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THE GENETICS OF PLANT-NEMATODE PARASITIC SYSTEMS[1]

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ABSTRACT

The genetic basis of plant-nematode interactions is discussed with special emphasis on potato-*Globodera rostochiensis/pallida*, barley/oat-*Heterodera avenae*, soybean-*H. glycines* and tomato-*Meloidogyne incognita* parasitic systems. The basis of physiological specialization and its role in breaking down cultivar resistance is explored. There are numerous reports on the inheritance of resistance as compared to a few on the inheritance of virulence. These reports indicate certain general characteristics of the host-parasite genes. The genes for resistance and virulence are conditional in the sense that the expression of one is dependent on the presence of the other in the association. Therefore, host-parasite genes, often exhibit a gene-for-gene interaction, the basis of which is discussed and illustrated. Also, the genetics of the complex parasitic systems is briefly mentioned. Fungal and nematode parasitic systems are compared throughout the review.

ZUSAMMENFASSUNG

Diese Zusammenfassung bespricht die genetische Basis der Interaktion zwischen Pflanzen und Nematoden, mit spezialer Rücksicht auf die parasitischen Systemen: Kartoffel-*Globodera rostochiensis/pallida*, Gerste/Hafer-*Heterodera avenae*, Sojabohne-*Heterodera glycines*, und tomate-*Meloidogyne incognita*. Man untersucht die Basis der physiologischer Spezializierung sowie seine Rolle im Zusammenbruch der Pflanzenresistenz. Es gibt viele Berichte über Resistenzvererbung, aber wenige über Vererbung der Virulenz. Solche Berichte deuten auf bestimmte allgemeine Merkmale den Resistenzgenen und Virulenzfaktoren. Die Genen für Resistenz und die für Virulenz sind einander abhängig, im Sinn, dass der Ausdruck dem Wirtresistenzgen die Anwesenheit dem Parasitvirulenzgen benötigt. Deshalb, stellen diese Genen oft eine Gen-fur-Gen Interaktion dar, dessen Basis hier besprochen und illustriert wird. Auch bespricht mann in Kurz die Genetik complizierten parasitischen Systeme. Durchaus dieser Zusammenfassung, vergleicht man die Pilze und Nematoden systemen.

RESÚMEN

Se discute la base genética de las interacciones planta-nemátodo con especial énfasis en los sistemas parasíticos papa-*Globodera rostochiensis/pallida*, cebada/avena-*Heterodera avenae*, soya-*H. glycines* y tomate-*Meloidogyne incognita*. Se explora la base de la especialización fisiológica y su papel en romper la resistencia característica de variedad. Hay numerosos reportes sobre la transmisión genética de la resistencia, en

comparación con unos pocos sobre la transmisión genética de la virulencia. Estos reportes indican ciertas características generales de los genes huésped-parásito. Los genes responsables de la resistencia y la virulencia son condicionales en el sentido de que la expresión de uno de ellos depende de la presencia del otro en la asociación. En consecuencia, los genes huésped-parásito generalmente exhiben una interacción gene-por-gene, la base de la cual se discute e ilustra. También se menciona brevemente la genética de los sistemas parasíticos complejos. Los sistemas parasíticos de hongos y nemátodos se comparan a lo largo de esta revisión.

I. INTRODUCTION

The control of crop pests and diseases by breeding and growing resistant cultivars is one of the oldest and safest methods of improving crop yields. In the middle of the 19th century, potatoes resistant to the late blight fungus, *Phytophthora infestans,* became the first "man-made" disease resistant crop. However, it was not until early in the 20th century that genetic resistance to nematode was found, namely in cowpea plants against root-knot nematodes (Orton, 1911). Biffen (1905) first demonstrated that plant resistance to disease was inherited in a typical Mendelian fashion. Since then the genetics of pest and disease resistance has been pursued intensively especially as many phytopathogenic organisms continually overcome the resistance of host varieties. The discovery of sex in smuts (Kniep, 1919) and rusts (Craigie, 1927) led to further studies on the genetic basis of virulence (pathogenicity) in fungal (Johnson and Newton, 1940; Nicolaisen, 1934) and non-fungal organisms (Sidhu, 1975). Subsequently, Flor (1955, 1971) ingeniously investigated the genetics of resistance in the host and of virulence in the parasite, using the flax-flax rust (*Melampsora lini*) system, and proposed the classic hypothesis of a gene-for-gene interaction.

Most of the studies on host-parasite genetics have been based mainly on the fungus-plant interaction and they have been extensively discussed and reviewed (Day, 1974; Robinson, 1976). The application of these basic principles of host-parasite relationships to plant nematode pests in recent years has led to an understanding of nematode plant parasitic systems. This paper discusses the inheritance of resistance and virulence in the nematode-plant interaction and its relationship to physiological specialization, and the genetics of nematode disease complexes. To facilitate interpretation we first define commonly used terms (see also Robinson, 1976).

A plant is *resistant* if a nematode species reproduces poorly or not at all in it, and is *susceptible* if a nematode reproduces and develops a large

population in it (cf. Rohde, 1972). The term *tolerance* is used when a plant can sustain the effect of invasion and subsequent feeding without serious loss of yield, and often with a modest increase in the nematode population.

Two kinds of host resistance are recognized in parasitic systems: *vertical resistance* (*specific*), associated with oligogenes or major genes that generally exhibit gene-for-gene interactions and often results in the recognition of physiological races or pathotypes, and *horizontal resistance* (*nonspecific*), associated with polygenes or minor genes and is often termed as general resistance.

The terms physiological or biological race, biotype or pathotype and trophotype have been used to define physiological and pathogenic variation in the nematode-plant interaction. In our opinion, the term "biological race," as used by nematologists, is synonymous with the term "physiological race" as used by the fungal plant pathologists and describes populations within a nematode species that are recognized as morphologically similar but have different host perferences. The botanical term "biotype," as defined by Johannsen (1903), should be applied only to those populations of a nematode species that reproduce parthenogenetically in addition to being morphologically identical but have different host preferences, e.g. several *Meloidogyne* species (Sturhan, 1971). The term "pathotype," as described by Cole and Howard (1966), should be applied only to those populations of a nematode species that are morphologically similar, have different host preferences and whose mode of reproduction is amphimictic, e.g. many *Heterodera* species. Hence, although these commonly used nematological terms "biological race," "biotype" and "pathotype" may be used synonymously, the purists may wish to adhere to the original definitions as long as this does not lead to confusion.

"Physiological races" of pathogens, and "cultivars" of hosts are currently accepted as the smallest units of the components of the host-parasite interaction. Physiological specialization is well documented for phytopathogenic fungi, but warrants further examination and discussion in terms of plant-parasitic nematodes.

