluation of potato cultivars, clones and a true seed population for resistance to *Rhizoctonia solani*. American Potato Journal, 70, 317-328

Lees AK, de Maine MJ, Nicolson MJ, Bradshaw JE, 2000. Long-day-adapted *Solanum phureja* as a source of resistance to blackleg caused by *Erwinia carotovora* subsp. *atroseptica.* Potato Research, 43, 279-285.

Lees AK & Bradshaw JE, 2001. Inheritance of resistance to *Fusarium sulphureum* in crosses between *S. tuberosum* potato cultivars measured on field and glasshouse grown tubers. Potato Research, 44, 147-152.

Lees AK, Cullen DW, Sullivan L & Nicolson MJ, 2002. Development of conventional and quantitative real-time PCR assays for the detection and identification of *Rhizoctonia solani* AG-3 in potato and soil. Plant Pathology, 51, 293-302.

Li Q, Xie QG, Smith-Becker J, Navarre DA, Kaloshian I, 2006. *Mi-1*-mediated aphid resistance involves salicylic acid and mitogen-activated protein kinase signaling cascades. Molecular Plant-Microbe Interactions, 19, 655-664.

Logan C, 1969. The survival of the blackleg pathogen overwinter. Record of Agricultural Research, Ministry of Agriculture, Northern Ireland, 17, 115-121.

Logan C, Woodward JR, 1971. Pathogenicity differences within *Phoma exigua* var. *foveata* Records of Agricultural Research. Ministry of Agriculture for Northern Ireland, 19, 27-31.

Logan C, Copeland RB, 1979. The effect of time of planting inoculated tubers on the incidence of potato blackleg and gangrene. Annals of Applied Biology, 93, 133-140.

Love SL, Herrman TJ, Thompson-Jones A, Baker TP, 1994. Effect of interaction of crop management factors on the glycoalkaloid concentration of potato tubers. Potato Research, 37, 77-85

Maas PW, 1975. Soil fumigation and crop rotation to control spraing in potatoes. European Journal of Plant Pathology, 81, 138-143.

Maas Geesteranus HP, Vruggink H, (1976) *Erwinia carotovora* (Jones) Bergey *et al.* var. *atroseptica* (Hellmers et Dowson) Dye. Seed tuber infection vs. symptom expression in the field. EPPO Bulletin, 6, 223-224.

Maciel JLN, Duarte V, Silveira JRP, 2004. Population densities of *Ralstonia solanacearum* on potato cultivars in the field. Ciência Rural, 34, 19-24.

Mai WF, Petertson LC, 1952. Resistance of *Solanum ballsi* and *Solanum sucrense* to the golden nematode, *Hereodera rostochiensis* Wollenweber. Science, 116, 224-225.

Maine MJ, Carroll CP, Stewart HE, Solomon RM, Wastie RL, 1993. Disease resistance in *Solanum phureja* and diploid and tetraploid *S. tuberosum* x *S. phureja* hybrids. Potato Research, 36: 1-28.

Malcolmson JF, 1958. Some factors affecting the occurrence and development in potatoes of gangrene caused by *Phoma* solanicola Prill. and Delacr. Annals of Applied Biology, 46, 639-650.

Malcolmson JF, Gray EG, 1968. The incidence of gangrene of potatoes caused by *Phoma exigua* in relation to handling and storage. Annals of Applied Biology, 62, 89-101.

Manzer FE, McKenzie AR, 1988. Cultivar response to bacterial ring rot infection in Maine. American Potato Journal, 65, 333-339.

Marshall B, Barker H, Verall SR, 1988. Effects of potato leafroll virus on the crop processes leading to tuber yield in potato cultivars which differ in tolerance of infection. Annals of Applied Biology, 113, 297-305.

Mcgee DC, Morton AP, Boyd AEW, 1972. Reaction of potato varieties to skin spot (Oospora pustulans) infection and transmission in different soils. Potato Research, 15, 302-316.

Mérida CL & Loria R, 1994. Effects of potato cultivar and time of harvest on the severity of silver scurf. Plant Disease, 78, 146-149.

Merz U, Martinez V, Schwarzel R, 2004. The potential for the rapid screening of potato cultivars (*Solanum tuberosum*) for resistance to powdery scab (*Spongospora subterranea*) using a laboratory assay. European Journal of Plant Pathology, 110, 71-77.

Mishra KK, Srivastava JS, 2001. Screening potato cultivars for common scab of potato in a naturally infested field. Potato Research, 44, 19-24.

Mizuno N, Yoshida H, Nanzyo M, Tadano T, 1998.Chemical characterisation of conducive and suppressive soils for potato scab in Hokkaido, Japan. Soil Science and Plant Nutrition, 44, 289-295.

Mizuno N, Nizamidin K, Nanzyo M, Yoshida H, Amano Y, 2003.Judging conducive soils from clay mineralogical properties and soil chemical method to suppress potato common scab. Soil microorganisms, 57, 97-103.

Mndolwa D, Boshop G, Corsini D, Pavek J, 1984. Resistance of potato clones to the green peach aphid and potato leaf roll virus. American Potato Journal, 61, 713-722.

Molina JJ, Harrison MD, 1980. The role of *Erwinia carotovora* in the epidemiology of potato blackleg. II. The effect of soil temperature on disease severity. American Potato Journal, 57, 351-363.

Momeni DA, Plaisted RL, Peterson LC, Harrison MB, 1969. The inheritance of resistance to the golden nematode (*Heterodera rostochiensis*) in *Solanum famatinae* and *S. neohawkesii*. American Potato Journal, 46, 128-131.

