Nature and Inheritance of Nematode Resistance in Cereals[1]

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Abstract: Resistance to a number of nematodes is present in varieties of temperate and tropical cereals. The occurrence, nature, and inheritance of varietal resistance in cereals is reviewed. Evaluation of the practical significance of nematode resistance in a particular host-nematode combination is discussed in relation to host efficiency, host sensitivity, genetic control of resistance, and presence of virulence in the nematode population.

This paper was originally prepared for the 2nd International Congress of Plant Pathology held in Minneapolis, Minnesota, U.S.A. on 5-12 September 1973. It was presented in a symposium on Nematode Resistance in Plants together with papers dealing with aspects of plant breeding (43), with characteristics of nematode reproduction and genetics (74), and with host plant biochemistry (30).

Other authors have reviewed nematode resistance (12, 40, 63, 64), and the nematode pests of cereals (44, 46, 49). There is no a priori reason to suppose that varietal resistance of cereals differs fundamentally, if at all, from that of other plants. However, because of the dependence of the world's peoples on various species of Gramineae cultivated as cereals, it is appropriate to consider separately the resistance of these crops to nematodes. There is continuing pressure towards cereal monoculture, or shortening of rotations, in many parts of the world. In spite of their importance, cereal crops will not generally support the cost of chemical control of nematode infestations. Development of resistant varieties is, therefore, a valuable means of safeguarding cereal production from losses due to nematode pests. Preliminary estimates for the United States of America put these losses at 5-6% (70), equivalent to the loss of over 8 million acres of cereals annually. The aims of this paper are to summarize information on the nature and inheritance of nematode resistance in cereals, and to illustrate some concepts underlying the successful implementation of plant breeding to control nematode pests.

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OCCURRENCE OF NEMATODE RESISTANCE

Rice (*Oryza sativa* L.): 1) *Aphelenchoides besseyi* Christie.—Varietal differences in reaction to the rice white-tip nematode have been reported from Japan (35, 60) and the U.S.A. (9), and summarized by Ichinohe (44, and Fortuner (28) who also mentioned resistant varieties from Italy and India. Differences in reaction exhibited by different varieties have been categorized as (a) susceptible, and showing white-tip symptoms, (b) susceptible but showing no symptoms, and (c) immune, with no indication of nematode attack (44). These different reactions, coupled with the fact that expression of typical white-tip symptoms is affected by environment (17, 44), make an accurate summary of varietal reactions difficult. In the U.S.A. many varieties have been considered resistant since they show no white-tip symptoms and their yields are relatively little affected by the nematode (9). In Japan, varieties have been rated as very resistant, intermediate-resistant, and susceptible. The variety 'Asahi 1' is classed as 'symptomless' by Nishizawa (60), but is susceptible according to Goto and Fukatsu (35) who recorded 'percentage diseased culms'. The latter authors studied the nature of resistance and found that attractiveness of plants to *A. besseyi* in laboratory experiments was highly correlated with their field susceptibility. On resistant varieties, reproduction of the nematode was reduced and adult females were smaller than in susceptible varieties. Tolerance, in varieties which can have nematodes in the panicle without symptom expression, was not correlated with decreased attraction to, or reproduction by, the nematode. In spite of difficulties of classifying resistance, useful resistance to *A. besseyi* exists in a number of local varieties of rice. Information on the inheritance of resistance is lacking, but it is said to be genetically controlled and carried by the variety 'Asa - Hi' (not 'Asahi') (60).

2) *Ditylenchus angustus* (Butler) Filipjev.—Differences in response of rice varieties to this nematode have been reported from Thailand. Laboratory inoculations of rice coleoptiles showed that 43% of plants of 'Khao-tah-oo' were infected, compared with 81% of 'Khao-tah-haeng 17'. Another test suggested that four *japonica* varieties tested were more resistant than three *indica* varieties. However, differences in degree of infection must be ascribed partly to the slower germination of the *japonica* varieties, which were, therefore, at a less suitable stage for nematode invasion than the *indica* varieties (41).

3) *Meloidogyne graminicola* (Golden and Birchfield).—*Indica* variety 'TKM 6' and five other varieties were shown to be highly resistant to *M. graminicola* in India (66). However, 80 *indica* and *japonica* varieties, including 'TKM 6', were all galled to various degrees in tests in Laos (53).