II. PHYSIOLOGICAL SPECIALIZATION

Any explanation of the evolution of race formation is likely based on the following assumptions: (i) chromosome changes and gene mutations are common in all organisms, (ii) natural selection favors beneficial mutations which are passed on to the next generation and (iii) genetic variability created by mutations lead to genetic drift in a given direction and, therefore, to new races (Kehr, 1966; Steiner, 1925). Most of the earlier observations on race formation involved the endoparasites *Tylenchus*

dipsaci (=*Ditylenchus dipsaci*), *H. marioni* (=*Meloidogyne* spp.) and *H. schachtii*. However, *H. marioni* was subsequently divided into a separate genus containing several species many of which are now regarded as sibling species with distinct biological characteristics and reproductive barriers (Sturhan, 1971). Further, it appears that physiological specialization to the level of pathotypes/biotypes is more often found in endoparasitic than in ectoparasitic species. In 1930 De Bruyn Ouboter claimed that selection pressure on genetic variability is the major source of development of new races in nematodes (quoted by Sturhan, 1971). The problem of race genesis in nematodes has been briefly considered by others (Bingefors, 1971; Hesling, 1966; Sturhan, 1971; Wallace, 1963; Webster, 1969) and on this basis we will now examine the genetic basis of race formation and adaptation.

GENETIC ADAPTATION

Genetic variability resulting from sexual recombination and mutation is screened by natural selection, and the individuals better suited to the environment survive and preferentially reproduce. Selection, therefore, operates on the genetic variability of populations and favors those genes and genotypes that are better suited to a given biotic and abiotic environment. As a corollary, changes in the environment facilitate a corresponding change in gene or genotype frequencies of the populations. Therefore, the environment plays a decisive role in the genetic adaptation of host-parasite populations.

The genetic basis of the association between the host and the parasite was considered by Person (1959, 1968). In parasitism one partner benefits at the expense of the other and the relationship between the host and the parasite is essentially antagonistic. This type of relationship becomes obvious in the case of obligate parasites where the host controls the parasite's environment for all or a significant part of its life cycle. Person (1968) remarks that

> . . . in this type of parasitic relationship, exemplified in fungi by the rusts, the environment of the parasite is completely provided by the host and, more importantly, is under the genetic control of the host. From the standpoint of the host, the presence of the parasite must be considered a *not* insignificant factor in its environment; this factor of the host environment is under the genetic control of the parasite.

Therefore, the basis of the host-parasite relationship is the interplay of the respective genetic systems of the participating organisms. The degree of specificity, however, is determined by the microevolutionary changes which take place in both the host and parasite populations. The understanding of this concept has mostly been obtained from fungal parasites of agricultural crops.

The most important source of variability is genetic recombination

through sexuality, and those nematode species which are amphimictic exhibit much physiological specialization. Lack of alternative mechanisms of variability in nematodes, such as heterokaryosis, mitotic recombination, parasexuality that are common among fungal pathogens, and the fewer number of generations per year, further put these organisms at an evolutionary disadvantage. Hence, the proliferation of new races is much slower than in fungi. However, different adverse influences on nematode populations could be expected to increase the rate of mutation and selection pressure and, therefore, accelerate race genesis, depending on whether they are r- or K-strategists. Nevertheless, most nematode species are polyphagous and this evolutionary adaptation may contribute to their better survival.

SPECIFICITY

Host specificity attains its ultimate expression in obligate parasites and biotrophy is an inevitable basis for this phenomenon in host-parasite interactions (Brian, 1976; Lewis, 1973). A moderate degree of specificity may be observed among widely different taxonomic groups of parasites and their hosts, which have evolved independently on several different occasions. However, taxonomically related pathogens which have coevolved with their hosts may not differ widely in their specificity. For example, the host range of *Globodera rostochiensis* and *G. pallida* on the Solanaceae can be understood in terms of coevolution because both parasite species and the potato host are known to have originated in South America and were later transported to Europe. Hence, divergent evolution of nematode parasites along with their solanaceous hosts has divergent but complementary genetic systems. However, some of the resistance genes in different solanaceous species would be homologous by descent (Jones, 1972a) and, hence, *G. rostochiensis* may completely overcome the resistance in a true host plant species (e.g. potato cultivar) but only moderately infect a closely related non-host species.

A similar coevolution may have occurred in the *H. avenae* group, but the evolution of grasses and their associated cyst nematodes is less well understood than that of the Solanaceae-cyst nematode complex (Jones, 1972a). In cereals and their *Heterodera* parasites, the genetic homology seems to be of wide occurrence, e.g. *H. avenae* can attack oat, wheat and barley equally well. This genetic homology which is common in solanaceous and graminaceous hosts against nematode parasites occurs also in relation to their fungal pathogens. Nevertheless, the level of specificity in fungal parasitic systems appears to be greater which may be due to some of the favorable factors that contribute to their successful microevolutionary adaptations.

Physiological specialization in nematodes, unlike that in fungal parasites, is mostly higher than the species level. For example, some nematode species attack various host species of the same genus. However, intensive cultivation of single host species in a particular locality over a long period probably has influenced the evolution of corresponding parasite species or races (e.g., Australian and Canadian races of *H. avenae*). Several nematode species have been divided into biological races, but the lack of inter-crossing and of different morphological characteristics among physiological races, often blurs such claims. Sturhan (1971) suggested that many biological races are probably confused with sibling species and this is supported by the occurrence of subsequently taxonomic redescriptions. For example, pathotypes of *G. rostochiensis* and *G. pallida* differ in female color, reproductive ability, fecundity and genetic incompatibility (Parrott, 1968). On the basis of female color and other differences Stone (1972) divided potato cyst nematodes into two distinct species, *G. pallida* and *G. rostochiensis* (see also Guile, 1967; Howard, 1972a; Jones et al., 1970).

Intra- and interspecific variation in plant-parasitic nematodes is influenced by many biotic and abiotic factors in the soil. However, there is no reason to discount the fact that physiological races of the type found in obligate fungal parasites should not exist in nematode parasites. In fact, races (biotypes, pathotypes) have been described in many nematode species. The pathotypes have been found mostly in sexually-reproducing nematode species as compared to the ameiotically-reproducing nematode species.

PHYSIOLOGICAL RACES

To produce better crops, heavy yielding cultivars have been bred through selection and breeding of plants showing specific taxonomic and physiological characteristics. By growing monocultures of genetically uniform cultivars man has also encouraged the corresponding evolution of parasites on these cultivars. This led Johnson (1961) to designate the evolution of agricultural parasitic systems as "man-made."

The identification of physiological races was demonstrated in cereal rusts (Stakman, 1914) and a similar approach has been taken with nematode parasites. A summary of physiological races, either suggested or demonstrated in various nematode species, is given in Table I.