Montarry J, Corbiere R, Lesueur S, Glais I, Andrivon D, 2006. Does selection by resistant hosts trigger local adaptation in plant-pathogen systems? Journal of Evolutionary Biology, 19, 522-531.

Moreno C, Mosquera PF, 1983. Changes in the probing behaviour of alate forms of *Myzus persicae* (Sulzer) on seven potato varieties and the disemmination of PVY and PLRV viruses. Revista Colombiana de Entomologia, 9, 31-36. (Through CAB Direct Record No. 19850524291)

Mulder A, Van der Wal AF, 1997. Relationship between potato cyst nematodes and their principal host. I. A literature review. Potato Research, 40, 317-326.

Murphy AM, d. Jong H, Tai GCC, 1995. Transmission of resistance to common scab from the diploid to the tetraploid level via 4x-2x crosses in potatoes. Euphytica, 82, 227-233.

Musetti L, Neal JJ, 1997. Resistance to the pink potato aphid, *Macrosiphum euphorbiae*, in two accessions of *Lycopersicon hirsutum f. glabratum*. Entomologia Experimentalis et Applicata, 84, 137-146.

Naerstad R, 2002. Exploitation of cultivar resistance in potato late blight disease management and some aspects of variation in *Phytophthora infestans*. PhD thesis, Agricultural University of Norway. 112 pp.

Nagdy GA, Boyd AEW, 1965. Susceptibilty of potato varieties to skin spot (*Oospora pustulans*) in relation to the structure of skin and eye. European Potato Journal, 8**,** 200-214.

Natsume M, Taki M, Tashiro N, Abe H, 2001. Phytotoxin production and aerial mycelium formation by *Streptomyces scabies* and *S. acidiscabies in vitro*. Journal of General Plant Pathology, 67, 299-302.

Navarre DA, Thomas PE, Brown CR, Kachroo P, 2003. Systemic acquired resistance in potato. Acta Horticulturae, 619, 177-181.

Nelson GA, 1982. *Corynebacterium sepedonicum* in potato: effect of inoculum concentration on ring rot symptoms and latent infection. Canadian Journal of Plant Pathology, 4, 129-133.

Nelson GA, Kozub GC, 1983. Effect of total light energy on symptoms and growth of ring rot-infected red Pontiac potato plants. American Potato Journal, 60, 461-468.

Nelson GA, Kozub GC, 1987. Effect of temperature and latent viruses on atypical ring rot symptoms of Russet Burbank potatoes. American potato Journal, 64, 589-597.

Nielsen LW, 1979. The epidemiology and control of potato brown rot. American Potato Journal, 56, 474.

Noble M, Glynne MD, 1970. Wart disease of potatoes. F.A.O Plant Protection Bulletin 18, 125-135.

Novy RG, Nasruddin A, Ragsdale DW, Radcliffe EB, 2002. Genetic Resistances to potato leafroll virus, potato virus Y, and green peach aphid in progeny of *Solanum etuberosum*. American Journal of Potato Research, 79, 9-18.

Olsson K, Jonasson T, 1995. Genotypic differences in susceptibility to wireworm attack in potato – Mechanisms and implications for plant breeding. Plant Breeding, 114, 66-69.

Osusky M, Osuska L, Hancock RE, Kay WW, Misra S, 2004. Transgenic potatoes expressing a novel cationic peptide are resistant to late blight and pink rot. Transgenic Research, 13, 181-190.

Park TH, Vleeshouwers VGAA, Kim JB, Hutten RCB, Visser RGF, 2005. Dissection of foliage and tuber late blight resistance in mapping populations of potato. Euphytica, 143, 75-83

Parker A, 1986. Evaluation of potato cultivar resistance to common scab, black scurf and tuber cracking. Tests of Agrochemicals and Cultivars 7 (Annals of Applied Biology 108 – Supplement), 158-159.

Parker WE, 1994. Evaluation of the use of food baits for detecting wireworms (*Agriotes* spp. Coleoptera, Elateridae) in fields intended for arable crop production. Crop Protection, 13, 271-276.

Parker WE, 1996. The development of baiting techniques to detect wireworms (*Agriotes* spp, Coleoptera: Elateridae) in the field, and the relationship between bait-trap catches and wireworm damage to potato. Crop Protection, 15 (6): 521-527.

Parker WE, 2004. Wireworms: end-user evaluation of click beetle pheromone traps as an aid to risk assessment. BPC Project Report No.2004/3.

Parker WE, Howard JJ, 1999. Wireworm biology, risk assessment and control. British Potato Council Project Report.

Parker WE, Howard JJ, 2001. The biology and management of wireworms (*Agriotes* spp) on potato with particular reference to the United Kingdom. Agricultural & Forest Entomology, 2, 85-98.

Parker WE, Howard JJ, 2001. Assessment of the relative susceptibility of potato cultivars to wireworm (*Agriotes* spp.) damage. Tests of Agrochemicals and Cultivars, 21, 15-16.

Parmeter JR, Sherwood RT & Platt WD, 1969. Anastomosis grouping among isolates of *Thanatephorus cucumeris*. Phytopathology, 59, 1270-1278.

Pelletier Y, Clark C, 2004. Use of reciprocal grafts to elucidate mode of resistance to Colorado potato beetle (*Leptinotarsa decemlineata* (Say)) and potato aphid (*Macrosiphum euphorbiae* (Thomas)) in six wild Solanum species. American Journal of Potato Research, 81, 341-346.

Perombelon MCM, 1973. Studies on the epidemiology and etiology of blackleg (*Erwinia carotovora* var. *atroseptica*) of potato. PhD Thesis, University of Dundee.