Corn (*Zea mays* L.): 1) *Ditylenchus dipsaci* (Kühn) Filipjev.—Damage caused to corn by this nematode has been reported from Europe (19, 39, 51) and differences occur between varieties in degree of infection and yield loss. In field tests in Hessen, Germany, only 13% of 'Inrakorn' plants were infected, compared with 52-72% for six other cultivars which yielded 18-31% less than 'Inrakorn' (51).

2) *Tylenchorhynchus claytoni* Steiner.—Nelson (55) reported that, of 30 inbred lines and 11 single cross corn hybrids tested against this nematode, nine of the inbreds suffered little damage. Further tests showed that numbers of *T. claytoni* declined under these inbreds and that there were more secondary roots and less browning of primary roots than in other inbreds and crosses. Progenies of two crosses of different resistant and susceptible inbred parents were susceptible, suggesting that resistance may be recessive.

3) *Meloidogyne* spp.—Resistance of unnamed inbred lines of corn to *Meloidogyne incognita* (Kofoid and White) Chitwood was reported by Nelson (56). Criteria were: number and size of galls, and nematode reproduction and its effects on root weight. Resistant lines had few, small galls and limited nematode reproduction. Certain heavily galled, susceptible lines showed no loss of root weight compared with uninoculated controls, which Nelson suggested may indicate tolerance. Crosses of resistant and susceptible inbreds always produced progeny at least as susceptible as the susceptible parent. Histologically a poor host for *M. incognita,* 'Pioneer 309B' often showed collapsed giant cells, with associated larvae apparently dead. Few females matured, although some eggs were produced. In contrast, a good host, 'Coker 911' had granular multinucleate giant cells. Fewer larvae invaded the poor host (11).

Varietal differences in response of corn to *M. arenaria* (Neal) Chitwood, *M. incognita,* and *M. javanica*(Treub) Chitwood have been reported (10). In general, 'Coker' hybrids supported greatest nematode egg production, whilst 'Pioneer' hybrids had moderate numbers of eggs and 'McNair' hybrids relatively few. There were differences in rates of egg production between nematode species and also between different field populations of both *M. arenaria* and *M. incognita.* Interactions between nematode populations and corn cultivars were also encountered. In some cases the resistant reaction appeared to be associated with root damage and consequently limited plant growth, at least temporarily. Differences in the response of corn to *M. incognita* and *M. javanica,* as measured by degree of galling resulting from controlled inoculations, have also been observed in Lebanon (72). Both nematode species were unable to stimulate gall formation on two varieties, 'Carmel Cross' and 'Span Cross'. Three varieties on which galls were produced were moderately resistant to *M. incognita,* but susceptible to *M. javanica.* In South Africa, resistance to *M. javanica* has been found in several varieties which remained almost free of galls and permitted no nematode reproduction (7, 8).

Barley (*Hordeum* spp.): 1) *Heterodera avenae* Wollenweber.—Resistance to *H. avenae* in barley was first reported in Sweden in 1920 (59). Little further work was done until the 1950's when, in several European countries, interest in resistance was rekindled by the great increase in barley acreages. Roots of resistant varieties were invaded by *H. avenae* larvae, but subsequent development to mature females was prevented (3). Histological investigations of the host-nematode relationship in the resistant variety, 'Sabarlis', showed that invading larvae stimulated the initiation of giant-cell feeding sites, but subsequently the cytoplasm of these

cells became sparse and vacuolated (Cook, *unpublished*). Development of males was not prevented by the resistant reaction (23). Comparison of resistant and susceptible isogenic barley lines established a 20% grain yield advantage of the resistant barley (23). Also, resistant barley reduced the population of *H. avenae* in the soil (1, 23). Since in much of Europe barley is grown continuously, these attributes permit farmers to control this nematode without departing from preferred cropping practices.

