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Co-evolution of potato cyst nematodes and their hosts: implications for pathotypes and resistance[1]

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The co-evolutionary process is believed to have resulted, through interaction of wild populations of potato cyst nematodes and their hosts in geological time, in the resistant hosts now utilized in plant breeding programmes and in nematode populations with genes for resistance-breaking or virulence. It is argued that all such interactions between highly adapted, truly parasitic plant nematodes and their hosts are likely to be governed by gene-for-gene interrelationships. Practical implications of this hypothesis are that only pathotypes (resistance-breaking races) defined against identified resistance genes are scientifically sound and of practical value; that, in the case of potato cyst nematodes, other pathotypes (Ro2, Ro3, Ro5 and Pa2 and Pa3) should be abandoned; and that oligogenically based resistance to potato cyst nematodes, especially important in providing resistance to *Globodera pallida*, is non-durable. Working definitions of the terms 'pathotype', 'host-race' and 'virulence' are provided.

Introduction

Few plant-feeding nematodes are true parasites. The majority browse on root systems, deplete cell contents and move on to feed elsewhere. However, some have evolved a true parasitic habit, typified by endoparasitic forms with sedentary females which modify host tissues to form long-term feeding sites (Jones, 1981). Specialization on a single host species or on a few closely related hosts is common to many parasite taxa because the parasite–host relationship is so intimate (Thompson, 1982), and this is true of the cyst nematodes (Krall & Krall, 1978; Stone, 1979).

The major nematode pests of agriculture throughout the world are generally those forms showing greatest adaptations to the parasitic habit. Of these, cyst nematodes are perhaps the most extreme in their adaptation. The consequences of their adaptations, and of the co-evolutionary process which has influenced their hosts as well as the nematodes themselves, make cyst nematodes difficult to control. But these consequences provide unique opportunities for nematode management through utilization of crop resistance, specifically adapted pathogens and behaviour-disrupting chemicals, as well as the established methods of control through crop rotation and nematicides. Today the increasing attention being paid to real or alleged dangers to health and the environment posed by nematicides may change the spectrum of control measures for cyst nematodes available to us in future. Aldicarb, oxamyl, dichloropropene and ethylene dibromide have all been reported from ground water in the United States, albeit in some cases from sites where patterns of nematicide usage and soil hydraulic conditions are exceptional, and availability of dibromochloropropane has been severely restricted because of risk to process workers. The likelihood of new compounds being developed as nematicides is not high because nematode control forms only a small portion of world pesticide usage while development and registration costs are escalating. Against this background, interest in alternative control

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measures is increasing. Of these, although resistant cultivars are a long established measure, there has nevertheless been a steady increase in interest in recent years (Fig. 1).

Co-evolution between cyst nematodes and their hosts resulting in species complexes, host resistance and resistance-breaking pathotypes of the nematodes has been discussed extensively elsewhere (Stone & Hill, 1982; Stone, 1983; Turner *et al.*, 1983). This paper considers some of the practical implications for control of potato cyst nematodes.

Terminology

The number of studies of intraspecific variation in relation to nematode behaviour on hosts has increased steadily during the last decade as an adjunct to the control of plant parasitic nematodes by crop rotation and the use of resistant cultivars. At first, such forms were loosely described as races, biological races, biotypes etc. The nomenclature is becoming more organized, however, and the following definitions are now widely accepted. *Host races* are races of a nematode species distinguished by inherited ability or inability to successfully parasitize certain *species* of plant. *Pathotypes* are races of a nematode species distinguished by inherited ability or inability to reproduce on specified *lines* of a host-plant species that embody different genes for resistance to the nematode. Pathotypes may exist within a host race. In considering potato cyst nematodes we need be concerned only with pathotypes. *Virulence* is defined as the ability to break resistance and to reproduce on a resistant host; thus the term is used in a sense very like Vanderplank's (1978) but different from that of many plant pathologists. This simple nomenclature provides the definitions needed and is self-explanatory and free from jargon.