Perombelon MCM, 1992. Potato blackleg – Epidemiology, host pathogen interaction and control. Netherlands Journal of Plant Pathology, 98, 135-146.

Perombelon MCM, Kelman A, 1987. Blackleg and other potato diseases caused by soft rot erwinias: a proposal for a revision of terminology. Plant Disease, 71, 282-285.

Perombelon MCM, Kelman A, 1980. Ecology of the soft rot erwinias. Annual Review of Phytopathology, 18, 361-387.

Perombelon MCM, Lowe R, 1979. Blackleg etiology: field studies. 25th Annual Report of the Scottish Horticultural Research Institute, 1978, p. 86.

Perombelon MCM, Lumb VM, Zutra D, 1987. Pathogenicity of soft rot erwinias to potato plants in Scotland and Israel. Journal of Applied Bacteriology, 63, 73-84.

Perombelon MCM, Lumb VM, Zutra D, Hyman LJ, Burnett EM, 1989. Factors affecting potato blackleg development. In: Proceedings of the NATO Advanced Research Workshop on the Interaction of Genetic and Environmental Factors in the Development of Vascular Wilt Diseases of Plants, Cape Sunion, Greece, May 1988 (Ed. By EC Tjames & CH Beckman), pp. 421-431.

Peters J & Lees AK, 2004. Regional variation among *Fusarium* spp. causing dry rot of potato. Final report for British Potato Council. Project Ref. 807/223

Peters J, Saunders S, 2005. Storage assessments of independent variety trials. Final report for British Potato Council Ref 807/223.

Peters JC, Hilton AJ, Brierley J, Gladders P, Bradshaw N, Wale SJ, 2005. Control of black dot on potatoes: Integrating agronomy and storage. Proceedings of 16th Triennial Conference of European Association of Potato Research, Bilbao, Spain, 372-374.

Peters RD, Sturz AV, Matheson BG, Arsenault WJ, Malone A, 2001. Metalaxyl sensitivity of isolates of *Phytophthora erythroseptica* in Prince Edward Island. Plant Pathology, 50, 302-309.

Peters RD, Sturz AV, Arsenault WJ, 2004. Tuber response of six cultivars to inoculation with *Phytophthora erythroseptica*, the causal agent of pink rot. Canadian Journal of Plant Pathology, 26, 63-69.

Peters RD, Sturz AV, 2001. A rapid technique for the evaluation of potato germ plasm for susceptibility to pink rot. Plant Disease, 85, 833-837

Phillips SL, Shaw MW, Wolfe MS, 2005. The effect of potato variety mixtures on epidemics of late blight in relation to plot size and level of resistance. Annals of Applied Biology, 147, 245-252.

Pietkiewicz JB, Jellis GJ, 1975. Laboratory testing for the resistance of potato tubers to gangrene (*Phoma exigua* var. *foveata*). Phytopathologische Zeitschrift, 83, 289-295.

Pratt M, 1974. Assessment of resistance to potato wart disease. Potato Research, 17, 357.

Priou S, Triki MA, Mahjoub Mel, Fahem M & Souibgui M, 1997. Assessing potato cultivars in Tunisia for susceptibility to leak caused by *Pythium aphanidermatum*. Potato Research, 40, 399-406.

Qaim M, 1998. Transgenic virus-resistant potatoes in Mexico: Potential socioecomic implications of North-South biotechnology transfer. ISAAA Briefs NO. 7. Ithaca, NY state:ISAAA [http://www.isaaa.org/bin/Briefs/7/briefs.htm]

Rahbe Y, Deraison C, Bonade-Bottino M, Girard C, Nardon C, Jouanin L, 2003. Effects of the cysteine protease inhibitor oryzacystatin (OC-I) on different aphids and reduced performance of *Myzus persicae* on OC-I expressing transgenic oilseed rape. Plant Science, 164, 441-450.

Read PJ, 1991. The susceptibility of tubers of potato cultivars to black dot (*Colletotrichum coccodes* (Wallr.) Hughes). Annals of Applied Biology, 119, 475-482.

Read PJ, Storey RMJ & Hudson DR, 1995. A survey of black dot and other fungal tuber blemishing diseases in British potato crops at harvest. Annals of Applied Biology, 126, 249-258.

Reed PJ, 2004. De-sheduling of previously infested sites for potato wart disease in England and Wales. Proceedings of Crop Protection in Northern Britain, Dundee 2004, p 333-336.

Reglinski T, Lyon GD, Newton AC, 1994a. Induction of resistance mechanisms in barley by yeast-derived elicitors. Annals of Applied Biology, 124, 509-517.

Reglinski T, Lyon GD, Newton AC, 1994b. Assessment of the ability of yeast-derived elicitors to control powdery mildew in the field. Journal of Plant Disease and Protection, 101, 1-10.

Ribeiro Do Vale FX, Parleviet JE, Zambolim L, 2001. Concepts in Plant Disease Resistance. Fitopatologia Brasileira, 26, 577-589. http://www.scielo.br/scielo.php?script=sci_arttext&pid=S0100-15820010003000001andIng=en&nrm=iso]

Rich AE, 1983. Potato diseases. New York: Academic Press

Rizvi SAH, Raman KV, 1983. Effect of glandular trichomes on the spread of potato virus Y and potato leafroll virus in the field. In: Hooker WJ, ed. Proceedings of the International Congress Research for the Potato in the Year 2000. Lima, Peru: International Potato Centre. 163-163.

Robert Y, 1999. Epidemiology of potato leafroll disease. In: Smith HG, Barker H, eds The Luteoviridae. Wallingford, UK: CABI Publishing. 221-228.

