RESISTANCE OF POTATO VARIETIES TO CYST NEMATODES

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ABSTRACT

Heterodera, the cyst forming nematode species affecting potatoes has a highly specialised method of survival for prolonged periods of time in the absence of host crops. Its insidious form of spread and the difficulty encountered in detecting its presence notably during early stages of infection, make this pest one of the most difficult organisms to control and eradicate through chemical therapeutants.

Eradication of the cyst nematode by the withdrawal of high value land from potato production is unlikely to succeed because there is no method which could stop the passive spread of cysts into other areas.

The general use of resistant potato varieties offers a cheap and lasting solution to the nematode problem.

A generalised form of resistance operative against all pathotypes of the type stemming from **Solanum vernei** has distinct advantages in the long run as opposed to the use of varieties incorporating strain specific resistance.

INTRODUCTION

The cyst forming nematode species, **Heterodera rostochiensis sensulato,** is specifically pathogenic to potatoes [**Solanum tuberosum** subsp. **tuberosum**] and tomatoes [**S. emasculatum**]. In spite of every attempt to prevent or slow down its spread, the nematode has found its way into practically every potato-growing country of the World (Jones and Jones, 1964). Some of the factors responsible for its widespread distribution are (a) Ease of spread, by any means involving movement of soil. (b) Highly specialised nature of its method of survival in the absence of host crops over prolonged periods of time. (c) Difficulty of detection. As it is a soil-borne organism, detection during early stages of infection is virtually impossible. Usually foci of infection are discovered only after populations have reached epidemic proportions.

Attempts to eradicate cyst nematodes through the use of toxic chemicals by other means have so far been unsuccessful. The cyst wall is highly impervious to chemical toxins, and moreover the small proportion of cysts deeper than 20 cm in the soil is not affected because of inadequate diffusion of the chemical.

Partial control through the regular use of nematicides is possible but has been too expensive to use on a field scale. It would allow a good crop to be grown, but it usually results in an increase in the nematode population, because the surviving larvae are able to develop successfully on the strong plants resulting from the initial kill. There is also the problem that repeated use of nematicides can lead to residues in the soil which will taint crops, and the long term effects on populations of other organisms in the soil are not known and may be undesirable.

The development of resistant varieties offers a cheap and lasting solution to the nematode problem. The general use of resistant varieties would appear to have two advantages, (a) it will help reduce the density of cyst nematode and, (b) it will eliminate yield losses due to nematode infection.

Nematode population density in the field

The weight per hectare of the top 20 cm of soil is approximately 2570 tonnes or 2.5×10^9g. When susceptible potatoes are grown continuously, there may be 100-500 nematode eggs per gram of soil. The terminal field populations are of the order of 10^{12} eggs per ha (Jones 1966). Periods of five or six years without susceptible host crops reduce populations to about 10^{11} eggs per ha. At this level of infestation, potato crops grow well and yield loss is slight or negligible. It is very rare for the pest to multiply to its maximum capacity, which has been estimated to be 22 cysts per cm of root length (Trudgill 1968).

It is difficult to generalise about the threshold levels at which losses begin to occur, because of such factors as soil type, moisture, temperature, carryover of residual cysts and the effect of predaecious fungi in the soil. However the National Agriculture Advisory Service in Britain used threshold values ranging from 10 to 50 eggs per gram of soil according to circumstances. Williams (1958) suggested a threshold level of 10-20 eggs/g for peat soils, while Jones (1966) suggested 10 eggs/g for light sandy soils.

Estimates of the loss in yield caused by nematodes under English conditions are 0.25 tonnes/ha per full cyst per 10 g soil, and 0.1 tonnes/ha per egg/g for silt soils (Brown 1969).

Nematode Pathotypes

Because of the 'founder' effect (Mayer 1964) of cyst colonies developing in close proximity to each other without having to interbreed, whereby a certain gene could be fixed in some populations and absent or in very low frequency in others, it is necessary to identify pathotypes within fields so as to make the best possible use of resistant varieties. Potato cyst pathotypes can be identified by cyst chromogenesis (Guile 1966), morphogenesis (Webley 1970), electrophoresis of proteins (Trudgill **et al.** 1971), and by pot tests using a series of differential host plants incorporating strain-specific resistance genes.

