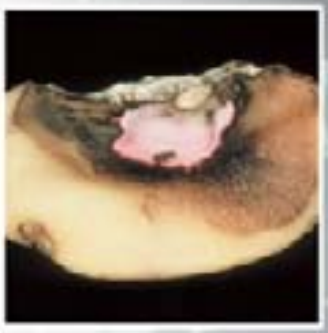
Diseases, Pests and Disorders of Potatoes

A Colour Handbook

Stuart Wale
H.W. (Bud) Platt
Nigel Cattlin



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Diseases, Pests and Disorders of Potatoes

A Colour Handbook

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Holt Studios: Images of Agriculture and Plant Science

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We dedicate this book to all who are involved with scientific investigation, education, extension, phytosanitary regulation, management and dealing with the consequences of potato diseases and pests. It is hoped that the material provided is beneficial.

We also acknowledge the dedicated interest and kind support of Shirley Jo-Ann Platt (1951–2007), Sue Wale, and others who have, over the years, provided assistance to all those who deal directly with potato disease and pest issues.

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Preface

DESPITE THE FACT that potatoes are immensely important as a food crop worldwide, and that the diseases, pests and disorders that affect them have received a great deal of investigation, there are few texts that have attempted to describe and encompass the crop protection problems in any depth. Only relatively recently has it been possible to reproduce high- quality colour photographs, integrated within text, in books. The *Colour Handbook* is one of the first to achieve this for the potato crop, and covers the majority of diseases, pests and disorders that occur.

High-quality photographs are important where accurate visual diagnosis is required. Where possible, and where they were available, 'typical' symptoms are represented here. Of course, there is always variation in the symptoms of any potato problem, but we have tried to find photographs that will help to distinguish one disease, pest or disorder from another.

Identification by comparison with photographs is only a start to confirming diagnosis. Confirming that the observed symptoms accord with a verbal description is an essential next step on the road to correct identification. In this book, each disease, pest or disorder has a detailed description of the symptoms and, where relevant, the vectors that transmit the disease.

While photographic and written symptoms can support an initial diagnosis, its confirmation will depend on isolating the micro-organism/pest involved or, in the case of disorders, establishing that no living pest or disease is involved. Absolute confirmation would depend on reproducing the symptoms, by infecting a healthy plant with the isolated micro-organisms or pest. However, disorders are more difficult to reproduce. This last step of absolute confirmation is not often undertaken, unless a problem is perceived as a new one, or a new strain of a disease or pest is suspected. Our intention in this book is to help readers reach the initial level of diagnosis.

Once you have confirmed the problem, if you are not familiar with it, some background will be required. By supplying information about the economic importance, life cycle and control of each disease, pest or disorder, we hope that an appreciation can be achieved. The selected references provided can act as a basis for further research, especially if there is little access to scientific literature databases. For some diseases and pests, scientific knowledge is published continuously, and it is impossible to include the most up-to-date references. For other problems, especially disorders, there has been little study or published work and, as a result, some of the references may be old.

This book is intended as a practical guide and tool. We expect and hope that it will be carried around and referred to in the field, as well as in the laboratory, on an everyday basis. We hope that it will prove a valuable asset to all who use it.

STUART WALE, BUD PLATT & NIGEL CATTLIN

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Introduction

PRAY FOR PEACE and grace and spiritual food for wisdom and guidance, for all these are good. But don't forget the potatoes.

John Tyler Pettee, 1822–1907

WHAT I SAY IS, if a man really likes potatoes, he must be a pretty decent sort of fellow.

AA Milne, 1882–1956

We began this venture with great enthusiasm and interest in not only bridging the 'waters' between our respective continents but also to provide a practical and useful reference tool for anyone involved with the major factors that impact on potato production, handling and storage worldwide. The format of the book provides a durable tool that is portable for field, vehicle or indoor reference. It is our sincere hope that as you use this book, you will find that we have fulfilled our objectives.

The World Bank and FAO have ranked the potato crop the fourth most important in the world and the most important non-cereal crop. Potatoes are grown in every continent and in over 130 countries. Through breeding and advanced agronomic practices, the crop can be grown under almost all climatic conditions. Although potatoes can be grown from true seed, most of the world's production stems from vegetatively propagated tubers. This makes it stand apart from other major world crops, such as cereals. Struik and Wiersema (1999) list seven aspects distinguishing the potato from other major world food crops:

- It has a low multiplication rate.
- Seed tuber costs are high.
- As a vegetative crop it is prone to 'seed degeneration'.
- Seed tubers usually show innate dormancy after harvest.
- Tubers on the same stem show a wide range in size, dry matter content, dry matter composition and physiological condition.
- In many regions potatoes are grown in different seasons of the year.
- A wide range of suitable techniques for the production of high-quality seed potato tubers is available.

It is the vegetative propagation of potatoes that exposes it to challenge from serious pest and disease attack. Unlike graminaceous crops, many diseases can be transmitted in seed potatoes, and there is a continuous challenge to generate healthy basic seed. The low multiplication rate means that several generations of seed are needed to reduce the unit seed price to acceptable levels. Throughout the seed multiplication phase tubers are prone to invasion by diseases that are carried from generation to generation on, or in, the seed tuber. Perhaps the most demonstrable seed-borne agents that affect vigour, yield and quality of potatoes are the viruses. These can debilitate potato production, and sophisticated propagation techniques and certification schemes are in place around the world to ensure freedom from viruses and other pathogens.

Additional features that distinguish the potato from other crops also impact on susceptibility to attack. The considerable variation in characteristics from tuber to tuber means that they may differ in susceptibility, and therefore consistency in applying control measures can be difficult. In countries where crops are grown in different seasons, a different range of pest and disease problems can occur, and this also presents the crop protectionist with different challenges.

Another difference between potatoes and graminaceous crops is that the storage organ, the tuber, which contains around 80% water and an ideal blend of nutrients, is highly suited as a food source for pests and microorganisms. Thus unless great care is taken, there is potential for huge losses in storage facilities. Losses of 100% have occurred, even in developed countries where, for example, bacterial soft rot has taken hold soon after harvest. Whilst storage in some countries is based on simple technology, in other countries great

advances including effective insulation, ventilation and refrigeration have substantially limited the devastating losses of the past.

Despite these technological improvements and removal of fears about sufficiency of supply, losses in storage facilities are still high because there has been a change in emphasis toward quality. What concerns potato pathologists and zoologists in the developed countries today is the production of potato tubers with excellent skin finish or ideal processing qualities. Many of the diseases and pests that impact on these quality factors were minor pathogens in the past but can make the difference between profit and loss now.

That is not to say that major diseases and pests do not threaten potato production. Potato late blight (*Phytophthora infestans*) continues to dominate. The widespread application of fungicides has reduced the disease to manageable proportions in developed countries, but the potential of the pathogen to mutate or produce new sexual strains, adapt and overcome efforts to suppress it has confounded generations of scientists. In recent years, this tendency has resulted in new strains of the pathogen which attack potato crops across North America (Goodwin *et al*., 1994a, 1994b, 1995, Peters *et al*., 1998) while, in Europe, more virulent strains are causing growers in some countries to more than double the number of fungicide applications (Flier, 2002).

There is a worldwide effort to tackle potato late blight, and the sharing of knowledge about the biology of the pathogen is proceeding at an unprecedented rate. But other diseases are also of great importance, even if they are on a more localized scale. For example, brown rot (*Ralstonia solanacearum*) can be devastating in tropical and sub-tropical countries. Potato cyst nematodes (*Globodera rostochiensis* and *G. pallida*) have become major pests in parts of Europe. Large quantities of nematicide are used annually to suppress these pests, with potentially severe environmental consequences. Countries free from these devastating nematode problems take immense precautions to avoid importing them with seed tubers.

Identification and diagnosis are critical to implementing effective control measures. Luckily, there are many technical aids available to scientists, field scouts, agronomists and farmers to assist identification. An overview of these is given in the next chapter. However, visual diagnosis remains the first line of identification and is a skill that is honed with training and practice. With an increasing emphasis on molecular aspects of plant pathology and zoology, there are fewer plant pathologists throughout the world who are field-based and have developed the skill of diagnostics. We hope that books like this will encourage scientists not to lose touch with field-based plant pathology or zoology.

In the visual identification of potato pests and diseases, a diagnostician needs a clear indication of symptoms, both written and visual. It is perhaps surprising that so few colour guides to pests and diseases of potatoes are available for this major crop. The American Phytopathological Society has released a second edition of its *Potato Disease Compendium* (Stevenson *et al*., 2001). This excellent book contains good overviews of diseases and disorders. What we hope makes our book stand out is the inclusion of pests, the emphasis on symptoms and above all, the quality of the colour photographs.

From the start, we did not set out to cover our topic in an exhaustive manner. We listed all known pests and diseases and ranked them in order of importance, particularly with respect to the Northern Hemisphere. Those considered of minor importance were omitted. No doubt there will be those who will argue with the choices made, but we have attempted to include all the pests, diseases and disorders that are most likely to be encountered in North America or Europe.

The book has been prepared using a format that will enable quick access to the most relevant information on all of the major diseases, pests and disorders of potatoes. Each chapter includes the basic information on **Symptoms** (including photographs)**, Status of the disease, Pest** or **Disorder, Life cycle and biology** or **Cause, Control** and **Key references**. To avoid conflicts among crop, disease and pest management treatment recommendations that are often site-specific, the chapters include very general information on control treatments.

For each chapter, only a few key references are listed. These are selected, in the main, from major journals that are most likely to be accessible in scientific libraries. They are only a starting point for a literature search. The availability of abstract databases and journals electronically has opened opportunities to reach a great deal of the scientific literature. Even more readily available on the Internet is a vast amount of information on potato diseases, pests and disorders, which can be accessed through search engines. Some are cited in this book. However, unlike scientific publications in established journals, Internet web sites are usually not peer reviewed, and care is needed when using such information.

Current philosophies in the development of sustainable agricultural systems rely on the implementation of integrated pest management (IPM) practices. Modern pest and disease control goals include not only successful suppression of causal agents (and therefore disease losses), but a reduction in the use of synthetic pesticides and farming methods that negatively impact on the environment and human health. Successful IPM relies heavily on early and accurate detection of pests and diseases and their causal agents. In this way, our hope is that this field guide will contribute to the development and implementation of IPM programmes that ensure sustainable agricultural production, healthy soil, water and natural environments, food safety and human health.

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Disease, pest and disorder diagnostics

As much an art as a science

Diagnosis of diseases and other problems in potatoes is as much an art as a science. Descriptions in books, even this one, can never fully describe the range of symptoms that may develop in the field. Photographs often show 'typical' symptoms, but from this norm variations occur due to resistance of the host, the prevailing environmental conditions or interaction with other pathogens or pests. An example of this variability is manifest in viruses. In one cultivar, a virus may exhibit severe mosaic symptoms, yet in another, the symptoms may be milder or non-existent. However, in this second cultivar, if an additional virus is present symptoms may be catastrophic, leading to severe stunting or death. Confusion in visual diagnosis of symptoms can be overcome by resorting to specific laboratory (or field) diagnostic tests. But even then, where a pathogen, pest or disorder is confirmed and seems to be the obvious cause, it is essential that the diagnostician eliminates other causes or implicatory factors. Where a diagnostician relies solely on visual symptoms, perhaps in the field situation, diagnosis may not be clear-cut. A preliminary diagnosis, before confirmation in the laboratory, may be based on a large proportion of the key diagnostic characteristics of a causal agent or the symptoms being present.

Know the appearance and development of healthy potato crops

The first step in diagnosis is to be aware of what healthy plants or tubers look like. This may seem obvious, but there are parts of the world where potato production is so affected by disease or pest problems that the appearance of the crops they affect is considered normal. The poor growth induced by extensive potato cyst nematode infestation in soil, or 100% virus infection, are such examples. Where widespread problems like these do not exist, diseases, pests or disorders are usually identified by their relative impact on some plants compared with healthy neighbours. Even here, the diagnostician must be clear about how a healthy crop should develop in his/her locality.

Field observation aids diagnosis

To be certain of diagnosis, access to the problem in the field or potato storage facility is essential. Many clues to the cause of a problem lie in the distribution of symptoms. When a sample is sent to a laboratory for diagnosis, it may be possible to determine the pest or pathogen involved but not associated or contributory factors. A sketch of the distribution in the field or storage facility can help diagnosis in the laboratory immensely, but it is no substitute for on-site observation. For example, from a sample in the laboratory it would be possible to identify and isolate *Rhizoctonia solani* and ascribe poor growth to this pathogen. A visit to the field might reveal that the presence of the pathogen is exacerbated in certain areas by soil compaction, or that the problem only occurs in the lightest soil in the field. The use of images taken *in situ* with digital cameras and rapidly dispatched to a diagnostic laboratory has both speeded diagnosis and improved appreciation of the field situation.

Access to all relevant information is crucial

Whether you are attempting to diagnose a potato problem in the field or in a laboratory, complete diagnosis requires a full knowledge of the field in which the crop is grown, the agronomy of the crop itself and as many observations of the crop as possible. Full background information is needed if advice is to accompany the diagnosis. It may be quite straightforward to diagnose a problem, but without clear knowledge of the background information, advice may be inappropriate or misleading. For example, the problem may be very minor and action to control it unnecessary. Alternatively, the problem may be a symptom of poor husbandry, in which case control measures should be directed at the

source of the problem. At the end of this chapter there is a checklist of some of the information that may be required for diagnosis in the field or that should accompany a sample for diagnosis in the laboratory. A simple note to a laboratory asking 'What is it?' is unlikely to get a sympathetic reception.

How to sample

Representative plants showing the full range of symptoms should be sampled. Healthy plants should be included for comparison, and both healthy and poor plants should be labelled clearly. Samples of symptomatic weeds from in-field sites and sites adjacent to cropping areas may assist in accurate diagnosis. When sampling in the field, it is advisable to sample whole plants, including roots. Plants should be dug and not pulled, since diseased or damaged roots, stems or stolons may drop off when plants are pulled out. It is often useful to the diagnostician if soil is sampled along with the roots; otherwise separate soil samples should be taken from the root zones of poor and healthy plants. These may be required for separate chemical analysis.

Sampling to assess the incidence and severity of a problem should be carried out randomly. In this instance, unbiased sampling – by selecting plants or tubers from the field or storage facility in a stratified manner – is needed. Typical patterns of sampling are: across one or both diagonals of a field, or in a 'W' pattern. For some pests, such as potato cyst nematode, very specific sampling procedures have been developed.

Investigations of the impact of crop damage on yield or profit require careful sampling to indicate the extent of damage and likelihood of loss. For example, with the increasing instances of glyphosate contamination of growing crops as a result of spray drift from a neighbouring crop, sampling at regular intervals from the boundary of the crop is required to determine how far downwind the glyphosate has drifted and what proportion of tubers are contaminated. Tubers must be sampled and set up for chitting (green-sprouting) before the extent of damage can be determined, since even low doses accumulate in tubers and affect sprout development.

Sampling can be difficult in the potato storage facility, whether tubers are kept in piles or in boxes. In these conditions, samples should be taken from as many points as possible; sampling from the surface only can give a misleading result. When sampling a crop to assess pesticide residues in tubers, correct stratified sampling is critical, especially where potential litigation may result.

Without correct sampling, assessment of the extent of a problem cannot be made fully. A simple example will explain this point. In the UK, seed tubers of susceptible cultivars are sampled and tested in the laboratory for contamination by *Pectobacterium atrosepticum* (*Pa*), the causal organism of blackleg. This test provides a snapshot of the level of contamination and thus the risk of blackleg developing in the field in the subsequent crop. A person who wished to buy a batch of seed sampled the surface tubers of the topmost boxes in the storage facility. He argued that as heat rises, condensation would be greatest on the surface of the uppermost boxes and contamination worst there. When tested, the result indicated a contamination of around 10^5 Eca per tuber – a very high count. On hearing the count the grower resampled, but from the surface of the lowermost boxes. The test for this sample indicated a contamination of 10^1 Eca per tuber. With such a disparity in results, the testing laboratory was asked for an explanation. A person trained in sampling was dispatched to the storage facility and sampled in a stratified way from boxes at different heights and locations. When this correctly drawn sample was tested, the contamination was found to be approximately 10^3 Eca per tuber. Both the seed buyer and seed grower had made fundamental errors in sampling and, in consequence, were misled about the true extent of contamination.

This example can be extended to explain other risks in sampling. If, by chance, a tuber was sampled that was touching a rotting tuber, a tuber with an exceptional level of contamination could unfairly bias the assessment of Eca. Thus, sample tubers should be taken away from rotting tubers, although the fact that some tubers were rotting should be noted. A similar error can occur if sampling takes place too soon after lifting. Eca contaminate both the tuber surface and lenticels or wounds of a tuber. Normally, once tubers are dried, a large proportion of the bacteria on the tuber surface die, leaving viable Eca mainly in the lenticels or wounds. If sampling takes place soon after lifting, a false impression of the extent of Eca contamination may be gained.

Dispatch of samples

As with any samples destined for a laboratory, careful packaging and rapid delivery are essential. Haulm of potatoes is easily desiccated if left exposed to a dry atmosphere. Thus it is often dispatched within a polythene bag. If the time between dispatch and arrival is too long, green leaf tissue, and particularly necrotic tissue, may be invaded by secondary organisms. This may be reduced if dry tissue or newspaper is wrapped around the haulm before placing it in the bag. Otherwise, wrapping damp (not soaking) tissue or newspaper around the roots and enclosing the roots in a polythene bag whilst wrapping the entire sample in a paper bag can keep haulm tissue in a sound condition.

- Tubers should be placed in a cardboard box or padded envelope for dispatch and not in a plastic bag or box.
- Where pests are involved, separate secure tubes containing live suspect specimens should accompany the potato samples.
- Samples should arrive within 24 hours and on a day when a diagnostician is available to examine them.
- Shipment of samples in the early part of a week is preferable to shipment on days prior to a weekend or holiday.

Examining samples

It is good practice to avoid the temptation of examining poor plants first and to look initially at healthy plants. These are the plants with which poor specimens are compared. On the apparently healthy plants, there may be diseases or pests, or their symptoms, which have a bearing on the poor plants but which otherwise might go unnoticed. Since above-ground symptoms may be influenced by below-ground problems, the roots, seed tuber or seed piece and below-ground stems and stolons should be examined first.

Before washing roots, look for signs of pests on them or in the soil around them. A low-power binocular microscope is usually sufficient for this. Satisfied that no pests are involved with the roots, the shoot bases and seed tuber/seed piece should be examined, also before washing. Shaking the soil from the root zone into a container will allow examination of this later if symptoms indicate a below-ground cause.

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1 Standard diagnostics using stereo (left) and transmission (right) microscopy.

After washing, the below-ground parts can be examined for lesions, root, stolon or stem pruning, rotting or evidence of pest injury. Again, a low-power binocular microscope (**1**) or hand lens is usually sufficient for this examination. Comparing the extent of lesions or damage found with the 'healthy' plants will indicate if the problem is localized or graded across the field. A sharp knife or scalpel can be used to section roots, stems, tubers, seed tuber or seed piece. This can indicate if wilt or tuber pathogens are involved. Comparing the extent of below-ground development in the poor and healthy samples can be instructive, provided samples have been carefully taken. Localized soil compaction can have an effect on plant growth and make the ingress of pest or disease more likely. Limited or distorted below-ground growth points to problems of cultivation or compaction.

Moving to above-ground parts, each part of the stem, petioles, leaves and leaflets of the haulm should be examined. Notice should be taken of the location of symptoms: whether on upper or lower surfaces of leaflets, which leaves are infected and which stems are affected. In addition to obvious lesions, associated symptoms or conditions should be recorded. These include yellowing (senescence) or other colouring, chlorosis or necrosis, and wilting or curling of leaflets. Stems should be sectioned at various points to determine if the vascular tissue or central pith shows signs of discoloration. It may be necessary to assess the percentage of leaf area or number of stems affected in order to judge the extent of the problem.

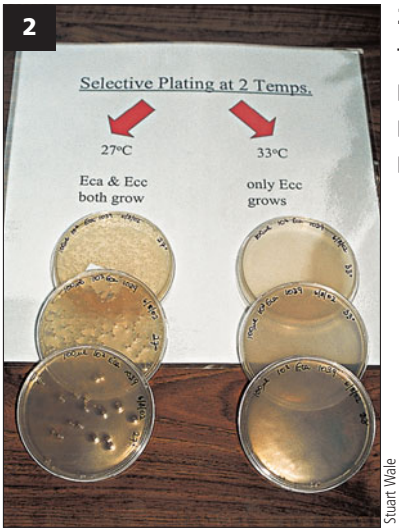
Make concise but comprehensive notes of what you find. Trying to remember features of a specific sample even a few days later when the specimen is no longer available is difficult. It is also good practice to note the number of plants or tubers in the sample as well as the stage of plant growth.

Diagnostic tests

To support diagnosis, there is an increasing array of tests used to confirm the presence of a pathogen and sometimes to quantify it. Diagnostic tests are not the subject of this book, but a brief overview will indicate the range of tests available.

The most basic laboratory technique used in the diagnosis of fungal, and sometimes bacterial, pathogens is the damp chamber. Incubating a sample in high humidity at a favourable temperature encourages sporulation of fungi. Lesions can be viewed microscopically and preliminary diagnosis of the presence of a pathogen made from the spore-bearing structures emanating from them. When a sample of leaf tissue suspected to have late blight *(Phytophthora infestans)* fails to exhibit sporulation on the lower surface on receipt, incubation in a damp chamber for 24 hours at 20° C is sufficient for sporulation to be initiated and the pathogen to be identified. Usually, if no sporulation occurs, the lesion is not that of late blight. If necessary, samples of the sporulating tissue may be transferred to slides for closer, more detailed examination under a microscope. Sections of diseased tissue can be stained and examined under the microscope for fungal spores or bacterial cells.

For decades, plant pathologists have relied on culturing bacteria and fungi from lesions on appropriate media in order to identify the cause of a disease. Today, this medium is usually agar-based. A specific medium can be highly selective for the pathogen, or suitable for a wide range of organisms. In both instances, the chances of isolating the causal organism are increased by plating tissue from the leading edge of a lesion. Sometimes isolation is preceded by surface sterilization of the affected tissue. The frequent isolation of an organism from a lesion is highly indicative that it is involved in a disease – but not proof. For proof, Koch's postulates must be fulfilled, where the isolated organism is inoculated into healthy tissue, the symptoms are reproduced and the same organism re-isolated.



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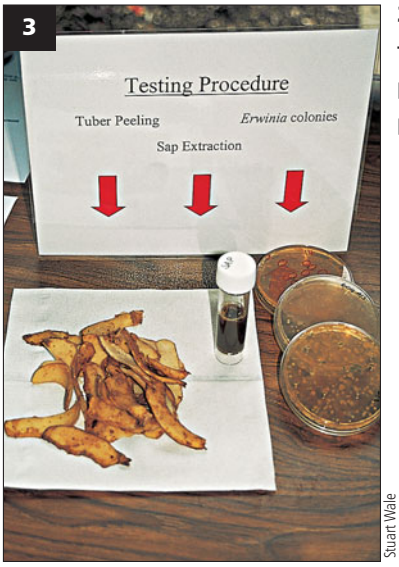
2 Microbiological testing for blackleg pathogens in tuber periderm using pectate medium.



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3 Microbiological testing for blackleg pathogens in tuber periderm.

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4 Rapid serologically based test kits for specific pathogens.

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5 Rapid serologically based test kits for specific pathogens.

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6 Real-time PCR diagnostic equipment.

Some fungi and bacteria require special techniques for isolation (**2, 3**). For example, *Clostridia* bacteria only grow under anaerobic conditions. Some fungi and all viruses and mycoplasmas are obligate parasites and, as yet, techniques have not been found to culture them *in vitro*. For these organisms, other techniques are required. To confirm the presence of viruses or mycoplasmas, electron microscopy is frequently used. Access to such specialist equipment is not always possible. Prior to the availability of such equipment, techniques of extracting and concentrating the organism and inoculation into indicator plants were used to confirm presence.

In recent decades, serological techniques have been used to detect bacteria, fungi, and viruses. Using Enzyme-linked ImmunoSorbent Assay (ELISA), a range of rapid and accurate techniques have been developed to assist diagnosis (**4, 5**). The technique is still used widely for certain pathogens, particularly in research and in certification schemes. One drawback is the complicated process by which antibodies are produced. These antibodies are then used to detect the antigen of interest. The reaction is tested for its specificity to the pathogen in question. Generally, there are two types of antibody:

- polyclonal antibodies, which detect a broader spectrum of antigens that might be produced by several microorganisms (e.g. genus or species levels)
- monoclonal antibodies, which detect more specific antigens that might be produced by a much smaller number of microorganisms (e.g. species, subspecies or strain levels).

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7 Real-time PCR diagnostic equipment.

Modern techniques

In the last few years, more sophisticated and potentially more rapid and specific tests have become available, based on Polymerase Chain Reaction (PCR) techniques. Enzymes are used to excise unique fragments of nucleic acid (DNA sequences) from tissue or soil containing a suspect organism. The sequence is replicated many times using a 'soup' of nutrients and a sequence of temperatures (thermocycling), and the quantity of the nucleic acid produced is compared against replication of a similarly treated but known quantity of the unique sequence of bases for a particular organism artificially produced. PCR and its derivative techniques also have the advantage of being relatively inexpensive. However, they require specialist equipment (**6, 7**) and an operator experienced in the technique. In time, it is

likely that portable PCR kits will enable sophisticated diagnosis to take place in the field.

To detect RNA, such as is found in many viruses, an extra step is required when using PCR. This step involves reverse transcription of the RNA genome into a single strand of DNA using a suitable reverse transcriptase enzyme. This is done prior to thermocycling.

The technology for detecting DNA or RNA fragments has advanced rapidly in the last decade. Variations in PCR have been made to increase the sensitivity and specificity of detection. For example, Immunocapture PCR (I-PCR) combines PCR and the ability of virus-specific antibodies to bind and concentrate virus particles on a solid surface. Another technique is Nested PCR. This increases sensitivity by using two pairs of oligonucleotide primers, where the annealing sites of the second pair of primers are within the sequence of the amplified fragment of the first pair.

Restriction Fragment Length Polymorphism (RFLP) is another development that allows discrimination between similar DNA samples at a species or strain level. Extracted DNA is digested and cleaved at specific recognition sites. The resulting restricted fragments are separated by electrophoresis and differences between isolates or strains determined from differences in the bands on the gel. An extension of this technique is Amplified Fragment Length Polymorphism (AFLP), where the individual fragments are amplified to improve detection.

The presence of multiple pathogens can now be tested at the same time from very small quantities of test tissue using multiplexing with real time PCR technology. Microarray technology is likely to become a powerful tool in diagnostics.

Microsatellite (mSAT) DNA technology (also called simple sequence repeats) involves short sequences of one to five nucleotides, repeated in tandem, that are distributed within eukaryotic genomes. Repeated sequences are usually polymorphic with a variable number of repetitive elements. PCR primers flanking microsatellite DNA or microsatellite-based restriction fragment length polymorphic probes are used as molecular markers to fingerprint genomic DNA. Microsatellite polymorphisms are particularly useful for the molecular identification of plant cultivars.

Checklist of factors to consider in the field or record for samples sent for diagnosis

- Name, address and contact details of sender
- Name, address and contact details of grower
- Date sent
- Cultivar(s) involved
- Location of crop/storage facility (provision of field location – e.g. grid reference – permits soil maps to be consulted)
- Area of crop and area affected/tonnage of stored tubers and tonnage affected
- Distribution of symptoms in the field or storage facility (a sketch map is usually sufficient)
- Brief description of symptoms (including when first noticed and development of symptoms over time)
- Previous crop(s); if necessary, pesticide use on previous crops
- History of previous potato cropping and any prior problems
- Soil type
- Soil drainage class
- Soil analysis (e.g. pH, organic matter, P, K, Mg, S and if available micronutrient levels)
- Soil series or association
- Stoniness of soil
- Handling of seed prior to planting (how stored, whether chitted, state of sprout growth at planting)
- Seed history (from seed supplier – including pesticide applications to seed and certification label)
- Date of seed cutting and handling/treatment afterwards
- Cultivation (including type of machinery used, soil conditions during cultivation)
- Date of planting
- Fertilizer applications
- Other soil applications (e.g. lime or soil fumigation)
- Irrigation programme (dates, quantities applied, method of irrigation, information used for irrigation scheduling)
- Pesticide applications (date, dose of pesticide, water volume applied, crop growth stage of application, weather at application)
- Haulm destruction method(s); date, techniques, dose of chemical desiccants and water volume used
- Date of harvest

- Condition of tubers at harvest, including degree of skin set, assessment of damage and tuber temperature
- Harvester details (type, whether picking area is present)
- Records of any field inspections by grower or adviser
- Handling of potatoes into the storage facility (e.g. whether loaded into boxes from harvester or into trailer for dispatch to the storage facility, whether split graded into the storage facility)
- Details of curing including duration, temperature of tubers and presence of condensation
- Layout of storage facility(ies) including location of the doors, ventilation fans, louvres, fridge unit, etc.
- Location of suspect potatoes
- Storage temperature records from loading of the storage facility
- Pattern of loading the storage facility
- Ventilation and refrigeration run hours
- Pesticide treatments in the storage facility (fungicides, sprout suppressants etc. – dose, date and how applied)
- Date of grading
- Grading equipment and assessment of damage
- Details of warming or washing of tubers prior to grading
- Date of dispatch
- Details of transport of potatoes

For chemical damage claims, details of crops in surrounding fields and their pesticide applications may be required as well as detailed meteorological data.

Equipment for field diagnosis

- Fieldguide for identifying diseases and pests
- Sharp knife
- Hand lens (*c.* 10×)
- Camera (digital and/or slide film with close-up or 'macro' capability)
- Notebook and pen/pencil
- Portable computer/data recorder
- Wellington boots/rubber boots
- Overtrousers/coveralls
- Disposable footwear and gloves
- Disinfectant spray
- Sample bags (plastic and paper) and tubes/boxes of various sizes
- Marker pen to label bags, tubes or boxes
- Loops in sterile sealed jars
- Shovel, trowel or soil sample probe
- Pail
- Container of disinfectant and clean water if possible

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Bacterial diseases

OF THE HUNDREDS OF THOUSANDS of species of bacteria, relatively few are significant pathogens of potatoes, but of the pathogens that do attack potatoes, bacterial diseases are some of the most intractable. There are a number of reasons for this. Firstly, bacteria have the potential to multiply rapidly where favourable conditions persist, increasing inoculum and disease pressure in a relatively short time period. Secondly, bacterial pathogens are very contagious and spread rapidly. Thirdly, during the early stages of disease development for some pathogens, infection may be latent or unseen. Fourthly, bacterial diseases are difficult to control: for example, there are few instances where chemicals or antibiotics are useful. Combinations of these reasons mean that bacterial pathogens represent real challenges where they occur and considerable effort is spent attempting to limit spread by quarantine and other measures.

Clavibacter michiganensis* subsp. *sepedonicus
(Spieck. *et* Kotth.) Davis *et al.*
(syn. *Corynebacterium sepedonicum*)

BACTERIAL RING ROT

Symptoms

HAULM: Ring rot symptoms may first appear 60–70 days after planting but disease expression is dependent on temperature and potato cultivar. Under warm growing conditions all of the plant symptoms may be detected, while under cooler conditions, few or no symptoms may be seen. The first symptoms appear as a pale yellow mottling in the lower leaves (**8**), which usually show signs of wilting. Leaf margins often curl upward and roll inward, and eventually dead brown areas develop. These symptoms usually begin in the lower leaves and proceed upward as the disease progresses. Leaf yellowing may intensify, producing bars of bright yellow tissue between the veins. Symptoms may first be limited to one stem but with severe infection, the whole plant may wilt and die. At an advanced stage, a brown ring can be visible in a cut stem and a cheesy exudate squeezed out from the vascular ring. In cool climates, haulm symptoms may be difficult to detect.

8 Yellow mottling of leaves.

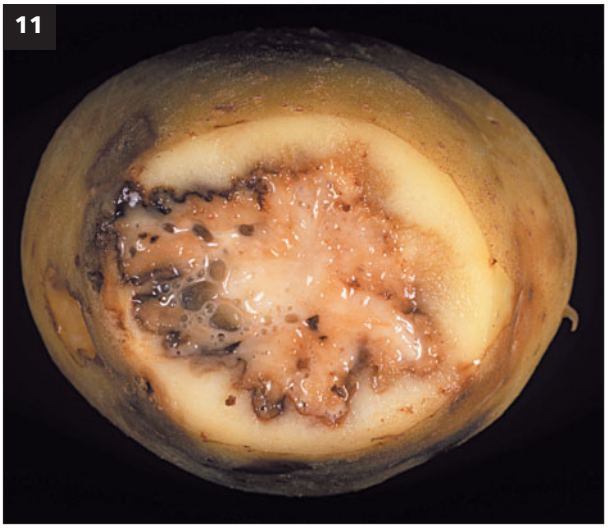
Roots: No distinct symptoms are normally seen.

Stolons: No distinct symptoms are normally seen.

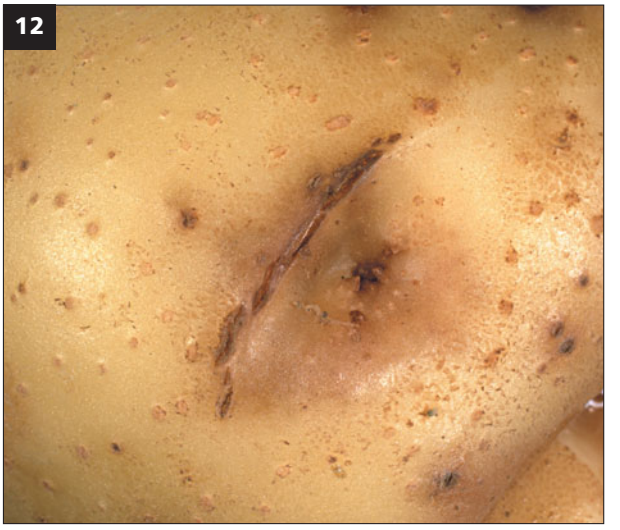
Tubers: Tuber symptoms may be visible at harvest or develop during storage. Depending on the cultivar and growing conditions, infected tubers may not show symptoms. However, ring rot bacteria can be recovered from infected, symptomless tubers. Tuber symptoms usually begin at the stolon attachment end (**9**). An initial symptom can be water-soaked translucent tissue in the vascular tissue when an infected tuber is cut. As symptoms develop, creamy yellow to tan areas form in the vascular tissue (**10**). When the cut tuber is squeezed sticky, cheesy bacterial ooze can be forced from the vascular ring. As the disease progresses, the tissue surrounding the vascular ring becomes infected and often the entire centre of the tuber disintegrates (**11**) leaving the outer shell of the potato. Externally, the tubers from diseased plants may appear normal. If badly infected, the skin may have pale reddish-brown patches, which gradually darken and become slightly sunken (**12**). Irregularly shaped cracks may appear in these areas. At this stage the tuber may become infected with common soft rot bacteria and become slimy and give off an offensive odour.

9 Ring rot symptoms at the point of stolon attachment.

10 Lesions in the vascular ring.



11 Disintegration of central tissue in tuber.



12 External lesion of ring rot at an eye.

Status of the disease

Bacterial ring rot caused serious crop losses until intensive quarantine measures, seed inspection procedures, disease control programmes, and legislation were adopted. These measures have kept disease outbreaks to a minimum. There is a zero tolerance for this disease in seed potatoes. Today the incidence of ring rot is so low that field and tuber symptoms are rarely seen. Serological or PCR test methods are required to detect the disease. Ring rot is found in parts of North America and the former Soviet Union and is established in northern and eastern Europe.

Life cycle and biology

The seed tuber is the main source of the pathogen, although in some soils the bacteria may overwinter in volunteers, in cull piles or in debris from infected crops. Contaminated waste is another potential source of infection. Disease spread is most frequent when seed tubers are cut and bacteria from infected tubers are transferred onto freshly cut seed surfaces. The pathogen is easily spread on equipment used in planting (e.g. picker-type planters), harvesting, handling, storing and transporting potatoes. Bacteria can persist for years in dried slime on potato bags, bins, machinery and storage walls and floors.

Control

The only practical methods of control are to plant certified, disease-free, tested seed tubers and to follow strict sanitation procedures. All machinery used in planting, cultivating and harvesting, bins, barrels, boxes, sacks, etc., should be thoroughly cleaned of caked mud and debris by water under pressure and plenty of scrubbing. Disinfectants are not effective unless surfaces are relatively clean. Disinfectants must be mixed to the proper strength and come in contact with all surfaces, which must remain moist for 30 minutes or more after treatment. Steam delivered at 690 kPa pressure is highly effective at cleaning and 'disinfecting'. Cutters, conveyors, planters, etc. should be cleaned and disinfected between seed lots and every few hours when cutting and planting large seed lots. In countries or localities where ring rot is present, where machinery is shared with another grower, it should be cleaned and disinfected before using or storing it. Remove all cull potatoes and destroy by burying, freezing or composting. Do not plant potatoes in fields where ring rot was found for at least 2 years or until all volunteer potatoes have been destroyed. Store seed in a clean, disinfected area. Dispose of all used potato bags. Quarantine procedures to prevent import of infected seed and regular inspection and testing of seed lots can restrict entry into non-indigenous areas.

Stuart Wale

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Pectobacterium atrosepticum **(van Hall) Garden *et al.* (*Pa*) (syn. *Erwinia carotovora* subsp. *atroseptica*), *Pectobacterium carotovorum* subsp. *carotovorum* (Jones) Hauben *et al.* (*Pcc*) (syn. *Erwinia carotovora* subsp. *carotovora*), *Dickeya* spp. (including *D. dianthicola, D. dadantii, D. zeae*) (*Dsp*) (syn. *Erwinia chrysanthemi*)**

BLACKLEG AND BACTERIAL SOFT ROT, HARD OR PIT ROT

Symptoms

Haulm: All three species may cause non-emergence through rotting of the mother tuber or seed piece.

With *Pa*, if emergence does occur, the blackleg symptom is first detectable as a stunting of growth relative to neighbouring plants. Affected stems become restricted in growth with inward rolling of topmost leaves, the foliage begins to yellow (**13**) and the haulm progressively wilts. Not every stem from a seed tuber may exhibit symptoms. Above ground, a soft slimy and often black lesion develops on the stem (**14**) developing progressively upwards. It is this lesion that gives the disease its name of blackleg.

Occasionally *Pa* and *Pcc* bacteria can splash up from soil or basal blackleg lesions and invade stems, petioles or leaves at a damage point, often where leaves join the stem, and cause an aerial blackleg symptom (**15**). Above the lesion the foliage yellows, wilts and dies progressively. The stem is soft and slimy at the lesion, exuding bacteria. If the stem is sectioned at the leading edge of the lesion, the vascular tissue and pith show brown discoloration or blackening (**16, 17**).

Dsp causes wilting and desiccation but not the soft slimy black lesions of *Pa* or *Pcc*. Browning of the vascular tissue in the stem develops into a necrotic lesion and internal desiccation occurs. Wilting and foliage symptoms tend to occur later than with *Pa* or *Pcc* and the disease has been called slow wilt.

Roots : No distinct symptoms normally visible.

Stolons: No distinct symptoms normally visible.

Tubers: With *Pa* and *Dsp*, bacterial invasion of the stem extends from the mother tuber up the stem. Bacteria can pass along stolons from the stem to developing tubers. If bacterial numbers are sufficiently high a black, soft lesion can develop at the point of tuber

13 Yellowing of foliage – early blackleg symptoms.

14 Typical blackleg caused by *Pectobacterium atrosepticum*.

15 Aerial stem lesion.

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16 Sections of diseased stem showing stem base lesion and vascular discoloration caused by *Pectobacterium atrosepticum,* compared to a healthy stem.

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17 Sections of diseased stem showing stem base lesion and vascular discoloration caused by *Dickeya* spp., compared to a healthy stem.

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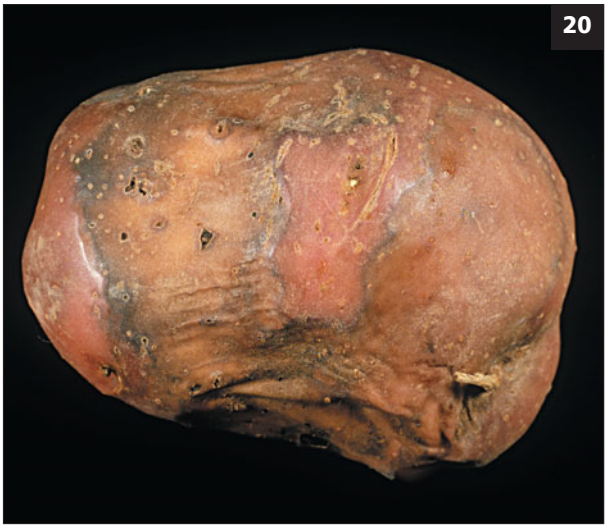
18 Invasion and rotting of the stem end of tubers after movement of bacteria along stolon.

19 Bacterial soft rot caused by *Pa* or *Pcc.*

attachment, which starts small but can develop and rot the whole tuber (18).

Tubers can develop a bacterial soft rot in the ground or during storage. If they carry a high burden of bacterial numbers of *Pa*, *Pcc* or *Dsp*, or if bacteria have invaded wounds, a progressive soft rot may develop. Where no wounds are present, infection of tuber tissue begins around a lenticel. Such lesions look circular and brown when viewed externally. The pectolytic enzymes produced by the bacteria break down the pectin binding cells together. Initially, infected tissue in the lesion is granular and cream coloured (19). Infected tissue is sharply delineated from healthy tissue (20). Subsequently, the infected tissue turns black. In advanced states of rotting, liquid containing bacteria exudes from tubers and onto adjacent tubers in store,



20 Tuber exterior with bacterial soft rot.

21 Pit rot lesions.

initiating further rotting. As bacterial soft rot proceeds in a tuber, invasion by secondary fungi and bacteria often occurs. Once this happens, confirmation that blackleg bacteria are the primary cause can be difficult. Occasionally, rotting can be contained within the skin of a tuber, the fluid only leaking out if damage occurs. Early limited infection by *Erwinia* is often odourless but a characteristic of bacterial soft rot, particularly once secondary organisms invade, is a pungent odour.

During lesion development arising from lenticels, if external conditions become dry and unfavourable for the disease, the lesion may be halted and a wound periderm forms beneath the lesion preventing its progression. This is bacterial hard rot or pit rot (**21**). The external diameter and depth of hard rot lesions vary from 1–2mm to 4–5mm.

Status of the disease

These bacteria species may cause disease wherever potatoes are grown, although climatic conditions dictate which species predominates. *Pa* is most important in cool wet climates such as northern Europe. *Dsp* has a wide host range but is found primarily in lowland warm to hot climates. *Pa* and *Dsp* are the principal causes of blackleg symptoms in the field. *Pcc* exists in all climatic zones and has a very wide host range. It is the principal pathogen associated with soft rotting in tubers.

Under favourable conditions, blackleg symptoms and/or soft rot can be extremely serious. Pre-emergence rotting causes non-emergence which, if extensive, will affect yield seriously. Blackleg can affect certification of seed in some countries (e.g. the UK) and by creating uneven plant stands can affect tuber size distribution. Soft rotting, if left unchecked, can reduce a stock of potatoes to a liquid mass as the rotting generates heat thus creating ideal conditions for further rotting.

Life cycle and biology

Blackleg: Seed tubers and seed pieces are the primary source of inoculum for *Pa* and *Dsp*. *Pcc* may also be found on seed tubers but is adapted to survival in soil. Investigations of the build up of contamination on seed through generations of seed production have shown that the pathogens can be introduced to a healthy stock in a number of ways. These include from the soil (*Pcc* and *Dsp* mainly), from rain, by insects from cull piles, by mechanical transmission during seed cutting and in aerosols after haulm pulverization in wet climates. Contaminated seed can break down when bacterial levels reach a threshold. Investigations in Europe have shown that seed with contamination by *Pa* of log 10^3 per tuber or above can result in significant blackleg, although soil conditions play a large part in development of the disease. By contrast, blackleg from *Dsp* can develop from initial low levels of tuber contamination. When soil conditions are cool and wet, seed-tuber or seed-piece breakdown by *Pa* and *Pcc* can be rapid. The breakdown results in release of bacteria into the soil where multiplication may occur in the rhizosphere of certain weeds. The bacteria (particularly *Pa* and *Dsp*) can invade stems from the mother seed tuber, which can develop blackleg symptoms. The development of blackleg depends on the time at which the mother tuber rots. Rotting soon after planting leads to non-emergence, while rotting late in crop development may result in little or no blackleg symptoms developing. High levels of nitrogen can delay blackleg expression. Infection of seed by *Fusarium* spp. can predispose it to *Pectobacterium* soft rot and blackleg development.

Bacteria from rotting seed or, to a lesser extent, blackleg stems can contaminate daughter tubers, water aiding dispersal of the bacteria through the soil. Survival in the soil depends greatly on soil conditions. If the period prior to harvest is warm and dry bacterial contamination of daughter tubers can be limited. Conversely, wet conditions in the period leading to harvest can result in high levels of tuber contamination. In general, bacterial contamination of tubers increases the later the harvest.

At harvest, blackleg bacteria can be found on the tuber surface, within wounds or within lenticels. In wet conditions, lenticels can invert and allow bacteria easy entry. Rapid drying after harvest will normally lead to death of surface bacteria but those in lenticels are protected from the storage environment, declining in numbers only slowly if unfavourable conditions persist. Numbers of blackleg bacteria in lenticels can increase if favourable conditions for bacterial growth occur. These include warm storage temperatures and the presence of surface moisture through condensation. At grading, rotting tubers can spread the bacteria on the machinery to many other tubers.

SOFT ROT: Invasion of tubers by bacteria can occur in a number of ways. If *Erwinia* numbers are high, rotting can develop in wounds and through lenticels where conditions favour the bacteria. High temperatures (15–25°C) and the presence of free water are conducive conditions. *Pa* and *Pcc* are facultative anaerobes, and the presence of free water for a period of time can induce anaerobic conditions at the tuber surface and enhance infection. At temperatures of 6°C or below, growth of these bacteria is very slow and rotting inhibited. *Erwinia* often invade lesions made by other pathogens including *Fusarium* spp., *Phoma exigua* var. *foveata*, and occasionally *Streptomyces scabies* and *Spongospora subterranea*. Other bacteria such as *Pseudomonas* spp., *Bacillus* spp. and *Clostridium* spp. can cause soft rotting but these are mostly favoured only at very high temperatures. Immature skins on tubers, wounding and high nitrogen fertilization predispose tubers to soft rot.

Control

Reduce contamination of seed tubers

- This is achieved by using limited generation seed, harvesting early in dry conditions, and by minimizing damage by correct harvester setting and lifting tubers with a good skin set.

Minimize disease spread

- Remove mother tubers and rotting tubers at harvest. Do not plant diseased or damaged tubers, especially those with damage to sprouts. Plant when conditions are favourable for rapid growth.
- Select well-drained fields and use recommended rates of fertilizer. Rogue out infected plants (seed crops). Avoid waterlogging by irrigation. Do not pulverize haulm during wet conditions. Rapid desiccation of seed crops (e.g. with sulphuric acid) is advantageous.

Good storage

- Dry crops immediately after harvest, dry cure and reduce tuber temperature steadily thereafter. Avoid condensation and CO_2 build up during storage by effective ventilation. Pick off rotted tubers early in the grading process, clean the grader between stocks and especially after grading a stock containing bacterial soft rot. Warm tubers before grading to at least 8°C to minimize damage.
- Maintain good hygiene by cleaning and disinfecting stores, machinery, boxes, seed cutting equipment and trays between seasons. With tubers washed before sale, use clean wash water or treat water with a suitable disinfectant to reduce bacteria and dry tubers rapidly after washing.

STUART WALE

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Ralstonia solanacearum (Smith) Yabuuchi *et al.* (syn. *Pseudomonas solanacearum, Burkholderia solanacearum*)

BROWN ROT (BACTERIAL WILT, SOUTHERN BACTERIAL WILT)

Symptoms

Haulm: In the growing crop, the first symptoms are wilting of younger leaves during the heat of the day. Initially, this may occur in only one stem. The wilting may recover at night, but the wilting is progressive, eventually becoming permanent and leading to stunting, yellowing and haulm death. Stems may show brown streaks externally from soil level upward. When cut, stems exude a grey-brown bacterial ooze from the vascular tissue.

Roots: No distinct symptoms are normally visible.

Stolons: Discoloration along the stolon may be visible.

Tubers: In tubers, an early symptom is necrosis of the vascular ring (**22**). The area of necrosis can extend from the vascular ring into the parenchymatous tissue. When cut, a milky fluid exudes from the vascular ring (**23**). Infection by some strains of *R. solanacearum* does not result in vascular browning. Bacteria can exude from eyes (**24**), especially at the rose end and the stolon attachment, resulting in soil particles adhering to the tuber. Not all tubers show symptoms; some can be infected latently.