Robert Y, Woodford JAT, Ducray-Bourdin DG, 2000. Some epidemiological approaches to the control of aphid-borne virus diseases in seed potato crops in northern Europe. Virus Research, 71, 33-47.

Robinson DJ, Dale MFB, 2004. Factors Affecting the Incidence of Potato Spraing Disease Caused by Tobacco Rattle Virus. Proceedings Crop Protection in Northern Britain 2004, 243-248.

Rodriguez D, Secor GA, Gudmestad NC, Grafton K, 1995. Screening tuber-bearing *Solanum* species for resistance to *Helminthosporium solani*. American Potato Journal, 72, 669-679.

Rodriguez D, Secor GA, Gudmestad NC, Franci LJ, 1996. Sporulation of *Helminthosporium solani* and infection of potato tubers in seed and commercial storages. Plant Disease, 80, 1063-1070.

Rogers WG, Killick RJ, 1974. Factors affecting the assessment of resistance of potatoes to gangrene. Annals of Applied Biology, 81, 51-59.

Ross H, 1986. Potato breeding – problems and perspectives. Advances in Plant Breeding, 13.

Rouselle-Bourgeois F, Mugniery D, 1995. Screening tuber-bearing *Solanum* ssp. for resistance to *Globodera rostochiensis* R1A Woll. and *G. pallida* Pa2/3 Stone. Potato Research, 38, 241-249.

Rykbost KA, Charlton BA, James SR, Hane DC, 1998. Evaluation of clones of Russet Norkotah, Russet Burbank and Shepody. In Research in the Klamath Basin 1998 Annual Report. 48-55. [http://oregonstate.edu/dept/kes/transgenic98.pdf]

Saguez J, Hainez R, Cherqui A, Van Wuytswinkel O, Jeanpierre H, Lebon G, Noiraud N, Beaujean A, Jouanin L, Laberche JC, Vincent C, Giordanengo P, 2005. Unexpected effects of chitinases on the peach-potato aphid (*Myzus persicae* Sulzer) when delivered via transgenic potato plants (*Solanum tuberosum* Linne) and in vitro. Transgenic Research, 14, 57-67.

Salas B, Secor GA, Taylor RJ & Gudmestad NC, 2003. Assessment of resistance of tubers of potato cultivars to *Phytophthora erythroseptica* and *Pythium ultimum*. Plant Disease, 87, 91-97.

Salas B, Stack RW, Secor GA & Gudmestad NC, 2000. The effect of wounding, temperature and inoculum on the development of pink rot caused by *Phytophthora erythroseptica*. Plant Disease, 84, 1327-1333.

Sandgren M, 1995. Potato mop-top virus (PMTV): distribution in Sweden, development of symptoms during storage and cultivar trials in field and glasshouse. Potato Research, 38, 379-389.

Scholte K, 1989. Effects of soil-borne *Rhizoctonia solani* Kuhn on yield and quality of ten potato cultivars. Potato Research, 32, 367-376.

Seppanen E, 1982. Further studies on varietal differences in resistance to potato gangrene. Ann. Agric. Fenn., 21, 155-161.

Serrano C, Arce-Johnson P, Torres H, Gebauer M, Gutierrez M, Moreno M, Jordana X, Venegas A, Kalazich J, Holuigue L, 2000. Expression of the chicken lysozyme gene in potato enhances resistance to infection by *Erwinia carotovora* subsp. *atroseptica*. American Journal of Potato Research, 77, 191-199.

Sharma R, Cammack, 1976. A modification of the Glynne-Lemmerzahl method for testing resistance of potato varieties to wart disease, *Synchytrium endobioticum* (Schilb.) Perc. Potato Research, 19, 165-167.

Shaw DS & Kiezebrink DT, 2005. Late blight resistance in Sarpo clones: an update.

Sherf AF, 1944. Infection experiments with potato ring rot and the effect of soil temperature on the disease. American Potato Journal, 21, 27-29.

Sicilia C, Copeland RB, Cooke LR, 2002. Comparison of the interactions of *Erwinia carotovora* ssp. *atroseptica* with *Phytophthora infestans*, *Phoma foveata* and *Fusarium coeruleum* in rotting potato tubers. Potato Research, 45, 237-246.

Slack, SA, 2001a. Potato leafroll virus. In: Stevenson WR, Loria R, Franc GD, Weingartner, eds. Compendium of Potato Diseases. St Paul, Minnesota: The American Phytopathological Society. 63-64.

Slack, SA, 2001b, Potato Virus X. In: Stevenson WR, Loria R, Franc GD, Weingartner, eds. Compendium of Potato Diseases. St Paul, Minnesota: The American Phytopathological Society. 69.

Solomon-Blackburn RM, Barker H, 2001a. A review of host-major-gene resistance to potato viruses X, Y, A and V in potato: genes, genetics and mapped locations. Heredity, 86, 8-16.

Solomon-Blackburn RM, Barker H, 2001b. Breeding virus resistant potatoes (*Solanum tuberosum*): a review of traditional and molecular approaches. Heredity, 86, 17-35.

Speiser B, Tamm L, Amsler T, Lambion J, Bertrand C, Hermansen A, Ruissen MA, Haaland P, Zarb J, Santos J, Shotton P, Wilcockson S, Juntharathep P, Ghorbani R, Leifert C, 2006. Improvement of late blight management in organic potato production systems in Europe: Field tests with more resistant potato varieties and copper based fungicides. Biological Agriculture and Horticulture, 23, 393-412.