Populations of *H. avenae* which have different virulence spectra as measured by differential barley varieties have been identified in Europe (2, 24, 50, 52, 71, 76), Australia (15), and India (31). Information on these races, or pathotypes, has been summarized (22). Currently, nematologists and plant breeders from eleven countries are cooperating, using standardized methods and differential varieties of all temperate-zone cereals, to characterize these pathotypes and to identify useful sources of resistance. Preliminary conclusions, based on results obtained by this '*Heterodera avenae* group', have been reported (58). The single dominant gene Ha_2, from barley No. 191, confers resistance to the two pathotypes common in Britain and Denmark and in some other European countries. Pathotypes virulent on varieties incorporating this gene are known, but additional resistance sources are available except possibly in Rajasthan, India, and in Sweden.

Inheritance of resistance and interrelationships of genes for resistance have been the subject of study (5, 6, 25, 42). Single dominant genes have been found to control resistance in most of the genotypes which were investigated. Genes at a minimum of three loci have been identified, with close linkage of two of the loci on the long arm of chromosome 2 (25). In the variety Harlan 43, which has partial resistance to British pathotypes 1 and 2, several genes may be involved (Cook, *unpublished*). Attempts to relate plant resistance genes to virulence genes in the nematode are perhaps premature. Genetics of virulence has been studied (4), but experiments are difficult to carry out. There is evidence that the genic background of a variety may affect expression of resistance genes.

Oat (*Avena* spp.): 1) *Ditylenchus dipsaci.*—Oat varieties with resistance to *D. dipsaci* have been grown in Britain for many years and the nature and inheritance of resistance investigated by a number of workers (13, 18, 32, 36, 38, 47). In varieties with resistance of the type present in *A. sativa* L., 'Grey Winter', the nematode fails to elicit the compatible (susceptible) response, although initial invasion is the same in both resistant and susceptible oats. In the latter, cells of the stem base enlarge and separate from each other, due to breakdown of middle lamellae; cytoplasm is withdrawn from the cell walls, which collapse to create a cavity around the nematode. In resistant plants there

TABLE 1. Nature of resistance to *Ditylenchus dipsaci* in oats (*Avena* spp.).[a]

Reaction to *D. dipsaci*	Genotype	Host reaction[b]		Nematode reproduction
		Swelling of stem base	Stunting of seedling	
Susceptible	many varieties of *A. sativa* and *A. byzantina*	+++	+++	+++
Resistant	'Early Miller' 'Record' *A. byzantina* (unnamed types)	++	+	+
Resistant	*A. sativa* cv. 'Grey Winter' and other 'land races' Derived varieties	−	+	+
Resistant	*A. ludoviciana*, Cc4346 Cc4347	−	−	−

[a]Sources cited in text.
[b]Relative scales from none (−) to high (+++).

is little or no cell hypertrophy and separation, but withdrawal of cytoplasm occurs and cell walls collapse. This leads to the isolation of invading nematodes in local lesions. Subsequent growth and reproduction of the nematode is much slower than in susceptible plants, so that large nematode populations do not develop in resistant plants.

The morphological responses of resistant and susceptible plants are also different. Either may be killed by very high initial invasion, but the usual susceptible response is the 'tulip root' symptom in which cellular hypertrophy results in a much-swollen stem base. Resistant varieties do not show this symptom although they may produce an abnormal number of tillers. In Britain, autumn-sown oats are more affected by *D. dipsaci* because they are growing only slowly during periods of relatively low temperatures when the nematode is capable of activity. Spring-sown oats may escape damage through rapid early season growth before high populations of *D. dipsaci* develop in the stems.

Resistant varieties of oats have been developed also in Belgium (20); resistance of the type described above has been used. In California, U.S.A., resistant varieties have been selected on the basis of total yield produced in the presence of *D. dipsaci* (62).

