STUDIES ON BLACK SCURF OF POTATO CAUSED BY *RHIZOCTONIA SOLANI* KUHN

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by

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SUMMARY

1. The Symptoms of black scurf disease of potato caused by Rhizoctonia solani Kuhn are described and literature relevant to the control of tuber-borne inoculum reviewed.

2. Pathogenic strains of R. solani were present on all 49 certified seed lines screened during 1971 and 1972.

3. Fifteen fungicides varied in their ability to prevent growth of R. solani in poison food tests. An organic mercury compound was the most effective, there being complete suppression of growth at 0.5 ug/ml a.i. Thiabendazole, benomyl, carboxin, chloroneb, thiram and two experimental compounds (BAS 3201F and BAS 3192F) were also highly effective with ED_{50} values of less than 5 ug/ml a.i.

4. The commercially recommended dipping time and concentration for use with organic mercury compounds inactivated sclerotia. However, the mercury dip treatment as practised by farmers was considerably less effective.

5. Very high concentrations of benomyl (5000 to 7500 ug/ml a.i.) when used as a tuber dip were required to inactivate sclerotia.

6. Increasing dipping time increased the effectiveness of an organic mercury compound but not of benomyl.

7. In glasshouse and field trials benomyl, thiabendazole and carboxin (all 10% a.i.) when applied as tuber dusts gave excellent control of shoot lesioning arising from tuber-borne inoculum. Dip treatment with an organic mercury compound also gave good control. Captan, metiram (both 10% a.i.) and mancozeb (8% a.i.) were less effective but still gave appreciable control.

8. Evidence was obtained indicating that benomyl, thiabendazole, carboxin, metiram, captan and mancozeb when applied as dusts to tubers had a fungistatic effect on sclerotia, whereas an organic mercury dip treatment was fungicidal.

9. Benomyl was systemic in potato plants, the extent of uptake being dependent on the planting medium and the method of application. Uptake was greatest in perlite following

drench application.

10. When benomyl and thiabendazole were applied as tuber dusts uptake was limited to the basal regions of shoots.

11. Benomyl, thiabendazole and carboxin applied as dusts to seed tubers provided some protection against inoculum of R. solani added to the planting medium.

PREFACE

The potato, Solanum tuberosum L. is second only to wheat in importance as a food crop in New Zealand. During the last five years the total area of potatoes grown has fluctuated between 8,000 and 10,000 hectares, and the yield has increased steadily to over 25 tonnes per hectare (Table I).

Table I Potato production in New Zealand. Area and production for several recent seasons*

Season	Area (ha)	Total Yield (tonnes)	Yield (tonnes/ha)
1966-67	8,020	187,267	23.35
1967-68	9,517	235,831	24.78
1968-69	10,132	256,542	25.32
1969-70	9,928	253,263	25.51**
1970-71	7,689 (est.)		

* New Zealand Official Year Book, 1972. (Yield figures converted to metric equivalents).

** = 10.15 tons/acre

Although potatoes are grown in all parts of New Zealand, the bulk of the market is supplied from three areas. The Pukekohe district supplies the early markets from September to December and also substantial quantities of mid-season and main-crop potatoes. The February to July market is supplied principally from the Manawatu-Rangitikei district, and winter supplies (May to November) are drawn mainly from Canterbury, Otago and Southland (Claridge, 1972; Baxter, 1972).

The bulk of seed potato requirements are provided by South Island growers, but increasing quantities are being produced in the North Island on properties situated at elevations greater than 305 metres (1,000 feet) above sea level.

Although many potato varieties are grown in this country, according to an estimate made for the 1967-68 season six varieties accounted for 89% of the total area (Claridge, 1972). Figures extracted from the 1971-72 list of growers of provisionally certified crops (Miller and Wilson, 1972) indicate much the same situation today (Table II).

Table II The relative importance of the main potato varieties grown in New Zealand.

Variety	Percentage of total area occupied by each variety 1967-68	Percentage of area grown for certified seed occupied by each variety 1971-72
Ilam Hardy	35	45
Aucklander Short Top	14	6
Sebago	12	7
Rua	11	18
Katahdin	10	6
Red King Edward	7	6
Total	89	88

Driver (1963) considers the following to be the most important diseases of the potato in New Zealand.

(1) Late blight (Phytophthora infestans (Mont.) de Bary).

(2) Early blight (Alternaria solani (Ell. and Mart.) Sorauer).

(3) Wilt (Verticillium albo-atrum Reinke and Berthold; Verticillium dahliae Kleb.)

(4) Blackleg (Erwinia aroideae (Townsd.) Holland; Erwinia atroseptica (van Hall) Jennison; Erwinia carotovora (Jones) Holland)

(5) Black scurf (Rhizoctonia solani Kuhn)*

* Although the existence of a perfect stage of this fungus has been known since 1903, the mycelial (Rhizoctonia) stage is predominant and for the purposes of this thesis the fungus causing black scurf of potatoes will be referred to as Rhizoctonia solani. The perfect stage is a basidiomycete, Thanatephorus cucumeris (Frank) Donk.

(6) Scab (Streptomyces scabies (Thaxt.) Waksman and Henrici)
(7) Powdery scab (Spongospora subterranea (Wallr.) Lagerh.)
(8) Pinkrot (Phytophthora erythroseptica Pethybr.)
(9) Virus diseases (leafroll, viruses X, Y, A, S and M).