Starling NC, 1987. Potatoes. Powdery scab and viruses. Plant Breeding Annual Report for 1986, 50-51.

Staples RC, 2004. Race non-specific resistance for potato late blight. Trends in Plant Science, 9, 5-6.

Starr GH, 1947. The effect of different concentrations of bacterial suspensions used in inoculations upon subsequent ring rot symptoms in the potato plant. American Potato Journal, 24, 151-156.

Stead DE, Elphinstone JG, Pemberton AW, 1996. Potato brown rot in Europe. Brighton Crop Protection Conference: Pests & Diseases – 1996, 1145-1152.

Stead D, Wale S, 2004. Non-water control measures for potato common scab. British Potato Council Research Review, Reference R248, 49pp.

Stevenson WR, Loria R, Franc GD, Weingartner DP. Eds. Compendium of Potato Diseases. St Paul, Minnesota: The American Phytopathological Society.

Stewart HE, Bradshaw JE, Wastie RL, 1994. Correlation between resistance to late blight in foliage and tubers in potato clones from parents of contrasting resistance. Potato Research, 37, 429-434.

Stewart HE, Wastie RL, Bradshaw JE, 1996. Susceptibility to *Phytophthora infestans* of field- and glasshouse-grown potato tubers. Potato Research, 39, 283-288.

Stewart HE, Bradshaw JE, Pande B, 2003. The effect of the presence of R-genes for resistance to late blight (*Phytophthora infestans*) of potato (*Solanum tuberosum*) on the underlying level of field resistance. Plant Pathology, 52, 193-198.

Sturtz AV, Ryan DAJ, Coffin AD, Matheson BG, Arsenault WJ, Kimpinski J, Christie BR, 2004. Stimulating disease suppression in soils: sulphate fertilisers can increase biodiversity and antibiosis ability of root zone bacteria against *Streptomyces scabies*. Soil Biology and Biochemistry, 36, 343-352.

Sutton MW, Robinson DL, Dixon GR, Wilson F, 1988. The growth and yield of virus-tested and visually healthy commercial narcissus stocks from different localities. Journal of Horticultural Science, 63, 479-487.

Swiezynski KM, Domanski L, Zarzycka H, Zimnoch-Guzowska E, 2000. The reaction of potato differentials to *Phytophthora infestans* isolates collected in nature. Plant Breeding, 119, 119-126.

Swiezynski, KM, Chrzanowska M, Domanski L, Zimnoch-Guzowska E, 2001. Comparison of resistance evaluation in potato variety assessment. Potato Research 44, 25-31.

Swiezynski KM, Zimnoch-Guzowska E, 2001a. Breeding potato cultivars with tubers resistant to *Phytophthora infestans*. Potato Research, 44, 97-117.

Swiezynski KM, Chrzanowska M, Domanski L, Zimnoch-Guzowska E, 2001b. Comparison of resistance evaluation in potato variety assessment. Potato Research, 44, 25-31.

Tamada T, Harrison BD, Roberts IM, 1984. Variation amongst British isolates of potato leafroll virus. Annals of Applied Biology, 98, 107-116.

Taylor RJ, Salas B, Secor GA, Riveria V, Gudmestad NC, 2002. Sensitivity of North American isolates of *Phytophthora erythroseptica* and *Pythium ultimum* to mefenoxam (metalaxyl). Plant Disease 86, 797-802.

Thomas JE, Gans PT & Kenyon DM, 2005. Resistance to disease in commercial potato cultivars and its use in disease management. Aspects of Applied Biology, 76, 121-126.

Thomson AJ, Lacey CND, Negus RM, Squire AM, Taylor L, Jellis GJ, Boulton RE, Martlew ED,

Tingey WM, 1991. Potato glandular trichomes – defensive activity against insect attack. ACS Symposium Series, 449, 126-135.

Torres H, Pacheco MA, French ER, 1995. Resistance of potato to powdery scab (*Spongospora subterranean*) under Andean field conditions. American Potato Journal, 72, 355-363.

Toxopeus HJ, Huijsman CA, 1953. Breeding for resistance to potato root eelworm. Euphytica, 2, 180-186.

Trognitz BR, 1998. Inheritance of resistance in potato to lesion expansion and sporulation by *Phytophthora infestans*. Plant Pathology, 47, 712-722.

Toxopeus HJ, 1958. Some notes on the relations between field resistance to *Phytophthora infestans* in leaves and tubers and ripening time in *Solanum tuberosum* subsp. *tuberosum*. Euphytica, 7, 123-130.

Tsror L, Erlich O, Hazanovsky M, 1999. Effect of *Colletotrichum coccodes* on potato yield, tuber quality, and stem colonisation during spring and autumn. Plant Disease, 83, 561-565.

Tsror L, Petrez-Alon I, 2002. Reduction of silver scurf on potatoes by pre- and post-storage treatment of seed tubers with imazalil. American Journal of Potato Research, 79, 33-37.

Turkensteen LJ, 2005. Leak (Watery wound rot). In Potato diseases ed: Mulder A & Turkensteen LJ, Nivap Holland & Arderappelwereld, pp 34-35.

Turkensteen LJ, Flier WG, 2003. Host and non-host resistance against *Phytophthora infestans*, the causal organism of late blight of potatoes and tomatoes. In: van Hintum ThJL, Ledeba A, Pink D, Schut JW, eds. Eucarpia Leafy Vegetables. [http://www.leafyvegetables.nl/papers.htm]

Umaerus V, 1975. Screening methods for resistance to mechanical damage. Proceedings European Association for Potato Research Triennial Conference, 1975, 16-17.