Inheritance of resistance to *D. dipsaci* in a number of oat genotypes has been studied (18, 32, 38). Resistance from 'Grey Winter', which has been used in most plant breeding programs, either directly or from derived varieties, is dominant and inherited as a single gene pair (38). Other resistant varieties probably derive from European 'land races', morphologically and physiologically similar to 'Grey Winter', and which may carry the same factor for resistance. In other hexaploid species of oats, *A. ludoviciana* Dur. and *A. byzantina* C. Koch, forms resistant to *D. dipsaci* are found. The resistance of *A. ludoviciana* types is well defined, but that of *A. byzantina* less so, since in these latter genotypes resistance is associated with rapid differentiation and stem elongation. Therefore, infection or damage may be avoided, and a certain amount of nematode reproduction tolerated without severe stunting. In Scotland, breeding resistant varieties has involved studying several types of resistance (18, 36, 37). Table 1 summarizes the nature of resistance in these different genotypes. Results of inheritance studies support the conclusion that the 'Grey Winter' type of resistance is inherited as a single factor pair, responsible for the inhibition of nematode reproduction. In 'Early Miller' the 'tolerance' type of reaction is governed by a single gene pair, independent of the inhibitant factor, but subject to modification according to the genic background. Resistance of *A. ludoviciana* is said to be controlled by two independent genes and appears to combine the effects of the other two types of resistance. Crosses between 'Early Miller' and 'Milford', whose resistance derives from 'Grey Winter', have produced resistant progeny similar in reaction to *A. ludoviciana* (36).

2) *Heterodera avenae.*—Resistance to cereal cyst nematode in oats is expressed by

TABLE 2. Genetics of resistance of oats, *Avena* spp., to *Heterodera avenae*.

	Genotype	Mode of inheritance	Reference
A. sterilis,	I. 376 (Cc4658)	three dominant genes (or) two complementary dominant genes	(5) (26)
A. byzantina,	P.I. 175021 (Cc4701)	one dominant gene	(26)
A. sativa,	C.I. 3444 P.I. 175022 'Silva'	one dominant gene at the same locus	(5)
	'Nelson'[a] 06334Cn[b]	one dominant gene at the same or closely linked loci	(Cook, *unpubl.*)
	'Mortgage Lifter'	two independent recessive genes	(26)

[a]Resistance derived from *A. sativa,* C.I. 3444.
[b]Resistance derived from *A. sterilis,* I. 376.

failure of females to mature. The effect of resistant oat varieties in reducing *H. avenae* populations is, therefore, similar to that of resistant barleys. However, it appears that preventing maturation of females does not confer on resistant oats the same yield advantage as that demonstrated for resistant barleys. Histological examination of the reaction of these resistant varieties showed that invasion of roots was normally followed by hyperplasia and necrosis, and not by initiation of giant cells (Cook, *unpublished*). The adverse effects of this hypersensitive resistance mechanism may override any yield advantage from preventing female feeding and development.

Resistance, as measured by absence or reduction of cyst formation, is present in varieties of a number of oat species. The diploid and tetraploid species have the most complete resistance (5), but resistance more easily exploited in plant breeding is available in hexaploid species. In general, the differences in virulence of *H. avenae* populations, as identified on barley differentials, are not recognized on oats. Thus the resistance of *A. sterilis* L., I. 376 (Cc 4658) is effective against all European populations so far encountered, and also against Australian populations (15, 16, 58). In India, some *A. sterilis* genotypes are susceptible, but I. 376 was not tested (31). However, differences in virulence of *H. avenae* on oats do exist since certain European populations have been identified which are unable to reproduce on oats (50, 71). The suggestion that the resistance of *A. sativa* (C.I. 3444) is less effective against Netherlands pathotype C than against a similar pathotype in Denmark (50) has not been proved. Further differences in virulence of *H. avenae* on oats might exist or develop in response to selection.

Genetic investigations of a number of oat genotypes indicate that several genes for resistance may be available (5, 26) (Table 2). The interrelationships of these resistance genes are not fully understood, since the hexaploid constitution of cultivated oats makes investigation and interpretation difficult. However, it is of interest to relate the available genetic information to the effectiveness of resistance. In the *A. sativa*