Of the above diseases, only five are considered by growers to be of sufficient significance to warrant control measures. Control of late blight is based on extensive protective fungicide spray programmes, and the virus diseases are kept in check by planting certified seed and the use of systemic insecticides. By using certified seed, some degree of control of blackleg and wilt is obtained since the certification scheme also takes these diseases into account.

In the belief that shoot infection arising from tuber-borne sclerotia of R. solani significantly affects stand establishment many growers take steps to control this phase of the black scurf disease. Control in recent years has depended almost entirely on the dipping of seed tubers in solutions of organic mercury compounds to inactivate sclerotia. Because of the persistent nature and high mammalian toxicity of these mercury fungicides the Agricultural Chemicals Board in October 1971 advised that use of such chemicals will be banned after two years (J.D. August, pers. comm.). This announcement has had the effect of stimulating considerable interest in alternative tuber treatments to control black scurf.

The present study relates to the seed-borne nature of black scurf and control of tuber-borne inoculum by seed treatments with alternative chemical compounds.

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INTRODUCTION

Black scurf of potatoes, a disease of world-wide distribution (Walker, 1952), is also known by a great variety of other common names, e.g. scurf, black speck, stem canker, little potato disease, Rhizoctonia disease, black scab (Dana, 1925; Heald, 1926) and Corticium disease (in reference to the generic name earlier applied to the perfect stage of the fungus). In the present study "black scurf" is the preferred name as this is the one most commonly applied in New Zealand, and aptly describes the evidence available to growers that inoculum is associated with a particular seed line.

On account of its ready transmission by way of seed tubers, black scurf was probably present in New Zealand from the time of the first introduction of the potato. However, it was not officially recorded until 1922 (Cunningham, 1922) and currently is widely distributed throughout New Zealand (Claridge, 1972). In spite of its prevalence and the widely held belief of its economic significance there have been no serious studies on the disease in New Zealand since those of Cunningham (1925a, 1925b), Cunningham and Neill (1926) and Chamberlain (1931a, 1931b, 1932a, 1932b, 1935).

The following review aims at summarizing salient features of the disease cycle and other information considered relevant to the objectives of the present study.

A. SYMPTOMS

Black scurf presents a great variety of symptoms and signs which may be expressed on shoots, stems, stolons, roots, tubers and in the aerial parts of the plant.

(i) Shoots and Stems: When seed tubers sprout the growing tip is particularly susceptible to Rhizoctonia attack which is manifest as a reddish-brown

dry necrosis, which in turn induces shoot death. In such cases dormant buds may grow out from below the necrotic area or from the base of the original shoot to form secondary shoots (Figure 1). Depending on the prevailing soil conditions so these secondary shoots may or may not escape infection. Lesions may also develop along shoots as slightly sunken, reddish-brown, elongate necrotic areas which in extreme cases may girdle the stem (Figure 2). It is this conspicuous phase of the disease which arouses the concern of potato growers.

In extreme cases where shoots fail to reach the soil surface the effect is one of "gappiness" in potato crops (Figure 3). In less severe cases where secondary and subsequent shoots develop the effect is that of delayed and uneven emergence. When cankers completely girdle stems immediately below ground level, the normal downward flow of carbohydrates to the developing tubers is disturbed, and their accumulation in the tops is accompanied by stunting, rosetting, formation of aerial tubers in axes of branches and petioles, and enhancement of purpling due to excessive anthocyanin formation (Walker, 1952).

(ii) Stolons and Roots: Reddish to dark-brown lesions similar to those occurring on shoots are frequently found on stolons. The tips of stolons can be attacked (Figure 4), thereby preventing the formation of tubers, or stolons can be girdled at a later stage resulting in the production of undersized potatoes. A consequence of death of some stolons on the one plant may be the production of oversized tubers on the surviving stolons, due to reduced competition for nutrients.

Root damage is said to be an important phase of the disease, especially under dry conditions where the formation of new roots is retarded (Dana, 1925). Yellowing and rolling of leaves may accompany such damage (Dana, 1925) and it is claimed by Gussow (1917) that the death of fine feeding roots, rather than stem lesions, account for the secondary symptoms in the aerial parts of plants.

(iii) Tubers: Various tuber defects associated with R. solani have been described:

(a) tuber pits (Morse and Shapovalov, 1914; Ramsey, 1917);

(b) tuber rots (Shapovalov, 1922);

(c) irregular, malformed tubers and surface russetting (Morse and Shapovalov, 1924; Dana, 1925).

However, doubt has been expressed as to whether R. solani is the primary pathogen causing tuber pits and irregular malformed tubers (Heald, 1926).

A secondary consequence of damage to both stems and stolons is the formation of "little potatoes" in clusters around the base of stems, or directly from the seed tuber on short stolons (Dana, 1925). The "little potato" condition is illustrated in Figure 5.

