POTATO STORAGE DISEASES

A. E. W. Boyd,

The Edinburgh School of Agriculture

The potato crop, with the exception of early varieties grown for immediate consumption, is stored in a variety of ways and for a variety of purposes. Each of the methods employed tends to be accompanied by a different spectrum of diseases. The most common method is the bulk store with or without artificial ventilation but 10 cwt (508 kg) pallet boxes are also commonly used. Some crops are still kept in outdoor clamps and, subsequent to separation of the ware, seed crops may then be stored in sprouting boxes or in hessian sacks. Small lots of 'pre-packaged' ware are stored in polythene bags.

For all practical purposes, with almost all storage diseases infection occurs prior to storage. It is introduced with or in the tubers and the incidence of gross symptoms is largely influenced by damage or by the storage environment. Perhaps with the exception of blackleg and other soft rotting bacteria, there is little evidence that tuber to tuber spread of a primary pathogen occurs during storage.

THE BACTERIAL SOFT ROT SYNDROME

Bacterial soft rot is the final symptom for which there are a number of causes. It is very often the result of infection by the coliform soft rot bacterium *Erwinia carotovora* (Jones) Bergey *et al.* var. *atroseptica* (van Hall) Dye (*E. atroseptica* (van Hall) Jennison), the organism responsible for typical 'blackleg' rotting of the basal parts of the stems of potato plants and which probably occurs in all countries where potatoes are grown.

Another organism of the soft rot coliform group, *E. carotovora* (Jones) Bergey *et al.*, is also involved in tuber decay in storage but is not, under normal circumstances, found associated with blackleg symptoms in the field. Dye (1969) proposed that *E. aroideae* (Townsend) Bergey *et al.*, sometimes mentioned in connection with tuber soft rot, be merged with *E. carotovora.* Isolations made from rotting tubers by Pérombelon (1968) showed that *E. carotovora* occurred to the extent of 17%, compared with 83% for the blackleg bacterium. Isolations made by Malcolmson (1959) from tuber soft rot and blackleg were in almost the same proportions. From this and other evidence it would seem that *E. atroseptica* is the predominant soft rot coliform bacterium associated with tuber rotting. Other bacteria, e.g. *Pseudomonas* sp. and *Bacillus* sp., although isolated occasionally, are not thought to be of general importance.

Methods for the isolation and identification of *Erwinia* spp. are described by Graham (1972) and the presence of *E. atroseptica* in potato stems and tubers can usually be determined serologically (Novakova, 1957; Graham, 1963) provided sufficient rotted tissue is available. The serological method is quick but is not completely reliable because some strains of *E. carotovora* give an agglutination reaction with *E. atroseptica* antiserum.

Blackleg infected plants usually develop from diseased seed tubers and in turn the progeny becomes infected, characteristically at the stem end by means of the stolons, but also through lenticels and damaged areas. Infection may also be contracted by stems which have been injured (Ramsey, 1919).

Bonde (1950) found that soft rot organisms may survive the winter in considerable numbers in soils in S. Carolina but to a very limited extent in Maine. He suggested that this was due to climatic differences and was not convinced that soil was the source of blackleg epidemics in the latter area. On the other hand there is considerable evidence from a number of countries that the blackleg organism does not overwinter in field soils (Ramsey, 1919; Graham, 1958; Lazar & Bucur, 1964; Logan, 1968). Even ploughing in of diseased plants did not provide a source of infection in the following year (Voronkevich & Butsevich, 1964). But although it is agreed that infection is carried over almost entirely by seed tubers, and Ramsey (1919) commented that all volunteer plants growing in spring are free from blackleg, Pérombelon & Lowe (1971) detected contamination by *E. atroseptica,* as well as *E. carotovora,* on most of the tubers produced by groundkeepers growing in barley 4

years after the previous potato crop, as well as from rotting carrots in land where potato groundkeepers were present.

Field symptoms of blackleg are well known and need no further description here. Their incidence, however, may be altered by a number of factors, some of which may have a bearing on subsequent storage performance of the crop. Symptom expression is reduced by increasing fertiliser application (Graham & Harper, 1966). Perhaps, in view of this, the diminishing nutritional status of the soil during the course of the growing season, or simply the maturity of the crop, is involved in the gradual increase in observed symptoms as the crop reaches maturity, so that a count of 3·1% in mid-July may have risen to 27·3% by the beginning of September (Graham & Harper, 1967). Blackleg symptoms may be increased in plants grown from seed which has been washed or in wet areas in a field or in seasons of heavy rainfall. Thus there is a variation from year to year (Graham & Harper, 1967) and this depends also on storage conditions of the seed rather than on the amount of disease in the previous crop (Logan, 1968).

Daughter tubers may be infected directly through the stolon end from infected plants. Such tubers may begin to rot before harvest, particularly under wet soil conditions, and at maturity it is the largest tubers which show the highest incidence of symptoms (Harper *et al.*, 1963). However, many tubers from diseased plants may show no obvious sign of infection and the problem of latent infection still remains.

Although the organism does not survive in soil over winter, it may easily be spread during the growing season by movement of soil water (Graham, 1962) and pectolytic *Erwinia* spp. have been detected more than 3 m along a row from naturally infected seed tubers (Pérombelon & Lowe, 1971). The main source of contamination is the decaying mother tuber and in this way contamination of lenticels occurs, the critical period under Scottish conditions being about the end of September (Pérombelon, 1969), less being detected in late October and November and none before mid-August.

The blackleg bacterium cannot penetrate the periderm directly but enters through wounds and lenticels, provided liquid water is present, so that cell proliferation ruptures the suberised lenticel plug. Having penetrated, the bacterium is confined to the intercellular spaces and rarely enters the host cells but dissolves the pectin layer of the middle lamellae, which causes cell separation and finally cytoplasm disintegration (Fox *et al.*, 1971).

In some seasons, when irregular rainfall induces second growth cracking in the tubers, it also encourages soil contamination by rapid breakdown of seed tubers and release of bacteria from the decaying stems. Growth cracks may be invaded in this way (Harper *et al.*, 1963) and present symptoms very similar to those of watery wound rot (*Pythium ultimum*).

Thus there is every opportunity for tubers to become at least contaminated if not infected before harvest and it was shown that virtually all tubers of all 23 commercial stocks tested were contaminated by *Erwinia* spp. (Pérombelon, 1969). This is probably further enhanced by contact with lifting machinery on which infected tubers have disintegrated. Mechanical damage to tubers in this process or on the separating riddle may also serve as points of entry for the bacterium.

The blackleg bacterium, *E. atroseptica*, is the most important pathological cause of primary soft rotting of stored potatoes under UK conditions, but there is no clear relationship between the disease in the field and storage rotting, since the storage environment greatly influences the development of rots. If affected tubers are present even in relatively low numbers the products of their disintegration may affect neighbouring tubers and the area of decay extends so that in a short time the whole of the stored crop may be lost. The active rotting is accompanied by considerable heat production and temperatures of up to 71°C have been recorded (Cotton, 1921).

Again, when the stored crop is being separated for ware or seed extraction, considerable delay, often of economic importance, may result from the necessity to clean the machinery and remove decayed tubers by hand. Furthermore, when such potatoes do disintegrate, the surrounding tubers may not only be extensively contaminated but also directly infected through wounds sustained on the riddle and they often rot when subsequently stored in sacks.

It has been shown that high levels of fertiliser decrease field symptoms of blackleg but tend to increase tuber soft rotting, particularly on growth cracks. It is possible that excessive amounts of fertilisers may, either by increasing tuber susceptibility or by delaying maturity, cause tubers to decay more readily in storage. What would appear to be circumstantial evidence for this was observed in 1970 during storage of many crops of the variety Pentland Crown in Scotland, since where extensive

soft rotting occurred it was associated with infection with *E. atroseptica*, often at the stolon end. Because of an atypical rainfall pattern, most fertiliser uptake had occurred at a relatively late growth stage and thus delayed maturity. Bétencourt & Prunier (1965) suggested that tuber immaturity at harvest encouraged lenticel rotting caused by *E. carotovora*. However, even although the blackleg organism is present in many of the decaying tubers, this is not unequivocal proof that it is the primary cause.

The effect of environment upon the rapidity with which tuber breakdown progresses once it has been initiated has been noted by many workers. Thus Nielson (1968) showed that bacterial soft rot was associated with accumulation of CO_2 which varied with the temperature of the respiring tubers and that, in practice, when potatoes were being transported the decay could be reduced by ventilation and reduction of temperature. Lund & Nichols (1970) were able to increase rotting by replacing air by nitrogen during experimental storage of tubers inoculated with *E. atroseptica*. Under anaerobic conditions they also noted that spreading soft rots were associated with the presence of *Clostridium* spp. and Rudd Jones & Dowson (1950), under similar conditions, found *Clostridium* sp. apparently acting together with *Bacterium carotovorum* (Jones) Lehmann & Neumann in the production of a gassy, pink rot. The exact identity of this last-named organism is not known; it could have been either *E. carotovora* or *E. atroseptica*.

The polythene bag environment of prepackaged potatoes together with immaturity of the tubers is conducive to decay by bacteria and Scholey *et al.* (1968) showed that the incidence of soft rot was higher when tubers were riddled, washed and bagged immediately after harvest rather than 8 weeks later. In Florida, control of the soft rot was obtained by subjecting the washed potatoes for 4 min to a current of air at 150°F (65°C) and pre-cooling them before transport (Ruehle, 1940).

Under relatively low temperature and humidity (5–10°C and 80% R.H.) the tuber forms a barrier of suberised cells and finally periderm to exclude further bacterial advance, but this may be penetrated when temperature and humidity are increased and bacterial multiplication encouraged (Rudd Jones & Dowson, 1950). In practice Burton (1963) contended that rotting does not spread from initial foci unless the temperature exceeds 4–7°C, depending on the organism, and there is a film of water on the tubers.

The progress of lenticel infection may be arrested by exposure to a dry environment and the affected tissues collapse to form a small usually circular 'pit rot' lesion. In some cases where tubers have been removed from areas of active rotting, the surface may be disfigured by many coalescing pits. Sometimes gangrene-like lesions known as 'hard rot' (Logan, 1964) develop in dry storage following the drying-out of superficial tissues which have been invaded by the blackleg bacterium after potatoes have been washed. Tubers in such a condition should never be used for seed purposes. When potatoes are washed before storage, as in Maine, bacterial lenticel infection can be a limiting factor, but can be reduced by using a 500 ppm chlorine solution with a biodegradable detergent (Wilson & Johnston, 1967).

Since the organisms responsible for the breakdown of potatoes in storage are invariably present on the tubers both in the field and in the store, control of deterioration involves careful growth of the crop, harvesting in reasonably dry conditions, a minimum of damage and attention to ventilation and temperature.

Careful husbandry includes the judicious use of fertilisers and the roguing as far as possible of affected plants. The latter may assist to some extent in removing obvious sources of infection in small stocks of high grade seed but since symptoms appear progressively the procedure becomes almost impossible to implement thoroughly.

Care at harvesting involves the lifting of tubers which are fully mature and minimising avoidable damage. It means the exclusion of loads of potatoes which have been subjected to rain between the field and the store, because in practice rapid tuber breakdown can often follow (Anon., 1970). There is a difference, as Burton (1963) pointed out, between the condition of such potatoes and those taken from wet soil. The film of moisture at the interfaces of already wet potatoes is very difficult to remove even by forced-draught ventilation and it is at these points that bacterial action starts. This is probably not due to asphyxiation of the tissues unless the film of water covers the whole surface (Burton & Wiggington, 1970) but perhaps pressure and waterlogging of lenticels play a part. If any danger from soft rotting is thought to be likely, it can be avoided or reduced by storage at about 4°C after the wounds have been healed (Burton, 1963). However, in practice, such temperatures may not easily be achieved in autumn except in refrigerated storage.

Storage in sprouting boxes or in 10 cwt (508 kg) pallet boxes usually allows adequate ventilation without special precautions, but in a bulk store ventilation is of primary importance in reducing the possibility of moisture deposition on tuber surfaces through respiration and evaporation. Where no air recirculation system is employed, the immediate application of an adequate layer of loose straw, about 0·5 m in thickness, over the top of the potatoes is essential (Burton, 1963) so that the moisture in the rising current of air will condense in the straw. Otherwise it is deposited in the cooler top 30–45 cm of the potatoes. Many cases of extensive bacterial soft rot have been initiated through neglect of this simple precaution.

As well as mechanical damage, tissue breakdown following low temperature injury during storage must be avoided since this allows entry of soft rotting bacteria, or at least stimulates the development of those already present. Bonde (1950) found that seed stocks injured by frost during storage produced a high incidence of blackleg. Also tubers affected by other diseases, such as blight, dry rot or pink rot, may act as foci of secondary bacterial soft rot if stored in wet conditions and according to Bonde (1950) lesions of *Phoma tuberosa* Melhus *et al.* and even powdery scab (*Spongospora subterranea*), allow entry of soft rotting organisms.

In view of the difficulty of controlling what is essentially a ubiquitous internal tuber-borne disease, the production in Scotland of stocks derived from blackleg-free stem cuttings gives promise of a widespread reduction in incidence of the disease. These virus-tested stem cutting stocks (VTSC) have been grown experimentally and then commercially since 1967 with considerable success, although with the constant hazard of recontamination from other crops by means of machinery and possibly birds and insects (Graham & Hardie, 1971). With the wider use of such stocks in future the potential sources of reinfection should be diminished.

BLIGHT—*PHYTOPHTHORA INFESTANS* (MONT.) DE BARY

Late blight of potatoes has two distinct phases, one on the haulm and the other on the tuber. Only the tuber aspect and factors immediately connected with this will be discussed here.

Tuber infection occurs commonly when spores (sporangia) produced on the infected haulm are washed down by rain water through the soil. They penetrate through eyes, lenticels or small wounds but not through the intact skin. Potato varieties differ in their susceptibility to infection through these avenues of entry, e.g. Arran Banner is more susceptible to lenticel infection while King Edward eyes are more readily infected (Lapwood, 1967; Lacey, 1967b). Entry to the tubers may also be gained through pustules of powdery scab (*Spongospora subterranea*) (Bonde, 1955).