RACE VS. CULTIVAR

Physiological races are identified by their reaction to a set of test host cultivars known as "differentials." A chosen set of "differentials" repre-

Table I

Physiological races either suggested or demonstrated in sexually and asexually reproducing plant parasitic nematodes on various hosts

Nematode spp.	No. of races	Mode of reproduction	Reference(s)
Ditylenchus:			
dipsaci	20	Amphimictic	Steiner, 1956; Sturhan, 1971
destructor	4	Amphimictic	Smart and Darling, 1963
radicicola	?	Amphimictic	S'Jacob, 1962
Globodera:			
rostochiensis	5+	Amphimictic	Dunnett, 1957; Huijsman, 1974; Jones and Parrott, 1965; Toxopeus, 1956
pallida	5+	Amphimictic	Kort et al., 1977; Stone, 1972
Heterodera:			
avenae	3 (B)[a]	Amphimictic	Cotton, 1962; Fiddian and Kimber, 1964; Hayes and Cotton, 1971; Saynor, 1975
	2 (D)[a]	Amphimictic	Andersen, 1959
	6 (G)[a]	Amphimictic	Jones, 1972b; Luke, 1969
	4 (N)[a]	Amphimictic	Kort et al., 1964
	2 (S)[a]	Amphimictic	Cook and Williams, 1972
schachtii	?	Amphimictic	Shepherd, 1959
glycines	5	Amphimictic	Epps and Golden, 1967; Golden et al., 1970; Triantaphyllou, 1975
Meloidogyne:	B-races	Parthenogenetic	Goplen et al., 1959; Netscher, 1976; Price et al., 1978
incognita	4	Parthenogenetic	Sasser, 1979
arenaria	2	Parthenogenetic	Sasser, 1979
Pratylenchus:			
penetrans	2	Amphimictic	Olthof, 1968
Radopholus:			
similis	3	Amphimictic	DuCharme and Birchfield, 1956
Tylenchus:			
semipenetrans	2	Amphimictic	Baines et al., 1969
Other species	?		Sturhan, 1971

[a] B = Britain. D = Denmark. G = Germany. N = The Netherlands. S = Sweden.

sents a series of independent biological systems which, taken as a group, define the physiological race (Person, 1958). The differences expressed in "differentials" are usually only physiological as distinct from the primary morphological characters associated with species differentiation. Hence, two physiological races may be morphologically identical but may show different pathogenicity on a given host cultivar, and these differences may or may not have an identical genetic basis. Irrespective of the morphological differences, the characteristic of parasites which concerns us here is their ability to reproduce and induce disease in the host.

In terms of two vertical responses, i.e. resistance vs. susceptibility on the part of a host, and virulence vs. avirulence on the part of a parasite, a host cultivar can identify two physiological races of a parasite or conversely a physiological race can define two host cultivars. The basic expression 2^n, which leads to a geometric series, determines the total number of possible races where n = cultivars, and the converse applies to the host. The genes for pathogenicity expressed by a given physiological race on a set of differentials correspond to the number of host-cultivars present in the set. Segregations at an individual virulent locus are revealed only on a single cultivar. Similarly, segregations at an individual resistance locus are obtained against a single physiological race. Therefore, the essential units of host-parasite interaction are the "cultivar" and the "physiological race" on the basis of which the current host-parasite relationships are founded. These units, when translated into the segregating unit of genetics—the gene—form the basis of the genetic interpretation of the host-parasite relationship. The factors involved in breeding for resistance to plant-parasitic nematodes have been mentioned in other reviews (Bingefors, 1971; Hare, 1965; Harrison, 1960; Hunt et al., 1975; Kehr, 1966; Malo, 1964; Rohde, 1972; Sidhu and Webster, 1981) whereas the present review is examining the practical and theoretical basis of the genetics of nematode parasitism in plants.

III. GENETICS OF RESISTANCE

BEFORE 1950

The discovery by Biffen (1905) of resistant genes (R-genes) in wheat against yellow rust initiated interest in the inheritance of resistance in other parasitic systems. Orton (1911) was the first to consider the genetics of resistance to nematodes in his study of the resistance of cowpea (*Vigna sinensis*) to a root-knot nematode species previously shown to be resistant by Webber and Orton (1902). He tested F_1 and F_2 progenies but no discernible ratios were reported. He noticed great variation in disease responses in the F_2 progeny, and postulated that selection plays a major role in nematode resistance. Nearly ten years later Nilsson-Ehle (1920), reported on the resistance in barley to *Heterodera schachtii* Schmidt. He

Table II

A partial summary of genetic studies of host resistance to nematode species published before 1950

Parasitic system		Mode of inheritance[a]	No. of genes identified	Reference
Alfalfa:	*D. dipsaci*	? (D)	?	Barham and Sasser, 1956
Barley:	*H. avenae*	Mono (D)	one	Nielsson-Ehle, 1920
Beans:	*M. incognita*	Di (R)	two (?)	Barrons, 1940
Cowpeas:	*H. radicicola*	? (D)	?	Mackie, 1934
Cotton:	*M. incognita*	? (R)	?	Smith, 1941
Peach:	*M. incognita*	? (D)	?	Weinberger et al., 1943
Tobacco:	*M. incognita*	Poly (R)	?	Clayton and Foster, 1940
Tomato:	*M. incognita*	Poly (D)	?	McFarlane et al., 1946
		Mono (D)	one (?)	Watts, 1947
		Mono, Di or Poly (D)	?	Frazier and Dunnett, 1949

[a] Mono = monogenic; Di = digenic; Poly = polygenic; D = dominant; R = recessive.

crossed resistant and susceptible barley cultivars and from studying the responses of the F_1, F_2 and F_3 progeny concluded that resistance was controlled by a single dominant gene. His work was the first attempt to elucidate the inheritance of resistance to plant-parasitic nematodes. Burkart (1937) searched for resistance in alfalfa to *Ditylenchus dipsaci* but no specific mode of inheritance was found. During the 1940s many scientists investigated inheritance of resistance to *Meloidogyne* spp. (see Table II) but most results were inconclusive. This was due probably to the use of mixtures of species or biological races.

AFTER 1950

Identification of R-genes in the host is largely dependent on the use of appropriate cultures of the pathogen. Christie and Albin (1944) reported that several populations of *G. marioni* exhibited variations in their ability to reproduce (pathogenicity) on selected host species. However, Chitwood (1949) differentiated between several species and subspecies of *Meloidogyne* and so enabled subsequent workers to use identifiable species in their experiments. This recognition and isolation of biological races (biotypes or pathotypes) of nematodes gave impetus to the study of the genetics of host-parasite interrelations and resulted in the identification of R-genes in various host species (Table III).

Most of the genetical studies have involved the more host-specific nem-

atode species of the three economically important genera *Globodera*, *Heterodera*, and *Meloidogyne*. Species of these genera are sedentary and endoparasitic and these characteristics influence their specificity. The following parasitic systems have been most extensively studied and will now be considered in detail: (i) Potato-*G. rostochiensis/pallida*; (ii) Barley/Oat-*H. avenae*; (iii) Soybean-*H. glycines*; (iv) Tomato-*M. incognita*.