It should be noted that some of the symptoms attributed to this disease, particularly aerial symptoms and "little potatoes" can arise from other sources. For example virus diseases may induce rosetting, stunting, yellowing and leaf rolling (Dana, 1925), and the "little potato" condition may result from the use of old or poorly stored seed tubers (Driver, 1972). Further, Edson and Shapovalov (1918) produced stem lesions on potatoes similar to those caused by R. solani by inoculating with several other species of fungi. Throughout the present study isolations were made in all instances where the cause of stem, stolon and root lesions was in doubt. Isolation and identification procedures are detailed in Appendix II.

B. SIGNS

Dark brown or black sclerotia on tubers are the most characteristic and diagnostic signs of the disease (Figure 6). Such sclerotia are irregular in size and shape ranging from minute specks to large angular aggregates an inch or more across. They are entirely superficial but are firmly attached to the tuber surface and leave no perceptible scar when

removed. Dark brown runner hyphae of the fungus can be seen on the tuber surface connecting sclerotia, which are often located very close to eyes (Figure 7) and may even form over eyes.

Frequently a conspicuous brown mantle of interlacing hyphae forms on shoots, roots and stolons (Figure 8). Such mycelium may be abundant without causing any definite lesions, while in other cases lesions may be evident, and the mycelium relatively inconspicuous. Sclerotia are frequently formed from these runner hyphae on shoots, roots and stolons (Figures 1 and 8).

Under warm, moist, conditions, mycelium may aggregate around stem bases to form a whitish weft or mantle (hymenium) which bears the perfect stage of the fungus.

Figure 1 Potato shoot symptoms caused by R. solani, showing the development of secondary shoots following death of primary shoots.

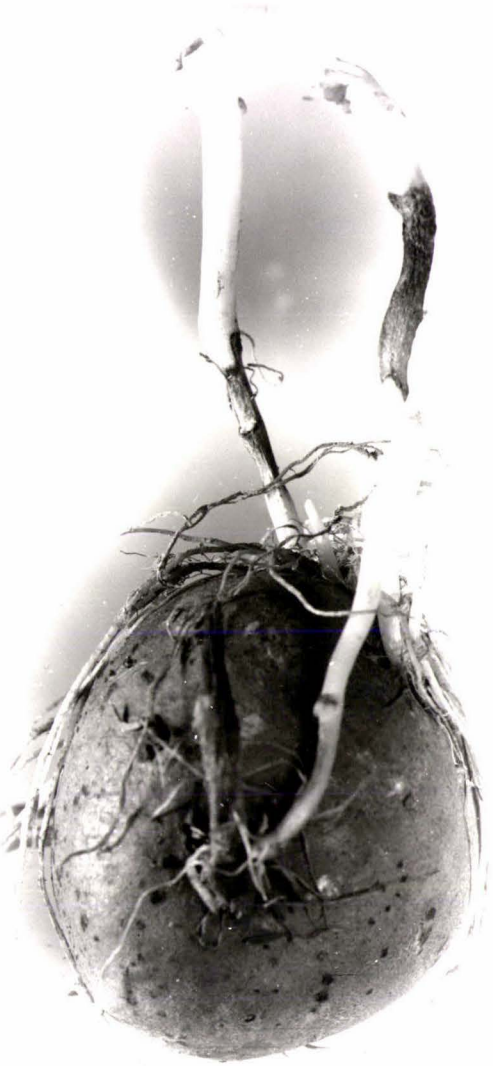

Figure 2 Girdling of potato shoots, caused by R. solani.

Figure 3 "Gappiness" in a commercial potato crop as a result of death of shoots caused by R. solani.

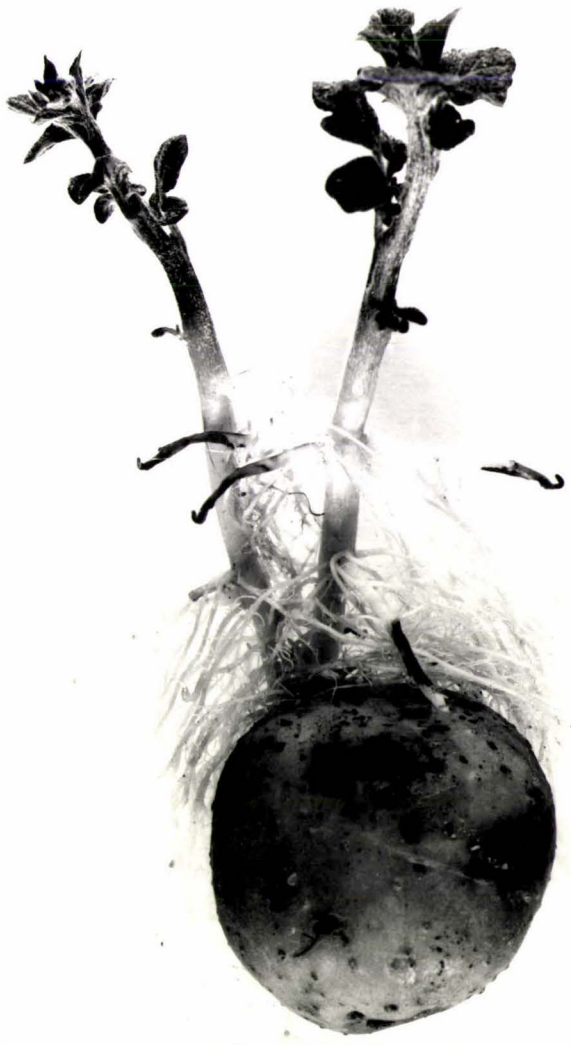

Figure 4 Death of stolon tips, caused by R. solani.

