Parasitic nematodes infect thousands of plant species, but some plants harbor specific resistance genes that defend against these pests. Several nematode resistance genes have been cloned in plants, and most resemble other plant resistance genes. Nematode resistance is generally characterized by host plant cell death near or at the feeding site of the endoparasitic worm. The timing and localization of the resistance response varies with the particular resistance gene and nematode interaction. Although there is genetic evidence that single genes in the nematode can determine whether a plant mounts a resistance response, cognate nematode effectors corresponding to a plant resistance gene have not been identified. However, recent progress in genetics and genomics of both plants and nematodes, and developments in RNA silencing strategies are improving our understanding of the molecular players in this complex interaction. In this article, we review the nature and mechanisms of plant–nematode interactions with respect to resistance in plants.

Introduction

The Nematode comprise the largest animal phylum in terms of numbers of individuals and arguably numbers of species [1]. Most species, including the model animal Caenorhabditis elegans, are free living. However, animal and plant parasitic nematodes have received much attention owing to the devastating disease and damage that they can cause [2,3]. Worldwide losses as a result of plant-parasitic nematode infection have been reported to amount to >$100 billion per year [4]. Although several different groups of nematodes are plant parasites, the most damaging are the root-knot nematodes (Meloidogyne spp.) and cyst nematodes (Heterodera and Globodera spp.) [5] (Box 1). These sophisticated soil-dwelling pests parasitize plant roots, usurping the nutritional resources of their hosts while evading or suppressing host defenses. Although nematicides have been successfully used to control nematodes, host resistance is a preferable alternative because of the expense and environmental toxicity of nematicides. To protect themselves from invading pathogens, hosts have evolved defense mechanisms including chemical and physical barriers and a highly specialized resistance (R) gene-mediated defense (Box 2). In recent years, it has been revealed that plants use similar R-gene-based resistance mechanisms to protect themselves from nematodes [6].

Structure of nematode resistance genes

Several nematode resistance (Nem-R) genes have been isolated from plants, all conferring resistance against sedentary endoparasites (Table 1). The first nematode resistance gene (see Glossary) to be cloned was Hs1pro-1 from sugar beet, which confers resistance against the sugar beet cyst nematode [7]. The encoded protein does not have obvious similarities to known plant genes. However, other cloned Nem-R genes closely resemble known plant R-genes in their domain structure (Figure 1 in Box 2). Four of these genes, Mi-1, Hero A, Gpa2 and Gro1-4, all cloned from tomato or potato relatives, fall into the NBS-LRR class of R-genes (Box 2). The tomato genes Mi-1 and Hero A confer broad-spectrum resistance against several root knot nematode species [8,9] and against several pathotypes of two potato cyst nematode species [10], respectively. By contrast, the potato genes Gpa2 [11] and Gro1-4 [12] confer resistance to a narrow range of pathotypes of a single potato cyst nematode species. Mi-1, Gpa2 and Hero A are members of the NBS-LRR class of plant R-genes that does not contain an N-terminal toll-interleukin receptor-like (TIR) domain. The Hero A gene product is 32% identical to Mi-1 and ~22% identical to Gpa2 at the amino acid level.

Glossary

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resistance gene (R-gene)</td>
<td>A plant gene encoding a specificity determinant for activation of the plant defense response to a pathogen.</td>
</tr>
<tr>
<td>Avirulence gene (Avr gene)</td>
<td>A pathogen gene that encodes a protein that is recognized, or produces a product or modification that is recognized, by the host with the corresponding R-gene to elicit a defense response.</td>
</tr>
<tr>
<td>Gene-for-gene interaction</td>
<td>The allele-specific interaction between a host R-gene and a pathogen Avr gene that leads to a host resistance response.</td>
</tr>
<tr>
<td>Avirulence</td>
<td>Inability of a pathogen to reproduce on a host containing a particular R-gene.</td>
</tr>
<tr>
<td>Virulence</td>
<td>Ability of a pathogen to reproduce on a host containing a particular R-gene.</td>
</tr>
<tr>
<td>Pathogenicity gene</td>
<td>A pathogen gene whose direct or indirect product has a role in successful interaction with the host.</td>
</tr>
<tr>
<td>Elicitor</td>
<td>A protein or other compound from the nematode that is recognized by the host R-gene.</td>
</tr>
<tr>
<td>Hypersensitive response (HR)</td>
<td>Localized programmed cell death that occurs after recognition of the pathogen by a specific R-gene.</td>
</tr>
<tr>
<td>Pathotype</td>
<td>For some cyst nematode species, a strain or isolate is defined by its qualitative or in some cases quantitative success or lack of success at increasing in number on a set of specific host genotypes each with a known complement of R-genes.</td>
</tr>
</tbody>
</table>

Corresponding authors: Williamson, V.M. (vmwilliamson@ucdavis.edu); Kumar, A. (akumar@scri.ac.uk). Available online 24 May 2006
Box 1. Life cycle of cyst and root-knot nematodes

