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Some Aspects of Seed Infection and Control of the
Collar-rot Complex of Peas (Pisum sativum L.),
caused by Mycosphaerella pinodes (Berk. and Blox.)
Verstergr., Phoma Medicaginis var. pinodella (Jones)
Boerema, and Ascochyta pisi Lib.

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by

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SUMMARY

1. The collar-rot complex of peas caused by Mycosphaerella pinodes, Phoma medicaginis var. pinodella and Ascochyta pisi is present in the Manawatu, but only mycosphaerella blight (caused by M. pinodes) is prevalent.
2. The component diseases and causal fungi of the complex are readily identified on the basis of their symptoms and pycnidiospore morphology.
3. Symptoms induced by A. pisi are distinct from those of M. pinodes and P. medicaginis var. pinodella. Although symptoms induced by the latter two pathogens are indistinguishable, in the field situation M. pinodes generally induced more profuse lesioning.
4. The pycnidiospores of M. pinodes and A. pisi are large and uniseptate, those of M. pinodes being broader (11.5×4.3 u) than those of A. pisi (13.2×3.7 u). Pycnidiospores of P. medicaginis var. pinodella are non-septate (or occasionally uniseptate) and smaller (7.8×2.9 u). Mycosphaerella pinodes can also be identified by the production of ascostromata and ascospores on agar and on infected plants.
5. The disease complex was present in 60% of 86 New Zealand produced pea seedlines from the 1969/70 and 1970/71 harvests.

The level of infection in individual seedlines was low, only 18% being infected to more than 1%.

6. Mycosphaerella pinodes constituted 66.9% of the collar-rot fungi isolated to agar from commercial seedlines, the remainder being P. medicaginis var. pinodella. In no instance was A. pisi detected.
7. The infection level of individual seed lines can be increased by macroscopic selection on the basis of seed discolouration, fluorescence under ultraviolet light and smaller seed size.
8. All three pathogens are highly sensitive to benomyl and thiram, mycelial growth being suppressed by 50% (ED₅₀) at rates of less than 10 ug/ml a.i.
9. Benomyl is taken up and systemically translocated in pea plants when applied as a soil drench, foliar spray and seed dressing.
10. When applied as a soil drench benomyl is taken up and translocated at concentrations of 250 to 1,000 ug/ml a.i. In plants drenched with the higher concentrations translocation is more widespread and accumulation is higher.
11. By the third week after drenching at 250 to 1,000 ug/ml a.i. the distribution of benomyl in all treated plants is bimodal, being concentrated mainly in the leaves of the lower and upper

nodes (nodes 2 and 10 respectively). Within individual leaves, benomyl is mainly concentrated in the leaf tip and margins.

12. When drenched at 500 ug/ml a.i., benomyl persists up to nine weeks in treated plants and peak accumulation occurs at approximately the third week. The chemical is translocated to leaves, pedicels, sepals, podwall and maturing seeds, but is not detected in the petals.
13. When applied as a foliar spray at concentrations of 62.5 to 250 ug/ml a.i., benomyl is taken up by pea plants but translocation is not widespread.
14. In glasshouse trials benomyl applied as a soil drench and a foliar spray was systemically absorbed and provided significant but not complete protection.
15. Benomyl applied to pea seeds as a dust or slurry (1 to 6 oz Benlate/bushel) is taken up by germinating seeds and accumulates mainly in the cotyledons and the hypocotyl/epicotyl regions of the seedlings. In glasshouse and field trials the absorbed chemical inactivated deep seated mycelium of M. pinodes and P. medicaginis var. pinodella in most but not all infected seeds.

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Chapter I

INTRODUCTION.

The green pea, Pisum sativum L., is one of the principal field crops in New Zealand (Anon, 1970a), and essentially two varieties, P. sativum var. sativum (garden peas) and P. sativum var. arvense (field peas) are grown. Peas of the garden variety are used for human consumption and may be consumed fresh, processed or marketed as split peas. The field variety is mainly used for animal consumption. However both varieties are widely grown for seed production and are important to New Zealand in international seed trade.

The cultivation of green peas in New Zealand has increased steadily over the last few years. In 1966-67 approximately 28,000 acres were sown, while in 1968-69 this had increased to approximately 50,000 acres (Anon, 1970a). The main pea producing areas are Canterbury, Wellington, Marlborough, Hawkes Bay and Otago, with Canterbury alone being responsible in 1967-68 for three-quarters of the total production in New Zealand (Anon, 1969). In 1968-69 export of seed peas and artificially dehydrated peas alone resulted in total earnings of more than two million dollars (Anon, 1970b).

The average yield of peas for the last few years has been approximately 35-40 bushels/acre (Anon, 1969). However this does not reflect the genetic potential of the crop owing to the influence of a variety of adverse environmental factors, of which diseases frequently play an important part. Some fifteen fungal diseases have been recorded

on peas in New Zealand (Dingley, 1969), and of these the collar-rot complex, and pea wilt (Fusarium orthoceras Appel. and Wollenw. var. pisi Linford; synonym Fusarium oxysporum Schlecht. f pisi) are considered the most important (Brien *et al.*, 1955).