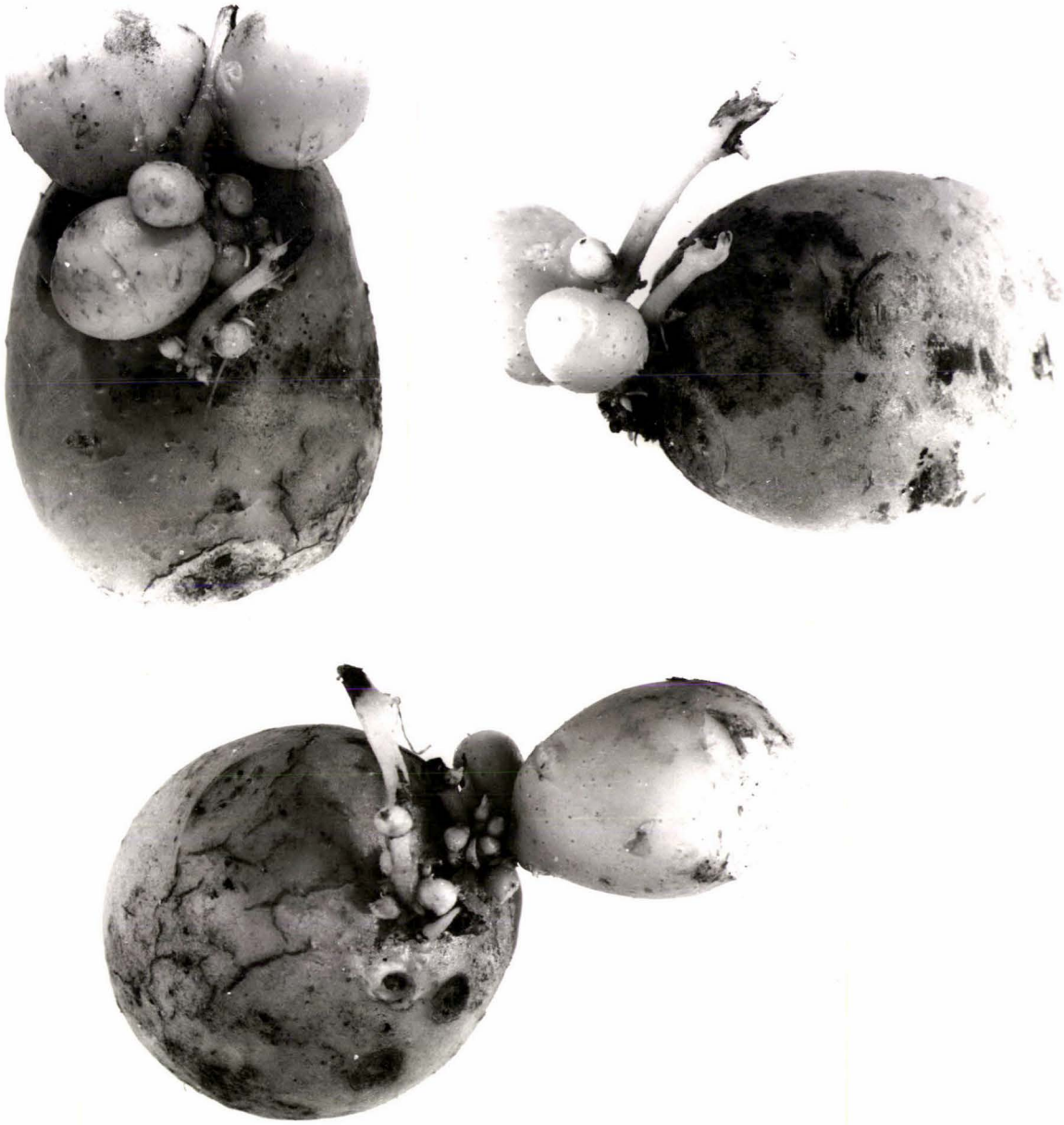

Figure 5 The "little potato" condition.
Note lesions on shoots, caused by R. solani.