Symptoms on tubers are first seen as brown irregular areas on the skin and these patches tend to become sunken during storage. Slightly infected tubers may readily be overlooked as the crop is being sorted, particularly if it is a coloured variety. Internally the affected tissues are dry rusty-brown, sometimes occurring in small superficial speckled patches not penetrating beyond the vascular ring and often not unlike the lesions caused by invasion by the tuber eelworm, *Ditylenchus destructor*. In other cases the infected areas extend in lobes into the central pith. In box storage tubers with blight lesions frequently sprout in advance of the healthy tubers but certain other rots or mechanical damage often have the same effect.

Tuber infection depends on a number of factors and susceptibility may not always be related to that of the haulm (Müller *et al.*, 1939). It varies during the development of the plant and is higher in immature tubers (Boyd & Henderson, 1953; Lacey, 1967b) possibly because lenticels become more resistant as tubers mature (Zan, 1962), for differential suberisation of lenticels was suggested by Löhnis (1925) as a factor influencing varietal resistance of tubers.

Rainfall obviously plays an important part in tuber infection which may be independent of the severity of the haulm attack and may be encouraged by irrigation (Lapwood, 1965a). If this occurs early in the season when tubers are immature, infection may be particularly severe. Such early epidemics often progress slowly at first and tubers are thus subjected to an extended period of potential attack so that a surprisingly high incidence of diseased tubers may develop. It follows therefore that any measure taken to delay the onset of the disease should tend to diminish the extent of early tuber attack.

Naturally a high spore load usually leads to an enhanced degree of infection and the spore concentration which reaches the tubers largely depends on the haulm susceptibility of the variety concerned and its potential for spore production on haulm lesions (Lapwood, 1965b).

The growth habit of some varieties influences the chances of tuber infection depending on the route whereby the rainwater spore suspension is channelled. With King Edward, for example, much of the water which runs down the stems becomes highly charged with spores from the abundant stem lesions characteristic of this variety (Lapwood 1964). Moreover, tubers of some varieties are formed on short stolons near the stem base or near the soil surface and these may be more exposed to infection. Thus King Edward tubers in the top 2 in (51 mm) are more frequently infected than those at deeper levels. In the shallow set tubers also, infection tends to occur at the apical end while in the deeper ones stolon end infection is more prevalent (Lacey, 1963, 1966). Again, in practice small tubers are more likely to be infected than large ones (Boyd, 1960) but it is not clear whether this is the result of immaturity or spacial arrangement in the row. However, there is usually a higher proportion in seed than in ware and thus in any direct comparisons, stocks should be assessed on equally sized tubers.

Resistance of tubers to blight in inoculation tests was reported to be increased by treatment of the seed tubers with superphosphate before planting (Mishchuk, 1964). Herlihy (1970) also recorded that phosphate application reduced and nitrogen increased the proportion of blighted tubers. He suggested that nitrogen application may have encouraged more extensive sporulation on the haulm and thus provided a greater chance of tuber infection or else, since it tends to retard tuber maturity, it may also increase susceptibility to natural infection.

A mechanical method of disease escape can be provided by well formed ridges which protect the developing tubers and a case can be made for the final ridging operation to be made some time after full emergence, for two reasons. If it is carried out soon after planting and application of the pre-emergence herbicide, ridges have a longer period in which to weather and to lose their form. Furthermore, if the final ridge is made before emergence, attack of the developing sprouts by *Rhizoctonia solani* is encouraged, particularly in light, dry soil.

The type of soil (de Bruyn, 1922) and its moisture content also influence the chances of tuber infection. Löhnis (1925) found that in sandy soils lenticel suberisation was greater than in clay soil and tuber infection correspondingly less. Attack of young tubers has been recorded where there were no symptoms on the associated haulm (MacAlpine, 1911; Löhnis, 1922; Hirst & Stedman, 1962b; Gray, 1965), the fungus presumably being transmitted from diseased seed tubers on which sporulation occurred in the soil. Soil infestation occurring earlier than the first symptoms on the leaves has also been noted by Fedotova & Bliznets (1969). Overwintering in soil of oospores of *P. infestans* formed parthenogenetically in potato tissues has been reported in USSR (Yurova, 1960; Malenev, 1962) but this has not been confirmed elsewhere than in Mexico. Lacey (1962) detected as many as 2100 sporangia per ml in surface soil after 0·77 in (19·6 mm) rainfall towards the end of the growing season. Infectivity could be detected at a depth of 8 in (2·03 mm) and was found in the surface soil 32 days after the haulm of the crop had been destroyed by sulphuric acid although there was a rapid decline after the first 7 days (Lacey, 1963). Under laboratory conditions, however, Murphy (1922) showed that contaminated soil maintained undiminished infectivity even after 44 days and suggested that persistence of the fungus is increased by sporangial germination in the soil. Zan (1962) and Lacey (1965) found persistence of infectivity for 11 weeks with different soils in the laboratory and Zan (1962) suggested that, in practice, cracks in the soil surface appear necessary for extensive tuber infection.

Murphy (1921a) first demonstrated that, by protecting tubers from infection from blighted haulm or contaminated soil, they remained virtually free from the disease. He also showed (Murphy, 1921b) that tuber infection was increased by omitting later applications in a protective spray programme, because although the life of the haulm was extended it eventually became extensively infected. Thus protective fungicide applications to the haulm may not always effect a reduction in tuber blight.

Chemical control of blight in the tuber has been approached in three ways; by the use of fungicides which both protect the haulm and affect the sporangia in the soil; by direct application of soil-acting fungicides; and by early destruction of infected haulm. Skaptason *et al.* (1940) recorded that the continued use of Bordeaux mixture in Long Island over periods of up to 32 years had increased the copper content of the soil to such an extent as to account for the low incidence of tuber blight in that area. A combination of foliar treatment with Bordeaux mixture and soil application of difolatan before the last row cultivation was found significantly to

decrease sporangial germination in the top 1–2 in (25–51 mm) layer of soil (Cetas & Leach, 1969).

Reduction of tuber infection has also been effected by the organo-tin compounds fentin acetate and fentin hydroxide, used as foliar sprays in comparison with copper oxychloride and dithiocarbamates (Last, 1961; Holmes & Storey, 1962; Jarvis *et al.*, 1967). Dusts containing fentin acetate and triphenyl tin chloride applied to soil ridges in June or July have also given significant decreases (McIntosh, 1965) although protective sprays, directed to the lower parts of the stem, or ridge application of skin-forming materials were unsuccessful (McIntosh, 1969). In Northern Ireland, incorporation of sulphur into the soil (McCreary, 1967) before planting to give a pH of *c.* 5·1 controlled tuber blight and some other tuber diseases almost completely.

It is generally agreed that a protective spray programme should always be completed by chemical or mechanical destruction of the haulm and that tubers should not be harvested while any green leaf or portion of stem which potentially can harbour the disease is present (Murphy & MacKay, 1925; Bonde & Schultz, 1949). There have been many instances of extensive harvest infection of tubers where this advice has been ignored. Furthermore, sufficient time must be allowed after destruction of the haulm for soil contamination to subside. A minimum of 10 days has been suggested but normally this period may be as long as 2–4 weeks, particularly for seed crops, although this increases the risk of *Rhizoctonia* contamination of the tubers.

The timing of the haulm killing procedure can be critical, particularly with tuber-susceptible varieties, and it has been suggested that with a variety such as King Edward the yield of healthy tubers does not increase after haulm blight has reached the 5% (BMS scale) stage (de Lint, 1961; Lacey, 1963; Hirst *et al.*, 1965). It would seem likely that this could depend on subsequent weather conditions, and contrasting experimental results given by Boyd *et al.* (1967) for two successive years illustrate this. In practice it is difficult to persuade growers to implement the advice to destroy haulm which is 95% blight-free.

Mechanical destruction of potato haulm by means of a rotabeater or rotaflail is frequently employed, but sulphuric acid remains the most efficient chemical method although many other substances such as sodium chlorate, dinoseb in oil and diquat are used. Under very dry and warm soil conditions, application of some compounds for this purpose may be followed by stem end necrosis of the tuber or even rotting and a warning system with regard to the application of diquat and based on soil moisture deficit was described by Headford & Low (1970).

The disease continues to develop in storage but does not spread from tuber to tuber (Murphy, 1921b) and further advance of the fungus may be completely inhibited by storage at 2°C (Müller *et al.*, 1939). It has been suggested (Murphy & MacKay, 1925) that all lesions become visible within the first month of clamp storage, although there is less development when the potatoes are kept in the drier environment of boxes. Some lesions may remain small and difficult to observe when the tubers are graded and Gray (1965) found that symptoms continued to appear until late in the storage season.

Tubers may be freed from blight infection by subjecting them to hot air treatment at 40–45°C for 1–8 hours (Førsund, 1960) and, under dry storage conditions, the tissues of blighted tubers usually become desiccated and turn brown (Wilson & Boyd, 1945) or may be invaded by dry rot type fungi, often *Fusarium* spp. Thus, if the presence of tuber blight is suspected at the time of lifting, great care must be taken so that by adequate ventilation no excessive condensation is allowed. Such a case was observed recently in which a store containing 400 tons of the variety Kerr's Pink with 15% blighted tubers in November was maintained in a dry condition with no further deterioration until spring.

In a damp storage environment, secondary invasion by bacteria may cause rapid breakdown of the tissues. Dowson & Rudd Jones (1951) noted the development in blighted potatoes of free liquid in the diseased areas. Infected tissues had a higher sugar content and osmotic pressure than healthy tissues and this liquid which often appeared on the tuber surface was able to support the active growth of *Erwinia carotovora* and other bacteria which penetrated the blighted tubers and caused soft rotting.

Varieties carrying major gene resistance in haulm and tuber have been developed but when these are intensively cultivated and grown in conditions of high spore intensity they may be overcome by new virulent races of *P. infestans.* An instance of this occurred in 1968 with the R_1 R_2 R_3 variety Pentland Dell, tubers of which were very heavily attacked over a wide area in the UK (Malcolmson, 1969).

The physiological basis for tissue resistance of tubers to infection has been the subject of extensive research and several phenolic substances have been shown to be

important. Also the activity of the enzyme polyphenoloxidase increases after infection of a resistant variety but not of a susceptable variety (Sokolova *et al.*, 1958). Moreover, when tubers of a resistant variety are inoculated with an avirulent race of *P. infestans* the cells in areas near the site of the inoculation are stimulated to form phytoalexins (Müller & Börger, 1941), nonspecific compounds which inhibit penetration even by races of the fungus to which the tubers are normally susceptible.

Losses caused by blighted tubers vary greatly with season and variety and even from crop to crop but have been estimated for all varieties in England and Wales in the mid 1950s to be about 3% of the crop (Cox & Large, 1960). In USSR, Belozor (1960) noted that annual losses from blight amounted to 2–2·5 million tons which may be calculated as about 1 ton per acre; and in India from 1961 to 1963 losses were estimated to be about 40% in the Simla Hills (Dutt, 1965). By any standard, losses on such a scale are still unacceptably high and although such a volume of research has been undertaken on blight control the disease continues to be one of the chief sources of loss of potato tubers in most areas where the crop is grown.

Blight control by breeding is now concentrated upon the search for a high degree of polygenic or field resistance both in haulm and tuber, possibly in combination with major gene resistance, but even now the blight problem could be minimised by a reduction in the cultivation of highly tuber-susceptible varieties.

PINK ROT—*PHYTOPHTHORA ERYTHROSEPTICA* PETHYBR.

Pink rot occurs in many areas where potatoes are grown. Infection of the stem base and stolons takes place in contaminated soil producing wilt symptoms (Cotton, 1922) but this occurs mostly towards the end of the growing season and may be confused with natural senescence or other diseases. Tuber infection characteristically occurs through the stolon but the fungus may also penetrate directly through an eye, particularly in very wet soil (Cairns & Muskett, 1939) and in advanced cases the tuber shows a dark discoloration of the lenticels (Jones, 1945). The invading mycelium is intercellular and the tissues remain coherent.

Symptoms are often not noticed until harvest; affected potatoes are rather soft and rubbery as if they had been severely frosted. When the tuber is cut the diseased tissues are off-white or yellowish but become uniformly salmon pink after exposure to air for about 30 min. This symptom provides the descriptive name for the disease but it may not be observed in every case and the pink cut surface later becomes dark purple or black on further exposure. The rot starts typically at the stolon end and is most frequently observed in the largest tubers (Van Haeringen, 1938; Boyd, 1960). Entry of the fungus may also occur through wounds or possibly contact with diseased potatoes under suitable conditions in storage.

White (1946) observed that the pink coloration is found in the zone of invaded but dead tissue where the fungus survives saprophytically, and is formed in an oxidising reaction by tyrosinase.

Occasionally atypical brown necrotic symptoms similar to those of blight (*Phytophthora infestans*) have been observed during the growing season on immature tubers, sometimes associated with small cracks.

Other *Phytophthora* spp. have been shown to cause typical pink rot symptoms either naturally, as with *P. megasperma* (Cairns & Muskett, 1933a) or experimentally, as with *P. cryptogea* and *P. cactorum* (Cairns & Muskett, 1933b).

Development of the disease occurs fairly rapidly, particularly under warm soil conditions. However, at harvest, many of the slightly affected tubers may not be noticed and, as they may not readily disintegrate, they are included among the stored potatoes. With the rise of temperature during early storage observable rotting is hastened and the affected tissues finally decay with bacterial soft rots. High relative humidity and poor ventilation also encourage rapid decay in storage and Cairns & Muskett (1933b) recorded up to 50% loss in this way. Since it is generally the larger tubers which are attacked, the effect of the spread of decomposed tissues on to the surrounding potatoes is out of proportion to the number of tubers originally affected. The results are therefore similar to those of the inclusion of blackleg affected tubers in the delay caused by removing rots by hand and cleaning machinery when potatoes are being taken from store.

It is generally accepted that pink rot is a disease of wet soil conditions and it has often been recorded from wet patches in fields. It was found to be prevalent in the Netherlands in reclaimed marsh land especially in dry, warm seasons, when soil temperature was high, e.g. 20°C or more at 20 cm, and water was withdrawn from the subsoil (van Haeringen, 1938). In UK, pink rot is often associated with

such seasons in soils with a heavier texture which, however, maintain the moisture likely to encourage the pink rot fungus.