Status of the disease

The bacterium is widely distributed in the tropics, the sub-tropics and warm temperate regions and can be significant in limiting potato production. Yield losses up to 75% have been recorded in tropical countries. It has a wide host range, including the Solanaceae and a number of important crops including tobacco, pepper, groundnut and banana. Certain weeds can act as host reservoirs of infection. *R. solanacearum* has been classified into five races based on host range and five biovars based on chemical tests. Race 1 is mainly found in tropical and sub-tropical areas including parts of North America and Africa. Race 3, biovar 2 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. Some countries remain free of the pathogen and impose quarantine measures to prevent importing it.

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22 **Necrosis of the vascular ring.**

Life cycle and biology

Seed-borne infection is the most important source of the pathogen, but infection can occur via the soil especially in warmer climates. Seed-cutting knives can spread the bacterium from infected tubers to the cut surfaces of healthy tuber pieces. The bacterium can persist in crop debris and on weed hosts and survive in the soil for a period of time. However, in temperate climates, survival in soil more than two years after harvest of an infected crop is unlikely, provided there are no groundkeepers. Where soil is contaminated, infection can occur by invasion of the roots at root emergence points or at wound points (e.g. caused by nematode activity). The pathogen moves through the vascular system, multiplies and blocks xylem vessels through extracellular polysaccharide production and aggregation of bacteria, thus causing wilting. The bacterium also passes along stolons to daughter tubers. In warmer climates and in wet conditions, the pathogen multiplies faster and the development of symptoms is rapid. In cool climates (soil temperatures below 15°C) it may take several years for the disease to manifest itself during multiplication, and it can also be latent and symptomless. The pathogen

23 Cut tuber showing necrotic vascular ring and milky exudates from ring.

24 Exudation from eyes of tuber infected by ***Ralstonia solanacearum***.

can cause brown rot in a wide range of soil types and levels of acidity. In cool temperate climates, *R. solanacearum* has been found to persist and multiply on the roots of *Solanum dulcamara*, in or on the edge of watercourses. Irrigation using water from these sources can spread the bacterium to uninfected potato crops.

Control

Differential resistance (tolerance) to *R. solanacearum* has been identified. However, whilst symptoms may be suppressed, a large population of the pathogen may build up on resistant cultivars. This has implications for the spread of the disease. Long rotations, dense cover crops and effective weed control to prevent survival of the bacterium on weed hosts or volunteers is important. However, the principal control measure is to plant healthy, uncontaminated seed. To achieve this, routine testing of seed stocks for the presence of *R. solanacearum* is carried out in some seed-producing countries. In Europe, this follows EPPO guidelines and consists of testing 200 tubers per 25 tonne seed lot. Various methods are employed to test seed lots including serology, PCR and plating on to selective media. Legislation, quarantine measures and certification schemes linked to seed and watercourse testing are used to ensure freedom from brown rot. Disinfection of cutting knives is important to prevent spread within a seed lot.

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Streptomyces scabiei **(Thaxt.) Waksman & Henrici (syn. *S. scabies*)**

COMMON SCAB

Whilst *S. scabiei* is considered the most widespread and common species, a further nine species have been associated with common scab: *S. acidiscabiei*, *S. caviscabiei*, *S. europaeiscabiei*, *S. luridiscabiei, S. niveiscabiei, S. reticuliscabiei*, *S. stelliscabiei*, and *S. turgidiscabiei.*

Symptoms

Haulm, Roots, and Stolons: Disease symptoms are normally not seen.

Tubers: There are three basic common scab types – raised, pitted and superficial. There is some correlation with the causal *Streptomyces* spp., but there is also some correlation with cultivar and environmental conditions. For example, a species that causes one symptom in one cultivar may cause a different symptom in another or in the same cultivar grown under different conditions. The disease caused by *S. scabiei* is usually characterized by corky erumpent or pitted lesions on the surface of the tubers (**25**, **26**). Lesions are tan to brown and roughly circular in shape, although in severe cases they may coalesce and cover much of the surface area. Other *Streptomyces* species may cause similar symptoms.

Pitted scab lesions are raised but often deeply pitted and crater like. Deep pitted scab has been associated with *S. caviscabiei.* Symptoms of superficial lesions include netted scab and russet scab, which are often confused with each other. Netted scab (**27**) in Europe can be caused both by *S. reticuliscabiei* and *S. europaeiscabiei.* Superficial russeting symptoms accompanied by necrosis of the root system have been attributed to species of *Streptomyces* such as *S. reticuliscabiei* and appear to have different etiology.

Status of the disease

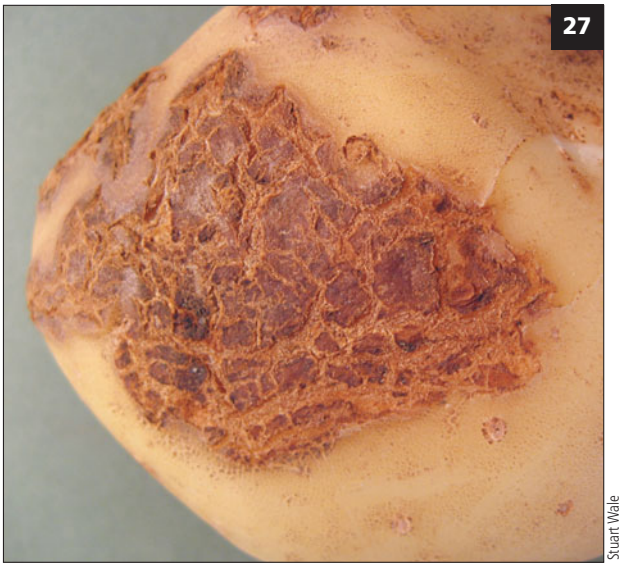
Potato scab occurs in potato production soils throughout the world and reduces the quality of processing, table and seed potatoes because of the unsightliness of infected tubers and the tuber-borne nature of the disease. Increased peeling costs, diminished marketability and increased seed grade-out are the major economic effects. Total yields are seldom reduced.

25 Common scab lesions.

Life cycle and biology

Streptomyces species are classified as aerobic bacteria yet they have some characteristics, including filamentous morphology, resembling fungi and hence are termed actinomycetes. Aerial mycelia are usually mouse grey in colour. Sporulating scab lesions may be observed sometimes on tubers. In culture, a characteristic earthy smell is emitted. *S. scabiei* produces phytotoxins called thaxtomins, which elicit scab symptoms. Thaxtomins are likely to play a role in pathogenicity. The organism is tuber-borne and is also a well-adapted saprophyte that persists in soil on decaying organic matter and manure. (*S. acidiscabiei* causes similar symptoms but has different morphological and physiological characteristics.) Infection occurs mainly through immature lenticels making the tubers most susceptible for six to eight weeks following tuber initiation, since they rapidly enlarge. Infection of the stolons and fibrous roots may also occur. The optimum temperature for the growth of *S. scabiei* is 30°C. Soil properties influence the extent of symptom development. The disease is usually most severe in dry soils with a pH of 5.2–7.0, although *S. acidiscabiei* has caused scab in soils with a pH of 4.5. High soil moisture inhibits growth and offers a means of control. Common scab does not spread in storage.

26 Common scab lesions on red tubers.



Stuart Wale

27 Netted scab lesions.

Control

Differences exist in the susceptibility of potato cultivars to scab, but as yet no cultivars are totally resistant. Under conditions that are highly conducive to common scab development, even the more resistant cultivars may develop symptoms. Potato-breeding programmes around the world seek new sources of resistance in relatives of the cultivated potato to combine with agronomically acceptable characteristics. Lowering soil pH with acid-producing fertilizers or sulphur applications has reduced the levels of scab in some cases, but this may negatively influence fertilizer absorption and yield. Soils with lower pH may also limit the choice of rotation crops. Over-liming of fields is to be avoided. Increasing the interval between potato crops may reduce the soil population but is unlikely to eliminate the organism. Irrigation to maintain high soil moisture during tuber formation has effectively controlled scab development in the UK. Soil and seed treatments are costly, not particularly reliable and may not be available or acceptable in all production areas.

Agnes Murphy

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CHAPTER 2

Fungal and fungal-like diseases

THE DIVERSITY OF FUNGAL ORGANISMS that cause disease in potatoes is great. It includes relatively primitive organisms (Plasmodiophorales) that are more closely associated with protozoa. The two representatives described in this book cause powdery scab and wart diseases. Also included are water moulds (Oomycetes) which are strictly not fungi either, although they do produce mycelium. Within the Oomycetes is, perhaps, the most destructive potato pathogen, *Phytophthora infestans*, which causes potato blight.

The effect of fungal pathogens on the potato crop varies enormously, ranging from foliage lesions, and wilting to tuber rots, blemishes, and malformations. In developed countries, fungal pathogens of potatoes have increased in importance, particularly where they affect quality.

In consort with the diversity of fungi involved, the biology and epidemiology of fungal pathogens is equally diverse, and so control strategies vary substantially. With many of this group of potato pathogens, there are fungicides available to add to potential control measures. Potato blight control, in particular, still relies heavily on fungicides in many countries. However, control using host resistance, manipulation of the environment , and good husbandry are equally possible with this group.

The identification of fungal pathogens is possible usually from the reproductive structures of the fungi involved. Although they are microscopic, a hand lens or optical microscope is usually sufficient for identification.

Alternaria solani **Sorauer**
Alternaria alternata **(Fries.) Keissler (syn. *A. tenuis*)**

EARLY BLIGHT/TARGET SPOT

Symptoms

HAULM: Leaf, and to a lesser extent, stem lesions are most frequently observed on mature tissues or on tissues weakened by other diseases or environmental and nutritional stresses. Small black spots (1–2 mm) develop into large, brown to black, circular to ovoid lesions (**28**) often restricted by leaf veins (**29**) but can also be associated with lenticels. Characteristic 'target board' concentric rings of raised and depressed necrotic tissue can be observed, often with a chlorotic halo surrounding the lesion. The under-surface of leaf lesions

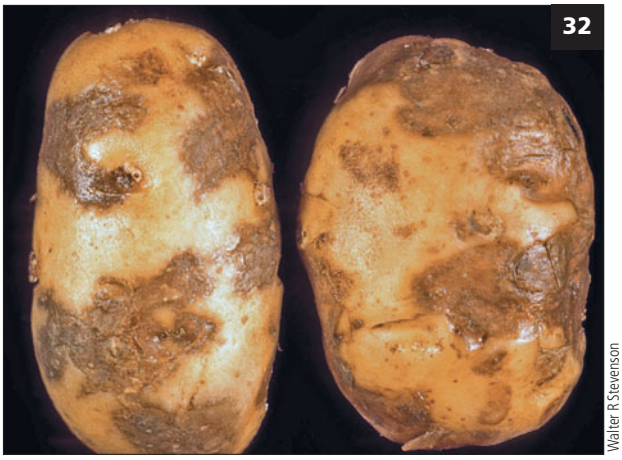
28 **Circular target lesions of *Alternaria*.**

29 Early blight lesions, sometimes restricted by veins.

30 Severe symptoms of early blight on haulm.

Stuart Wale

31 *Alternaria* tuber symptoms.



Walter R Stevenson

32 *Alternaria* tuber symptoms.

will not have a bordering halo of white, fluffy fungal mycelium and spores as occurs with late blight, but on the necrotic tissues of the lesion some dark mycelium and spores can be observed. As old and new lesions develop, the whole leaf becomes chlorotic, then necrotic (**30**), often remaining attached to the stem as a desiccated, brown to black parchment.

Roots and Stolons: Infrequently observed and reported.

Tubers: Circular or irregularly shaped, dark, sunken lesions on tuber surfaces with raised, purple to dark brown borders can be seen at, or after, harvest (**31, 32**). Below the tuber surface, tissues are dry, leathery to corky, and dark brown with yellowish, water-soaked borders. The dry rot phase of tuber symptoms develops slowly.

Status of the disease

Early blight occurs in all potato production areas, but there are significant impacts on tuber yield and quality only when warm, wet conditions favour early season and rapid disease development. When epidemics result in excessive defoliation, tuber yield losses occur in excess of 20–30%. When conditions favour late season or slow development of the disease, tuber yield and quality losses do not occur. In these cases, late season disease can aid in maturation and desiccation of the foliage.

Life cycle and biology

Alternaria solani persists as mycelium and spores in crop and other host debris, soils, and infected tubers. Tan to olive-brown spores (15–19 × 150–300 µm) are normally formed singly and are eight- to ten-celled, ellipsoid to oblong, with a long, narrowing terminal cell or beak. *A. alternata* also infects solanaceous plants, forming lesions similar to those caused by *A. solani*. However, the spores are smaller (20–63 × 9–18 µm), formed in chains, and do not have a long beak. *A. alternata* can cause brown spotting on the leaves and black pitting in tuber tissue. It is frequently isolated from various diseased sites but is generally considered a weaker pathogen than *A. solani.*

Spores formed in the spring serve as the primary inoculum source and are carried by wind, water, and equipment from field to field. Alternating wet and dry periods favour spore formation and dissemination, which peaks during the morning drying periods. Spores germinate and penetrate leaf tissues directly through the epidermis, stomata and wounds. Actively growing, newly formed foliage and plants heavily fertilized are less susceptible than mature or weakened (diseased or stressed) foliage. Spores produced on infected tissues provide further inoculum sources throughout the growing season for this multicyclic disease. Lesion development progresses until all healthy tissues succumb and the plant is defoliated or dies.

Immature tuber skin or wounded tuber surfaces are more susceptible than mature tubers. Spores germinate and penetrate lenticels and wound sites in the presence of free moisture. Viable spores can also be carried on tuber surfaces into storage prior to germination and infection. Lesions continue to expand slowly in storage causing a dry rot.

Control

Disease management strategies include cultivar resistance, use of foliar fungicides, maintaining optimum agronomic inputs and harvesting when tuber skin is set. Few cultivars with resistance are grown commercially, but several potato breeding programmes have lines under development, and rate-reducing resistance-slowing disease development has been identified. In general, disease susceptibility is less in late-maturing cultivars than early-maturing cultivars. Use of resistant cultivars can reduce the need for fungicides in disease management. Several protectant (e.g. chlorothalonil, dithiocarbamates, fentin hydroxide) and systemic (e.g. azoxystrobin, difenoconazole, tebuconazole) fungicides are available for effective protection against the initial development of early blight. Fungicide applications are most efficacious when properly timed for forecasted disease threat due to spore production early in the season.

Reduction of inoculum sources with proper crop rotation, crop debris disposal, weed management and the use of healthy seed will lessen the occurrence and impact of the disease. Weakened or stressed plants are more susceptible, so measures such as proper crop fertilization, water supply, cultivation, disease management and other crop and land management strategies that improve crop health will limit the occurrence and damage due to early blight. Potato tubers with mature and intact skins are less susceptible to the disease. The harvesting of tubers with advanced skin maturation, as well as the harvesting and handling of tubers in ways that minimize bruising and mechanical damage, will reduce disease incidence and impact. *A. alternata* has been reported to cause a black pit disease, and the use of a post-harvest tuber treatment fungicide reduces disease incidence during storage.

H W (Bud) Platt

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Botrytis cinerea **Pers. (teleomorph: *Botryotinia fuckeliana* [deBary] Whetz.; syn. *Sclerotinia fuckeliana* [deBary] Fuckel)**

GREY MOULD

Symptoms

Symptoms of grey mould can sometimes be confused with those of late blight, since *Phytophthora infestans* also produces a profusion of spores during humid conditions. However, sporulation of *P. infestans* tends to produce a sparse, white, fuzzy growth, mainly on the underside of leaves. If doubt exists in identification, the two pathogens are readily distinguished upon microscopic examination. *B. cinerea* conidia are produced in grape-like clusters on conidophores that are more or less straight, branched and brown in colour. Single-celled conidia are obovate or ellipsoidal, hyaline to brown, or in-mass, grey-brown and measure 9–15 × 6.5–10 µm in size.

Haulm: Infected flowers often provide the first evidence of the presence of the grey mould pathogen in a potato field. Flower petals become covered with a grey, fuzzy growth as the disease progresses under cool, moist weather conditions. Infected flowers can initiate the foliar phase of the disease when they fall into the plant canopy. During cool, wet weather or in lush, irrigated fields, *B. cinerea* can spread from fallen flowers to infect damaged or mature leaves. Leaf lesions are initially tan coloured, often wedge shaped and bordered by major veins (**33**). Wide concentric zonation is sometimes apparent. The remnants of the floral parts which initiated the lesion are often still visible in the centre of the lesion. A brown, slimy rot can develop on stems infected via wounds or as the result of disease progress from an infected petiole. In humid conditions, the diseased tissue develops a dense, grey or brown, fuzzy growth due to the production of mycelium and large numbers of conidia.

Roots and Stolons: Infections do not usually occur.

Tubers: Infections of tubers are rare, but when they occur they are usually initiated by wounding during harvest, transit and grading operations. Infected tubers have wrinkled skin and soft, wet, sub-surface tissues (**34**) which become darkened and develop into a dry rot unless secondary wet rot pathogens are present.

33 Foliage symptoms of grey mould with sporulation on dead tissue within lesion.

SASA © Crown copyright

34 Tuber symptoms of grey mould.

SASA © Crown copyright

35 Sporulating *B. cinerea* lesion on a tuber.

The sunken, pitted and discoloured areas usually penetrate less than 1 cm into the tuber. Grey, fuzzy growth (mycelium and conidia) can also develop on diseased tubers under humid conditions (**35**).

Status of the disease

B. cinerea has a wide host range and infects many types of vegetable, fruit and ornamental plants worldwide. In general, grey mould is not an economically important disease of potatoes. However, it can contribute to poor crop growth and the establishment of other diseases. Grey mould can also contribute to difficulties in assessing proper disease management strategies, since its symptoms are similar to those of other diseases such as late blight (see page 48).

Life cycle and biology

The fungus forms hard, black sclerotia (variable in size) which can overwinter in field soils. Although the source of primary inoculum is unclear, it may be ascospores or conidia from fruiting sclerotia found in crop debris. A period of cold temperatures is required to form the apothecia. Apothecial stalks are up to 3 cm long and 1–2 mm thick; the discs are concave, yellow-brown, and up to 8 mm in diameter. Asci are cylindrical, and the ascospores are ellipsoidal to fusiform, 9–15 × 4–7 µm in size and uninucleate. Conidia or ascospores of the fungus are carried by wind and water to host plants where infection is initiated. Although *B. cinerea* can penetrate directly into plant tissues following spore germination, the fungus is a saprophyte and a weak pathogen requiring cool, wet conditions to facilitate infection, disease initiation and development.

Similarly, wounded or diseased tissues, weak or senescent plant parts, and prolonged wet periods increase the opportunity for disease. Disease initiation and development are prevented or inhibited by warm, dry weather, but are favoured by frequent periods of moisture (rain, dew or fog), shaded conditions, heavy irrigation when temperatures are low, and dense or fallen canopies, particularly as plant tissues begin to mature. Latent infections appear to occur commonly; disease development is initiated by plant stress factors and conducive environments. Conidia are produced abundantly on infected and decaying tissues, particularly during cool, wet periods. These are carried by wind and water to new host plants and initiate secondary infections. Tubers can become infected in the field or during harvest and grading operations, especially if they are wounded or if wet conditions occur.

Control

Dense crop canopies create microclimatic conditions conducive to disease development. Therefore, avoiding excessive canopy development by over-fertilization and irrigation is an important form of cultural control. Most contact fungicides prevent disease if plant tissues are adequately covered prior to the arrival of spores. Fungicides vary in their efficacy for grey mould control, and fungicide resistance may occur when they are routinely used. Integrated strategies, incorporating various control methods and climatic models, may be beneficial in the future. The incidence of tuber infection can be reduced by harvesting when the plant foliage is completely dead and dry weather conditions prevail. Reducing tuber wounding during the harvest, grading, and transport operations also decreases the occurrence of grey mould and other diseases caused by soil-borne pathogens. Applications of fungicides during the growing season may help to reduce tuber rots in storage, including those caused by *B. cinerea*. Proper tuber curing and storage will also limit tuber infections.

Rick Peters

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Colletotrichum coccodes (Wallr.) Hughes
(syn. *C. atramentarium* [Berk. & Br.] Taub.)

BLACK DOT

Symptoms

Haulm: Symptoms on leaves are less common than stem or tuber symptoms in the field, but under experimental conditions, spray inoculations with conidia have caused sunken, necrotic lesions on stems, leaves and petioles, as well as vein and leaf necrosis, dieback and premature vine death. Infection of vascular tissue and girdling stem lesions (**36**) can induce yellowing and wilt-like symptoms, which generally progress from plant apices to lower portions of the plant. Wilt symptoms may be confused with those caused by other pathogens such as *Fusarium* or *Verticillium* (see pages 36 and 68). The disease is sometimes associated with the potato early dying syndrome. Small, black, dot-like sclerotia (microsclerotia) are formed abundantly in stem lesions, particularly late in the growing season, and are visible to the naked eye. Under the microscope, examination of sclerotia reveals acervuli bearing setae, which are diagnostic. Cutting open dried stems often reveals an amethyst coloration inside the vascular cylinder. Sclerotia may form in internal tissues as well. Leaf lesions are mostly associated with hot dry climates where wind-blown sand particles facilitate transport of the pathogen.

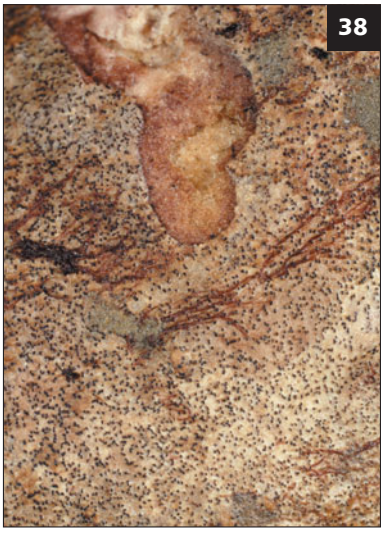
Roots: Silvery brown lesions are reminiscent of those caused by *Rhizoctonia*. However, characteristic microsclerotia are readily formed – aiding diagnosis – and as the infection develops, peridermal tissues are sloughed away.

Stolons: Lesions are also reminiscent of those caused by *Rhizoctonia*, but microsclerotia are readily formed on stolons. Infected remnants of stolons often adhere to tubers at harvest.

Tubers: Tubers infected with *Colletotrichum* develop dark, greyish lesions (**37**), which appear similar to silver scurf (*Helminthosporium solani*) (see page 41), and both pathogens can appear on the same tuber. However, black dot lesions are more irregular, with undefined margins. They also usually contain microsclerotia (**38**) which are smaller than those on stolons. Where black dot affects tubers, symptoms are

36 Stem lesions of black dot.

37 Lesions of black dot with microsclerotia.



38 Severe black dot on tubers.

39 Black dot lesions developing from the stolon.

commonly seen near the stolon attachment end (**39**). Extensive tuber blemishes may increase tuber respiration, resulting in shrivelling and tuber shrinkage. Prior to harvest, tubers are more frequently infected on the underside where damper soil conditions persist.

Status of the disease

Black dot is commonly found in most potato-growing regions. It is generally considered to be a surface-blemishing disease of tubers which downgrades potatoes destined for table markets and may affect seed tuber sales due to disease tolerance restrictions. Total tuber yields are rarely affected, but research has shown that the number of small tubers is sometimes increased in affected fields. In some studies where fungal inoculum has been added to the soil, tuber yields and plant growth have been somewhat reduced. In addition, foliar infections have been shown to cause yield losses when pathogen populations are artificially enhanced. Recent studies indicate that the fungus may be associated with the potato relatively early in the growing season, and with many plants over a wide geographic area. Therefore, yield effects may be more significant than formerly assumed.

Life-cycle and biology

C. coccodes overwinters as microsclerotia (100–500 µm in diameter) occurring free or on colonized plant debris in the soil. Studies have shown that the fungus can persist in the soil for at least eight years. The fungus may also overwinter as sclerotia on infected seed tubers and, therefore, infection of plants during the growing season may be due to tuber-borne and/or soil-borne inoculum. Some studies indicate that the contribution of soil-borne inoculum is at least twice that of seed-borne inoculum. However, other studies have shown that plants grown from infected seed are infected earlier, resulting in increased disease incidence and severity. In addition, tuber-borne inoculum is important in the long-distance spread of pathogen strains. Variation in the pathogenicity of isolates of *C. coccodes* has been reported.

Conidia probably serve as the primary inoculum for infection. Under conducive conditions, conidia are formed in acervuli on the surface of sclerotia. Acervuli also contain septate, pointed setae that vary in length from 80–350 µm. Conidia are hyaline, guttulate and 18–22 µm × 3–8 µm in size. In culture, masses of conidia appear yellow to pink, depending on growth medium. Conidia do not germinate at 7°C; the optimum temperature for germination and infection is between 22 and 28°C. Roots are the organ most susceptible to infection; stems generally become diseased only after the fungus is well established on the underground system of the plant. Infection can develop on roots and stem bases soon after plant emergence. Research indicates that the fungus may colonize plant tissues as a latent endophyte without causing disease symptoms until conducive conditions (including various plant stress factors such as the presence of other pathogens or natural senescence) occur. Black dot is commonly associated with high temperatures, poor soil drainage and sandy soils, and low nitrogen levels. Other solanaceous plant species and several weed species also act as hosts for *C. coccodes*.

Tubers can be infected in the field and probably also in storage. The severity of black dot tends to increase the longer tubers are left in the soil (i.e. a later harvest date usually leads to more disease). The incidence of black dot also increases the more frequently a field is used for potato cultivation. In storage, infection and symptom development are favoured by warm, humid conditions. Studies have shown an increase in disease in

tubers held at 15°C compared with those kept at 5°C. However, tuber necrosis may be accentuated at lower temperatures. Soil adhering to tubers in storage could be a source of inoculum contributing to disease development after harvest. Acervuli on microsclerotia in tuber lesions will produce conidia, which may infect adjacent tubers, under warm, moist conditions .

Control

Black dot tends to be more prevalent in irrigated crops and when the interval between successive potato crops is short. Therefore, long rotations (at least five years between potato crops) and good irrigation management are recommended. Maintaining adequate soil fertility is also important. Since inoculum can be tuber-borne, planting disease-free seed is essential. Various fungicides (e.g. prochloraz) inhibit mycelial growth of *C. coccodes in vitro* and may be useful as seed treatments, but the fungus is not controlled by some of the seed treatments commonly used to control scurf diseases. Seed treatments tend to be less effective when soil inoculum levels are high. In experiments, soil fumigation has reduced levels of soil-borne inoculum and decreased the incidence of black dot in progeny tubers, although its use is not always practical or appropriate. Recently, soil applications of azoxystrobin have shown efficacy against soil-borne *C. coccodes*. Some species of *Trichoderma,* when added to infested soil, have reduced disease in research plots. Controlling weed hosts is beneficial.

Harvesting early generally decreases black dot in tubers. Washing and then drying tubers has been shown in experiments to limit pathogen spread in storage. Studies indicate that drying tubers for two weeks at 10–15°C prior to storage at 5°C decreases the severity of black dot in storage. There is variation in the response of cultivars to disease; early cultivars are generally more susceptible to infection than later-maturing genotypes. Thin-skinned cultivars also appear to be more susceptible to disease than thicker-skinned cultivars. No cultivars with very high levels of resistance are currently available.

Rick Peters

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***Fusarium* spp.**

FUSARIUM DRY ROT/WILT

Dry rot species

***Fusarium coeruleum* (Lib.) ex Sacc.**
(syn. *F. solani* var. *coeruleum* [Sacc.] C Booth)

***Fusarium sulphureum* Schlect**
(teleomorph: *Gibberella cyanogena* [Desm.] Sacc.)

***Fusarium sambucinum* Fuckel**
(teleomorph: *Giberella pulicaris* [Fr.] Sacc.)

Wilt species

***Fusarium oxysporum* f.sp. *tuberosi* Snyder & Hansen**
(syn. *F. oxysporum*)

***Fusarium eumartii* (C.W. Carp.) (teleomorph: *Nectria haematococca* var. *haematoccocca*)**

***Fusarium javanicum* Koord.**
(teleomorph: *Nectria ipomoeae* Halsted)

***Fusarium solani* (Martius) Sacc. (teleomorph: *Nectria haematococca* var. *brevicona* [Wollenw.] Gerlach.)**

***Fusarium avenaceum* (Corda) Sacc.**
(teleomorph: *Gibberella avenacea* Cooke)

Other species recorded as pathogenic on potatoes

***Fusarium arthrosporioides* Sherbakoff**
(similar to *F. avenaceum*)

***Fusarium sporotrichioides* Sherbakoff**

***Fusarium trichothecioides* Wollenw.**

***Fusarium equiseti* (Corda) Sacc.**
***(Gibberella intricans* Wollenw.)**

***Fusarium graminearum* Schwabe.**
***(Gibberella zeae* [Schweinitz] Petch)**

Symptoms

Dry rots

Haulm, Stem Bases, Roots and Stolons: Missing plants or severely stunted, chlorotic and necrotic haulm. Roots and stolons have abnormal growth symptoms.

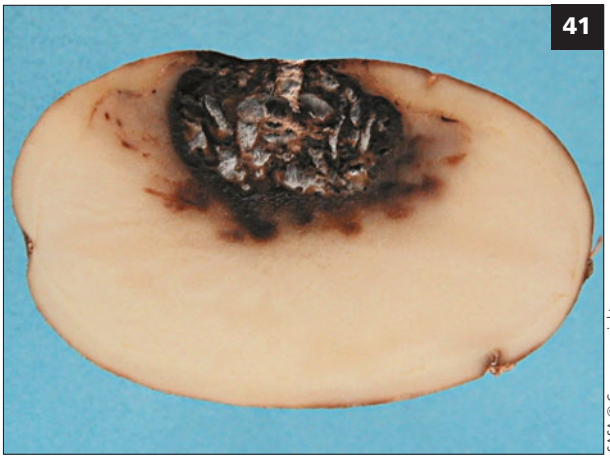
Tubers: On the surface, a sunken lesion with concentric rings of shrivelled skin is visible (**40**). Small cushions of white mycelium can erupt on the lesion surface, sometimes merging into irregular mats. Neither of these symptoms is diagnostic, since they can be associated with other diseases (e.g. gangrene). In section, lesions caused by *F. sulphureum* (syn. *F. sambucinum* Fuckel f.6 Wollenw.) are light to dark brown in colour, dry and crumbly in texture with a distinct dark brown but irregular margin to the lesion (**41**). Fungal growth within the lesion can vary in colour from white, through yellow to red. In section, lesions caused by *F. coeruleum* are dark brown in the older part of the lesion, fading to a lighter brown which merges into the healthy tissue (**42, 43**). *F. coeruleum* lesions have a more regular edge than those of *F. sulphureum*. Cavities may develop within lesions with age and may be lined with mycelium and spores. When fully invaded, tubers lose moisture and shrivel to a dried 'mummy'. Invasion by secondary organisms can occur and confuse identification. Seed tubers infected by the dry rot fungus prior to planting or seed pieces infected at cutting may rot and fail to emerge.

Wilts

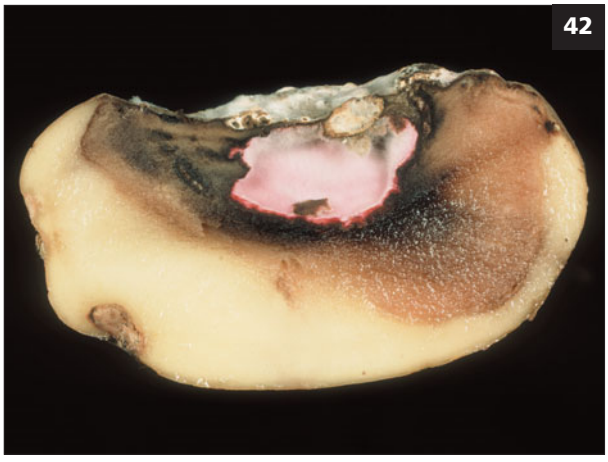
Haulm, Stem Bases, Roots and Stolons: Symptoms vary according to the pathogen involved. Generally, there is a decay of roots and lower parts of stems. Vascular discoloration frequently develops in the stem. Above-ground symptoms usually start on the lower leaves with veinal chlorosis and/or yellowing followed by wilting. Browning or purpling as well as incurling of leaves can occur. Leaf symptoms may appear on one side of a plant associated with partial stem infection. As the wilting progresses, streaks may develop on the stem, the cortical tissue of the stem is invaded, symptoms progress to upper leaves, leaves become necrotic and ultimately stems collapse. Affected plants tend to occur in foci which may increase in size with time.

Wilting caused by *Fusarium oxysporum* f.sp. *tuberosi* develops rapidly causing early haulm death but usually after the crop has established. Vascular discoloration is usually found at or below the soil line. Vascular discoloration of tubers can occur but is uncommon. With *Fusarium eumartii* the pith of the stem is often discoloured at the nodes. Underground parts of the stem may rot at the end of the growing season, when the wilting usually becomes apparent. The vascular necrosis can extend into the tubers. At the point of stolon attachment a brown lesion may be apparent on the surface. Internally, development is variable. Browning of the vascular tissue can extend from the rot and a light brown, water-soaked area may extend out from the vascular tissue into the tuber flesh. Vascular symptoms can extend to the eyes, which appear brown when viewed externally.

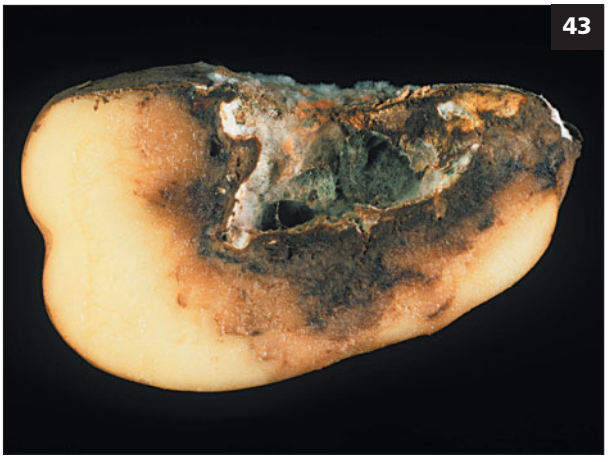
40 Wrinkled surface of lesion with white cushions of mycelium-bearing spores.



41 Tuber symptoms of *F. sulphureum* – light to dark brown lesions.



42 Tuber symptoms of *F. coeruleum* showing pink mycelium.



43 Tuber symptoms of *F. coeruleum* showing white to grey mycelium lining a cavity.

Wilting caused by *Fusarium avenaceum* can occur at the same time as those of *Fusarium oxysporum* f.sp. *tuberosi*, from mid-season onward. Like most wilts, symptoms are more severe in dry, hot conditions, and a sudden collapse of the plant is possible. Whilst symptoms may be worse on one side of a plant, general stunting of growth may occur and aerial tubers may develop, as well as reddening of stems. Vascular discoloration in the stem occurs below, but also sometimes above, the ground.

Rotting of the roots and below-ground stems characterizes wilting caused by *Fusarium solani*. Vascular staining is usually absent. Aerial tubers may develop as a response to infection. A dry stem-end rot and vascular discoloration of tubers may develop. Wilting caused by *F. javanicum* is similar to that of *F. eumartii*.

Tubers: Some wilt pathogens cause symptoms in daughter tubers. These appear as vascular browning and a sunken rot initiating from the stolon end.

Status of the disease

Dry rot and wilt caused by *Fusarium* spp. have a worldwide occurrence and can have a major impact on potato production. *F. coeruleum* and *F. sulphureum* are the most important dry rot pathogens. The former is more common in cooler climates and is the predominant species causing dry rot in northern Europe. *F. sulphureum* is a major pathogen in North America, causing seed piece decay and storage rots. Increased use of machinery to harvest tubers has resulted in a rise in importance of these wound pathogens. Of the wilt pathogens, their importance varies from region to region. For example, *F. oxysporum* f.sp. *tuberosi* is mainly of significance in tropical regions. Wilt diseases caused by *F. eumartii* and *F. javanicum,* whilst of little significance in Europe, are relatively more important in North America. *F. solani* and *F. avenaceum* are weak parasites, often relying on other factors to create conditions conducive for infection.

Life cycle and biology

The teleomorph stages have no apparent role in disease development. Tuber infection by dry rot *Fusarium* spp. only occurs through wounds. These wounds may occur whenever the tuber is handled, for example, at harvesting or at seed cutting or through lesions caused by pests such as slugs. Infection of seed tubers may occur through damaged sprouts after planting. Dry rot can occur from soil- or seed-borne inoculum, although the latter is probably more important. Seed tubers with lesions rot down and release spores into the soil, which contaminate daughter tubers at harvest. Seed tubers or seed pieces contaminated by spores may become invaded and rotted and release spores into the soil. Although the optimum temperature for growth of *F. coeruleum* and *F. sulphureum* is much higher, they can infect successfully at low storage temperatures (3–4°C). Low temperatures slow the process of wound healing and permit the pathogens to establish. Symptoms can take months to express themselves when tubers are cold, but at higher temperatures symptom expression can occur in weeks. Susceptibility to infection changes with time during storage; tubers often being most susceptible late in storage when they are graded.

Wilt can arise from soil-borne inoculum or from infected seed. Resting spores (chlamydospores) are produced by *Fusarium oxysporum* f.sp. *tuberosi*, *F. eumartii* and *F. javanicum* and can persist in plant debris in soil for years. Penetration by *F. oxysporum* f.sp. *tuberosi* and *F. eumartii* occurs in the root elongation zone and may be enhanced where nematodes are also attacking the roots. The pathogen spreads up the vascular tissue, often in the form of microconidia. Wilting is a result of blocking of the vascular tissue. Infection of stems by *F. eumartii* can also occur from infected mother tubers/seed pieces. By contrast, *F. javanicum* only occurs from infected mother tubers.

Inoculum can be introduced to a location by planting contaminated seed or where soil containing resting spores is spread on farm machinery, by wind and by irrigation, etc. Factors that exacerbate wilting are those that cause crop stress. Where stress is relieved, the impact is reduced. High temperatures, rapid growth and transpiration are conducive to Fusarium wilt.

Control

Dry rots

Avoid damage by ensuring a good skin set pre-harvest and correct setting of harvesting or other handling machinery. Dry curing using ventilation at temperatures of 10°C or higher to remove moisture after harvest or tuber cutting heals wounds and reduces ingress of the pathogen. Similarly, warming seed tubers before grading reduces wounding. Maintenance of store hygiene reduces inoculum spread to healthy tubers.

Treating tubers with fungicides at, or within, 24 hours of harvest or at grading can prevent infection of wounds and restrict spread to the daughter tubers. Some pathogens (e.g. *F. sulphureum*) have developed resistance to MBC-generating fungicides. Cultivars vary in their susceptibility to dry rot.

Wilts

Soil contamination can be reduced by long rotations between potato crops (if volunteer potatoes are eliminated). Planting healthy seed and adopting sanitation measures to avoid contamination of soil from dust, irrigation and machinery will reduce disease risk. Soils suppressive to Fusarium wilt pathogens through antagonistic microorganisms have been recorded. Seed piece treatment to prevent infection of the pathogens into cut surfaces can be effective if applied soon after cutting, but the use of fungicide drenches has proved impractical, and resistance to certain fungicides (e.g.MBC- generating fungicides) has been recorded.

Ammonium nitrogen, potassium deficiency and acid soils favour Fusarium wilt, as do high temperatures and rapid transpiration with associated rapid growth. Whilst environmental factors are unavoidable, using appropriate fertilizers, liming soils and minimizing crop stress will reduce the impact of the wilt pathogen. However, liming soils may increase the risk of common scab. Some cultivars are resistant to races of some wilt species.

Stuart Wale

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***Helicobasidium purpureum* (Tul.) Pat.**
(anamorph: *Rhizoctonia crocorum* [Pers.] DC; syn. *Rhizoctonia violacea*)

VIOLET ROOT ROT

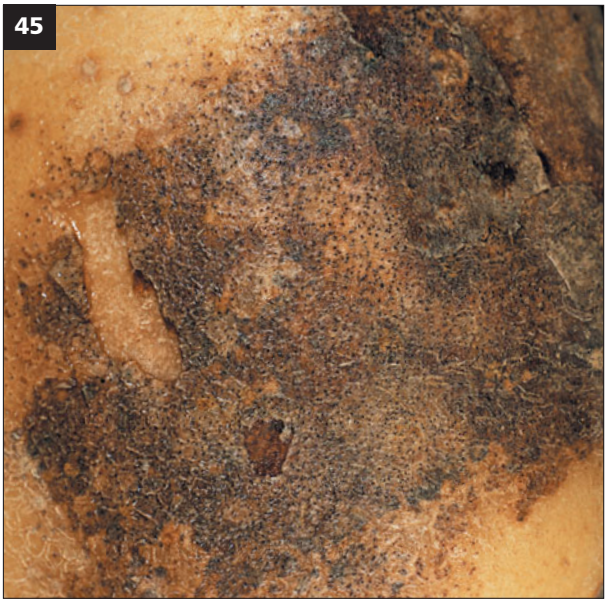
Symptoms

Haulm: Above-ground plant parts may become chlorotic and affected plants often wilt and subsequently senesce. Infected plants tend to occur in localized patches in the field. Diseased plants are difficult to diagnose using foliar symptoms alone.

Roots and Stolons: Distinct symptoms are rarely observed.

Tubers: The presence of violet or purple-brown mycelium on the tuber surface is diagnostic (**44**). This pigmented mycelium grows sparsely but evenly on the tuber surface and coalesces to form branched, root-like

44 Tuber symptoms of violet root rot.



45 Close up of violet root rot symptoms showing small sclerotia on the tuber surface.

mycelial strands up to 1 mm in diameter. Mycelium is septate, with branches arising at right angles, and often turns cinnamon-brown with age. The fungus also produces dark purple to black resting structures called sclerotia (**45**), which may be found in association with dark grey, sunken spots under the mycelial network. The fungus is generally limited to tissues near the tuber periderm; however, invasion by secondary pathogens can lead to more advanced wet rots and the disintegration of internal tuber tissues.

Status of the disease

Violet root rot occurs wherever potatoes are grown, but the incidence of the disease is rare. Reports from Europe indicate that the disease was more frequent in the 1930s than in recent years.

Life cycle and biology

The fungus can survive as sclerotia in the soil for a number of years in the absence of a host. Sclerotia are round to oval, dark brown to purplish-black, often covered with a thick felt and vary in size from a few millimetres to several centimetres in diameter. Sclerotia can germinate in the spring to initiate infection. The sexual phase of the life cycle is rarely seen, but results in the formation of basidiospores (10–12 × 6–7 µm) from sterigmata (10–35 µm in length) which arise from each cell of a hyaline, two- to four-celled basidium. The basidial stage is sometimes found as a white growth in association with potato stems near the soil surface. Airborne basidiospores may spread to adjacent plants to cause infection, but their importance in the epidemiology of the disease is unknown.

Isolation of the pathogen on artificial media can be difficult because its slow growth is often overtaken by competing contaminant organisms. Washing the tuber surface and surface-sterilization of infected tissues in a sodium hypochlorite solution (4–10%) is recommended prior to attempting isolation. Antibiotics may be added to the culture medium to inhibit the growth of other microorganisms. The optimum temperature and pH for growth of *H. purpureum* in culture are 30°C and between 5.5 and 7.0, respectively.

H. purpureum can infect a wide variety of hosts, and there is no evidence to date of strains that display host-specificity. Some of the more important hosts of the fungus include carrot, alfalfa, asparagus and sugar beet, as well as a variety of weed species such as *Sonchus* spp., *Rumex crispus*, *Urtica dioica*, and *Convolvulus arvensis*.

Control

Rotation with non-susceptible crops may be effective. Growing susceptible crops too closely in succession can lead to a build-up of inoculum in the soil. Adequate weed suppression and control of volunteer plants may also be beneficial. Infected tubers should be carefully disposed of and not simply ploughed under or left in cull piles. The movement of infested soil from one field to another on farm machinery should be avoided.

Rick Peters

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Helminthosporium solani **Dur. & Mont.**

SILVER SCURF

Symptoms

Haulm: There are no above-ground symptoms of the fungus.

Roots: There are no symptoms on roots.

Stolons: Lesions can be observed on stolons soon after tuber initiation.

Tubers: *H. solani* causes a superficial disease of the tuber periderm. The lesions are roughly circular in size, expanding up to several centimetres (**46**). Lesions may coalesce. The edge of the lesion is regular. The disease gets its name because the lesions are mostly silvery in colour. At times, bronze or gold discoloration of the outer layers of periderm can also occur. Loss of pigment in periderm cells results in the metallic appearance of the lesion. In soil, established lesions expand rapidly within a few weeks of planting infected seed tubers. New lesions may develop from spores present on the tuber surface at planting. Lesions on progeny tubers spread slowly on the surface when in soil. The lesions are usually small at harvest but enlarge during storage. Infection can occur in storage from spores present on the tuber surface after harvest or landing on the surface during storage (**47**).

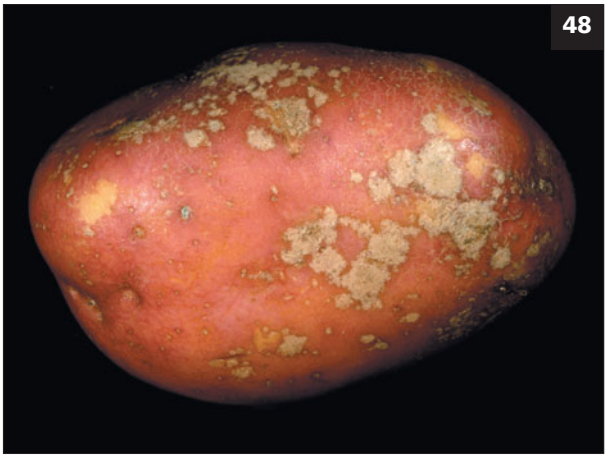
The disease occurs on white-, russet- and red-skinned cultivars, but symptoms are most noticeable on red-skinned potatoes (**48**). In severe cases the pathogen causes skin freckling of the tubers – often called 'elephant ear' texture skin.

Status of the disease

Silver scurf occurs wherever potatoes are grown. It is a common storage disease in Australasia, Canada, Israel, the Netherlands, the USA and the UK. In the past 10 years silver scurf incidence has increased, and it has become an economically important disease through reduction in cosmetic quality of washed fresh-packed potatoes. Silver scurf does not usually cause yield loss, but severe seed infection can affect vigour. The disease is also becoming important in potato processing, because crisps made from potatoes with severe silver scurf infection may result in blackened edges, making the product unmarketable. Because of silver scurf and

46 Circular lesions of silver scurf.

47 ***H. solani*** **conidiophores and conidia on the tuber surface.**



48 Silver scurf is more obvious on red tubers.

skin freckling, retail grocery stores reject consignments of fresh market potatoes. Fresh weight reduction of tubers may also occur due to excessive moisture loss from the tubers through lesions.

Life cycle and biology

Large, cylindrical (7–8 × 18–64 µm), dark, three- to ten-celled, slightly curved conidia with thick walls and up to eight septa are produced successively on new growing tips of dark, septate, irregular conidiophores; the structures often resemble Christmas trees under the microscope. In most cases conidia and conidiophores of successive generations are produced at the junction of dead symptomatic tissue and healthy tissue. In the soil, spores are released from seed tubers and stolon lesions and spread passively to daughter tubers, particularly during harvest. Conidia produced in storage conditions are released and carried to other tubers via circulating air. Under favourable conditions – moderate to warm temperatures (10–32°C) and very high humidity or free water – conidia germinate on plant tissue by polar germ tubes and cause infection of the tuber. The mycelium invades parenchymatous tissue and the tissues lose pigmentation and collapse, resulting in a silver tuber lesion. Where long rotations exist, the source of inoculum of the pathogen is considered to be seed-borne (e.g. in Europe), however, in other regions (e.g. North America) the source of primary inoculum can be seed-borne and/or soil-borne. Therefore, transmission of silver scurf can occur through infected seed introduced into soil or through conidia present in soil.

Control

Once infected, there is no effective control against silver scurf, especially in storage. Tubers treated with various fungicides at planting, at harvest, or at both times can reduce silver scurf infection, but post-harvest fungicide treatments have not proved particularly effective in reducing the disease in storage. Seed tuber treatments, such as benomyl, captan, imazalil, thiophanate-methyl and thiabendazole, reduce the severity of silver scurf at harvest; however, their effects do not usually extend into storage. In recent years, *H. solani* has developed resistance to thiabendazole through its extensive use. No cultivars carry high resistance to silver scurf.

Development of management practices that reduce silver scurf is a key element in control of the disease. These include avoiding a delay in harvest and exposure of tubers to the pathogen in the soil, rapid drying of tubers after harvest, avoiding long periods of condensation on tuber surfaces in store and good store hygiene. These measures will reduce tuber infection and disease spread. In addition, as the pathogen can be introduced into soil by seed tubers, planting silver scurf-free seed is also recommended.