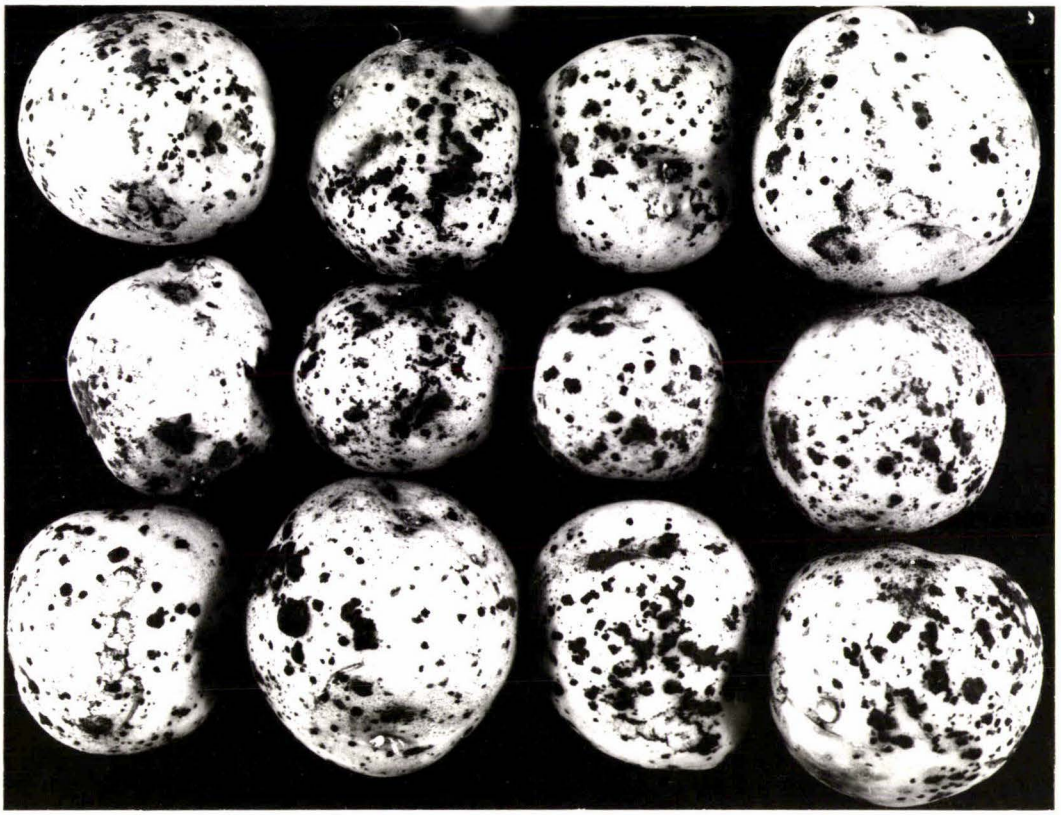

Figure 6 Sclerotia of R. solani on seed tubers (var. Ilam Hardy, certified seed).

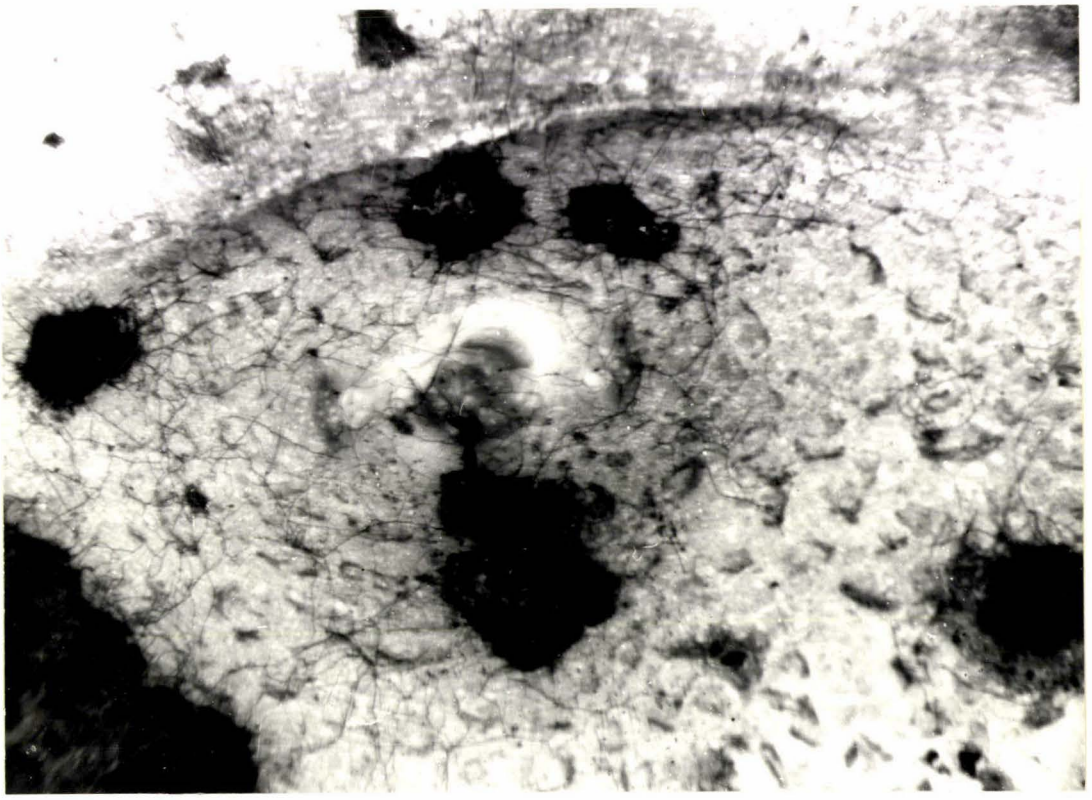

Figure 7 Close-up view of tuber surface showing proximity of sclerotia and hyphae of R. solani to eyes.

Figure 8 Runner hyphae and a sclerotium of *R. solani* on a potato shoot.

C. ETIOLOGY

Primary infections may arise from either of two sources - the soil, or sclerotia adhering to seed tubers (Heald, 1926; Butler and Jones, 1949; Walker, 1952). Pathogenic strains of the fungus may be present in soil as a result of planting infected tubers and conceivably may survive there in three ways:

(i) As a saprophyte. The competitive saprophytic ability of R. solani has been established (Blair, 1943; Papavizas and Davey, 1962).