The so-called collar-rot complex is in fact three distinct diseases grouped for convenience on account of the similarity of the symptoms induced and the close mycological relationship of the three causal fungi. The causal organism and common name of each component of the complex is as follows:

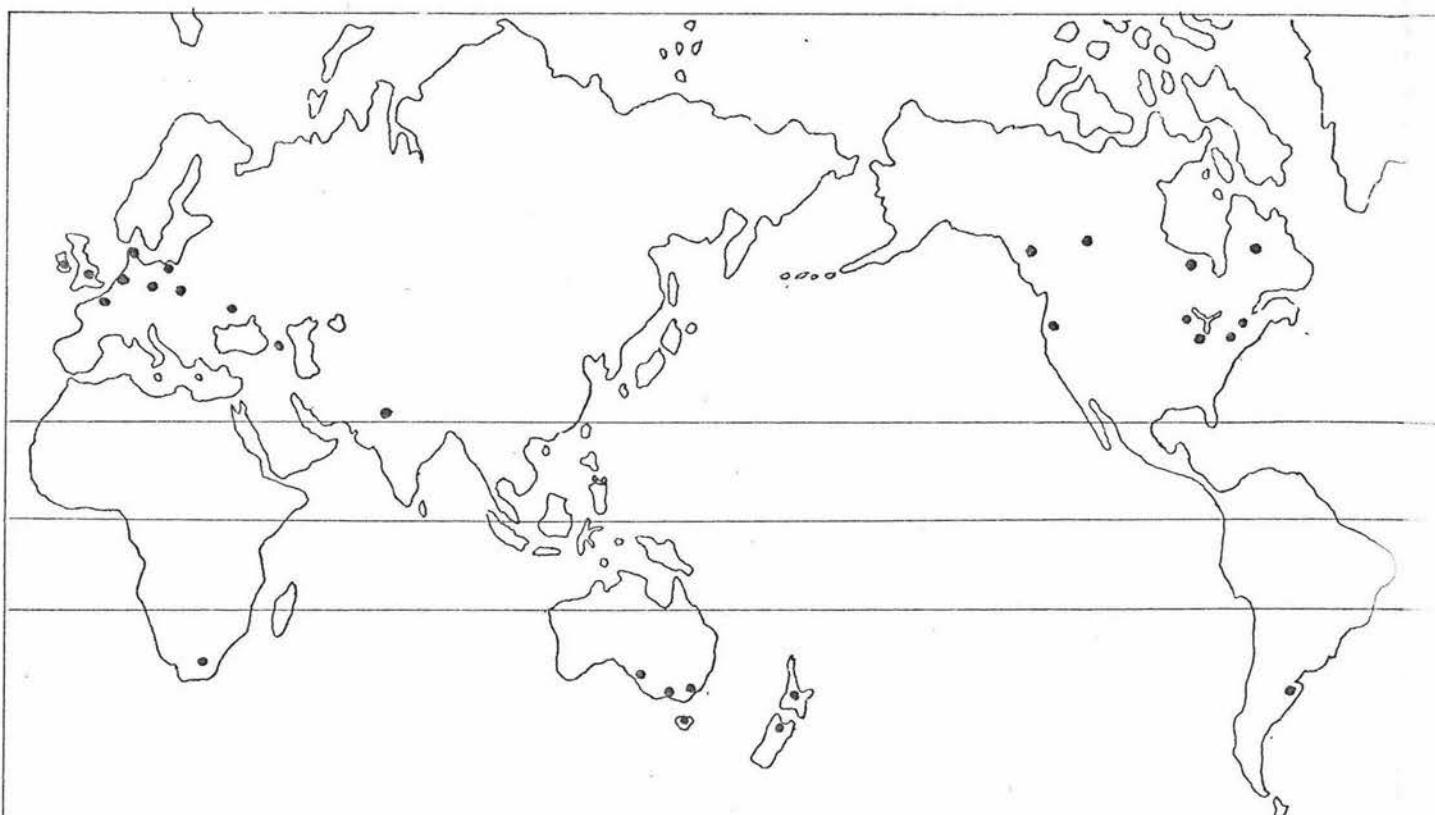
- (i) Mycosphaerella pinodes (Berk. and Blox.) Vestergr. (Synonym: Ascochyta pinodes Jones), causing mycosphaerella blight
- (ii) Phoma medicaginis var. pinodella (Jones) Boerema, causing ascochyta footrot
- (iii) Ascochyta pisi Lib., causing ascochyta leaf and pod spot.

Ascochyta pisi attacks pods, seeds, leaves and sometimes stems, while P. medicaginis var. pinodella and M. pinodes cause lesions on pods, seeds, leaves, stems and roots (Hare and Walker, 1944; Brien *et al.*, 1955). In all cases, where infections are severe the result is a significant reduction in both the quantitative and qualitative yield of seeds (Jones, 1927; Wallen, 1965; Maude, 1966).

The collar-rot complex is of world wide distribution (Map 1) and has been the subject of a great deal of research. The essential features of the epidemiology of each pathogen has been determined and these are summarised in Table I.