Deena Errampalli

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Phoma exigua* Desm. var. *exigua

POCKET/BUTTON ROT

Symptoms

Haulm: Pycnidia can be detected on dead or senescent haulm, but there is usually little evidence of infection before senescence.

Roots and stolons: No distinct symptoms are recognizable.

Tubers: Small dark and usually circular depressions develop in the tuber skin, most frequently at wounds, eyes or lenticels. The hard lining covering the depressions can easily be removed to leave a smooth cavity, delimited by the wound cork, which prevents the further spread of the fungus. Rots caused by *Phoma exigua* var. *exigua* are smaller and less severe than those caused by *Phoma exigua* var. *foveata*, the causal agent of gangrene (see page 44). A mild infection may give rise only to a form of skin necrosis. In the final stages of the disease the dry internal tissue may vary from greyish

pink to black, and mycelium in the cavities is white or grey and may bear black pycnidia which burst through the skin to give black single or cluster 'pin heads', usually arranged in curved lines.

Status of the disease

Phoma exigua var. *exigua* is a weak but widespread pathogen that occurs in most European countries, Russia, the United States, Canada and Australia. This pathogen has been isolated from more than 200 genera of phanerogams. The disease is less severe than gangrene caused by *Phoma exigua* var. *foveata,* and it is restricted to some parts of a field or some fields in a production area. It is only infrequently reported as a serious disease on current commonly used potato cultivars. The causal agent invades wounds effectively under conditions which adversely affect the formation of wound periderm.

Life cycle and biology

Pycnidia are usually globoid (90–200 µm) and dark brown to black. Initially subepidermal, they become erumpent and extrude hyaline, non-septate, cylindrical pycnidiospores (4–5 × 2–3 µm). In culture on 2% maltose *Phoma exigua* var. *exigua* produces zonate colonies, and it can be distinguished from *P. exigua* var. *foveata* by the lack of anthraquinone pigments that are present in the latter species.

Infected seed tubers produce diseased stems in which infection remains latent during the growing season. Usually, pycnidia appear associated with nodes as stems begin to senesce, and they can provide an ample source of pycnidiospores which can be washed into the soil to act as inoculum on the tuber surface and on nearby plants. Rots in mother tubers continue to be active in the soil, produce pycnidia and constitute another source of inoculum at tuber harvest. Before harvest, tuber infection may occur through eyes and proliferated lenticels, usually when soil moisture is high. Most of the disease development occurs after harvest, through damage of the tuber skin. Wounding introduces infection from contaminated soil on the tuber surface or stimulates development of the fungus already latent in the periderm. *P. exigua* var. *exigua* can be easily isolated from arable soils, and it seems to persist in the soil for longer periods without a host plant than *P. exigua* var. *foveata*.

The optimal temperature for the germination of conidia is 25°C and the maximum is 28°C, while optimal growth occurs at 22–26°C, with a maximum at 30°C and a minimum at 4°C.

Metabolites identified include an antibiotic substance E active against both bacteria and other fungi (e.g. *Fusarium coeruleum* and *Phytophthora infestans*) and the cytochalasins A and B with phytotoxic properties. Wet soil conditions, night frosts and low day temperatures (less than 12°C) around harvest time and low store temperatures (2–10°C) favour disease incidence.

Control

Pocket rot is a tuber and soil-borne disease. Infected seed tubers can produce diseased haulm pycnidiospores that can infect daughter tubers. Most of the infection that develops in seed stocks is associated with damage, and disease development may not reflect initial inoculum levels. Infections can spread in store from diseased to healthy tubers either through the buds or, under moist conditions, through enlarged lenticels. There are differences in resistance to pocket rot among potato cultivars, and in general the disease is more frequent in early than late cultivars. Disease incidence can be reduced by applying general measures to control storage diseases; avoiding damage during harvest, removal of damaged tubers, not harvesting from wet soils, drying tubers after harvesting, respecting the appropriate period for wound healing (extra oxygen at 10–15°C for at least 10 days), no movement of tubers during storage and chemical treatment of seed tubers. Factors that retard tuber skin set at harvest, such as high rates of nitrogen fertilizer, may influence the extent of pre-lifting infection. A range of fungicide treatment options is available including fumigation with 2-aminobutane several weeks after harvest, mist sprays of thiabendazole and imazalil at or immediately after harvest and fungicidal dips.

Ruth Heinz & H W (Bud) Platt

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Phoma exigua var. *foveata* (Foister) Boerema
(Syn. *P. solanicola* f. *foveata*, *P. foveata*,
P. exigua f. sp. *foveata*)

GANGRENE

Symptoms

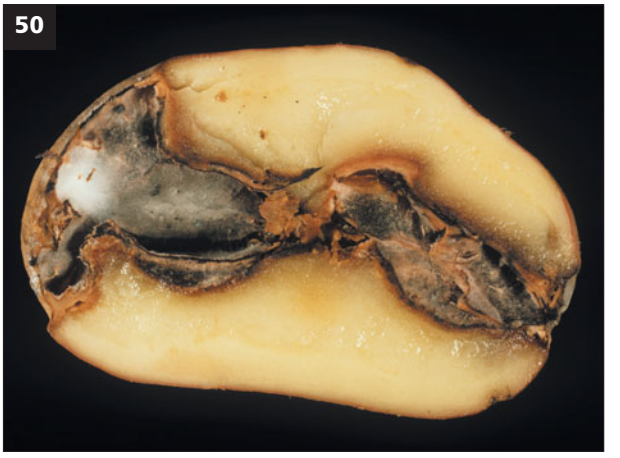
Haulm, Roots and Stolons: No distinct symptoms are normally seen. The stem base, junctions of petiole and stem on the haulm, stem bases, stolons and other underground parts may become infected during growth. However, the presence of the fungus is only detected easily once the haulm senesces or is killed, at which time clusters of black pycnidia develop on the dead or dying tissue.

Tubers: After infection, small thumb-mark depressions develop on the surface (**49**). The lesions are dark brown or black in colour. With time, lesions expand and small black pycnidia develop on the surface. When the tuber is cut across the lesion, a dark brown, purple or black rot extends into the flesh (**50, 51**). The extent of the rot can vary; at one extreme it may be confined to the surface, and at the other much of the tuber tissue can be taken up by the lesion. The area of the lesion at the tuber surface may give no indication of the extent of internal rotting. The margin of the lesion is clearly demarcated from healthy tissue. Within the lesion, cavities may form in various dark hues. Pycnidia form on the lining of the cavities.

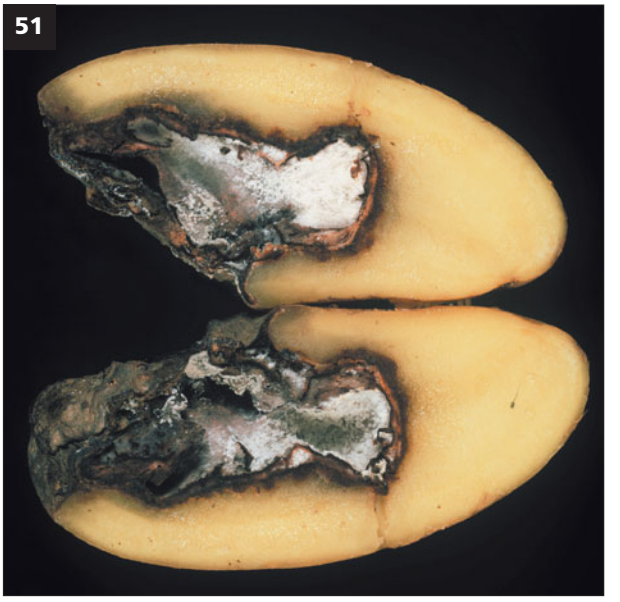
Status of the disease

Phoma exigua var. *foveata* is only of importance in northern Europe, although it has been reported in Australasia and other parts of the world. By contrast with the related weak pathogen *P. exigua* var. *exigua* (see Pocket Rot), *P. exigua* var. *foveata* is only short lived in soil (<2 years), although it can persist longer if volunteers and certain weeds are present. The tuber rot is of importance in cool, temperate climates. At worst, whole stocks have been discarded. More usually it is of sporadic occurrence in susceptible cultivars after late harvest in cold, wet seasons.

49 External tuber symptoms of gangrene.



50 Internal symptoms of gangrene.



51 Internal symptoms of gangrene.

Life cycle and biology

Pycnidiospores (4–5 × 2–3 µm) can persist in dust in store – especially on a grading line – and contaminate otherwise healthy tubers. Infected or contaminated tubers are the most important source of the fungus to the growing crop. After planting, the fungus on the tuber surface or released from a rotting tuber can infect stem bases, stolons and other underground parts. Above-ground infection of haulm may occur as growth proceeds. Pycnidia can spread in aerosol droplets generated by rainfall and infect healthy crops. The number of spores spread in this way increases as crop senescence or haulm destruction proceeds. Lesions both above and below ground remain restricted until senescence or the haulm is killed, when the lesions develop and produce clusters of pycnidia (90–200 µm). Pycnidiospores are either released into the soil or washed into the soil by rainfall. Spore numbers increase with time around the daughter tubers. Latent infection can occur before harvest when the fungus penetrates the tuber periderm and forms a small pad beneath it. This latent infection can be stimulated to develop by movement of the tuber, for example, at harvest or grading. However, the most important mechanism of infection is through wounds created during the handling of tubers.

There is evidence that the main source of spores for infection is infected underground parts and not rotting mother tubers or above-ground lesions. In general, the later the harvest the greater the build-up of spores around the daughter tubers and the greater the risk of infection. This is particularly true when soil conditions are wet and tuber temperatures low. Low temperatures increase the risk of wounding, delay wound healing and make it more difficult to dry tubers. Cultivars differ in their susceptibility to gangrene; however, susceptibility to *P. exigua* var. *foveata* has been found to increase during the storage period.

Control

The earlier that haulm destruction and harvest take place the less the build-up of inoculum around the daughter tubers. Reducing damage during harvesting and subsequent handling is a key element of a control strategy. Rapid drying of tubers after harvest and curing for 7–14 days in a warm but dry (<80% relative humidity) atmosphere creates conditions favourable for wound healing but unfavourable for infection. Various fungicides, such as thiabendazole and imazalil applied as spray applications at, or soon after, harvest, or fumigation with 2-aminobutane once wounds have healed after harvest can effectively reduce infection levels. However, fungicides work best when combined with early harvest and dry curing. Avoiding spore contamination during multiplication by good hygeine in store and early harvest can minimize build-up of the pathogen.

Stuart Wale

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Phytophthora erythroseptica Pethybr.

PINK ROT

Symptoms

Haulm: Pink rot does not normally occur on potato leaves and stems except when extensive infection of underground plant parts results in plant wilt, at which time leaf and stem chlorotic and necrotic symptoms can be observed. Lesions may form at the stem base, and infected stems turn brown with water soaking and light brown vascular discoloration. Severely infected plants may wilt and die prematurely.

Roots: Symptoms of pink rot on potato roots typically involve a light brown discoloration or streaking along the roots.

Stolons: Symptoms of pink rot on potato stolons typically involve a light brown discoloration or streaking along the stolons.

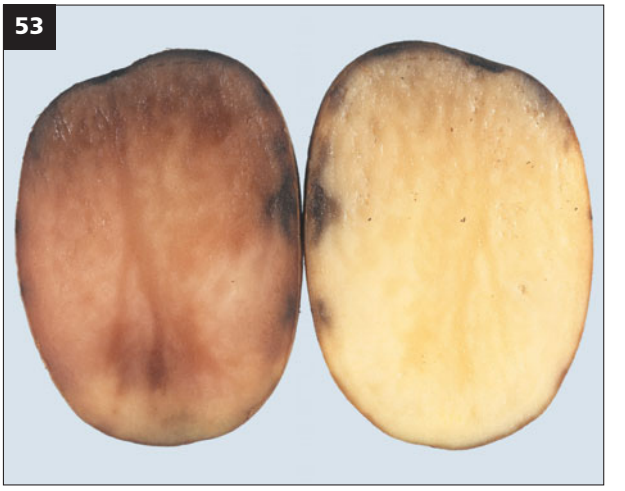
Tubers: Tubers become infected through the stolons, eyes, lenticels and wounds. Exterior surfaces of *P. erythroseptica*-infected tubers have irregularly shaped, small to large, dull brown-coloured lesions with dark skin tones around the eyes and lenticels (**52**). A dark line on the tuber surface delineates the extent of infection, and the infected tuber tissues have a soft, wet, spongy texture. Internally, rotting of the tuber tissue often begins at the stem end and is creamy or light brown. Cut tubers can ooze a clear, odourless liquid when squeezed. Cut surfaces change colour to pink (**53**), brown and then black following exposure to air for several hours. Continued disease development results in a wet rot and complete tuber tissue breakdown.

Status of the disease

Pink rot is one of the most damaging potato tuber diseases, since harvested tubers that appear healthy can rot completely within a few weeks after being placed in store. The causal agent of pink rot, *Phytophthora erythroseptica*, occurs in soils in all potato production areas of the world. It causes disease symptoms, such as root rots, leaf spots, crown rots, fruit rots, wilt, and leaf and petiole blights, on several plant species. In addition to potatoes, this pathogen attacks lilies, peas, raspberry, sugarcane, tomatoes, tulips, vetch, wild rice and other species, and it has been isolated from the roots of many non-solanaceous plants including wheat and rye.

Pink rot is favoured by cool, moist, environmental conditions and is enhanced in monoculture or short crop rotation systems. Recent increases in disease occurrence and severity have been related to the development of strains of the pathogen resistant to the fungicide metalaxyl.

52 External symptoms of pink rot.



53 Internal symptoms of pink rot: pink coloration after cutting (left) and freshly cut (right).

Life cycle and biology

Originally considered a fungus, *Phytophthora erythroseptica* is now termed a water mould and classed in the kingdom Chromista, phylum Oomycota, class Peronosporomycetidae, order Pythiales and family Pythiaceae.

Asexually produced sporangia (39–47 × 25–29 µm) of *P. erythroseptica* are hyaline, variable in shape, ellipsoid, thin walled, and non-papillate. Sporangia can germinate directly or cleave to form zoospores that move through soil via water films. Oogonia form readily in single isolate culture on V8 agar; they are globose and 34–36 µm in diameter. Antheridia are spherical to ovoid and 14–16 × 11–13 µm. Sexually produced oospores are 28–31 µm in diameter with walls about 3 µm thick.

Pink rot is most damaging at soil temperatures above 20°C in waterlogged soils. The pathogen is active at temperatures of 5–33°C (optimum 20–27°C). Germ tubes from sporangia, zoospores or oospores enter the host via open lenticels, wounds and tuber eyes. Sporangia will form on sporangiophores and mycelium growing on the surface of the host tissue at the edge of the diseased area. The soil-borne pathogen can persist in soil for many years, since oospores are disseminated from diseased potato stems, roots and stolons. Zoospores are probably the main infective agent in soil and can swim through soil water to the host, where they encyst and germinate to form a germ tube which penetrates the host. Following infection of below-ground plant parts, mycelium grows via stolons into daughter tubers. As infected plant parts decay, oospores formed previously will be released into the soil. However, potato tubers with eye infections caused by zoospore attack may not rot prior to harvest. Tubers with superficial infections may have viable mycelium and oospores but appear to be healthy when placed in storage, only to rot a few weeks later or remain healthy until planted – resulting in disease initiation at new sites. The pathogen can also be transmitted in storage or during tuber handling periods to healthy tubers which appear sound but carry oospores or mycelium in the skin or eyes from which infections can develop after planting.

Control

Potato disease management strategies require the use of a large number of disease control tools. These include:

- Elimination or reduction of inoculum sources is the most important measure, and new molecular biology-based methods for pathogen detection have been developed since detection based on disease symptoms is not always effective.
- In areas where oospores survive in potato field soils and plant debris, or where buried tubers survive between growing seasons (volunteer potatoes or groundkeepers), crop rotations of four or more years and/or the use of soil treatments (e.g. solarization, chemicals) may be needed. Areas with infected non-potato hosts or with exposed waste piles of infected potato tubers and debris may require destruction via composting, burial of the diseased material, covering with polythene or treatment with chemicals to prevent sprout growth.
- Fields or field sites with a history of pink rot should be avoided and cropped to non-hosts to reduce soil-borne pathogen population levels.
- Selection of potato cultivars with resistance to pink rot is recommended, but most of the current commercially available cultivars only vary slightly in their susceptibility to the disease. However, potato breeding programmes are developing new cultivars with greater levels of disease resistance.
- Seed tubers should be disease-free and handled to prevent the spread of disease by avoiding moisture conditions that activate mycelial growth, spore production and germination, and tissue invasion. Pink rot symptoms are not easily detected during grading. Visual inspection of washed seed tubers will indicate the extent of attack in a stock. Seed treatment fungicides (e.g. mancozeb) may be used to limit spore development but are not 100% effective.
- Planting in warm, well-drained soils will hasten healthy plant growth and limit plant-to-plant disease spread.
- During the growing season, inspect the below-ground plant parts for symptoms of pink rot. Infected plants should be removed from the field and destroyed by burning or composting. Where resistance to metalaxyl is not present, the fungicide may be applied at planting or to foliage when newly formed tubers are 2–3 cm in diameter as a means of reducing disease occurrence.

- Crop harvest should be delayed until the foliage has died naturally or following application of desiccants to encourage tuber skin development. Late season tuber infections will occur most frequently in moist soils.
- Harvest tubers when conditions limit tuber damage and the amount of soil adhering to the tuber surface. Wet areas in the field should be harvested separately to reduce the spread of disease from tuber to tuber during the harvest and handling operations.
- Avoid grading pink rot-infected tubers with secondary wet rotting, since this spreads the pathogen to healthy tubers. Ventilate to dry up wet-rotting tubers. Grade to remove diseased tubers and establish potato storage conditions that prevent excess moisture to limit pathogen growth and disease spread.

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Phytophthora infestans (Mont.) de Bary

LATE BLIGHT

Symptoms

Haulm: Late blight symptoms on potato leaves and stems begin as pale green, water-soaked, irregularly shaped spots that expand quickly under favourable environmental conditions. As the spots enlarge to brown or purplish-black necrotic lesions (**54**), a pale green to yellowish halo appears at the edge of the diseased area. The disease can quickly spread to entire leaflets (**55**), petioles and stems (**56**), eventually killing the plant. During moist conditions, a fluffy white growth of mycelium and asexually produced sporangia appears, generally on the under-surface of the leaf, at the leading edge of the diseased area (**57**). With excess moisture, spore production can be observed on upper surfaces of leaves, and lesions may have a 'greasy' appearance. Diseased plants have a distinctive, somewhat fishy odour.

Roots and Stolons: Symptoms of late blight on potato roots and stolons are rare.

Tubers: Exterior surfaces of late blight-infected tubers have irregularly shaped, small to large, slightly sunken areas of leathery, purplish-brown skin (**58**). Below the skin, potato tissues are tan or light brown in colour with a dry granular rot extending into the tubers (**59**). Lesion development is slow under dry, cool conditions, while under moist conditions a fluffy, white growth of mycelium and sporangia can occur. In addition, a wet rot may occur if secondary fungal and/or bacterial pathogens invade the diseased areas.

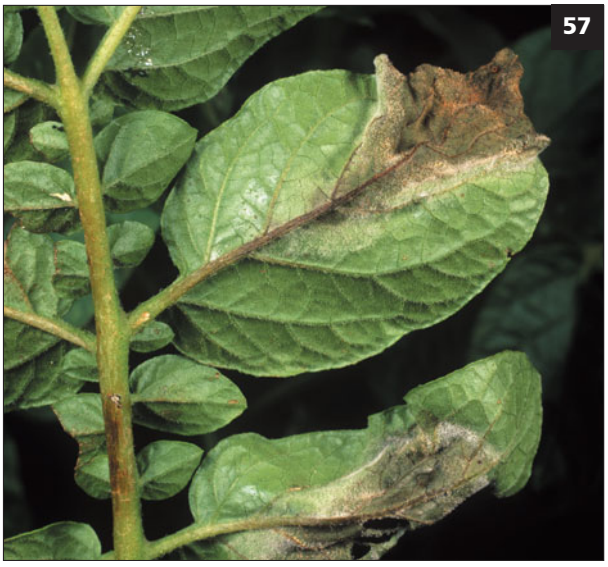
Status of the disease

Late blight is one of the most damaging diseases of potatoes worldwide, sometimes causing widespread agricultural disruption. One of the best-documented effects of the disease was the Irish potato famine of the 1840s, which had a severe impact on human health and social conditions nationally and internationally. Potato late blight occurs annually in most potato production areas in the world and can result in 100% crop losses. Originating in the highlands of central Mexico, the pathogen was transported during the 1830s and 1840s via infected potatoes to Europe and the USA and subsequently worldwide, either as spores carried by wind

54 Symptoms of late blight on upper surfaces of leaflets.

55 Late blight: infection of growing point.

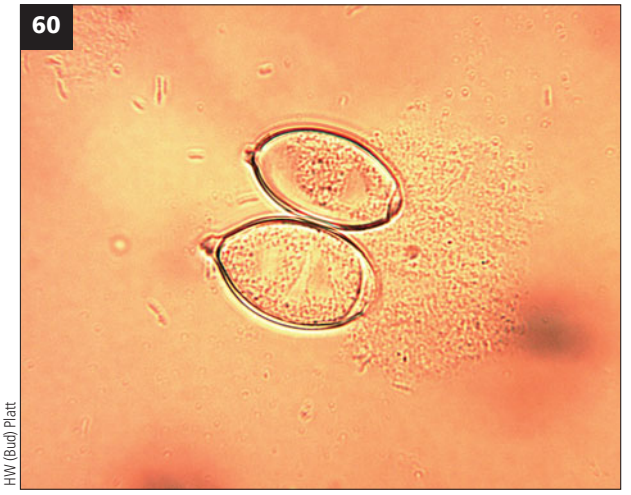
56 Symptoms of late blight on stem.



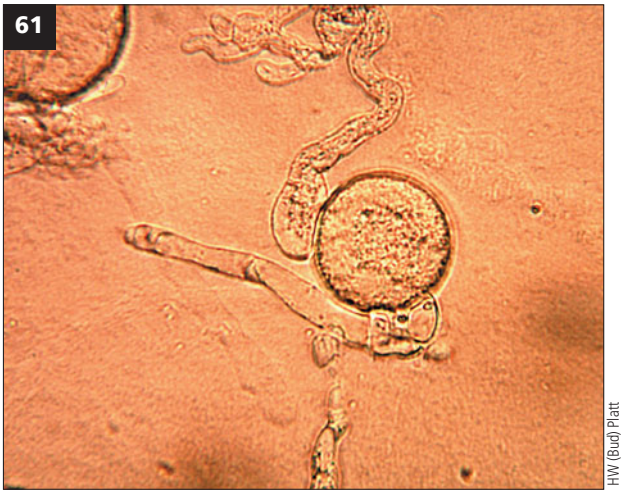
57 Symptoms of late blight on lower surface of leaflets.

58 External symptoms of late blight on a tuber.

59 Internal symptoms of late blight in a tuber.



60 Sporangia of *P. infestans*.



61 Oospore of *P. infestans*.

and water or in diseased potatoes and tomatoes. While the pathogen exists as both A1 and A2 mating types in central Mexico, the original spread globally involved only the A1 mating type. Despite the dependence on asexual reproduction of these A1 mating type populations, the pathogen has successfully become established around the world and has maintained its presence by adapting to a variety of disease management strategies introduced during the past 150 years.

Late blight is favoured by the cool, moist environmental conditions prevailing in various maritime climates worldwide, where the disease occurs annually or very frequently. In drier regions, such as central North America, it may occur much more infrequently – such as only once every decade or so. During the late 1980s and 1990s, late blight occurrence and severity dramatically changed worldwide in areas of both cool, moist and warm, dry conditions. This second movement of the pathogen, involving the A2 mating type, occurred from central Mexico to northern Mexico, Europe, the USA, Canada, and subsequently most other potato producing countries. In most countries, introduction of the A2 mating type included pathogen genotypes with resistance to metalaxyl, a highly favoured fungicide in most potato production areas because of its curative effect. The new genotypes were also more aggressive, since the old pathogen populations were quickly displaced and different pathogen populations evolved worldwide. For example, by the late 1990s, a diverse genotype profile, involving both A2 and new A1 genotypes generally at similar levels, occurred in European pathogen populations, while a single genotype, involving an A2 mating type with resistance to metalaxyl, predominated in populations in Canada and the USA.

Life cycle and biology

Originally termed a fungus, *Phytophthora infestans* is now classified in the kingdom Chromista, phylum Oomycota, class Peronosporomycetidae, order Pythiales and family Pythiaceae.

Asexually produced sporangia (21–38 × 12–23 µm) of *P. infestans* are hyaline, lemon shaped, thin walled, and relatively short lived (hours to days) outside living host tissue (**60**). Moisture above 90% relative humidity and temperatures of 3–29°C (optimum 16–21°C) are needed to stimulate spore production and germination. At temperatures of about 24°C this involves formation of a germ tube, while at about 12°C this involves formation of biciliate zoospores that swim freely in water prior to encysting and forming germ tubes. Airborne sporangia can be carried from several to hundreds of kilometres to new host plants. When transported by water they can travel to other plant foliage and potato tubers. Germ tubes enter the host via stomata or form an appressorium prior to the hyphae directly penetrating the host tissue. Inside the plant, non-septate mycelium grows intercellularly and intracellularly via haustoria that extend into cells; growth is most rapid at temperatures of 21°C. Sporangia form on

sporangiophores and mycelium growing on the surface of the host tissue at the edge of the diseased area.

Sexually produced oospores (24–46 µm) of the pathogen are reddish brown, circular, thick walled, and relatively long lived (weeks to years) outside of living host tissues (**61**). Oospores are formed when mycelium of an A1 and an A2 mating type come into physical contact and form oogonia and antheridia that unite. Oospores germinate via a germ tube with a terminal sporangium, which subsequently germinates directly or forms zoosporangia prior to germination.

Foliar disease generally originates from diseased seed, airborne sporangia, or soil-borne oospores, but within a few days plant parts can become infected due to the formation and transport of sporangia on diseased tissues of the same or neighbouring plants. As the growing season continues, sporangia and/or zoosporangia can be carried to, and infect, potato tubers. During non-cropping seasons, the pathogen survives as mycelium in living plant tissues, most often as a slow-growing dry rot in potato tubers in potato fields or stores. In some parts of the world, oospores survive long periods in soil and potato debris. In the spring, infected tubers, planted or buried overwinter in field soils, are often exposed to moisture and temperature conditions that result in the infection of sprouts or in the formation of sporangia that infect other seed tubers, emerging plant parts, or plant foliage (leaf and stem tissues) in contact with spores at the soil surface. Similarly, sporangia are formed on other diseased hosts (tomato, petunia, and some weeds such as nightshade) and transported to emerging potato plants. Infected potato tissues become sources of more sporangia to continue the disease cycle.

Control

Potato disease management strategies require the use of a large number of disease control tools. These include:

- Elimination or reduction of inoculum sources. In areas where oospores survive in potato field soils and plant debris or where buried tubers survive between growing seasons (volunteer potatoes or groundkeepers), crop rotations of four years or more or the use of soil treatments (e.g. solarization, chemicals) may be needed. Areas with infected non-potato hosts, or with exposed waste piles of infected potato tubers and debris, may require destruction via composting, burial of the diseased material, covering with polythene or chemical treatment to prevent sprout growth. Feeding infected and waste tubers to livestock or cutting and spreading the tubers on non-potato fields during the winter are effective alternatives to burial if the material is fully consumed or rotted prior to new crop emergence.
- Selection of potato cultivars with resistance to late blight.
- Seed tubers should also be disease-free or handled in a way that reduces or eliminates the spread of disease by preventing moisture conditions that cause spore production and germination. Late blight dry rot symptoms are not easily detected during grading. Visual inspection of washed seed tubers will indicate the extent of tuber blight symptoms in a stock. Seed treatment fungicides (e.g. mancozeb) may be used to limit spore formation and germination but are not 100% effective. Field inspection of seed lots the previous year can be helpful but does not guarantee seed tuber disease freedom the following year.
- Planting in warm, well-drained soils will hasten healthy plant growth. Ensure that an adequate hill (ridge) is made to prevent formation of potato tubers near the soil surface, which would increase the opportunity for infection.
- When crop emergence is 90%, or earlier if disease risk is high, inspect plants for symptoms of late blight and apply protectant fungicides (e.g. chlorothalonil, copper oxychloride, fluazinam, mancozeb, metiram) to the foliage. Initiate disease forecasting systems for local or region monitoring of disease risk conditions.
- Continue to inspect plants for disease symptoms and continue foliar fungicide treatments when the foliage within the rows is touching or when disease risk is high. Initiate regular fungicide application programmes based on late blight forecasting system estimates of disease risk or based on disease presence in the area. Apply fungicides that enter plant tissues (e.g. cymoxonil, dimethomorph, propamacarb) during periods of high disease risk, rapid plant growth, or disease-favourable conditions when disease is present in the area. Ensure that fungicide application systems are properly calibrated and adjusted in terms of pressure, volume, and so on, to maximize coverage of the foliage.

Duncan Smith/FLPA

62 Focus of potato blight in a crop.

- Destroy sources of inoculum in the area that cannot be successfully managed, such as 'hotspots' (focus of infection) in a field or fields with excessive amounts of disease (**62**).
- Continue crop inspection for disease. Continue a disease prevention fungicide programme until the foliage is completely dead naturally or following application of desiccants. Late season tuber infections will occur if sporangia are transported to moist soils.
- Harvest tubers when conditions limit damage and the amount of soil adhering to the tuber surface.
- Avoid grading blight-infected tubers with secondary wet rotting, since these spread the pathogen to healthy tubers. Ventilate to dry up wet rotting tubers. Grade and remove diseased tubers, apply post-harvest fungicides and establish potato storage conditions that prevent excess moisture and the formation of spores.

H W (Bud) Platt

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Polyscytalum pustulans (Owen & Wakef.) Ellis

SKIN SPOT

Symptoms

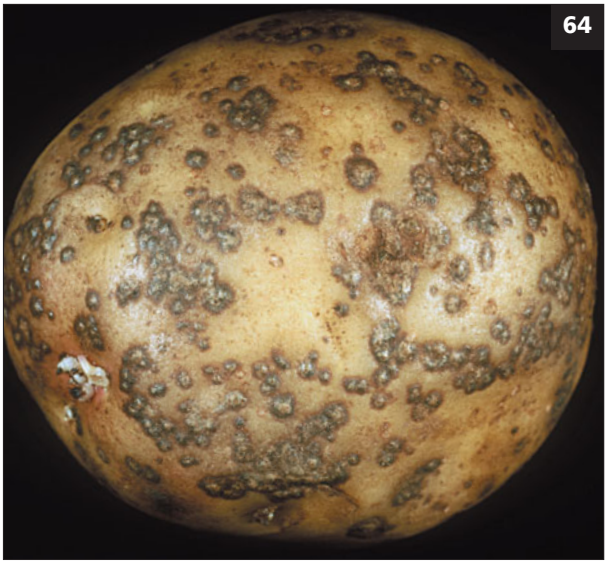
Haulm, Stem bases, Roots and Stolons: Initially, small, light brown spots develop, which may subsequently coalesce to form larger areas (**63**). These larger lesions are superficial and may have longitudinal cracks along them.

Tubers: Black or dark purple spots (1–3 mm diameter) develop on the tuber surface (**64**). The spots are sometimes slightly raised and may occur singly or in groups anywhere on the tuber surface, including around eyes or on damaged epidermis. The spots are usually superficial (1–2 mm deep) and easily removed by a single stroke of a potato peeler, but are sometimes deeper. Confirmation of the disease can be made by incubating tubers with spots or lesions on below-ground parts at 12–15°C in high humidity for 7–14 days. The typical chains of single-celled spores (2–3 × 6–18 µm) develop from infected tissue.

Status of the disease

The disease is of most importance in cool temperate regions of the world but has been of particular importance in northern Europe. The spots detract from the tuber's appearance and can reduce the market value of washed ware potatoes. Processors have experienced increased peeling with tubers showing extensive skin spot. The early use of sprout suppressants during storage can inhibit wound periderm formation and increase the incidence and severity of skin spot. Perhaps the most important effect of skin spot is that it can cause non-emergence or uneven emergence of seed tubers after planting as a result of invasion of eyes and buds. In severe cases, extensive areas of crop may fail to emerge. Stem numbers may be reduced as a result of eye infection, and this can influence tuber size distribution. The extent of non-emergence or uneven emergence can depend on the vigour of the variety and the soil conditions at planting. Cool conditions that inhibit sprout growth will allow the fungus to have a greater effect.

63 Stem infection by *P. pustulans*.



64 Skin spot on the tuber surface.

Life cycle and biology

The fungus can survive as microsclerotia (1 mm diameter) in crop debris and dust in store and in soil. Survival in crop debris can be for up to eight years, and healthy seed can become contaminated in store.

Survival in soil is usually for up to four years, and frequent cropping can result in infection from inoculum in the soil. However, under longer rotations (five or more years), soil inoculum is much less important than inoculum on seed.

Buds that emerge infected also develop infected stems. The fungus spreads up the stem and on to stolons and roots. Lesions sporulate, releasing spores into the soil. Infection of developing daughter tubers occurs through eyes initially, and then lenticels, with the tubers nearest to the mother tuber most affected. Spread and germination of spores are favoured by wet soil conditions, particularly in the period before harvest. Contamination of tubers at harvest and cool (<5°C), wet conditions during the early period of storage permit infection of tubers especially through wounds and abrasions. The fungus has a long latent period after infection, and typically symptoms will not develop for several months after harvest. Further infection can occur in store if damp conditions persist, and spores may then spread to other tubers.

Control

The use of disease-free seed is a key control measure. Initiating healthy stocks from stem cuttings or micropropagated tubers, long rotations and reduced generations of seed production all prevent build-up of disease in stocks. Store hygiene both during storage and between storage seasons is also important to reduce spread to healthy stocks. Early lifting in warm, dry conditions minimizes tuber infection. After lifting, 'dry' curing for 10–14 days will prevent infection. Prevention of condensation during storage and during sprouting will prevent spread later in storage. The use of fungicides at or post-harvest has been shown to give good control. Options vary from country to country. In some countries fumigation with 2-aminobutane is particularly effective. The use of benomyl, thiabendazole and mercury dips are all reported as effective control measures. However, dipping is not favoured and the use of spray applications is more usual, but less effective. Fungicide options include thiabendazole, imazalil and a mixture of the two. Where skin spot is present on seed at planting, the use of spray-applied fungicides will reduce development of the disease on the growing crop and progeny tubers. However, the level of disease in the progeny crop is not always related to the level of infection in seed. Cultivars differ markedly in their susceptibility to skin spot. On the whole, thin-skinned cultivars are more susceptible.

Stuart Wale

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***Pythium ultimum* Trow. (syn. *P. debaryanum*), *P. aphanidermatum* and other *Pythium* spp.**

WATERY WOUND ROT/LEAK

Symptoms

Haulm, Roots and Stolons: No distinct symptoms normally seen.

Tubers: Externally, there may be little evidence of internal rotting while the skin is stretched over a lesion. However, if the skin is broken fluid may leak out. On advanced lesions the surface is uneven and soft to the touch. When cut across, infected tubers show a range of symptoms depending on the state of disease development. Initially, infected tissue is only slightly discoloured compared with the colour of the rest of the tuber flesh and may not be easily demarcated from it (**65**). The texture of the infected tissue is creamy or cheesy. With time, the infected tissue darkens, and the junction with healthy tissue becomes demarcated by a dark brown margin (**66**). Vacuoles appear in the infected tissue, but no mycelium is visible in the vacuoles or on the tuber surface. Infected tissue frequently becomes invaded by secondary bacteria (**67**). Before bacteria invade, infected tissue is sweet smelling.

Status of the disease

Watery wound rot is a sporadic disease but recorded in many temperate and hot countries. In hot climates, the symptoms develop rapidly and are obvious soon after lifting. In cooler climates, symptom development can take considerably longer, especially if tubers are cooled after lifting and held in refrigerated storage. It is not possible to predict those fields where the risk of watery wound rot is high.

Life cycle and biology

The *Pythium* spp. that cause the disease are common soil inhabitants, persisting as thick-walled oospores. Where freshly cut seed pieces are planted, the fungus can invade the cut surface and cause seed piece decay. Infection of progeny tubers can occur at the point of stolon attachment following damage as a result of haulm desiccation. However, the principal way the fungus invades tubers is through wounds at harvest or through damage by sunburn or sunscald. Warm conditions at lifting favour infection, and the disease rapidly develops at temperatures between 16°C and 32°C.

65 Early stages of watery wound rot with infected tissue not clearly demarcated.

66 Later stages of watery wound rot showing darkened tissue and demarcated margins to the lesion.

67 Tubers infected by *P. ultimum*, showing secondary rotting by bacteria.

Control

Seed pieces should be treated with a fungicide or cured before planting. Although *Pythium* spp. are common in soils, where extensive watery wound rot has occurred in a field, that field should be avoided in the future. Avoiding desiccant injury to the stolon end of tubers and harvesting in cooler weather, where possible, will reduce conditions favourable to the fungus. The fungus is a 'water' mould and requires moisture to infect. Thus dry curing after lifting will make conditions for infection less suitable. Minimizing damage at harvest will reduce the risk of infection. No cultivar differences in resistance to watery wound rot are known, but those less prone to wounding are likely to be at lower risk.

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***Rhizoctonia solani* Kühn (teleomorph: *Thanatephorus cucumeris* [A.B. Frank] Donk)**

RHIZOCTONIA DISEASE: STEM, STOLON AND ROOT CANKER/ BLACK SCURF

68 Stem canker lesions on stems.

Symptoms

Haulm: Brown to reddish-brown lesions along the surface (**68**) or at the end of shoots result in pruning of stems as the lesion extends. Sometimes weakened, stunted plants will emerge with partial or complete girdling of the stem at the soil surface; these often die prematurely. Extensive sprout attack by the fungus results in no, or delayed, plant emergence and uneven stands of weakened plants. Further disease development on the sprouts can lead to sunken cankers girdling stem bases, premature death of smaller stems, chlorosis of main stems, upward curling, purple colouring of leaves and formation of aerial tubers (**69**). Depending on environmental conditions, signs of the sexual phase of the fungus can be observed; a white, powdery mould forms at stem bases with cankers at, or below, the soil surface (**70**). Mycelial mats can also be seen on stems, particularly on below-ground tissues.

69 Formation of aerial tubers as a result of *R. solani* infection.

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70 White collar (sexual) phase of stem canker (*T. cucumeris*) at soil level.

71 Stolon canker and pruning with secondary stolon forming.

Roots: Brown to reddish-brown lesions and/or mycelial mats along the roots or at root ends result in pruning of roots as the lesion extends around and through the tissue. The disease reduces the root mass and function, preventing normal plant growth and development.

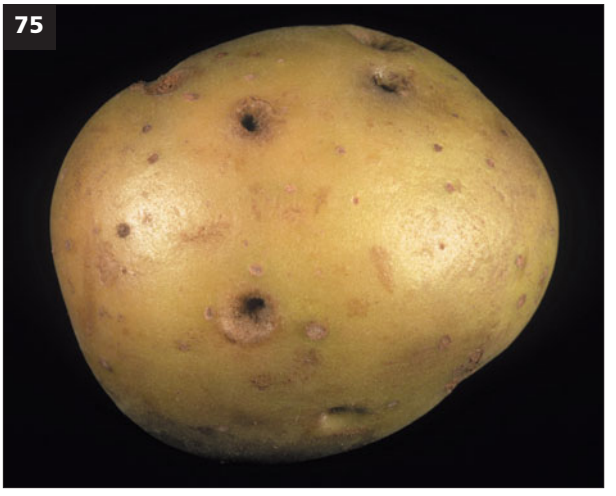
Stolons: Brown to reddish-brown lesions and/or mycelial mats along the surface or at the end of stolons (**71**) result in pruning of stolons as the lesion extends around and through the tissue. This phase of the disease causes malformed tubers. Stolon pruning also reduces the number of tubers formed.

Tubers: Black scurf is easily observed on the surface of washed or soil-free potatoes (**72**). The fungus produces black or dark brown structures called sclerotia which adhere to the surface of the tuber. Sclerotia are of

72 Black scurf symptoms.

73 Misshapen tuber and skin netting as a result of *R. solani* infection.

74 Surface pitting caused by *R. solani*.



75 'Holes' resulting from *R.solani* infection.

various shapes and sizes and can be observed shortly before harvest but generally form or enlarge at, and shortly after, harvest. The fungus also produces dark reddish-brown mycelial strands or networks on the surface of the tuber. Tuber symptoms do not cause extensive damage to the tuber tissues below the skin and can often be scraped from the tuber leaving no disease symptoms. However, disease occurrences on plant roots, stolons and haulms can result in misshapen tubers (**73**), tuber cracking and skin surface pitting (**74**, **75**) or netting (**73**), and stolon-end necrosis.

Status of the disease

Rhizoctonia disease occurs wherever potatoes are grown, and is associated with many crop and non-cropped plant species. Several strains or anastomosis groups (AG) of the fungus exist in soils, and not all cause all plant symptoms to the same degree on their various hosts; AG-3 is generally associated with the black scurf phase of the disease on potatoes. Diseased plants can usually be found in every field of potatoes, but disease incidence and severity vary greatly. Similarly, depending on the potato cultivar, timing and severity of disease, damage to the plants and effects on tuber yield and quality may be limited or extensive, but severe disease in the haulm does not always cause significant tuber yield losses, although tuber quality may be reduced.

Life cycle and biology

Rhizoctonia solani can survive for long periods on crop debris and in the soil as mycelium and sclerotia but also overwinters in a dormant state on potato tubers. Limiting the frequency of cropping with susceptible hosts and potato cultivars can reduce soil levels of the fungus. After planting, sclerotia germinate and mycelium invades roots, sprouts, and emerging stems particularly when cool, moist soils slow plant growth. Once they have emerged, infection of stems declines, but roots, stolons, and plant stems can be attacked throughout the growing season. The fungus can enter plants directly, but plant tissues wounded naturally or by cultivation methods are particularly susceptible. After the start of crop senescence, the rate of development of *R. solani* is more rapid. As plants mature and soil conditions become less favourable for the fungus, sclerotia are formed on tuber surfaces.

Control

No single disease control method is highly effective, and successful management must include strategies that reduce levels of the pertinent strains of the fungus in the soil and eliminate or limit the occurrence and disease potential of sclerotia on tuber surfaces. No potato cultivars are currently available with commercially acceptable levels of disease resistance.

Since sclerotia and mycelium of the fungus on potato tubers pose disease threats, use of seed with little or no fungus present will reduce disease risk. Fungicides can also be used for disease control, but currently available fungicides provide disease protection and not curative activity. Use of seed treatments with efficacious fungicides (e.g. thiabendazole, fludioxinil, tolclofos-methyl, pencycuron) will reduce the disease threat caused by sclerotia and mycelium of the fungus on the seed tubers. However, none of the currently available fungicides is 100% effective, and the fungus also exists in the soil so this must also be managed. Crop rotations involving production of potatoes once every three to four years and inclusion of cereal, grass and other less susceptible crops will reduce soil-borne levels of the fungus. Soil treatments (e.g. pentachloronitrobenzene, tolclofos-methyl, pencycuron) can also be used in some countries to reduce soil-borne inoculum levels, but the cost-effectiveness of these measures is not always favourable.

As the fungus is opportunistic and favoured by conditions that slow plant growth or result in weakened plants, strategies that improve plant growth and health will lessen disease risk and impact. Use of green-sprouted tubers, planting in warm, well-drained soils, and limiting the depth of the hill (ridge) to encourage rapid emergence of healthy plants will reduce early season disease occurrence and severity. Similarly, implementation of local crop management recommendations to enhance healthy crop growth and tuber production will limit disease impact.

Separation of haulm, roots and stolons from tubers prior to harvest (e.g. by haulm pulling) reduces black scurf development. Biological control of *R. solani* has been achieved by applying *Verticillium biguttatum* to tubers in a two-stage lifting process. Early harvest reduces black scurf especially when skins are mature and when done in a manner that reduces tuber wounding and soil adherence to the tubers. Conversely, delaying harvest of mature tubers and storage of potatoes coated with soil enhances the occurrence of black scurf. Immediate post-harvest tuber treatments (e.g. thiabendazole), when applied at recommended rates and to tubers without adhering soil, will also limit the formation, development and viability of the sclerotia and mycelium of the fungus.

H W (Bud) Platt

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Sclerotinia sclerotiorum (Lib.) DeBary

WHITE MOULD/ SCLEROTINIA STALK ROT

Symptoms

Haulm: The disease occurs on the stem either at the soil line, where the host plant makes contact with soil, or at the junction with leaf petioles (**76**). Early symptoms on stems are the appearance of water-soaked areas on which white fluffy mycelial growth subsequently develops. As the lesion dries out, infected areas are bleached to beige in colour, sometimes with a dark brown border. In early stages of stem lesion development, the foliage may not show any symptoms until the fungus grows and the stem rots. Then, the foliage above the lesion wilts and dies (**77**). With aerial symptoms, infection will start at a wound, usually where the leaf joins the stem, and moves into the stem. In the later stages of symptom development, large, dark, compact resting sclerotia are formed in the stem core (**78**). Sclerotia are white at first but become black and hard on the outside with sizes ranging between 2 and 10 mm in length. Most sclerotia are flattened and elongated.

Roots and Stolons: There are no symptoms on roots or stolons.

Tubers : Potato tubers are rarely infected by *S. sclerotiorum* in the field.

Status of the disease

Sclerotinia stalk rot occurs in temperate zones where potatoes are grown. The disease has been reported from the northern USA, the UK, Ireland and Canada. The disease has become increasingly important where environmental factors, such as high humidity from sprinkler irrigation and dense canopies of foliage, favour the development of the disease and where susceptible crops are intensively grown.

Life cycle and biology

The fungus *S. sclerotiorum* overwinters as mycelium in dead or living plants, but primarily as the hardened black sclerotia in soil. The sclerotia can survive for more than three to five years in this way. *S. sclerotiorum* operates at low temperatures and can cause infection from 10–27°C. In the spring or early summer, the

76 Infection at junction of stem with leaf petiole.

77 Girdling of stems by *Sclerotinia* lesions which result in premature senescence.

78 Resting sclerotia formed within the stem core.

79 Apothecia of *S. sclerotiorum*.

sclerotia germinate and produce from two, to many more, slender stalks that terminate in 5–15 mm diameter, cup-shaped apothecia, or fruiting bodies (**79**). Large numbers of ascospores are ejected from the apothecia into the air for a period of several weeks. The number of ascospores peaks at about full flower bloom. Following dispersal by the wind, the ascospores land and germinate to cause infection on potato stems and leaves. Stem lesions occur after row closure and blossom drop. Under prolonged periods of moist conditions, direct infection is caused by mycelium produced from germinating sclerotia. The mycelial strands attack the potato stem and leaves at the soil line.

Control

Control of this pathogen is very difficult because of its wide host range and persistent sclerotia. In the field, white mould can be limited by crop rotation, foliar fungicides and modification of the micro-environment. Crop rotation with cereals or grasses for four or more years limits the incidence of white mould. Rotations that include broad leaf crops susceptible to *S. sclerotiorum* increase the risk of disease in potatoes.

Control of white mould can be achieved by foliar spays of protectant fungicides (e.g. fluazinam or iprodione) and biocontrol agents (e.g. *Ulocladium atrum, Coniothyrium minitrans*). Foliar applications of protectant fungicides, prior to full blossom bloom and row closure to coincide with ascospore release, can limit the disease. However, one or more fungicide applications are needed where ascospores are discharged over an extended period of time. Systemic fungicides such as benomyl limit the pathogen, but resistant *S. sclerotiorum* isolates are likely to develop.

In fields where irrigation is used, the management of irrigation timing can avoid stimulation of the disease. Alterations to timing and frequency of irrigation during ascospore production – the two-week to eight-week period beginning at row closure – will reduce the chances of ascospore germination on the plant canopy.

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Spongospora subterranea (Wallr.) Lagerh.
f.sp. *subterranea* Tomlinson

POWDERY SCAB

Symptoms

Haulm: There are no above-ground symptoms of the disease.

Roots: The swimming spores of the pathogen can infect root hairs and surface cells of the root. Only when examined microscopically can these infection structures be seen. Galls can form on roots (**80**). Galls are irregular in shape but usually 0.5–2.0 cm in length.

Stolons: Infection of stolons can occur and occasionally results in small galls forming on them. The galls can superficially resemble those of root knot nematodes but can be differentiated by their internal structure.