(i) Potato-*Globodera rostochiensis/pallida*

Screening of *Solanum* spp. for resistance to *G. rostochiensis* was started by Ellenby (1952, 1954), but the breeding of potato cultivars resistant to *G. rostochiensis* was initiated by Toxopeus, Huijsman, and Howard (see Jones, 1954). However, Toxopeus and Huijsman (1952) were the first to publish the hereditary nature of resistance to the potato cyst nematode. Theoretically expected ratios between different levels of ploidy in potato i.e. duplex × duplex (36R:1S), duplex × simplex (11R:1S) and simplex × simplex (3R:1S) were in close agreement with the experimental results (Toxopeus and Huijsman, 1953). This provided the first conclusive evidence of a single gene namely H_1 (*Heterodera* resistance gene number one), which was found in *andigena* clones of the Commonwealth Potato Collection (C.P.C.) numbers 1673 and 1685. Later, an identical R-gene in clone C.P.C. 1690 was described (Cole and Howard, 1957).

A resistance-breaking pathotype evolved against R-gene H_1, presumably in Perú, and was discovered in field populations in England (Dunnett, 1957; Huijsman, 1958; Jones, 1958). This led to a new phase in breeding for nematode resistance. Dunnett (1961) tested the wild type diploid species, *S. multidissectum*, for resistance to new pathotype(s) and identified another R-gene, H_2. Soon, more pathotypes were discovered and schemes for their identification were proposed (Cole and Howard, 1957; Huijsman, 1962; Ross and Huijsman, 1969). British pathotypes A (*G. rostochiensis*), and B and E (*G. pallida*), and Dutch pathotypes A, B, and C (*G. rostochiensis*) and D (*G. pallida*) were recognized. Resistance gene H_1 was effective against Dutch pathotype A but not against B whereas R-gene H_2 gave the converse response. During the same period it was reported from England (Guile, 1967; Jones and Pawelska, 1963; Jones and Parrott, 1965) and, subsequently, from Germany and the Netherlands (Ross and Huijsman, 1969) that in addition to populations of pathotypes A and B there also existed a third pathotype, C, which could nullify the resistance of both the H_1 and H_2 R-genes. Clones having either one or both of the R-genes were rendered susceptible and this led to the discovery of a third R-gene, H_3, in clones C.P.C. 2775 and 2802 from an *andigena* source (Howard, 1972b; Howard et al., 1970; Parrott and Trudgill, 1970).

Table III

A partial summary of genetic studies of host resistance to nematode species published after 1950

Parasitic system	Mode of inheritance[a]	No. of genes identified	Reference(s)
Alfalfa:			
D. dipsaci	Mono (D)	one, poly	Grundbacher and Stanford, 1962
M. hapla	Mono (D)	one	Goplen and Stanford, 1960
M. javanica	Mono (D)	one	Goplen and Stanford, 1960
Barley:			
H. avenae	Mono (D)	two	Andersen, 1959
	Mono (D)	two	Andersen and Andersen, 1968
	Mono (D)	three	Cotton and Hayes, 1969
	Mono (D)	four	Hayes and Cotton, 1971
	Mono (R)	one	Hayes and Cotton, 1971
	Mono (R)	one	Cook, 1974
Beans:			
M. incognita	Di (R)	two (?)	McGuire et al., 1961
	Poly	—	Blazey et al., 1964
	Tri	three (?)	Hartman, 1971
Beets:			
H. schachtii	(D) ?	—	Savitsky and Price, 1965
Citrus:			
T. semipenetrans	(D) ?	?	Cameron et al., 1954
Cotton:			
M. incognita	Poly (R)	—	Smith, 1954
	Poly (R)	—	Wiles, 1957
	Di (R)	two	Turcotte et al., 1963
Oats:			
H. avenae	Mono (D)	one	Andersen and Andersen, 1970
	Poly (PD)	—	Andersen and Andersen, 1970
	Mono (D)	three	Andersen and Andersen, 1970
	Di (D)	two	Cotton and Hayes, 1972
	Di (R)	two	Cotton and Hayes, 1972
	Mono (D)	one	Cotton and Hayes, 1972
	Mono (D)	one	Cook, 1974
D. dipsaci	Mono (D)	one	Griffiths et al., 1957
	Di (?)	two	Cook, 1974

Table III
Continued

Parasitic system	Mode of inheritance[a]	No. of genes identified	Reference(s)
Peach:			
M. incognita	(D) ?	?	Sharpe, 1958
Pepper:			
M. incognita	Mono (D)	one	Hare, 1957
Potato:			
G. rostochiensis	Mono (D)	one	Toxopeus and Huijsman, 1953
	Mono (D)	one	Cole and Howard, 1957
	Mono (D)	one	Dunnett, 1961
	Di (D)	two	Ross, 1962
	Mono (D)	one	Howard et al., 1970
	Mono (D)	one	Parrott and Trudgill, 1970
	Di (D)	two	Jones, 1972a
	Poly (?)	—	Huijsman, 1974; Ross, 1969
Red clover:			
D. dipsaci	Poly (?)	—	Bingefors, 1957
Soybean:			
H. glycines	Mono (R)	three	Caldwell et al., 1960
	Mono (D)	one	Matson and Williams, 1965
M. incognita	Di (PD)	two	Boquet et al., 1976
Tobacco:			
M. incognita acrita	Mono (D)	one (?)	Drolson et al., 1958
Tomato:			
M. incognita	Mono (D)	one	Gilbert and McGuire, 1956
	Mono (D)	one	Thomason and Smith, 1957
	Mono (PD)	one	Barham and Winstead, 1957
	Mono (D)	one (?)	Harrison, 1960
	Mono (D)	two	Sidhu and Webster, 1973
	Mono (R)	one (?)	Sidhu and Webster, 1973
	Mono (D)	two	Fatunla and Salu, 1977
	Mono (D)	one	Sidhu and Webster, 1980
Meloidogyne spp.	Mono (D)	one (?)	Barham and Sasser, 1956
	Mono (D)	one	Barham and Winstead, 1957
G. rostochiensis	Mono (D)	one	Ellis and Maxon Smith, 1971

Table III
Continued

Parasitic system	Mode of inheritance[a]	No. of genes identified	Reference(s)
Wheat:			
H. avenae	Mono (D)	one	Nielsen, 1966; Slootmaker et al., 1974
Vitis:			
M. incognita	Mono (D)	one	Lider, 1954

[a] Mono = monogenic; Di = digenic; Tri = Trigenic; Poly = polygenic; D = dominant; R = recessive; PD = partially dominant.