From the above table it is apparent that for all three components of the complex seed is considered an important source of primary infection.

Map 1. World distribution of the collar-rot complex in peas.



REFERENCES:

North & South America

- a. Alberta: Skolko *et al.*, 1954.
- b. Br. Columbia: "
- c. Quebec: "
- d. Ontario: "
- e. New York: Jones, 1927.
- f. Pennsylvania: Weaver, 1946.
- g. Minnesota: Starr, 1932.
- h. Wisconsin: Hare & Walker, 1944.
- i. Washington: Gould, 1949.
- j. Argentina: Jauch, 1941.

Australia & New Zealand

- a. N.S. Wales: Walker, 1961.
- b. S. Austr.: Carter & Moller, 1961.
- c. Victoria: Stubbs, 1942.
- d. Tasmania: Geard, 1961.
- e. New Zealand: Dingley, 1969.

Europe & Russia

- a. Czechoslovakia: Zacha, 1967.
- b. Denmark: Neergaard, 1940.
- c. England: Moore, 1946.
- d. France: Anselme & Champion, 1962.
- e. Germany: Mansurat & Stephan, 1962.
- f. Netherlands: Schoorel, 1963.
- g. Poland: Bajan, 1968.
- h. Republic of Ireland: Ryan, 1966.
- i. Armenia: Teterevnikova-Babayan, 1963.
- j. Moldavia: Balashova, 1965.

Africa

- a. S. Africa: Doidge *et al.*, 1952.

India

- a. Punjab: Sattar, 1934.

Table I. Salient features of the epidemiology of the diseases caused by M. pinodes, P. medicaginis var. pinodella and A. pisi (Jones, 1927; Hare and Walker, 1944; Carter and Moller, 1961 and Wallen et al., 1967b).

Phase of disease cycle	<u>Mycosphaerella pinodes</u>	<u>Phoma medicaginis</u> var. <u>pinodella</u>	<u>Ascochyta pisi</u>
<u>Overwintering Phase</u>	1.In seed. 2.In infected pea plant debris. 3.In soil.	1.In seed. 2.In infected pea plant debris. 3.In soil.	1.In seed. 2.In infected pea plant debris.
<u>Primary Inoculum</u>	1.From infected seeds. 2.Pycnidiospores from infected pea plant debris. 3.Ascospores from plant debris. 4.From soil-borne inoculum.	1.From infected seeds. 2.Pycnidiospores from infected pea plant debris. 3.From soil-borne inoculum.	1.From infected seeds. 2.Pycnidiospores from infected pea plant debris.
<u>Secondary Inoculum and spread</u>	1.Localised spread by rainsplashed pycnidiospores from primary lesions. 2.Long distance spread by airborne ascospores from primary lesions.	1.Localised spread by rainsplashed pycnidiospores from primary lesions.	1.Localised spread by rainsplashed pycnidiospores from primary lesions.

For this reason considerable emphasis has been placed on the establishment of crops using pea seed free of infection (Cruickshank, 1957; Wallen, 1965). Ideally seed should be produced in climatic areas not conducive to disease development, thereby ensuring the production of pathogen free seed. In all pea seed producing areas in New Zealand climate does have some restrictive influence on the collar-rot complex but insufficient to guarantee freedom from the component pathogens. For this reason there is need for a practical seed treatment method to inactivate inoculum associated with seed. Non-systemic fungicidal dusts applied either in dry form or as a slurry are relatively ineffective on account of the pathogens being established deep within the seed (Maude, 1966; de Tempe, 1968b), and hot-water seed treatments have failed to eradicate the pathogens without impairing seed viability (Ogilvie, 1933). The thiram soak method of seed treatment developed by Maude (1966) is effective against M. pinodes and A. pisi, but is impractical on account of the difficulty of drying large quantities of seed.

A recent development showing great promise for the control of seed-borne diseases has been the introduction of fungicides with systemic activity. When used as a seed dressing such fungicides are absorbed and inactivate deep-seated infection without adversely affecting seed germination (Catling, 1969; Maude and Shuring, 1969). In the United Kingdom, Maude and Kyle (1970) have demonstrated complete control of A. pisi using the systemic fungicide benomyl as a seed dressing, but no reference was made to its effectiveness in controlling P. medicaginis var. pinodella or M. pinodes. Since Cruickshank (1957) has shown these latter two species to be also commonly associated with New Zealand produced seed peas there is obvious need to explore the effectiveness of benomyl

against all three components of the collar-rot complex under New Zealand conditions. Such an investigation constituted the main theme of the present study.

However before this work could proceed it was first necessary to conduct symptomatological and mycological studies to enable ready identification of the component diseases and their causal fungi. Further, to ascertain the relative prevalence of the three fungi in New Zealand produced seed a survey was undertaken over two years of the state of health of commercially available seed lines.

In summary then, the main objectives of the present investigation were:

- (i) to determine the distinctive features of the component diseases and their causal fungi
- (ii) to survey the health status of pea seed lines harvested in New Zealand in 1969/70 and 1970/71
- (iii) to investigate the effectiveness of some recently available fungicides for the control of the collar-rot complex when applied as a seed dressing.