Valkonen JPT, 1994. Natural genes and mechanisms for resistance to viruses in cultivated and wild potato species (*Solanum* spp). Plant Breeding, 112, 1-16.

Valkonen JPT, Jones RAC, Slack SA, Watanabe KN, 1996. Resistance specificities to viruses in potato: standardisation of nomenclature. Plant Breeding, 155, 433-438.

Van de Graaf P, Lees AK, Cullen DW, Duncan JM, 2003. Detection and quantification of *Spongospora subterranea* in soil, water and plant tissue samples using real-time PCR. European Journal of Plant Pathology, 109, 589-597.

Visker MHPW, Keizer LCP, Budding DJ, van Loon LC, Colon LT, Struik PC, 2003. Leaf position prevails over plant age and leaf age in reflecting resistance to late blight in potato. Phytopathology, 93, 666-674.

Visker MHPW, van Raaij HMG, Keizer LCP, Struik PC, Colon LT, 2004. Correlation between late blight resistance and foliage maturity type in potato. Euphytica, 137, 311-323.

Visker MHPW, Heilersig HJMB, Kodde LP, van de Weg WE, Voorrips RE, Struik PC, Colon LT, 2005. Genetic linkage of QTLs for late blight resistance and foliage maturity type in six related potato progenies. Euphytica, 143, 189-199

Wale S, 2005. Potato seed treatment decision trees. BASF information leaflet

Wale SJ, 2002. Potential for chemical control of *Spongospora subterranea*, cause of powdery scab of potatoes and vector of Potato mop-top virus. The BCPC Conference – Pests and diseases 2002, pp 129-134.

Wale SJ, Clayton R, 2002. Relationship between powdery scab inoculum on seed and disease development on progeny tubers. Proceedings Crop Protection in Northern Britain 2002, 287-292.

Wale SJ, Hilton A, Burgess P, Lees A, van der Graaf P, 2004. Evaluating integrated control of powdery scab. Proceedings Crop Protection in Northern Britain 2004, pp 285-290.

Wale SJ, Hilton A, Tait M & Mitchell T, 2004. The control of disease of potato caused by soil-borne *Rhizoctonia solani* using soil application of azoxystrobin. Proceedings Crop Protection in Northern Britain 2004, 291-296.

Wale SJ, Sutton M, 2004. Supplement to the literature review on non-water control measures for potato common scab. British Potato Council Research Review, Reference R248, 31pp.

Wale SJ, van de Graaf P, Lees A, 2005. Epidemiology, autecology and control of *Spongospora subterranea* cause of potato powdery scab. Project report reference 807/211. British Potato Council. 123pp.

Wale S, Bradshaw N, Hilton A, Gladders P, Lees A, Brierley J & Peters J, Unpublished.. Developing effective control measures for the control of black dot. Second year report for the British Potato Council.

Walker RR, Wade GC, 1976. Epidemiology of potato gangrene in Tasmania. Australian Journal of Botany, 24, 337-347.

Walters DR, Newton A, Lyon G, 2005. Induced resistance: helping plants to help themselves. Biologist, 52, 28-33. [http://www.iob/downloads/919.pdf]

Wander JGN, Spits HG, Kessel GJT, 2006. Exploiting potato late blight cultivar resistance using DSSs: 4 years of field experiments. In: Proceedings of the Ninth Workshop of an European Network for development of an Integrated Control Strategy of potato late blight. Eds. Westerdijk, CE & Schepers, HTAM. PPO Special Report no. 11, 113-119.

Wang AiXia, Lazarovits G, 2005. Role of seed tubers in the spread of plant pathogenic *Streptomyces* and initiating potato common scab disease. American Journal of Potato Research, 82, 221-230.

Wastie RL, 1984. Inoculating plant material by jet inoculation. Plant Pathology, 33, 61-63.

Wastie RL, 1991. Breeding for resistance. In: Ingram, DS & Williams PH (eds) Advances in Plant pathology. Volume 7, Academic Press, London, pp. 193-224.

Wastie RL, 1991. Resistance to powdery scab of seedling progenies of *Solanum tuberosum*. Potato Research, 34, 249-52

Wastie RL, Caligari PDS, Wale SJ, 1988. Assessing the resistance of potatoes to powdery scab (*Spongospora subterranea* (Wallr.) Lagerh). Potato Research, 31, 167-71.

Wastie RL, Stewart HE & Brown J, 1989. Comparative susceptibility of some potato cultivars to dry rot caused by *Fusarium sulphureum* and *F. solani* var. *coeruleum*. Potato Research, 32, 49-55.

Wastie RL, Bradshaw JE, 1993. Inheritance of resistance to *Fusarium* spp. in tuber progenies of potato. Potato Research, 36, 189-193.

Weingartner DP, 2001. Potato mop-top virus. In: Stevenson WR, Loria R, Franc GD, Weingartner, eds. Compendium of Potato Diseases. St Paul, Minnesota: The American Phytopathological Society. 64-65

Weingartner DP, Shumaker JR, 1987, Development of bacterial wilt and tuber brown rot in six potato cultivars. American Potato Journal, 64, 465.

Wellving A, 1976. Studies on the resistance of potato to storage rots caused by *Phoma exigua* var. *foveata* and *Fusarium* spp. The Swedish Seed Association, Svalov, Sweden. 131 pp.

Westra AAG, Arneson CP, Slack SA, 1994. Effect of interaction of inoculum dose, cultivar and geographic location on the development of foliar symptoms of bacterial ring rot of potato. Phytopathology, 84, 410-415.

Westra AAG, Slack SA, 1994. Effect of interaction of inoculum dose, cultivar, and geographic location on the magnitude of bacterial ring rot symptom expression in potato. Phytopathology, 84, 228-235.