80 Powdery scab galls on roots.

81 Scab form of powdery scab.

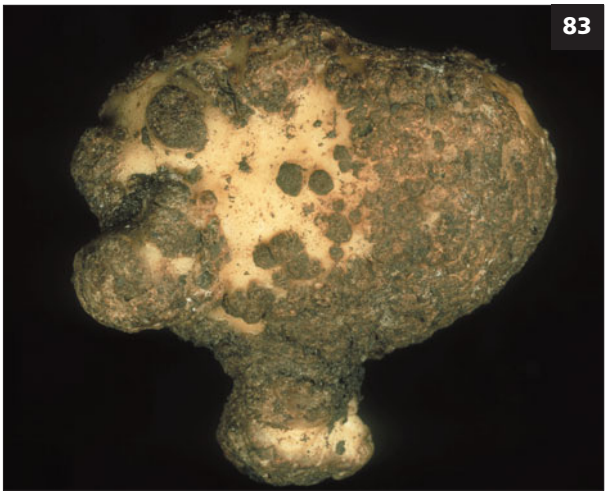
Tubers: There are two forms of the disease on the tuber, a scab form (**81, 82**) and a canker form (**83**). The scab form results from infection of lenticels in unsuberized tissue of the developing tuber. After infection, the pathogen develops slowly and is unseen with the naked eye for several weeks. A small purple-brown pimple (c. 2 mm) develops on the tuber surface then expands, turns brown in colour and bursts to reveal and then release a brown powdery mass of sporeballs (cystosori) within. The edges of the lesion are small flaps of loose skin with a smooth outline. In severe attacks the scabs can merge to form large areas of infected tissue. The lesions do not usually penetrate more that a few millimetres. The canker form is the result of infection of eyes. Infection stimulates the tuber tissue to grow and results in a cankerous outgrowth. The new growth is initially unsuberized and frequently becomes severely affected by the scab form.

Both forms are unsightly and result in tubers being discarded because of poor quality. Yield of the crop is rarely affected by infection, but it has been established that severely infected tubers when planted produce fewer stems and thus a reduced yield.

Status of the disease

Powdery scab occurs wherever potatoes are grown but is mainly of importance in cool temperate climates. Powdery scab is a particular problem in northern Europe, Australia, New Zealand and Pakistan. There have also been outbreaks recently in North America.

82 Close-up of powdery scab showing clusters of powdery spore balls after eruption of the skin.



83 Tuber showing cankers as a result of infection by *S. subterranea*.

The demand for blemish-free table potatoes in the western world, and the introduction of susceptible cultivars, has raised the status of this disease in the last two decades. Losses are incurred as a result of more critical grading to achieve a high standard of tuber quality. In the UK the disease became significant when a highly susceptible cultivar (Pentland Crown) rose to prominence. Although the area given over to the growth of this cultivar fell rapidly, a large number of susceptible cultivars followed it in popularity and the disease has become established. The pathogen is important as a vector of *Potato mop-top virus*.

Life cycle and biology

Cystosori (19–85 µm across) comprise a mass of thick-walled resting spores. They can survive in the soil for more than 10 years. In response to the presence of a host crop, spores within the cystosori germinate, releasing relatively short-lived, single, uninucleate primary zoospores. If free water is present, these zoospores swim to the host and infect it. Zoospores can infect root hairs and surface cells of roots. They become attached to the root, encyst and penetrate. Once inside the root the pathogen forms a multinucleate plasmodium by mitotic division. The plasmodium cleaves into segments, and each segment forms a zoospore. These secondary zoospores are indistinguishable from the primary zoospores and can further infect roots, thus mutiplying the inoculum. Both primary and secondary zoospores can infect tubers once they are formed. The phase of development when tubers are most susceptible to infection is during early formation when the tissue is unsuberized. However, as the tuber swells, there is always an area of unsuberized tissue at the rose end, and infection can occur throughout tuber development. Once in the tuber a multinucleate plasmodium forms, as in the roots. Infected cells are stimulated to multiply and cytoplasmic cleavage results in uninucleate cells around which cell walls develop. These are the spores which together form the cystosorus. Swelling resulting from hyperplasia and hypertrophy ultimately ruptures the epidermis.

Infection occurs at temperatures between about 9°and 17°C and is dependent on a high soil water content. However, the worst infection occurs when alternating high and low soil water contents occur. Clearly, the ability of a soil to hold water and the quantity and duration of precipitation (whether by rain or irrigation) can have a major impact on the extent of infection.

Control

If soil moisture and temperature are unfavourable for infection, although tubers may be at a susceptible stage they may escape infection. Thus in warmer climates where high soil temperatures persist at, and after, tuber initiation, there is less disease. However, the extensive use of irrigation can both reduce soil temperature and create a high soil water content. Careful use of irrigation is thus required, especially around the susceptible phase

at tuber initiation. In cooler, wetter climates where soil temperatures frequently fall within the optimum range for the pathogen, the quantity, duration and timing of rainfall can have a marked impact on whether disease occurs. In these climates, selecting free draining soils and preventing soil compaction by creating a good soil structure reduce the risk of disease.

The most effective and durable means of control is cultivar resistance. However, in some countries the cultivars most sought after for production are susceptible, and there is little opportunity to grow resistant ones. Where susceptible cultivars must be grown, selecting fields without a history of powdery scab can help avoid the disease. However, the pathogen can be introduced into a field on the seed planted or where manure from animals fed with infected potatoes has been spread. When growing in uncontaminated soil, a high degree of seed health is desirable. Where soil is contaminated, planting healthy seed is of less significance. Targeting resistant cultivars in contaminated fields will reduce disease risk. Methods of detecting the level of soil contamination in fields are being developed and these should assist matching cultivars to fields.

Many potential fungicides have been tested for the control of powdery scab as soil or tuber treatments, but with little success. Zinc has been shown to reduce disease severity when applied to the tuber or soil but is only of limited effectiveness. Fluazinam and flusulphamide have shown promise as soil treatments, but the effects are variable.

Stuart Wale

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Synchytrium endobioticum (Schilb.) Perc.

WART

Symptoms

Haulm: Aerial symptoms are often absent. Plants may have poor vigour and take on a darker than normal green colour. Sometimes galls form at the base of the stem (**84**), and occasionally they develop on upper stems, leaves or flowers. The above-ground galls tend to be green to brown, rather than the white to brown colour of subterranean galls.

Roots: Roots do not develop galls.

Stolons: Galls form at meristematic tips and vary from pea-sized to more than 10 cm in diameter.

Tubers: Galls resembling warty tumours and composed of convoluted masses of cauliflower-like excrescences form usually at tuber eyes and may completely disfigure tubers (**85**). Galls are white to brown in colour and are susceptible to decay by soil organisms.

Status of the disease

The disease occurs in limited areas on all continents except Australia. Quarantine measures restrict its distribution. Potato production is often restricted in known infected fields. The disease may cause only a slight reduction in quality or cause total loss of a crop. Disease severity varies with cultivar, pathogen strain, level of infection and weather conditions.

Life cycle and biology

The causal agent is *Synchytrium endobioticum*, which produces resting spores that survive in soil for more than 40 years. Sporangia germinate into uninucleate zoospores which penetrate host epidermal tissue. Zoospores encyst in host tissue and develop into sori which release haploid zoospores into neighbouring host cells. Host cells surrounding the infected ones are subject to hyperplasia and hypertrophy, resulting in the characteristic warts. Some zoospores conjugate and form a zygote which develops into a meio- (resting) sporangium. Sporangia are released into soil after the decay of host tissue and are disseminated on tuber surfaces, via soil movement, and perhaps by wind.

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84 Cankers of wart on stems.

85 Cauliflower-like cankers of wart on tuber.

Control

Quarantine measures are widely used to prevent spread of the wart pathogen into disease-free areas. Once introduced into an area, the contamination cannot be eradicated. Soil amendment with crushed crab-shell suppresses the disease. Resistant cultivars utilizing major gene resistance are available for growing in contaminated regions.

Solke H De Boer

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Thecaphora solani
(Thirumulachar & O'Brien) Mordue
(syn. *Angiosurus solani*)

POTATO SMUT

Symptoms

Haulm: No symptoms are visible on leaves.

Stems, Stolons and tubers: Malformed tubers are conspicuous, but wart-like galls may also be formed below ground on stems and stolons . Generally, galls are hard and vary from oval to an irregular shape. The biggest galls are those formed first on underground stems, as compared with those developing on tubers and stolons. Galls can be formed anywhere along the length of stolons. At harvest time, two kinds of symptoms are observed on tubers:

- Slight protuberances (less than 1–5 mm in diameter) that can sometimes be confused with symptoms caused by root-knot nematodes. These small galls (**86**) become sunken and suberized injuries two to three months later. Then, when the sprouting starts under storage conditions, new galls develop on the tuber surface – near sprouts (**87**), or on the tip of the sprouts.
- Small galls, mostly at the apical end, and some of them developing as secondary growth. Oval to irregular locular sori containing reddish-dark, granular-powdery masses of teliospores (**88**) can be easily seen when a gall is cut transversally. (Teliospores are spore balls composed of two to eight moderately firmly united ustiliospores.) Sori are distributed in a radial pattern (**89**).

Status of the disease

Potato smut is indigenous to the Andean region. It has been reported in Venezuela, Colombia, Ecuador, Peru, Bolivia and Chile, and there are also reports from Panama and Mexico. There are no reports of potato smut elsewhere in the world. *T. solani* develops in different kinds of soils and climates. This pathogen is prevalent in the cool, mountainous regions (2,500 –3,800 metres above sea level) of tropical zones, but it has also been found in the warmer coastal climates (100–400 metres above sea level) of sub-tropical zones. EPPO consider *T. solani* as an A1 quarantine pest, and it is also of quarantine significance for CPPC.

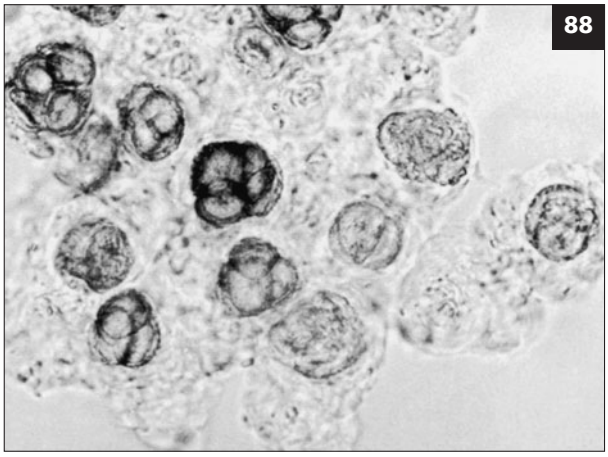
86 Tuber symptoms of potato smut.

87 Gall of *Thecaphora solani* on apical end of tuber.

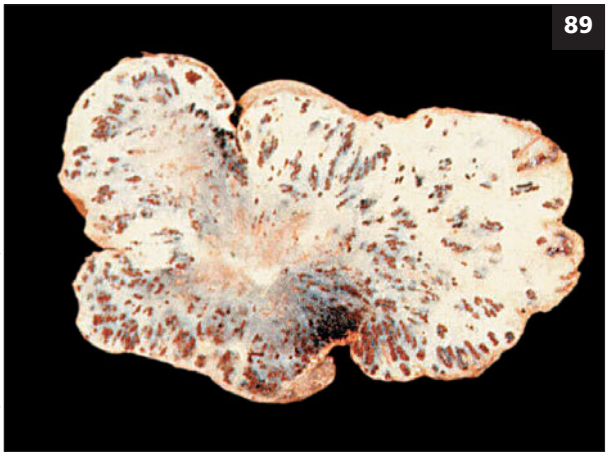
Biology and life cycle

Phylogenetic analysis of the large subunit (LSU) rDNA data of the genus *Thecaphora* and other smut fungi confirmed the initial classification of the pathogen as *T. solani*. It produces subglobose to ovoid or irregular spore balls in locular sori that are surrounded by a periderm six to eight cells deep (**88, 89**). Each spore ball, when mature, contains two to eight rust-brown spores. Spores are pressed together, but when they are teased apart they become free spores.

In nature *T. solani* survives in soil (seven years or more) or in tuber debris. Infection starts a few days after planting, especially in meristematic regions of young sprouts, underground stems, stolons or eventually in tubers. After they penetrate the cortex, hyphae ramify intercellularly, growing toward the phloem and



88 Teliospores (spore balls) of *Thecaphora solani*.



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89 Cross-section of gall showing radial distribution of locular sori.

parenchyma and causing hypertrophy in these tissues. The spores originate by compaction of the thick-walled hyphae at certain intervals.

The literature mentions that the infection is generally stimulated by high humidity in the soil during the first stages of growth. However, contradictory results from greenhouse studies indicate that different levels of humidity in the soil (20–25% and 50–55%) did not influence the development of the disease, potato smut being similar in both levels of humidity. There are no studies about optimal temperature for the development of potato smut, but in Peru it develops where the potato plants grow at between 5 and 20°C.

The principal hosts of this pathogen are *Solanum tuberosum, S. andigenum, S. stoloniferum* and other species of *Solanum*, although *Lycopersicon* and weeds such as *Datura stramonium* are also reported as natural hosts. Successful *in-vitro* cultivation of *T. solani* has been reported recently.

Control

The principal control measure is to plant smut-free seed potatoes in non-infested soils. Long rotations, effective control of volunteer plants or weeds (especially *D. stramonium*) and elimination of smutted galls after harvest are also recommended for infested fields. In countries where the disease is not present, inspections of imported vegetative materials from infested countries are necessary in order to find malformed tubers. However, a quarantine period to ensure freedom from the disease is required. A fragment belonging to ribosomal region ITS 1-5.8S - ITS2 from *T. solani* is being studied in order to detect latent infection using Polymerase Chain Reaction (PCR) tests. This could serve to test different materials including soil samples.

Different levels of resistance in commercial varieties and accessions from potato germplasm have been reported in several countries. Many potential fungicides have been tested for control of potato smut as soil disinfectants or tuber treatments, but with limited success. Carbendazim and thiabendazole have shown a reduction of attack of *T. solani,* but not the elimination of this pathogen.

Perez Willmer & Torres Hebert

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***Verticillium albo-atrum* Reinke & Berthier, *V. dahliae* Kleb.**

VERTICILLIUM WILT/ POTATO EARLY DYING

Symptoms

Haulm: Symptoms include wilting, yellowing and premature death which first appear 40–60 days after planting (**90**). Plant wilting and leaf chlorosis generally begin at the base of the plant and progress upward over time. They may be restricted to one side of a compound leaf or leaves, one side of the plant or even one half of a leaflet. Interveinal chlorosis, leaflet curling and leaf-tip necrosis (**91**) may occur, and a browning of the vascular tissue (**92**) of stems can usually be observed. Premature plant death occurs and scattered diseased patches are found in potato fields.

Roots: Although not easily or frequently observed, discoloured vascular tissues can occur.

Stolons: Diseased stolons typically have a brown to dark brown discoloration of the vascular ring.

90 Wilting and senescence symptoms of Verticillium wilt.

91 Leaflet curling and leaf-tip necrosis.

92 Browning of the vascular tissue (left) as a result of *Verticillium* infection.

Tubers: Potato tubers do not always exhibit disease symptoms despite the presence of the pathogen but when visible, the symptoms include a brown to dark brown discoloration of the vascular system (**93**). The discoloration is often first seen at the stolon end of the tuber and in the vascular ring of the whole tuber; in severe cases, discoloured blotches or cavities can develop in the tuber pith. These disease symptoms can sometimes appear similar to those caused by vine desiccants, *Potato leaf roll virus* and cold temperature injury.

HW (Bud) Platt

93 Vascular symptoms of *Verticillium* infection.

Status of the disease

Two fungi, *V. albo-atrum* and *V. dahliae,* cause Verticillium wilt. The pathogen is able to infect more than 50 species of plant and occurs in all potato production areas. The disease results in premature plant death and reductions in tuber yields. When the pathogen is present in tubers, it causes losses in their seed quality. In processed or non-processed potatoes for consumption, there is discoloration of the tuber tissues. Most commercial potato cultivars are susceptible to the disease, with recorded disease incidences of 10–80% resulting in 5–30% tuber yield losses and 5–25% tuber quality losses.

Life cycle and biology

Colourless, vegetative hyphae produce condiophores, arranged in whorls, upon which numerous conidia (3–6 × 2–3 µm) are formed. Conidia of *V. albo-atrum* are slightly larger than those of *V. dahliae*, and both are short lived and susceptible to drying. Dark mycelium (melanized hyphae) of *V. albo-atrum* and microsclerotia (30–60 µm across) of *V. dahliae* enable the fungi to

survive for months in unfavourable conditions in potato stems and tubers, and for years in the soil. Soil type, temperature, microbial activity and other factors greatly affect survival of these structures, which germinate to produce mycelium and then conidia which can be carried by equipment, wind, and water from field to field. Disease spread can also occur by root contact. While both species are often found in field soils, one will predominate due to the effects of climate and soil conditions; *V. albo-atrum* predominates in cooler (16–22°C), more moist and heavier soil conditions, and *V. dahliae* predominates in warm (22–27°C), drier and lighter soil conditions. Although these fungi are recognized as the primary pathogens involved in potato early dying (PED), co-infections with root lesion nematodes (*Pratylenchus penetrans*) have also been implicated.

Disease occurs as a result of planting diseased seed or the presence of soil-borne inoculum. Infection is generally through root hairs, root wounds and sprouts or leaf surfaces. Hyphae invade intracellularly and intercellularly to the xylem in the vascular tissues. Fungal proliferation leads to production of conidia and substances which plug the vascular system and result in restricted water movement, wilting and death. Extensive formation of dark mycelium and microsclerotia in these diseased potato plant parts and tubers provides a means of survival until the next host crop.

Control

No single disease control method is 100% effective, and an integrated disease management approach is required. While most commercially acceptable cultivars are susceptible, use of resistant cultivars (e.g. Desiree, Ranger Russet, Reddale) is important although most resistant hosts often succumb to high inoculum levels. In addition, some cultivars may be resistant to only one of the pathogens. Chemical seed treatments (e.g. benomyl, captan, mancozeb, thiophanate-methyl) and soil fumigants (e.g. 1,3-dichloropropene, metham sodium) have been used to reduce disease. However, the use of these is restricted in some countries. Currently available fungicides provide disease protection but not curative activity.

Disease management strategies that reduce initial inoculum levels in soils are the most reliable over the long term. In some locations, reduction in soil-borne populations of *Verticillium* spp. and root lesion nematodes are required to lessen damage due to PED syndrome. The use of healthy seed provides the first step in reducing inoculum sources of the pathogens, which involves selection or testing of seed to eliminate contaminated (pathogen in soil on tuber) and infected (pathogen inside) seed tubers. Fields with a history of disease, or with soil test levels of large numbers of pathogen propagules, should be avoided. Crop rotation is very important for soil-borne inoculum level reduction and disease management. In most areas, potatoes or other host crops should only be grown once every three or four years or more, and groundkeepers eliminated. Soil-borne inoculum levels are reduced whennon-host crops, such as grasses, cereals, or legumes, are grown. In addition, fields must be managed to eliminate the occurrence of susceptible weed species including *Capsella bursa-pastoris, Chenopodium album, Equisetum arvense*, and *Taraxacum* spp. As host crops harbour large quantities of 'overwintering' inoculum, the disposal of crop debris by removal from the field and composting or burning will reduce soil-borne inoculum levels.

H W (Bud) Platt

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Virus and phytoplasma diseases

There are over 50 viruses to which *Solanum tuberosum* is susceptible. Eight of the most important are covered in this book; the rest are, fortunately, only of academic interest or limited in their distribution and significance. Viruses are sub-microscopic particles comprising a core of nucleic acid within a protein coat. They infect other organisms by invading host cells and using the cells' genetic material to make copies of themselves. Viruses, and the even smaller viroids, require specialized detection methods such as ELISA or molecular techniques. Viruses are spread either by contact or by insects and other vectors. Control relies on controlling vectors, limiting sources of virus, and utilizing host resistance.

Phytoplasmas cause virus-like symptoms in plants, and for many years such problems were attributed to viruses. However, in the 1960s, phytoplasmas were discovered in phloem tissues. They are actually related to bacteria, although they lack cell walls, and are curently placed in the class Mollicutes. Viroids also cause virus-like symptoms and, like viruses, hijack a cell's mechanisms to replicate themselves, but, lacking a protein coat, are a quite different entity.

There is a range of symptoms that viruses produce in potato haulm; some are characteristic of a particular virus, but many are general symptoms that may be ascribed to several different viruses. Thus in certification schemes, viruses are often visually classified in general terms such as 'mild mosaic' (**94**) or 'severe mosaic' (**95**) without knowing with certainty which virus is causing the symptom until further diagnostic tests are applied. To add confusion to diagnosis, different cultivars when infected by the same virus may exhibit different symptoms. In addition, where mixed virus infections occur, symptoms may be very different from those exhibited by either virus singly (**96, 97**). Most viruses associated with potatoes are described in the CAB descriptions of viruses (see reference list) and at various websites.

Although potato viruses rarely kill the plants they infect, they can be devastating in their impact on yield and quality and are considered a major cause of 'degeneration' of the potato crop. Since potatoes are vegetatively multiplied and most viruses can be transmitted to progeny tubers, the proportion of plants infected increases as a seed stock is multiplied. Seed production and certification schemes have been devised to ensure that foundation seed is free from viruses and, as far as possible, seed is kept free during multiplication. It is possible to eliminate virus from a stock by meristem tip culture, with or without heat therapy, and healthy foundation seed is produced in this way. In most seed producing countries, healthy cultures of potato varieties are maintained *in vitro* and from these new seed stocks are generated either as virus-tested stem cuttings or mini-tubers.

94 Mild mosaic symptoms.

95 Severe mosaic symptoms.

96 Mixed infections by *Potato virus X* and *Y*.

97 Mixed infections by *Potato virus X* and *Y*.

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Aster yellows phytoplasma

ASTER YELLOWS

Symptoms

Haulm: Symptoms generally appear after mid-season and may resemble Fusarium or Verticillium wilts. Initially, the basal region of upper leaves rolls upward and the leaves often become distinctly purple, red, or yellow in colour (**98**). The rolling gradually spreads to leaves lower down the plant and these may become flaccid and wilted (**99**). New leaves fail to enlarge normally, and apical dominance is lost causing aerial tubers and/or shoots with swollen bases to grow from axillary buds. Lower stems may develop cortical necrosis and vascular discoloration. Some stems on plants may remain uninfected and symptomless.

Roots: Roots on infected plants do not produce symptoms.

Stolons: Aster yellows symptoms have not been described in stolons.

Tubers: Infected tubers are small, malformed, and sometimes soft and spongy. Infected tubers often fail to sprout but if they do, sprouts are long and spindly. If infected tubers are planted they either fail to establish a new plant or produce weak, spindly plants.

Status of the disease

The causal agent of aster yellows is a phytoplasma. It is not known whether other similar diseases described as stolbur, purple top, or marginal flavescence are in fact caused by the same or related phytoplasmas. Symptomology and epidemiological characteristics of these diseases overlap.

If infection levels are high, disease losses can be considerable. However, the phytoplasmas are transmitted to potato by leafhoppers from nearby weeds, and the efficiency of transmission is low. The disease is not propagated via seed because infected potatoes used for seed generally do not produce plants, or if they do they emerge late, are weak and stunted, and do not produce progeny tubers. Spread from infected tubers is not known to occur. Aster yellows and allied diseases are a significant problem in potato, mainly in areas which have relatively warm climates during the growing season – which favours the leafhopper vectors.

98 Rolling of upper leaflets and discoloration.

99 Symptoms spread to lower leaves, which become flaccid and wilting.

Life cycle and biology

The phytoplasmas are transmitted by several different species of leafhoppers and perhaps some other insects. In North America, the primary vector is *Macrosteles quadrilineatus*, the aster leafhopper. The potato leafhopper reproduces on potato but is not a vector. Weeds and other host crops are important reservoirs of the pathogen. Leafhoppers need to feed for several hours on infected plants in order to acquire the phytoplasma, and an incubation period of about two weeks

is required before the insects become 'phytoplasma-liferous'. Potatoes are not a preferred host for leafhoppers, and although they will feed on potato they do not breed on these plants.

Control

In geographic regions where the disease is a problem, control consists largely of suppressing weeds which may serve as a reservoir for the phytoplasma and control of leafhopper vectors. In arid regions, planting and harvesting may need to coincide with periods of other vegetative growth so leafhoppers are not attracted to potato as the only green vegetation available. Since infected tubers do not sprout adequately to produce progeny, the disease is not tuber borne.

Solke H De Boer

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Potato leaf roll virus

POTATO LEAF ROLL

Symptoms

Haulm: Primary infection, arising from infection in the growing season, appears in the youngest leaves and mostly results in a pale discoloration and in-rolling of leaflets starting at the leaflet base. Some purple discoloration of affected leaflets may occur. Primary symptoms tend to occur only where infection of the plant occurs early in crop development or in hot climates. Secondary infection, where symptoms develop from infected tubers, is always more severe. Inward rolling of lower leaflets (**100**), extending ultimately to the upper leaves, is typical. The leaves become dry and brittle, and if touched the plant makes a characteristic rustling noise. Leaves are chlorotic and often show purple discoloration. Once symptoms appear, a necrosis of phloem tissue in the haulm is characteristic of the virus infection in the field. *Potato leaf roll virus* (PLRV) infected plants are usually stunted and erect (**101**) and produce normal-shaped, but small, tubers.

Roots and Stolons: No distinct symptoms are normally visible.

100 In-rolling of lower leaflets as a result of *Potato leaf roll virus* infection.

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101 Stunted and erect plant infected by *Potato leaf roll virus*.

SASA © Crown copyright

102 Net necrosis of vascular tissue of tubers infected by *Potato leaf roll virus*.

Tubers: Susceptible cultivars may produce a necrosis of the phloem tissue of tubers. This is a network of brown necrotic tissue extending through the vascular tissue of the tuber and is called net necrosis (**102**). Net necrosis may not be apparent at harvest but can develop in store. It can develop in tubers from plants showing both primary and secondary haulm infection.

Status of the disease

PLRV is found wherever potatoes are grown. It is primarily found in members of the Solanaceae. Single infected plants can have their yield reduced by 50% or more, although neighbouring uninfected plants will compensate. However, where widespread infection is present, severe yield reduction will occur. PLRV is probably the most important potato virus worldwide. One estimate has suggested the virus is responsible for 20 million tonnes yield loss globally.

Life cycle and biology

The virus is transmitted by aphids, principally by *Myzus persicae*. It is not sap transmissible. Transmission from the aphid is much slower than for *Potato virus Y* (PVY). The virus persists in the aphid throughout its life cycle. Aphids transmit the virus in a persistent (circulative) way. All instars (stages) of the aphid can transmit the virus, but the nymph stage is more efficient than the adult. The extent of transmission depends on the degree of acquisition of the virus from an infected host and this, in turn, depends on the duration of feeding. Similarly, the transmission to an uninfected host depends on the amount of virus in the aphid and the duration of feeding. Several strains of the virus have been identified, based on the severity of symptom expression in an indicator species. The virus can be spread long distances by winged aphids. Virus accumulates in tubers and, if planted, the virus is transmitted to daughter tubers as well as into the foliage. Some cultivars exhibit high resistance to PLRV.

Control

The production of healthy certified seed – from virus-free clonal material initially and then multiplied in areas of low risk of virus spread – is the most important control measure. Elimination of sources of the virus by roguing seed crops and killing volunteers reduces the risk of spread. Additionally, the application of insecticides to reduce aphid numbers is also important. The extent of tuber infection declines the later in crop growth the virus is transmitted, thus restriction of virus spread early in crop growth is important. Systemic insecticide granules applied into the soil at planting can give protection over the period of early growth. However, systemic insecticides applied to the foliage to coincide with the arrival of aphids can also be effective.

Some seed-growing countries operate aphid warning schemes to alert growers to the need for insecticide. In seed crops, early haulm destruction before aphid numbers build up reduces the risk of infection. In chitting sheds aphids can spread PLRV and other viruses from tuber to tuber by feeding on sprouts, and aphid control is important in this environment. The degree of infection of tubers in a stock can be determined by a 'growing-on' indexing test or more accurately by an ELISA serological test on sap from sprouts. Because tubers have a low virus content, PCR testing is more sensitive directly from tuber tissue.

Stuart Wale

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Potato spindle tuber viroid

SPINDLE TUBER

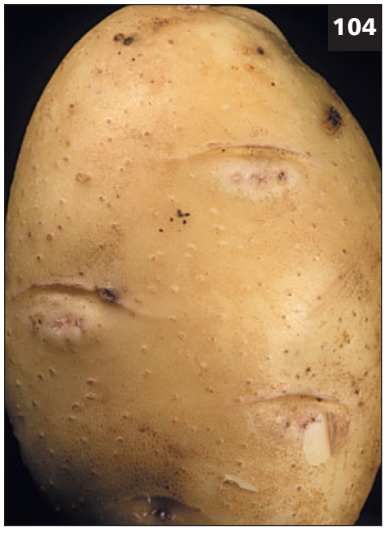
Symptoms

HAULM: Aerial symptoms develop in warmer conditions but are masked in cooler ones. Primary haulm symptoms are seldom evident in potato plants. Stem and blossom pedicels are slender, longer than normal, and remain erect. Leaflets are slightly reduced with fluted margins, tend to curve inward and overlap the terminal leaflet. Angles between stems and petioles are more acute than normal. Leaves near the ground are noticeably shorter and erect, contrasting with healthy leaves, which rest on the ground. As the season advances, diseased plants are restricted in growth (**103**) and become harder to identify because of intertwining with neighbouring healthy plants. Severe strains cause enhanced symptoms, twisting of leaflets, and rugosity of leaf surfaces. Under some light conditions, plants with *Potato spindle tuber viroid* (PSTVd) show a dull leaf surface that is less reflective of light than normal leaf surfaces.

ROOTS AND STOLONS: No distinct symptoms.

TUBERS: Tubers tend to be somewhat elongated, with pointed ends, and – in contrast to the flattened shape of normal tubers – round in cross-section. Eyes appear to be more numerous; they have characteristic indentation with 'heavy brows' or enhanced edges (**104**).

103 Stunted plant infected with *Potato spindle tuber viroid*.



104 Characteristic indentation of eyes with heavy brows of tuber infected by *Potato spindle tuber viroid*.

105 Surface cracking as a result of *Potato spindle tuber viroid* infection.

Surface cracking, usually parallel to the long axis, is frequent (**105**). Tuber symptoms become more marked as the season advances. Russet skins become smooth; red skins become pink and purple skins turn a lighter, lavender colour. Not all tubers from diseased plants show all or any of these symptoms. In contrast, tubers from healthy appearing plants grown in warm climates have all these symptoms. Therefore, symptoms are not conclusive for PSTVd infection.

Status of the disease

Spindle tuber is caused by a viroid. Viroids are the smallest-known infectious agents causing diseases in higher plants. They consist of small (241–399 nucleotides), single-stranded circular RNAs. Potato spindle tuber was the first member identified in 1971 as 'viroid'. PSTVd has been reported mainly from the USA, Canada and Russia. However, in the last 20 years it has not been observed in North America. Under field conditions, mild strains with indistinct symptoms outnumber severe strains by a ratio of 10:1 and cause yield losses of 15–25%, whereas severe strains with distinct symptoms cause 65% yield loss. PSTVd is probably limited to untested wild potato collections and potato breeding lines in different countries.

Life cycle and biology

The viroid is highly contagious and readily transmitted to plants by contaminated cultivating and seed-cutting tools. There are some reports of transmission by chewing and sucking insects, which are probably due to physical contamination of insects by the viroid. The viroid is transmitted through pollen and true potato seed; therefore, breeding and release of new cultivars can be one of the sources of its introduction to fields. Plants infected in the current season are difficult to diagnose, thus, diseased plants could multiply for several years before being diagnosed. Laboratory testing of viroids by return polyacrylamide gel electrophoresis (R-PAGE); nucleic acid spot hybridization (NASH); or reverse transcription polymerase chain reaction (RT-PCR) can be used to detect the viroid infection. Several viroids infecting horticultural and ornamental crops, wild *Solanum* species and tomato plants can infect potato plants and cause PSTVd-like symptoms.

Control

Strict adherence to potato seed certification is necessary to control PSTVd. Certification has been very effective in reducing PSTVd infection. Use of seed tubers known to be free from PSTVd, avoidance of contaminated machinery for planting, and decontamination of knives and other equipment with disinfectants such as sodium hypochlorite, should be practised to control the outbreak of the disease. Since insect vectors do not transmit PSTVd, PSTVd-free nuclear stocks remain free from PSTVd under field conditions.

Rudra Singh

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Potato virus A

POTATO VIRUS A

Symptoms

Haulm: *Potato virus A* (PVA) causes mild mosaic symptoms not dissimilar to those caused by *Potato virus X* (PVX) (**106**). Differences can be difficult to detect visually, but PVA mottles may appear on the veins, and infected leaves look shiny. Infected plants may have a more open habit. Although visually similar to PVX, this virus is related to *Potato virus Y* (PVY) and is spread by aphids.

Roots, Stolons and Tubers: Typical symptoms are not normally visible.

Status of the disease

PVA is confined to members of the Solanaceae. Widespread infection of a crop can cause up to 40% yield loss. The virus is found wherever potatoes are grown. PVA is considered less important that PVY.

106 Mild mosaic symptoms in a plant affected by *Potato virus A*.

Life cycle and biology

At least seven aphid species are capable of transmitting PVA (including *Aphis frangulae, Macrosiphum euphorbiae,* and *Myzus persicae*). The virus is non-persistent and is lost from aphids as they go through their life cycle. As with PVY, the virus can be acquired rapidly from an infected plant (<1 minute) and transmitted equally rapidly. Some cultivars are resistant to PVA and others show field resistance by hypersensitivity. In general, cultivars resistant to PVY are also resistant to PVA.

Control

Control measures are similar to those for PVY; planting certified seed free from virus, elimination of sources of virus by roguing seed crops and destroying volunteers, early harvest of seed crops and the use of foliar applied insecticides with a repellent action. Detection and separation from PVY can be achieved serologically using ELISA.

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Potato virus X

POTATO VIRUS X

Symptoms

Haulm: *Potato virus X* (PVX) is one of the potato viruses that cause mosaic symptoms. Visually, symptoms range from absent through a faint or fleeting mottle to a severe necrotic streak. The light conditions can affect symptom detection, with low-light conditions making them more apparent. When symptoms are expressed, there is a pattern of light and dark green on leaflets; the lighter, small, irregular blotches being between the veins. Only occasionally does leaf distortion, rugosity, necrotic spotting or stunting occur. More severe symptoms can occur when PVX is present with other viruses. In combination with PVA or PVY it causes leaf distortion and crinkle.

Roots, Stolons and Tubers: Typical symptoms are not normally visible.

Status of the disease

PVX is found wherever potatoes are grown. In nature it is largely confined to members of the family Solanaceae. When occasional plants are infected, yield loss is negligible. With more widespread infection, yield losses of 15–20% can occur. However, through effective control, in many countries the virus has become of limited importance.

Life cycle and biology

Depending on how they are determined, there are a number of different strains and variants of PVX. Some cultivars are extremely resistant to most strains – but not all. Some cultivars are field resistant, developing a hypersensitive reaction to the virus. Transmission is by contact, either plant to plant or by humans, animals or machinery passing through a crop. PVX is highly contagious, and once attached to a surface such as clothing the virus can remain infective for many hours provided the surface remains wet. Consequently, a virus picked up from an infected plant can be transmitted to many other plants when moving through a crop. The virus accumulates in tubers, and the process of cutting seed tubers can spread the virus from one tuber to another. PVX is not transmitted by true seed or by aphids. There are some reports of transmission by chewing insects, but this is probably limited. Other reports of transmission by zoospores of the wart pathogen (*Synchytrium endobioticum*) or powdery scab pathogen (*Spongospora subterranea*) have been disproved or are considered unlikely. The virus occurs in high concentration in plant tissues, and it can be readily detected serologically by precipitation when antisera are mixed with leaf or sprout sap on a slide. The extent of infection in a stock of potato tubers can be determined by testing leaves of plants grown from the tubers, but this is less effective than serological or direct PCR tuber tests.

Control

Schemes to produce virus-free foundation clones, to minimize spread during multiplication by inspection, testing when required, and certification, have been effective in minimizing the significance of this disease. After passage through a crop, machinery and clothing should be disinfected before entering a seed crop. Seed-cutting machinery should similarly be disinfected during and after cutting each stock. Resistant cultivars can be used where the disease is a major problem.

Stuart Wale

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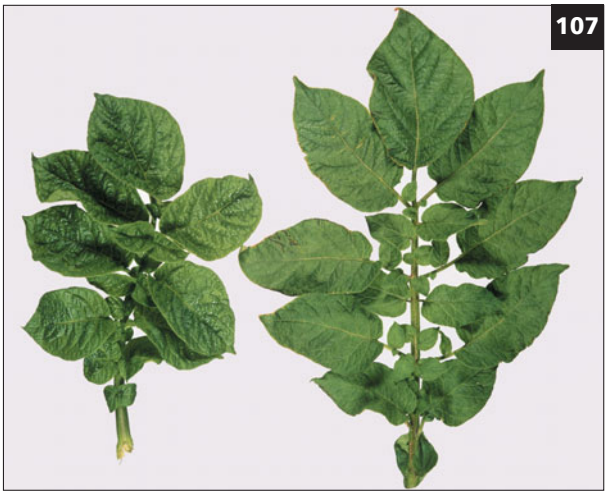
Potato virus Y

POTATO VIRUS Y

Symptoms

HAULM: *Potato virus Y* (PVY) contains strain groups which cause different symptoms in potato and other Solanaceous crops, thus varying in importance according to the crop. Primary symptoms of common strain (PVY^O) and stipple-streak strain (PVY^C) are necrosis, mottling, yellowing of leaflets (**107**), leaf drops and premature death of plants. Necrosis of foliage is a hypersensitive-type of reaction and generally starts at a veinal point on foliage and may result in spots or rings. Affected leaves may drop (leafdrop streaks) or remain clinging to the stem. These symptoms may be restricted to a few leaves or to a single shoot. Secondary symptoms result in a dwarf plant with mottled or crinkled foliage (**108, 109, 110**). Necrosis of foliage may also accompany these symptoms in certain cultivars. Some PVY^O (rugose strain) symptoms resemble those of early blight.

ROOTS AND STOLONS: Distinct symptoms are not normally seen.



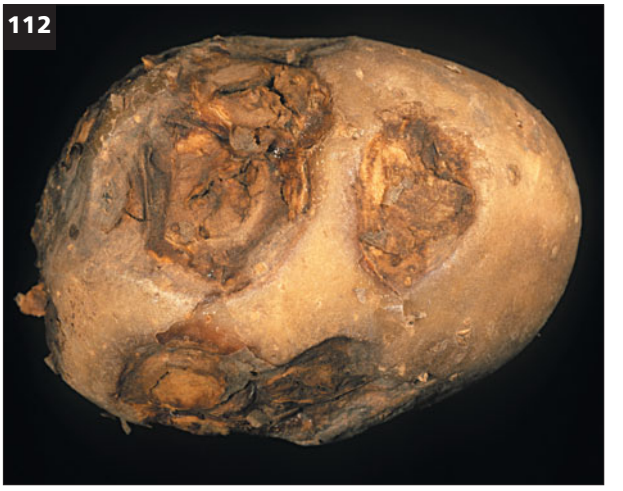
107 Comparison of healthy leaf (right) and leaf with *Potato virus Y* infection (left).

108 Severe mosaic – PVY^O symptoms.

109 Changes in leaf shape as a result of *Potato virus Y* infection (compare with 110).

110 Leaves of a healthy plant.

111 Tuber symptoms of *Potato virus Y*^{NTN}: necrotic ring spots.



112 Tuber symptoms of *Potato virus Y*^{NTN}: sunken ring spots.

Tubers: While primary symptoms of tobacco veinal necrosis strain (PVY^{N}) in potato foliage are vague and lack tuber symptoms, there are strains that cause tuber ring spot necrosis (PVY^{NTN}) (**111, 112**). The PVY^{NTN} strains cause mild mosaic to brightly chlorotic foliage and clearly visible raised necrotic ring spots on the tuber surface. The rings may remain isolated or coalesce together to cover the whole tuber. Under the surface of necrotic rings the tissues may be thick in contrast to adjacent watery flesh. Rings may become sunken and skin cracked. Necrosis often increases during storage.

Status of the disease

PVY^{O} occurrence is worldwide. However, PVY^{N} and PVY^{NTN} strains are limited to certain countries; therefore, these are of quarantine significance. PVY^{O} is one of the most damaging potato viruses in terms of yield loss. In combination with *Potato virus X*, it causes an even more destructive disease known as rugose mosaic. Besides potatoes, PVY is damaging to pepper, tomato and tobacco crops.

Life cycle and biology

The main sources of PVY inoculum are infected seed tubers. Aphids feeding on plants emerging from infected tubers acquire PVY within a few seconds and also inoculate the virus to healthy plants within seconds. Thus, aphids probing on potato plant are potential vectors of PVY. The peach potato aphid, *Myzus persicae* (see page 118) is the most efficient vector in many areas. This type of virus spread cannot be controlled effectively by the use of insecticides, because no pesticide can kill the aphids rapidly enough to prevent infection. Where insecticides are used, they tend to act as deterrents to aphids landing on a crop. Virus spread mainly takes place by winged aphids. In most areas infected tubers are the initial source of virus, although in tropical countries Solanaceous weeds may also serve as a reservoir of virus for vectors.

Control

In most countries, seed tubers are multiplied from virus-free nuclear material in areas with a low risk of virus spread under a seed certification programme. However, depending on the cultivar, seed tubers can be subjected to a post-harvest virus test to ensure a low level of infection. Some cultivars are practically symptomless when infected by PVY. Modern methods of reverse transcription polymerase chain reaction (RT-PCR) are available which can detect PVY in dormant tubers soon after harvest. RT-PCR primers which can differentiate various strains of PVY or other potyviruses are also available. Elimination of sources of virus by roguing diseased plants reduces its spread. However, this should be done early in the growing season. In areas where virus inoculum and aphid pressure are high, oil sprays prior to the arrival of aphids can be helpful. The spread of non-persistently transmitted virus like PVY cannot be reduced by controlling aphids with insecticides. In fact, insecticide sprays stimulate aphids to probe more profusely, which may result in more virus spread.

Rudra Singh

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Potato yellow vein virus

POTATO YELLOW VEIN

Symptoms

Haulm: Primary infection: first symptoms caused by *Potato yellow vein virus* (PYVV) appear 10–15 days after infection as a bright yellowing of small (tertiary) leaf veins. Sometimes only a few yellow spots have been observed in the leaf lamina (**113**). As the plants develop, secondary veins become affected. Later, leaf lamina between veins also become yellow. PYVV rarely affects the main (primary) veins, which may remain green until the plant dies. Secondary infection: symptom development in secondarily infected plants begins in the same way as in primarily infected plants. When symptoms appear early, the whole plant can become affected. The foliage of some infected plants exhibits a whitish coloration instead of the bright yellowing commonly associated with potato yellow vein disease. Some plants derived from tubers of infected plants are asymptomatic and the progeny of these individuals may develop both symptomatic and asymptomatic plants.

Roots and Stolons: Distinct symptoms are not seen.

Tubers: Except for a reduction in number and size, tubers collected from PYVV-infected plants appear identical to those from healthy plants.

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113 Symptoms of *Potato yellow vein virus*.

Status of the disease

At present, PYVV occurrence is circumscribed to South America (Colombia, Ecuador, Venezuela and Peru). Sporadic outbreaks of the disease were first reported in Antioquia-Colombia in the early 1940s by potato growers and rapidly reached a high incidence and caused severe yield reductions. It seems that PYVV began to spread from Colombia and Ecuador to neighbouring countries due to informal trade in potato seed that initially provided the major pathway for virus dissemination. Recently, increase of its whitefly vector favoured it spreading to new potato growing areas in South America. PYVV was also reported to have spread from South American potatoes to nuclear potato stocks growing nearby in the same greenhouse in Cambridge, England, in the 1970s. Several researchers have determined the effect of PYVV on yield, and all studies report yield reductions of about 50% in plots where all plants were infected. PYVV is considered a quarantine pathogen and a potential threat to world potato production.

Life cycle and biology

PYVV is a whitefly-transmitted potato pathogen and a proposed member of the genus *Crinivirus*, family Closteroviridae. Like most other closteroviruses, the virus does not appear to be mechanically transmissible. The main sources of PYVV inoculum are infected seed tubers and weeds. Plants emerging from infected tubers serve as a primary virus source for *Trialeurodes vaporariorum* Westwood, its natural vector. The virus can also infect and survive in weeds that play an important role in virus spread and establishment. Whiteflies acquire PYVV within a few hours and also inoculate the virus to healthy potato plants within hours. The inability of aphids (*Myzus persicae* and *Macrosiphum euphorbiae*) and mites (*Eutetranychus telarius* and *Faratomus yusti*) to transmit PYVV has been demonstrated. The abuse in use of pesticides has contributed to increased vector populations and resistance to the pesticides used.

Control

Use certified seed or seed produced in virus-free areas and eliminate infected plants by roguing early in the growing season to reduce spread of the virus. Currently, there is a reliable and sensitive method for PYVV diagnosis by NASH technique based on its HSP-70 and CP-genes. So far, no resistance to this virus has been found in the cultivated potato.

Giovanna Müller

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Potato mop-top virus (PMTV)
Tobacco rattle virus (TRV)

SPRAING

Symptoms

HAULM: Plant stunting, leaf yellowing, and other symptoms (see below) can be seen.

ROOTS AND STOLONS: No distinct symptoms are normally visible.

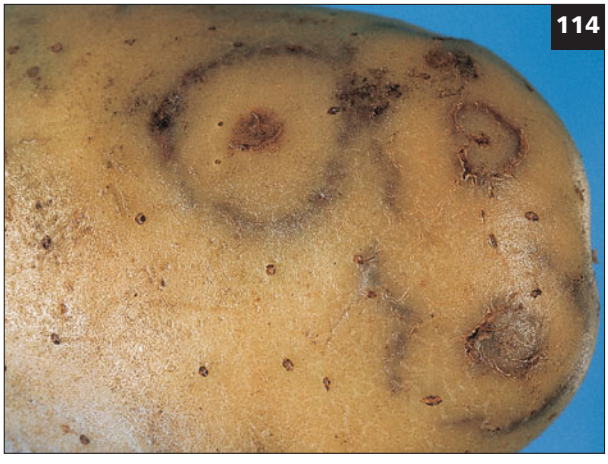
TUBERS: Both viruses cause a symptom called 'spraing' in tubers. Symptom expression varies greatly and is affected by cultivar and environmental conditions. Cultivars may be classified into three types:

- Resistant – usually no symptoms or virus present.
- Sensitive – symptoms develop as a hypersensitive reaction but virus is present at low concentrations.
- Tolerant – no or slight symptoms develop but the virus is present.

Primary infection: tubers

PMTV: On the surface, dark lines and rings can be seen, which are often slightly raised (**114, 115**). Internally there are arcs, lines or brown flecks visible (**116**). Tuber symptoms can develop after a sudden drop in temperature.

TRV: Arcs, lines or flecks are visible in the tuber flesh but they are not always visible on the tuber surface. The symptoms include a layer of corky tissue (**117**).



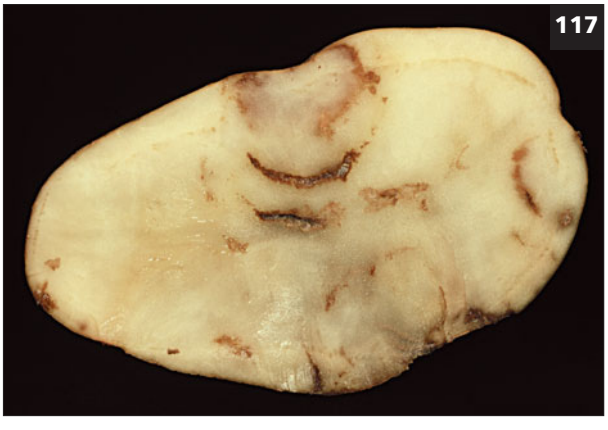
Stuart Wale

114 External tuber symptoms of *Potato mop-top virus*.

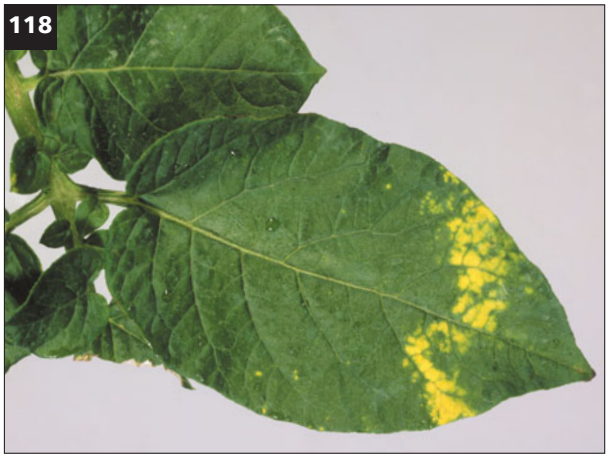
115 External symptoms of *Potato mop-top virus* around a powdery scab lesion.