(ii) As an active parasite on other crop or weed hosts. R. solani has a very wide host range (Baker, 1970).

(iii) In a dormant state. The pathogen could well persist either as dislodged sclerotia, or as sclerotia associated with crop debris.

It is questionable whether strains of R. solani pathogenic to potatoes are present in virgin soils. Pratt (1918) in the United States obtained sclerotia on progeny tubers from sclerotia-free, treated seed and also isolated R. solani directly from soil. However, he did not conduct any pathogenicity tests. Thornton (1958), in New Zealand, isolated R. solani from virgin soils. Although, in the experiments of Morrison, Ross and Thornton (1959), these isolates were not pathogenic to potatoes, no isolate of known pathogenicity was included as a control and so these results are questionable.

There are differing opinions as to the relative importance of seed and soil inoculum, although few definitive experiments have been conducted.

In the United States the experiments of Coons (1918), Melhus and Gilman (1921), Schultz, Gratz and Bond (1930), Schaal (1935) and Raleigh and Bond (1936) indicate that the inoculum potential of R. solani in their soils was very low and that tuber inoculum could be an effective source of the disease. In other experiments in the same country, however, the soil inoculum load has been so high that seed treatments have been of little value (Bisby, Higham and Groh, 1923; Clayton, 1929; Dana, 1925).

Experiments in Canada (Sanford, 1936) to determine the effect of potato tuber treatment on stem infection showed that one-third of the lesions which developed from planting non-treated tubers could be traced to soil inoculum, although great variability existed between individual experiments. Sanford's studies involved 34 experiments conducted over three years under a wide range of field conditions which included different crop sequences, culture and soil types.

In the Netherlands, tuber-borne inoculum is considered the most important source of infection (Emden, Labruyere and Tichelaar, 1966) and all tubers destined for seed purposes are treated in that country (Ir. Th. de Bruin, pers. comm.).

Hide, Hirst and Griffith (1969) state that black scurf may as often, or even more often, arise from soil-borne inoculum as that carried on tubers. However, their surveys of the health status of seed tubers revealed sclerotia of R. solani to be present in numbers considered sufficient to warrant seed treatments (Hirst, Hide, Griffith and Stedman, 1970). In Scotland it is not known how much infection is attributable to soil-borne inoculum under their conditions (A.E.K. Boyd, pers. comm.).

In Victoria (Australia) tuber-borne inoculum is regarded as an important source of infection - probably at least as important as soil-borne inoculum, but the relative importance varies from district to district (D.E. Harrison, pers. comm.).

Experiments in New Zealand indicated that R. solani in the soil can give rise to sclerotia on progeny tubers (Chamberlain, 1931b, 1935). However, the pathogenicity of these strains was not established.

One would be inclined to accept seed-tubers as the more important source of inoculum but for the fact that several workers have shown only a small percentage of isolates from sclerotia on tubers to be pathogenic to potatoes. Sanford (1938a) assigned 20-50% of 114 isolates from random sclerotia on random tubers from four fields to the zero or marginal classes of pathogenic rank. In Australia, tests with sclerotial isolates from a large number of tubers indicated that less than 10% were pathogenic to solanaceous hosts (Flentje, 1967). Person (1945) demonstrated that 70% of 70 sclerotial isolates from potatoes from

six States were either non-pathogenic or only slightly pathogenic, and only 7% were strongly pathogenic.

Basidiospores are commonly produced in potato crops, but the part they play in the disease cycle is obscure. It is stated, without evidence, that basidiospores can germinate to establish mycelium in the soil (Heald, 1926) and since basidiospore isolates have proved to be pathogenic to potato stems (Sanford, 1938a) it is conceivable that air-borne basidiospores may function to increase the soil inoculum load.

D. EPIDEMIOLOGY

Many soil factors, temperature, moisture, aeration, pH, texture, structure, nutrient status and microbial interactions may influence the development of black scurf, and of these, temperature and moisture are considered the most important (Heald, 1926; Butler and Jones, 1949).

Richards (1921), using Wisconsin soil temperature tanks, unsterilized soil and naturally infected tubers, demonstrated that lower soil temperatures were most conducive to the development of stem lesions. He obtained lesions on stems at temperatures between 9C and 27C, with the most severe attack occurring in the 12C to 21C range (optimum 18C). However, relatively serious damage frequently occurred at temperatures as low as 9C. Richards further observed that the destruction of growing points was most severe between 12C and 18C, but negligible at and above 21C. He also found that at 24C potato shoots emerged in about one-third of the time it took at 15C and this fact he suggested provided one explanation for the "escape" of shoots from injury at higher temperatures and the ability of later-formed secondary shoots to escape infection.

Richards (1923) also conducted field experiments involving different planting dates and different years and successfully correlated the prevalence and severity of the disease with lower soil temperatures.

In a series of tests with many isolates each uniformly incorporated in unsterilized soil, Sanford (1938b) was unable to

confirm Richards' results. He concluded that the results obtained in separate tests were not sufficiently definite or consistent to warrant the conclusion that disease expression was greater at 16, 18, 20 or 23C, or that growing point destruction was more severe at the lower temperatures.