The confusion over the potato cyst nematode species (Jones et al., 1970) was resolved by Stone (1972) who described the pathotypes of *G. rostochiensis* with white or cream colored females as belonging to the species *G. pallida* (British pathotypes B and E, and the Dutch pathotype D) and those with golden-yellow colored females as belonging to *G. rostochiensis* (British pathotype A and Dutch pathotypes A, B, and C) (see also Table IV). Dutch pathotype B has not been found in Great Britain and British pathotype E equates with Dutch pathotye D (Howard, 1972a). The recognition of additional pathotypes due to selection pressures could be expected. Pathotype E of *G. pallida,* which existed with A of *G. rostochiensis* in low frequencies, has increased in Great Britain. Also mixtures of A of *G. rostochiensis* and B or E of *G. pallida* were found (Howard, 1972a; Howard and Fuller, 1971). The recognition of a relatively widespread distribution or more than one virulent gene necessitated the development of a broader base of resistance in potato hosts, especially against the newly discovered species, either through the combination of existing R-genes or by identifying new R-genes.

Fuller and Howard (1974) compared resistance to the most prevalent pathotype E of *G. pallida* derived from the diploid wild potato species *S. vernei* with that from *andigena* clones. It had been claimed that *S. vernei* contained polygenes (Goffart and Ross, 1954; Huijsman and Lamberts, 1972; Kort et al., 1972), or few major genes plus modifiers, or even an H_1 type R-gene (De Scurrah et al., 1973; Huijsman, 1974; Ross, 1969). However, bad cooking quality and inconclusive data on the mode of inheritance of resistance in *S. vernei* discouraged plant breeders from incorporating it into the breeding of new cultivars. Therefore, a combination of R-genes, H_3 from *andigena* C.P.C. 2775 or 2802 and H_2 from *S. multidissectum* against pathotype E of *H. pallida,* was suggested (Fuller and Howard, 1974; Howard and Fuller, 1975).

Table IV

Source and response of resistant genes (R-genes) in potato (*Solanum*) to British (I), Dutch (II), and New (III) pathotyping schemes of *Heterodera* (=*Globodera*) *rostochiensis* and *Heterodera* (=*Globodera*) *pallida*

		Response of pathotypes of								
		G. rostochiensis					G. pallida			Scheme
		A					B		E	I
	Resistance gene	A	B	C	F			D	E	II
Potato clone	symbol	Ro1	Ro2	Ro3	Ro4	Ro5	Pa1	Pa2	Pa3	III
S. tub. ssp. *tuberosum*	H_0	S	S	S	S	S	S	S	S	
S. tub. ssp. *andigena* CPC 1673, 1960	H_1	R	S	S	R	S	S	S	S	
S. multidissectum P. 55/7	H_2	S	S	S	S	S	R	S	S	
S. tub. ssp. *andigena* CPC 2775	H_3	S	S	S	S	S	R	R	R	
S. multi. × *S.* ssp. *andigena* K3/5	H_1H_2	R	S	S	R	S	R	S	S	
S. ssp. *andigena* × *S.* ssp. *andigena* AND 586/1	H_1H_3	R	S	S	R	S	R	R	R	
S. multi. × *S.* ssp. *andigena*	$H_1H_2H_3$	R	?	?	R	R	R	R	R	
S. kurtzianum	K_1	R	R	S	S	S	S	S	S	
S. vernei	Poly	R	R	R	R	R	R	R	R	

Recently two international schemes for designating the pathotypes of potato cyst-nematodes *Globodera* (=*Heterodera*) *rostochiensis* and *G. pallida* have been suggested (Canto Saenz and De Scurrah, 1977; Kort et al., 1977). In the scheme suggested by Kort et al. (1977), a similar nomenclature is applied to both the pathotypes and the differentials. These authors point out that such a scheme is useful for farmers, extension workers and nematologists. However, we think that it is not meaningful to those researchers who are not familiar with nematode parasitic systems. For example, symbols Ro (*rostochiensis*) and Pa (*pallida*) are used for both the pathotypes and the cultivars. In our opinion a descriptive scheme should be one which readily differentiates the parasitic system, the participating members and, wherever possible, the R- and V-genes. Therefore, we share the view (Howard, 1972a) that the R-genes, rather than the names of clones or cultivars, should be used for designating the differential hosts. Such a nomenclatorial scheme would be

helpful to geneticists, plant breeders, nematologists and educated growers alike, and has been described in detail by Sidhu (1976). The second scheme (Canto Saenz and De Scurrah, 1977) seems to incorporate some of the suggestions made above and elsewhere (Sidhu, 1976).

Currently a number of R-genes against both *G. rostochiensis* and *G. pallida* are known in various *Solanum* species (Table IV). In addition to the R-genes listed in Table IV, there are other major genes referred to in wild species, e.g. *S. spegazzinii* contains Fa and Fb R-genes (Ross, 1962) and *S. vernei* contains polygenes (Goffart and Ross, 1954) or a complex of major and minor genes (Huijsman, 1974; Ross, 1969).

(ii) Barley-*Heterodera avenae*

After the report by Nilsson-Ehle (1920), there were no significant studies on the inheritance of resistance until the isolation of two pathotypes, 1 and 2, in Denmark (Andersen, 1959). Later, two independent dominant R-genes were identified (Andersen and Andersen, 1968; Andersen and Andersen, 1970)—one present in the cultivars Drost and Fero effective against pathotype 1, and the second in cultivar No. 191 effective against both pathotypes 1 and 2. A third R-gene in cultivar No. 14, effective against both the pathotypes, was indicated in their studies and it was later confirmed to be different from but closely linked to the R-gene present in cultivar No. 191 (Cotton and Hayes, 1969).

The comprehensive studies by Cotton and Hayes (1969) reaffirmed the results (Andersen and Andersen, 1968) and also showed that the R-gene present in No. 191, which was designated as *Ha* (*Heterodera avenae*), is located on chromosome 2. The other R-genes present in No. 14, C.1. 8334 and C.1. 3902 were shown to be different from but closely linked with the *Ha* gene. The location of these R-genes on chromosome 2 was ascertained in relation to other genes controlling morphological characters (Andersen and Andersen, 1973; Cotton and Hayes, 1969). However, no linkage values are available between the R-genes per se. Currently, there are at least six R-genes against the various pathotypes of *H. avenae* (Hayes and Cotton, 1971). A recent report has indicated the presence of at least three dominant R-genes with the possibility of two others, in the barley cultivars Athinais, C98147, Marocaine 079, Nile and Morocco (O'Brien et al., 1979). However, no attempt has been made to correlate these R-genes with other *Ha* genes. A summary of the genetic studies with respect to R-genes and their reaction to British, Danish, Dutch and Swedish pathotypes is given in Table V. Discussion of the relationships of the various pathotypes (Cook and Williams, 1972; Saynor, 1975) and the genetics of resistance in barley (Andersen and Andersen, 1970; Hayes and Cotton, 1971), is available in the literature.