Stuart Wale

116 Internal symptoms of *Potato mop-top virus*.



117 Internal symptoms of spraing caused by *Tobacco rattle virus*.



118 Yellow chevrons of the foliage symptom of secondary *Potato mop-top virus*.

119 Cracking and malformation of tubers as a result of secondary *Potato mop-top virus* infection.

120 Brown, red or yellow margins and tips to leaflets as a result of secondary *Tobacco rattle virus* infection.

Secondary infection: foliage and tubers

PMTV: In certain cultivars, yellow chevrons or aucuba symptoms develop on the leaves (**118**). Cool temperatures or fluctuations in temperature favour foliage symptoms. In plants grown from infected tubers there may be shortening of internodes or stunting and typical mop-top (bunching of leaves at the top of the plant) from which the virus name is derived. Secondary infection in tubers is manifested as cracking, malformation and surface blotches (**119**).

TRV: Plants grown from tubers showing spraing symptoms may develop a 'stem' mottle on one or two stems per plant. These stems grow less vigorously and have mottled leaves. A portion of tubers produced from affected plants may have spraing symptoms. Plants grown from symptomless tubers of tolerant cultivars can show delayed emergence, stunting and leaf curling. Later in the season, brown, red or yellow marks appear near the tips and edges of leaves (**120**). Progeny from infected plants are smaller and misshapen with yield being affected.

Confusing symptoms

- Internal rust spot can be confused with both types of spraing.
- PVY^{NTN} can be confused with primary symptoms of PMTV.
- Foliage symptoms of TRV may be confused with wind damage or chemical scorch.
- Yellow chevrons of PMTV on foliage are diagnostic.
- The presence or absence of powdery scab is not indicative of PMTV or TRV.

Status of the disease

Spraing in the tubers has a direct effect on quality for both ware and processing markets and can lead to rejection of a stock. Secondary infection can affect both yield and quality. Some countries impose a tolerance for imported seed. The disease is sporadic in occurrence and affected greatly by seasonal conditions. Thus the significance of the disease is difficult to determine in financial terms. However, rejection of stocks can cause considerable financial loss to individual growers.

Life cycle and biology

PMTV: The virus is transmitted by *Spongospora subterranea* (see Powdery Scab), which persists for many years as resting spores in soil, and thus the potential for PMTV transmission is believed to also persist. Infection of roots and tubers is favoured by high soil water content, and PMTV can occur in any soil type. Weeds are thought to be relatively unimportant reservoirs of the virus. Transmission of the virus from an infected mother tuber to foliage is limited, and not all progeny tubers are infected. Thus PMTV is gradually self eliminating from potato stocks if grown in virus-free fields. Little is known about the conditions that favour virus propagation. PMTV can be established in a new location by planting infected tubers, but seed tubers carrying sporeballs of *S. subterranea* containing PMTV are probably the main cause of transmission.

TRV: The vectors of TRV are species of *Paratrichodorus* and *Trichodorus* nematodes (see Stubby Root Nematodes). There are a number of serotypes of TRV (currently 10 have been identified, but there are probably more) and each is transmitted by just one, or a few, species of nematode. An infected nematode can transmit the virus to several plants in series, and non-feeding nematodes retain the virus for several months. However, the virus is not retained through moulting or passed through eggs. A shallow topsoil, high soil water content and an optimum soil temperature of 15–20°C favour vector activity.

TRV has the widest-known range of any plant virus, occurring in wild plants and weeds. The potato is unimportant for the survival of the virus, and the interval between crops is probably irrelevant. TRV is seed-borne in some weed species. Virus isolates can differ in their effect on different cultivars and the ease with which they are transmitted from mother to daughter tubers. However, in TRV-susceptible cultivars where symptoms occur, infection is usually self-eliminating, but the milder the spraing symptoms the greater the level of transmission. In tolerant cultivars, all daughter tubers may be infected. Infected tubers can be sources for virus acquisition by nematodes, but those with spraing symptoms are usually not. For a strain of TRV to be introduced to a new site by planting infected seed tubers, the corresponding vector species must be present.

Control

Breeding for resistance to either virus is a low priority for plant breeders. However, resistance to PMTV and TRV is known, and through genetic modification it may be introduced into future cultivars.

PMTV: Measures to control powdery scab should reduce the risk of PMTV (see Powdery Scab). In seed multiplication, removing plants displaying foliage symptoms should hasten self-elimination of the virus provided the field is virus free. Planting uninfected tubers and avoiding tubers carrying resting spores of the vector will reduce spread of the virus. Commercial testing of tubers for PMTV is available in some countries.

TRV: Effective weed control practised through the rotation may reduce virus levels in a field. Barley and wheat are hosts for the nematode but not for the virus. Thus several years of growing cereals between potato crops with good weed control can reduce the virus but not the vector. Plants are probably most sensitive to infection at tuber initiation, and irrigation in the early stages of crop growth can help nematode motility. Avoiding excessive irrigation at this time will help.

Assessing free-living nematode populations in soil is of little help without knowing the level of virus infection. Determination of TRV in free-living nematodes extracted from soil is available commercially in the UK. Knowledge of prior occurrence of TRV-spraing in a field, and of the cultivars grown, can be used to select a resistant cultivar. It can also determine whether there is a need for nematicides or nematistats and whether to avoid growing potatoes in the field. Where no TRV-spraing occurred in a field, there is less likelihood of disease if the same cultivar is grown. DNA tests to detect TRV in tubers are available commercially in some countries, and it is possible to avoid planting infected but symptomless tubers.

Stuart Wale

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Tomato spotted wilt virus

TOMATO SPOTTED WILT

Symptoms

Symptoms vary with cultivar, strain of the virus and time of infection in relation to crop growth.

HAULM: Primary infection shows as necrotic spots or rings on youngest leaflets (**121**). The spots may have concentric zonation and may be confused with early blight. Before leaf symptoms occur, the foliage may become paler. Brown necrotic lesions develop on petioles and stems and within the stem. Death of one or more stem tops may occur or, in extreme cases, the whole plant may die (**122**). In secondary infection, plants arising from infected tubers are stunted and exhibit bunching of leaves. The leaflets are coarse and may desiccate and turn brown. Early death may occur.

ROOTS AND STOLONS**:** Root and stolon symptoms do not normally occur.

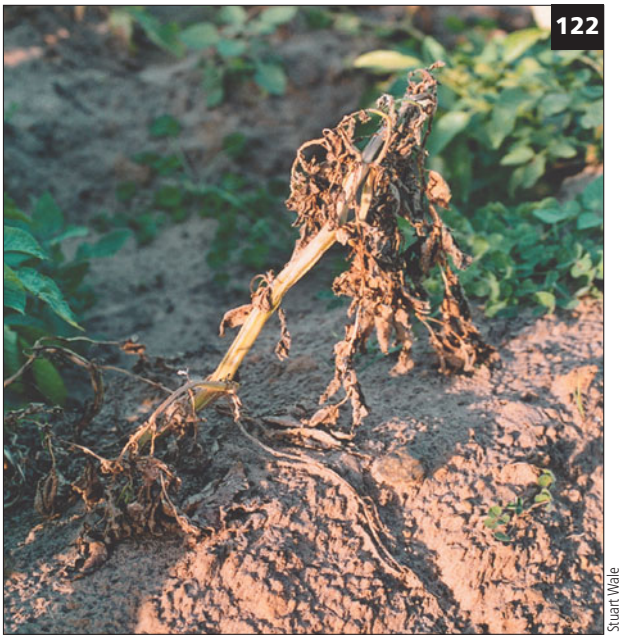
TUBERS: Infected plants may appear normal or may show distortion or cracking but are usually small in size. Sunken black spots may develop on the surface in susceptible cultivars. Internally, symptoms may range from flecking through dark shadowing to severe dark necrosis (**123**).

Status of the disease

Tomato spotted wilt virus (TSWV) can cause disease and serious losses in over 70 plant families and over 1000 species, including both monocotyledons and dicotyledons. Many weeds may act as reservoirs of the virus. The virus was originally a major problem in tropical and sub-tropical regions, but through movement of virus-infected plant material and the western flower thrips vector it now occurs virtually worldwide. Potatoes are less severely damaged by *Tomato spotted wilt virus* than other food crops, and outbreaks in potatoes are sporadic and often localized. Outbreaks are frequently related to the spread of vectors carrying the virus from other infected hosts. Infected plants produce either no tubers or a very low yield. Neighbouring plants often compensate for this lost yield. Tubers with symptoms are unmarketable, and their presence in seed crops can result in failed certification. However, symptoms are rarely present in more than 5% of tubers.

Stuart Wale

121 Foliage symptoms of T*omato spotted wilt virus*.



Stuart Wale

122 Death of *Tomato spotted wilt virus* infected plant.

Life cycle and biology

The vectors of *Tomato spotted wilt virus* are species of thrips, including *Thrips palmi*, *Thrips setosus*, *Thrips tabaci*, *Frankliniella fusca*, *Frankliniella occidentalis*, and *Frankliniella schultzei*. The vector of most importance varies according to location. Thrips are very small (<2 mm), just visible with the naked eye. The virus is acquired by the larval stages during feeding, after eggs are laid on an infected host. Only a short period of feeding is required for virus acquisition and larvae are infective after an incubation period of 3–10 days. Adults transmit the virus during feeding and, since they are winged and can be carried long distances on the wind, are responsible for virus spread. Spread generally occurs in late spring or early summer, and a high thrip population is required for significant disease to develop. Adults do not pass the virus to their progeny but overlapping stages of the life cycle can provide continuous virus spread. Conditions that favour thrip population increase and spread depend on the thrip species involved. For example, in Australia, warm, moist conditions are ideal, but in other countries dry conditions favour the vector. Under varying conditions, thrips may spread in infection waves.

The virus can be spread by planting infected seed tubers. There is no evidence that seed-cutting tools spread the virus.

Stuart Wale

123 Severe internal necrosis in potato tubers.

Control

Early crops can escape disease by passing the susceptible young stage before virus-containing vectors arrive. Siting potato crops in locations away from virus reservoirs, such as other susceptible crops, may permit disease escape. Controlling weeds within and around a crop reduces a potential virus reservoir and hosts for thrips to survive on. The use of granular persistent and systemic insecticides or foliar insecticides may reduce vector numbers. These can be applied to the potato crop or border catch crops. Insecticides are only

effective when thrips are actively feeding as larvae or adults. Sticky traps have been used to monitor thrip movement into crops. Cultivars differ in their susceptibility to *Tomato spotted wilt virus*, and resistant cultivars should be grown where there is a high risk of infection.

Stuart Wale

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Witches' broom phytoplasma

WITCHES' BROOM

Symptoms

Haulm: Primary infection results in an erect growth habit, rolling of leaflets and chlorosis – particularly at margins – and may resemble virus diseases. Secondary infection (that is, plants grown from infected tubers) has more severe and characteristic symptoms. Stems and petioles on affected plants are round and smooth, and leaves may be simple rather than compound. Plants are dwarf and exhibit a bushy appearance due to a proliferation of slender, spindly shoots that have an upright growth habit and extensive branching. Flower and seed berry formation may be increased, but phyllody of flowers may develop.

Roots: Symptoms of witches' broom have not been described on roots.

Stolons: Stolons may elongate abnormally and bear many small tubers either singly or in chains.

Tubers: Primary infected tubers appear normal but have a shortened dormancy period and give rise to dwarf plants. Virtually all eyes of infected tubers sprout, but sprouts are spindly or hair-like and often branched. Tubers produced on secondarily infected plants are small and usually do not sprout.

Status of the disease

The disease is worldwide in occurrence but of little economic importance in potato generally. It is sometimes serious as a local problem.

Life cycle and biology

The causal agent is a phytoplasma that is vectored by leafhoppers including *Scleroracus flavopictus*, *S. dasidus*, and *S. balli*. Other insects associated with witches' broom transmission include an Andean psyllid (*Russelliana solanicola*). Leafhoppers do not acquire the phytoplasma from potato, so transmission to neighbouring plants or other hosts is not possible. Important reservoirs of the disease include legumes such as alfalfa or lucerne, clover and birds-foot trefoil (*Medicago sativa*, *Trifolium repens*, *Trifolium pratense*, *Melitlotus alba* and *Lotus corniculatus*). These plant species are also the preferred hosts for leafhoppers.

Control

The disease is largely self-eliminating, because infected tubers either do not sprout or produce weak, unproductive plants.

Solke H De Boer

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Nematodes

Sometimes known as roundworms or eelworms, these small, often microscopic, animals are found ubiquitously: some are plant or animal parasties, others are free-living in the soil or water. Plant-parasitic nematodes are characterized by the hollow needle-like structure – stylet – with which they penetrate the plant's cell walls. Many crops are prone to attack by nematodes and the potato is no exception.

There are several groups of nematode which affect potato, and these are categorized according to their habits. There are nematodes that penetrate the root (endoparasites) and those that live externally in the soil or roots, feeding on the outside surfaces (ectoparasites). The former group includes root-knot nematodes, potato cyst nematodes, root lesion nematodes and the potato rot nematodes, while the latter group contains stubby-root nematodes. Within each group there may be more than one genus or species involved. Most potato nematodes are active below soil level but some may also be found on above-ground parts. Similarly, some nematodes are very host specific and some are not. Potato-pathogenic nematodes can be identified by microscopic examination. Their size and structure, particularly those of the stylet, are used for identification.

The life cycles of nematodes can be complex and vary between species, and this affects key characteristics such as hatching dates, with resulting influences on potential control measures. Nematodes can be very damaging to potatoes, affecting both the yield and the quality of tubers. When attacked severely, crop growth is reduced and potential production limited. Damage to tubers can impact on marketability. Damage is often related to population size, so control measures are often targeted at this. Restrictions can be severe in relation to seed potatoes, as transport of nematodes to otherwise uninfested areas is undesirable.

Ditylenchus destructor (Thorne)

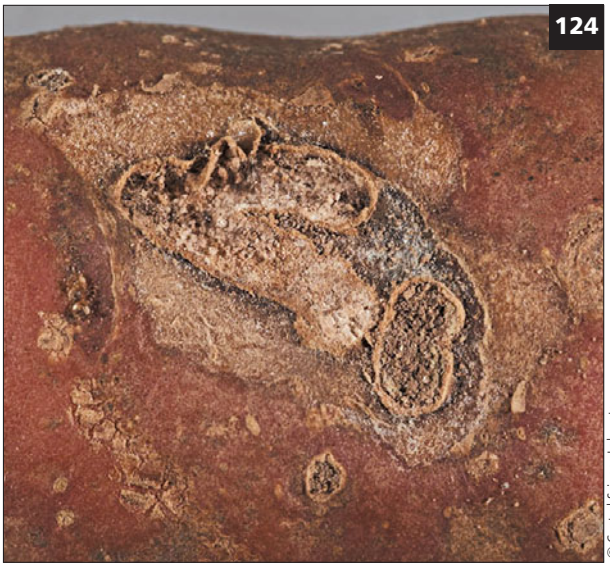
POTATO TUBER NEMATODES/ POTATO ROT NEMATODES

Symptoms

HAULM: No distinct symptoms, but heavily infested tubers may give rise to weak stems.

ROOTS AND STOLONS: No distinct symptoms are normally seen.

TUBERS: The nematode enters through lenticels, and white mealy spots form beneath the surface. Infested areas enlarge and coalesce, and the spots become light brown lesions visible from the surface (**124**). The texture of the lesion is granular and dry. As the infestation progresses, the affected tissues desiccate and shrink. The skin becomes dry, papery and cracked. With time, infested tissues darken and are prone to secondary invasion and rotting.



124 Tuber symptoms of potato tuber nematode.

Status of the pest

Ditylenchus destructor is mainly a pest of potatoes in temperate climates and is widely distributed across Europe (including countries of the former USSR), North and South America, Australasia and also in certain parts of Africa and Asia. However, the European Plant Protection Organisation considers it a minor pest. *D. destructor* exists as host-specific races, but it has been recorded as attacking over 70 hosts.

Life cycle and biology

Optimum temperature for infestation is reported as 15–20°C, but there is evidence of adaption to differing climatic conditions and infestation may occur in a range from 5°C to over 30°C. The nematode also requires moist soils. Unlike the closely related species *D. dipsaci* (the stem nematode), *D. destructor* does not produce a structure that can resist desiccation. The nematodes may overwinter as eggs or as adults or larvae which feed on alternative weed hosts. Eggs hatch in the spring and larvae are immediately able to attack hosts.

Control

Dispersal is usually by movement of infested seed potatoes, and the primary control measure is to plant healthy seed tubers. Crop rotation may reduce soil populations but is rarely effective as the nematode has a wide host range. However, non-host crops grown with effective weed control can substantially reduce populations. It is rarely a problem in warm, dry soils. Reducing soil populations by fumigation is possible but often uneconomical. The use of soil-applied nematicides can achieve a large reduction in population but is also an expensive option. Cultivar resistance has been identified, but most commercial cultivars are susceptible.

STUART WALE

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Yellow potato cyst nematode *Globodera rostochiensis* (Wollenweber) Behrens (syn. *Heterodera rostochiensis*)
White potato cyst nematode *Globodera pallida* (Stone) Behrens (syn. *Heterodera pallida*)

POTATO CYST NEMATODES

Symptoms

Haulm: Below a critical level of attack, there are no obvious above-ground symptoms. Above the critical level, water and nutrient supply is limited and haulm growth exhibits stunting, wilting, yellowing (**125**) and death (**126**). Above-ground symptoms occur in foci.

Roots, Stolons and Stem Bases: When roots are substantially invaded they develop less well and produce more laterals, which are often stubby. The characteristic cysts (200–500 µm) can be seen clinging to the surface of roots, stolons and even tubers (**127, 128**). Separation of the species is possible when cysts are present.

Tubers: Under severe attack, tuber numbers and size are affected. Occasionally, quality is affected through pitting of the tuber surface by the nematode (**129**). When attacked by cyst nematodes, plants are more susceptible to attack by pathogens such as *Rhizoctonia solani* and *Verticillium* spp.

Status of the pest

Although originating in the Andes of South America, both potato cyst nematode (PCN) species are widely distributed around the world, being found on all continents. In 1994, the European Plant Protection Organization reported that *G. rostochiensis* was in 65 countries and *G. pallida* in 41 of these. However, occurrence in some countries is limited, and strict quarantine regulations are imposed. PCN is of most significance in temperate regions and is particularly severe in Europe. Of the two species, *G. pallida* is frequently reported as less extensive than *G. rostochiensis*, but the use of *G. rostochiensis*-resistant cultivars has led to a shift in the relative occurrence in some countries. Yield losses due to attack by PCN depend on the degree of attack. In England and Wales, where two-thirds of potato fields are infested, estimated yield losses are £50 m (about 67 million Euros or 98 US$) per annum. The annual cost of control would be around a quarter of the loss. To prevent spread and ensure seed is produced in PCN-free areas, there is an annual cost of soil testing and purchase of certified seed.

Life cycle and biology

Both cyst species have similar life cycles. Normally, there is a single life cycle for each crop. Cysts are dead remnants of the female nematode, containing hundreds of eggs per cyst. The eggs within cysts survive between crops in the soil. A proportion of the eggs hatch spontaneously in the spring over a number of months each year (**130**). Whilst the nematodes can live in the soil for several weeks, in the absence of a suitable host they die. The proportion of eggs that hatch depends on soil and environmental factors. Where a host is present, root exudates stimulate a large proportion (up to 90%) of eggs to hatch. Within the egg, the juvenile nematode moults once before hatching.

When the juveniles reach the host roots they use their stylets to pierce epidermal cells and crevices where roots emerge. They then embed themselves near the vascular tissues in the cortex. The root responds by producing a synctium or food transfer cell. At this stage, the sex of the juvenile is determined by nutritional status. Those associated with large synctia become females, otherwise they develop into males. Juveniles remain embedded until maturity and affect water and nutrient movement in the vascular system. In response to invasion, the potato produces lateral roots which, in turn, may be invaded. The juveniles swell and become flask shaped.

After a final moult within the host tissue, the males burst out into the soil. The females swell until round and burst out of the roots but remain attached. The males are attracted to the females and fertilize them. The females swell into cysts which are visible with the naked eye. Initially all cysts are white, but those *of G. rostochiensis* turn a golden yellow. Those of *G. pallida* remain white but ultimately turn deep brown. The life cycle from egg hatch to egg formation varies from 50 to 100 days depending on environmental conditions.

Control

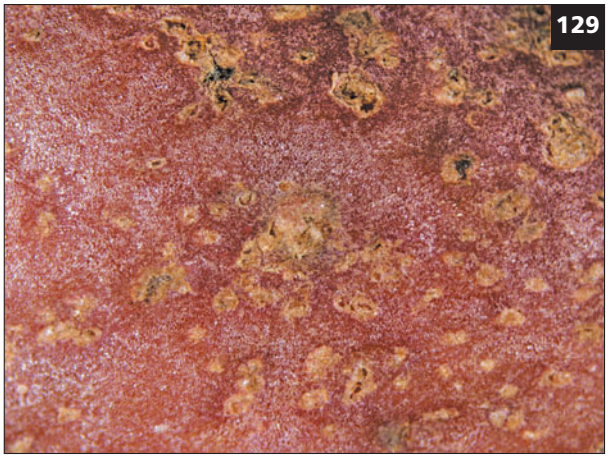
In the absence of a host the viable eggs in the soil will decline, and long rotations contribute to maintaining low populations. Natural decline in eggs has been estimated at 30% per annum but varies depending on soil factors. The interaction between host and parasite is complicated. The multiplication of the nematode in

125 Above-ground symptoms of severe potato cyst nematode attack. The crop on the right has been treated with nematicide.

126 Haulm death as a result of potato cyst nematode attack.

127 Cysts of *Globodera rostochiensis*.

128 Cysts of *Globodera pallida.*



129 Close-up of pitted tuber surface.

130 Hatch of eggs of *Globodera rostochiensis*.

the presence of a host will depend on host resistance, nematicide use and competition between nematodes. If the host is resistant, multiplication will be limited. Resistance does not decrease the rate of egg hatch but does impair the development of synctia. Pathotypes of both species exist. The most widespread pathotypes of *G. rostochiensis* are Ro1 and Ro4, and host resistance to both is conferred by a single gene. Pathotypes of *G. pallida* are less easily distinguished as several genes act in concert to limit development of this species. Complete resistance to *G. pallida* is not known.

Fumigant and non-fumigant nematicides may be used to control PCN. Fumigant options include 1,3-dichloropropene and methyl isothiocyanate. The most commonly used non-fumigant options are aldicarb and oxamyl. The effective incorporation of a nematicide at planting can limit multiplication, although *G. pallida* is generally slower hatching and may not be fully controlled. To limit multiplication, it has been estimated that a nematicide must be at least 94% effective. When nematode levels are extremely high, the use of soil sterilants such as metham sodium may be the only option. In heavily infested soils competition for feeding sites and extensive root damage can result in few females being produced and hence a population decline. However, at low densities (e.g. 3–5 eggs/g soil) populations can increase up to 50-fold. Some cultivars produce large root systems and may be tolerant of invasion whilst supporting large populations of nematodes.

The ability of a cultivar to limit multiplication is indicative of its resistance. To achieve maximum control and minimize multiplication, integration of rotation, cultivar resistance and nematicide use is required. Soil testing to measure the species present and their level is routine in Europe and provides a basis for integrated control measures. Early detection of *G. pallida* is crucial if measures are to be taken to prevent development to significant threshold levels. Below a threshold of 5 eggs/g soil little damage is likely, but above 10 eggs/g crop damage would be expected.

Legislation to prevent the export or import of PCN is common in many countries. Thus seed multiplication is usually carried out in soils tested for absence of PCN to prevent spread on seed. Similarly, importers of seed may test batches for the presence of PCN, especially where the pest is rare or absent. Other control measures include soil solarization and trap cropping.

Stuart Wale

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Northern root-knot nematode
***Meloidogyne hapla* Chitwood**
Southern root-knot nematode
***Meloidogyne incognita* (Kofoid & White) Chitwood**
Columbia root-knot nematode
Meloidogyne chitwoodii* Golden *et al.

ROOT-KNOT NEMATODES

Other root knot nematode species attacking potato include *M. arenaria* (Neal) Chitwood, *M. fallax* Karssen, *M. javanica* (Treub) Chitwood, *M. thamesi* Chitwood

Symptoms

Haulm: High populations of root-knot nematodes may cause varying degrees of stunting, chlorosis or wilting. Symptoms are more evident under conditions of temperature or moisture stress.

Roots: *Meloidogyne* spp. invasion is identified by the presence of knots or galls, usually irregular in shape (**131**). Galls induced by the feeding of second-stage juvenile nematodes consist of hypertrophied cortical cells surrounding the nematode. The nematodes also induce the formation of syncytia or 'giant cells' in the vascular tissue, which are multinucleate with dense

cytoplasm and highly invaginated cell walls. The extent of galling varies with species: those caused by *M. chitwoodii* are small and difficult to see, those of *M. hapla* are distinct with root proliferation around the galls and those of *M. incognita* are more pronounced. In some instances, galls may be absent.

STOLONS: Symptoms are not usually evident.

TUBERS: The outer layers of tubers are sometimes invaded when large nematode populations are present in soil. Symptoms are not always apparent but may appear as wart-like protuberances (**132, 133**), and thin slices of tuber tissue may reveal adult females below the swelling (**134**). The females have pearly white, round to pear-shaped bodies surrounded by a brown layer.

Status of the pest

M. hapla parasitizes potatoes worldwide, especially in cooler regions. In temperate regions, it may cause serious economic losses by direct damage to the plant and through reduced tuber quality. *M. incognita* is more limited in distribution and is favoured in hotter climates as are *M. arenaria, M. javanica*, and *M. thamesi*. *M. chitwoodii* is a major pest of potatoes in the USA and has been found in localized parts of northern Europe. *M. fallax* is closely related morphologically to *M. chitwoodii* and is localized in a few locations in the Netherlands and Belgium, where mixed infestations may occur. It is believed to have a similar pest status to *M. chitwoodii*. Because of its limited national and international distribution, *M. chitwoodii* has a quarantine status in many countries.

131 Knots or galls in roots caused by root-knot nematode.

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132 Wart-like protuberances caused by root-knot nematode.

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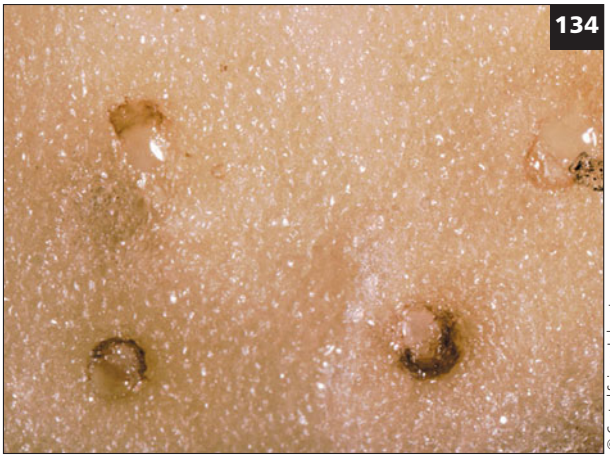
133 Cross-section of tuber invaded by *Meloidogyne* sp.



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134 Adult females beneath swelling on tuber.

Life cycle and biology

All root-knot nematodes display marked sexual dimorphism; the females are pyriform or saccate and the males are vermiform. With only slight variations, root-knot nematodes follow a similar life cycle. Embryonic development results in the first-stage juvenile, which moults once in the egg and hatches as a vermiform mobile second-stage juvenile. This stage migrates through the soil and enters a potato root where it moves through tissue to a suitable feeding site. It becomes sedentary and initiates the formation of syncytia or multinucleate giant cells from which the nematode derives its food. The nematode becomes flask-shaped and without further feeding moults three times into the third, fourth and adult stages. Shortly after the final moult, the saccate adult female resumes feeding and produces hundreds of eggs that are laid in a gelatinous matrix attached to the posterior of the nematode. For males, there is a change from a saccate to a vermiform shape during the fourth-stage juvenile stage. After the final moult, a fully developed vermiform male emerges, migrates through the soil, but does not feed. In *M. hapla*, in temperate zones where potatoes are grown, generation time is about four to six weeks, and overwinter survival is mostly in the egg stage.

Control

Microscopic species identification is possible for some species but is highly specialized. Closely related species such as *M. chitwoodii* and *M. fallax* usually require molecular methods. The occurrence of biological races has resulted in greater reliance on molecular methods. As spread can readily occur through the movement of infested seed potatoes, quarantine regulations preventing import exist in many states and countries. Local spread can occur in soil on farm machinery, in irrigation water or on animals moving from infested to uninfested fields. Care can be taken to avoid spread in these ways, but once established in an area spread may be difficult to restrict.

Soil fumigants usually offer good control of root-knot nematodes but they are expensive and may not be cost-effective for the grower. Organophosphate and oxime carbamates are also effective and are less expensive, but these compounds are very toxic to mammals, and they may also leave residues in tubers, soil and groundwater. Consequently, the use of fumigants and nematicides is now severely restricted in many regions. No commercial resistant potato cultivars are available to date. Crop rotation can reduce root-knot nematode populations, but host ranges differ between species and races, and identification is critical. *M. hapla* does not usually reproduce on various grasses and cereals, *M. incognita* does not usually reproduce on nematode-resistant tomato cultivars and *M. chitwoodii* does not usually reproduce on alfalfa. A few plant species such as marigold (*Tagetes* spp.) reduce the numbers of root-knot nematodes, but these crops are generally of little economic value. A weed-free fallow can reduce populations by 80–90%. Biological control methods using fungi, bacteria and sporozoans in commercial potato production still require considerable research.

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Trichodorus spp., *Paratrichodorus* spp.

STUBBY-ROOT NEMATODES

Symptoms

HAULM: High populations may cause varying degrees of stunting and wilting in potato foliage.

ROOTS: Stubby-root nematodes usually feed in the meristematic region of roots. Epidermal tissue may turn brown, and numerous stunted roots may develop which gives a 'stubby-root' appearance (**135**). However, symptoms on potato roots are uncommon.

STEMS AND STOLONS: Symptoms are uncommon.

TUBERS: Stubby-root nematodes can transmit *Tobacco rattle virus* (see Spraing, page 85).

Status of the pest

In general, *Trichodorus* spp. are found in cooler regions and *Paratrichodorus* spp. are found in warmer tropical and subtropical soils. Although direct feeding can cause significant damage to the host, the interest in stubby-root nematodes is largely due to their ability to transmit *Tobacco rattle virus*. Each species appears to vector a distinct serotype of the virus.

Life cycle and biology

Reproduction is usually amphimictic or parthenogenetic. There are six stages in the life cycle: the egg, four juvenile stages and the adult. All the juvenile stages, as well as the adult, can vector viruses. Depending on the species, virus particles are selectively adsorbed on the lining of the oesophagus and dissociation occurs as the nematode saliva is injected into the host. Life cycle periods depend largely on temperature and can vary from 16 days at 30°C for *Paratrichodorus minor* to about 45 days at 15°C for *Trichodorus viruliflerus*. Stubby-root nematodes are migratory ectoparasites that feed in the zone of root elongation and appear to migrate with the roots as they grow.

Control

The emphasis on control of stubby-root nematodes is usually due to their importance as virus vectors rather than from the direct effects of parasitism. Volatile fumigant nematicides offer good nematode control, but they are expensive and may not be cost-effective.

Andy Evans/SAC

135 Thickened, branched roots and stunting (right) compared to an unaffected plant (left).

Non-volatile nematicides such as fenamiphos, aldicarb, carbofuran and oxamyl are also effective in reducing nematode numbers and virus transmission and are less costly than fumigants. However, they can leave residues in tubers, soil and groundwater. Consequently, the use of fumigants and nematicides is now severely restricted in many regions. Stubby-root nematodes have a wide host range, so crop rotation is usually impractical. Resistant potato cultivars are available and may be a management option in some pathosystems. Repeated cultivation of soil has an adverse effect on nematode numbers, but it is not usually considered to be an adequate control method. The biological control of stubby-root nematodes and the viruses they vector requires more research.

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Pratylenchus penetrans **(Cobb) Filipjev & Schuurmans Stekhoven and other *Pratylenchus* spp. including *P. andinus* Lordello, Zamith & Boock, *P. brachyurus* (Godfrey), *P. coffeae* (Zimmerman), *P. crenatus* Loof, *P. neglectus* (Rensch), *P. scribneri* Steiner, *P. thorneyi* Sher & Allen, *P. vulnus* Allen & Jensen**

ROOT-LESION NEMATODES

Symptoms

Haulm: High populations of root-lesion nematodes give the crop the appearance of typical plant stress. There may be areas of poor growth, the plants appear to lack vigour, and the foliage loses its rich green texture. Infected plants mature earlier, and in association with other organisms such as *Verticillium* spp., potato early dying (PED) disease may be evident (see Verticillium Wilt). Symptoms similar to those occurring on roots sometimes occur on lower stem tissue.

Roots: *P. penetrans* is primarily a parasite of the root cortex, migrating through and between parenchyma cells. The area of the roots where the nematodes feed usually exhibits dark-coloured necrotic lesions (**136**). Root necrosis may be accelerated by invading secondary organisms and root-rot symptoms.

Stolons: Symptoms in stolons are not usually evident.

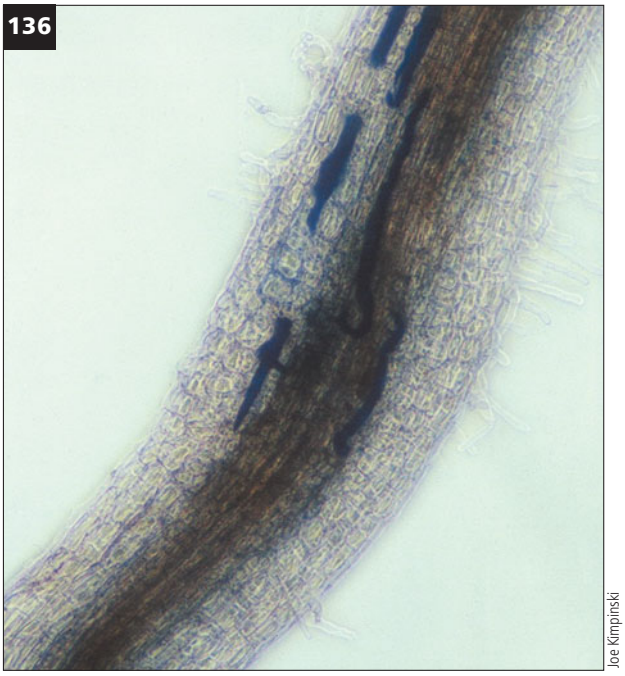
Tubers: The outer layers of tubers are sometimes invaded when large nematode populations are present in soil, and symptoms may appear as wart-like protuberances (**137**).

Status of the pest

P. penetrans parasitizes potatoes worldwide, especially in the north temperate regions. It is considered to be the most important plant parasitic nematode in northeastern North America. Economic studies have shown that this nematode species can reduce tuber yields by over 50% when populations are high. Since the distribution of the nematode is so cosmopolitan, lower populations still cause losses over very wide areas.

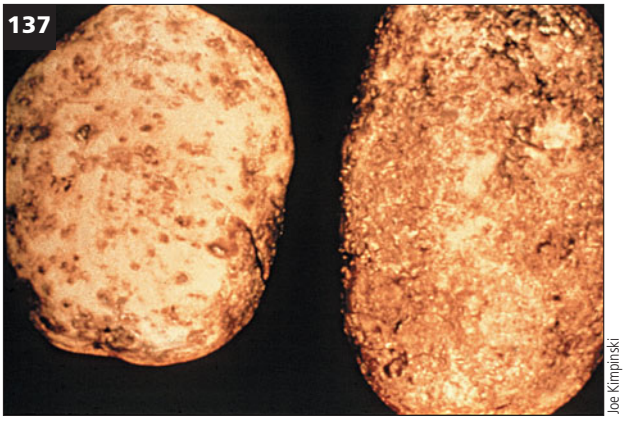
Life cycle and biology

P. penetrans (**138**) usually enters and inhabits roots, though tubers and rhizomes are sometimes invaded. This nematode may also leave the roots and migrate through the soil in search of fresh root tissue. In some cases, ectoparasitic feeding occurs on root hairs.



Joe Kimpinski

136 Root lesions caused by root lesion nematode. Blue streaks are the stained nematodes in the cortical tissues.



Joe Kimpinski

137 Tuber symptoms. The nematodes are within the tuber, usually near the surface.

Joe Kimpinski

138 Root-lesion nematode (*P. penetrans*).

All stages from the second juvenile stage to the adult invade roots, usually penetrating the zone of root elongation. Once inside they migrate through root tissue, feeding on cortical cells. The females lay eggs inside the roots, and depending on the temperature during the growing season, generation time is 30 to 60 days. Nematodes become inactive in late autumn and overwinter in the stage to which they have developed.

Control

Soil fumigants usually offer good control of root-lesion nematodes, but they are expensive and may not be cost-effective. Organophosphate and oxime carbamates are also effective and are less expensive, but these compounds are very toxic to mammals and they may leave residues in tubers, soil and groundwater. Consequently, the use of fumigants and nematicides is now severely restricted. Resistance to *P. penetrans* as a means of control has not been fully explored. Russet Burbank is more tolerant to root-lesion nematode attack than cultivars such as Superior, but the former does not reduce the size of nematode populations. Crop rotation is not highly effective since *P. penetrans* has a very wide host range (e.g. maize or wheat is a good host crop). Some plant species such as marigold (*Tagetes* spp.) reduce the numbers of root-lesion nematodes, and several cultivars have been developed specifically for this purpose. However, the crop is generally of little economic value, and growers are reluctant to use this method of nematode control.

JOE KIMPINSKI

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Insects and other pests

DIVERSITY is a characteristic of potato pests. Whilst most pests are insects, other arthropod groups are also represented. Potato pests are frequently mobile and may travel or be transported large distances. Given the ability to multiply rapidly where suitable environmental conditions exist, pests can be extremely damaging. A good example of the potential to cause severe damage is Colorado beetle, which can strip a crop of foliage if left unchecked.

The diversity of pests is mirrored by a diversity of life history and resulting approaches to control. Many insects pass through several stages in their life cycle and potatoes are not always attacked by all stages, so an understanding of life cycle is crucial to ensuring that control measures are effective. With many pests, insecticides or other pesticides are available. However, their use is not always acceptable, nor are they always affordable. Because of their size, in comparison to other potato pathogen groups, pests are relatively easily identified, and monitoring numbers is often part of a control programme.

Many insect pests attack above-ground haulm or below- ground stem tissue: relatively few attack tubers. Exceptions are potato tuber moth and wireworm. Slugs, which are molluscs, also attack tubers. Some pests are also vectors of viruses. Most notably, aphids (especially *Myzus persicae*) can carry a number of important viruses and efficiently transmit them into potato plants. In this instance, aphid numbers are less important than whether they are carrying virus. Knowledge of both aphid and virus is important to create effective control measures.

Larvae of click beetles including *Agriotes* spp., *Athous* spp., *Conoderus* spp., *Ctenicera* spp., *Limonius* spp., *Melanotus* spp.

WIREWORMS

Symptoms

Haulm: Potato haulms can be invaded, but plant growth is not normally affected.

Roots and Stolons: No distinct symptoms are normally seen.

Tubers: Small round holes mark the entry point of wireworms, but they may tunnel within the tuber extensively (**139**). This damage may occur in newly planted seed tubers but rarely affects growth.

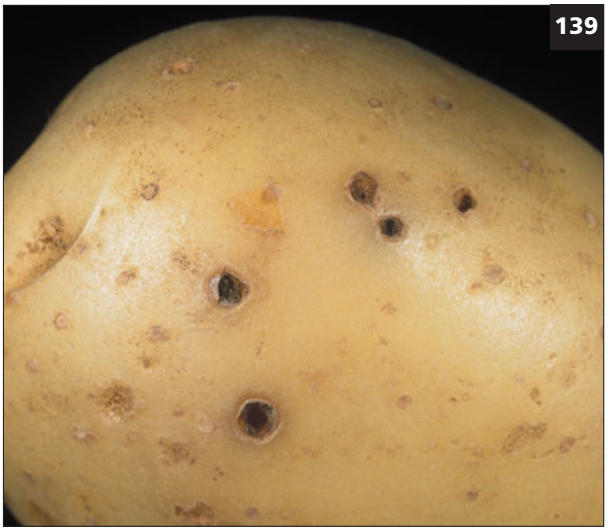
Status of the pest

There are at least 39 species of wireworms that attack potatoes. Damage to progeny tubers affects quality and marketability. With increasing emphasis on blemish-free tubers, even limited damage can be significant. Wireworms are mainly of concern in Europe and North America.

Life cycle and biology

Wireworms are the larvae of 'click' beetles (**140**) in the family Elateridae, named because of the noise the beetles make when flicking themselves upright after falling on their back. The adults (**141**) lay eggs in batches of 50–150 just below the soil surface in the spring or early summer. After several weeks, the eggs hatch into larvae. Initially they are hardly visible (at 1–2 mm in length) and transparent. As they grow their colour darkens to a golden brown. The larvae have a distinct dark brown head with biting mouthparts. The body is cylindrical with a tough skin. Three pairs of legs are found behind the head. The life cycle takes up to four or five years to complete. Larvae moult twice annually in late summer and early autumn. After two or three years they are large enough to cause economic damage, reaching 12–40 mm in length.

Feeding damage occurs in the periods prior to moulting in spring and early autumn. At the end of the larval period, when mature, they burrow deep in the soil, create a 'cell' and pupate. About a month later they emerge as adults.



139 Entry holes of wireworm into tuber.

140 Wireworm larva tunnelling into tuber flesh.

141 Click beetle – adult wireworm.

Typically, eggs are laid in pasture (grass, clover, alfalfa), especially long-term grassland. Only when it is ploughed for potatoes does damage occur. Although in the first two or three years after ploughing damage is minimal as the larvae feed on the decaying organic matter, even low levels of damage can seriously affect quality. Several years after ploughing, once the organic matter has declined, wireworms can cause severe damage as they seek other food sources. More recently, wireworm damage has been found after the growing of long-term small grain crops in Europe ('arable' wireworm).

Control

Avoiding fields with high populations is recommended. This may mean avoiding fields that follow crops into which adults lay their eggs (e.g. grass) or by testing fields for the level of infestation. Baiting methods comprise placing a food source, typically whole or ground-up cereal grain, in a mesh bag or container with holes and burying in the soil up to 15 cm deep for several days. Baits are buried at random sites across a field. After removal, the number of sites where larvae are found is determined and related to a threshold. Thresholds vary from country to country depending on the quality criteria, but one or more per square metre can cause economic losses with washed table potatoes. Soil conditions influence the extent to which baits are colonized, and this test is usually only used as an indicator. Pheromone traps in the crop preceding potatoes have also been used to estimate populations and predict the risk of damage.

Where possible, a fallow period over summer during which soils dry out will reduce wireworm numbers. Avoid planting in poorly drained or wet fields, since these favour survival of the pest. Wireworms are usually less prevalent with arable crop rotations. In temperate climates, autumn cultivations during arable rotations tend to result in a decline in wireworm numbers.

A number of soil-applied insecticides are effective against wireworms. These include aldrin, diazinon, ethoprop and fonofos. Specific recommendations for their use against wireworms are approved in some countries. In other countries, the insecticides are used against other pests and have a secondary effect against wireworms.

There are no cultivars of potatoes resistant to wireworms.

Stuart Wale

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***Arion* spp. (e.g. *A. hortensis* [Ferussac], *A. distinctus* Mabille), *Milax* spp. (e.g. *M. budapestensis* [Hazay], *M. sowerbyi* [Ferussac]), *Deroceras reticulatum* (Müller), *Limax maximus* L. and other species**

SLUGS

A number of slug species are associated with potato damage, including those named above.

Symptoms

Haulm, Roots and Stolons: Occasionally slugs feed on sprouts or the haulm but with little significant effect. Damage to sprouts or haulm is irregular grazing (**142**) with ragged edges typical of slugs.

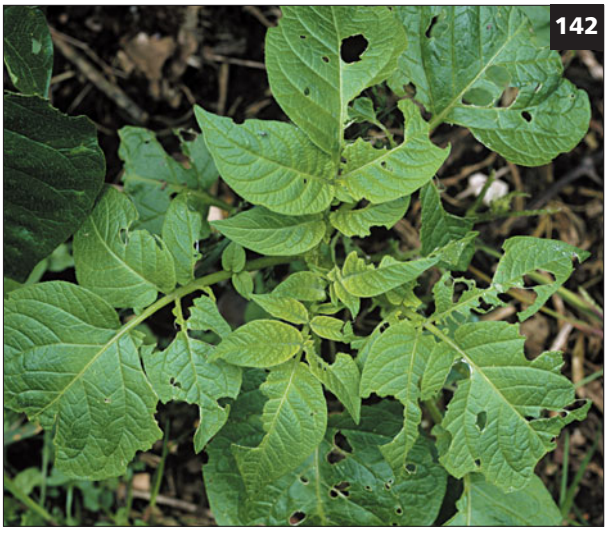
Tubers: Damage consists of an entry hole (**143**) and an excavated cavity beneath (**144**). The cavity may be extensive. A trail of slime is an indicator of slug activity. Occasionally bacterial or fungal secondary rots may develop from tuber damage by slugs.

Status of the pest

In the production of blemish-free tubers for the washed potato market, the presence of slug damage can have a major impact on value. On individual tubers, a single slug hole can result in rejection during grading. They are also important in processing outlets where excessive peeling may be required. Whilst all cultivars may be attacked potentially, some are much more susceptible than others. Damage varies from season to season; in one quoted example it varied from 0.2% to 55%. Slugs occur in many parts of the world but are a problem mainly in temperate, wetter, often maritime, climates such as northern Europe. They are a particular problem in heavy, moisture-retentive soils where a fine tilth is difficult to achieve. During wetter seasons, slug damage is also possible in lighter soils. Delayed harvest in wet seasons can result in more damage.

Life cycle and biology

Slugs require mild, moist conditions to be active. In dry or cold weather they move down the soil profile, under stones, or into organic matter. Slugs are generally more active at night. Numbers present in a field are influenced by cropping history and previous weather. Eggs are laid in clusters in organic matter and if laid before winter may not hatch until spring. However, hatching can be as short as 3–4 weeks if conditions are good.



142 Slug damage to leaves.

143 Slug entry holes may disguise the greater level of damage within.

144 Slug damage to tubers.

145 The grey field slug *Deroceras reticulatum*.

146 The garden slug *Arion hortensis*.

Of the most comon pests, *Deroceras* slugs (**145**) go through one complete and one half generation each year, while *Arion* (**146**) and *Milax* (**147**) species have annual life cycles, breeding at a time of year particular to each species. Individual growth rates, as well as life cycles, vary in length, and in any field the population may consist of several species of differing ages and sizes.

Control

Control is difficult and may be considered a matter of judgement rather than science. There is no simple relationship between slug numbers and damage, as the weather and soil conditions play a major role. Under ideal conditions for slugs, small numbers can have a big effect. Risk needs to be assessed and based on local experience. Slug numbers can be assessed using traps or by observing feeding damage on weeds, etc. but the soil type, previous cropping history, irrigation, soil tilth as well as weather conditions all need to be considered. Lighter soils may be considered a lower risk, but these are most frequently irrigated and this can exacerbate damage. Harvesting early may reduce the effects of slugs, but this depends on weather conditions and is no guarantee against damage. There are differences in susceptibility of cultivars to slug damage, and growing less susceptible cultivars in high-risk fields, and the use of molluscicides, are the main control measures. Molluscicides are applied in the form of pellets to the surface of the crop. A number of applications may be required for effective control. Since damage is done below ground, the use of molluscicides is not always successful.

Stuart Wale

147 Keeled slug: ***Milax/Tandonia* spp.**

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Empoasca fabae **(Harris),** ***Macrosteles quadrilineatus*** **(Forbes),** ***Aceratagallia sanguinolenta*** **Provancher,** ***Circulifer tenellus*** **(Baker)**

POTATO LEAFHOPPERS

Symptoms

HAULM: Potato leafhoppers feed on leaf tissue. Excessive feeding results in 'hopperburn' – damage to the foliage which is seen as a brown, triangular lesion at the tip of the infested leaflet. Lesions spread backwards and inward from the leaf margins eventually destroying the entire leaflet (**148**). Destruction of foliage can result in yield losses.

ROOTS, STOLONS AND TUBERS: Symptoms are not normally found.

Status of the pest

Potato leafhoppers are strong fliers and are important because of the direct feeding damage they cause. Both adult and nymphs of the potato leafhopper (*Empoasca fabae*) damage vascular tissues (phloem) by mechanical disruption and/or release of toxins. Late-instar nymphs can reduce yields to a greater extent than can a similar number of adults.