Using British and Dutch notifications the following pathotypes have so far been identified (Table 1).

The nomenclature of cyst nematode pathotypes/species needs redefining because the present system of notifications is inadequate and confusing. It is likely that with the discovery of further sources of resistance we will be able to identify new pathotypes.

Breeding for pathotype A resistance

Pathotype A is the most widely distributed of the cyst nematodes in Europe and America.

Breeding for resistance commenced with the discovery of near immunity in a few clones of **Andigena** potatoes (Ellenby 1952). As **Andigena** potatoes are tetraploids and closely related to the domesticated potato, the resistance gene designated H1 (Toxopius & Huijsman 1953) was easily incorporated into existing breeding programmes. Several resistant varieties including Maris Piper, Ulster Glade, Pentland Javelin, and Hudson are now available.

Resistant potatoes produce a root diffusate with a hatching power similar to that of susceptible varieties. Nematode larvae invade their roots in numbers comparable to those invading the roots of susceptible varieties, but for some unknown reason females fail to mature.

The growing of resistant potatoes decreases nematode populations by 50 to 70% (Cole and Howard 1962) as compared with a 25% reduction from a non-host crop.

Other sources of resistance to pathotype A have been discovered in **S. spegazzinii, S. gourlayi** and **S. maglia** (Anon. 1957 to 1963), all wild relatives of the potato, none of which has so far been used in the breeding of commercial potato varieties.

Breeding for resistance to Pathotype B

Dunnett (1957) discovered in Scotland a population of cyst nematodes which produced many cysts on potatoes with gene H1. Subsequent surveys (Jones 1958, Huijsman 1962, Goffart 1957) indicated that similar populations were to be found in other parts of Europe as well.

S. multidissectum, a wild diploid species, was found to have resistance designated gene H2 (Dunnett 1961) to the new pathotype B, but not to populations to which gene H1 gave resistance.

Although up to now no variety has been released for commercial use incorporating the resistance gene H2, a number of varieties incorporating resistance gense H1 and H2 are in their final stages of selection. The advantage of such a variety is that it can be grown in fields where pathotypes A and B occur as mixtures, which are quite common.

Resistance to pathotype B has also been discovered in **S. sanctaerosae** (Anon. 1957), **S. juzepeczukii** (Cole and Howard 1962) and **S. oplocense** (Anon. 1965), although these sources have so far not been used in the breeding of commercial varieties.

Breeding for resistance to Pathotype E [H. pallida]

A number of nematode populations have been found capable of overcoming the resistance to genes H1, H2 and H1H2 (Jones **et al.** 1970) and have been designated pathotype E. Because of colour differences during the maturation of cysts of pathotype A and E (Guile 1966), differences in first-instar larvae, in length and in size and shape of mouth stylet (Webley 1970) and differences in proteins (Trudgill **et al.** 1971), pathotype E is now considered a separate species, **H. Pallida.** The inability of pathotypes A and E to interbreed is not conclusive. The low compliment of eggs that resulted from single larvae matings of pathotype A and E (Parrott 1972) could have arisen because of fitness differences between larvae. Under natural conditions multiple fertilisations are known to occur in which a number of males are involved possibly of the one pathotype, resulting in a large number of eggs being produced.

Because of fitness difference between Pathotypes A and E (McKenna **et al.** 1972) it is natural for one pathotype to dominate a second pathotype if they occupy the same ecological niche. Such a situation however does not preclude the existence of genes in the nematode gene pool, which manifest themselves as new pathotypes when selection is altered, with the introduction of a resistant variety, (Cole and Howard, 1966). The complex that has arisen between pathotypes (some now called species) appears to be a form of gentic polymorphism akin to that observed in **Cepea nemoralis** and other organisms (see Ford, 1965).