Table V

A summary of resistance genes (R-genes) found in the barley-*Heterodera avenae* parasitic system

Cultivar	Resistance due to R-gene			Resistance to pathotypes from[a]				Reference(s)
	Symbol[b]	Allelic	Linked	B	D	N	S	
Drost, Fero	Ha_{df}	—	—	1	1	ABD	S_1	Andersen and Andersen, 1968
No. 191	Ha_s	—	—	1,2	1,2	ACD	S_1,S_2	Andersen and Andersen, 1968
No. 14	Ha_c	—	Ha_s	$1,2_p$[c]	1,2	ACD	S_1,S_2	Andersen and Andersen, 1968; Cotton and Hayes, 1969
Marocaine, Morocco	Ha_m	Ha_s,Ha_c (?)	Ha_s,Ha_c (?)	1,2	1,2	ABCD	S_1,S_2	Cotton and Hayes, 1969; Kort et al., 1964
Harlan 43	ha_h (?)	—	—	1_p,2_p,3	—	DC_p	—	Cook, 1974; Hayes and Cotton, 1971

[a] B = Britain, D = Denmark, N = The Netherlands, S = Sweden.

[b] Ha = *Heterodera avenae* and letters in subscript denote the name of the cultivar in which R-genes were originally discovered; Ha and ha indicate R-gene being dominant and recessive, respectively.

[c] p = partial resistance.

Table VI

A summary of resistant genes (R-genes) found in the oat-*Heterodera avenae* system

Host species	Cultivar or selection no.	Mode of inheritance		Reference(s)
		No. R-genes[a]	Relationship	
A. byzantina	Cc 4701	one (D)	—	Cotton and Hayes, 1972
A. sterilis	Cc 4658	three (D)	independent	Andersen and Andersen, 1970
	Cc 4658	two (D)	epistatic, allelic	Cotton and Hayes, 1972
A. sativa	C.1 3444 P.1 175022 'Silva'	one (D)	same locus or different alleles	Andersen and Andersen, 1970; Cook, 1974
	'Nelson'	one-two (?) (D)	same locus or linked genes	Cook, 1974
	Mortgage lifter	two (R)	independent	Cotton and Hayes, 1972

[a] D = dominant, R = recessive.

(iii) Oat-*Heterodera avenae*

Pathotypes of *H. avenae* which attack barley also attack oats. Strong resistance in oats has been found in wild diploid and tetraploid species and breeding for resistance has been most readily exploited in the cultivated hexaploid oats. Unfortunately, the identification and interrelationships of R-genes in hexaploid species remains obscure due to the greater genome complexity. Nevertheless, some genetic studies on the inheritance of resistance are available (Andersen and Andersen, 1970; Cook, 1974; Cotton and Hayes, 1972). Approximately five R-genes are known and a summary of their mode of inheritance is given in Table VI.

(iv) Soybeans-*Heterodera glycines*

Relatively more is known about the genetics of resistance in soybeans against *H. glycines*. Ross and Brim (1957) tested 2800 plant introductions and cultivars of soybeans and found that three strains, Peking, P.I. 90763 and P.I. 84751 possess effective resistance based on the absence of white females on the roots. From the crosses between the above mentioned resistant strains and susceptible cultivars Lee, Hill and D53-354, three independent recessive R-genes rhg_1, rhg_2 and rhg_3 were identified (Caldwell et al., 1960). However, there was a reduction in the resistant class

which was suspected due to zygotic and/or gametic elimination. Another dominant R-gene, Rhg_4 was discovered (Matson and Williams, 1965) in cultivar Peking at a separate locus from that previously identified for the three recessive genes. They (Matson and Williams, 1965) also discovered that the R-gene Rhg_4 was closely linked with the *I* locus for seed-coat color. All the resistance genes mentioned above were identified using field populations of *H. glycines*. Thereafter, four *H. glycines* races designated as 1, 2, 3, 4 were described on a standard set of differentials (Golden et al., 1970). This lead to the identification of other resistance genes, namely one recessive gene against race 2 (Hartwig and Epps, 1970), and one dominant and two recessive genes against race 4 (Thomas et al., 1975). It is interesting to note that resistance in soybeans to *H. glycines*, often seems to be inherited as a recessive characteristic which is in sharp contrast to other plant parasitic systems especially those involving nematodes.

(v) Other hosts vs. *Heterodera* spp.

Genes controlling resistance to *H. avenae* in wheat and rye are also known (see Table III). One R-gene in the wheat cultivar Loros on chromosome 2B is effective against Australian and European pathotypes (Cook, 1974; Nielsen, 1966; Slootmaker et al., 1974). No specific mode of inheritance in rye has been studied. A single R-gene, *Hero*, is known in tomato, *Lycopersicon pimpinellifolium*, against *G. rostochiensis* (Ellis and Maxon Smith, 1971).

(vi) Tomato-*Meloidogyne incognita*

Inheritance of resistance to *Meloidogyne* spp. has been studied in various host species, but the tomato-*M. incognita* system is the most explored. As early as 1949 it was reported that resistance in tomatoes appeared to be dominant and controlled by a small number of R-genes (Frazier and Dunnett, 1949). The *Mi* gene (=*Meloidogyne incognita*), was identified (Gilbert and McGuire, 1956). This R-gene, present in linkage group IV, is effective also against all other *Meloidogyne* spp. that attack tomato, except *M. hapla*. Several other workers reported a single dominant or incompletely dominant R-gene effective against *Meloidogyne* spp. (Barham and Winstead, 1957; Cordner et al., 1965; Thomason and Smith, 1957; Winstead and Barham, 1957). A similar study (Barham and Sasser, 1956) showed that resistance to *Meloidogyne* species was controlled by one or more dominant genes and it was suggested that it may be due to a single dominant gene or a block of genes acting as a unit (Harrison, 1960).

Nearly all other reports indicate a non-specific mode of inheritance of resistance. This may be due to the use of impure cultures or to the origin of B-races in situ. Netscher (1976) suggested that B-races may develop by growing resistance cultivars in *Meloidogyne*-infected soils, or may occur spontaneously. The occurrence of B-races in a single egg mass culture of *M. incognita* on a resistant cultivar Hawaii 5229 was shown (Riggs and Winstead, 1959). The potential for giving rise to new biotypes was subsequently confirmed (Triantaphyllou and Sasser, 1960).

If B-races exist there is no reason to discount the possibility that R-genes effective against them should also exist. Hence, an investigation was undertaken to discover additional R-genes in existing cultivars (Sidhu and Webster, 1973). Two dominant and one recessive R-genes, designated as $LMiR_1$, $LMiR_2$ and $LMir_3$, were identified. The dominant R-genes $LMiR_1$ and $LMiR_2$ are possessed by the cultivars Nematex and Small Fry-1 respectively, whereas the recessive R-gene $LMir_3$ was indicated to be present in Cold Set-1. However, inheritance of the recessive R-gene ($LMir_3$) was later found to be unstable due, probably, to cytoplasmic influences and temperature sensitivity. The R-gene $LMiR_1$ is identical or allelic to the previously identified *Mi* locus whereas $LMiR_2$ is closely linked to $LMiR_1$ and/or *Mi* locus and is 5.65 mμ apart on linkage group IV (Sidhu and Webster, 1975). Another incompletely dominant R-gene in cultivar Rosol is reported, which is different from the $LMiR_1$ gene in Nematex (Fatunla and Salu, 1977). The relationship of the Rosol gene with the $LMiR_2$ gene is currently not known. Recently Sidhu and Webster (1980) reported an allele ($LMiR_1^1$) of $LMiR_1$ for partial resistance in cultivar Rutgers. It is possible that this allele is identical to the Nematex gene but manifests a different level of resistance when present in a different genetic background.