Oospores of *P. erythroseptica* are formed in the roots, stolons and stem bases of affected plants but not frequently in the tubers. Recontamination of the soil is believed to be maintained by the oospores in the breakdown of these tissues and de Bruyn (1922) noted that the fungus could overwinter in soil. Cairns & Muskett (1933b) believed that the fungus could persist indefinitely in the soil, even in the absence of the potato, although it is a poor competitor in field soil. Vujicic & Park (1964) considered that it is unable to colonise dead plant material and persists in soil as oospores. The fungus can attack tulips (Buddin, 1938) and it is possible that it may survive on the seedling roots of other plants such as wheat, peas and creeping thistle (*Carduus arvenis*) (Whelan & Loughnane, 1969). In practice the disease is not infrequently found quite severely attacking potatoes in fields which have not grown this crop for 7 years.

Transmission by or with tubers was suggested by Cotton (1922) and Blodgett (1945). It is unlikely that infected potatoes are implicated in this respect because of their rapid breakdown, although Cairns & Muskett (1933b) recorded systemic infection causing blanking and 'pink rot wilt' after planting. Transmission by soil on the surface of seed tubers is possible although it has never been demonstrated experimentally. Cotton (1922) blamed the use of contaminated manure containing vegetable debris in the spread of pink rot and Blodgett (1945) stated that heavy application of sheep manure or ploughed-in lucerne increased infection.

Application to the soil of sulphur (McKee, 1968) and copper oxychloride (Blair, 1959) has given some reduction of pink rot and crop rotation, destruction of diseased refuse and careful selection of tubers before storage have all been advocated to control the disease, but some of these measures are hardly feasible in practice. The use of sprouted seed and early harvesting (Cairns & Muskett, 1933b) would seem to conflict with the growing of varieties which retain luxuriant foliage until late in the season (van Haeringen, 1938). The most satisfactory control appears to lie in some method of improving the structure of the soil, whether this be by effective drainage, by liberal application of stable manure or by increasing the permeability of the sub-soil as suggested by van Haeringen (1938). It may be that the continual use of heavy machinery on normally heavy soils will encourage the incidence of pink rot but it is not yet known whether sub-soiling will act as a satisfactory means of control.

WATERY WOUND ROT—*PYTHIUM ULTIMUM* TROW

This disease, originally attributed to *P. debaryanum*, can cause extensive and rapid rotting of potato tubers immediately after harvest. It does not affect the other underground parts but follows upon damage, principally that made during lifting operations. The fungus does not penetrate the unbroken skin (Pethybridge & Smith, 1930; Jones, 1935), but having invaded the tissues the mycelium is both inter- and intracellular (Pethybridge & Smith, 1930). Tissue disintegration, therefore, occurs very rapidly under warm and humid conditions at harvest and during early storage, e.g. 20°C or above (Brien, 1940), or in transport. Larger and immature tubers are generally more likely to be affected, possibly because of their greater liability to sustain gross wounds.

Typically the central pith tissues are rotted and become soft and watery very quickly and have a greyish or sometimes pink or finally almost black coloration, usually with a dark zone at the edge of the diseased area. This, however, is not entirely diagnostic because such colour changes can occur with other diseases, e.g. blackleg. With pink rot, on the other hand, the diseased area is usually more uniformly discoloured and is initially coherent. Because the outer cortex remains intact, watery wound rot is sometimes known as 'leak' or 'shell rot' (Blodgett & Ray, 1945).

In North America the disease also causes rotting of cut setts after planting, particularly in wet and heavy soils; the use of some fungicidal dusts has given only limited success (Newton & Lines, 1947).

P. ultimum is soil borne and has a wide range of host plants, including turnip, carrot and beet (Jones, 1935; Tomkins *et al.*, 1939). No reproductive bodies have been found in diseased potato tissues but oospores can be formed in soil when the moisture content is high and the soil pores are water-filled (Bainbridge, 1970).

In UK, watery wound rot is occasionally troublesome but seldom presents a serious storage problem, possibly because temperatures at the time of normal harvest

are not sufficiently high. The progress of the disease is retarded by storage at 40°F (4·4°C) (Anon., 1950).

DRY ROT—*FUSARIUM* SPP.

Various species of *Fusarium* are responsible for causing symptoms of a dry rot type. In UK and Europe generally the most common is *F. solani* var. *coeruleum* (Sacc.) Booth (*F. coeruleum* [Lib.] Sacc.), although *F. avenaceum* ([Corda] Fr.) Sacc. is sometimes involved and occasionally *F. arthrosporioides* Sherb., *F. tricinctum* (Corda) Sacc. (McKee, 1952), and *F. sporotrichioides* Sherb. and *F. oxysporum* Schlecht. (Upstone, 1970a, b). In North America dry rot is mostly associated with *F. sulphureum* Schlecht. (*F. sambucinum* Fuckel f.6 Wollenweber) but *F. s.* var. *coeruleum* is also very common and *F. trichothecioides* Wollenw. *F. oxysporum* and *F. solani* (Mart.) Sacc. are also found.

Symptoms of infection with each of the *Fusarium* spp. show minor differences. With *F. s.* var. *coeruleum* the lesion is externally dark brown without necessarily a clearly defined edge. In dry conditions, such as in sprouting trays, the tissues shrink and the skin develops a series of concentric wrinkles round the site of infection. The affected flesh is usually fawn or light brown, often with darker streaks and the margin of the rot is diffuse and merges into the healthy area. Because of the moisture loss from the tissue, cavities often lined with mycelium are formed below the site of infection. Sporodochial pustules are frequently formed on the tuber surface and these may be pink or white when exposed to light and blue, or at least with a blue base, when light is excluded as in clamp storage. In damp conditions, e.g. in a clamp or store, there is much less desiccation and a more rapid advancement of the rot (Moore, 1945), while secondary soft rotting organisms hasten the decay of the tubers.

Symptoms caused by *F. avenaceum* are externally often similar to those of *F. s.* var. *coeruleum* but internally the tissues become grey-brown or black with a sharper margin between healthy and affected areas. Sporodochial formation is less common but occasionally typical red mycelium is found in the cavities (McKee & Boyd, 1952) and the optimum temperature for spread of lesions (20–25°C) is higher than for *F. s.* var. *coeruleum* (15–20°C). The latter acts as a true parasite with mycelium which is intercellular and adjacent host cells which remain alive for some time, but with *F. avenaceum* the advancing mycelium kills and penetrates the cells (McKee, 1954).

F. sulphureum causes a dry, rather mealy, brown rot with a fairly well defined edge and extensive cavitation of the tissues. Externally the lesion is somewhat similar to those of gangrene (*Phoma exigua* var. *foveata*) and dry rot (*F. s.* var. *coeruleum*) and internally the appearance has many of the characteristics of gangrene. This has so far been recorded only once in UK (Boyd & Tickle, 1972).

There are two aspects to rotting caused by Fusaria. The first is as a storage rot and the second is a 'seed-piece decay'. For all practical purposes the dry rot fungi are wound parasites and rotting from this cause is never observed before lifting, either in growth cracks or through slug or wireworm damage (Foister *et al.*, 1952). However, Pethybridge & Lafferty (1917) obtained experimental infection by *F. s.* var. *coeruleum* through lenticels, eyes, sprouts and through 'scab' pustules (most likely those of *Streptomyces scabies*, although this is not stated). Foister *et al.* (1952) obtained no infection this way, although natural infection associated with powdery scab (*Spongospora subterranea*) was not uncommon.

Small (1944a) and Foister *et al.* (1945a) showed that *F. s.* var. *coeruleum* was present in a viable condition in soil both in the field and on the tuber surface and damage to tubers then brings contaminated soil in contact with the exposed tissue. In practice, this damage occurs (i) at harvesting, (ii) on grading, (iii) during transport, (iv) in handling when seed tubers are boxed and (v) in cutting if, as in North America, seed pieces are used for planting.

A very important factor is the fluctuation of tuber resistance. Many workers have noted the increase in susceptibility with tuber maturity (Pethybridge & Lafferty, 1917; Moore, 1924; Small, 1945; Lansade, 1949). At the time of harvest tubers appear to have a maximum resistance after inoculation and storage at 15°C in optimum conditions for wound healing. Susceptibility rises throughout storage to a maximum in spring (Boyd, 1952b), although less rapidly with *F. avenaceum* than with *F. s.* var *coeruleum* (McKee, 1954). A high peak of susceptibility also occurs in immature tubers in the post-flowering period. When the haulm is removed at that time, however, susceptibility falls very quickly (Boyd,

1967) and is closely related to the content of sucrose, which temporarily accumulates in the tuber because of the vigorously growing haulm. Cases where dry rot develops on seed tubers harvested early with no delay after haulm removal can be explained on this basis. After harvest, increase in the content of mainly reducing sugars is accompanied by increase in susceptibility, but these are not directly related.

Under normal conditions in UK, mechanical damage at the time of harvest does not lead to excessive rotting during storage where the initial temperature is allowed to rise in the curing period. However, if the damage is followed by low temperature storage, rotting develops more extensively although much more slowly (Boyd, 1952d). Wounds are decreasingly open to infection for several days and McKee (1954) suggested that the increase of resistance was not simply due to suberisation in the wound healing process but to resistance of the cells near the wound, for lesions resulting from delayed inoculations are smaller than those developing on fresh wounds. He suggested that the initial response of the tissues to infection by Fusaria is similar to that due to *P. infestans*, for Müller & Börger (1941) had shown that the phytoalexin formed after inoculation with an avirulent race of *P. infestans* also restricted the growth of *F. s.* var. *coeruleum*. Wound healing as such is also important and inhibiting factors, e.g. IPPC (McKee, 1955) and MANA (Cunningham. 1953), considerably reduce resistance.

Under UK conditions, the highest incidence of dry rot follows wounding in the riddling process (Foister *et al.*, 1952) which is normally done between November and March while potatoes gradually become more susceptible. In this operation almost every tuber is damaged to some extent, particularly if the riddle screen is of unprotected wire mesh (Foister *et al.*, 1952) and McKee (1954) showed that scarified wounds were more susceptible than clean cuts. In practice the potatoes, having been riddled, are bagged and transported and only several weeks or even months later, depending on conditions, does dry rot become evident. Hence it is primarily a disease of seed potatoes since ware is normally utilised immediately and, although large tubers are, in fact, more susceptible than small ones (Boyd, 1952a), this is not simply because they may be more liable to damage. It follows, therefore, that to re-store ware potatoes or to re-riddle seed stocks is potentially to encourage heavy losses.

Extensive infection of seed of susceptible varieties is often caused if the riddling is carried out not long before planting and the planting delayed by inclement weather or followed by dry conditions in the soil. The rotting continues in the soil, and depending on the lesion size at planting can cause a high incidence of blanks particularly in the variety Redskin.

Damage can be further inflicted during transport (Foister *et al.*, 1952) or in sprout removal in handling the seed tubers before planting (Mooi, 1950). *F. s.* var. *coeruleum* spores may also be present in the air in potato stores (Pethybridge & Bowers, 1908; Small, 1945; Lansade, 1949) or in dust (Small, 1945) or as contamination in sprouting trays (Pethybridge, 1917) but these sources present a negligible risk unless the tubers have been freshly wounded (Small, 1944b; Foister *et al.*, 1952). Even so, Martinović (1961) noted that disinfection of storehouses with 5% formalin provided a good control.

The last occasion of injury to tubers is just before planting and is carried out deliberately in cutting tubers to provide 'seed pieces'. This procedure is widely adopted in North America but rarely undertaken in Europe. *Fusarium* spp. are one of the chief sources of 'seed piece decay' (Cunningham & Reinking, 1946). When cutting seed was carried out experimentally with the susceptible variety Ninetyfold (Small, 1944b), infection by *F. s.* var. *coeruleum* was increased seven-fold and disinfection with an organo-mercury compound immediately afterwards gave little control.

The *F. s.* var. *coeruleum* infectivity of field soils stored in the laboratory can be maintained for at least 5 years (McKee & Boyd, 1952) or in one case for almost 10 years (Boyd, 1970), and Schippers (1962) noted that soil from tubers on the riddle was usually more highly contaminated than that in which the potatoes had been growing. Boyd (1971) found a very high degree of infectivity both of *F. s.* var. *coeruleum* and *P. e.* var. *foveata* in soils associated with potato riddles. Planting seed affected by dry rot leads to increased contamination of the soil as assessed at harvest, and also of the soil on the surface of the young tubers with consequent dry rot infection (Ayers & Robinson, 1956; Nadvodnyuk, 1960; Boyd, 1970). The disease may thus be perpetuated by planting diseased or contaminated seed.

Dry rot has been recorded as being more severe after dry and warm growing seasons which Mooi (1950) considers is related to the effect of higher temperature in advancing the rate of maturity and thus of susceptibility. Ayers & Ramsay (1961)

concluded that the warm, dry soil favoured the propagation of the pathogen, in their case *F. trichothecioides*, while Lansade (1949) suggested that the operative factor was the higher dry matter content of the tuber associated with such conditions. Tubers may also sustain more damage during lifting from dry soil.

In many respects susceptibility does appear to be associated with higher dry matter content, e.g. the stolon end of a tuber is more susceptible than the rose end (Boyd, 1952a) and smooth-skinned Olympia tubers which had a higher dry matter content are more susceptible than rough-skinned tubers (Schoene, 1967). Again, susceptibility is reduced together with dry matter if the growing period is shortened either by late planting or early haulm removal (Boyd, 1967). However, application of nitrogen alone, which lowered dry matter content, also tended to increase susceptibility (Schippers, 1962; Boyd, 1967), so that any such association would appear to be indirect.

Some substances present in the tuber before infection, e.g. chlorogenic acid (Bate-Smith, 1956) and some produced by infection, e.g. rishitin (Metlitskii & Ozeretskovskaya, 1970) inhibit the growth of *F. s.* var. *coeruleum* and *F. solani* respectively *in vitro*. However, in spite of a number of investigations, the nature of the defence mechanism against *Fusarium* infection is still not clear and neither solanin nor chaconine (McKee, 1961), sugar content (Moore, 1924; Boyd, 1967), osmotic pressure (Radtke, 1969) nor orthodihydric phenols (Griffin, 1964) have been directly associated.

One of the most obvious variables in dry rot susceptibility is the varietal reaction which has been mentioned by many workers; e.g. Ayers & Ramsay (1961) noted that economic losses from dry rot (*F. sulphureum*) had not been encountered in Prince Edward Island before the introduction of the variety Sebago.