Resistance to pathotype E has been reported to occur in some clones of **Andigena** potatoes, and has been designated H3 (Howard **et al.** 1970, Howard and Fuller 1971). The gene H3 gives no resistance to pathotype A, but high resistance to B and E. A similar resistance gene has been reported to occur in **S. multidissectum** derivatives, (Parrott **et al.** 1970 1972).

S. vernei, a diploid species from South America, has shown a generalised form of resistance to all known pathotypes including pathotype E (Rothacker 1957, Rothacker 1959, Dunnett 1960). Breeding of potato varieties using this source of resistance has reached a fairly advanced stage in Scotland and Germany, and the release of commercially suitable cultivars is imminent (Dunnett, pers. comm.).

The use of resistant potato varieties

The decrease in nematode populations in the absence of host crops probably varies to some extent with soil types. Over a 10-11 year period, in the absence of potatoes, under British conditions populations have declined by 95%. However, Brown (1972) is of the opinion that this decrease is likely to be lower in future because of natural selection for longevity.

The principle governing the use of resistant varieties is based on maintaining cyst numbers below the critical threshold level. The effect on a nematode population of growing resistant potatoes depends upon the pathotypes in the population and the resistance of the clones used to the different pathotypes. In areas where pathotypes are predominantly of one type, resistant varieties can be grown successfully for a number of years. The reduction in cyst numbers would then allow regrowing of susceptible varieties.

In areas where mixtures of pathotype occur, as is the case in most fields, resistant varieties have to be used with some degree of caution. It has been shown by Cole and Howard (1962) and Huijsman (1963) that the growing of four or five successive crops of potatoes incorporating gene H1, in infested soil in which the nematode was originally predominantly pathotype A, led to a population which was mostly not pathotype A.

In such fields a susceptible variety grown alternatively with a resistant variety would slow down the genetic change in the nematode population, while still maintaining it below critical levels. When potatoes are grown in a three or four course rotation, it would take over 20 years for the nematode pathotype to change completely.

For areas which follow short rotations, or no rotation at all, an integrated approach using resistant and susceptible varieties and nematicides would seem most feasible. The Dutch claim that this integrated control permits good yields of potatoes for starch production (Nollen and Mulder, 1969).

The potato cyst nematode was first reported in New Zealand in a paddock near a residential area in Pukekohe in August 1972. Based on infection levels it was suspected the infestation was at least six years old and possibly older. Prior to its discovery it is likely for cysts to have spread to other areas on soil adhering to potatoes, on tractors and other farm machinery, feet of animals and boots of farm workers, potato containers whether they be sacks, wooden cases or pallet boxes, on roots of transplants, and through wind and water erosion. Following the initial outbreak and as a result of field surveys conducted by Officers of the Ministry of Agriculture and Fisheries, over 45 ha on 14 properties, on which the cyst nematode has been found are now under quarantine.

Eradication of the potato cyst nematode from Pukekohe is undoubtedly a very attractive proposition, because this will remove the threat of crop losses from this pest for all time to come. Extermination of the potato cyst nematode by chemical means has proved an expensive failure wherever it has been practiced because a small fraction of cysts escape treatment. For example, on Long Island, U.S.A. persistant efforts at eradicating the pest using toxic chemicals over a 20 year period have failed and subsequent to this the cyst nematode has spread into upper New York State and into parts of New Jersey.

Eradication of the potato cyst nematode through the isolation of paddocks that have been treated by a nematocide is unlikely to succeed because it could take 15 to 20 years and possibly longer for the cysts to become completely empty through spontaneous hatching. During this period of time no solanaceous hosts could be grown. However, there is no guarantee that the balance of 2% or so viable cysts still remaining in the soil will not spread by any of the means already enumerated into other areas which may be growing potatoes. Such a system of eradication is very expensive to operate because more and more high value land is likely to go out of production, which imposes considerable hardship upon growers.