Whitehead AG, 1998. Plant Nematode Control. CAB International, Wallingford.

Wicks TJ, Davoren CW, Hall BH, 2000. Fungicidal control of *Phytophthora erythroseptica*: the cause of pink rot on potato. American Journal of Potato Research 77, 233-240.

Wiersema HT, 1977. A quick laboratory test for assessing resistance of potato tubers to gangrene (*Phoma exigua* var. *foveata*). Potato Research, 20, 267-268 (abstract).

Wild N, 1929. Untersuchungen uber den Pulverschorf der Kartoffelknollen (*Spongospora subterranea* (Wallr.) Johnson). Phytopathologische Zeitschrift, 1, 367-452.

Wilson CR, 2001a. Variability within clones of potato cv. Russet Burbank to infection and severity of common scab disease of potato. Journal of Phytopathology, 149, 625-628.

Wilson CR, 2001b. Development of extreme resistance to common scab. Potato Australia, 12, 28.

Woodhall JW, 2004. Characterisation of *Rhizoctonia solani* anastomosis groups and their pathogenicity to potato. PhD Thesis. Harper Adams University College, Newport, Shropshire, UK.

Xenophontos S, Robinson DJ, Dale MFB, Brown DJF, 1998. Evidence for persistent symptomless infection of some potato cultivars with tobacco rattle virus. Potato Research, 41, 255-265.

Zadina J, Dobias K, Horackova V, 1975. Resistance to *Streptomyces scabies* (Thaxt.) Waksman et Henrici in the varieties of the world collection. [Czech]. Ochrana Rostlin, 11, 195-204.

Zambolim L, Parizza P, Matsuoka K, Vale FXR do, Chaves GM, 1995. Powdery scab of potato. Fitopatologia Brasileira, 20, 5-12.

Zimnoch-Guzowska E, Lebecka R, Pietrak J, 1999. Soft rot and blackleg reactions in diploid potato hybrids inoculated with *Erwinia* spp. American Journal of Potato Research, 76, 199-207.

Zimnoch-Guzowska E, Marczewski W, Lebecka R, Flis B, Schafer-Pregl R, Salamini F, Gebhardt C, 2000. QTL analysis of new sources of resistance to *Erwinia carotovora* ssp. *atroseptica* in potato done by AFLP, RFLP, and resistance-gene-like markers. Crop Science, 40, 1156-1167.

Appendix 1

A1 Evaluating risk from variety disease and pest resistance ratings

The table below provides an interpretation of resistance ratings that may be made by a grower for varieties for a specific market. It suggests how the information on plant resistance may be used and what actions a grower may make to minimise risks.

A1.1 Maris Peer & Desiree for seed

	Maris Peer		**Desiree**	
	Rating	**Comment**	**Rating**	**Comment**
Foliage blight	4	Moderately susceptible for both foliage and tubers. Little opportunity for reducing blight programme in relation to rating	4	Moderately susceptible for both foliage and tubers. Little opportunity for reducing blight programme in relation to rating
Tuber blight	4		5	
Blackleg	4	Moderately susceptible. Blackleg could be an issue. Look out for levels of blackleg in previous crop and rotting of tubers in store. Avoid knocking off sprouts at planting	4	Moderately susceptible. Blackleg could be an issue. Look out for levels of blackleg in previous crop and rotting of tubers in store. Avoid knocking off sprouts at planting
Common scab	5	Moderately susceptible. Cannot avoid scab in a dry season in an unirrigated crop but of lower risk in a dry seed field than Desiree	4	Moderately susceptible. Lesions tend to be large when they develop and very obvious. Place in heavier soils to minimise risk of common scab but be aware of later harvest.
Powdery scab	6	Moderately resistant. Can still be infected if wet conditions occur at tuber initiation and inoculum is high but risk is much lower than for many varieties and could be considered for a field with known contamination	7	Moderately resistant. Can still be infected if wet conditions occur at tuber initiation and inoculum is high but risk is much lower than for many varieties and could be considered for a field with known contamination
Gangrene	6	Moderately resistant. As small round seed tubers damage may be lower thus reducing risk. Take action with fungicides at harvest if disease seen in seed at planting	4	Moderately susceptible. When gangrene was a major disease Desiree could be badly infected. Check seed for the disease at planting and apply fungicide after harvest if seen. Take care to avoid damage at harvest.
PLRV	4	Moderately susceptible. Aphid control important and insecticide applications are crucial and best timed when vector aphids arrive in the crop	4	Moderately susceptible. Aphid control important and insecticide applications are crucial and best timed when vector aphids arrive in the crop
PVY	3	Susceptible. As PVY is rapidly transmitted from aphids it is crucial to monitor the crop for aphid colonization and apply insecticides when the first vectors arrive	7	Resistant. Risk of PVY is much lower than Maris Peer but because the variety is moderately susceptible to PLRV careful timing of insecticide use will still be required.
Spraing	3	Susceptible. Whilst not a major issue for most seed crops, testing soil for spraing prior to planting would be	3	Susceptible. Whilst not a major issue for most seed crops, testing soil for spraing prior to planting