Boyd (1952c) distinguished two factors in the assessment of field susceptibility of varieties, the physiological resistance of the tissue to infection and the mechanical resistance of the periderm to wounding. In standard tests, Arran Victory and Arran Banner were the most highly resistant and Catriona and Dunbar Standard the most susceptible of a wide range of British varieties to *F. s.* var. *coeruleum*. Varietal susceptibility to different Fusaria appears to vary, e.g. Doon Star is more susceptible to *F. s.* var. *coeruleum* and King Edward to *F. avenaceum* (Moore, 1945). Ayers (1961) also found differences in varietal reaction to *F. sulphureum* and *F. s.* var. *coeruleum*.

Extensive losses during storage and as 'seed piece decay' involving different tuber-rotting Fusaria have been documented from many parts of the world. In UK, such heavy losses of seed tubers were experienced, prior to planting, by growers of the susceptible variety Doon Star that the seed acreage in Scotland dropped from 12,000 in 1942 to 2000 3 years later (Boyd, 1952c). Another aspect of loss caused by planting infected seed is that, even where emergence is not reduced, the plants may show a higher incidence of blackleg (Conroy, 1958; Boyd & Logan, 1967).

Satisfactory chemical control of dry rot can be achieved by the use of organo-mercury compounds as disinfectant solutions with seed tubers separated immediately after harvest (Foister, 1940; Foister & Wilson, 1943; Boyd, 1960) or on certain other occasions depending on the time of bruising (Small, 1945, 1946). Even washing tubers immediately after lifting can remove much of the inoculum (Boyd, 1960). Where seed piece decay is involved, treatment must also be effective against invasion by other fungi and bacteria as well as *Fusarium* spp. Control has been effected by the use of dithiocarbamates, organo-mercury compounds (usually semesan-bel, (Cunningham & Reinking, 1946)) with and without antibiotics, although agrimycin increases susceptibility to *Fusarium* rot (Bonde & Hyland, 1960).

Dusting tubers with thymol (Foister *et al.*, 1945b) or better with tecnazene (Anon., 1948; Foister & Wilson, 1950; Harrison & Downie, 1960) at the time of lifting has also provided effective control and the latter acts also as a sprout depressant. McKee (1950), however, obtained resistant strains of *F. s.* var. *coeruleum* which were induced by exposure to this compound. Leach (1971) found that both dusts and dips containing benomyl, thiabendazole or zinc+maneb effectively controlled dry rot (*Fusarium sambucinum*) in wounded and inoculated tubers.

The antibiotic activity of *Trichoderma koningii* has been utilised in the USSR in the application of a conidial suspension to tubers before storage and Nadvodnyuk (1962) claimed 50% reduction in infection.

In UK since about 1950 the importance of dry rot has considerably declined and *Phoma exigua* var. *foveata* has become the dominant dry-rot type of organism affecting seed tubers. The reason for this is still not clear and *Fusarium*-susceptible varieties are still grown. It has been shown (Ivashchenko, 1962; Boyd, 1967) that NPK fertiliser application reduces susceptibility to dry rot and MacKenzie (1968) showed

that susceptibility to gangrene in artificial inoculations was enhanced by low rates of NPK fertilizer, but increased NPK application made tubers more susceptible to damage and thus increased the amount of gangrene which developed from natural infection. It may be that changes in fertiliser practice have played some part for, when the incidence of dry rot was severe during and just after the second world war, the rate of NPK fertiliser applied to seed crops was considerably less than that given at present.

GANGRENE—*PHOMA* SPP.

Gangrene is the most serious fungal storage disease of seed potato stocks in UK at this time. Symptoms are in some respects similar to those of dry rot (*Fusarium* spp.); the two diseases were first differentiated by Alcock & Foister (1936) in Scotland, where gangrene had probably been present unrecognised for many years. The causal fungus was described and named *Phoma foveata* Foister (Foister, 1940b). *P. foveata* has now been recorded from Australia (Harrison, 1959), Tasmania (Sampson & Fountain, 1960), West Germany (Kranz, 1958), Netherlands (Boerema, 1967) and Rumania (Puşcaşu & Popescu, 1970). Similar symptoms have been recorded in W. Europe, USSR, USA and Canada and attributed to other *Phoma*-type fungi, *P. eupyrena* Sacc., *P. tuberosa* Melh. Rosenb. & E. S. Schultz and *Phomopsis tuberivora* Güss. & Foster. The condition known as skin necrosis has been associated with both *P. foveata* and also *P. eupyrena* (Foister, 1952; Malcolmson & Gray, 1968a).

Recently there have been several changes in nomenclature and the generally accepted name of the causal fungus is now *P. exigua* var. *foveata* (Foister) Boerema (=*P. foveata*=*P. solanicola* f. *foveata* (Malcolmson, 1958)=*P. exigua* f.sp. *foveata* (Malcolmson & Gray, 1968c)). This is a pigment-producing variety and is thus differentiated from *P. exigua* var. *exigua* (=*P. solanicola*=*P. tuberosa*), while *P. eupyrena* is regarded as being non-pathogenic to potato (Boerema & Höweler, 1967; Logan & Kahn, 1969), although it can be associated with gangrene lesions and both Foister (1952) and Kranz (1963) noted that it could attack tubers.

In UK, *P. e.* var. *exigua* can also cause gangrene, but not commonly. Logan (1967a), Todd & Adam (1967) and Fox & Dashwood (1967, 1968) all found that about 90% or more of gangrene lesions are caused by *P. e.* var. *foveata.*

Furthermore, two strains of *P. e.* var. *foveata* may be identified, only one of which produces an antibiotic substance 'E' (Logan & O'Neil, 1970).

Gangrene symptoms usually take the form of black, circular, thumb-mark type lesions, or larger, irregularly shaped depressions on the surface of the tuber where pycnidia may develop in damp conditions. The extent of the lesion is often not related to the depth of tissue invasion, and small superficial lesions may hide extensive and deep penetration with variously shaped cavities. These are often lined with dark brown or deep purple mycelium in which pycnidia are sometimes found. The diseased tissues are typically black and well defined from the healthy area and the cavities may be surrounded by only a narrow black or brown diseased edge. Deep lesions tend to be associated with warm storage conditions and shallow lesions, particularly of the skin necrosis type, with low temperature (Malcolmson & Gray, 1968a). Skin necrosis lesions are completely superficial, extensive, dark and irregularly shaped (Foister, 1952), not unlike those caused by tobacco necrosis virus.

One of the main differences between dry rot and gangrene concerns infection of the haulm. Pycnidia of *P. e.* var. *foveata*, *P. e.* var. *exigua* and *P. eupyrena* may all be detected on dead or senescent haulm (Malcolmson, 1958), the first mentioned being usually predominant where gangrene-affected seed tubers have been planted (Todd & Adam, 1967; Logan, 1967a) and *P. e.* var. *exigua* being predominant when the seed is free from gangrene contamination. The pycnidia on the haulm can provide an ample source of pycnospores which can be washed into the soil to act as an inoculum on the tuber surface. This may explain Foister's observation (1952) that gangrene was most prevalent after excessive rainfall in 2 of the 3 months August to October. The importance of this is illustrated by Gray & Malcolmson (1966) who noted that the time of haulm death or haulm removal before harvest influences the pre-harvest infection of the tubers. A high incidence of tuber infection followed inoculation of the haulm with *P. e.* var. *foveata* but susceptibility of the haulm is not related to that of the tubers (Fox & Dashwood, 1969; Fox *et al.*, 1970). Stems may be infected at any time, but the fungus is usually localised while the stems are green, and in this connection, the observation (Fox *et al.*, 1970) of the presence of pycnospores in the transpiration stream is of interest. Infection does not become

obvious until the beginning of senescence and the formation of pycnidia on the moribund parts of the stems (Khan & Logan, 1968). It is of considerable significance that pycnospores may be released gradually throughout the harvest period (Todd & Adam, 1967). In respect of pycnospore production gangrene may be regarded as splash-borne if not wind-borne and no doubt neighbouring plants can be infected but there is no record of the extent of such spread.

The tuber-borne nature of gangrene is quite clear; the infected seed tuber can produce diseased haulm, pycnospores from which can infect tubers of the following crop. Foister & Alcock (1936) showed that gangrene could be induced without wounding the tubers and Malcolmson (1958) obtained infection through lenticels, particularly those which were proliferating, and also through eyes. Not only so, but exposure to contamination may lead to a degree of true latent infection where the periderm is penetrated but no further development occurs. The tuber appears healthy although the fungus can be recovered from within the periderm in viable condition (Todd & Adam, 1967). Sometimes *P. e.* var. *exigua* and *P. eupyrena* can be isolated also in this way.

In practice, most of the gangrene which develops in seed stocks is associated with damage and the disease development may not reflect the initial inoculum. Wounding the tuber acts like an inoculation with contaminated soil on the tuber surface or with the fungus already in the periderm. As with dry rot, wound infection may occur at lifting, grading, transport or at any time of handling. Gangrene appears to follow lifting injury more than does dry rot and the reason for this is not clear. Possibly pre-harvest latent gangrene infection may be activated by lifting wounds. Gangrene incidence is increased by lifting machinery which is likely to increase tuber damage but it is not so frequent on clean cuts (Malcolmson & Gray, 1968a, b). Apart from lifting, the degree of riddle wounding (Griffith, 1969) is very important in relation to gangrene, as indeed it is to dry rot.

Establishment of infection is considerably influenced by temperature and, because of the inhibition of wound periderm formation (Kranz, 1958), exposure to cold conditions immediately after the tubers are damaged increases the incidence of the disease (Malcolmson & Gray, 1968), particularly if they had been stored warm before inoculation (Malcolmson, 1958a; Kranz, 1959b). The maximum amount of rotting occurs in a dry environment at 5°C (Malcolmson, 1958a; Kranz, 1959c) and wounds are open to infection at this temperature for at least 4 days (Malcolmson, 1958) while storage at 21°C for 10 days can arrest rotting.

During the growing season tuber susceptibility tends to be high in the early stages of development but gradually decreases towards maturity (Fox *et al.*, 1970) and finally rises again during storage (Malcolmson, 1958; Kranz, 1959c; Fox *et al.*, 1970). Malcolmson & Gray (1968a) suggest that factors which retard the maturing of tubers at harvest, possibly the skin-setting process, influence the extent of pre-lifting infection. For example, although increasing rates of NPK fertilisers reduce tissue susceptibility, the incidence of natural infection can be increased possibly by greater liability to mechanical damage or by delaying maturity of the tubers (Mackenzie, 1968).

Gangrene is also soil-borne and contamination with *P. e.* var. *foveata* (Foister *et al.*, 1945a) was confirmed more convincingly by Malcolmson (1958) using fresh soil with higher moisture contents, and by Kranz (1958). Malcolmson & Gray (1968a) considered that this aspect of the disease was more important than the influence of infected haulm and showed that the distribution of the disease was related to soil moisture. It did not depend on previous cropping and the fungus could be recovered from soil where no potatoes had been grown for 7 years. Khan & Logan (1968) recorded at least 5 years. It has been suggested also that moister soils may tend to delay tuber maturity and so make tubers more susceptible to infection. In spite of this, potatoes harvested from wet soils often develop less gangrene since they tend to be buffered against the damage of the lifting machinery (Malcolmson & Gray, 1968a) but this was not confirmed in detail by Khan & Logan (1968).

Although *P. e.* var. *exigua* and *P. eupyrena* can be isolated easily from arable soils, *P. e.* var. *foveata* does not appear to be a persistent inhabitant and was much more restricted to the potato plant, according to Todd & Adam (1967). On the other hand, while it may not be well adapted to persist in soil in the absence of a host plant, Fox *et al.* (1971) have recorded its presence in inoculated seedlings of other crop plants and in self-sown weed seedlings of a number of species growing in contaminated soil.

Tubers of all commercial potato varieties can develop gangrene under suitable conditions but, as with dry rot, there are considerable varietal differences. Gangrene was observed more frequently in early than in late varieties (Alcock & Foister, 1936; Kranz, 1959a; Logan, 1967b) but Malcolmson (1958), in uniform tests of 39 varieties, noted that susceptibility was not correlated with maturity types. According to Langton (1970), differences in varietal reaction may be detected more accurately by incubation at 10°C than at 5°C.

Varietal differences extend even to reaction to *P. e.* var. *foveata* and *P. e.* var. *exigua*, e.g. Arran Pilot and Golden Wonder are more susceptible to the former and Kerr's Pink, Doon Star and Catriona to the latter (Malcolmson & Gray, 1968c); nor do haulm and tuber reactions necessarily correspond (Fox & Dashwood, 1969).

There are several aspects of the control of the disease, particularly where chemical control is concerned, and much depends on pre-harvest infection. Immersion in an organo-mercury disinfectant solution, usually methoxy ethyl mercuric chloride, either with or without prior washing of the tubers, can provide a very satisfactory reduction of gangrene if it is carried out immediately after lifting (Boyd, 1960; Harrison & Downie, 1960; Logan, 1967c; Boyd & Penna, 1967). In north-east Scotland, however, this method of control has not been so uniformly successful (Gray & Malcolmson, 1967) possibly because of pre-harvest establishment of infection.

The use of dusts, e.g. tecnazene (Harrison & Downie, 1960; Gray & Malcolmson, 1967) has not been effective in all cases but application of the systemic fungicides thiabendazole and benomyl both as dips and as dust formulations immediately after lifting has given promising results (Hide *et al.*, 1969). A most effective control has been achieved by fumigating seed tubers within a short time of lifting with *sec*-butylamine vapour (Graham & Hamilton, 1970).

Since there is considerable evidence that the disease can be controlled chemically by treatment at or shortly after lifting, it would seem that in most cases only a small degree of pre-harvest infection has been established at that time. Thus, if the haulm which is the source of much of the inoculum is removed it reduces the chances of contamination of the tuber surface, and uniform damage tests (Logan, 1970) demonstrated control of tuber infection by pulling and removing the haulm before lifting. On the other hand, Malcolmson & Gray (1968a) suggested that the reduction of natural infection which followed rapid haulm destruction was probably a reflection of the maturity of the tubers.

Kranz (1958) provides evidence that temperatures close to 0°C and high spore concentration favour tuber infection. From this it follows that liability to pre-lifting infection will be increased by exposure of the tubers to low temperatures in the field. Not only so, but the damage caused in the lifting process may be more severe and therefore more open to infection. This may well explain the occurrence of very extensive gangrene in stocks which have been lifted after a period of ground frost although this may not have been severe enough to damage the potatoes. Furthermore it lends weight to the advice that the disease can be minimised by early lifting in warmer soil conditions.