Some consequences of the co-evolutionary process

The evolution of wild plant populations with resistance genes to nematode parasitism, and wild nematode populations with virulence genes and pathotypes, is a process which occurred in geological time but which affects us by making available resistant germplasm for breeding programmes and pathotypes of the nematodes in agricultural field populations. The reality of this hypothesis and its practical consequences are readily demonstrated. Firstly, resistance is

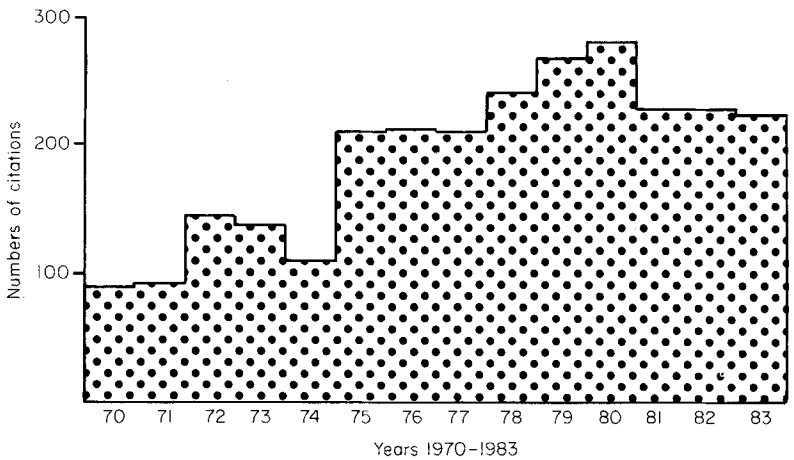

Fig. 1. Numbers of citations of host resistance to plant parasitic nematodes appearing in *Helminthological Abstracts Series B*, 1970–83.
Nombre de citations concernant la résistance des plantes-hôtes aux nématodes parasites relevées dans *Helminthological Abstracts Series B*, 1970–83.

largely confined to the highly specialized, truly parasitic plant nematodes: citations indexed under resistance in *Helminthological Abstracts, Series B* from 1970 to 1983 alluded to cyst nematodes in 33%, root-knot nematodes in 47%, other forms with sedentary females 3%, and specialized foliar parasites 9%. Secondly, pathotypes or host-specific races have been demonstrated only in species of the more highly adapted parasitic forms (Table 1). Thirdly, an examination of the origins of wild hosts with resistance to nematodes demonstrates that the distribution of resistance genes is largely confined to the natural distribution of the appropriate parasite: potato cyst nematodes provide an example, known sources of resistance originating chiefly from the Highlands of Peru, Bolivia and Argentina—the indigenous distribution of potato cyst nematode species.

The co-evolution hypothesis suggests, on the principle of the most parsimonious solution, that resistance in plant hosts and virulence in nematodes is most likely to be mediated by genes operating on a gene-for-gene basis as proposed by Flor (1956). Such genes have specific action, the effect of a particular resistance gene in the host being matched or overcome by the effect of a particular virulence gene in the parasite. Specific action implies that single or few genes will be involved in conferring resistance, i.e. major or oligogenes. For cases where the genetic nature of the resistance has been investigated, the great majority of examples of resistance to plant parasitic nematodes fall into this category (Table 2). For potato cyst nematodes up to 12 major resistance genes have been proposed with only three cases of oligogenic or polygenic resistance (Table 3).

F.G.W. Jones & D.M. Parrott (Parrott, 1981) have provided evidence of gene-for-gene relationships governing interactions between *Globodera rostochiensis* pathotype Ro1 and gene *H1* derived from *Solanum tuberosum* ssp. *andigena*, and *G. pallida* pathotype Pa1 and gene *H2* from *S. multidissectum*.