This apparent conflict can be explained when one considers the source of infection in the two series of experiments. In Richards' studies, where tuber-borne inoculum was involved, it would seem reasonable for emerging shoots to "outstrip" the pathogen at higher temperatures. By contrast, in Sanford's experiments infections arose from inoculum uniformly distributed throughout the test soil, in which case shoot infections would be more likely to occur irrespective of soil temperature. Perhaps the work of Richards and Sanford allows the generalization that low soil temperature enhances the possibility of shoot infections, irrespective of inoculum source, and that athigher soil temperatures shoot destruction will be minimized if seed tubers are the main inoculum source.

No definite conclusion can be drawn as to the importance of soil moisture levels in governing the extent and severity of black scurf, as apparent from the studies of Sanford (1938b). He noted that the recovery of the host by means of secondary and tertiary shoots was better in a wet soil than in a dry one, regardless of temperature, but was unable to conclude whether a dry soil or a wet soil was more favourable for disease development. However, according to Boyd (1969) and McKay (1955) the disease is more severe under dry conditions, and Clark and Martin (1931) demonstrated that cankering of stems and stolons was much more severe in soils with a 20% moisture content than in those with a higher moisture content. By contrast, Rolfs (1904) found that heavy, poorly drained soils favoured the development of disease, and Schaal (1935) demonstrated that heavy irrigation increased the contamination of tubers with sclerotia.

The growth of R. solani through soil is restricted by poor aeration (Blair, 1943) and since aeration is influenced by soil

moisture, structure and texture, all these factors could possibly affect disease development.

Soil reaction is regarded by Heald (1926) as an important factor influencing the severity of black scurf. He suggests that R. solani thrives best in acid soils and reports that liming can reduce the severity of the disease. Morse and Shapovalov (1916) increased the infection of potato stems by 20% to 30% with row applications of sulphur, which presumably lowered the soil pH. However, McGreary (1967) reduced the incidence of sclerotia on tubers with soil applications of sulphur and increased the incidence with lime applications. Rhizoctonia solani grows through soil over a broad pH range with maximum growth at pH 7.0 (Blair, 1943) and Danish work (Frederiksen, Jorgenson, and Nielsen, 1938) demonstrated that soil pH within the range 4.7 to 7.6 was of little importance in the development of stem lesions. It would seem that pH within the range of most agricultural soils has little influence on the disease, although indirect effects of soil pH on available nutrients and the soil microbial population may be important.

The nutrient status of the soil, both organic and inorganic, will no doubt influence disease development because of effects on either the host, the pathogen or the other soil micro-organisms. Microbial interactions (synergism, antibiosis, hyperparasitism, fungistasis and lysis, competition) will also influence disease development. The effect of nutrition and microbial interactions on Rhizoctonia diseases in general and on the growth of R. solani in soil are considered by Baker and Martinson (1970) and Papavizas (1970).

According to workers in the Netherlands potato shoots become more resistant to attack following emergence (Emden et al., 1966). Thus, in any situation where shoot emergence is delayed, the chances of severe disease developing will increase, all other factors being equal. Factors which could reduce the rate of emergence of shoots include:-

(i) unfavourable temperature and moisture conditions;

(ii) reduced vigour of shoots because of virus diseases, or poor storage treatment of seed tubers;

(iii) planting depth. Stormer and Ebell (1944) obtained over a 50% increase in the number of severely diseased shoots by increasing planting depth from 5 cm to 15 cm. Thus some control of the disease could be obtained by shallow planting of tubers and using subsequent cultivations to form moulds. This would increase the rate of emergence of shoots, firstly because of the shorter distance they have to grow to reach the soil surface, and secondly, because of higher temperatures within this shallower covering.

A common cultural practice used in New Zealand for weed control is to harrow down moulds shortly before the first shoots emerge. It would seem that this practice has been an unconscious method of minimizing the disease by allowing shoots to emerge more quickly.

By contrast, cultural practices associated with use of pre-emergence herbicides enhance the likelihood of shoot infection. Weed control in this manner involves moulding the crop immediately prior to emergence followed by spray application of the herbicide. Since the moulds are subsequently left undisturbed, potato shoots are required to grow up through a considerable depth of soil with greatly increased chances of shoot infection occurring. This would apply more particularly to such varieties as Rua and Sebago which inherently are slow emerging.

E. EXTENT OF LOSSES

Reports as to the loss directly attributable to black scurf are conflicting.

Estimates from the United States between 1928 and 1939 claim an average loss in yield of 2.5%, with up to 10% loss reported from some States (Wood, 1932; McCallan, 1946). Reports of individual experiments conducted in the United States involving seed or soil treatments and/or seed selection indicate losses as either, severe (Cook and Lint, 1916; Richards, 1923), moderate but still significant (Dana, 1925; Leach, Johnson and Parson, 1929; Schultz *et al.*, 1930; Raleigh and Bond, 1936; Livingston, Oshima and Morrill, 1962; Cetas, 1971; Davis, Groskopp and Callihan, 1971), slight (Coons, 1918; Schaal,

1935; Schultz, 1966), or negligible (Heald, 1926; Biehn, 1969; Cetas, 1969a, 1970; Easton, Maxwell, Oldenburg and Anderson, 1970; Harrison, Johnson and Barmington, 1970).