Many R-genes against *Meloidogyne* spp. are known in other agricultural hosts such as alfalfa, beans, cotton, peach, pepper, soybeans, tobacco and *Vitis*. Most of these R-genes are monogenic and dominant in action (see Tables II and III).

(vii) Other parasitic systems

Knowledge of the inheritance of resistance to other nematode species in plants, other than the ones mentioned above, is scarce. The inheritance of resistance in alfalfa to the stem eelworm *Ditylenchus dipsaci* is one of the more extensively studied but the genetic results are usually inconclusive. The mode of inheritance of resistance in parasitic systems that have been relatively less explored is given in Tables II and III.

From the studies on mode of inheritance of resistance we learn that: (i) resistance is most often monogenically controlled; (ii) genes for resis-

tance are often dominant; (iii) in a few cases linkage and allelic relationships among R-genes occur. These characteristics are in common with those of other parasitic systems (Person and Sidhu, 1971; Sidhu, 1975) which suggest that genetic response(s) are elicited by plant hosts irrespective of the animal (nematode, insects) or fungal parasitic species. Such similarities may have their counterparts in identical biochemical reactions in the host.

Our knowledge of nematode resistance in plants is limited as compared with that of fungal parasitic systems (Sidhu, 1975) and in view of the economic importance of plant parasitic nematodes, studies in this field should be intensified. Such studies should consider not only the inheritance of resistance but also the inheritance of virulence, whenever possible.

IV. GENETICS OF VIRULENCE

Very little is known about the inheritance of virulence in plant nematodes. It has been attempted only for *G. rostochiensis/pallida, H. avenae* and *H. glycines*. Jones and Parrott (1965) first advanced the hypothesis that the dominant R-genes are normally matched by the corresponding and related recessive V-genes in the potato-cyst nematode. Later, a single recessive V-gene in *G. rostochiensis* corresponding to R-gene H_1 was discovered (Parrott and Berry, 1974). Further crosses between *G. pallida* populations have shown another V-gene which can nullify the resistance conferred by R-gene H_2 (Parrott, 1978). The inheritance of two dominant V-genes was reported also for *H. avenae* (Andersen, 1965). Similarly, the inheritance of virulence in *H. glycines* on soybeans was found to be dominant (Koliopanos, 1970). Triantaphyllou (1975) attempted the genetic analysis of some of *H. glycines* races and showed that different field populations (races) possess different groups of V-genes which can multiply on corresponding R-genes in soybeans. He postulated that the race pattern changes due to the shift in V-gene frequencies.

Recently, controlled matings were made among four *H. glycines* races using simple females from each (Price et al., 1978). When race 1 or 3 were crossed with race 2 or 4, a reduction in female numbers was found in the progeny (Price et al., 1978). This indicates either sexual incompatibility or the presence of lethal genes which influence the segregation of female genotype. However, crosses 1×3 and 2×4 exhibited a normal segregation. The virulence on cultivar Pickett was controlled by one or two dominant genes present in the races 2 and 4. A recombinant from the cross 3×4 appeared which was virulent on the resistant genotype PG903763.

The evolution of new races through genetic recombination is a usual phenomenon, however, genetic adaptation also plays a definitive role in the evolution of plant nematode parasitic systems. Price et al. (1978) showed that repeated inoculation of races 2 and 4 on resistant cultivar Pickett enhanced the virulence through genetic adaptation. Unless the nature of inheritance of virulence is studied extensively, it may be impossible to control nematode parasites effectively by the use of resistant cultivars.

V. GENE-FOR-GENE RELATIONSHIP

Specificity is a common attribute of most obligate parasitic systems and is controlled by the interacting genomes of the host and the parasite. Such intrinsically antagonistic interactions are controlled by resistant genes in the host and virulent genes in the parasite. H. H. Flor (1955, 1971) demonstrated a numerical equivalence between the R- and V-genes, and experimentally proved that every R-gene in the host is matched by a related and corresponding V-gene in the parasite. This discovery led Flor (1971) to formulate his now widely accepted hyopthesis of a gene-for-gene interaction. This hypothesis was extended and popularized by Person (1959) who concluded that gene-for-gene relationships should occur as a general rule in nature.

Natural parasitic systems are difficult to analyze but we know from the man-manipulated systems (e.g., agricultural crops) that gene-for-gene relationships have either been suggested or demonstrated in about three dozen parasitic systems involving fungal, bacterial, insect, viral as well as nematode parasites (Sidhu, in press). Here we will discuss gene-for-gene relationships with particular reference to plant-parasitic nematodes.

The R-genes in the host and V-genes in the parasite are conditional in the sense that the presence of one is contingent on the presence of the other in the interacting host-parasite populations. It is reasonable to assume, therefore, that this general statement probably applies to all obligate parasitic systems that evolve through microevolutionary changes occurring in the interacting host-parasite populations. Jones and Parrott (1965) postulated that the major genes for resistance in potatoes are matched by the corresponding recessive genes in the nematode. This hypothesis takes into account the potential for the infective larvae to become either male or female according to the gene products in the nematode gland secretions and host transfer cells (Jones, 1974). It would seem, therefore, that the oesophageal gland secretions of the recessive V-gene (*nn*) larvae evoked the susceptible response in the host cells which led to the formation of transfer cell-like structures (feeding site) or giant cells. The secretions of comparable larvae of genotype *NN* or *Nn*

(*N* being the avirulence allele for nematode) fail to incite such a response in a resistant host and so the larvae become male.

To test this hypothesis, controlled crosses between different proportions of pathotypes of *G. rostochiensis* were attempted. However, the information could not establish unequivocally the genetic basis of virulence because of the difficulty of accounting for multiple matings of females. To solve this problem expected frequencies of new cysts in given nematode cultures were deduced on the basis of two hypotheses which presuppose (i) females being *nn*, males *NN*, *Nn* or *nn*, and (ii) females being *NN*, *Nn*, males *NN*, *Nn*, *nn* (Jones, 1967). Jones (1967) calculated that females being *nn* tended to support the hypothesis and indicated that a female mated an average six to seven times. These hypotheses were the results from controlled matings of single females on potato cultivars with single R-genes.