Table 1. Nematode species with proposed or demonstrated pathotypes (Sidhu & Webster, 1981).
Espèces de nématodes chez lesquelles l'existence de pathotypes a été proposée ou effectivement mise en évidence (Sidhu & Webster, 1981).

Ditylenchus dipsaci	*H. schachtii*
D. destructor	*Meloidogyne incognita*
D. radicicola	*M. arenaria*
Globodera rostochiensis	*Radopholus similis*
G. pallida	*Tylenchulus semipenetrans*
Heterodera avenae	*Pratylenchus penetrans*
H. glycines	

Table 2. Cases of types of resistance proposed or identified since 1950 (Sidhu & Webster, 1981).
Cas de types de résistance proposés ou identifiés depuis 1950 (Sidhu & Webster, 1981).

	Major genes/oligogenes	Polygenes
Heterodera avenae	13	1
H. glycines	2	
Potato cyst nematodes	12	3
Meloidogyne hapla	1	
M. incognita	15	3
M. javanica	1	
Ditylenchus dipsaci	3	2

Table 3. Some known or proposed genes conferring resistance to potato cyst nematodes.
Quelques gènes connus ou proposés conférant une résistance aux nématodes à kystes de la pomme de terre.

Source	Gene symbol	Gene type	References
S. tuberosum ssp. *andigena*			
CPC 1673, 1685, 1690, 2775	*H1*	major gene	Toxopeus & Huijsman, 1952, 1953; Cole & Howard, 1957; Howard *et al.*, 1970
CPC 2775, 2802, 2805	*H3*(?)	polygenes	Thomson, pers. comm.
S. vernei	—	polygenes	Various
	V1–V4 (?)	(+major genes)	Ross, 1969
S. multidissectum			
P.H. 1366	*H2*	major gene	Dunnett, 1961
	—	(+polygenes?)	(Dunnett, 1961)
S. kurtzianum	*A, B*	major genes	Huijsman, 1960
S. spegazinii	*Fa, Fb*	major genes	Ross, 1962
S. oplocense	*O1, O2*	major genes	Ross, 1969

The nature of resistance to *Globodera pallida*

Resistance to *G. pallida* derived from *S. vernei* is of major importance in potato breeding programmes. This resistance is unusual amongst cases of resistance to nematodes in that it is oligogenic or polygenic and has been held to be of the horizontal or field type (*sensu* Vanderplank; Vanderplank, 1978) and hence durable. Turner *et al.* (1983) tested this hypothesis by examining the durability of resistance in ex *vernei* hybrids to *G. pallida*. Selection of potato cyst-nematode populations on the resistant ex *vernei* hybrids which are used to differentiate *G. pallida* pathotypes (Kort *et al.*, 1978) resulted in an increase in virulence of these populations on the resistant clones on which they were selected. Despite doubts about interpretation of the data (Caligari & Phillips, 1984), subsequent results support the hypothesis that selection for virulence has occurred (Table 4). In some cases very considerable increases in virulence have taken place during selection through only six generations. The fitness of such highly selected populations in field conditions remains to be demonstrated. However, loss of durability of ex *vernei* resistance to *G. pallida* has been clearly demonstrated and the rapidity of selection suggests that this resistance is based upon a small number of genes participating in gene-for-gene relationships. This interpretation is in accordance with a view that both so-called horizontal and vertical resistance in plants involve gene-for-gene relationships as proposed by Parlevliet & Zadoks (1977). The frequently observed variable, or quantitative, resistance to *G. pallida* in ex *vernei* hybrids can be explained by assuming that the population level of resistance is determined by the number of individuals in a given nematode population with sufficient virulence genes in their genomes to overcome the complement of resistance genes in the genome of the plant on which the population is tested.