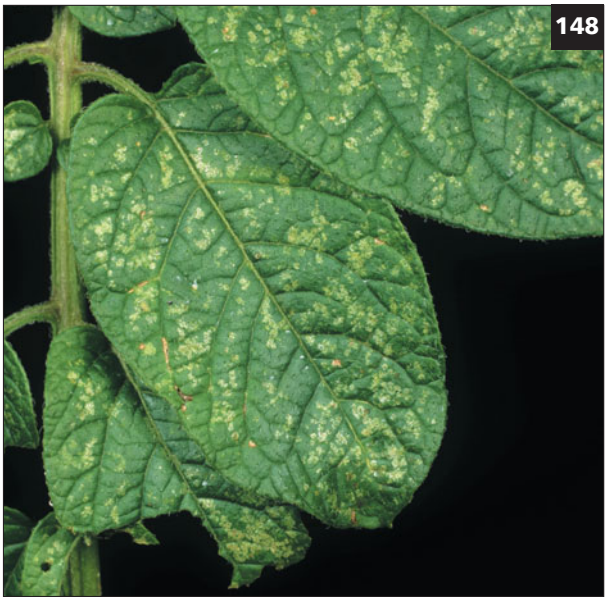
The aster leafhopper (*Macrosteles quadrilineatus*) can also transmit the aster yellows phytoplasma (see page 74), causing aster yellows disease (also known as purple top) in potato. The potato leafhopper reproduces on potato but is not a vector. Meanwhile, the clover leafhopper (*Aceratagallia sanguinolenta*) and beet leafhopper (*Circulifer tenellus*) can transmit *Potato yellow dwarf virus* and *Beet curly top virus*, respectively.

Life cycle and biology

The potato leafhopper is generally found in warm climates and does not occur in all potato-producing areas. The insects are wedge shaped with a broad head and thorax, and tapering along the wings (**149**). Leafhoppers have three life stages: egg, nymph (five instar stages) and adult. On potato, the insect can have several generations during the growing season. The adult potato leafhopper begins laying eggs when about six days old and can live for 30–40 days, resulting in generation overlaps. Insect development and population levels on potato are regulated by temperature and date of arrival on the crop, except for the aster leafhopper, which does not reproduce on potato. Eggs hatch in about 10 days and the yellow-green nymphs, which resemble adults, require about 12 days to mature. Leafhoppers that attack potato overwinter as adults in warm climates, and the aster leafhopper can overwinter as eggs. Adult leafhoppers are carried long distances on updrafts and fast-moving weather systems.

Control

Potato leafhopper populations should be monitored by sweep-netting of adults or counting nymphs on mid-plant leaves. Differences in threshold values for treatment application can vary, but population densities


148

148 **Leafhopper damage.**


149

149 **Adult leafhopper.**

of nymphs greater than 10 per 100 plants is used in some areas to warrant a treatment with an efficacious registered insecticide. Dynamic models for action thresholds for specific production areas may be useful when this pest is causing economic losses. Insecticide resistant populations have not been reported in most areas, but monitoring is essential for the support of efficacious treatments. Leafhoppers have few natural enemies but an insect-pathogenic fungus (*Erynia radicans*) is occasionally found. Roguing plants with purple-top does not prevent spread of the aster yellows phytoplasma since the aster leafhopper cannot acquire it from potato.

H W (BUD) PLATT

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***Epitrix cucumeris* (Harris), *Epitrix tuberis* Gentner, other *Epitrix* spp., *Psylliodes* spp.**

POTATO FLEA BEETLES

Symptoms

HAULM: Feeding on leaves by adults produces round feeding scars, 0.1–5.0 mm in diameter, which frequently penetrate through the leaf to form a hole. When the potato flea beetle is abundant, potato leaflets receive multiple perforations, eventually exhibiting a 'shot-hole' appearance (**150**). The vertical distribution of the flea beetles or their damage can be influenced by previous damage and by meteorological conditions. Larvae of all flea beetle species feed on roots, stolons, and tubers, but only the tuber flea beetle (*E. tuberis*) causes extensive tunneling damage in tubers.

ROOTS AND STOLONS: Symptoms are not normally found.

TUBERS: Symptoms are not normally found but, where present, the larvae mostly cause cosmetic tuber damage in the form of pimpling, surface channels and shallow networks of fine tunnels. The damage can usually be removed by peeling.

Status of the pest

Flea beetles (family Chrysomelidae) are a widespread insect group with many genera: some are agricultural pests while others are beneficial in their control of certain weeds. Several species of the genus *Epitrix* infest potato in South, Central and North America and several species of the genus *Psylliodes* in Eurasia. The best-known species is the potato flea beetle (*E. cucumeris*) followed by the tuber flea beetle (*E. tuberis*). The potato and tuber flea beetles are primarily pests of potato, but they also attack cucumber, egg plant, pepper, tomato and other plants. Flea beetles are important insect pests but not primary pests in commercial potato production in America, although yield losses of 10–25% have been shown locally. They are marginal pests in Europe.

Early-season feeding by adults is not normally considered significant. The most significant direct damage is from late-summer feeding by adults in crops that do not receive insecticide late in the season. Mid- and late-season potato crops are subject to severe damage from second- and third-generation larvae when the tubers are

maturing. In first- and second-year potato fields, flea beetles from surrounding headlands tend to migrate mostly into the outer rows, where the greatest damage occurs. By their feeding, larvae may directly transmit pathogens to tubers and roots and increase secondary infection of damaged tubers. Adults may spread pathogens when they emerge from pupation sites in the ground. Fungal diseases associated with flea beetles include common scab, Fusarium dry rot, Rhizoctonia, and Verticillium wilt. In addition, bacterial diseases and *Potato spindle tuber viroid* may be transmitted mechanically. The incidence of the potato flea beetle and the severity of common scab have been correlated positively in Manitoba.

150 Flea beetle damage on potato foliage.

151 Adult flea beetle.

Life cycle and biology

The potato flea beetle adult (**151**) is 1.7 mm long and 1 mm wide. It is black with brown legs and brown antennae. The tuber flea beetle is similar in size but black and shiny. The femur of the hind legs is thicker and darker than the other leg segments in all species. When disturbed, they use these legs to rapidly jump away like a flea. Adults overwinter at the soil surface among litter or undergrowth, either in or near the potato fields where they fed the previous summer. In spring, when the temperature rises above 10°C, they resume activity, feeding on herbaceous plants before moving to potato fields to feed on the foliage of newly emerged potato plants. The beetles may be found on all above-ground parts of the plant and on the soil surface. They feed on both the upper and lower leaf surfaces, but more frequently on the upper surface. If potato plants have not yet emerged, they feed on weed hosts.

Females lay eggs in the soil around potato plants and then die. Usually all overwintered adults die by first flowering of the potato crop. The larva is small, slender and white with a dark brown head and minute legs. When fully grown, it is about 5 mm in length. On occasion, they may enter the tubers, forming small hollows at the point of entry and tunnels, which are usually straight, about 0.8 mm in diameter and less than 6 mm long, and filled with corky tissue. This type of tuber injury contrasts with the deeper, more penetrating tunnels made by the tuber flea beetle. Soils that have high moisture levels and relatively high temperatures, such as organic soils, favour population growth. Thus, potato crops in muck soils are usually at greater risk than those in mineral soils. Following larval pupation in the soil, adult beetles emerge, crawl to the surface and feed on potato foliage. Adult feeding continues on potato leaves until the weather becomes too cold or the foliage becomes unsuitable. Adults that leave potato plants may feed on other favoured hosts before entering overwintering sites. One to three generations per year are produced, depending on species and location.

Control

Monitoring the adult flea beetles throughout the growing season ensures efficient and cost-effective control. Economic thresholds for tuber flea beetles are based on visual and sweepnet counts of adults. In British Columbia, Canada, if an average of one beetle per 60 inspected plants is observed, it is recommended

that a spray be applied to the inner rows or to the perimeter rows. Plants taller than 30 cm should be sampled with a 30 cm sweepnet. A spray is recommended if an average of more than one beetle per 10 sweeps is observed in either the outer or the inner rows. An economic threshold of up to 100 adult potato flea beetles per plant has been calculated for the cultivar Norland in Manitoba, Canada, in August, although yield declines sharply when beetle numbers exceed that density. However, because adult flea beetles are extremely active and are easily disturbed when approached, it is difficult to monitor adults. It is easier to sample potato foliage for feeding damage by counting the number of shotholes per leaflet. Unfortunately, at least in the case of *Epitrix cucumeris*, there does not appear to be a good relationship between the number of shotholes per leaflet and effects on yield. A conservative action threshold is 50 shotholes per leaflet. Thresholds may vary for different cultivars and at different times during the season. Early-maturing cultivars, such as Superior, are less tolerant of feeding injury late in the season than are later-maturing cultivars, such as Russet Burbank. A preliminary estimate of the economic threshold for the cultivar Norland in Manitoba is 65–75 feeding punctures per terminal leaflet from the lower third of the plant taken two weeks after first flowering. Later in the season, however, the number of feeding punctures may exceed this threshold without signifying a need for control. The threshold also may differ for other cultivars and in other regions. In Atlantic Canada, an economic threshold of 15 feeding punctures on the fourth terminal leaflet (counting down from the apex of the plant) has been advocated, but this number now appears to be too low. The thresholds may need to be lowered for plants that are under stress from weather or from attack by other insects or pathogens.

There is rarely any need for chemical control measures specifically for these insects, since insecticides applied to control Colorado potato beetles and aphids usually keep flea beetle populations on the potato crop in check. For instance, granular systemic insecticides applied at planting may be effective against the potato flea beetle for the whole season. Where systemic insecticides are not used at planting, the need for control must be based on monitoring and population sampling. Foliar sprays must be applied against the unprotected adult stage. Resistance to currently used insecticides is not generalized but has been reported in the case of the tuber flea beetle. Normally, only mid- and late-season potato cultivars require chemical control of the tuber flea beetle. The potato flea beetle tends to be more abundant in parts of potato fields that are adjacent to uncultivated areas. Such areas often have suitable food hosts for the adult beetles and in some countries retain snow, which may enhance their survival over winter. Because adult flea beetles do not readily fly, the separating of uncultivated areas from potato fields may reduce populations. Eliminating volunteer potatoes and other hosts may also be used to starve overwintered adults in the spring before potato crops emerge. Yearly crop rotation is the key cultural practice, because flea beetle populations build up in fields planted repeatedly to potato.

None of the commercial cultivars of potato is known to be resistant to the potato flea beetle, nor are there biocontrol agents commercially available.

Gilles Boiteau

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Leptinotarsa decemlineata (Say)

COLORADO POTATO BEETLE

Symptoms

Haulm: The adult and all larval stages of the Colorado potato beetle (CPB) (Coleoptera: Chrysomelidae) feed mostly on the foliage of host plants, chewing irregular holes in, and along, leaf margins (**152**), but they also attack stems. The fourth larval instar and the adults are the most destructive stages. Hosts of the CPB are limited to the wild and cultivated Solanaceae. The beetle prefers potato, but it also attacks egg-plant (aubergine), tomato and weeds such as ground cherry, wild tomato and climbing nightshade. High populations can completely defoliate plants, locally or throughout a field (**153**). Extensive feeding at any time during the season, but especially when the crop is in bloom, can reduce yield. The suggestion that the CPB might be a vector of certain bacterial and viral diseases of potato has not been confirmed.

Roots and Stolons: Symptoms are not normally found.

Tubers: Symptoms are rare (**154**).

152 Early symptoms of Colorado potato beetle damage – holes in leaves.

153 Complete defoliation by Colorado potato beetle.

154 Colorado potato beetle larvae attacking potato tubers.

155 Adult Colorado potato beetle.

156 Eggs of Colorado potato beetle.

157 Larva of Colorado potato beetle.

Status of the pest

The CPB originated in Mexico, from where it spread throughout North America and crossed the Atlantic to the Eurasian continent. Today, it is a significant pest of the potato crop in America and in Eurasia, where it is now reaching China. Uncontrolled, the CPB can reduce potato yields by an average of 30% in North America.

Life cycle and biology

The CPB adult is about 10 mm long, 7 mm wide and somewhat rounded. Its head and anterior thorax (pronotum) are brown-orange to yellow and covered with variously shaped black markings (**155**). Ten black lines run the length of the forewings (elytra), which otherwise are pale yellow. Females can be recognized by their greatly distended abdomen and the absence of a depression in the last abdominal segment when viewed from below. Adults overwinter or aestivate in the soil of potato fields or in adjacent hedges or grassy borders. As the temperature rises again, the beetles move up through the soil. They disperse by walking if temperatures are cool and may fly short distances if temperatures are high coupled with continuous insolation. They feed for a few days, after which mating and egg laying occur. Individual females lay 300 to 500 eggs over a period of up to two months. During this period, they may move from older to younger plants. The eggs are elongate and yellow to orange, and usually they are laid on the underside of leaves of the host plant in clusters of about 30 (**156**). They must not be confused with the eggs of the Coccinellidae, which are smaller and usually a lighter colour.

The extended oviposition period means that hatching larvae may be present in the field for three to five weeks, although larval development from hatch to pupation may require only two to three weeks. The larva is humpbacked, and red-orange with two rows of black spots along the sides of the body (**157**). There are four larval stages. These differ in size but are best differentiated by measuring their head width. Pupation occurs in the soil. CPB development from the egg to the adult requires 21–56 days depending on temperature (15–28°C) in North America but may require more time in Europe, suggesting the existence of genetically different populations. One to four generations per year can occur depending on the climate and the availability of host plants.

Control

Monitoring

The density of egg, larval, and adult stages can be estimated by visual counts on a fixed number of whole plants or plant stems chosen randomly from different parts of the field. Sampling by sweep net is not suitable for estimating numbers. Sampling sites are not selected in border rows of fields, because beetles may be concentrated in these areas at the beginning of the season. The average number of beetles found in the field might be distorted by counts from border rows. However, concentrations of beetles on field borders are important and should be reported separately from overall counts. Currently, recommendations to commence control are based solely on empirical observations of leaf damage or beetle abundance for different regions or countries.

In Canada, a count threshold of two to eight large larvae or adults per plant is used, depending on the province. In some areas the threshold is set at two beetle equivalents per plant on the basis of one adult = three large larvae = eight small larvae. Elsewhere, the threshold is set at 2% of the plants with one or more compound leaves 50% defoliated. It is advisable to consult with local advisers for thresholds adapted to specific areas. Thresholds tend to be lowest during bloom when potato plants are most vulnerable. In countries where it is absent, CPB is a notifiable pest.

Chemical control

An array of systemic and foliar insecticides is available. Growers are encouraged to spray only when necessary and to alternate their choice of insecticides from among the different families of chemicals available. Resistance to insecticides is a more serious problem in North America than in Europe, where outbreaks seem more sporadic. Spot treatment with foliar insecticides should be applied against adults early in the season only where numbers warrant it. Otherwise, insecticides are best limited to the plant-bloom period where numbers exceed the economic threshold, and at the end of the season if unusually high numbers are present to overwinter. In fields that have not been rotated for many years, or were surrounded by fields with extremely high beetle infestations the year before, the application of a systemic insecticide in the furrow at planting will effectively control the colonizing population. Alternative controls should be used whenever possible to reduce the risk of insecticide-resistance development.

Cultural practices

Apart from the use of insecticides, crop rotation is one of the few control techniques currently available to potato growers. It is best to avoid planting in fields or adjacent to fields where high numbers of adult beetles were present at the end of the last growing season. Rotation can significantly reduce beetle numbers, and it also results in the initial localization of incoming beetles at the periphery of the potato field, where insecticides can then be applied as a spot treatment. Growers may not have the land needed for rotation, and alternative crops tend to be less profitable than potato. Planting all fields of a region within a short period of time can help reduce the reproductive potential of females. Hilling (earthing-up) can bury egg masses deposited on the lower leaves. In addition, planting one to three rows of potato along the edge of the field to 'trap' and destroy (by insecticide treatments) the beetles can prevent them from entering the crop.

Physical control

Plastic-lined trenches or other barriers surrounding newly planted potato fields can reduce crop colonization in the spring by overwintered adults by some 50%. This barrier can also reduce oviposition and emigration to the overwintering sites at the end of the season. Propane burners and vacuum machines can also successfully control beetles accumulating at the periphery of potato fields in the spring.

Resistant cultivars

No commercial cultivars of potato are resistant to the CPB at present.

Biological control

Native predators, which include the ground beetles *Lebia* spp. and *Pterostichus* spp., the two-spotted stink bug *Perillus bioculatus* (Fabricius) and the spotted lady beetle *Coleomegilla maculata* (DeGeer), and the parasites *Edovum puttleri* Grissell and *Myiopharus doryphorae* (Riley) rarely have a significant impact on the abundance of the CPB. A strain of the bacterium *Bacillus thuringiensis* Berliner has been registered for foliar application against the small larvae of the CPB. Formulations of the fungus *Beauveria bassiana* have shown efficacy against the small larvae of the CPB in the USA and in some Eastern European countries.

Gilles Boiteau

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Liriomyza huidobrensis **Blanchard (syn.** ***Agromyza huidobrensis*** **Blanchard),** ***L. cucumifoliae*** **Blanchard,** ***L. langei*** **Frick,** ***L. dianthi*** **Frick**

LEAFMINER FLY

Symptoms

Haulm: Newly hatched larvae feed under the leaf epidermis mainly close to the leaf midrib, creating characteristic serpentine 'mines' which increase in diameter as the larvae grow (**158**). Affected leaf tissue becomes necrotic and brownish. Older, lower leaves show the first damage. Middle and upper leaves are progressively damaged as the plant develops and infestation continues. As necrotic areas coalesce in highly infested leaves, the leaves dry out and die. Accordingly, highly infested potato fields appear burned.

Roots, Stolons and Tubers: No symptoms.

Status of the pest

Lyriomiza huidobrensis is a destructive, polyphagous insect pest in the tropics and subtropics. It has been reported from Peru, Chile, Argentina, Brazil, Central American countries, Mexico, Northern Africa, Kenya, Indonesia, Malaysia and Israel. The leafminer fly has become a key pest in all countries where it has been unintentionally introduced, capable of completely

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158 **Typical leafminer fly damage.**

159 Leafminer larval stage.

160 Leafminer pupa surrounded by puncture holes.

destroying potato fields if no control is applied. In many regions, leafminer flies have become resistant to commonly used insecticides (e.g. carbamate, organophosphate and pyrethroids) that are used to control larvae and adults. Without control, yields are often reduced by 50%. Potato farmers in the coastal valleys of Peru try to control the pest with 8–13 sprayings at regular intervals each season. Therefore, insecticides often present the highest input cost in potato production. Most of the leafminer fly research of the past 20 years has been oriented towards finding new chemical compounds to replace those insecticides that have lost their efficacy. In this context, the surge of leafminer fly as a pest has been attributed to the destruction of its natural enemies. As a result, alternative control methods have been investigated, including the evaluation and development of tolerant potato varieties, the role and use of natural enemies, cultural practices, trapping devices, and the selective application of larvicides. These control tools, combined with monitoring of the fly population, form the basis for developing integrated pest-management strategies.

161 Leafminer adult stage.

Life cycle and biology

A leafminer fly has four life stages: egg, larva (**159**), pupa (**160**) and adult (**161**). Adults are small (1.7–2.3 mm) black flies with bright yellow spots on the thorax, with females being slightly larger than males. Peak emergence of adults occurs before midday and males usually emerge first. Mating and oviposition occur after 6–24 hours and over a period of 1–3 days, respectively. A single mating is sufficient to fertilize all the eggs. Females use the ovipositor to make holes, so called 'feeding punctures', in the top and/or bottom of leaves, promoting the production of exudates that will feed both males and females. Wounds prepared for egg laying are called 'oviposition punctures' (**160**). On average, females lay 8–14 eggs per day, which are inserted just below the leaf surface. Eggs are oval and translucent, about 0.3 × 0.1 mm. At lower temperatures oviposition is increased; 18°C being the optimum temperature for development. Larvae hatch within 2–5 days, according to temperature, and start feeding in the spongy mesophyll of the leaf. Larvae develop in three distinguishable larval stages (4–10 days) that can be

recognized by the length and thickness of the leaf mine. The third-instar larva measures 3.2 × 1 mm. Mouth hooks remaining in the mines after each successive moult can be used for stage identification. Due to their mining habits, the larvae reduce the photosynthetic abilityof the plant, and that causes the main damage. Leafminer fly pupae develop either on the plant foliage or just beneath the soil surface. Pupae vary in size (1.6–3.25 mm long, 0.7–1.1 mm wide) and colour (light brown to almost black). Adults emerge 7–17 days after pupal development. With a life cycle of only 2–5 weeks, up to 15 generations per year may develop.

Fourteen families of plants have been recorded as hosts, without a clear preference for any particular family. Included in these are *Amaranthus* spp., *Aster* spp., aubergine, beet , red pepper, celery, chrysanthemum, cucumber, *Dahlia* spp., *Dianthus* spp., faba bean, garlic, *Gypsophila* spp., hemp, *Lathyrus* spp., lettuce, lucerne, melon, onion, pea, common bean, potato, *Primula* spp., radish, spinach, tomato, *Tropaeolum* spp., *Verbena* spp. and *Zinnia* spp.

Control

Ecological and economical control of the leafminer fly is best realized when based on integrated pest management by promoting natural regulation and combining cultural practices with physical and chemical control.

Conserving beneficial insects

Leafminer flies are controlled by a large number of beneficial insects, which are either predators or parasitoids (see next page). The species involved depends on the local agroecology. In regions rich in natural enemies it has been proven that leafminer flies are less damaging. Strategies to conserve beneficial insects can be manifold and include diversified cropping systems, high structural floristic diversity in agricultural landscapes, special weed-management practices and the reduced use of selective insecticides.

Crop management

Healthy, vigorously growing plants are able to counteract the damaging effect of leafminers, particularly during the vegetative phase. The fast-growing foliage enhances the egg extrusion reaction of the foliar tissue. Plants deficient in water and fertilizer, or grown from from low-quality seed (e.g. virus infested seed) show damage much earlier and mined leaves dry more rapidly. Under these circumstances, the negative effects of leafminer fly infestations further reduce low yields due to inadequate agronomic practices.

Adequate N-fertilization

High N-content of leaves promotes leafminer fly development. Therefore, a balanced N-fertilization should be considered.

Crop rotation

The continuous availability of food supplies through repeated crop planting will favour an abundance of the leafminer fly. It is therefore recommended to rotate with non-hosts.

Monitoring pest populations

Counting the number of flies captured in yellow sticky traps (see below) can monitor adult leafminer flies. Counting the number of larvae, or fresh tunnels per leaflet, by sampling the bottom, middle, and top parts of the plant can monitor larvae infestation. Careful monitoring using a combination of both methods can avoid unnecessary applications of insecticides.

Physical control

Yellow attracts leafminer fly adults. The use of mobile and stationary yellow sticky traps has been proved to effectively reduce leafminer fly populations, decreasing insecticide application costs by more than 50%.

Chemical control

Insecticides must be used only according to the monitoring results and when the leafminer population is expected to cause economic damage. Owing to frequent spraying, leafminer flies have become rapidly resistant to insecticides. Systemic insecticides with translaminar properties are most effective in controlling the larvae. The products ciromazine, abamectin and spinosad can be recommended. Ciromazine has a residual effect of more than 20 days, followed by abamectin and spinosad. Abamectin is most specific with fewer negative effects on beneficial insects.

Prospect for biological control

Parasitoids play a very important role in the natural regulation of leafminer fly populations but are often suppressed by insecticide applications. In fields where insecticides are applied, parasitism is low compared to non-treated ones. For example, in Peru, a complex of 17 larval parasitoids, with *Halticoptera arduine*, *Chrysocharis flacilla*, *Diglyphus websteri* and *Chrysocharis caribea* as the most abundant species, is involved in natural regulation. These observations stimulated worldwide biological control efforts and research and implementation, largely focused on the use of inundative and inoculative biocontrol for open-field and glasshouse conditions. Glasshouse applications have been mostly developed for horticultural industries in Western Europe and North America with *Dacnusa sibirica, Opius pallipes* and *Diglyphus isaea* as the most efficient parasitoids used. Classical biological control has been realized against *L. trifolii* and *L. sativa* with some notable achievements in vegetable production. Through the introduction and release of the cucoilid *Ganaspidium utilis*, in 1977 in Hawaii and in 1988 in Tonga, both leafminer species are successfully held in check, making chemical control unnecessary. Also, the fungal insect pathogen *Paecilomyces fumosoroseus* is being considered as a promising biocontrol agent. A natural mortality of 60% of leafminer fly adults was recorded in beans (*Phaseolus vulgaris*).

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PEACH POTATO APHID *Myzus persicae* (Sulzer),
POTATO APHID *Macrosiphum euphorbiae* (Thomas),
GLASSHOUSE POTATO APHID *Aulacorthum solani* (Kaltenbach),
BUCKTHORN POTATO APHID *Aphis nasturtii* (Kaltenbach)

APHIDS

Of the many aphid species that colonize potatoes, the peach potato aphid, (**162**), potato aphid, (**163**), glasshouse potato aphid, (**164, 165**) and buckthorn potato aphid (**166, 167**) are the most significant. Aphids are important pests of potatoes for two reasons. Firstly, they are effective vectors of viruses (especially *Myzus persicae*) and, secondly, they can cause direct feeding damage.

Symptoms

HAULM: When present in large numbers, aphids can cause wilting and a loss in yield by sucking sap from the leaves. This is most prevalent in crops under stress from a lack of water. Another foliage symptom resulting from feeding damage is top-roll. This symptom occurs on young, upper leaves. Light-green spots appear on affected leaves and precede an upward rolling. The leaf margins may develop a purple discoloration and become necrotic. When top-roll occurs tuber yields are usually affected.

ROOTS, STOLONS AND TUBERS: These are not normally affected by aphids.

Status of the pest

Aphids occur on potato crops throughout the world. Of the species found on potatoes, *Myzus persicae* is the most important because of its ubiquity and because it is the most effective vector of both persistent and non-persistent viruses. Its success in virus transmission is partly because it has highly specialized mouthparts but also because of its habit of continually probing leaves and moving from plant to plant. Other aphids (e.g. *Macrosiphum euphorbiae*) tend to be poorer transmitters of virus, although still significant because they occur in larger numbers. Due to their numbers, their direct feeding impact on a crop may also be greater. Large infestations have been found to reduce yield by more than 60%. Occasionally aphids can be found colonizing below-ground parts. One example is the bulb and potato aphid (*Rhopalosiphon latysiphon* [Davids.]), which infests stolons.

Life cycle and biology

Aphids exhibit a range of life cycles depending on species and climatic factors. They can be holocyclic, in which they have a sexual stage, or anholocyclic, in which they do not. Irrespective of the type of life cycle, aphids are also able to multiply parthogenetically. At different stages in their life cycle aphids may be winged (alate) or wingless (apterous). The complexity of life cycles is exemplified by *Myzus persicae*. In temperate climates, it can have a holocyclic or an anholocyclic life cycle. In the former, the aphids fly to a woody host (usually *Prunus* spp.) during the winter. Cold-tolerant eggs are laid in the bark after mating. These hatch into parthogenetic females in the spring. In turn these give birth to around 50 wingless progeny viviparously. Further wingless progeny are produced on the woody host before winged forms develop which move to secondary crop plants including potatoes.

On the secondary crop, generations of wingless aphids are produced parthogenetically. Sporadically, winged aphids develop that migrate within the field or to other crops. In response to declining day length and temperatures, winged males and females fly back to the primary woody host. The females produce sexual females, which mate with the males and lay eggs. In the anholocyclic life cycle, aphids remain active on weeds and crops. In this way, the development of large numbers on crop plants occurs earlier but they are more prone to low winter temperatures (a threshold of –10°C to –15°C). In some localities, individuals of *M. persicae* can occur that are neither holocyclic nor anholocyclic (androcyclic), in which a proportion of males contribute to the sexual phase. In warm climates, *M. persicae* is only anholocyclic. Aphids may be found in potato storage and chitting houses. Virus spread is possible, since the aphids can feed once sprouts develop.

Control

The degree to which control measures are applied depends on the aphid pressure and the market for the crop. Because minimization of virus is so important in seed crops, much greater effort is taken to prevent infestation. Growing seed in areas of low aphid numbers and where numbers of potato crops are limited achieves this. Statutory schemes of seed classification also impose strict virus tolerances on seed, which minimizes aphids picking up virus within a crop. In seed-growing areas, non-seed growing crops may also be subject to

162 Peach potato aphid *Myzus persicae.*

163 Potato aphid *Macrosiphum euphorbiae.*

164 Glasshouse potato aphid *Aulacorthum solani*.

165 *Aulacorthum solani*: close-up of proboscis.

166 Buckthorn potato aphid *Aphis nasturtii*.

167 *Aphis nasturtii*.

restrictions to reduce virus levels. Aphids have many predators including various beetles (e.g. ladybirds – Coccinellidae), hoverflies (Syrphidae), lacewings (Chrysopidae), wasps (Hymenoptera) and birds. Numbers of predators tend to follow the build-up of aphid numbers. The deliberate introduction of predators is one form of biological control. An example is the successful introduction of the European ladybird (*Coccinella septempunctata*) onto potatoes in New York State, USA.

Insecticides remain a major element of aphid control in developed countries where virus pressure is low. They are applied either as granules into the soil at planting or as foliar spray applications. The success of the method of application depends on the insecticide used and whether the virus involved is a persistent or non-persistent one. With the former, virus acquisition and transmission are slow. In consequence, insecticides have time to be effective. Non-persistent viruses are rapidly acquired or transmitted, and thus insecticides that repel aphids or prevent them feeding (e.g. pyrethroids) are used. Continuous use of insecticides often affects predator populations and can lead to a build-up of aphicide resistance. Multiple resistance to organochlorine, organophosphorus, carbamate and pyrethroid insecticides has been found in *M. persicae*. Targeted use of insecticides, by applying only when aphids reach a threshold level, can reduce insecticide use. Integrated control, where insecticides are used along with other control measures, and which take the population of predators into account, is difficult where a high-value crop is being grown.

Aphid resistance in wild potato species has been identified. Frequently, resistance relates to the 'hairiness' (relative abundance of trichomes) of leaves. However, breeding for resistance to viruses is likely to be more productive than resistance to aphids

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Ostrinia nubilalis (Hubner)

EUROPEAN CORN BORER

Symptoms

Haulm: Newly hatched larvae feed briefly on the leaf surface surrounding the egg mass from which they hatched. This feeding causes minor pitting and scarring on the leaf surface. Young larvae may tunnel into the midrib or petiole, but most often tunnel into main stems at the leaf axil (**168**). Larger larvae tunnel in stems. Damage results from feeding on the pith and vascular tissue by larvae tunnelling within stems. Points of larval entry into the stem can be recognized by an accumulation of brown or greyish-brown, sawdust-like faeces produced by the tunnelling larva (**169**). Foliage on damaged stems often wilts during periods of water stress. Damaged stems are readily broken by strong winds or by farm equipment moving through the field (**170**). There is evidence that feeding by European corn borer larvae predisposes the plant to bacterial soft rot caused by *Erwinia* spp. Extensive research has failed to demonstrate any suppression of tuber yield or specific gravity in the cultivar Atlantic when the infestation occurred during the bloom stage. Effects on yield in other potato cultivars have not been adequately investigated, and the possibility of reductions in yield or quality should not be dismissed in the absence of clear evidence. Heavily damaged vines may senesce prematurely, especially under hot, dry conditions.

Roots, Stolons and Tubers: Symptoms are not normally found.

Status of the pest

The European corn borer occurs throughout most of Europe and much of Asia and North America. It was first introduced into North America from Europe during the early 1900s and has since spread through most of the major maize-producing regions of the United States and Canada. The European corn borer reportedly attacks over 200 plant species and is a pest on a number of crops, including maize, peppers, green

168 European corn borer larva tunnelling in a stem at a leaf axil.

169 Accumulation of brown or greyish-brown, sawdust-like faeces produced by the tunnelling larvae.

170 Stem broken at the point of damage by European corn borer larvae.

beans, egg plant and potato. Among crops, maize is its preferred host. The importance of European corn borer as a pest of potato has not been adequately defined.

Life cycle and biology

Moths, which have a wingspan of approximately 25 mm, are active primarily at night. They are pale yellow to light brown, with irregular darker bands running diagonally across the wings. The female is lighter in colour and has a wider abdomen than the male. Eggs are deposited in white to cream-coloured masses of approximately 5–35 eggs on the undersides of leaves. Egg masses are covered with a shiny, waxy substance. Females may lay over 500 eggs during their lifetime. All eggs within a mass hatch simultaneously, typically in 3–12 days, depending on temperature, but may take considerably longer under cool conditions. A black spot develops at the centre of each egg shortly before it hatches. The newly hatched larva is approximately 1.5 mm long. It has a black head and a pale yellow body bearing several rows of small black or brown spots and five pairs of prolegs. The larvae develop through five instars and reach a length of about 25 mm at maturity. Larval development varies with temperature, but in potato typically requires approximately four weeks to complete. Pupation occurs within the stem of the host plant. Pupae are 13 to 15 mm long and have a smooth, brown, capsule-like body.

European corn borers overwinter as diapausing 5th instar larvae within the stems of host plants. There is little relationship between overwinter survival and population size the following season. The emergence of moths of the overwintered generation is determined by spring temperatures and typically occurs over a period of four to six weeks. Emergence may begin as early as mid-March or as late as early July, depending on temperature and location. In North America there are three known ecotypes of the European corn borer that differ in number of generations per year (1-, 2-, and multi-generation ecotypes) and there are two strains that differ in the blend of chemicals produced by females as a sex pheromone to attract males. More than one ecotype and sex pheromone strain may occur in the same locality, complicating our ability to predict the dynamics of local populations. Typically, however, only one generation of European corn borer occurs in potato each year; any additional generations occur in other crop and weed hosts, most often maize.

Control

Monitoring

Populations of European corn borer moths can be monitored using blacklight traps or sex pheromone-baited traps to determine when fields should be scouted. Blacklight traps are very effective monitoring tools but require a source of power. In addition, because they attract a large number of insect species, considerable time must be spent sorting through the insect catch to identify corn borer moths. Sex pheromone-baited traps have neither disadvantage. However, they must be placed correctly and the sex pheromone blend used as bait must be appropriate for the population of moths in the area to be monitored. Traps should be located within the potato field or in unmowed patches of grass along the field margin. They should not be placed in areas having no vegetation or in grassy areas that are kept mowed. The effectiveness of pheromone traps is influenced by trap height. Traps should be positioned so that the entrance to the trap is slightly above the top of the plant canopy. Field scouting should begin when trap catches indicate significant moth activity.

Because the abundance of egg masses is not a reliable predictor of larval infestations in potato, reliable control decisions cannot be based on egg-mass abundance. Control decisions are most reliable when based on the incidence of European corn borer-infested stems. Scouting entails examining leaf axils and petioles for evidence of entry by small larvae. Stems are classified as infested if they contain evidence of larval entry into stems or petioles; otherwise they are classified as non-infested. A typical sampling plan used by commercial scouts entails examining 10 stems in each of 10 sample sites located randomly in an area 30 hectares or less. Where a high level of control is desired, insecticide treatments are recommended when 5–10% of the stems are classified as infested. A sequential sampling system that is easy to use and more efficient than the sampling plan described is also available for use in timing the application of control measures. European corn borer infestations tend to be highest in potato plantings having the largest plants and the most well-developed crop canopy at the time of oviposition. Consequently, early plantings tend to be at the greatest risk of heavy infestations, and it is advisable to give these plantings priority in scouting.

Chemical control

Several foliar insecticides are available. Applications are most effective when based on thresholds for the area and directed against newly hatched larvae. Applications directed against larger larvae tunnelling in stems are ineffective. In general, applications made using ground equipment are more effective than are aerial applications. A single, well-timed application of an effective insecticide will typically provide acceptable control, except during cool seasons when oviposition and egg hatch occur over an unusually long period. In such cases, two applications may be necessary. The need for this should be determined by continued scouting following the initial application. Spot treatments with foliar insecticides are not advised. In the absence of scouting, insecticide applications should be made every 7–10 days during periods of significant moth activity, as indicated by light or sex pheromone-baited traps.

Insecticide resistance has not been documented in European corn borer populations, but applications to control corn borer have the potential to select for resistance in local Colorado potato beetle populations. Decisions regarding the use of insecticides to control European corn borer should be made with this in mind.

Cultural practices

Because maize is a preferred host of the European corn borer and large populations can overwinter in maize, isolation of potato plantings from maize can reduce the risk of high infestations in potato. Similarly, because the moths prefer crops with well-developed canopies, later planted fields of potato have a lower risk of heavy infestations than early planted fields. No commercial potato cultivars are currently known to be resistant to European corn borer, although Atlantic is extremely tolerant of injury caused by corn borer infestations that result from egg hatch during the bloom stage.

Other factors in population suppression

An array of predaceous insects and parasitoids attack European corn borer and are capable of causing extensive mortality, especially in eggs and small larvae. However, these natural enemies cannot be relied upon to prevent the development of heavy larval infestations. Newly hatched larvae are extremely sensitive to cool, wet conditions. Prolonged periods of cool wet weather may prevent infestations from becoming established.

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Phthorimaea operculella Zeller
(syn. *Gnorimoschema operculella* Zeller)

POTATO TUBER MOTH

Symptoms

Haulm: Mining of the main veins, petioles or young shoots can occur caused by the larvae eating the mesophyll without damaging the upper and lower epidermis (**171**). Larvae also enter into leaf axils and growing points of young plants. This can result in wilting and premature senescence of leaves.

Tubers: Larvae enter tubers through the potato eyes. They can mine into tuber flesh forming irregular cavities and galleries (**172, 173**). These may run into the interior or remain directly under the skin. Excrement (frass or detritus) lines the cavities as a result of the consumption of the tuber tissue. The mining produces weight losses of tubers, which is exacerbated by increased transpiration through the wounds, causing tubers to shrink. The wounds provide entry points for microorganisms and cause secondary infection, particularly by species of *Penicillium*. Heavily infested tubers, and those with secondary rotting, can render tubers unsuitable for human consumption. Besides losing value for feed, damage to the eye can reduce sprouting potential. At harvest, tubers do not always show signs of damage but may harbour eggs and early instar larvae. This may result in severe post-harvest losses of stored potato in the absence of adequate control measures.

Status of the pest

The potato tuber moth originated in tropical mountainous regions of South America. Today it has a worldwide distribution and is considered the most damaging potato pest in the developing world. It is present in almost all tropical and subtropical regions of the world, in North, Central and South America, Africa, Asia, Australia, and Europe. In many regions the moth is mainly considered as a storage pest of potato, with losses of up to 100%. Either the pest is transferred to the stores within already infested tubers or adults enter storage facilities. The severity of tuber infestation and consequently the quality loss of harvested tubers are closely related to the population build-up of the moth during the potato-growing season. Yield losses of up to 45% are therefore largely the result of early tuber

171 Mining of young shoots by larva of potato tuber moth.

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172 Tuber damage by larvae of potato tuber moth.

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173 Tunnels in tuber produced by larvae of potato tuber moth.

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174 Eggs of potato tuber moth.

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175 Pupa of potato tuber moth.

infestation in the field, generally where females are able to directly deposit eggs on tubers through soil cracks. Leaf infestation becomes highest when potato is cultivated during warm dry seasons, especially under furrow irrigation. Mean temperatures of more than 20°C favour a quick population development. Under these conditions, the highest plant damage has been observed in the Republic of Yemen, with more than 30 mines per plant that directly reduces the productivity of the plant and causes yield losses. Heavy rain or regular sprinkler irrigation influences the flight activity of adults and limits infestation. In rain-fed potato crops in the Andean highlands leaf infestation is low, but delayed harvest during the dry season increases tuber infestation substantially.

Life cycle and biology

Adults are small brownish-grey-coloured moths (7–9 mm), with fraying on the posterior edge of the forewings and on both posterior and inner edges of the hind wings. At rest the wings are folded to form a roof-like shape. The wingspan is 12–16 mm. The tip of the female's abdomen is cone-shaped, whereas the males possess two claspers at the hairy tip of their abdomen. The male's sexual organs are situated in the middle of the ninth abdominal segment, the females in the middle of the eighth. The sexual pheromones by which females attract males have been isolated and identified as trans-4, cis-7-tridecadien-1-ol acetate (PTM1) and trans-4, cis-7, cis-10-tridecatrien-1-ol acetate (PTM2). A mixture of the two is far more effective than one component used by itself.

Potato tuber moth eggs are too small (0.5 × 0.35mm) to be visible to the naked eye on potato leaves and tubers. The freshly laid eggs are cream-coloured and deposited singly or in small clutches resembling strings of beads (**174**). As they develop, they take on a yellowish tinge and, before hatching, the black head capsule of the tiny larva can be seen through the thin eggshell. Of all the host plants, the females prefer to lay eggs on potato. The eggs are laid on the undersides of leaves, on stems, and on the tubers, the eyes in particular. The females lay between 100 and 300 eggs, depending on temperature and food availability, with a sex ratio of 1:1. The first instar larva (L1) is barely 2 mm long, while the fourth instar larva (L4) measures 9–13 mm before pupation (**175**). The colour of the larvae depends on their diet. In tubers they are a whitish-purple, but those on the potato leaves are purple to green. A fully developed larva has six ocelli on each side of the head, mouth parts with a silk gland, a prothoracic and an anal plate, nine pairs of spiracles, and five pairs of prolegs on abdominal segments III – VI and X. The full-grown larva usually leaves the plant or tuber to pupate, which takes place either in the soil or on tubers. The pupa reaches 6–7 mm. At first it is brownish in colour, turning to dark brown one day before the emergence of the moth.

The potato tuber moth can adapt to the most diverse climatic conditions. Development is possible within a temperature range of between 10°C and 35°C. The population growth rate peaks at temperatures of between 20°C and 25°C. At a constant temperature of 26°C, the development of eggs takes four to five days; the larval stage is completed within 12 days and the pupal stage within six to seven days. Daily fluctuating temperatures having the same mean temperature do not affect development times differently. In Yemen, the moth developed eight generations over a year, while for the Andean highlands only three to four generations were determined. The potato tuber moth can survive low temperatures for short periods at all developmental stages. The species does not respond to unfavourable conditions by entering diapause. For development to continue, short spells of higher daily temperatures are necessary. The moth survives intervening periods on leftover potato or by adults re-colonizing fields after storage. Most importantly, infested tubers often used as seed in developing countries initiate colonization in the next growing season. Certain weeds can also serve as hosts, but it has been observed that they are rarely infested.

Many cultivated and wild species of the family Solanaceae are hosts: *Solanum tuberosum* (potato), *S. melongena* (aubergine), *S. incanum* (apple of Sodom), *S. muricatum* (melon pear, sweet cucumber), *S. aviculare* (poroporo), *S. nigrum* (black nightshade), *Nicotiana tabacum* (tobacco), *Lycopersicon esculentum* (tomato), *Datura stramonium* (thorn apple), *Physalis peruviana* (Cape gooseberry), *Capsicum annum* (paprika), *Hyoscyamus albus* (henbane). Only a few host plants are described from other families, e.g. *Beta vulgaris* (beetroot) (Chenopodiaceae).

Control

Before the advent of insecticides, a number of cultural practices were used to reduce the impact of potato tuber moth. These included crop rotation, deeper planting of seed, and continual ridging up. The latter two are effective, as tubers closest to the surface are most affected. Today, in view of the pattern of damage development, control of the potato tuber moth must take place both in the field and in storage. Farmers tend to revert to the use of highly toxic chemicals to control this pest. However, it has been shown that, with the implementation of integrated pest management, the problem can be economically reduced, in both field and store, in an environmentally friendly way.

Control of leaf and tuber infestation during potato cultivation

- **Use of healthy non-infested seed** prevents re-colonization.
- **Timely planting.** Based on the seasonal occurrence of the pest, planting is best done in seasons or at temperatures that do not favour moth development. Pheromone traps can best be used to monitor the flight activity of the moth all year round.
- **Deep planting.** Planting seeds to a depth of 10 cm reduces the possibility of females laying eggs in newly developed tubers.
- **High and regular hilling (earthing-up)** protects developing tubers from ovipositing females and reduces the possibility of larvae reaching bulking tubers. High hilling can reduce damage by 30%.
- **Adequate irrigation** prevents soils from cracking and allowing females to reach potato tubers for oviposition. Sandy soils are less vulnerable to cracking than clayish soils. Sprinkler irrigation alone reduces damage by 30%.
- **Regular monitoring of field infestation.** Depending on the capability of the moth to build up a high population during the potato-growing season, either leaf infestation can directly cause yield losses or tuber infestation will be increased at harvest. The control threshold concept should be applied to predict the necessity and right time for chemical control; e.g. for the Republic of Yemen a control threshold of one mine/two potato plants at growth stage 20–30 has been determined. A sequential sampling procedure can be used to monitor leaf infestation.
- **Timely harvesting.** During the last crop growth phase (tuber filling and plant senescence), the infestation rate of tubers accelerates. Delaying harvest by 1 to 2 months can increase tuber damage by as much as 70%.
- **Protect tubers after harvest.** Harvested tubers should not be exposed to ovipositing females overnight. If they cannot be stored immediately, tubers should be covered; otherwise the infestation rate can increase rapidly within a few days.

- **Avoid leftover potatoes and destroy crop residues.** *P. operculella* complete larval development in tubers and dry stems left in the field. Moths from these pupae may infest the crop the following season. Also, tubers left in the field are additional food sources and become volunteer plants in the next season.

Reduction of tuber damage in potato stores

- **Cleaning stores and physical control.** Stores should be carefully cleared of old potatoes and should be totally moth-free to avoid re-infestation. Furthermore, the entrance of adults should be prevented by using screens at windows and doors.
- **Storing healthy tubers.** Tubers should be sorted and only healthy non-infested ones stored. Tubers exposed to moth oviposition should not be stored, as eggs are commonly overlooked during sorting. Storing infested tubers or those that have been exposed to moth oviposition along with healthy tubers may result in the infestation of the entire stock within some months.
- **Using *Po*GV granulovirus.** The *Phthorimaea operculella* granulovirus (*Po*GV) applied before storage as a powder formulation at a dose of 5 kg/t of tubers is most effective and used in Bolivia and Peru. Besides its direct effect, the powder formulation, consisting of inert material (talcum), provides a physical protection. In situations where the Andean potato tuber moth (*Symmetrischema tangolias*) co-exists, a mixture with *Bacillus thuringiensis* (*Bt*) or *Bt* alone should be used. If potatoes are stored over a long period, developed young sprouts are not protected.
- **Repellent plants**. The foliage of plants rich in essential oils, such as *Eucalyptus* spp., *Lantana camara*, *Schinus molle* or *Minthostachys* spp., repels adult moths and can be used for covering potato tubers.
- **Use of pheromone traps.** Pheromone traps may be used to monitor the moth population and to help farmers to recognize the presence of moths in storage.

Transgenic potatoes have been developed with resistance to potato tuber moth.

Prospects for biological control

Parasitoids of the families Braconidae, Encyrtidae and Ichneumonidae are major natural enemies of the potato tuber moth. Many polyphagous species have accepted the moth as an additional host in regions where the moth has been introduced. Classical biological control dates back to the 1940s to establish 18 different parasitoids by mass release in at least 20 countries. Among those parasitoids that were established in more than one country were species of the family Braconidae, including *Apanteles subandinus* Blanch., *Orgilus lepidus* Mues., and *Bracon gelechiae* Ashm., and from the Encyrtidae *Copidosoma koehleri* Blanch. Apart from *B. gelechiae*, which originates from North America, all the others are native to South America. A granulovirus of *P. operculella* (*Po*GV) that naturally infects the moth population has been isolated in many countries. While its efficacy under field conditions is often too low due to a rapid ultraviolet-inactivation it has been developed into a biopesticide for potato tuber moth control in storage. *Bacillus thuringiensis* is less effective in fields but highly recommendable for storage control. The use of genetically modified *Bt* potatoes is not recommended in all countries and regions where potato is native. In general, the potato tuber moth can be controlled effectively by IPM. Therefore, considering potential risks, the usefulness and deployment of *Bt* potato has to be well thought out.

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***Premnotrypes* spp. (syn. *Trypopremnon* [Pierce], *Solanophagus* [Hustache], *Plastoleptops* [Heller])**

ANDEAN POTATO WEEVIL

Symptoms

Haulm: Adults feed on leaf margins causing characteristic half-moon feeding damage; in general this damage is not significant but indicates the presence of weevils in potato fields.

Roots and Stolons: None.

Tubers: The larvae produce deep galleries (**176**), lowering tuber quality and rendering tubers useless for consumption or use as seed. On emerging from the tubers, larvae produce characteristic circular holes. Larval development may be completed in the field before harvest, otherwise weevils will complete their life cycle in potato stores.

Status of the pest

Andean potato weevil larvae can complete their development only on potatoes; adults can occasionally feed on other plants. Andean potato weevils (APW) are the most serious pests of potatoes (*Solanum* spp.) at high altitudes (between 2,800 and 4,700 m) in the Andean region, where wild and cultivated potato species are their host plants. They are distributed from Argentina to Venezuela, covering a mountain territory of about 5,000 km in length. Without control, tuber damage can be up to 50–80% at harvest. In commercial production, highly toxic insecticides are usually applied at planting as soil treatment or post-emergence on potato foliage.