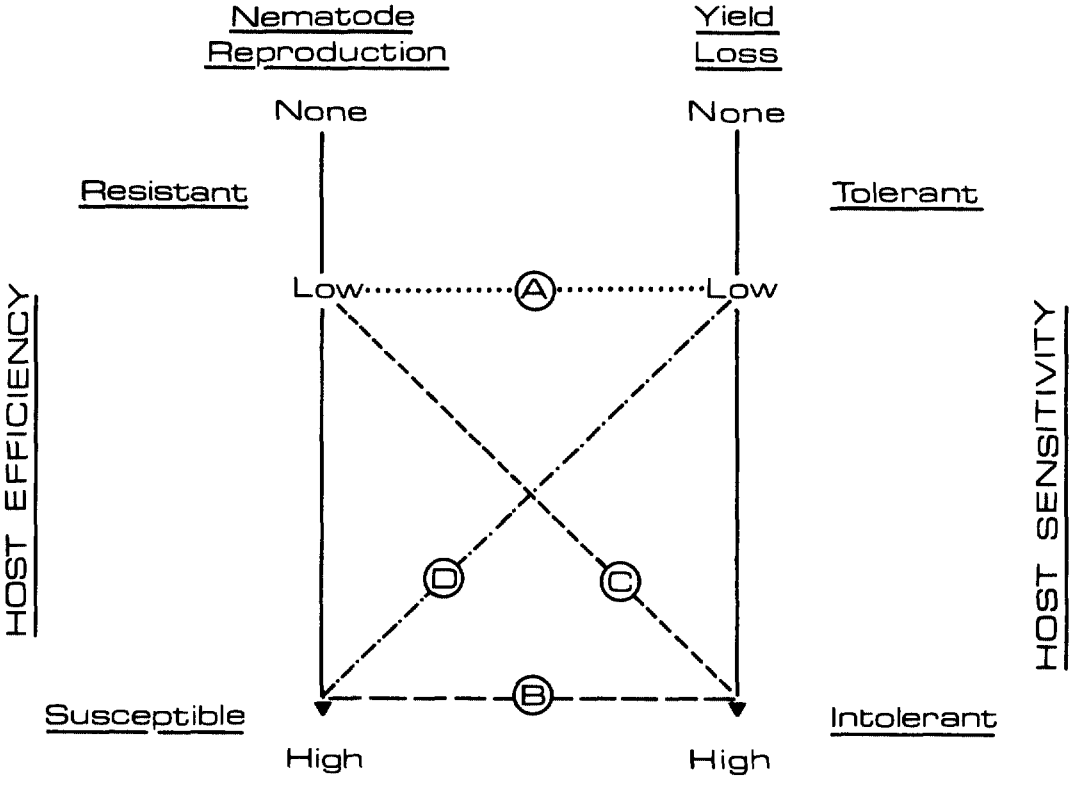

FIG. 1. Concepts of host efficiency and host sensitivity in plant-nematode relationships (See text for explanation).

genotypes C.I. 3444, P.I. 175022, and Silva, the same locus is involved, although in tests against British populations of *H. avenae* the resistance of Silva is less effective in suppressing female maturation than that of the other two genotypes. This indicates that different alleles may be present at a single locus or that the genetic background affects expression of resistance. Conflicting reports on the numbers of resistance genes present in *A. sterilis,* I. 376, suggest that there may be differences in the number of genes for virulence in British and Danish populations of *H. avenae.* However, difficulties in classifying plants with low numbers of cysts as resistant or susceptible in segregating generations also contribute to this conflict. In segregating populations from crosses between 'Nelson' and the Welsh Plant Breeding Station resistant oat 06334Cn, no susceptible plants have been found. This suggests that the same or closely linked loci may be involved.

3) *Meloidogyne* spp.—Nine of 18 oat varieties were classified as resistant to *M. incognita acrita* Chitwood in the state of Delaware in the U.S.A. (27).

Oats also exhibit varietal differences to *M. naasi* Franklin. In Illinois, U.S.A., 11 of 47 varieties tested were not galled by this nematode, and 31 were classified as susceptible on the basis of degree of galling and nematode reproduction. The other varieties were only slightly susceptible (68, 73). In Europe, oats have been classed as resistant to *M. naasi* (29, 33), although resistance was lost in tests carried out at 25 C (34). Welsh populations of *M. naasi* reproduced on 11 oat varieties in a glasshouse test, and there were varietial differences in numbers of females produced (21).

Rye (*Secale cereale* L.): 1) *Ditylenchus dipsaci.*—Resistance to *D. dipsaci* has been incorporated into winter rye in the Netherlands. The source of resistance was a local 'land race' variety, 'Ottersumse' (49).

2) *Heterodera avenae.*—Most rye varieties are susceptible to *H. avenae,* although less so than other cereals. In Australia, the local variety 'South Australian' is resistant (16), and recent tests suggest it may also be resistant to British populations.