Unfortunately, resistance to *G. pallida* derived from a second source, *S. tuberosum* ssp. *andigena* CPC 2802 (Howard *et al.*, 1970), thought to be due to a major gene designated H3, is believed to be polygenic, with resistance being inherited quantitatively as in the case of that derived from *S. vernei* (Dale & Phillips, 1982). This resistance, too, may prove vulnerable to selection for virulence and it would seem wise for the breeding base for resistance to *G. pallida* to be widened to include other sources.

Table 4. Mean numbers of cysts produced by British *Globodera pallida* populations on resistant ex *vernei* potato clones in the first and sixth generations, expressed as percentages of numbers of cysts produced by the same populations on susceptible cv. Arran Banner in each generation.
Nombres moyens de kystes produits par les populations britanniques de *Globodera pallida* sur des clones de pommes de terre ex *vernei* résistants (1e et 6e générations), exprimés en pourcentages des nombres de kystes produits par les mêmes populations sur le cv. sensible Arran Banner.

Nematode population	Potato clone	Generation	
		1st	6th
Glarryford	62.33.3	17	68
Glarryford	65.346	52	97
Crowle CA	62.33.3	11	93
Crowle CA	65.346	15	72
Crowle CH	62.33.3	9	51
Little Ouse C	62.33.3	9	62
Wainfleet	62.33.3	3	36
Wainfleet	65.346	29	59
Frenswegen	62.33.3	66	90
Frenswegen	65.346	22	90

Pathotypes of potato cyst nematodes

The current 'European' scheme for pathotyping potato cyst nematodes (Kort *et al.*, 1978) was proposed in order to provide a common, international nomenclature for use by nematologists, plant breeders and advisory workers. It embodied the disadvantages as well as the advantages of the separate Dutch, German and British schemes then in operation.

Proper definition of pathotypes requires that each one is defined against identified resistance genes. Unfortunately, the current potato cyst-nematode scheme is scientifically unsound because only three of the differential clones (*S. tuberosum* ssp. *andigena* CPC 1673 hybrid, *S. kurtzianum* hybrid 60.21.19 and *S. multidissectum* hybrid P55/7) have identified resistance genes. Thus pathotypes Pa2 and Pa3 of *G. pallida* and Ro2, Ro3 and Ro5 of *G. rostochiensis* are defined by performance on differential clones of unknown genetic constitution. If, as is proposed, there is a continuum of a small number of resistance genes available from each of the sources from which these hybrids were bred, and there is also a continuum of virulence genes available in nematode populations and acting against those resistance genes, then departures from the clear-cut positive or negative response required in a pathotype test may occur in any populations other than those reference populations on which the national schemes were based. Even with the reference populations results may sometimes be equivocal, as Table III in Kort *et al.* (1978) demonstrates. Consequently, we may assume that pathotypes Pa2, Pa3, Ro2, Ro3 and Ro5 are artefacts of the test procedure in which reference nematode populations were found to behave in a differentially distinctive way on particular resistant potato hybrids.

The variable nature of virulence in *G. pallida* populations, demonstrated by selection experiments, has indicated the unsatisfactory nature of the distinction between pathotypes Pa2 and Pa3. A survey of British *G. pallida* field populations supports this contention. Of 100 *G. pallida* field populations tested by members of the Agricultural and Development Advisory Service Potato Nematodes Working Party, 33 were impossible to attribute to either Pa2 or Pa3 alone while in many of the cases where a single attribution was made the response was equivocal. Consequently, it is proposed that pathotypes Pa2 and Pa3, being artefacts of no useful application, be abandoned. Although field experience with pathotypes of *G. rostochiensis* other

than Ro1 is lacking in the United Kingdom, it seems likely that experience elsewhere may result in similar conclusions in relation to pathotypes Ro2, Ro3 and Ro5.

Conclusions

The co-evolution hypothesis and the balance of evidence suggest that resistance to plant-parasitic nematodes is likely to operate on a gene-for-gene basis and be non-durable, whether mediated by major genes or oligogenes.