Another means of reducing gangrene incidence is the encouragement of rapid wound periderm formation after lifting by means of temperature control. In practice, this emphasises the importance of the 'curing period' of storage immediately after harvest to heal lifting wounds (Malcolmson & Gray, 1968b). There is no doubt also that after grading or indeed after any operation where damage might be sustained gangrene can be reduced if the tubers are placed in warm storage conditions. However, although storage for a period of 10 days at 21°C immediately after infection can prevent progressive rotting when the tubers are subsequently kept at 10°C (Malcolmson, 1958), with tubers in which infection is already established in storage at 5°C, subsequent storage at 20°C can allow the rot to develop (Anon., 1967). Gray & Paterson (1971) showed that storage at 15–17°C for 5–10 days after grading or after delivery of seed potato consignments reduced gangrene incidence but also pointed out the practical complications which beset the application of this procedure. Moreover, the effectiveness of this practice may depend on the depth and nature of the wound (Griffith, 1969) and also possibly on the additional presence of the dry rot fungus, which can develop rapidly at higher temperatures particularly later in the storage period. Thus too much reliance cannot be placed on this method as a means of effective control. Prevention of exposure of tubers to cold draughts can be effective in gangrene reduction (Malcolmson & Gray, 1968b) and in practice the incidence of the disease is frequently greater nearer the inlets of the ventilation ducts in potato stores.

Gangrene is relatively more severe in seed principally because of the grading damage and the length of the storage period thereafter but it rarely leads to secondary soft rotting. It occasionally causes blanks in the field after planting (Boyd & Logan, 1967), often because the lesions are overtaken by dry rot, but unless the lesions are very large or the soil conditions unsuitable, planting of infected tubers usually does not significantly reduce yield (Malcolmson & Gray, 1968a). However, it is possible that such plants are more likely to show blackleg symptoms.

In view of the tuber-borne nature of the disease, it is hoped that from stocks of seed tubers which have been raised initially from stem cuttings in UK (Hirst & Hide, 1967), associated with additional chemical treatment, the level of gangrene will be reduced to and maintained at negligible levels in all certified seed potato stocks (Graham & Hardie, 1971).

SKIN SPOT—*OOSPORA PUSTULANS* OWEN & WAKEF.

Symptoms of what was most likely skin spot were first distinguished by Carruthers in England in 1904, although the disease was probably known for many years before this. The first full account with a detailed description of the causal organism was given by Owen (1919). The fungus has a relatively low optimum for growth of 8–13°C (Kharkova, 1961b) and, as might be expected, the disease appears to be confined to the temperate or cooler regions of the world. It is present in most Northern European countries, Canada and USA but it is from UK (Anon., 1953), Norway (Førsund, 1966) and certain parts of USSR (Znamenskaya, 1960) that serious effects have been reported.

Tuber symptoms become obvious only after several months' storage and accurate assessment can be made only after tubers have been washed. They consist of small discrete black or purplish pimples up to 2mm in diameter, occurring either singly or aggregated in groups covering a considerable area of the surface. A high proportion of affected tubers carry a small area of infection at or near the stolon attachment (Boyd, 1957). Eyes are often affected and pustules or small areas of necrosis may be formed at these points.

In the Murmansk region of USSR other symptom types have also been recognised (Kharkova, 1961a), such as black superficial stains and depressed patches or spots of a 'pit rot' type. The former can be seen sometimes in artificial inoculations and the latter have been noted by Ives (1955) in tubers treated with the sprout depressant IPPC which inhibited wound periderm formation, and by Boyd & Lennard (1962) in tubers overwintered in the soil. Together with *Helminthosporium solani*, *O. pustulans* is the cause of another symptom known as 'black skin' (Gomolyako, 1959).

Penetration of the tissues is normally limited to about 12 layers of cells and occurs through lenticels and buds (Allen, 1957) or by superficial wounds (Fuchs, 1954; Boyd & Lennard, 1961; Kharkova, 1961a). About 2 months after infection the cork layer at the outside of the infected tissue breaks down and the 'spot' begins to become visible (Allen, 1957).

Extensive infection makes tubers unattractive and unfit for the 'washed ware' trade. However, the more important aspect is as a seed tuber disease in causing the death of the bud tissues of the eyes, although sometimes eye infection may be accompanied by very few, if any, surface symptoms and the presence of the fungus may be detected only by incubation of eye cores, as in the method described by Hide *et al.* (1968).

Infection of the stem bases, roots and stolons causes light brown superficial spots or patches fairly early in the growth of the plant (Hirst & Salt, 1959) and later these become darker brown (but never as dark as lesions of *Rhizoctonia*) and tend to crack. Conidia developed on such infected areas can provide an abundant source of inoculum for infection of young tubers and this may occur not long after tuber initiation and may become well established under field conditions in the buds and bud scales as early as July (Hirst *et al.*, 1963).

During growth, there is evidence that infection occurs first of all in the eyes (Hide *et al.*, 1968) and this is supported by Lennard (1967) in periodic lifting observations and by Edie & Boyd (1966) who found that, after harvest, infection of the eyes was established beyond the influence of mercury disinfection before that of the skin.

During storage, penetration of surface and eye continues but the degree to which this occurs depends largely on the environment. Under cold (4°C), damp (almost 100% R.H.) conditions this may be extensive. It is minimised at 14·5°C and 75%

R.H. but these conditions must be maintained for upwards of 3 months for control to be really effective (Lennard, 1967). In practical terms, this allows of some control by storage in the dry atmosphere of sprouting trays immediately after lifting instead of in bulk in a clamp or potato store. At the same time it means that, although the warmer conditions of the 'curing period' of 10–14 days may in some cases be beneficial, this is not sufficient for an effective reduction of skin spot, nor is it likely that the relative humidity in bulk storage would be of an order low enough to restrict infection satisfactorily.

During storage, the number of dead, infected buds increases, but at the same time the total number of infected buds also rises (Edie, 1966; Hide, Hirst & Mundy, 1969). This almost certainly indicates a spread of the organism during storage, an observation which was first recorded by Kharkova (1961a), for conidia of *O. pustulans* have been detected in the air in potato stores (Kharkova, 1961a) and in the air among stored tubers (Hide, Hirst & Mundy, 1969). In damp storage conditions, the fungus can often be seen sporulating on the surface of tubers and on young sprouts. Such spread in storage is not generally regarded as of great importance with normal commercial stocks but low temperatures during late storage are suspected of favouring severe bud damage.

After the planting of infected seed tubers, the fungus colonises the sprouts and parts of the stem bases nearest to the tubers, then the stolons and young tubers. This pattern develops more rapidly with increasing degrees of tuber infection and in heavier soils (Morton *et al.*, 1970).

Transmission of the disease from contaminated soil and carry-over on plant debris until the following year appears to be of importance in USSR. *O. pustulans* forms microsclerotia (Kharkova, 1961a) and is thus adapted for at least limited survival in the absence of a crop. In UK, the extent to which the fungus persists in soil through a rotation is not clear although microsclerotia have been found (Hirst *et al.*, 1965; Lennard & Boyd, 1965), and these were capable of producing conidia after burial in soil for 7 years (Hirst *et al.*, 1971). However, it is generally accepted that transmission from the seed tubers is largely, if not entirely, responsible for tuber infection (Boyd & Lennard, 1961; Hirst *et al.*, 1963) and the degree of infection on the parent seed is reflected in that of the progeny (Boyd *et al.*, 1968).

The fungus has been isolated directly from soil (Bannon, 1966) but was not detected beyond 60 cm from infected plants. It can also be detected on roots of tomato seedling test plants (Salt, 1957) or on roots of potato stem cuttings. Znamenskaya (1960), in the Murmansk region of USSR, noted that infection was highest in light sandy soils, less in humus iron podzols and least in peat bog soils. In UK, soil plays a part in influencing conditions for infection, both of stem bases (Salt, 1964) and tubers; sandy loam allows considerably less transmission of infection to the progeny than clay loam (McGee *et al.*, 1972) from the same initial seed infection source.

There is considerable fluctuation in the seasonal incidence of the disease. It seems reasonable to assume that two of the most important environmental factors concerned in tuber infection are soil moisture and temperature. Boyd & Lennard (1962) observed that, over a period of 34 years in UK, incidence of skin spot above an arbitrary normal in Scottish seed stocks in spring usually followed a 41-day lifting period when the rainfall was above average (4·10 in : 104 mm) and a subsequent early period of storage (October–December) when the temperature was below average.

Considerable differences in susceptibility of varieties have been observed in those areas where skin spot is prevalent. Boyd (1957), Boyd & Lennard (1961) and Nagdy & Boyd (1965) tested varietal reaction to natural infection and to standard inoculations and assessed susceptibility to surface and eye infection as separate indices. With variable conditions of field infection, surface and eye infection indices are not necessarily related but in standard tests there is a significant correlation between them. Manturova (1960) noted that some USSR tuber selections with thick rough periderms were practically non-susceptible, and in UK skin resistance to skin spot was correlated with periderm thickness. Thus, of 30 varieties, Golden Wonder and Dunbar Rover were most resistant and Arran Banner and King Edward were most susceptible (Nagdy & Boyd, 1965).

The order of susceptibility of tubers, however, is not comparable with that of the stem bases as given by Salt (1964). Furthermore, the surface and eye of the tubers may not by themselves indicate the field performance after planting. The two varieties King Edward and Kerr's Pink are good examples of this. Both are equally very susceptible in surface and eye but with Kerr's Pink seldom does even a high degree of seed infection result in blanking, whereas it is usually extensive with

King Edward (Boyd *et al.*, 1968; Bjor, 1970). This difference appears to be related to a varietal difference in the vigour of the sprouts and, although the nature of this is as yet unknown, it is a factor to be considered when assessing the field performance of a variety.

Skin spot is the most important cause of dead buds on potato tubers. The fungus is widespread on commercial stocks in UK and a survey of 200 stocks of the variety King Edward (Hirst & Hide, 1964) showed it to be present in every stock on average on about 75% of the tubers and about 50% of the eyes.

The most obvious effect of planting infected seed tubers is failure of emergence. In field surveys there is sometimes a tendency to overestimate the extent of blanking due to skin spot because of other factors. In surveys of King Edward seed stocks grown in the East of Scotland the highest figure recorded was 26·7% directly attributed to skin spot (Lennard, 1966), and over the 9-year period 1961–69 the average degree of blanking was only 4·4% (Boyd *et al.*, 1970), which by itself would hardly affect total yield. Znamenskaya (1960) noted that in some years in the Murmansk region of USSR 60–80% of the seed tubers had to be rejected because of skin spot.

As well as serving to transmit the disease, tuber infection causes a delay in emergence of the plants which are produced and, because of blanking, can alter the ratio of ware and seed in the progeny. Again, because of reduced numbers of viable eyes, fewer stems and thus fewer tubers may be produced (Boyd & Lennard, 1961).

Control of skin spot has been achieved by disinfection, at the time of lifting of tubers, in an organo-mercury solution (Greeves & Muskett, 1939; Foister, 1943; Boyd, 1957, 1960) either with or without prior washing. In later work the material used was methoxy ethyl mercuric chloride. The potatoes have to be dried immediately after disinfection and this is at once a drawback, because of additional labour, and an advantage, since the process of boxing in dry conditions by itself confers a benefit in reduction of skin spot. Edie & Boyd (1966), however, pointed out that the efficiency of the method did not depend on the lifting process as such but on the date at which this was carried out. They showed that there was a progressive decrease in effectiveness of disinfection corresponding to delay in lifting treatment, starting several weeks before normal lifting in early October. Along with this, penetration of the eye tissues occurred more rapidly than that of the skin. Greeves & Muskett (1939), on the other hand, obtained no additional control by lifting in August but Lennard (1967) confirmed the benefit of earlier lifting and found that this depended on temperature conditions since, if August-lifted tubers were stored at 4°C immediately after lifting, they could be just as heavily infected as those lifted later. Hence in practice natural infection tends to be established more extensively in colder conditions normally associated with later lifted stocks; for example, in the seed-growing areas in Scotland, the average weekly mean air temperature falls from 10°C to 7·2°C during October and to 4·4°C by the end of November.

Exposure of tubers to heat after harvest, e.g. 43°C for 2 hours (Førsund, 1966) can greatly reduce infection. The systemic fungicides thiabendazole and benomyl, applied to tubers after harvest, the former as a dip (Hide, Hirst & Griffith, 1969) and the latter as a dust (Morton *et al.*, 1969), can also provide an effective control, and when benomyl dust was applied to seed some time before planting it was found to reduce skin spot in the progeny tubers (Hide, Hirst & Griffith, 1969). The latter method would have little effect on the seed to which it was applied but it would be suitable for treatment on individual farms and, if carried out annually, might restrict any build-up of infection on seed stocks.

The application of *sec*-butylamine as a fumigant soon after lifting to potatoes in bulk (Graham & Hamilton, 1970) is the most recent and, from the limited number of tests carried out so far, appears to be the most effective chemical method of control of skin spot and, as mentioned previously, of gangrene also.

Finally, the development of stocks derived from skin spot-free stem cuttings (Hirst & Hide, 1967) represents a considerable advance in the control of skin spot as well as a number of other tuber-borne diseases, provided great care is taken to avoid re-contamination in commercial production.

SILVER SCURF—*HELMINTHOSPORIUM SOLANI* DUR. & MONT.

Silver scurf occurs in most countries where potatoes are grown. The symptoms, which are confined to the tubers, take the form of grey silvered patches of skin and are most obvious towards the end of the storage period.

Infection occurs during the growth of the tubers but obvious symptoms are sometimes difficult to detect at harvest. Those which do occur appear mainly at the stolon end of the tuber as small pale brown spots. Burke (1938) considered that susceptibility increased with maturity, but severe disease development has been recorded in late July or August (Mooi, 1968) and, even where there are no obvious symptoms, the fungus can be detected in early tuber growth by the plug tissue method (Hide, Hirst & Mundy, 1969).

Minimum conditions for lesion inception are 2·8°C and 90% R.H. and the optimum temperature for growth is 24°C (Burke, 1938), while extension of the affected areas and continuing infection can occur gradually during storage in conditions of high temperature and high R.H. This was demonstrated by Hide & Stedman (1968) who obtained infection on initially 'healthy' tubers by exposing them in a bulk of infected potatoes. Lennard (1969b) showed that development of the disease was directly influenced by different periods of storage at 13–16°C before lowering the storage temperature to 0–6°C, but that the normal curing period recommended in practice, i.e. 10–14 days of 15°C, and high R.H., would have little effect on symptom expression if the subsequent temperature was low.