Wheat (*Triticum* spp.): 1) *Heterodera avenae.*—Resistance of the wheat variety, 'Loros', is manifested by failure of the nematode to produce cysts and it is believed to be controlled by a single dominant gene (57). Other resistant wheat varieties give less complete suppression of cyst formation; e.g., 'Red Egyptian', and 'Iskamish Katagan'. In Haryana, India, the varieties 'Kalyan Sona' and 'PV18', although susceptible, supported fewer cysts than other varieties (54). In Rajasthan, India, three of 193 selections of *T. aestivum* L. were identified tentatively as completely resistant (69). These were 'P.I. 183868' from Turkey, and 'P.I. 185205' and 'P.I. 185207' from Portugal. Other selections were only slightly susceptible and could provide useful sources of resistance. Some varieties of *T. durum* Desf. from Australia appear to have resistance to European populations.

2) *Anguina tritici* (Steinbuch) Chitwood.—In Rumania, three winter wheats and three spring wheats have been reported to show a measure of resistance to *A. tritici* (65). In India, three varieties, including 'Sonora 64' are said to show tolerance to 'tundu' disease, caused by *A. tritici* and *Corynebacterium tritici* (48).

DISCUSSION

In order to evaluate the significance of varietal resistance in a particular host-nematode combination, several aspects must be considered. Agronomically, a resistant variety must compare favorably with other available means of control in terms of its yield on infested land and its effects on nematode populations. Incorporation of resistance into new varieties depends upon an identifiable host response which must be transferable through hybridization (i.e., it must be hereditable). Thirdly, the range of effectiveness of the resistance against different populations of the nematode must permit use of the resistant variety throughout the area of its agronomic adaptation.

Use of resistant varieties should maximize and stabilize yields through their effects on nematode population dynamics. Crop yield depends upon initial nematode density and rate of reproduction, and the inherent sensitivity of the host. Introduction of resistance into a crop may affect all of these factors. The value of a resistant variety, therefore, depends upon the interrelationships of the mechanism of resistance with the biology of the host and the

nematode. Some of these different interrelationships are illustrated by comparison of the effects on yield of resistance to *D. dipsaci* in oat and to *H. avenae* in both oat and barley. Oats resistant to *D. dipsaci* suffer less damage than susceptible varieties. The resistant reaction is less harmful than the susceptible one, but, more importantly, the resulting suppression of nematode multiplication reduces damage. In the examples of resistance to *H. avenae,* the hypersensitive reaction of oat contrasts with the initial compatibility of the reaction in barley. The resistance mechanism in oat apparently damages the plant as much as the susceptible response. Since *H. avenae* produces only a single generation per season the benefits of suppression of multiplication may not be seen unless a second cereal crop is grown after the resistant cereal. On the other hand, barley varieties show yield benefits from their resistance in the first crop on *H. avenae*-infested fields.

The range of varietal resistance summarized in the survey, and the different methods of selection used to identify resistance, indicate the need for a definition of terms. Figure 1 attempts to provide a diagrammatic characterisation of host-nematode relationships in terms of the concepts of resistance and tolerance. Variables in this diagram are: *host efficiency* as measured by degree of nematode reproduction, and *host sensitivity* as measured by yield loss. The definition of resistance in terms of reproduction of the nematode is one commonly adopted by plant nematologists (64). In the context of plant breeding, the level of host efficiency defined as 'resistant' will depend upon that which is available and is not, therefore, the same for all host-nematode combinations. Tolerance, 'equivalent severity of disease without equivalent damage or loss' (67), seems a particularly useful concept for plant nematology. Most reports of tolerance to plant disease relate to host-pathogen relationships in which the plant part harvested is distinct from the part diseased (67). With the exception of *Anguina tritici* and *Aphelenchoides besseyi,* the nematodes considered in this review do not attack the harvested part (grain) of cereals. Problems of distinguishing effects of tolerance from those attributable to different levels of resistance may be overcome by relating the concepts of 'host efficiency' and 'host sensitivity'. In Fig. 1, hatched lines A, B, and C represent three such relationships for *Heterodera avenae* and cereals. Line A shows the relationship for a barley variety possessing a gene preventing female nematode development, thus supporting little reproduction and suffering little damage. Line B represents the relationship for a susceptible variety of spring sown oat permitting nematode reproduction and suffering damage, and line C that for a spring oat with a gene suppressing nematode reproduction, but suffering damage. Line D indicates the relationship of host sensitivity to nematode density since a tolerant host which permitted high reproduction, would eventually so increase the nematode population as to become sensitive or intolerant. Fig. 1 requires a third dimension to represent factors which may alter the relationship between efficiency and sensitivity; e.g., initial nematode population and environmental conditions. Nevertheless, the diagram provides a model for identifying and comparing varietal differences in response to nematodes in a way relevant to selection in plant breeding programs.