Although species of the genus *Premnotrypes* are the most important and widely distributed Andean potato weevils, the first taxonomic descriptions were made in the year 1824 for the genera *Phyrdenus,* and in 1906 for the genus *Rhigopsidius*. All species of both genera attack potato and are native to the Andes. Pierce (1914) established the genus *Premnotrypes* and described two species from infested tubers in Peru. Today 12 *Premnotrypes* species have been described: *P. solani* Pierce, *P. latithorax* (Pierce), *P. vorax* (Hustache), *P. solanivorax* (Heller), *P. fractirostris* Marshall, *P. sanfordi* (Pierce), *P. clivosus* Kuschel, *P. zischkai* Kuschel, P. *pusillus* Kuschel, *P. solaniperda* Kuschel, *P. suturicallus* Kuschel and *P. piercei* Alcalá. Since most of the species occur in Peru and Bolivia, these countries are considered as the center of origin. Ten of twelve *Premnotrypes* species have been identified in Peru. The most widespread and damaging species are *P. vorax*, *P. latithorax* and *P. suturicallus,* which all have a similar behaviour.

Life cycle and biology

Adult weevils are dark brown ground beetles 6.0–9.2 mm in length (**177**). Most species have tubercles or sculpturing on the elytra, and in some the abdomen is

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176 Tuber damage by Andean potato weevil.

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177 Adult Andean potato weevil.

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178 Larva of Andean potato weevil.

squarely truncated posteriorly. Larvae (**178**) and pupae are typical for Curculionidae: 6.2–10 mm in length and whitish in colour. The eggs are 1–1.5 mm long.

During the day, male and female adults remain hidden beneath soil clods, stones, dry leaves or any other shelter, including soil cracks near the potato plant. In the evening, weevils climb to the foliage to feed and mate. Females lay eggs inside straw or other plant debris near potatoes. As the larvae hatch, they seek potato tubers. Larvae feed in tubers until they have completed their larval development, leaving them as the fourth instar larva before pupation. The larvae prepare pupal cells in the soil, in which they hibernate. Emergence of the adults starts with the onset of rains.

In the high Andes of Peru and Bolivia, with one potato crop per year, species of the genus *Premnotrypes* develop one generation per year. Development is well synchronized with climatic conditions, the cropping system and the phenological development of potato. The following developmental times have been reported: egg (26.5–47.7 days), larva (59.2–117.9 days), prepupa (26–46.6 days), pupa (28.5–54.4 days), hibernating adult (65.9–134.6 days), longevity of active adults (159.2–276.8 days) and total life cycle (424–499.3 days). In Ecuador and Colombia, *P. vorax* develops more than one generation per year when potato cropping is year-round. Weevils remain in the field if potato cropping continues in the next season, or they migrate to new potato fields. When immigration occurs, infestations are more pronounced at field borders. In Peru, adult emergence lasts from the end of October until the end of February. Weevils migrate into potato fields between December and February. The highest adult populations are observed in February. Larval tuber damage starts at the middle of March and increases until April. Pre-pupae occur from April to September, pupae from May to October, and hibernating adults are found from June to November. Hibernating adults are not active while in the process of chitinization, changing colour from yellow to dark brown or black.

Control

IPM strategies for APW aim to reduce the weevil population in fields, interrupt the migration of weevils to new fields and reduce the hibernating weevil population.

Reducing field infestation

- **Early planting.** The emergence of adults coincides with the onset of the rainy season. As more adults emerge, there is a continuous increase in the number of females depositing their eggs. Early planting and the use of early-maturing varieties reduce weevil infestation and damage.
- **Timely harvest**. Delayed harvest extends tubers' exposure to weevils and increases infestation.
- **Handpicking of adults.** Adult weevils in the potato field are active in the evening, climbing the foliage for feeding and mating. They can be captured easily by shaking the foliage over a container. Farmers have adopted this practice.
- **Avoidance of monocropping and destruction of volunteer plants.** Weevil infestation is highest when one potato crop is followed by another potato crop. Crop rotation with any other crop, or a fallow period, should be practised. Larger distances between new and former potato fields also reduce the probability of weevil infestation because of a limited migration capability. Further, volunteer potato plants in rotations are significant sources of weevil infestation. It is therefore recommended that they be removed.

Interrupting adult migration and larval movement for pupation

- **Barriers.** Migratory weevils can be caught and controlled by insecticides applied in peripheral trenches around potato fields. Also, band spraying with pesticides within the first two metres of potato fields

has been recommended. Early planting of field borders with other Andean crops as a vegetal barrier (tarwi, *Lupinus mutabilis* or mashua, *Tropaeolum tuberosum*) also disrupts the migration of weevils into fields.

- **Shelter traps.** Weevils hide in the shade during the day. Straw bundles, pieces of jute (sisal), or plastic sheets and other materials can be used to provide shelter to weevils, facilitating their capture. An alternative is to treat potential shelters with insecticides (potato foliage collected from volunteer plants can be sprayed and placed under the shelter). These shelter traps are particularly effective prior to the emergence of potatoes.
- **Ground sheeting.** At harvest, full-grown larvae leave the tubers to pupate in the soil. Larvae can be intercepted on their way to the soil by placing harvested tubers on plastic sheets or other materials. The collected larvae can then be fed to chickens or killed.

Reducing hibernating weevils

- **Winter ploughing.** Ploughing fields two or three months after harvest can destroy large numbers of larvae and pupae. Chickens can also help destroy exposed larvae and pupae. Abandoned potato fields are a major source of weevil reproduction and migration. Ploughing the soil during winter can also reduce the development of volunteer plants.
- **Control of hibernating weevils under former potato piles.** If sheets are not used at harvest, large numbers of larvae dig into the soil. This also occurs during sorting potatoes prior to storage. Sites where potatoes have been piled should be marked, and two to three months later underground larvae and pupae should be destroyed to reduce sources of new infestation. Small-scale farmers have adopted the control of hibernating weevils under former potato piles because those are covering small areas near houses. There are good examples from Taracollo, Bolivia and Cusco, Peru.

Prospects for biological control

Ground beetles (*Harpalus turmalinus* and *Hylitus* spp.; both Carabidae) and *Metius* spp. (Tenebrionidae) are predators of eggs and larvae. Further, ants (*Iridomirmex* spp.; Formicidae) that predate on larvae have been described. Important entomopathogenic fungi are *Beauveria brongniartii* and *Metharrizium anisopliae* that infest larvae, pupae and adults of Andean potato weevils. Recently, a native entomopathogenic nematode (*Heterorhabditis* sp.) attacking the larvae of Andean potato weevil has been reported from Peru.

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Symmetrischema tangolias (Gyen), (syn. *Phthorimaea plaesiosoma, Symmetrischema plaesiosema* [Turner])

ANDEAN/SOUTH AMERICAN POTATO TUBER MOTH

Symptoms

Haulm: The larva enters the stem, making a small hole in the plant axils (between stem and lateral petioles). From this hole, galleries made by the larvae run downward within the stem. Excrement is pushed out through the initial hole. When stems are severely damaged the upper part of the stem may wilt or the whole plant collapses (**179**). Young plants can suffer tip death from boring larvae. Eggs may be found in slits on the stem of a food plant.

Roots and Stolons: None.

Tuber: Larvae enter tubers through potato eyes. Initially, the small hole can hardly be seen by the naked eye. Larval excrement is pushed out through the hole, which becomes apparent after several days of mining activity. In the tuber, the larva tunnels just under the surface at first, but later penetrates more deeply. If infection is severe, i.e. after a longer storage period, the whole tuber may be eaten out, leaving only debris (**180**). Infested tubers develop a bitter taste and are unsuitable for human or livestock consumption. Mining of the larvae and increased transpiration through the wounds cause tuber weight loss and tuber shrinking. Wounds also provide entry points for microorganisms that cause secondary infections.

Status of the pest

The Andean potato tuber moth (PTM), probably originating in the mountainous region of Peru and Bolivia, has become a serious pest of potatoes in the Andes of South America during the last few decades. Especially in Peru and Bolivia, *S. tangolias* has become an economically significant species of the PTM pest complex, which attacks potatoes in both field and storage. Today, in many mid-elevated valleys and highlands *S. tangolias* has displaced the common potato tuber moth *P. operculella* as the most important PTM. In some zones the two species coexist, but the species-specific interaction and competition are not yet well understood. In Australia and New Zealand, where the pest was accidentally introduced from South America, this microlepidopteran is more recognized as a pest of tomato, poroporo, sweet cucumber and other solanaceous crops and commonly referred to as 'tomato stem borer'.

In the Andes, the extent of damage by this pest varies depending upon the storage methods and environmental conditions. Compared with other species of the potato tuber moth complex, *S. tangolias* is more adapted to cooler conditions. It prevails in mid-elevated regions of the Andes (Peru, Bolivia, Ecuador and Colombia), but is not a pest at low altitudes, where *P. operculella* develops several generations per year. Damage in the field may reach up to 30%, but the most economically significant damage occurs when infested tubers are transferred to potato storage, where re-infestation takes place. There are areas where farmers lose all of their stored potatoes within three to four months of the initial infestation if it is not controlled.

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179 Potato plant damaged by *S. tangolias* (infested potato plant, attacked stems and leaves).

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180 Potato tuber attacked by *S. tangolias.*

Sources of infestation are potato stores and potato fields, especially those fields where potato residue is allowed to remain after harvest. Moths commonly move in both directions. Field-infested tubers, or tubers infested at harvest, carry initial infestations to stores. On the other hand, the use of infested seed contributes significantly to initial infestation in potato fields.

Host plants of the Andean potato tuber moth are: *Solanum tuberosum* (potato), *S. muricatum* (melon pear, sweet cucumber), *S. muricatu*m (pepino), *S. aviculare* (poroporo, New Zealand nightshade, kangaroo apple), *Lycopersicon esculentum* (tomato).

Life cycle and biology

The adult moth is brownish-grey (silver-grey), with characteristic black triangular spots at the lateral edges of the forewings (**181**). Fine hairs fringe the edges of the forewings, while the hindwings, which are shorter and narrower than the forewings, are bordered with pale ochre scales. The length of the resting moth is 9–12 mm, while the body length ranges from 6–9 mm. Wingspan is 18–19 mm. Males are smaller than the females with a cone-shaped abdomen, while the female abdomen is wider with a blunt ending.

Adult females lay 170 to 185 eggs, attaching them to the host plant. In the field, over 90% of the eggs are deposited – singly or in small clutches – into leaf axils of host plants. In storage, eggs are laid on potato tubers. Freshly laid eggs are whitish and oval (0.7 × 0.4 mm). During embryogenesis, eggs become orange-yellow and turn dark grey shortly before larval hatching. The larva passes through five larval stages. Emerging larvae (L1) measure just about 1 mm in length, while L5 reach 13 mm before pupation. The coloration of the larvae depends on the feeding sources. Larvae mining in potato tubers appear whitish-beige, and those mining in plant stems are greenish (both with a dark head capsule). Characteristic for *S. tangolias* larvae from L3 on are three reddish longitudinal stripes found on the upper part of the thorax and abdomen (**182**).

S. tangolias makes characteristic tunnels within the tubers and stems. Full-grown larvae usually leave the feeding medium and spin a silken cocoon in some convenient place on the plant epidermis or among the debris of the attacked plant. Pupation also occurs inside potato tubers and plant stems more frequently than with *P. operculella*. Pupae are 7–8 mm long, first brownish in colour, later turning dark brown and then almost black one day before emergence.

The life cycle depends strongly on prevailing temperature. Development is possible within the temperature range of less than 10°C to approximately 24°C. It seems that *S. tangolias* is more adapted to cooler temperatures that the common PTM *P. operculella*. In the Andean regions *S. tangolias* may develop three to five overlapping generations a year. The insect does not show a hibernation period. Depending on temperature, egg development takes around 10 days at 20°C to 17 days at 13°C. Larval development lasts 30 to 60 days at 20°C and 13°C, respectively, while pupal development takes approximately 18 and 32 days. Immature mortality was noted to be very high (approximately 75%), but under the different climates potatoes are grown the influence of abiotic factors on the presence

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181 Adult Andean potato tuber moth: brownish-grey with a dark mark on each forewing.

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182 Andean potato tuber moth larvae.

and severity of *S. tangolias* is still not clearly understood. After a preoviposition period of several days, adult females lay new eggs. Therefore, the whole cycle from egg to egg, in most mid-elevated zones within the Andes, may last over 100 days depending on environmental conditions.

Control

Farmers commonly use organic insecticides to control *S. tangolias*, including highly toxic organophosphorous compounds, chlorinated hydrocarbons, carbamates (methomyl) and pyrethroids. The usual practice is to target the pest in storage, using chemical dust formulations. However, spraying, dipping or fumigating tubers is routine in several countries. The most widely used fumigant is aluminium phosphide (phostoxin). This unilateral use of chemical pesticides often leads to serious problems of pest resistance and accumulation of residues in potatoes. Fortunately, there is an increasing awareness of the need to reduce the harmful effects of pesticides on the environment and a growing interest in safer alternatives through the development of integrated pest management (IPM). Hence, strategies to minimize or even exclude the use of insecticides for potato tuber moth management have been investigated.

Various IPM components have been designed to reduce pest movement among fields and stores and to lower larval and adult moth densities:

Measures to protect the potato crop from planting to harvest (primarily cultural control methods)

- **Healthy uninfested** *seed* inhibits or may prevent initial infestation in the field.
- **Timely planting.** Based on the seasonal occurrence of the pest, optimum planting dates have been determined to coincide with lower temperatures and the onset of rains. Farmers who plant in the dry period face high moth infestations.
- **Deep planting.** Covering tuber seed to a depth of 5–10 cm prevents female moths from ovipositing in seed tubers and keeps larvae from migrating to tubers from infested above-ground sprouts. Neonate larvae of *S. tangolias* can burrow to a depth of 5 cm; those of *P. operculella* as much as 10 cm to the seed tubers.
- **High hilling.** High hilling (earthing up) of growing plants protects the developing tubers from ovipositing females and reduces the possibility of larvae reaching the bulking tubers. High hilling can reduce damage by 30%.
- **Frequent irrigation.** Adequate watering and cultivation prevent soil cracking. Soil cracks allow female moths to reach the potato tubers for oviposition, and provide shelter for adult moths. Sprinkle irrigation alone reduces damage by 30%.
- **Pheromone traps.** Commercial pheromones are available for *S. tangolias*. Mass trapping of male moths reduces the probability of moths mating, thus causing a drop in egg fertility. Pheromone traps can reduce infestation by 50%, but due to relatively high prices of the pheromones this approach does not seem to be economically feasible.
- **Early harvest.** During the last phases of the crop (tuber filling and plant senescence), the infestation rate accelerates. Delaying harvest by one or two months can increase damage by as much as 70%.
- **Protect harvested tubers.** Harvested tubers should not be exposed to ovipositing females overnight. If they cannot be stored immediately, tubers should at least be covered; otherwise the infestation level could reach 60% within a few days.
- **Avoid left-over potatoes and destroy crop residues.** *S. tangolias* (to a larger extent than *P. operculella*) pupate in tubers and dry stems left in the field. Moths from these pupae infest the crop the following season. Also, tubers left in the field become volunteer plants in the rotation crop.

Measures to avoid damage in stores (protecting stored tubers can reduce moth damage by 70–95%)

- **Storage hygiene.** Cleaning floors, walls and ceilings of rustic stores before storing healthy tubers destroys pupae and other life stages of the moth.
- **Storing healthy tubers.** Tubers should be sorted and infested ones discarded before storing. Tubers exposed to moth oviposition should not be stored, since eggs are commonly overlooked during sorting. Storing infested tubers, or those that have been exposed to moth oviposition along with healthy tubers, may result in infestation of the entire store in as little as four months.

- **Bacillus thuringiensis (Bt).** A dust formulation of *Bt* at a dose of 5 kg/t tubers has recently been under investigation. Besides the effect of *Bt* the inert material (talcum) provides physical protection. Tubers should be treated before storage.
- **Repellent plants.** The foliage of some plants rich in essential oils, such as *Eucalyptus* spp., *Lantana camara*, *Schinus molle* and *Minthostachys* spp., repels potato moths. The leaves are dried under shade, crushed, and then used to cover the stored tubers. On average, protected tubers are 70% less damaged than non-treated tubers.
- **Pheromone traps.** Commercial pheromones disrupt mating of the moth during storage and catch male moths. Tubers stored with pheromone traps are about 95% less infested than the control stores.
- **Diffused-light stores.** Potatoes stored in diffused light are generally about 70% less infested than those stored in the dark. Illumination results in greening of the tubers (with glycoalkaloid formation), which is unfavourable for the moth. In addition, the arrangement of tubers in diffused-light stores facilitates the periodic elimination of damaged tubers.

Prospects for biological control

The role of parasitoids on *S. tangolias* populations has not been studied in detail yet. In Peru, species of *Macrocentrus* and *Apanteles* (Hymenoptera: Braconidae) have been observed attacking *S. tangolias*. Furthermore, *Copidosoma koehleri* Blanchard (Hymenoptera: Encyrtidae), a parasite of the common potato tuber moth (*Phthorimaea operculella*) also attacks *S. tangolias*. The use of entomopathogenic fungi (i.e. *Bauveria* spp., *Metharhizium* spp.) does not seem to be a feasible approach for controlling *S. tangolias* because of relatively dry climatic conditions in the Andean highlands. While *P. operculella* can be controlled by a highly species-specific granulovirus (*Po*GV), a search to find a species-specific baculovirus infecting *S. tangolias* which could be produced alongside *Po*GV in Andean countries, failed. Recent research to manage the pest focuses on the use of *Bacillus thuringiensis* Berliner.

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Tecia solanivora **(Povolny)**
(syn. *Scrobipalpopsis solanivora* [Povolny])

GUATEMALAN POTATO TUBER MOTH

Symptoms

Haulm, Roots and Stolons: None.

Tubers: Initially larvae feed just under the surface but later they create deeper galleries (**183**). Externally, the tubers show almost no symptoms of damage until larvae leave the tubers to pupate. The galleries are filled with larval excrement and exuvia. The larvae leave the tubers by creating a circular hole 2–3 mm in diameter with regular edges, which is characteristic for this pest.

Status of the pest

Tecia solanivora probably originates from Guatemala and is endemic throughout Central America. In 1983, the pest was unintentionally introduced into Venezuela by contaminated seed imported from Costa Rica. From there, it invaded Colombia two years later. In the absence of natural enemies, it rapidly established itself in both countries in mountainous areas between 1,350 and 3,000 metres above sea level, where it developed huge populations. It is reported that in many cases half of the crop is lost and all stocks are infested and destroyed. In Ecuador, *T. solanivora* was first noticed in 1996. It spread quickly through trade movements into the country's interior. In 2000, the moth also reached the Canary Islands (Tenerife). Since then the pest is

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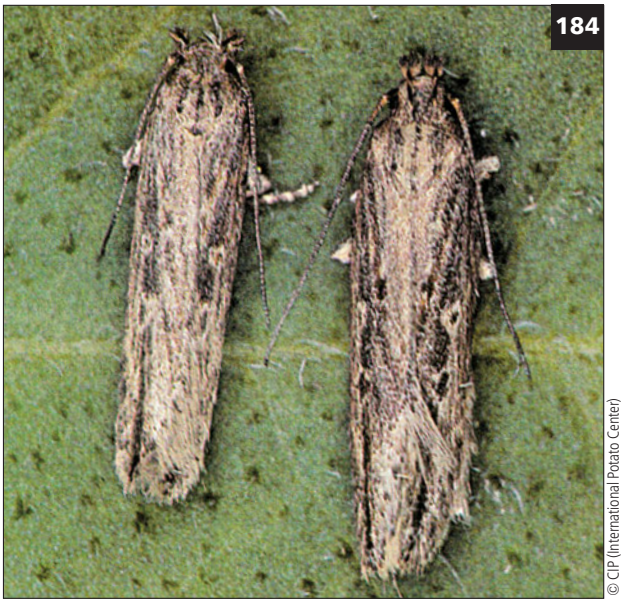
183 **Tuber damage by Guatemalan potato tuber moth.**

considered to be a major threat to potato crops throughout southern Europe and is listed as a quarantine pest by the European and Mediterranean Plant Protection Organization. No presence of the moth has been reported from Peru. However, uncontrolled trade in potatoes between Ecuador and Peru is frequent, posing a potential risk for a further expansion of the pest. Peru is therefore taking all possible measures in their quarantine programme to keep the pest out of the country.

Life cycle and biology

The adult is brownish, with longitudinal dark lines running from base to apex (**184**). Females and males measure on average 13 × 3.4 mm and 9.7 × 2.9 mm, respectively. Additionally, both sexes can be differentiated by the fact that females have three stains or marks on each wing, whereas males only have two. The adults are active during twilight and the first night hours. During the day, they remain in dark sites, hidden under foliage or plant stubble. Adults are able to fly relatively long distances. The sex ratio is 1:1. Females attract males through sex pheromones which have been identified as (E)-3-dodecenyl acetate and (Z)-3-dodecenyl acetate. Copulation may start one day after hatching.

Eggs are placed in groups around stems in the soil in the first day of oviposition. Afterwards, they are deposited more individually. They are oval (0.5 × 0.4 mm) and pearly white at first, turning yellow and dark brown with age. After hatching, larvae search for potato tubers to penetrate. Larvae pass through four larval stages. L1 larvae are transparent white and measure about 1 mm in length. The head and prothoracic shield are dark brown. L1 larvae penetrate into tubers, making imperceptible holes. L2 larvae, which are cream coloured with dark coffee coloured spots (**185**), mine superficially in tubers. L3 larvae turn yellow-green, and the spots along the body are more visible. L4 larvae measure 12–14 × 2.5 mm. The body is purple on the back and green in the ventral area. Unlike the closely related moth species *Phthorimaea operculella* and *Symmetrischema tangolias*, which are stem borers and leaf miners, the larvae of *Tecia solanivora* feed exclusively on potato tubers. Pupae have a light coffee colour, which later darkens (**186**). The average pupa measures either 8.5 × 2.9 mm (female) or 7.8 × 2.4 mm (male). Pupation takes place in the soil or on tubers; in some cases also within tubers.



184 Adult of Guatemalan potato tuber moth.

185 Larva of Guatemalan potato tuber moth.

186 Pupae of Guatemalan potato tuber moth.

Infested tubers cannot be used as seed for either human or animal consumption. The wounds made by the first instar larvae of *Tecia* provide entry points for pathogens that may cause secondary infections.

Tecia solanivora has adapted to diverse environmental conditions; it can be found in more temperate areas at 1,000 metres above sea level as well as in colder regions at 3,500 metres above sea level. The duration of the life cycle depends strongly on temperature. A significant reduction in the developmental time is observed as temperature increases. At constant temperatures of 20 to 10°C, egg, larva and pupa development times vary between 10 and 26 days, 21 – 57 days and 17 – 91 days, respectively. Accordingly, the potato moth is capable of having two generations per year at 10°C and 10 generations at 25°C. At the same temperatures, female egg-laying capacity has been determined at between 156 and 360 eggs, and their lifespan varies between 20 and 25 days.

Tecia solanivora survives between cropping seasons on leftover potatoes or in potato storage, from where adults invade new potato fields. Field infestation starts with the beginning of tuberization. Storage infestation can be the result of an insufficient selection of healthy tubers or can start as a consequence of the migration of adults toward potato stores.

Control

Farmers are mainly using insecticides to control *T. solanivora*. This reliance on chemical insecticides, which are often highly toxic, not only increases the costs of potato production but also compromises farmers' health and has deleterious effects on the environment. Further, moths might develop insecticide resistance, and in some places secondary pests might occur. Various IPM components have been designed to reduce pest movement between field and potato storage and to lower larval and adult moth densities.

Reducing field infestation by applying cultural control methods

- **Good soil preparation** helps to destroy hibernating stages (mostly pupae) before planting.
- **Timely planting**, based on population dynamics of the pest, coincides with lower temperatures and the onset of rains. Farmers who plant in the dry season face higher moth infestations.
- **Regular hilling** (**earthing up**) protects tubers from ovipositing females and reduces the possibility of larvae reaching tubers.
- **Frequent irrigation** prevents soil cracking. Cracks allow moths to deposit eggs directly onto tubers.
- **Pheromone trapping for monitoring flying activity.** Moth catches can be used for defining control thresholds. In Costa Rica, the application of pesticides is recommended if 60 moths/ha/week are trapped during the dry season crop.
- **Avoiding leftover potatoes** helps to reduce sources of infestation before the next planting season.

Measures to prevent tuber infestation in potato storage

- **Immediate storage of tubers after harvest** avoids new tuber infestation through egg laying by moths overnight.
- **Cleaning stores** prevents infestation from surviving moths on formerly stored potatoes.
- **Physical control** by using nylon gauze at windows or covering tubers with nets prevents access of moths to tubers.
- **Pheromone trapping** for monitoring flight activity and the presence of moths.
- **Chemical control.** Many chemical insecticides have been evaluated to control *T. solanivora* in field and in storage: organophosphates, carbamates and pyrethroids are the most commonly used insecticides. Insecticides applied to the foliage and stem base at the beginning of tuberization have reduced infestation.

Prospects for biological control

The egg parasitoid *Chelonus phthorimaea* was found in the presumed centre of origin, in Guatemala. After *T. solanivora* spread to Colombia, the native egg parasitoids *Trichogramma lopezandinensis, T. pretiosum* as well as *Apanteles* spp. have been found. *Copidosoma koehleri,* a parasite of *Phthorimaea operculella,* also attacks *T. solanivora*. Entomopathogenic nematodes of the genus *Heterorhabditis* and *Steinernema feltiae* have been isolated from soils of Venezuela and Colombia, respectively. Preliminary results showed that *T. solanivora* larvae are highly susceptible. By contrast, the entomopathogenic fungi *Beauveria bassiana* and *Metarhizium anisopliae* have been proved less effective.

Further, *Bacillus thuringiensis* showed promising potential for successful integration into IPM of *T. solanivora,* especially to protect tubers in storage. The granulovirus infecting the closely related potato tuber moth *Phthorimaea operculella* (*Po*GV) also showed efficacy against *T. solanivora*. A native granulovirus has been isolated from naturally infected *T. solanivora* larvae in Venezuela. Granulovirus is an interesting microbial agent to be integrated into control strategies for *T. solanivora* because of its environmental safety and its known capacity for mass production.

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Non-infectious disorders

UNLIKE the product of other major crops, potato tubers contain around 80% water. As a result, they are relatively fragile and are prone to damage in many ways other than attack by pests or diseases. The extent to which disorders affect marketability depends on the sophistication of the market. At one extreme, where food is in short supply, disorders are relatively insignificant provided they do not threaten storability or edibility. In the developed world, disorders – including surface blemishes – become increasingly important, and more attention is paid to minimizing them. Disorders also affect the non-edible parts of the crop.

Disorders can be categorized into those that are chemical, nutritional or physical, those that have environmental causes, and those that are physiological in nature.

Care is needed in the cultivation of potato crops world-wide to ensure that all elements required for nutrition are supplied. This balance of nutrients becomes more critical where high yields are expected. An under-supply of a nutrient as a result of insufficient fertilization, or occasionally over-supply, or because of environmental conditions, can result in a plethora of foliage and tuber symptoms. It is critical to correct these if yield and quality are not to be impaired.

Damage can be inflicted through the misuse of chemicals applied to protect the crop or manage crop growth. The tuber can also be physically damaged, either externally or internally, through mis-handling, or sometimes as a result of the conditions in which the tuber develops.

Environmental disorders are not always possible to avoid. They may be the result of excessive wind, extreme temperatures, or oxygen deficit. Sometimes they are caused by pollution in the atmosphere. Where tubers are exposed to light, perhaps inadvertently, tuber greening can occur, making tubers unfit for consumption.

Finally, through environmental and other factors, the potato plant sometimes develops abnormally, producing undesirable haulm or tuber symptoms. Where it is possible to mitigate the contributory factors, physiological disorders can be limited.

CHEMICAL DAMAGE

Opportunities for chemical damage can occur at all stages of potato production and storage. Damage arises through the misuse of chemicals specifically used for a potato crop (*Table 1*), or through inadvertent treatment of potatoes by chemicals that are not meant for the crop (*Table 2*). The tables give examples of both categories of damage, but the list is by no means exhaustive. Symptoms depend on the nature of the damage. They are usually recognized by reference to unaffected ('normal') plants or by uneven growth. Potentially, chemical damage can occur with fertilizers or pesticides, often resulting from misuse or inadvertent use. The degree of damage depends on the quantity or dose that comes in contact with the potato crop.

TABLE 1 Misuse of chemicals specifically used on the potato crop

Nature of damage	Cause	Symptoms
Fertilizer scorch	High concentrations of artificial fertilizer placed too close to seed tubers or seed pieces	Non emergence; bleaching and death of growing points of haulm; bleaching and death of roots close to fertilizer
Damage to emerging sprouts by non-specific broad-spectrum pre-emergence herbicide	Herbicide (e.g. paraquat/diquat) applied after haulm has started to emerge	Chlorosis and necrosis of foliage
Herbicide yellowing	Can occur even where post-emergence herbicides are used according to label recommendations	Transient yellowing or mottling of leaves. May be confused with virus symptoms
2-aminobutane tuber damage	Fumigation by the tuber fungicide after harvest but before wound healing is complete	Sunken black lesions on the tuber of varying size and shape penetrating several mm into the tuber. May be confused with pit rot or gangrene
Heel-end browning	Translocation of haulm desiccants (e.g. diquat) to the tuber. Occurs where the soil is dry and transpiration is limited	Brown lesions, sometimes sunken, at the point of stolon attachment. Browning extends to different depths into the tuber flesh and along the vascular tissue, depending on the degree of translocation
Non emergence	Application of seed tuber fungicides after sprouting is initiated	Death of eyes and failure of primary or secondary sprouts to develop

TABLE 2 Inadvertent treatment of potatoes by chemicals not intended for potatoes

Nature of damage	Cause	Symptoms
Tuber contamination by glyphosate	Glyphosate contamination of the growing crop through drift from a neighbouring crop, spray tank contamination or spot spraying of weeds in seed crops	Glyphosate concentrates in tubers. Symptoms vary depending on concentration in tuber: no sprouting; multi- or 'cauliflower' sprouting; development of weak, spindly sprouts. Tuber damage can occur even when foliage symptoms are absent. At high foliage concentrations, growth is arrested and tubers develop abnormally, with cracking
Herbicide damage	Herbicide contamination of the growing crop through drift from a neighbouring crop or spray tank contamination. Many herbicides can affect potatoes, including those that have a hormonal effect (e.g. MCPA) and sulphonylureas	Symptoms vary according to the herbicide. Hormonal herbicides cause deformation of leaflets and haulm and thickened veins. Depending on the dose, sulphonylureas may stop or reduce haulm development and the foliage takes on a colour change from yellow through to red. Tuber distortion and cracking can occur where foliage symptoms are severe
Damage by residues of soil-persistent herbicides	Planting	Uneven emergence, distortion of foliage and poor haulm development. For example, residues of clopyralid can cause stems to emerge like a fern frond

187 Metribuzin damage due to overdosing or application to sensitive varieties.

188 Multisprouting as a result of glyphosate contamination.

189 Glyphosate-contaminated tuber with premature tuber formation.

Overdosing (187)

Accidental application at higher-than-recommended doses may occur. For example, some soil-acting herbicides have recommendations for different doses on different soil types. If used at a higher dose than recommended for a soil type, it may cause damage. The pattern of damage can indicate the cause. Thus narrow strips of damage in the growing crop may indicate overlapping spraying. Semicircles of damage at the edge of a crop indicate the sprayer was not turned off when turning, and the affected areas were overdosed.

Contamination (188, 189)

Where sprayers are used to apply a diverse range of pesticides, failure to empty out sprayer contents or ineffective cleaning before application to potatoes can result in damage. Some herbicides (e.g. certain sulphonylurea herbicides) can cause damage at a very low concentration. Even residues clinging to the undersurface of the spray tank can cause distortion and stunting of a potato crop. Contamination can occur in pesticide storage areas where a leaking container could contaminate the contents of another container.

Spray or vapour drift (188, 189)

Spraying crops adjacent to potatoes during windy conditions can result in drift of chemical and damage to the potato crop. The type of damage depends on the pesticide being applied. Drift of herbicide can have a marked effect on potato haulm. Hormone or sulphonylurea herbicides will cause stunting, distortion, chlorosis and necrosis and discoloration of haulm, but the extent of the damage is usually readily visible. High doses of glyphosate drift, increasingly used in Europe as a pre-harvest cereal treatment, are also easily noted as causing yellowing of the growing point or stunting. However, low-dose drift can be difficult or impossible to detect, since it may give no visible haulm symptoms but the herbicide will concentrate in the tubers. If grown for seed, low doses in tubers will lead to weak sprouts, multi-sprouting, no sprouting or non-emergence.

Leaching

The activity and movement of herbicides in soil are complex. Many pesticides applied to the soil surface or haulm of a crop are strongly adsorbed onto soil particles and move little through soil. Only where seed tubers or seed pieces are planted shallowly and heavy rain follows application of a relatively mobile pesticide is damage by leaching likely. In practice, it is a rare occurrence.

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190 Damage to developing sprouts from trifluralin residues.

Residues in soil (190, 191, 192)

Persistent soil-acting herbicides can remain active in soil for many months after application. Many manufacturers recommend minimum intervals before planting a succeeding susceptible crop; sometimes ploughing may be recommended to dilute the chemical by mixing in a greater volume of soil. Where label instructions are not met or overdosing has occurred (e.g. through a burst hose on the sprayer) residues sufficient to affect crop growth can occur. The addition of crop residues contaminated with herbicides to soil prior to planting potatoes can also result in damage. The pattern of symptoms is helpful in diagnosing the cause of damage. Symptoms will depend on the herbicide, but may include thickening of stems, distortion of foliage, and non-emergence of sprouts. If the persistent herbicide chlopyralid is present in soil or on crop residues in manure spread on arable land, it can cause stem foliage to emerge hooked over like a bishop's crozier or the neck of a fiddle.

191 Damage to developing sprouts from residual soil herbicide.

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192 Rolling of foliage from chlopyralid residues in soil.

193 2-aminobutane damage to tuber after fumigation was applied before wound healing.

194 Internal appearance of 2-aminobutane damage.

Application at the wrong stage or in the wrong place (193, 194)

Artificial fertilizer placed too close to emerging roots of seed tubers or seed pieces can scorch the roots, causing bleaching and death of the roots or the growing points of the haulm and even non-emergence. Post-emergent application of the herbicide metribuzin to certain cultivars causes damage, whereas pre-emergent may not. The seed tuber fungicide 2-aminobutane, used as a fumigant, can cause severe pitting of the tuber surface when it is applied before wound healing is complete.

Difficulties of diagnosis

Where visible damage to the potato has occurred, the pattern of damage may point to a specific application of fertilizer or pesticide, and the cause is easily deduced. Symptoms can then be compared with known symptoms for that herbicide. More difficult to diagnose are symptoms that cannot be traced to a known fertilizer or pesticide application. As several pesticides can give similar symptoms, visual diagnosis may not be possible. A first step in diagnosis is to attempt to eliminate pests, diseases or disorders as the cause. For example, wind damage to leaves can be similar to scorch from some pesticides. Where all other causes are eliminated, soil or plant tissue residue analysis can determine whether a fertilizer or pesticide is involved. However, there are so many potential pesticides, and the cost of analysis is so high, that preliminary diagnosis is needed to determine the most likely culprit. Even residue analysis may not guarantee identification of the cause, as with time residue levels may have degraded or the residue is below the limit of detection. Where residues are detected, careful interpretation of the result is required, and this requires skill and experience. Not least in this interpretation step is knowledge of the level of a pesticide that will cause damage and how the chemical may degrade with time. Equally, contributory soil or environmental factors that exacerbate damage need to be considered. For example, periods of high transpiration, dry soil, water stress or soil compaction may have increased susceptibility to damage.

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NUTRIENT IMBALANCES

Symptoms and diagnosis

Diagnosis of a nutrient imbalance, a deficiency or an excess, can only reliably be achieved by chemical tissue analysis. Symptoms may be the result of a true shortage or excess of a nutrient but may be induced as a result of an environmental, physical or pathological stress. The first step in diagnosis is to know what the growth pattern and appearance of a healthy plant are when adequately supplied with nutrients. The comparison of plants showing abnormal symptoms with healthy plants provides a guide to the imbalance. However, confirmation of a deficiency requires tissue analysis to determine if nutrient levels fall within the expected ranges. Correction of a deficiency may require correction of the stress rather than supplementing the nutrient level. Most deficiencies, when severe, affect the development of both above- and below-ground parts.

Nitrogen deficiency

Plants turn pale green and chlorotic. They subsequently progressively turn yellow, brown and necrotic starting from the lower, older leaves. During this change in colour the veins may remain green after the interveinal tissue has become yellow. Where nitrogen is in short supply, growth is stunted and slower. With a prolonged shortage, plants take on an erect stunted appearance with reduced leaf size.

Phosphorus deficiency

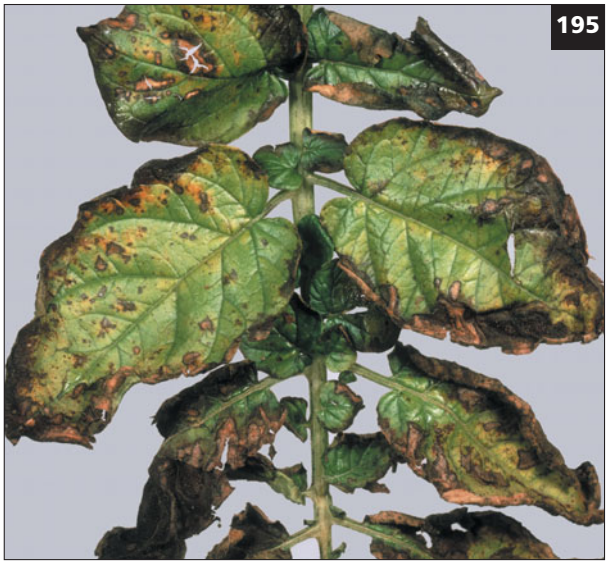
Plants deficient in phosphorus are stunted and darker green in colour than normal. Leaflets develop abnormally and may appear crinkled or show cupping. Lower leaves may drop off. This nutrient is vital for early plant growth and tuber formation. In consequence, fewer stolons and tubers are produced. Severe deficiency can result in rusty brown marks in the tuber flesh. Certain soil types and low temperatures can restrict the uptake of phosphorus.

Potassium deficiency

Young leaf blades are dark green and glossy with some crinkling. The leaves may have bronzing on the upper surface. Light blotches may appear within the veins and give the affected leaves the appearance of mild mosaic virus infection. When potassium is in particularly short supply marginal leaf scorch occurs, which spreads and leads to leaf death (**195**). The growing point may also be affected, leading to die-back. A shortage of potassium in the tuber is a predisposing factor to black spot and enzymatic blackening after cutting.

Magnesium deficiency

Symptoms first appear on older leaves as magnesium is rapidly transported to young growth. Starting mostly from the margins and spreading inwards between the veins, leaflets turn progressively pale green, yellow, then brown and necrotic (**196**). Leaflets ultimately die and



195 Potassium deficiency.

196 Magnesium deficiency.

become brittle but remain attached to the plant. Dry soil conditions resulting in poor uptake of nutrient, even where magnesium is abundant in the soil, can cause the deficiency.

Calcium deficiency

Calcium is a relatively immobile ion. In seed tubers, because of a deficiency in the flesh or poor mobility, sprout tips may turn black. Continued shortage of calcium results in sprouts becoming spindly. However, once planted, if a supply of calcium is present in the soil the sprouts absorb the element and can develop normally. Occasionally, in extreme situations, small tubers can form before sprouts develop. Plants that are deficient in calcium are spindly with small, deformed leaflets developing chlorotic margins which subsequently turn necrotic. However, the most common symptom of a shortage of calcium is internal rust spot in the tuber (**197**). This is scattered brown necrotic flecks sometimes in the vascular ring but also within the pith. The scattered and irregular flecks distinguish it from brown spot (see Hollow Heart), which is central to the tuber. This syndrome is most frequent on light, acidic soils where irrigation is not applied or is sporadic. It has been found on other soil types even where soil calcium levels are adequate. Some cultivars are particularly susceptible.

Sulphur deficiency

Sulphur is a macronutrient. Symptoms are similar to those of nitrogen deficiency: leaves turning pale and subsequently chlorotic. All leaves are affected including the youngest. In more advanced deficiency, leaflets curl upwards.

Boron deficiency

This is a rare condition but striking when it occurs, affecting the growing points. In the foliage, growing tips die and lateral shoots develop, the growing points of which also die. In time, the above-ground parts take on a bushy appearance. Leaflets crinkle, deform and curl upwards – symptoms that may be confused with those of virus infection. Leaf margins may turn brown. Below ground, root tips die and roots thicken and are stunted. Tuber production and growth are impaired. Tubers may show surface cracking and brown marks under the skin, especially near the point of stolon attachment. Potatoes are also sensitive to surplus boron.

197 Calcium deficiency – internal rust spot.

Iron deficiency

Iron deficiency is another relatively rare problem, most likely to be found on calcareous soils. Symptoms are a yellowing of younger leaves, with veins staying green longest. In severe deficiency the leaves sequentially turn yellow, bleached and necrotic.

Zinc deficiency

As with iron deficiency, younger leaves are affected, with veins staying green longest. However, a severe deficiency of zinc has a dramatic effect on plant development and morphology. Plants are stunted and leaflets are reduced in size, narrow and curl upwards. This effect can cause confusion with virus symptoms and gives a 'fern-like' appearance. Prolonged deficiency results in a bronzing of leaves and subsequently necrosis especially at leaf tips. Zinc deficiency is most likely on alkaline soils and when very high levels of phosphorus have been applied.

Manganese deficiency

This is not uncommon on alkaline soils, but is not often a severe problem. In deficient plants, interveinal chlorosis of younger leaves is the first symptom. If the deficiency persists, characteristic brown or black spotting develops along the edges of veins and the midrib (**198**). With extreme deficiency, the chlorosis becomes severe and leaves become bleached.

198 **Manganese deficiency.**

Manganese toxicity

Although uncommon, the potato is sensitive to high manganese soil concentrations. There are some cultivar differences in sensitivity, but typical symptoms are necrotic streaks or spots on stems or petioles. These may progress into more extensive areas. Symptoms start near soil level initially and progress upwards, achieving maximum expression after flowering. In the worst cases stems become brittle and may break off. Plants affected by manganese toxicity have pale leaves with interveinal chlorosis and are stunted. Manganese becomes more available at low soil pHs.

Aluminium toxicity

This is a manifestation of potatoes being grown in low pH soils. Potatoes are relatively tolerant of low pH, but depending on the organic matter content of soil and nutrient status, at extremely low pH levels roots become thickened and stubby with few root hairs, preventing the effective uptake of nutrients.

Copper deficiency

Young leaves become uniformly pale green. However, unlike other deficiencies with the same symptoms, copper deficient leaves roll inwards and upwards.

Control

Generally, nutrient imbalances are managed through the addition or removal of various nutrients through fertilizer application amendments, soil pH adjustment, crop rotation, crop selection and tillage modifications. The course of action depends on the specific nutrient involved, growing and cropping conditions, soil type, and other plant status factors. These are normally outlined in local or regional crop production recommendation guides.

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STEM-END BROWNING

Symptoms

Haulm, Roots and Stolons: Symptoms are not normally visible.

Tubers: At the point of stolon attachment to the tuber, collapse of cells to produce a brown necrotic lesion is indicative of stem-end browning (**199**). The lesion size can vary from 2–3 mm to 10 mm or more. Browning may extend from the stolon attachment along the vascular ring (**200**) for some distance in the worst cases. Pathogens sometimes invade the necrotic tissue.

Status of the disorder

Provided no pathogens invade the lesion, the presence of the lesion has no effect on the viability of seed tubers. However, tubers with stem-end browning of more than a few millimetres may be rejected as eating or processing potatoes.

Cause

One cause of stem-end browning occurs when the plants are under stress and the haulm is killed rapidly. Another cause is the occurrence of Verticillium wilt and the movement of the pathogen and its toxins into the stolon and stem end of the tuber. More usually, certain chemicals used to kill haulm may be drawn down to the tuber when the crop is under moisture stress. Diquat can produce stem-end browning in this way and is not recommended for use where the moisture deficit is high (see Chemical Damage).

Control

Take all measures possible to avoid placing crops under stress, particularly moisture stress, especially where desiccants are being used to kill the haulm.

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199 Stem-end browning lesion.

200 Cut tuber showing stem-end vascular browning.

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TUBER SURFACE INJURIES & CRACKS

Disorders caused by mechanical wounding, physiological disorders and virus infection.

Symptoms

Haulm: No foliar symptoms are known to be associated with tuber surface injuries or cracking, except as a result of infection of tubers through wounds (see specific pathogens).

Roots and Stolons: No symptoms have been reported.

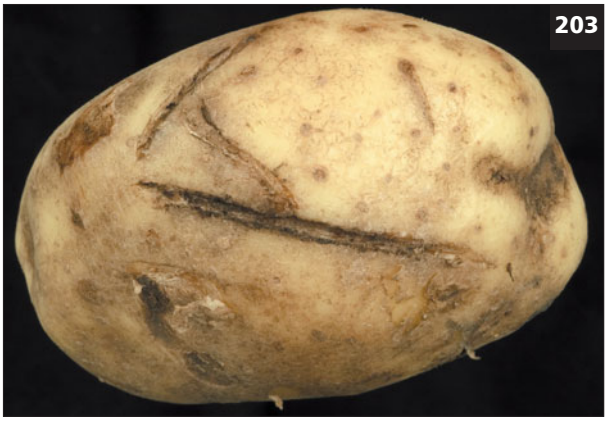
Tubers: Surface damage through improper handling or low humidity conditions can manifest itself as surface abrasions such as skinning or feathering, deep or shallow wounds such as cuts, scuffing, thumbnail cracks or shatter cracking – multiple cracks when a tuber impacts on an object. Longitudinal or growth cracking can develop during tuber development as can russetting of the tuber surface.

When surface abrasion occurs in immature tubers during harvest, it results in loose skin hanging in shreds (skinning or feathering) on the surface (**201**), exposing the underlying tissue. The exposed tissue can heal but may desiccate, become sunken and turn brown. Under humid or wet conditions it may develop a sticky surface due to bacterial growth. Although less prone, mature tubers may also suffer skinning during harvest or grading operations if handled badly, and damaged tissue can develop sunken scald spots if tubers are rapidly dried before storage. Surface discoloration of wounds, with associated rot problems, can develop especially on tubers that are kept at low temperature before wound healing is complete. These tubers may become flaccid from dehydration. Surface abrasions without loss of skin can also develop on mature tubers. Wounds vary in size depending on the surface they impact on and the intensity of impact.

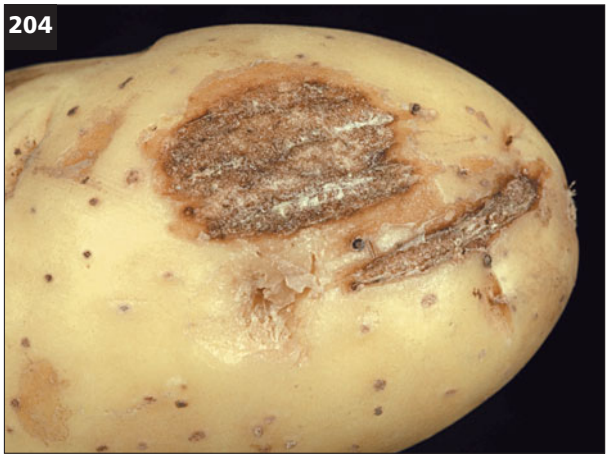
Thumbnail cracks (**202**) are usually crescent shaped, resembling a tear made with a thumbnail. They are the result of a tuber impacting on a projection. They are usually shallow (1–2 mm deep). Though more frequent when grading cold tubers, they can also occur when harvesting tubers under dry and low-humidity conditions. Wounds (**203**) that are produced by mechanical damage are more serious on turgid tubers and worse on larger tubers. Impacts upon turgid tubers, particularly when cold, can cause cracks to a depth of 5 mm or more, sometimes several centimetres in length.

201 Loose skin (skinning or feathering).

202 Thumbnail cracks.



203 Wounds.



204 Scuffing damage (larger lesion) that has darkened with drying.

205 Section through shatter cracking, also showing internal bruising.

206 Growth crack.

207 Star cracking.

Scuffing injuries are abrasions to the skin surface (**204**), while shatter cracks may penetrate deep into the tuber flesh (**205**).

Tuber cracking, caused by internal pressure during tuber enlargement (growth cracks), usually follows the long axis of the tuber (**206**). These cracks occur more frequently towards the apical or bud end of tubers. Small limited cracking called star cracking can occur (**207**). Growth cracks in the field frequently heal and, when they occur early in tuber growth, become relatively shallow and of little consequence (see Secondary Tuber Growth). Tuber cracks can also be induced by virus infection such as *Potato mop-top virus* or by *Potato spindle tuber viroid*. These growth cracks are indistinguishable from those caused physiologically, and diagnosis involves eliminating virus or viroid options by examining for disease symptoms and using laboratory tests.