Selection for virulence against such resistance may occur rather readily and has been demonstrated experimentally in the case of resistance to *G. pallida* derived from *S. vernei*. More widely based resistance-breeding programmes are required.

Pathotypes of potato cyst nematodes defined against differential clones with unknown resistance genes are likely to be artefacts and should be abandoned.

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Co-évolution des nématodes à kystes de la pomme de terre et de leurs hôtes: implications pour les mesures de contrôle

Par l'interaction des populations sauvages de nématodes à kystes de la pomme de terre et de leurs hôtes au cours des temps géologiques le processus de co-évolution aurait donné pour résultat les hôtes résistants maintenant utilisés dans les programmes de sélection ainsi que les populations de nématodes possédant des gènes de virulence. Toutes ces interactions entre les nématodes hautement adaptés au parasitisme et leurs hôtes seraient probablement régies par des relations gène à gène. De cette hypothèse découlent les conséquences pratiques suivantes: 1) seuls les pathotypes définis contre les gènes de résistance identifiés ont une valeur scientifique solide; 2) dans le cas des nématodes à kystes de la pomme de terre, les pathotypes Ro2, Ro3, Ro5 et Pa2, Pa3 devraient être abandonnés; 3) la résistance oligogénique aux nématodes à kystes de la pomme de terre, surtout importante contre *Globodera pallida*, n'est pas durable. L'auteur donne les définitions pratiques des termes pathotype, race-hôte et virulence.

Соэволюция цистообразующих нематод картофеля и их хозяев: выводы для защитных мероприятий

Предполагается, что процесс совместной эволюции за счет взаимодействия диких популяций цистообразующих нематод картофеля и их хозяев в геологический период привел к появлению устойчивых растений-хозяев, которые в настоящее время используются в селекционных программах, а также в появлении нематодных популяций, обладающих вирулентными генами, способными к разрушению этой устойчивости. Автор доказывает, что все эти взаимодействия между хорошо адаптировавшимися чисто паразитическими нематодами и их хозяевами, по всей вероятности, определяются связью «ген против гена». Практические выводы из этой гипотезы сводятся к тому, что только те патотипы (раса, разрушающая устойчивость), которые были определены как действующие против определенных генов устойчивости, являются научно обоснованными и имеют определенное практическое значение; в случае цистообразующих нематод на картофеле от остальных патотипов (Ro2, Ro3, Ro5 и Pa2 и Pa3) следует практически отказаться; а также, что олигогенная устойчивость к цистообразующим нематодам картофеля, имеющая особо важное значение для появления устойчивости по отношению к Globodera pallida, не обладает длительностью. Автором даются также практические определения таких терминов, как патотип, раса хозяина и вирулентность.

References

CALIGARI, P.D.S. & PHILLIPS, M.S. (1984) A re-examination of apparent selection in *Globodera pallida* on *Solanum vernei* hybrids. *Euphytica* **33,** 583–586.

COLE, C.S. & HOWARD H.W. (1957) The genetics of resistance to potato-root eelworm of *Solanum tuberosum* subsp. *andigenum*, clone C.P.C. 1960. *Euphytica* **6,** 242–246.

DALE, M.F.B. & PHILLIPS, M.S. (1982) An investigation of resistance to the white potato cyst-nematode. *Journal of Agricultural Science, Cambridge* **99,** 325–328.

DUNNETT, J.M. (1961) Inheritance of resistance to potato-root eelworm in breeding lines stemming from *Solanum multidissectum* Hawkes. *Report of the Scottish Plant Breeding Station for 1961*, 39–46.

FLOR, H.H. (1956) The complementary genic system in flax and flax rust. *Advances in Genetics* **8,** 29–54.

HOWARD, H.W., COLE, C.S. & FULLER J.M. (1970) Further sources of resistance to *Heterodera rostochiensis* Woll. in the Andigena potatoes. *Euphytica* **19,** 210–216.