		advisable.		would be advisable.
Black dot	-	No rating determined. However, as a second early it is usually one of the first crops to be lifted as the risk would be lower. However, if seed from the farm has a history of black dot a soil test would be useful	-	No rating determined. Red skinned varieties show silvering from black dot more than white skinned potatoes. Desiree may also be lifted latish. If seed from the farm has a history of black dot a soil test would be useful
Black scurf	-	No rating determined but known to develop black scurf. Take action against seed-borne Rhizoctonia if threshold exceeded. Amistar for soil-borne inoculum not advised on seed crops which normally have a wide rotation	-	No rating determined but known to develop black scurf. Take action against seed-borne Rhizoctonia if threshold exceeded. Amistar for soil-borne inoculum not advised on seed crops which normally have a wide rotation
Skin spot	-	No rating determined. Experience indicates that Maris Peer can develop skin spot and a careful watch is needed during seed multiplication for evidence of the disease. Effective fungicide use is the key control measure when present	-	No rating determined. Experience indicates that Desiree can develop skin spot and a careful watch is needed during seed multiplication for evidence of the disease. Effective fungicide use is the key control measure when present
Silver scurf	-	No rating known. Maris Peer can be infected and whilst silver scurf is not part of certification, disease may still develop. However, as it is generally harvested early, silver scurf should not be a problem if drying and good storage is available	-	No rating known. Silver scurf is very obvious on the red skin. Whilst silver scurf is not part of certification, disease development does mar a good sample. Seed treatment and careful drying and storage are crucial
Dry rot (*F. coeruleum*)	-	No rating known. Experience suggests that dry rot can be a problem for Maris Peer. Watch carefully for dry rot during multiplication and use fungicides effectively when found – even at low levels	-	Experience suggests that dry rot is not a major problem with Desiree (cf. Gangrene). Watch for dry rot during multiplication and use fungicides effectively when found – even at low levels
Dry rot (*F. sulphureum*)	-	No rating and little experience in Scotland. As for *F. coeruleum*, watch for dry rot developing during seed multiplication	-	No rating and little experience in Scotland. As for *F. coeruleum*, watch for dry rot developing during seed multiplication
Slugs	4	Not a major issue for seed and the variety matures earlier and is often lifted earlier meaning lower risk	4	Not a major issue for seed but it is later maturing and generally grown on heavier land. Single dose of slug pellets may be justified to minimise damage if season is wet.
PCN Ro1	S	Soil is tested for freedom from PCN in seed production	S	Soil is tested for freedom from PCN in seed production
PCN Pallida	S	Soil is tested for freedom from PCN in seed production	S	Soil is tested for freedom from PCN in seed production

A1.2 Maris Piper for ware

	Maris Piper	
	Rating	**Comment**
Foliage blight	4	Moderately susceptible for both foliage and tuber blight. Little opportunity for reducing blight programme in relation to rating
Tuber blight	5	
Blackleg	5	Moderately susceptible. Blackleg can occur in Maris Piper but is not usually a major issue. Check seed before planting for rots. Try to lift early and ventilate after lifting before keeping dry during storage. A blackleg tuber test is probably of limited value
Common scab	1	Very susceptible. In an unirrigated situation, select a field with a moisture retentive soil and preferably a pH as low as possible. Otherwise, irrigation is the only way to limit this disease. With restrictions on water use, ensure that the water is available and will be effectively scheduled
Powdery scab	3	Susceptible. If the spring is wet, Maris Piper can be badly affected by powdery scab. Like common scab, the key to reducing powdery scab is to select a field with a low risk. This is not an easy task as it relies on a knowledge of field history. Effective scheduling of irrigation should reduce risk of over-watering and hence development of powdery scab
Gangrene	6	Moderately resistant. This disease has declined in recent years and with this level of resistance, there should be little concern about gangrene unless it was evident on seed at planting
PLRV	4	Moderately susceptible. However, providing the seed is certified and has a low level of virus the only need for aphid control is where aphid damage is likely or a portion of the crop is being home-saved. If the seed to be planted is home-saved carry out a virus test to ensure it has low levels of virus
PVY	2	Very susceptible. As for PLRV above
Spraing	5	Moderately susceptible. Spraing is generally not a problem with Maris Piper but it may be worth testing soil before planting and considering nematicide use if TRV spraing detected
Black dot	4	Susceptible. If the farm has a history of black dot and/or the rotation is less than 7 years, aim to lift early to minimise risk. Consider a soil test to determine the level of soil-borne inoculum. Careful scheduling of irrigation and use of azoxystrobin where the risk is high will reduce black dot development. Consider swopping fields if the risk is considered high.
Black scurf	6	Moderately resistant. Whilst the rating is relatively high, this relates to black scurf and the impact of Rhizoctonia can still be substantial on tuber quality. Take action against seed-borne Rhizoctonia if threshold (1%) exceeded. If soil-borne inoculum is considered a high risk, perhaps because of previous crop history, a short rotation and/or the persistence of volunteers consider Amistar soil treatment
Skin spot	4	Susceptible. A careful examination is needed of seed to be planted. Effective fungicide treatment is the key control measure when found. Take greater care if long term storage or CIPC use is planned
Silver scurf	4	Susceptible. Maris Piper can be badly infected and disease may develop where care is not taken. As with all varieties, silver scurf should not be a problem with early harvesting, ventilation after harvest and dry storage
Dry rot (*F. coeruleum*)	3	Very susceptible. Dry rot (both species) can be very serious with Maris Piper. Watch carefully for the presence of dry rot on seed and apply a fungicide seed treatment when found – even at low levels
Dry rot (*F. sulphureum*)	2	
Slugs	2	Very susceptible. Slugs are a major issue for ware growers. Plan a programme slug pellets applications
PCN Ro1	R	Maris Piper is resistant to G. rostochiensis and this species of PCN presents no risk
PCN Pallida	S	Maris Piper is susceptible to G. pallida and soil should be tested prior to planting. If low levels are present either consider an alternative field or use a nematicide