Russetting takes the form of a regular criss-cross pattern on the tuber surface (**208**). It varies in its extent and can be confused with common scab and 'elephant hide' symptoms attributed to *Rhizoctonia*. However, russetting is usually regular in pattern and hardly raised above the tuber surface.

208 Russetting.

Status of the disorder

Where skin finish is critical to the marketable value of potatoes, such as in the washed-table-potato market, surface damage and cracks on tubers can have a major impact. This is particularly true in areas of the world where there is a well-developed washed-potato market for smooth-skinned cultivars, such as western Europe and Australasia. During grading for these markets, rejection rates can be high when a large proportion of tubers are affected.

Causes

Surface abrasion

This is more common when harvesting immature tubers, before the skin is set. Tubers with high turgor and low temperatures can suffer from superficial injuries during handling that are not related to impact.

Wounding

The overall healing process proceeds more slowly in bruise wounds than in cut wounds, and the capacity for wound healing decreases with increasing age of the tuber and time in storage.

Growth cracking

Cultivars differ greatly in their susceptibility to growth cracking. Severity of cracking is variable and depends partly on periderm strength. Growth cracks result from internal pressure exceeding the tensile strength. High internal turgor pressure develops from tissue expansion during rapid growth of tuber surface tissues during tuber enlargement. The use of fertilizer can also cause cracks because of the excessively rapid growth induced in tubers. Growth cracking is usually associated with irregular moisture levels and rapid water uptake. Such conditions can occur when heavy rain or irrigation follows a long dry period or during short periods of high-temperature stress. Wider plant spacing increases susceptibility to growth cracking. Low boron levels have also been reported to intensify growth cracking. Growth cracks heal with essentially normal skin, and are usually not infected by pathogens.

Mechanical cracking

Mechanical cracking during harvest and storage may follow sudden impacts. Immature tubers and large tubers are most easily injured. Severity is greatest when tuber temperatures are low and tissue is turgid. Extreme turgidity results when soil moisture levels are high and roots continue to function after vines have been suddenly killed by frost, by herbicides, or by harvesting tubers when vines are green. Severely cracked tubers are of little value, because wound healing is incomplete, dehydration is rapid and incidence of rot may be high.

Russetting

Cultivars vary in their propensity to russetting; however, the extent of russetting is influenced by soil type and soil conditions. More russetting occurs with coarse-textured soils and dry soil conditions.

Control

Careful harvesting and handling will minimize surface damage and wounding. This involves setting up machinery so that, for example, drops are minimized, tubers do not impact with protruding surfaces, stones and clods are separated from tubers, encrusted soil does not build up on equipment and sufficient soil is lifted with tubers during mechanical harvesting. It is equally important that tubers are in the correct condition for handling. A good tuber skin set will ensure that tubers resist damage. In turn, this depends on the cultivar grown, avoiding over fertilization, optimizing irrigation and allowing a sufficient interval from haulm destruction to harvest. Handling when tubers are cold increases the risk of tuber damage. Growing to secure an early harvest and warming tubers prior to grading reduce damage. Growth cracking can be avoided by applying irrigation to ensure an even development of tubers.

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AIR POLLUTION INJURY

Symptoms

Haulm: Symptom differences in the field between the several photochemical oxidants have not been determined in many cases. The symptoms reported here are mostly caused by ozone, peroxyacetyl nitrate, sulphur dioxide, nitrogen dioxide and related compounds. Upper leaf surfaces often present darkly pigmented spots (**209**) and have a bronzed appearance, sometimes with chlorosis. Injury is more severe in the lower, older leaves and progresses upwards. Later, plants become generally chlorotic, with premature leaf death progressing usually from the bottom upwards. Leaves eventually drop but do not abscise rapidly. Lower leaf surfaces may be light in colour, occasionally with a glazed or silvery sheen. Injury caused by sulphur oxides show interveinal necrotic areas that are light tan to white. Leaf area can be reduced by the effect of both sulphur dioxide and nitrogen dioxide. Symptoms can also occur on neighbouring plants and weeds in the field or adjacent areas, and examination of these areas can be helpful for accurate diagnosis.

Roots: Symptoms on roots have not been reported, except for a reduction in fresh and dry weights associated with high levels of sulphur and nitrogen dioxide. Retardation of root growth has been related to an interference by pollutant gases with the partitioning of dry matter from the leaves to the roots.

Stolons: Stolon symptoms have not been reported.

Tubers: Except for severe reduction in yields associated with very early senescence, tuber symptoms have not been reported.

Status of the disorder

Oxidant injury has been reported in North America along the Atlantic coast, in the Great Lakes region, the southeastern states and the Pacific southwest. There have also been reports of air pollution effects in European countries. Symptoms become evident within 24 hours following heavy exposure, but advanced necrosis and chlorosis may require 10–14 days. Photochemical oxidants accumulate either when relatively large areas of high atmospheric pressure are

Stuart Wale

209 Ozone damage.

present or when air masses move under a layer of warm air over cool land surfaces. The extent of injury is influenced by the concentration of oxidants, length and frequency of exposure, plant genotype and stage of plant growth. Field exposures of approximately 0.15 ppm ozone for a day or two are usually sufficient to injure exposed foliage.

The amount of damage is largely influenced by the density of the foliage mass. If this is large enough, a protection effect occurs, by which air pollutants are absorbed or adsorbed by leaf surfaces and removed from the immediate environment, thereby protecting nearby foliage. Thus, exposed leaves above the foliage canopy may be severely damaged while leaves within the canopy may escape injury. Similarly, injury may be more severe at field margins than in the centre. Plant injury can be more severe early in the season because plants are smaller, there is no protection effect, and therefore leaves and stems are completely exposed.

In the case of sulphur and nitrogen dioxides, the development of cuticular injury induced by these agents increases the water loss rate, and consequently the osmotic pressure in leaves is altered. Thus, it has been suggested that these pollutant gases may interfere with partitioning of dry matter from the leaves to the roots that leads to the reduction in root growth and tuber yields. As an indirect effect in potato crops, injury from ozone and other oxidants may increase susceptibility to pathogens such as the reported *Botrytis cinerea* infection.

Cause

Air pollution injuries are not caused by a biological organism. Photochemical oxidants such as ozone and toxic gases such as sulphur oxides and nitrogen dioxide are the more commonly cited air pollutants. Large circulating low-pressure cells move air pollutants great distances.

Control

Differences in tolerance to air pollutants exist within potato cultivars. Potato leaves are relatively resistant to injury by sulphur oxides compared with more sensitive crops (alfalfa, lettuce, sunflower, beet and bean). Injury should be anticipated in areas with air flow drainage patterns downwind from industrial plants. Cultural practices stimulating vigorous vine growth early in the season may help plants to avoid damage at the susceptible stage. Maintenance of a heavy foliage canopy until the tuber crop is assured may lessen or avoid mid-season injury.

Studies on the effects of anti-ozonant compounds such as ethylenediurea have shown that low concentrations of this product (given as a soil drench) can provide protection from accelerated foliar senescence induced by exposure to ozone. Higher concentrations of ethylenediurea can cause symptoms of toxicity and delayed senescence.

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FROST INJURY

Symptoms

Haulm: Frost injury is dependent on the temperature, the time of exposure, and the cultivar. Plant symptoms will range from a light-yellow to brown discoloration (**210**) on the top of the plant, especially at leaflet bases with temperatures above freezing, to completely frozen leaves that rapidly break down. Above freezing, chlorosis may appear as diffuse spots on the leaf surface area in some cases. Leaves exposed to freezing temperatures develop water-soaked tissues after thawing. Symptoms of low temperatures can be confused with those caused by viruses or pesticides. When low-temperature injury occurs in the field (**211**), only limited symptoms may develop at first. The full extent of damage may not be apparent until several days later.

Roots & Stolons: No symptoms known.

Tubers: Symptoms on tubers are essentially internal, but may be detectable on the tuber surface as well. In this case, they consist of diffuse, brown to black metallic-looking patches (**212**). Internal frozen tissue

210 Frost injury on potato foliage.

211 Field symptoms of frost injury.

212 Frost injury to potato tubers.

213 Cut tubers showing internal discoloration.

can easily be distinguished from unfrozen. It has a cheesy texture and is watery in appearance, resembling *Pythium* leak (watery wound rot). Other internal symptoms include small areas of necrosis in the flesh (**213**) and black spots at the vascular ring, due to the death of phloem tissue. These can resemble net necrosis and may be confused with symptoms of *Leaf roll virus*. After thawing, frozen tissue collapses but if dried it becomes chalky. Frost-injured seed tubers sprout poorly and may fail to produce plants. In addition to tuber damage, low temperatures may also predispose external tissues to surface mould growth. Tubers previously affected by low temperatures during storage will turn dark coloured on boiling.

Status of the disorder

Damage caused by low temperature has long been a significant concern to potato growers. In many production areas, frost injury may be a serious constraint to potato production, harvesting, and commercialization. Depending on the cultivar, some foliar symptoms may occur between 3 – 4°C. In general, symptoms develop most rapidly at temperatures ranging from slightly above freezing to below freezing. However, some primitive potato species grown in the Andes have tolerance to frost – some to as low as –5°C.

Low-temperature damage to potato tubers may develop in the ground before harvest, or later during storage or transportation, with the mature tubers being less severely injured than the immature ones. Low-temperature injury can increase losses as a result of growth retardation, delayed maturity of tubers, death of haulm and tubers in the field, damage to tubers during the storage period, damage to tubers during transportation, and difficulties in marketing damaged tubers. In addition, because symptoms are not always immediately apparent, merchants, processors and consumers may discover the consequences well after damage has occurred. Low temperatures will induce the formation of reducing sugars in tuber tissues, which will sweeten the flavour after cooking, or result in unacceptable fry colours.

Cause

This is a temperature related disorder and is not caused by a biological organism.

Control

Potato crops rarely justify frost protection measures such as smoke application or spray irrigation during low-temperature periods. However, the use of low temperature-tolerant hybrids may be helpful when cultivating potatoes in areas where potential chilling periods occur, such as at high altitudes. In general, to reduce the risk of low-temperature injury:

- Use early maturing cultivars where possible.
- Lift potatoes before there is a risk of frost; otherwise use frost-tolerant cultivars if available.
- Carefully manage nitrogen fertilization, because high concentrations of nitrogen in plant tissue increase the level of reducing sugars and delay harvest.
- Avoid storing field-frosted tubers or using them for seed.
- Hold storage temperatures around 4°C (for seed and fresh market potatoes) or above where possible (for processing potatoes). A lower temperature (down to 2°C) is possible for most commercial cultivars, provided an even temperature is maintained throughout the store.
- Maintain good air movement in storage.
- Provide good insulation for potato stores.
- Some authors recommend alternating temperatures during storage to reduce the risk of low-temperature injury. Tubers that have been kept at low temperatures for part of the storage period are believed to be less injured by a sudden drop in temperature than those stored at higher temperatures. However, their ability to form wound barriers in response to damage seems to diminish when tubers are returned to favourable temperature conditions.
- Observe the weather while transporting tubers, and avoid using open trucks during cold conditions. Warm potatoes prior to transport and use insulating material to protect potatoes during transport.

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HEAT NECROSIS

Symptoms

Haulm, Roots & Stolons: Typical symptoms are not normally visible.

Tubers: Symptoms of internal heat necrosis in potato tubers appear in the largest tubers first, at about the time of harvest. These can sometimes be confused with internal brown spot symptoms, which occur at various times throughout the growing season. These two common disorders may also be distinguished on the basis of the affected tissues within the tuber. Symptoms of heat necrosis primarily consist of tan to brown spots in the parenchyma tissues of the tuber internal to the vascular ring, but they can also be observed anywhere throughout the tuber tissues. Symptoms of brown spot occur in the central tissue of the tuber (see Hollow Heart). Necrotic tissue appears initially at the apical or bud end of the tuber. The colour intensity and the area affected increase over time, sometimes to a very severe level (**214, 215**). Nevertheless, these tissues remain firm, even after cooking.

External symptoms are usually not visible, except in some cultivars, where symptoms appear as blackened apical eyes, external tuber distortions, depressions in the tuber skin, or shrunken surface lesions. Heat necrosis may also be confused with the virus disease corky ring spot or calcium deficiency. The latter may be determined using laboratory tests.

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214, 215 **Severe symptoms of heat necrosis in tubers.**

Status of the disorder

It is well known that temperature has a controlling influence on plant growth. The potato was considered to be well adapted only to cool climates and to be adversely affected by high temperature, until the success of potato cultivation in arid and semi-arid regions. However, potatoes can be affected by excessively high temperatures, with the risk of economic loss. Heat necrosis of potato tubers is a good illustration of the negative effects of high temperatures on tuber health. While tolerance to heat has been reported to be genetically controlled, development of cultivars with resistance to heat necrosis is still in progress.

Internal heat necrosis damage varies with location, growing season and the cultivar used. It has been associated with growing seasons where above-normal temperatures and below-normal precipitation prevail, especially during the initiation and early development of tubers. Losses due to heat necrosis also occur in tubers formed near the soil surface, generally within the top 5 cm. Some reports suggest that the development of internal heat necrosis is not necessarily a simple response to high temperatures, but rather a combination of responses to several environmental factors. Exposure to excessive heat such as hot sun conditions may not only result in direct damage to tubers, but may have other effects such as delaying wound healing and encouraging the development of diseases such as Fusarium dry rot and Pythium leak.

Cause

Heat necrosis is a temperature-related disorder. It is not caused by a biological organism.

Control

Control depends on good crop management and careful attention to the factors over which growers have control. Every effort should be made in order to prevent exposure of tubers to high temperatures by:

- Applying proper and timely hilling (ridging) of tubers and emerging plants.
- Maintaining correct soil moisture levels where irrigation is in use.
- Encouraging good ground cover.
- Timing harvest to avoid high soil temperatures.
- Protecting tubers from extreme hot, sunny conditions at harvest.
- Appropriate storage environment and transportation conditions. It is recommended to avoid the use of open trucks under hot sun conditions, or to provide protection during transportation.
- In addition, tolerant cultivars may be used where available.

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OXYGEN DEFICIT/BLACKHEART

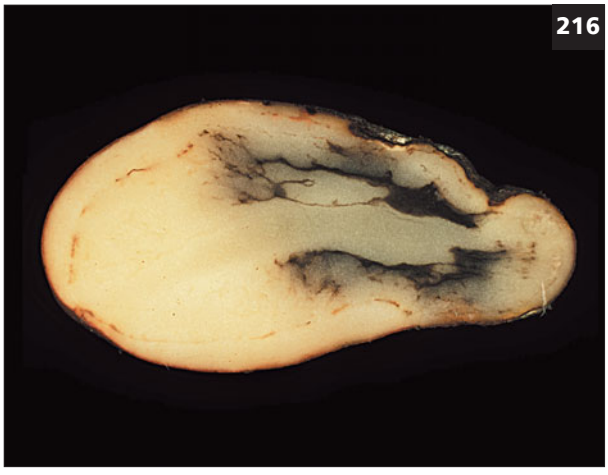
Symptoms

Haulm: A deficit in oxygen level may cause delayed plant emergence.

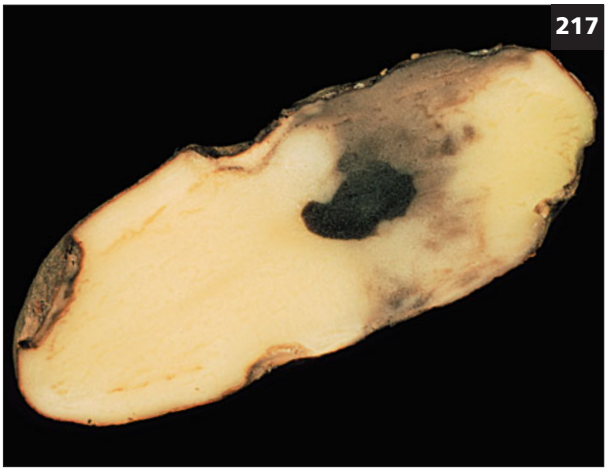
Roots: No specific symptoms are known.

Stolons: Abnormalities may be observed in the stolon shape.

Tubers: Development of the tubers after planting is affected, depending on the degree of oxygen deficit. Symptoms may vary, according to whether the tubers were exposed to high temperature with a normal air supply, or to low, high, or normal temperature with an insufficient air supply. Blackheart may develop as a result of oxygen deprivation in tubers. Symptoms are a brown, purple-black or black discoloration of an irregular area of the central tissues of tubers (**216, 217**). The discoloured area sometimes radiates to the skin. The coloration is usually uniform and is clearly demarcated from the healthy tissue. Blackheart is not usually visible externally. For a short time after the development of the internal lesion, the tuber retains its firmness. With time, through water loss, cavities form in the discoloured tissue, the tuber softens and the skin becomes leathery. Invasion of the necrotic tissue by bacteria and fungi can occur. Seed tubers with blackheart should not be used, due to increased susceptibility to soft rot and poor emergence problems.



216 Blackheart discoloration in a tuber.



217 Blackheart discoloration in a tuber.

Status of the disorder

Oxygen deficit is one of the physiological disorders that affect the quality of potato tubers and that may result in yield reduction. It can be caused by cultural and environmental factors, and may occur either during plant development or in tubers before harvesting or in storage.

Oxygen deficit during plant development

Tubers and other underground parts of potato plants require high oxygen concentrations for their normal development. Any reduction in oxygen availability or accessibility may result in delayed plant emergence and tuber and stolon abnormalities, as well as yield reduction. Damage is proportional to the level of oxygen deficit. This deficit may be due to the nature of the soil, since compact soils will generate more oxygen deficit problems than well-drained ones. The soil temperature is also very important, because high soil temperatures will result in high respiration rates of tubers. This generally leads to an oxygen deficiency, unless the soil is really well drained. Any excess of water (flooding) will prevent normal respiration, which will result in an oxygen deficiency in the below-ground plant tissue.

Blackheart development in the field or during storage

Blackheart is caused by a restriction of oxygen supply to the central tissues of tubers. The extent of discoloration depends on the degree of oxygen starvation. There are several potential causes of blackheart. At one extreme, when tubers are subjected to very high temperatures, the diffusion of oxygen to the central tissues is too slow to keep up with demand. This usually leads to a brown discoloration. Sub-oxidation of central tuber tissues can also occur when the external oxygen supply is restricted, for example, in closed bins, containers, clamps or deep piles without adequate ventilation. It can also occur when tubers are surrounded by a film of water, because oxygen diffuses only slowly through water. Thus blackheart can occur when soils are waterlogged prior to harvest. Blackheart can occur at freezing point, but above the temperature at which the tuber freezes. The rate of development of blackheart symptoms is temperature related; at lower temperatures a longer period of oxygen depletion is required. However, the development of internal symptoms at temperatures close to 0°C is faster than at 5°C.

Cause

This disorder is caused by a deficiency of oxygen and is not caused directly by a biological organism.

Control

- Avoid poorly drained soils and excess irrigation, to prevent flooding and oxygen reduction.
- Use cultural and tillage practices favouring good soil porosity, to improve air exchange.
- Avoid excessive heating, used to prevent chilling during winter transportation, to prevent excessive respiration which requires high oxygen levels. In general, avoid all conditions for excessively high respiration rates.
- Provide good ventilation in storage to make oxygen available for tubers.
- Avoid conditions leading to water condensation on tubers, because water films will stop exchange between the tuber and its environment.
- Do not hold seed in tightly sealed trailers for more than 12 hours, because it may reduce air availability.

Fouad Daayf, H W (Bud) Platt and Stuart Wale

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TUBER GREENING

Tuber greening is a physiological disorder and is not associated with a pathogenic organism.

Symptoms

HAULM, ROOTS & STOLONS: Typical symptoms are not normally visible.

TUBERS: Tubers exposed to light in the field or after harvest develop a green discoloration due to the accumulation of chlorophyll in the leucoplasts of affected tissues (**218**). Greening of surface tissues may also be accompanied by internal discoloration, which can extend to a depth of 20 mm or more. The intensity and extent of greening depend upon a variety of factors, such as duration and intensity of light exposure, environmental conditions, tuber cultivar and physiological age of tubers.

Status of the disorder

Tuber greening is found wherever potatoes are grown and conducive conditions occur. It can be a significant problem, because greened tubers are unmarketable. Exposure of potato tubers to light also results in an increase in alkaloid content. The glycoalkaloids solanine and chaconine, which tend to concentrate under the skin, have toxic properties and cause bitter, unpleasant flavours in cooked tubers. Concentrations of glycoalkaloids vary according to genetic and environmental factors, particularly exposure to light. Cultivars vary in their total tuber glycoalkaloid (TGA) content as well as in their rate of TGA increase after exposure to light. Although both TGA accumulation and chlorophyll production (tuber greening) are light-sensitive processes, the intensity of tuber greening after exposure to light is not correlated with percentage increase in TGA and, therefore, the processes are independent of each other.

Tuber greening in the field occurs when tubers are exposed to light, so this should be avoided. Some cultivars set tubers near the soil surface, and they are more susceptible to tuber greening. High plant densities can also cause tuber crowding, forcing tubers to the soil surface. Weather and cropping conditions that increase soil erosion can expose tubers. Depending on weather and soil type, cracks can form as the soil dries or as tubers enlarge, exposing tubers to light.

218 **Tuber greening after exposure to light.**

After harvest, the extent and intensity of tuber greening are related to the method of storage; exposure to light should be avoided. If light is present, storage at room temperature can increase the rate of tuber greening relative to cold storage. At the supermarket, the type of packaging (transparent or otherwise) and the amount of light tubers are exposed to in the display are critical factors in determining the extent to which tuber greening develops. Lighting in supermarkets and transparent packaging can cause increased greening, particularly with immature or 'new' potatoes, which green more rapidly than mature tubers.

Control

Proper hilling/ridging of plants can effectively cover tubers and reduce greening. In some locations, deep planting and more frequent hilling operations can reduce tuber greening caused by light filtering through soil cracks. Irrigation can reduce tuber greening by keeping the soil moist to prevent the formation of soil cracks. In some regions, mulch has been used effectively to cover soil cracks and reduce evaporation from the soil, thus preventing extensive crack development. Increasing the distance between rows can also reduce tuber greening. In a dense planting, high numbers of tubers within a restricted soil volume can force some of the tubers through the upper surface of the ridge, exposing them to light.

Following harvest, table potatoes should be stored in the dark. Experiments have shown that modifying storage atmosphere (high CO_2) and coating tuber surfaces with waxes or oils (modifying internal tuber conditions and gas exchange) reduce tuber greening, but results have been variable largely due to variability in the physiological age of tubers. The use of surfactants and calcium infiltration has also been effective in reducing tuber greening under experimental conditions. Gamma irradiation (also used for sprout control in some areas) has been shown to have some effect on reducing tuber greening and possibly extending the shelf life of tubers at market. Non-transparent packaging reduces greening of tubers in market displays.

Potato cultivars vary in their degree of greening in response to light exposure. External, internal and depth of greening parameters often vary independently among cultivars. In general, red cultivars tend to show fewer discolorations than white or russetted cultivars. Tuber greening appears to be quantitatively inherited, and broad sense heritability may be large enough to permit effective selection for germplasm with reduced levels of tuber greening in potato breeding programmes in the future.

RICK PETERS

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WIND INJURY

Symptoms

HAULM: Injury is evident on the upper surfaces of leaves that have been rubbed by other leaves due to the effect of wind. Discoloured tissue is usually brown when dry, varies in size, has a glistening or oily appearance and sometimes extends through the leaf. High winds can make the foliage appear 'hard'. Very cold winds can cause the under-surfaces of leaves, particularly those turned over by the wind, to become brown, sometimes with a silvery or glassy sheen. Leaf injury may be more severe at the edges of the field.

ROOTS & STOLONS: No effects have been reported.

TUBERS: Tubers in the field may be damaged by drying wind during harvest. Injury may become evident during storage, as sunken spots underlying the skinned portions of tubers. The surfaces may be overgrown with bacterial slime, causing decay in storage.

Status of the disorder

The effects of wind on potato plants can be direct or indirect. Direct injuries caused by wind on leaves affect the photosynthetic area and thus induce a reduction in carbon assimilates partitioned to the tubers. Consequently a reduced tuber yield is obtained. These effects are more severe in regions where cold winds last for long periods or when a combination of strong winds and high temperatures induces drying effects in the crop. Direct wind damage on tubers is very common at harvest time and is greater when immature tubers are harvested and when open mesh sacks are used.

Indirect wind effects include transportation of phytopathogenic fungal spores from field to field and even from distant regions, increasing disease incidence on potato crops. Spore migration has been documented for different pathogens such as *Phytophthora infestans*. On tubers, indirect effects of wind injury are detected in storage, when bacterial growth can take place on injured tuber surfaces more easily than on healthy tubers.

Cause

This disorder is caused by high wind speeds or gusts.

Control

- Grow potatoes in locations that are less exposed to severe winds, or that are close to natural barriers such as trees in critical regions.
- Harvest mature tubers, since they are more resistant to the drying effect of winds.
- Harvest tubers into tightly woven sacks, boxes or trailers instead of open mesh sacks to avoid drying effects.

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COILED SPROUT/HAIRY SPROUT

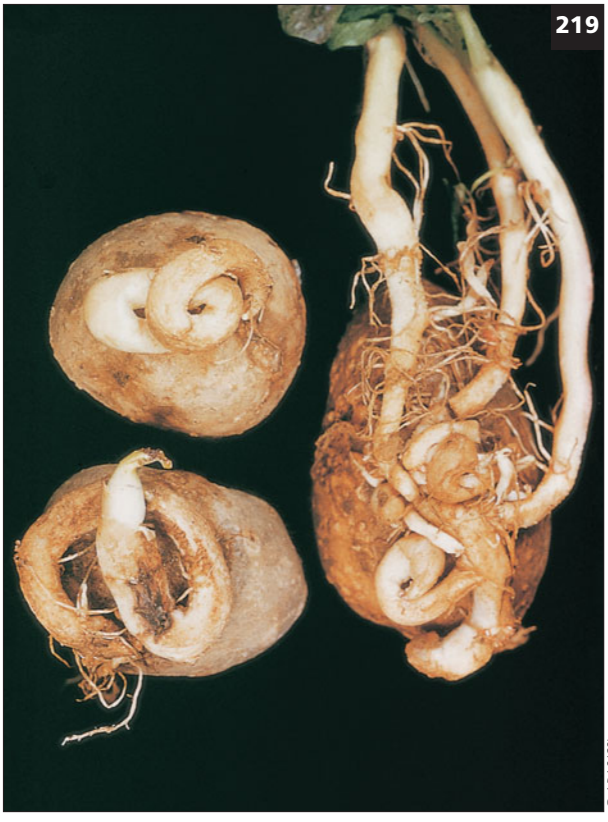
These conditions are associated with poorly specified physiological or pathological factors and/or agents.

Symptoms

Coiled sprout

HAULM: Sprouts fail to emerge or grow to the soil surface. They coil irregularly, are thickened and show cracking and sometimes brown lesions on the inner surface of the coil (**219**). The lesions are similar to those of skin spot (*Polyscytalum pustulans*). They are less well defined than those of stem canker (*Rhizoctonia solani*).

ROOTS, STOLONS & TUBERS: There are no distinct symptoms.



219 Coiled sprout symptoms.

Hairy sprout

Haulm: Thin weak sprouts develop from eyes and excessive root formation occurs (**220**).

Roots, Stolons & Tubers: There are no distinct symptoms.

Status of the disorders

These disorders are reported mainly from Europe, although they have occurred less frequently in the last two decades. They probably occur throughout the world.

Cause

Coiled sprout

The exact cause is unclear. A causal relationship with *Verticillium nubilum* Pethybr. has been demonstrated, but this fungus is frequently associated with potato tubers and is largely considered non-pathogenic. It can be found associated with both coiled and normal sprouts. However, sprout development at planting may also be a factor. Excessively mature tubers with well-developed sprouts, and those chitted in light, are more prone to the disorder, suggesting that physiological changes in sprout apical buds are involved. There may be an association with 'little potato' (see page 168) in that cold soil conditions may also induce coiled sprout.

Hairy sprout

This is also associated with excessively mature tubers that have received a high degree of heat input during storage after dormancy break.

Control

Well regulated storage conditions, controlling sprout development and good soil cultivation have largely prevented these problems in developed countries.

Stuart Wale

220 Hairy sprout.

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GENETIC DISORDERS

Symptoms

HAULM: Genetic mutations can result in a number of disorders: wildings, giant hill, etc. that produce abnormal haulm growth (**221–225**).

STOLONS: Abnormalities in stolon shape may be observed.

ROOTS: Increased root number and decreased size are often observed.

TUBERS: Development of tubers is altered in terms of number, size, colour, and shape.

221 Reduced chlorophyll content.

222 Reduced leaf size and altered haulm architecture.

223 Curled leaflets and increased rugosity.

224 Altered leaf arrangement and leaflet shape.

225 Intense rugosity of leaves.

Status of the disorders

Genetic disorders result in abnormal plant growth and tuber formation which affect crop yield and purity. They are a result of somatic mutations which occur spontaneously, or on rare occasions due to external factors influencing normal cell division and/or multiplication.

Wildings are typified by small, bushy growth of the potato plants. Stem numbers are greatly increased, but each is small and thin with leaflet number and size reductions. Numerous stolons form with small tubers that produce weakened sprouts.

Giant hill involves increased size and vigour of the haulm with thicker and/or coarser foliage. Maturity is often delayed, reducing tuber yield, and tubers sprout late. These plants often have delayed development of late blight. Many other disorders are also reported with a variety of plant growth and tuber abnormalities.

Cause

Genetic disorders are not caused by a biological organism but are a result of genetic mutation.

Control

Selection of healthy seed potatoes of registered and certified cultivars is important. Roguing the abnormal plants will lessen the effect of these genetic disorders.

H W (Bud) Platt

Key references

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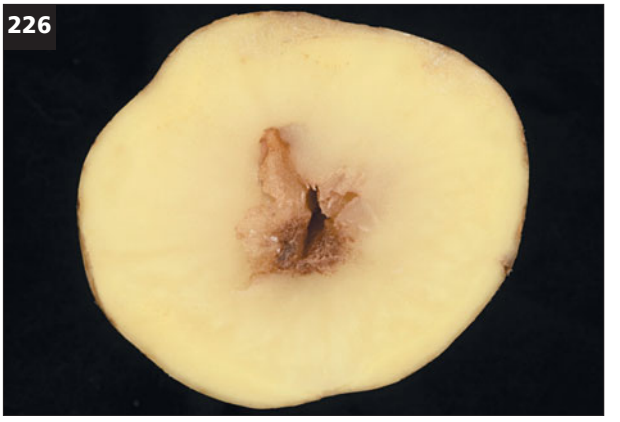
HOLLOW HEART

Symptoms

Haulm, Roots & Stolons: No distinct symptoms are normally seen.

Tubers: A cavity forms in the central tissue of tubers (**226**). The cavity is usually angular, and the walls of the cavity are frequently thickened and of a light brown colour. Usually the cavity cannot be seen from the tuber exterior, being revealed only after cutting. Prior to the cavity forming, the central cells appear water-soaked or translucent. Sometimes a brown diffuse area known as brown spot (**227**) develops as a precursor to cavity development. Occasionally, bacteria or fungi invade the cavity and rot the tuber from the inside. Hollow heart occurs mainly in large tubers of ware or table potato crops but early symptoms (brown spot) can occur in smaller tubers.



226 Symptoms of hollow heart.

227 Brown spot – a precursor to hollow heart.

Status of the disorder

Hollow heart does not affect the viability of a tuber and is thus of little significance in seed crops. However, in crops for eating or processing, the presence of hollow heart in a stock frequently leads to rejection of the stock for the chosen market. Occasionally up to 50% of large tubers can show hollow heart.

Cause

Hollow heart is a physiological disorder and develops where tuber growth is unusually rapid. Frequently it will occur where conditions for rapid growth follow a period of slow growth, such as where rainfall follows a period of moisture stress. Hollow heart is more common where plant spacing is uneven and few large tubers form. Varieties vary in their propensity to develop hollow heart.

Control

Maintaining adequate soil moisture throughout tuber swelling ensures steady tuber growth. Irrigation related to soil moisture deficit is thus important, particularly in varieties susceptible to hollow heart. Producing even crop stands by accurate planting, and minimizing seed tuber or seed piece decay by providing recommended rates and timings of nitrogen application and by optimizing seed spacing avoid uneven tuber size development. Where moisture deficits cannot be controlled, non-susceptible varieties to hollow heart should be used. The use of X-rays to identify and grade out tubers with hollow heart is very effective.

STUART WALE

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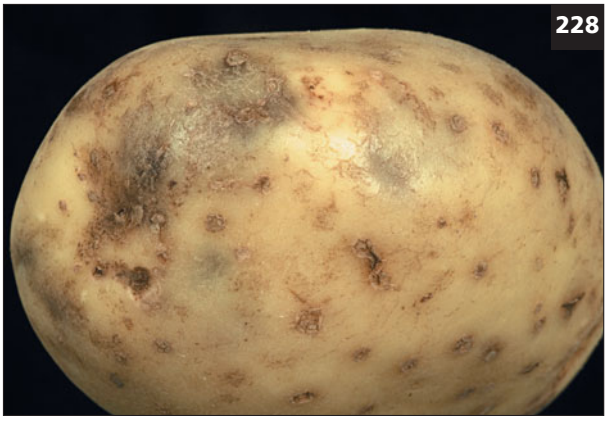
BLACK SPOT (INTERNAL BRUISING)

Symptoms

HAULM, ROOTS & STOLONS: Typical symptoms are not normally visible.

TUBERS: Internal black spot is a discoloration that occurs in tubers after a bruising injury. It is characterized by blue-grey to black patches, with diffuse margins, beneath the tuber skin (**228**). They may be of different sizes and shapes and rarely penetrate beyond the vascular ring. These symptoms are more detectable at the stolon end of the tuber, which is more susceptible. Symptoms are not easily observed on the tuber surface. They are normally visible only if the outer skin of the tuber is removed (**229**). Black spot can develop soon after injury, generally after a few hours, with maximum intensity about 24 hours later.



228 External symptoms of black spot bruising.

229 Internal symptoms of black spot bruising.

Status of the disorder

This disorder is at all times caused by bruising injury. Injuries causing black spot occur during harvest, handling or grading, and are due to impacts or, during storage, to pressure (pressure bruising). Where harvest and handling are mechanized, black spot is generally well known and can be a major cause of loss in potato production. The black discoloration is due to melanin pigments resulting from polymerization of quinones, obtained through oxidation of phenyl substrates such as tyrosine and chlorogenic acid. These biochemical reactions are initially induced by bruising impacts. Bruising impact severity depends on cell turgor pressure, temperature, cultivar and interaction with environmental factors. Where tubers are stored in bulk, especially where in deep piles, lower tubers may develop pressure bruising where their turgor pressure is not high. Although much work has been conducted on black spot, it is still not fully understood why certain tubers are more susceptible than others. There is variability in susceptibility to black spot from tuber to tuber.

Cause

Internal black spot is a physiological disorder, not caused by a biological organism. It is caused by an impact to the potato tubers.

Control

Control of black spot requires an integrated management programme from planting until the potatoes reach the consumer. In such a programme, many factors should be taken into consideration:

- Reducing physical impact during harvest and handling is the most important measure to reduce black spot symptoms. For this purpose, the use of appropriate equipment and careful handling of tubers, for example, by not dropping them from more than about 15 cm, is highly recommended.
- Potassium deficiency predisposes tubers to black spot, so adequate potash fertilization should be given special consideration.
- Provide good soil moisture, because tubers with low turgor pressure are most susceptible to bruising damage. Soils allowing good root development may also limit bruising damage.
- Do not let the potatoes dry out before harvest. Drying lowers their turgor pressure and makes them more susceptible to bruising damage.
- Maintain a high atmospheric humidity during storage to avoid dehydration of tubers, which predisposes them to bruising damage.
- Use resistant cultivars where possible.
- Avoid harvesting when the tuber flesh temperature is less than 8°C.

Fouad Daayf & H W (Bud) Platt

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INTERNAL TUBER SPROUTING

Symptoms

HAULM, ROOTS & STOLONS: Missing plants or severely stunted, chlorotic and necrotic haulm.

TUBERS: Sprouts develop within the tuber tissue or grow through the tuber tissue (**230, 231**).

Status of the disorder

This condition is relatively uncommon in modern potato production. It can be found in tubers stored in deep piles for a long time and in tubers stored at relatively high temperatures for processing (usually above 12°C). Internal sprouting has been linked with the use of sprout suppressants such as CIPC (isopropyl-m-chlorocarbanilate). Where concentrations are sub-optimal for preventing sprout development, internal sprouting may be encouraged. The sprout tips of internal sprouts are often blackened as the supply of nutrients, especially calcium, is restricted to the reserves in the mother tuber. Calcium is translocated relatively slowly in tubers sprouting in store.

Cause

Internal tuber sprouting is a physiological disorder usually induced by storage practices.

Control

No control measures are applied, since it is relatively uncommon. Uniform treatment with CIPC to completely prevent sprouting ensures that the disorder is rare in processing potatoes.

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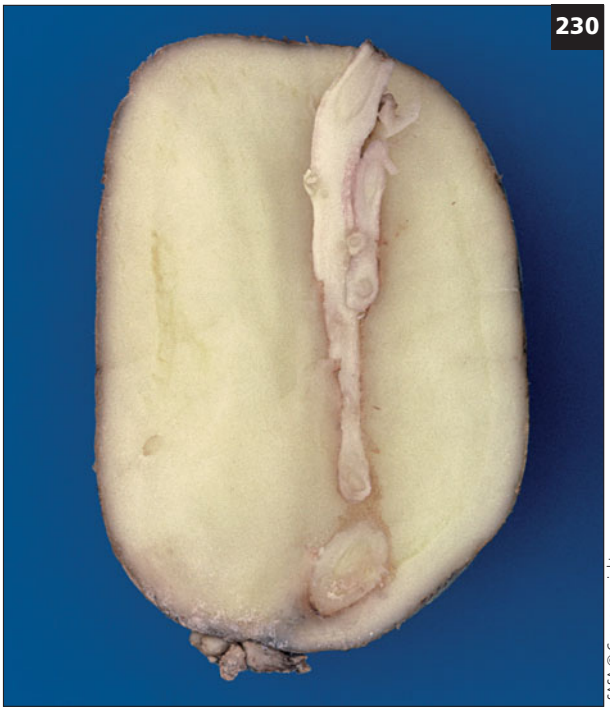

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230, 231 **Internal sprouts.**

SECONDARY TUBER GROWTH

Symptoms

Haulm, Roots & Stolons: No symptoms.

Tubers: There are several forms of secondary growth in tubers:

Extension of the rose-end

This condition is the regrowth of the meristematic tissue at the rose end to produce an elongated, dumbbell-shaped tuber. The starchy food reserves for the new growth are drawn from the original tuber tissue (**232**).

Jelly end rot

This occurs when the heel end becomes glassy and devoid of starch. The degree of glassiness depends on the extent of new growth. The tuber around the glassy area collapses, releasing liquid and the skin becomes wrinkled (**233**). Invasion by bacteria may occur through this tissue.

Gemmation

Gemmation (**234**) is the development of an eye, usually at the rose end, to produce a knob-like outgrowth.

Little potato and chain tuberization

Certain varieties of potato may form a stolon and another tuber from a single eye of a seed tuber instead of forming a stem. This is called little potato (**235**). Frequently, no foliage develops at all. Alternatively, in a normal growing crop, once tubers have formed, they may develop one or more secondary tubers in a similar way. This 'chain tuberization' may be the result of the proliferation of a single bud in an eye rather than all buds.

Cracking and hollow heart

When tuber development is held in check because of dry weather and the crop suddenly receives water, the tuber may swell rapidly. Cells within different parts of the tuber may develop unevenly and this can result in either longitudinal cracks from the apical or rose end to the stolon end or a cavity developing centrally in the tuber. See also Tuber Surface Injuries & Cracks (page 149), and Hollow Heart (page 164).

232 Secondary growth; extension of the rose end.

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233 Secondary growth; jelly end rot.

Cause

Little potato is considered to be the result of a check in development of seed tubers of certain cultivars. It can occur when tubers are well sprouted before planting but then experience a check in growth due to cold soil conditions. All the other secondary tuber symptoms are the result of irregular growth, particularly if a period of warm, dry weather is followed by rainfall.

234 Secondary growth; gemmation or development of knob-like outgrowth from eyes.

235 Secondary growth; little potato.

Control

Little potato may be avoided by planting recently sprouted or non-sprouted tubers from refrigerated storage directly into warm soils, but this can also cause seed decay if sprouts do not grow and emerge quickly. Late-planted cultivars often have this problem, since sprouts are quite old. Some cultivars form little potatoes in response to the fungicide thiabendazole, which should be utilized as per label directions.

Most of the other conditions can be controlled by an adequate supply of water throughout the crop growth period. Irrigation scheduling has limited the development of these adverse conditions.

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Glossary

Acervulus (pl. acervuli) Fungal structure bearing fungal conidia or spores.

Alate Winged.

Amphimictic By means of sexual reproduction.

Anastomosis groups Strains of a species characterized by the ability of mycelium to fuse together.

Androcyclic An aphid life cycle which is neither holocyclic or anholocyclic and where a proportion of males contribute to the sexual phase.

Anholocyclic Species with an incomplete life cycle reproducing only asexually (parthogenetically).

Antheridium (pl. antheridia) Organ in which male gametes are formed, e.g. in Oomycete species.

Apical At the apex or growing end of a root, shoot or leaf.

Apothecium (pl. apothecia) Cup-shaped fruiting body of ascomycete fungi. Asci, which form sexually-produced ascosphores, are borne on the inner surface.

Appressorium (pl. appressoria) Adhesive disc of fungal mycelium from which host tissue is penetrated.

Apterous Wingless.

Basidiospore Sexual spore formed on a basidium.

Basidium Spore-bearing structure of a basidiomycete fungus.

Biciliate Possessing two cilia or motile 'hairs'.

Chlamydospore A tough-walled resting spore of a fungus.

Chitinization The process of exoskeleton development in an insect.

Chitting Sprouting seed tubers under controlled conditions to enhance growth after planting.

Conidium (pl. conidia) An asexual fungus spore produced from a conidiophore.

Conidiophore A fungal hypha which bears one or more conidia.

Crop canopy The above-ground foliage of a potato crop.

Cull potatoes Rejected or discarded potatoes.

Cultivar / variety A distinct and uniform strain (of potato).

Cystosori Thick-walled single or multi-celled resting spores of the order Plasmodiophorales (e.g. powdery scab); sometimes called sporeballs.

Cytochalasin A compound produced by fungi which inhibit actin polymerization into microfilaments.

Diapause Spontaneous state of dormancy in the life cycle of insects.

Earthing up The process of drawing soil up over seed tubers. *See also* hilling, ridging up.

ELISA Enzyme Linked Immuno-Sorbent Assay. A biochemical test that uses antibodies to recognize target antigens of pathogens and other organisms.

Epidermis Outer layer or skin (e.g. of a leaf). *See also* periderm.

Eukaryon Organism with cells having a membrane-bound nucleus.

Exuvia Cast-off skin.

Genome An organism's genetic material.

Genotype The genetic constitution of an organism.

Glycoalkaloids A family of poisons commonly found in the plant genus *Solanum*.

Grading/sorting The process of separating out undesirable potatoes (cull or reject potatoes) from a stock.

Groundkeepers Tubers remaining in a field after harvest, which overwinter and grow in subsequent years. *See also* volunteer potatoes.

Guttulate Having small oily droplets (guttae) as markings.

Haulm A description of the stems, leaves and other plant structures produced above soil level. *See also* vine.

Haustorium (pl. haustoria) A hyphal branch which penetrates a host cell.

Heel end The end of the potato connected to the stolon. *See also* stolon end.

Hill A description of the soil covering the seed tuber. *See also* ridge.

Hilling The process of drawing soil up over seed tubers. *See also* earthing up, ridging up.

Holocyclic Having a complete life cycle. In particular, it refers to those aphids which alternate parthenogenetic (asexual) with sexual reproduction.

Hyaline Clear, transparent.

Hyperplasia Abnormal enlargement of plant tissue resulting from infection by a pathogen.

Hypertrophy The state of enlargement of host tissue.

Insolation Exposure to the sun's rays.

Instar Insect stage between moults.

Latent infection A quiescent infection; one that has not developed symptoms but may do so.

Lenticel A vestigial pore in the periderm of potato tubers.

Mating types Types of a pathogen that can undergo sexual reproduction.

Microconidium (pl. microconidia) Small, relatively simple spore (e.g. of the genus *Fusarium*).

Microsclerotium (pl. microsclerotia) Small sclerotium or firm, rounded mass of fungal mycelium – a resting body which may survive for long periods.

Muck soils Soils containing a high humus content (e.g. a peaty soil).

Multicyclic disease A disease consisting of many complete life cycles of a pathogen.

Mycelium A mass of microscopic threads or hyphae, that form the vegetative part of a fungus.

Neonate Newly born (e.g. larva).

Non-persistent (virus) A virus that infects its vector immediately after uptake but is retained only for a few hours.

Nymph A juvenile form of an insect without wings or with incomplete wings.

Ocellus (pl. ocelli) A simple eye or eye-spot marking in insects.

Oogonium (pl. oogonia) Female sex organ of Oomycete fungi.

Oospores A resting spore developing from a fertilized oogonium.

Oviposition The deposition of eggs on a surface.

Papilla (pl. papillae) A small, pimple-like projection or protruberance.

Papillate Covered with papillae.

Parasitoid An organism that is parasitic in its immature stages and free-living as an adult.

Parenchyma/Parenchymatous (tissue) In tubers, soft plant tissue composed of thin-walled, relatively undifferentiated cells which contain starch grains.

Parthenogenesis Reproduction from a female gamete without fertilization by a male.

Parthenogenetic Organisms produced by parthenogenesis.

Periderm The surface layer or skin of a potato tuber. It comprises three layers: the phellem – several layers of heavily suberized cells very much flattened; the phellogen; and the phelloderm.

Phanerograms Seed bearing plants.

Phyllody Metamorphosis of an organ into a foliage leaf.

Plasmodium (pl. plasmodia) An amoeboid body of fungal zoospores.

Polymorphism The occurrence of different forms, stages or types in individual organisms of the same species, independent of sexual variations.

Primary infection (viruses) Infection where symptoms occur in the year of infection. *See also* secondary infection.

Proleg An unjointed abdominal appendage of lepidopteran larvae and some other arthropods.

Proliferated lenticels Lenticels where the structure is inverted and raised above the tuber surface.

Pycnidium (pl. pycnidia) A flask-shaped fungal fruiting body bearing pycnidiospores.

Pycnidiospore A spore produced from a pycnidium.

Pyriform Pear shaped.

Ridge A description of the soil covering the seed tuber. *See also* hill.

Ridging up The process of drawing soil up over seed tubers. *See also* hilling and earthing up.

Rose end The apical end of a tuber where eyes are concentrated. *See also* heel end, stolon end.

Rugosity (leaves) With many wrinkles or ridges.

Saccate Pouched.

Sclerotium (pl. sclerotia) Firm, rounded mass of fungal mycelium – a resting body which may survive for long periods. *See also* microsclerotia.

Secondary infection (viruses) Infection where symptoms appear generally the year after infection occurred.

Seed berry/ball The fruit of potato formed from the flowers, which contains true potato seed.

Septate mycelium Hyphal threads of a fungus comprising cells separated by a partition or septum.

Serotype Subdivision of a species distinguished by its antigenic character.

Seta (pl. setae) Chitinous hair.

Sexual dimorphism Marked differences in shape, size, colour, etc between males and females of a species.

Sorus (pl. sori) A fruiting structure in certain fungi in Uredinales and Ustilaginales (e.g. potato smut).

Sporangium (pl. sporangia) A fungal reproductive structure that forms asexual spores.

Sporangiophore A specialized mycelial branch that bears sporangia.

Sporeballs Thick-walled single or multi-celled resting spores of the order Plasmodiophorales (e.g. powdery scab). More correctly called cystosori.

Sterigma (pl. sterigmata) An extension of a basidium wall which bears basidiospores.

Stolon end The end of the potato connected to the stolon. *See also* heel end.

Teleomorph The sexual stage of ascomycete and basidiomycete fungi.

Teliospore or **teleutospore** The spore from which basidiospores are formed in Ustilaginales (e.g. potato smut). Also known as ustilospore.

Tilth The structure and texture of soil.

Trichome Hair-like outgrowths from the epidermis.

Vermiform Worm-shaped.

Vine A description of the stems, leaves and other plant structures produced above soil level. *See also* haulm.

Viviparous Producing live young rather than laying eggs.

Volunteer potatoes Tubers remaining in a field after harvest, which overwinter and grow in subsequent years. *See also* groundkeepers.

Wound periderm Periderm formed underneath a tuber wound to prevent water loss and ingress of pathogens.

Zoospores Motile, flagellated asexual spores that swim in free water.

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