However, the losses reported by some of the above investigators varied from year to year. For example, Richards (1923) obtained a 50% yield increase by seed treatment in 1918 but only a 15% increase in 1919. Likewise, Schultz, et al. (1930) and Raleigh and Bond (1936) obtained yield increases of up to 20% with seed treatments in some years but no increase in other years.

In Canada (Sanford, 1937) extensive experiments failed to show any consistent effects of the disease on total tuber yield, or the yield of large size or small size tubers.

Small (1943, 1945) as a result of experiments conducted over four years, concluded that black scurf causes little, if any, loss under farm conditions in England. He failed to show any reduction in yield despite quite severe attacks on shoots and stolons. Other experiments in the United Kingdom involving seed selected for the extent of infection with sclerotia indicated only slight losses in yield in all instances (Hirst, et al., 1970). Only a 6% to 7% reduction in yield resulted from planting severely infected seed, while moderately infected and unselected seed gave yield reductions of 4% to 5% and 3% respectively.

Australian and Dutch workers consider that the most important effect of black scurf is on the quality (size, shape and colour) of harvested tubers (D.E. Harrison, pers. comm.; Emder et al., 1966). However, significant increases in yield have been obtained by control of seed and soil inoculum in both Australia (D.E. Harrison, pers. comm.) and the Netherlands (Ten Boer, 1958; Lint and Emden, 1957).

In New Zealand the experiments of Cunningham and Neill (1926) using seed treatments, and those of Chamberlain (1932b) involving seed selection and seed treatments failed to show any consistent effect of the disease on yield. Chamberlain concluded that treatment of seed potatoes under New Zealand conditions was not warranted, mainly because of the doubtful effect of black scurf on yield and the possible injurious effects

of the then available seed treatments.

In field trials in the Rangitikei, Booth (1963) obtained increases in the yield of table potatoes of up to 27% following seed treatment. However, there was no evidence provided to prove that this increased yield was in fact due to control of black scurf.

Several factors could account for the variability apparent in the experiments reviewed above.

(i) In studies involving seed and soil treatments, these treatments themselves could have reduced yields, for example in the experiments of Dana (1925), Chamberlain (1932b) and Sanford (1937).

(ii) Seed selection may not have been effective in providing inoculum-free seed. Minute sclerotia and mycelium on "clean" tubers could provide sufficient inoculum to confuse results.

(iii) In experiments involving seed treatments, soil inoculum could have confused the results obtained, and vice versa.

(iv) In individual tests unsuitable environmental conditions or the absence of pathogenic strains could well have accounted for the absence of any adverse effect on yield.

(v) Where misses occurred or plants were severely diseased then adjacent plants could have compensated to an extent that yield was not reduced. Hirst et al., (1970) have demonstrated in healthy crops that removing 24% of the plants from random positions at emergence only resulted in a 9% reduction in total yield. They also claim that to obtain a 5% decrease in yield (approximately the least decrease that experimentally can be reliably detected) at least 15% of the plants would have to be removed at emergence.

It must be appreciated that the above percentages relate strictly to the circumstances of their experiments. Prevailing environmental conditions, variety, seed size, within-row and between-row spacing are all factors which in any crop could influence the degree of compensation provided by adjacent plants.

F. DISCUSSION

From the above review it is clear that in spite of considerable research throughout the world, there is little agreement on such basic questions as to the relative importance of soil-borne and tuber-borne inoculum, the influence of specific environmental factors and the economic significance of the disease. This is understandable if one accepts that each planting is in effect a unique situation. In every situation there will be variation in the seed-tuber and/or soil inoculum load, and the extent to which inoculum from either source will cause loss will be dependent in part on the virulence of the strain of *R. solani* present, and the prevailing soil environmental conditions. Obviously then, there is danger in generalizing on any aspect of the potato black scurf disease. Nevertheless, in the present study one basic assumption is made:

- that in New Zealand tuber-borne inoculum is the more important source of infection.

This view is held for the following reasons:

(a) In most instances potatoes are grown in soil out of long established pastures. Chamberlain (1935) in this country demonstrated that returning soil to pasture for 3 to 4 years practically eliminated *R. solani*. It follows that with the exception of those areas where potatoes are continuously cropped (e.g. Pukekohe) the soil-borne inoculum load must inevitably be low or non-existent.

(b) In a health survey of certified seed potatoes (Appendix III) pathogenic strains of *R. solani* were present on all lines examined, indicating seed-tubers as a consistent source of inoculum.

An obvious corollary to the above-stated premise is that control of black scurf will be consequent on use of an effective method of inactivating inoculum associated with seed tubers. Since mercury based compounds currently used for dipping seed tubers are to be withdrawn from the New Zealand market later this year (J.D. August, pers. comm.), there is obvious need for assessing the effectiveness of alternative chemical compounds for seed tuber treatment. This has been the major objective of

the present study. Specifically the work has involved:

(a) Screening a range of fungicides as to their ability to prevent mycelial growth and inactivate sclerotia of R. solani under laboratory conditions.

(b) Determining the capacity of various fungicides to prevent development of shoot lesions when applied as seed tuber treatments under glasshouse and field conditions.

(c) Investigations on the systemic properties of some fungicides in relation to prevention of shoot lesioning.