The effectiveness of both systems of eradication hinges to the precision with which new infestations can be detected. Experimental studies on the biology and population dynamics of the cyst nematode have shown that a single cyst and its progeny are capable of eventually infecting an entire field. The pattern of infestation in fields in which the cyst nematode has been found, e.g. near points of entry, near areas used for filling up spray tanks with water, tends to suggest that these have arisen from the introduction of large numbers of cysts. The prospects of detecting infestations that have resulted from a few cysts are still extremely remote.

Control or suppression of the cyst nematode to below critical threshold levels by long term rotations in which potatoes are grown only once in every four to five years is a possibility, but practical only for those areas where nematode infestation level is low, or in those areas which would not be adversely affected by such rotations. In other areas where nematode infestations are relatively high, and potatoes are cropped intensively, as in Pukekohe, the use of resistant varieties offers a cheap and lasting solution.

The use of varieties incorporating resistance ex **S. vernei** which has a generalised form of resistance operative against all nematode pathotypes (Dunnet, 1960, Kort **et al.** 1972) has obvious advantages in the long term control of the nematode than varieties incorporating strain specific resistance. **S. vernei** derivatives do not stimulate a high rate of larval emergence from cysts of any of the known pathotypes, and further inhibits the maturity of both sexes of larvae (Williams 1958).

Table 1: Nomenclature of pathotypes (British and Dutch notations) in relation to their host plants

Host genotypes	Pathotype notations						
	British			Dutch			
	A	B	E	A	B	C	D
	Y	C	W	Y	Y		
Colour of cysts during maturation							
S. tuberosum subsp **tuberosum**	+	+	+	+	+	+	+
S. tuberosum spp. **andigena** H1	0	+	+	0	+	+	+
S. multidissectum H2	+	0	+	+			+
Andigena x							
S. multidissectum H1H2	0	0	+				
spp. **Andigena** H3	+	+	0	0			
S. multidissectum D40	+	+	0	0			
S. kurtizianum					0	+	+
S. vernei	—	—	—	—			—

\+ Inability to form cysts

0 Inability to form cysts

— Ability to form few cysts

Y = yellow cysts
C = cream cysts
W = white cysts

REFERENCES

Anon. 1957. Director's report. Report Scottish Plant Breeding Station 5-24.

———— 1958. Ibid 5-30.

———— 1959. Ibid 5-22.

———— 1960. Ibid 5-23.

———— 1961. Ibid 5-24.

———— 1962. Record Scottish Plant Breeding Station.

———— 1963. Ibid 5-25.

———— 1965. Ibid 7-25.

Brown, E.B. 1969. Assessment of the damage caused to potatoes by potato cyst eelworm, **Heterodera rostochiensis** Woll. Annals of Applied Biology **63:** 493-502.

Brown, E.B. 1972. Potato cyst nematode, some practical considerations. Annals of Applied Biology **71:** 294-6.

Cole, C.S. and Howard, H.W. 1962. Further results from a field experiment on the effect of growing resistant potatoes on a potato-root eelworm. (**Heterodera rostochiensis**) population. Nematologica **7:** 57-61.

Cole, C.S. and Howard, H.W. 1966. The effects on a population of potato-root eelworm [**Heterodera rostochiensis**] of growing potatoes resistant to pathotype B. Annals of Applied Biology **58:** 487-95.

Dunnett, J.M. 1957. Variation in pathogenicity of the potato root eelworm (**Heterodera rostochiensis** Woll) and its significance in potato breeding. Euphytica **6:** 77-89.

Dunnett, J.M. 1960. The role of **S. vernei** Bitt. et. Wittm in breeding for resistance to Potato Root Eelworm [**Heterodera rostochiensis** Woll.] Report Scottish Plant Breeding Station 39-44.

Dunnett, J.M. 1961. Inheritance of resistance to potato root eelworm in a breeding line stemming from **S. multidissectum** Hawkes. Report Scottish Plant Breeding Station 39-46.

Ellenby, C. 1952. Resistance to the potato root eelworm, **Heterodera rostochiensis** Wollenweber. Nature London **170:** 1016.