According to Mooi (1968), less disease develops on potatoes lifted in July than on those lifted in September, but the increase is greater if the tubers are kept in a clamp after lifting than if they are allowed to remain in the soil. On the other hand, early digging is advocated by Burke (1938), but this would have to be followed by suitable low temperature storage.

The fungus enters through lenticels or directly through cells of the periderm (Burke, 1938) and the mycelium becomes both inter- and intracellular. It disorganises the epidermal cells (Schultz, 1916) and is confined entirely to the phellem layers, while apparently utilising most of the starch in several layers of the outer cortical cells (Wenzl, 1969). In favourable storage conditions masses of olivaceous black conidia are formed on the surface of the tubers giving a sooty ring at the edge of the advancing lesion. Although Kramer (1942) has recorded secondary invasion, infection by *H. solani* is not generally regarded as leading to rotting of the storage tissues. Because of the disruption of the periderm, loosening and sloughing of some of the outer cork layers occurs, tubers shrivel prematurely and the skin becomes completely grey. In coloured varieties this feature is lost also (Schultz, 1916), thereby detracting from the appearance of the sample. There are some varietial differences, the American variety White Gold being outstandingly susceptible (Burke, 1938).

H. solani has never been recorded on any other plant and attempts to infect stems, stolons and roots have been unsuccessful (Schultz, 1916; Burke, 1938). Unlike *O. pustulans*, it has never been shown to attack bud tissues of the tuber eyes nor to cause blindness.

Planting infected seed is the main avenue of disease transmission (Burke, 1938; Santerre, 1967; Mooi, 1968) and international spread by this means has been claimed (Cristinzio, 1954). However, both Mooi (1968) and Lennard (1970) found that infection developed more on the progeny of slightly infected than of severely diseased seed. Young silver scurf lesions sporulate more profusely than old ones and the degree of infection on the crop depends on the level of active spore production on the seed rather than on the severity of symptoms. If, in fact, ground keepers do not form efficient sources of transmission, this may be the reason.

Taubenhaus (1916) considered that the soil was the major source of inoculum but the experience of later workers (Burke, 1938; Mooi, 1968) suggests that, apart from carry-over by volunteer tubers, with a crop rotation of 3 years or more, if soil contamination does exist it can be only very slight.

The soil environment, however, has some influence upon infection. There is more silver scurf when potatoes are grown in flat cultivation than in ridges (Mooi, 1968) or in sand rather than loam or clay soils (Lennard, 1970) or in peat bog soils than iron podzols (Hrobruih, 1953). Such evidence may indicate the importance of a critical soil moisture regime and Adams *et al.* (1970) noted a significant negative correlation between the soil moisture at a depth of 6 in (15 cm) and the incidence of silver scurf. Little is known about the influence of fertilisers on silver scurf and while in one case it was increased by excessive nitrate fertiliser (Cristinzio, 1954). in another it was reduced by nitrate (Adams *et al.*, 1970).

The skin blemishes and loss of 'bloom' caused by silver scurf have assumed more importance in recent years because of the increase in the pre-packed, washed ware trade in a number of countries (Busch, 1958; Harrison, 1962; Mooi, 1968) particularly towards the end of the storage season and with coloured varieties. The other important feature of the disease is the weight loss sustained by heavily infected tubers

in storage. Even a moderate degree of infection of tubers stored dry at as low as 4°C can give a measurable increase in fresh weight loss (Lennard, 1969a).

The severity of the disease can be reduced by leaving potatoes to dry in the field after lifting (Harrison, 1962; Hrobruih, 1953) or by storing them in a dry atmosphere (Mooi, 1968) such as in boxes.

There have been many attempts at chemical control using mercury compounds or tecnazene for soil application, or formalin (Johnson, 1903), organo-mercury or other compounds as seed tuber disinfectants (Busch, 1958). None of the results has been generally consistent and although the extent of infection can be considerably reduced by using an organo-mercury compound immediately after lifting, the subcutaneous mycelium may not be killed (Santerre, 1967) and may commence to sporulate several months later (Mooi, 1968). The reduction in silver scurf given by such a seed tuber treatment did not extend to the produce (Hide, Hirst & Mundy, 1969). Preliminary experiments using trametan dust or thiabendazole dip after harvest or benomyl dust before planting have shown a reduction in silver scurf infection of the progeny, benomyl being particularly effective (Hide, Hirst & Griffith, 1969).

Control of silver scurf by field drying of tubers at lifting time is limited in application by uncertain weather conditions. Reduction of the disease in a ware crop can be attempted by manipulation of the storage environment using a warm and dry ventilation current after lifting (Mooi, 1968) or storing under cold conditions (Schultz, 1916). Both of these methods, however, contain inherent difficulties because of possible interference with wound healing and the incidence of other diseases. The use of benomyl appears to offer a most promising means of control, especially if allied to the development of stocks derived from disease-free stem cuttings.

BLACK SPOT

Symptoms of black spot develop in potato tubers as a result of bruising under certain conditions. In some cases symptoms may be confused with those of a tissue rot, although no pathogenic organism is involved. These consist of grey or black spots or patches of variable size just under the skin, usually near the stolon end, and can be detected on peeling the tubers. These spots coincide with the site of the impact and occasionally there is a slight diffuse discoloration on the surface, although the periderm remains unbroken.

When the tuber is cut at this point the discoloration can be seen near the vascular ring, for in this area there is a marked plane of weakness, as evidenced by sloughing of the cortical tissues when tubers are cooked. It is probably here that the actual breakdown of the cells occurs by shock waves from the surface (Burton, 1969). It may be that this is the reason why black spot does not occur after a wound or 'shatter' bruise where the impact may have been taken up by the superficial tissues. Sometimes damage, particularly harvest bruising, of immature tubers can result in small 'starch pits' in the cortex, without the development of black spot.

Soon after bruising, which may not necessarily be severe, a pink or reddish coloration develops before the tissues become grey or black and, in severely affected potatoes, the flesh may be almost completely discoloured, such tubers often shrivelling to such an extent as to become quite hard and mummified.

The condition was first described by Horne (1913) in England but since then it has been recognised as an important cause of loss in other countries including the Netherlands and later USA. It has been recognised under many other names such as 'bruise', 'blue spot' and 'lead spot' and in fact the name 'black spot' sometimes does not adequately describe the observed symptoms. In recent years, growers have become more aware of its importance because of the increasing use of potatoes for crisps and chip manufacture. It has been acknowledged as one of the most important, if not the most important, physiological disorder in potato production in USA (Sawyer, 1959).

The discoloration is caused by the formation of the insoluble black pigment melanin which is formed by the action of the enzyme tyrosinase upon the amino acid tyrosin liberated when the cells are killed (Merkenschlager, 1929). The extent of cell breakdown governs the intensity of the discoloured area, but even severely affected tissue contains only a small proportion of damaged and discoloured cells (Vertregt, 1968). Contrary to the reaction of tuber tissue to normal wounds or penetration by some pathogens, no wound periderm is formed around the affected areas (Horne, 1912; Boyd, 1951).

Several interdependent factors, apart from the initial bruise, are involved in the development of black spot, but two of them, potassium content and dry matter content, are of considerable practical importance. The association between black

spot and the absence of sufficient available potassium during plant growth has been noted in a number of countries (Oortwijn Botjes & Verheuven, 1927; Boyd & White, 1956; Ophius *et al.*, 1958; Jacob, 1959) and it was found that such tubers contained a higher than normal amount of tyrosin (Mulder, 1949). Tyrosin is fundamental to the formation of melanin but its content alone is not sufficient to explain differences in black spot incidence (Vertregt, 1968). Possibly potassium contributes also to the strength of the cell walls and their resistance to damage, for this would appear to be the critical factor. In this connection, sloughing of the cortex has been shown to be directly proportional to potassium fertiliser application (Schippers, 1968). In practice, where potassium is deficient especially in presence of high nitrogen application, susceptibility to black spot is increased, but the trouble can occur even where potassium supply is apparently adequate.

The other important factor is dry matter content of the tubers and any procedure which influences this, either directly or indirectly by reducing turgidity, may alter the predisposition of the potatoes to black spot. The effect of dry matter has been shown by de Bruyn (1929) and by many other later workers. It may be illustrated even within an individual tuber, where both dry matter content and susceptibility increase from the apical to the stolon end. The disorder is also more prevalent in dry growing seasons when average dry matter content is usually higher than normal.

Loss of turgidity occurs gradually during storage either because of the length of this period, because of warm conditions or particularly because of sprouting (de Bruyn, 1929; Boyd, 1951; Ophius *et al.*, 1958; Sawyer & Collin, 1960). Consequently the disorder is more prevalent in spring after potatoes have been removed from storage and possibly desprouted and then bruised on a grading machine. Turgidity may also be reduced by severe infection by silver scurf. However, Van der Zaag & Meijers (1969) point out that tubers with high turgidity can also be susceptible and that there is an optimum at which they are least liable to black spot.

Varieties with high dry matter content are usually more susceptible to black spot but the grower often finds himself in a dilemma because it is just such varieties, e.g. in UK the variety Record, which are required for processing, and a relatively small proportion of affected tubers can lead to the rejection of the whole crop for this purpose. Sawyer & Collin (1960) suggest that varietal susceptibility to black spot can also be related to the structure of the phellem and the lenticels, since these tissues are involved in the prevention of shrinkage. Varieties which sprout strongly and early and thus lose turgidity can be very susceptible at the end of the storage season and this can be minimised by the use of sprout depressants.

Black spot is very difficult to avoid in varieties with a high dry matter content when prevailing conditions favour the development of high susceptibility. It may be possible, according to Van der Zaag & Meijers (1969), to predict the predisposition of a crop on the basis of analysis of the total potassium in the haulm. Such knowledge would be useful, although it is doubtful if much can effectively be done in practice except to reduce bruising to a minimum during lifting and riddling, but this should be attempted in any case. Howard *et al.* (1962) have suggested that aeration of the ridges before harvest is helpful because a high carbon dioxide content increased susceptibility, but this has not been confirmed. Dry soil conditions prior to harvest decrease turgidity of the tubers but this can be rectified by the use of irrigation (Kunkel & Gardner, 1965) with due regard to other effects of this procedure.

It is essential to maintain a high content of potassium in the soil, and heat conditioning of the crop immediately before, but not after, grading or other handling procedure, when the temperature both of the environment and the potatoes should be raised to about 20°C (Wiant *et al.*, 1951; Ophius *et al.*, 1958; Jacob, 1960), can reduce damage appreciably.

Sometimes black spot can be detected a few days after harvest has commenced in crops destined for processing and if it is possible to process each day's lifting within about 24 hours the crop can be salvaged in this way. Probably because of their greater weight and greater liability to bruise, ware potatoes are normally more affected than seed and are unacceptable when even slightly discoloured, but seed from such a crop, if satisfactory in other respects, is usually fit for planting.

CONCLUSIONS

If one views these 9 diseases in perspective, it is clear that 5 of them, soft rot, watery wound rot, dry rot, gangrene and black spot are governed by, or at least are associated to some degree with, one common factor—damage. Current husbandry methods involve pressures dictated by economics and climatic conditions. Not only is there increasing dependence upon machinery for handling perishable living

plant material but this has of necessity to be operated often at speeds which make it impossible to avoid injury. In this respect it is often injury of the less obstrusive kind which is the more insidious. Reduction of damage remains a most important sphere for continued research.

The development of seed stocks from stem cuttings can be instrumental in the reduction of blackleg, skin spot, silver scurf and possibly gangrene, if proper hygienic precautions can be maintained. To achieve this, and since it is unlikely that all damage will be eliminated in the forseeable future, methods of fungicidal control by the use of fumigants, dusts or liquids appear still to be necessary.

Of the last two diseases, pink rot presents a rather intractable problem but fortunately is only of local importance. Blight, on the other hand, although unlikely to be eliminated, can be minimised by the development of varieties with higher indices of polygenic resistance particularly of the tubers. The disease can be further reduced by more attention to the timing and efficiency of protective spray application and of haulm destruction and to the early destruction of overwintering sources of the fungus.