In order to be of value to a breeding program, varietal resistance must be hereditable unless the resistance source is suitable for commercial exploitation without hybridization. Few genetic studies on nematode resistance in cereals have been made, and detailed investigations on genetic interrelationships are limited to oat and barley. The full potential value of many of the cited examples cannot, therefore, be assessed. Relatively simple genetic control of resistance does occur, but it may be that this will be broken by the emergence of new, more virulent pathotypes.

Effectiveness of a resistant variety against the virulence spectrum of a nematode is the third consideration in assessing the usefulness of varietal resistance. Emergence of new virulent populations or pathotypes can take place by selection of pre-existing variation in a nematode population or by mutation followed by selection in an initially avirulent population. By analogy to other plant diseases, such changes in virulence are to be expected (75); and indeed, most nematode species do show variation in virulence (61). Information on the interrelationships of available resistance genes and detectable virulence genes in nematode populations

throughout the species range may enable local changes in virulence to be anticipated. Since most plant parasitic nematodes are soil-borne, the spread of new virulence genes should be relatively slow compared to that of some airborne pathogens, and, therefore, resistant varieties should remain effective for relatively long periods.

Varietal resistance under complex genetic control undoubtedly exists, but has been little studied. It is possible that poor hosts of root-knot and cyst nematodes have resistance of this type (64). The resistance of oat to *Meloidogyne naasi,* and less-than-complete resistance of some cereals to *Heterodera avenae,* may prove to be under polygenic control. This is more difficult to incorporate into agronomically adapted varieties. The phenomenon of tolerance may also be under complex genetic control.

Incorporation of genes for resistance to more than one nematode species, or more than one pathotype within a species, is possible. Oats resistant to both *Ditylenchus dipsaci* and *Heterodera avenae* are being developed at the Welsh Plant Breeding Station. Resistance to two pathotypes of *H. avenae* has been incorporated into barley varieties at several European plant breeding centers. Corn hybrids resistant to more than one species of *Meloidogyne* (10) also indicate that there are not necessarily barriers to multiple resistance. However, the existence of allelism in barley, with a single locus having different genes for resistance to different pathotypes in different host genotypes, suggests that difficulties may be encountered.

Resistance to nematodes which either feed ectoparasitically or are migratory endoparasites, is thought less likely to be found than resistance to sedentary endoparasites with complex host relationships (42, 64). There is, however, some evidence for resistance to these apparently less-specific feeders. Resistance of corn to *Tylenchorhynchus claytoni* (55) and differences in numbers of *Pratylenchus minyus* Sher and Allen in roots of barley varieties (42), indicate that varietal differences do exist. Of other members of the Gramineae, pearl millet, cv. 'Tiflate', has resistance to *Pratylenchus* spp. and *Belonolaimus longicaudatus* Rau (45). Forage grasses *Chloris* sp., *Cynodon dactylon* (L.) Pers., *Digitaria* sp. and *Paspalum notatum* Flügge, also exhibit varietal differences in host efficiency and sensitivity to *B. longicaudatus* (14). These examples suggest that it might be profitable to screen cereals for resistance to ectoparasitic and migratory endoparasitic nematodes. Whether such resistance is exploitable in plant breeding, depends upon the genetic mechanisms involved.

CONCLUSION

There is a wide range of varietal differences in host efficiency and sensitivity to nematodes of cereals. In some cases, selection or breeding of resistant varieties is being undertaken to provide control measures against nematodes. Future advances and best use of varietal resistance will depend on characterizing the effects of the resistance mechanism on host and nematode, investigating inheritance of resistance, identifying virulence in nematodes, and studying genetic control of virulence.

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