Separation of some pathotypes of *G. rostochiensis* into two species, *G. rostochiensis* and *G. pallida* which do not interbreed freely, created some doubt about the hypotheses (Jones, 1967; Jones and Parrott, 1965). Consequently, the hypotheses were retested by Parrott and Berry (1974) using individual species having pathotypes which do interbreed. They tested F_1 and F_2 progenies derived from a cross between pathotype A (Ro1) (see Table IV) and a Bolivian population of *G. rostochiensis* on a host containing R-gene H_1. The results clearly confirmed the hypothesis that only the recessive females nullified the resistance conferred by the R-genes. Similar results were obtained from studies with crosses of pathotypes B and E of *G. pallida* on the R-gene H_2 (Jones, 1974; Parrott, 1978).

It now is accepted that genes for virulence in nematodes are recessive which is in perfect accord with the situation found in fungal parasites. Accordingly, Jones (1974) assigned genotypes to some pathotypes of *G. rostochiensis* and *G. pallida*. Based on such observations, he formulated a hypothetical scheme of gene-for-gene relationships between known R-genes in the hybrid potatoes and postulated V-genes in the cyst-nematodes. This scheme has been reconfirmed (Parrott, 1978) and the genetic basis of the potato : cyst-nematode interaction is illustrated (Table VII). According to this scheme, the incompatible interaction occurs when the gene products of the dominant alleles for resistance and for avirulence interact specifically in the feeding site. A compatible interaction, however, follows when either one of the dominant alleles, or both of them are absent. This is in perfect agreement with other obligate parasitic systems except for the oat-*Helminthosporium* interaction, and the basis of which is inherent in a quadratic check (Table VII). A similar scheme was formulated for the barley-*H. avenae* system (Hayes and Cotton, 1971).

In the case of soybean-cyst nematode systems the R-genes are normally

Table VII

The basis of gene-for-gene interaction in potato : cyst-nematode system

Host genotype	Nematode genotype				Quadratic check	
	NN	Nn	nn		N–	nn
HH	–	–	+	H–	–	+
Hh	–	–	+	hh	+	+
hh	+	+	+			

Plus (+) and minus (–) indicate compatibility and incompatibility, respectively.

inherited as recessive whereas V-genes as dominant traits. However, the dominant and recessive relationships of host-parasite genes do not influence the basic pattern (quadratic check) of gene-for-gene interaction (see Sidhu, 1975). Therefore, the occurrence of a gene-for-gene relationship in this system can be envisaged. In fact, the interaction between the host cultivars Peking and PG88788, and the parasite races 2 and 4 (Price et al., 1978) does conform to a gene-for-gene pattern.

Sidhu (1975) described two criteria for evaluating gene-for-gene interactions, i.e. Flor's and Person's. Flor's criterion is applied to those parasitic systems where R-genes and V-genes are genetically identified, whereas Person's criterion can be applied to those parasitic systems where genetic studies have not been conducted or cannot be undertaken due to the inherent problems of studying segregations in the interacting species, e.g. asexually reproducing parasitic species or tree hosts. Both these criteria are probably applicable to plant-nematode parasitic systems. However, currently most of them can be studied only on the basis of Person's criterion because of difficulties in studying the genetics of virulence. Although Person's criterion is genetically less precise and explicit than that of Flor, it nevertheless provides a general guideline for the study of the genetic relationships of host-parasite systems in general.

Gene-for-gene relationships aid us in understanding the origin and source of parasitic variability and thus provide guidance in the deployment of R-genes in space and time and in synthesizing multigene and multiline cultivars. Also they can be used to study the mutability of R- and V-genes. Understanding the process of evolution of parasitism and of the R- and V-gene interaction is a major pursuit of researchers in the control of plant diseases. The little we currently know about the molecular interpretation of host resistance or parasite virulence is derived mainly from simple parasitic systems which readily show gene-for-gene relations (Albersheim and Anderson-Prouty, 1975; Callow, 1977).

VI. GENETICS OF COMPLEX PARASITIC SYSTEMS

Under natural conditions a plant is host to many incidental and true parasites and, hence, represents a miniature replica of a bigger ecological system. Different parasites on the same plant show inter- and intraspecific interactions which result in disease complexes, and these interactions may lead to susceptibility through predisposition, or resistance through preinduction of resistance against a particular parasite.

Plant-parasitic nematodes are known to predispose some plants to fungal pathogens (Bergeson, 1972; Sidhu and Webster, 1977). This type of interaction was genetically evaluated in the *Meloidogyne incognita-Fusarium oxysporum lycopersici* disease complex on tomato host (Sidhu and Webster, 1974). An F_2 progeny derived from a cross between tomato cultivars Small Fry-1 (resistant to both the pathogens) and Wonder Boy (susceptible to both the pathogens) was tested against each pathogen singly and against both the pathogens sequentially, i.e. the root-knot nematode followed by the wilt fungus. From single inoculations two independently segregating dominant R-genes were identified, one effective against the nematode and the other against the fungus. From the sequential inoculations of each F_2 plant genotype, a 9:3:4 ratio was obtained showing resistance to both, resistance to the nematode but susceptibility to the fungus, and susceptibility to both the pathogens, respectively. Such a ratio was possible due to the modification of the response of the F_2 plants which were genetically susceptible to the nematode but resistant to the fungus. As these plants were predisposed by the nematode to the fungus they also exhibited a susceptible reaction to the fungus.

A scheme depicting these modifications in the F_2 reaction classes due to sequential inoculations of each F_2 genotype, was illustrated earlier (Sidhu and Webster, 1974). It is interesting to note that if the F_2 progeny had been inoculated only with the wilt fungus without the knowledge of the presence of the root-knot nematode in the soil, the observed ratio probably would have been misinterpreted as 9:7 (R:S)—a characteristic of duplicate recessive epistasis. Modified epistatic ratios would also occur in those disease complexes where one parasite actually preinduces resistance against the other (Sidhu and Webster, 1979). The implications of such interactions have an important bearing on the practice of plant breeding against those organisms that form disease complexes (Sidhu and Webster, 1977).

Change in a genetic ratio obtained due to parasite-parasite interaction is not brought about by the corresponding change in the genome per se or by gene interaction in the interacting organisms. The modification is essentially biochemical and/or physiological in nature which eventually

influences the phenotypic response of the host to the parasite under study. As a result an epistatic genetic ratio occurs which suggests that there are two interacting R-genes—a situation which is genetically incorrect. Such a situation could mislead a plant breeder into incorporating R-gene(s), which do not exist in reality, into cultivars that subsequently would fail when exposed to a disease complex.

Nematode parasites probably influence the plant response to many soil borne fungal and bacterial parasites (Powell, 1979). Hence, breeding against nematodes, should be emphasized so as to achieve long term disease control of other parasites present in the rhizosphere. Shorter life spans of cultivars in relation to certain parasites may be due to predisposition rather than to the occurrence of virulent race or isolate with the relevant V-gene(s). Therefore, breeding against diseases warrants a new look at the complexity of parasitic associations as they are a common occurrence in nature.

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