REFERENCES

(Figures in square brackets refer to abstracts in the Review of Applied Mycology/Review of Plant Pathology)

Adams, A. P., Sandar, N. & Nelson, D. C. 1970. *Am. Potato J.* **47**:49–57.
Alcock, N. L. & Foister, C. E. 1936. *Scott. J. Agric.* **19**:252–257.
Allen, J. D. 1957. *Ann. appl. Biol.* **45**:293–298.
Anon. 1948. *Nature, Lond.* **162**:4126.
Anon. 1950. *Rep. Colorado agric. Exp. Stn 1949–50.* [**31**:104]
Anon. 1953. *Pl. Path.* **2**:139–140.
Anon. 1967. *Rep. Res. & tech. Work Minist. Agric. N. Ireland 1967*:85.
Anon. 1970. *Short Term Leafl. Minist Agric. Fish Fd* 118
Ayers, G. W. 1961. *Can. Pl. Dis. Surv.* **41**:170–171.
Ayers, G. W. & Ramsay, G. C. 1961. *Can. Pl. Dis. Surv.* **41**:199–202.
Ayers, G. W. & Robinson, D. B. 1956. *Am. Potato J.* **33**:1–5.
Bainbridge, A. 1970. *Trans. Br. mycol. Soc.* **55**:485–488.
Bannon, E. 1966. *Scient. Proc. R. Dubl. Soc.* Ser. B **1**:221–228.
Bate-Smith, E. C. 1956. *Rep. Fd Invest Bd, Lond. 1955*:53–62. [**35**:873]
Belozor, N. I. 1960. *Sborn. Trud. Aspir. molod. nauch. Sotrud. vses. Inst. Rastenievod. 1960*:305–310. [**41**:167]
Bétencourt, A. & Prunier, J. P. 1965. *Eur. Potato J.* **8**:230–242.
Bjor, T. 1970. *Proc. 4th trienn. Conf. Eur. Ass. Potato Res. 1969*:191–192.
Blair, I. D. 1959. *Tech. Publ. Canterbury agric. Coll.* 18, 23 pp. [**38**:766]
Blodgett, E. C. 1945. *Pl. Dis. Reptr* **29**:124–126. [**24**:285]
Blodgett, E. C. & Ray, W. W. 1945. *Am. Potato J.* **22**:250–253. [**25**:316]
Boerema, G. H. 1967. *Neth. J. Pl. Path.* **73**:190–192. [**47**, 1245]
Boerema, G. H. & Höweler, Louise H. 1967. *Persoonia* **5**:15–28. [**47**, 1442]
Bonde, R. 1950. *Bull. Me agric. Exp. Stn* 482, 31 pp.
Bonde, R. 1955. *Bull. Me agric. Exp. Stn* 583.
Bonde, R. & Hyland, Fay. 1960. *Am. Potato J.* **37**:279–288.
Bonde, R. & Schultz, E. S. 1949. *Bull. Me agric. Exp. Stn* 471, 16 pp.
Boyd, A. E. W. 1951. *J. hort. Sci.* **26**:148–156.
Boyd, A. E. W. 1952a. *Ann. appl. Biol.* **39**:322–329.
Boyd, A. E. W. 1952b. *Ann. appl. Biol.* **39**:330–338.
Boyd, A. E. W. 1952c. *Ann. appl. Biol.* **39**:339–350.
Boyd, A. E. W. 1952d. *Ann. appl. Biol.* **39**:351–357.
Boyd, A. E. W. 1957. *Ann. appl. Biol.* **45**:284–292.
Boyd, A. E. W. 1960a. *Pl. Path.* **9**:99–101.
Boyd, A. E. W. 1960b. *Eur. Potato J.* **3**:137–154.
Boyd, A. E. W. 1967. *Ann. appl. Biol.* **60**:231–240.
Boyd, A. E. W. 1970. *Proc. 4th trienn. Conf. Eur. Ass. Potato Res. 1969*:192–193.
Boyd, A. E. W. 1971. Abs. in *Potato Res.* **14**:337.
Boyd, A. E. W. & Henderson, J. Margaret. 1953. *Pl. Path.* **2**:113–117.
Boyd, A. E. W. & Lennard, J. H. 1961. *Eur. Potato J.* **4**:361–377.
Boyd, A. E. W. & Lennard, J. H. 1962. *Pl. Path.* **11**:161–166.
Boyd, A. E. W. & Logan, J. W. H. 1967. *Exp. Wk Edinb. Sch. Agric. 1966*:25–26.
Boyd, A. E. W., McGee, D. C. & Lennard, J. H. 1967. *Exp. Wk Edinb. Sch. Agric. 1966*:30–31.
Boyd, A. E. W., McGee, D. C. & Lennard, J. H. 1968. *Exp. Wk Edinb. Sch. Agric. 1967*:43–44.

Boyd, A. E. W., Morton, A. P. & Lennard, J. H. 1970. *Exp. Wk Edinb. Sch. Agric. 1969*: 21.
Boyd, A. E. W. & Penna, R. J. 1967. *Proc. 4th Br. Insectic. Fungic. Conf. 1967*: 294–300.
Boyd, A. E. W. & Tickle, J. H. 1972. In litt.
Boyd, A. E. W. & White, G. R. 1956. *Pl. Path.* **5**: 107–109.
Brien, R. M. 1940. *N.Z. Jl. Sci. Tech.* A **22**: 228–231. [**20**: 488]
De Bruyn, Helena L. G. 1922. *Meded. LandbHoogesch. Wageningen* **24**: 1–38. [**1**: 399]
De Bruyn, Helena L. G. 1929. *Tijdschr. PlZiekt.* **35**: 185–222.
Buddin, W. 1938. *Ann. appl. Biol.* **25**: 705–728.
Burke, O. D. 1938. *Bull. Cornell agric. Exp. Stn* 692, 30 pp.
Burton, W. G. 1963. *Eur. Potato J.* **6**: 77–92.
Burton, W. G. 1970. *Proc. 4th trienn. Conf. Eur. Ass. Potato Res. 1969*: 79–92.
Burton, W. G. & Wiggington, M. J. 1970. *Potato Res.* **13**: 180–186.
Busch, L. V. 1958. *Pl. Dis. Reptr* **42**: 441–443.
Cairns, H. & Muskett, A. E. 1933a. *Nature, Lond.* **131**: 277.
Cairns, H. & Muskett, A. E. 1933b. *Ann. appl. Biol.* **20**: 381–403.
Cairns, H. & Muskett, A. E. 1939. *Ann. appl. Biol.* **26**: 470–480.
Carruthers, W. 1904. *Jl R. agric. Soc. England* **65**: 261–263.
Cetas, R. C. & Leach, S. S. 1969. *Am. Potato J.* **46**: 174–181.
Conroy, R. J. 1958. *Agric. Gaz. N.S.W.* **69**: 299–302. [**38**: 30]
Cotton, A. D. 1921. *Misc. Publ. Minist. Agric. Fish.* **33**: 26–68. [**1**: 156]
Cotton, A. D. 1922. *Jl Minist. Agric.* **28**: 1126–1130.
Cox, A. E. & Large, E. C. 1960. *Agric. Handb. U.S. Dep. Agric.* 174, iv+230 pp.
Cristinzio, M. 1954. *Ric. fitop. Campan.* **12**: 35–47. [**35**: 540]
Cunningham, H. S. 1953. *Phytopathology* **43**: 95–98.
Cunningham, H. S. & Reinking, O. A. 1946. *Bull. N.Y. Sta. agric. Exp. Stn.* 721.
Dowson, W. J. & Rudd Jones, D. 1951. *Ann. appl. Biol.* **38**: 231–236.
Dutt, B. L. 1965. *Indian Potato J.* **7**: 81–84. [**45**, 2224]
Dye, D. W. 1969. *N.Z. Jl Sci.* **12**: 81–97. [**48**, 2737]
Edie, H. H. 1966. *Eur. Potato J.* **9**: 161–164.
Edie, H. H. & Boyd, A. E. W. 1966. *Eur. Potato J.* **9**: 216–225.
Fedotova, T. I. & Bliznets, N. I. 1969. *Mikol. i Fitopatol.* **3**: 53–56. [**48**, 1920]
Foister, C. E. 1940a. *Scott. J. Agric.* **23**: 63–67.
Foister, C. E. 1940b. *Trans. bot. Soc. Edinb.* **33**: 65–68.
Foister, C. E. 1943. *Ann. appl. Biol.* **30**: 186–187.
Foister, C. E. 1952. *Pl. Path.* **1**: 85–86.
Foister, C. E. & Wilson, A. R. 1943. *Agriculture* **50**: 300–303.
Foister, C. E. & Wilson, A. R. 1950. *Agriculture* **57**: 229–233.
Foister, C. E., Wilson, A. R. & Boyd, A. E. W. 1945a. *Nature, Lond.* **155**: 793.
Foister, C. E., Wilson, A. R. & Boyd, A. E. W. 1945b. *Nature, Lond.* **156**: 394.
Foister, C. E., Wilson, A. R. & Boyd, A. E. W. 1952. *Ann. appl. Biol.* **39**: 29–37.
Førsund, E. 1960. *Nord. Jordbr. Forskn.* Suppl. 1: 298–301. [**40**: 330]
Førsund, E. 1966. *Proc. 3rd trienn. Conf. Eur. Ass. Potato Res. 1966*: 221–222.
Fox, R. A. & Dashwood, Patricia. 1967. *Rep. Scott. hort. Res. Inst. 1966*: 31–33.
Fox, R. A. & Dashwood, Patricia. 1968. *Rep. Scott. hort. Res. Inst. 1967*: 31–32.
Fox, R. A. & Dashwood, Patricia. 1969. *Rep. Scott. hort. Res. Inst. 1968*: 32–33.
Fox, R. A., Dashwood, Patricia & Wilson, H. M. 1970. *Rep. Scott. hort. Res. Inst. 1969*: 30–31.
Fox, R. A., Dashwood, Patricia & Wilson, H. M. 1971. *Rep. Scott. hort. Res. Inst. 1970*: 33–34.
Fox. R. T. V., Manners, J. G. & Myers, A. 1971. *Potato Res.* **14**: 61–73.
Fuchs, W. H. 1954. *NachrBl. dt. PflSchutzdienst., Stuttg.* **6**: 75–76. [**34**: 59]
Gomolyako, L. G. 1959. *Biokhim. Plod. Ovoshch.* **5**: 159–164. [**39**: 189]
Graham, D. C. 1958. *Nature, Lond.* **181**: 61.
Graham, D. C. 1962. *Scott. Agric.* **41**: 211–215.
Graham, D. C. 1963. *Pl. Path.* **12**: 142–144.
Graham, D. C. 1972. *Proc. 3rd int. Conf. phytopath. Bact., Wageningen 1971*: 273–280.
Graham, D. C. & Hamilton, G. A. 1970. *Nature, Lond.* **227**: 297–298.
Graham, D. C. & Hardie, J. L. 1971. *Proc. 6th Br. Insectic. Fungic. Conf. 1971*: 219–224.
Graham, D. C. & Harper, P. C. 1966. *Eur. Potato J.* **9**: 141–145.
Graham, D. C. & Harper, P. C. 1967. *Scott. Agric.* **46**: 68–74.
Gray, Elizabeth G. 1965. Abs. in *Eur. Potato J.* **8**: 178–190.

Gray, Elizabeth G. & Malcolmson, Jean F. 1966. *Proc. 3rd trienn. Conf. Eur. Ass. Potato Res. 1966*: 217–218.
Gray, Elizabeth G. & Malcolmson, Jean F. 1967. *Proc. 4th Br. Insectic. Fungic. Conf. 1967*: 285–293.
Gray, Elizabeth G. & Paterson, M. Isabel. 1971. *Potato Res.* **14**: 251–262.
Greeves, T. M. & Muskett, A. E. 1939. *Ann. appl. Biol.* **26**: 481–496.
Griffin, G. J. 1964. *Phytopathology* **54**: 1275–1277.
Griffith, R. L. 1969. *Rep. Rothamsted Exp. Stn 1968*: 145–146.
Haeringen, G. H. van. 1938. *Tijdschr. PlZiekt.* **44**: 247–256. [**18**: 135]
Harrison, D. E. 1959. *J. Dep. Agric. Vict.* **57**: 381, 383–385, 387, 389. [**38**: 767]
Harrison, D. E. 1962. *J. Dep. Agric. Vict.* **60**: 530–531. [**42**: 276]
Harrison, D. E. & Downie, W. A. 1960. *J. Dep. Agric. Vict.* **58**: 372–373, 375–377, 378–381. [**40**: 485]
Harper, P. C., Boyd, A. E. W. & Graham, D. C. 1963. *Pl. Path.* **12**: 139–142.
Headford, D. W. R. & Low, A. J. 1970. *Proc. 4th trienn. Conf. Eur. Ass. Potato Res. 1969*: 185–186.
Herlihy, M. 1970. *Pl. Path.* **19**: 65–68.
Hide, G. A. & Stedman, O. J. 1968. *Rep. Rothamsted exp. Stn 1967*: 132–133.
Hide, G. A., Hirst, J. M. & Griffith, R. L. 1969. *Proc. 5th Br. Insectic. Fungic. Conf.*: 310–314.
Hide, G. A., Hirst, J. M. & Mundy, E. J. 1969. *Ann. appl. Biol.* **64**: 265–279.
Hide, G. A., Hirst, J. M. & Salt, G. A. 1968. *Ann. appl. Biol.* **62**: 309–318.
Hirst, J. M. & Hide, G. A. 1964. *Rep. Rothamsted exp. Stn 1963*: 113.
Hirst, J. M. & Hide, G. A. 1967. *Rep. Rothamsted exp. Stn 1966*: 129.
Hirst, J. M., Hide, G. A., Griffith, R. L. & Stedman, O. J. 1971. *Jl R. agric. Soc. England* **1**: 31–70.
Hirst, J. M., Hide, G. A. & Stedman, O. J. 1965. *Rep. Rothamsted exp. Stn 1964*: 138.
Hirst, J. M. & Salt, G. A. 1959. *Trans. Br. mycol. Soc.* **42**: 59–66.
Hirst, J. M., Salt, G. A. & Hide, G. A. 1963. *Rep. Rothamsted exp. Stn 1962*: 120–121.
Hirst, J. M. & Stedman, O. J. 1962. *Rep. Rothamsted exp. Stn 1961*: 113–114.
Hirst, J. M., Stedman, O. J., Lacey, J. & Hide, G. A. 1965. *Ann. appl. Biol.* **55**: 373–395.
Holmes, T. D. & Storey, I. F. 1962. *Pl. Path.* **11**: 139–146.
Horne, A. S. 1912. *Jl R. hort. Soc.* **38**: 40–50.
Howard, F. D., Yamaguchi, M. & Knott, J. E. 1962. *16th Internat. hortic. Congress* **2**: 584–590.
Hrobruih, N. D. 1953. *Bot. Zh. SSSR* **38**: 206–214. [**33**: 110]
Ivashchenko, O. V. 1963. *Visn. sil's'kogospod. Nauki* 7: 62–65. [**44**, 811]
Ives, June V. 1955. *Pl. Path.* **4**: 19–21.
Jacob, W. C. 1959. *Mem. Cornell agric. Exp. Stn* 368, 86 pp.
Jarvis, R. H., Short, J. L. & Shotton, F. E. 1967. *Pl. Path.* **16**: 49–53.
Jones, W. 1935. *Sci. Agric.* **15**: 402–410. [**14**: 607]
Jones, W. 1945. *Sci. Agric.* **25**: 596–600. [**25**: 8]
Johnston, T. 1903. *Econ. Proc. R. Dubl. Soc.* **1**(1899–1909): 161–165.
Khan, A. A. & Logan, C. 1968. *Eur. Potato J.* **11**: 77–87.
Kharkova, A. P. 1961a. *Bot. Zh. SSSR* **46**: 399–407.
Kharkova, A. P. 1961b. *Bot. Zh. SSSR* **46**: 1508–1516.
Kramer, M. 1942. *Biologico* **8**: 83–86.
Kunkel, R. & Gardner, W. H. 1965. *Am. Potato J.* **42**: 109–124.
Kranz, J. 1958. *Phytopath. Z.* **33**: 153–196.
Kranz, J. 1959a. *Phytopath. Z.* **35**: 135–147.
Kranz, J. 1959b. *NachrBl. dt. PflSchutzdienst., Stuttg.* **11**: 69–71.
Kranz, J. 1959c. *Phytopath. Z.* **36**: 101–110.
Kranz, J. 1963. *Sydowia* **16**: 1–40. [**42**: 701]
Lacey, J. 1962. *Rep. Rothamsted exp. Stn 1961*: 124.
Lacey, J. 1963. *Trans. Br. mycol. Soc.* **46**: 302.
Lacey, J. 1965. *Ann. appl. Biol.* **56**: 363–380.
Lacey, J. 1966. *Eur. Potato J.* **9**: 86–98.
Lacey, J. 1967a. *Ann. appl. Biol.* **59**: 245–255.
Lacey, J. 1967b. *Ann. appl. Biol.* **59**: 257–264.
Langton, F. A. 1971. *Potato Res.* **14**: 29–38.
Lansade, M. 1949. *Bull. tech. Inform. Ingén. Serv. Agric.* **41**: 419–432.
Lapwood, D. H. 1964. *Rep. Rothamsted exp. Stn 1963*: 111–112.
Lapwood, D. H. 1965a. *Rep. Rothamsted exp. Stn 1964*: 136–137.
Lapwood, D. H. 1965b. Abs. in *Eur. Potato J.* **8**: 178–180.
Lapwood, D. H. 1967. *Eur. Potato J.* **10**: 127–132.