HUIJSMAN, C.A. (1960) Some data on the resistance against the potato-root-eelworm (*Heterodera rostochiensis* W.) in *Solanum kurtzianum*. *Euphytica* **9,** 185–190.

JONES, M.G.K. (1981) The development and function of plant cells modified by endoparasitic nematodes. In *Plant Parasitic Nematodes* (eds Zuckerman, B.M. & Rohde, R.A.), vol. III, pp. 255–279. Academic Press, New York.

KORT, J., ROSS, H., RUMPENHORST, H.J. & STONE, A.R. (1978) An international scheme for identifying and classifying pathotypes of potato cyst-nematodes *Globodera rostochiensis* and *G. pallida*. *Nematologica* **23**(1977), 333–339.

KRALL, E. & KRALL, H. (1978) [The revision of plant nematodes of the family Heteroderidae (Nematoda: Tylenchida) by using a comparative ecological method of studying their phylogeny]. In *Printsipy i Metody Izucheniya Vzaimootnoshenii mezhdu Paraziticheskimi Nematodami i Rasteniyami*, pp. 39–56. Akademiya Nauk Estonskoi SSR, Tartu.

PARLEVLIET, J.E. & ZADOKS, J.C. (1977) The integrated concept of disease resistance; a new view including horizontal and vertical resistance in plants. *Euphytica* **26,** 5–21.

PARROTT, D.M. (1981) Evidence for gene-for-gene relationships between resistance gene H1 from *Solanum tuberosum* ssp. *andigena* and a gene in *Globodera rostochiensis* and between H2 from *S. multidissectum* and a gene in *G. pallida*. *Nematologica* **27,** 372–384.

ROSS, H. (1962) [Inheritance of resistance to potato cyst nematodes in crosses between *Solanum famatinae* and either *S. tuberosum* or *S. chacoense*]. *Züchter* **32,** 74–80.

ROSS, H. (1969) [Breeding potato cultivars with resistance to *Heterodera rostochiensis*]. *Mitteilungen aus der Biologischen Bundesanstalt für Land- und Forstwirtschaft, Berlin-Dahlem* **136,** 59–64.

SIDHU, G.S. & WEBSTER, J.M. (1981) The genetics of plant-nematode parasitic systems. *Botanical Review* **47,** 387–419.

STONE, A.R. (1979) Co-evolution of nematodes and plants. In *Parasites as Plant Taxonomists* (ed. Hedberg, I.), vol. XXII (4), pp. 46–61. Symbolae Botanicae Upsaliensis, Uppsala.

STONE, A.R. (1983) Three approaches to the status of a species complex, with a revision of some species of *Globodera* (Nematoda: Heteroderidae). In *Concepts in Nematode Systematics* (eds Stone, A.R., Platt, H.M. & Khalil, L.F.), pp. 221–233. Academic Press, London.

STONE, A.R. & HILL, A.J. (1982) Some problems posed by the *Heterodera avenae* complex. *Bulletin OEPP/EPPO Bulletin* **12,** 317–320.

THOMPSON, J.N. (1982) *Interaction and Coevolution*, 179 pp. John Wiley & Sons, New York.

TOXOPEUS, H.J. & HUIJSMAN, C.A. (1952) Genotypical background of resistance to *Heterodera rostochiensis* in *Solanum tuberosum* ssp. *andigenum*. *Nature* **170,** 1016–1017.

TOXOPEUS, H.J. & HUIJSMAN, C.A. (1953) Breeding for resistance to potato root eelworm, I. Preliminary data concerning the inheritance and nature of resistance. *Euphytica* **2,** 180–186.

TURNER, S.J., STONE, A.R. & PERRY, R.N. (1983) Selection of potato cyst-nematodes on resistant *Solanum vernei* hybrids. *Euphytica* **32,** 911–917.

VANDERPLANK, J.E. (1978) *Genetic and Molecular Basis of Plant Pathogenesis*, 167 pp. Springer-Verlag, Berlin.