Ford, E.B. 1965: "Genetic Polymorphism." Faber and Faber, London pp 101.

Goffart, H. 1957. Fortschritte angdem Gebiete der Fuchtung namatoden-resistanter Kartoffelsorten. Kartoffelbau. **8:** 194-5.

Guile, C.T. 1966. Cyst chromogenesis in potato root eelworm pathotypes. Plant Pathology **15:** 125-8.

Howard, H.W., Cole, C.S. and Fuller, J.M. 1970. Further sources of resistance to **Heterodera rostochiensis** Woll. in the **Andigena** potatoes Euphytica **19:** 210-16.

Howard, H.W. and Falker, J.M. 1971. Resistance to the cream and white potato cyst nematodes. Plant Pathology **20:** 32-5.

Huijsman, C.R. 1962. Physiological races in the potato-root eelworm, **Heterodera rostochiensis** Woll. European Potato Journal **5:** 106 (Abst.).

Jones, F.G.W. 1958. Resistance — breaking populations of potato-root eelworm. Plant Pathology **7:** 24-5.

Jones, F.G.W. and Jones, M.G. 1964. "Pests of fields crops". Edward Arnold London 406 pp.

Jones F.G.W. 1966. The population dynamics and population genetics of the potato cyst nematode **Heterodera rostochiensis** Woll. on susceptible and resistant potatoes. Report Rothamstead Experimental Station for 1965. 301-16.

Jones, F.G.W., Carpenter, J.M., Parrott, D.M. Stone, A.R., Trudgill, D.L. 1970. Potato cyst nematode: One species or two? Nature London **227:** 83-4.

Mayer, E. 1963. "Animal species and evolution". Harvard University Press. Cambridge, Massachusetts. pp 797.

Nollen, H.M. and Mulder, A. 1969. A practical method for economic control of potato cyst nematodes. Proceedings of the 5th British Insecticidal Fungicidal Conference 671-74.

Parrot, M. 1972. Mating of **Heterodera rostochiensis** pathotype. Annals of Applied Biology **71:** 271-3.

Parrott, D.M. and Trudgill, D.L. 1970. Variations in the resistance to **Heterodera rostochiensis** of potatoes derived from **Solanum tuberosum** sp. **andigena** and **S. multidissectum.** Annals of Applied Biology **65:** 385-91.

Parrott, D.M. and Trudgill, D.L. 1972. The resistance of hybrids to **Solanum tuberosum** sp. **andigena** and **S. multidissectum** to **Heterodera rostochiensis** pathotype E. Plant Pathology **21:** 86-8.

Rothacker, D. 1957. Beitrage zue Resistenzzuchturg gegen den Karteffelnematoden I. Prufung von Primitiv — und Wildkartoffeln augdas Verhaltengegenuber Kartoffelnematoden. Zuchten **27:** 124-31.

Rothacker, D. 1959. Symposion uber Fragen zur Zuchtung nematodenmiderstandsfabiger Kartoffeln in Gross-Lusewitz. 1958 Plant Breeding Abstracts **29:** 331.

Toxopeus H.J. and Huijsman, C.A. 1953. Breeding for resistance to potato root eelworm. I. Preliminary data concerning the inheritance and the nature of resistance. Euphytica **2:** 180-6.

Trudgill, D.L. 1968. The effect of environment on sex determination in **Heterodera rostochiensis** PhD Thesis, London University, pp 188.

Trudgill, D.L. and Carpenter, J.M. 1971. Disc electrophoresis of proteins of Heterodera species and pathotypes of **Heterodera rostochiensis.** Annals of Applied Biology **69:** 35-41.

Webley, D. 1970. A morphometric study of the three pathotypes of the potato root eelworm [**Heterodera rostochiensis**] recognised in Great Britain. Nematologica **16:** 107-12.

Williams, T.D. 1958. The potato root eelworm [**Heterodera rostochiensis** Woll.] studies on some relationships between plant and parasite in the family **Solanaceae.** PhD Thesis, Cambridge University, pp 152.