Last, F. T. 1961. *Rep. Rothamsted exp. Stn 1960*: 152.
Lazar, I. & Bucur, Elena. 1964. *Eur. Potato J.* **7**: 102–111.
Leach, S. S. 1971. *Pl. Dis. Reptr* **55**: 723–726.
Lennard, J. H. 1966. *Proc. 3rd trienn. Conf. Eur. Ass. Potato Res. 1966*: 222.
Lennard, J. H. 1967. *Proc. 4th Br. Insectic. Fungic. Conf. 1967*: 269–275.
Lennard, J. H. 1969a. *Exp. Wk Edinb. Sch. Agric. 1968*: 34.
Lennard, J. H. 1969b. *Exp. Wk Edinb. Sch. Agric. 1968*: 34–35.
Lennard, J. H. 1970. *Exp. Wk Edinb. Sch. Agric. 1969*: 24–25.
Lennard, J. H. & Boyd, A. E. W. 1965. *Exp. Wk Edinb. Sch. Agric. 1964*: 18–19.
Lint, M. M. de. 1961. *Proc. 1st trienn. Conf. Eur. Ass. Potato Res. 1960*: 282–283.
Logan, C. 1964. *Eur. Potato J.* **7**: 45–56.
Logan, C. 1967a. *Pl. Path.* **16**: 64–67.
Logan, C. 1967b. *Seed Potato* **7**: 78–80.
Logan, C. 1967c. *Rec. agric. Res. Minist. Agric. N. Ire.* **16**: 25–28.
Logan, C. 1968. *Rec. agric. Res. Minist. Agric. N. Ire* **17**: 115–121.
Logan, C. 1970. *Pl. Path.* **19**: 95–98.
Logan, C. & Khan, A. A. 1969. *Trans. Br. mycol. Soc.* **52**: 9–17.
Logan, C. & O'Neill, R. 1970. *Trans. Br. mycol. Soc.* **55**: 67–75.
Löhnis, Maria P. 1922. Thesis, Rijks Univ. Utrecht. [**1**: 253]
Löhnis, Maria P. 1925. *Meded. Wetensch. Comm. voor Advies en Onderzoek in het Belang van de Volkswelvaart en Weerbaarheid*, 129 pp. [**4**: 761–762]
Lund, B. M. & Nichols, J. C. 1970. *Potato Res.* **13**: 210–214.
MacAlpine, D. 1911. 215 pp., Dep. Agric. Victoria. (In Plant Pathology, E. J. Butler & S. G. Jones, Macmillan, London, 1949).
McCreary, C. W. R. 1967. *Proc. 4th Br. Insectic. Fungic. Conf. 1967*: 303–308.
McGee, D C., Morton, A. P. & Boyd, A. E. W. 1972. In litt.
McIntosh, A. H. 1965. *Proc. 3rd Br. Insectic. Fungic. Conf. 1965*: 177–181.
McIntosh, A. H. 1969. *Eur. Potato J.* **12**: 219–221.
McKee, R. K. 1950. *Nature, Lond.* **167**: 111.
McKee, R. K. 1952. *Ann. appl. Biol.* **39**: 38–43.
McKee, R. K. 1954. *Ann. appl. Biol.* **41**: 417–434.
McKee, R. K. 1955. *Ann. appl. Biol.* **43**: 147–148.
McKee, R. K. 1961. *Tagungsber. dtsch. Akad. Landwiss. Berlin* **27**: 277–289. [**42**: 45]
McKee, R. K. 1968. *Rep. Res. & tech. Work Minist. Agric. N. Ireland 1967*: 83–93.
McKee, R. K. & Boyd, A. E. W. 1952. *Ann appl. Biol.* **39**: 44–53.
Mackenzie, Mary. 1968. Abs. in *Eur. Potato J.* **11**: 193.
Malcolmson, Jean F. 1958. *Ann. appl. Biol.* **46**: 639–650.
Malcolmson, Jean F. 1959. *Trans. Br. mycol. Soc.* **42**: 261–269.
Malcolmson, Jean F. 1969. *Trans. Br. mycol. Soc.* **53**: 417–423.
Malcolmson, Jean F. & Gray, Elizabeth G. 1968a. *Ann. appl. Biol.* **62**: 77–87.
Malcolmson, Jean F. & Gray, Elizabeth G. 1968b. *Ann. appl. Biol.* **62**: 89–101.
Malcolmson, Jean F. & Gray, Elizabeth G. 1968c. *Trans. Br. mycol. Soc.* **51**: 618–620.
Manturova, I. M. 1960. *Zashch. Rast. Mosk.* **5**: 49–50. [**39**: 492]
Malenev, F. E. 1962. *Zap. Leningr. sel'skokhoz Inst.* **87**: 104–108. [**43**, 2394]
Martinović, Milica. 1961. *Zašt. Bilja 1961*: 21–26. [**42**: 401]
Merkenschlager, F. 1929. *NachrBl. dt. PflSchutzdienst* **9**: 20–21. [**8**: 598]
Metlitskiĭ, L. V. & Ozeretskovskaya, O. L. 1970. *Mikol. i Fitopatol.* **4**: 146–156. [**49**, 2743]
Mishchuk, E. M. 1964. *Sb. tr. Leningr. Inst. sov. torgovli* **23**: 157–164. [**45**, 1481]
Mooi, J. C. 1950. *Landbouwk. Tijdschr.* **62**: 712–724.
Mooi, J. C. 1968. *Meded. Inst. plziektenk. Onderz.* 482: 1–62.
Moore, Enid S. 1924. *Ann. Bot.* **38**: 137–161.
Moore, F. Joan. 1945. *Ann. appl. Biol.* **32**: 304–309.
Morton, A. P., Boyd, A. E. W. & Lennard, J. H. 1969. *Exp. Wk Edinb. Sch. Agric. 1968*: 22.
Morton, A. P., Boyd, A. E. W. & Lennard, J. H. 1970. *Exp. Wk Edinb. Sch. Agric. 1969*: 22.
Mulder, E. G. 1949. *Pl. Soil* **2**: 59–121.
Müller, K. O. & Börger, H. 1941. *Arb. biol. Abt. (Anst. Reichsanst.) Berl.* **23**: 189–231.
Müller, K. O., Meyer, G. & Klinkowski, M. 1939. *Naturwissenschaften* **27**: 765–768. [**19**: 496]
Murphy, P. A. 1921a. *Bull. Dom. Canada Dep. Agric. Exp. Farms* 44, 1–86. [**1**: 248]
Murphy, P. A. 1921b. *Sci. Proc. R. Dubl. Soc.* N.S. **16**: 353–368. [**1**: 251]

Murphy, P. A. 1922. *Sci. Proc. R. Dubl. Soc.* N.S. **16**: 442–466. [**1**: 251]
Murphy, P. A. & McKay, R. 1925. *Jl Dep. Lands Agric. Dubl.* **25**: 10–21.
Nadvodnyuk, Yu. N. 1960. *Nauch. Zap. belotserkov. sel.-khoz. Inst. 1960*: 205–212. [**41**: 613]
Nadvodnyuk, Yu. N. 1962. *J. Microbiol., Kiev* **24**: 38–43. [**42**: 143]
Nagdy, G. A. & Boyd, A. E. W. 1965. *Eur. Potato J.* **8**: 200–214.
Newton, W. & Lines, C. 1947. *Sci. Agric.* **27**: 72–73. [**26**: 313]
Nielson, L. W. 1968. *Am. Potato J.* **45**: 174–181.
Novakova, Jarmila. 1957. *Phytopath. Z.* **29**: 72–74. [**36**: 662]
Oortwijn Botjes, J. & Verhoeven, W. B. L. 1927. *Tijdschr. PlZiekt.* **33**: 56–96. [**6**: 632]
Ophuis, B. G., Hesen, J. C. & Kroesbergen, E. 1958. *Eur. Potato J.* **1**: 48–65.
Owen, M. N. 1919. *Kew Bull. 1919*: 289–301.
Pérombelon, M. 1968. *Rep. Scott. hort. Res. Inst. 1967*: 33–34.
Pérombelon, M. 1969. *Rep. Scott. hort. Res. Inst. 1968*: 34.
Pérombelon, M. & Lowe, R. 1971. *Rep. Scott. hort. Res. Inst. 1970*: 32–33.
Pethybridge, G. H. 1917. *J. Dep. Agric. tech. Instruct. Ire.* **17**: 601.
Pethybridge, G. H. & Bowers, E. H. 1908. *Econ. Proc. R. Dubl. Soc.* **1**: 547–558.
Pethybridge, G. H. & Lafferty, H. A. 1917. *Sci. Proc. R. Dubl. Soc.* **15**: 193–222.
Pethybridge, G. H. & Smith, A. 1930. *J. Minist. Agric.* **37**: 335–340.
Puşcaşu, A. & Popescu, Alexandra. 1970. *Studii Cerc. Biol.* Ser. Bot. **22**: 67–70. [**50**, 901]
Radtke, W. 1969. *Phytopath. Z.* **64**: 143–174. [**48**, 3131]
Ramsey, G. B. 1919. *Phytopathology* **9**: 285–288.
Ruehle, G. D. 1940. *Tech. Bull. Fla agric. Exp. Stn* 348, 36 pp. [**20**: 489]
Rudd Jones, D. & Dowson, W. J. 1950. *Ann. appl. Biol.* **37**: 563–569.
Salt, G. A. 1957. *Rep. Rothamsted exp. Stn 1956*: 112–113.
Salt, G. A. 1964. *Pl. Path.* **13**: 155–158.
Sampson, P. J. & Fountain, P. J. 1960. *Tasm. J. Agric.* **31**: 137–143. [**40**: 124]
Santerre, Jacquelin. 1967. *Can. J. Pl. Sci.* **47**: 695–702 [**47**, 1251]
Sawyer, R. L. 1959. *Am. Potato J.* **36**: 76–78.
Sawyer, R. L. & Collin, G .H. 1960. *Am. Potato J.* **37**: 115–126.
Schippers, P. A. 1962. *Eur. Potato J.* **5**: 132–146.
Schippers, P. A. 1968. *Eur. Potato J.* **11**: 88–99.
Schoene, Karin. 1967. *Phytopath. Z.* **60**: 201–236. [**47**, 1243]
Scholey, J., Marshall, C. & Whitbread, R. 1968. *Pl. Path.* **17**: 135–139.
Schultz, E. S. 1916. *J. agric. Res.* **6**: 339–350.
Skaptason, J. B., Peterson, L. C. & Blodgett, F. M. 1940. *Am. Potato J.* **17**: 88–92 [**19**: 490]
Small, T. 1944a. *Nature, Lond.* **153**: 436.
Small, T. 1944b. *Ann. appl. Biol.* **31**: 290–295.
Small, T. 1945. *Ann. appl. Biol.* **32**: 310–318.
Small, T. 1946. *Ann. appl. Biol.* **33**: 211–219.
Sokolova, V. E., Savel'eva, O. N. & Rubin, B. A. 1958. *C. R. Acad. Sci. U.R.S.S.* **123**: 335–338 [**39**: 340]
Taubenhaus, J. J. 1916. *Mem. N.Y. bot. Gdn* **6**: 549–560.
Todd, J. M. & Adam, J. W. 1967. *Proc. 4th Br. Conf. Insectic. Fungic.*: 276.
Tompkins, C. M., Ark, R. A., Tucker, C. M. & Middleton, J. D. 1939. *J. agric. Res.* **58**: 461–475. [**18**: 497]
Upstone, M. 1970a. *Pl. Path.* **19**: 148.
Upstone, M. 1970b. *Pl. Path.* **19**: 165–167.
Van der Zaag, D. E. & Meijers, C. P. 1970. *Proc. 4th trienn. Conf. Eur. Ass. Potato Res. 1969*: 92–103.
Vertregt, N. 1968. *Eur. Potato J.* **11**: 34–44.
Voronkevich, I. V. & Butsevich, L. A. 1964. *Dokl. vses. Akad. sel'.-khoz. Nauk 1964*: 30–33. [**45**, 1488]
Vujičič, Radmila & Park, D. 1964. *Trans. Br. mycol. Soc.* **47**: 455–458.
Wenzl, H. 1969. *Pflanzenarzt* **22**: 136–137. [**49**, 1123]
Whelan, J. & Loughnane, J. B. 1969. *Sci. Proc. R. Dubl. Soc.* Ser. B **2**: 171–178.
White, N. H. 1946. *J. Aust. Inst. agric. Sci.* **11**: 195–197. [**25**: 471]
Wiant, J. S., Findlen, H. & Kaufman, J. 1951. *Am. Potato J.* **28**: 753–765. [**31**: 513]
Wilson, A. R. & Boyd, A. E. W. 1945. *Agriculture* **51**: 507–512.
Wilson, J. B. & Johnston, E. F. 1967. *Am. Potato J.* **44**: 342.
Yurova, N. F. 1960. *Zashch. Rast., Mosk.* **5**: 29–31. [**39**: 614]
Zan, K. 1962. *Trans. Br. mycol. Soc.* **45**: 205–221.
Znamemskaya, M. K. 1960. *Zashch. Rast., Mosk.* **5**: 48–49. [**39**: 492]