Plant parasitic nematodes possess a specialized structure, called a stylet, a hollow, protrusible spear at their anterior end that is used to pierce plant cell walls. Secretions from the esophageal glands of the nematode are released through the stylet. It is also through the stylet that the nematode ingests cytoplasmic contents from plant cells. Cyst nematodes and root-knot nematodes are both sedentary endoparasites of plant roots and have similar life cycles (Figure I). Eggs hatch in soil as infective second stage juveniles (J2) (~0.4 mm long) [5]. Infective J2 penetrate the host root and then migrate towards the plant vascular system. In response to signals from the nematode, plant cells adjacent to the head of the nematode enlarge to form giant cells for root-knot or syncytia in the case of cyst nematodes, both large, multinucleate, metabolically active cells that serve as the source of nutrients for the developing endoparasite. For root-knot nematodes, swelling and cell division in the surrounding tissues leads to the formation of the galls or root-knots. Soon after feeding is initiated, nematodes begin to develop and become immobile. Adult females of both root-knot and cyst nematodes are bulbous and remain nonmotile, whereas males become vermiform, regain mobility and leave the root. Egg production begins 3–6 weeks after infection, depending on the species and conditions.

Figure I. The life cycle of a plant parasitic cyst nematode. Key stages in the life cycle of a cyst nematode female are shown in the center. (a) Infective second stage juvenile (J2) with stylet indicated by arrow (photo courtesy of J. D. Eisenback and E.C. McGawley; www.mactode.com). The J2 is ~0.4 mm in length. (b) The tail of a J2 (stained orange) can be seen as the nematode penetrates the plant root (adapted with permission from Ref. [33]). (c) An infected root, with a bulbous parasitic female (f), is shown on the left and an uninfected root is shown on the right (photo courtesy of J. D. Eisenback and U. Zunke, www.mactode.com). The elongated syncytiot (s) consists of plants cells that have fused together and serve as the feeding cell for the developing nematode. (d) Root sections with egg-filled cysts of Globodera rostochiensis (top root) and Globodera pallida (bottom root) appearing as golden or light colored globs, respectively, decorating the roots (photo courtesy of J. D. Eisenback and U. Zunke).

In both cases, the similarity is greatest in the NBS region, as is typical for R-genes. Mi-1 and Hero A share an extended N-terminal region in contrast to most other R-genes (Figure I in Box 2). Gpa2 has a much shorter N-terminal region than either Mi-1 or Hero A. Gro1-4 encodes a member of the subclass of NBS-LRR proteins characterized by a TIR N-terminal region [12].

Nem-R genes also include members of the class of pathogen R-genes encoding proteins with extracellular LRR motifs. In soybean, genetic analyses determined that two unlinked loci, Rgh1 and Rgh4, condition resistance to Heterodera glycines type 0 [13]. Rgh1 is necessary but not sufficient for resistance against all known biotypes. Rgh4 is necessary but not sufficient for resistance against H. glycines type 0. Candidate genes for Rgh1 and Rgh4 have been cloned and both encode putative proteins with extracellular LRRs, a transmembrane domain and a cytosolic serine–threonine kinase domain, structurally resembling the rice gene Xa21 that confers resistance against the bacterial pathogen Xanthomonas oryzae [14,15].

Evolutionary and functional relationships among R-genes

Plant R-genes generally belong to clustered multigene families ranging from a few to > 30 homologs [16]. This clustered organization might contribute to evolutionary changes in specificity of resistance, but our understanding of this process is limited [17]. Mi-1 and Hero A are found in clusters of seven and 14 homologous copies, respectively [10,18]. Gpa2, a Nem-R-gene, and Rx1, which confers resistance against potato virus X, are in the same cluster of four homologous NBS-LRR genes on chromosome 12 in potato [11]. These genes are 88% identical in amino acid sequence, but confer resistance to unrelated pathogens. Similarly, although the tomato Mi-1 and Hero A genes share an extended N-terminus, this motif has been noted in other R-genes in Solanaceous plants including the gene Rpi-b1b2 from wild potato Solanum bulbocastanum, which confers broad resistance to the oomycte Phytophthora infestans. [19]. In fact, Rpi-b1b2, which is positioned in the syntenic position to Mi-1, is its closest homolog with known function sharing 82% amino acid sequence identity. Conversely, although potato Gro1 and tomato Hero A both confer resistance against pathotype Ro1 of Globodera rostochiensis, the two genes occupy different positions in the highly syntenic potato and tomato genomes [20] and share little sequence similarity. This suggests that they recognize the presence of different nematode avirulence factors and also trigger different signaling pathways. The molecular and evolutionary mechanisms that produce the diverse recognition capabilities of R-genes are a matter of current debate [16,21,22].

Many Nem-R genes have been noted phenotypically and mapped genetically (Table 1). Both H1 and Mi-3 have been fine mapped with BACs and should be cloned in the near future [23,24]. Mi-9 maps to the same gene cluster as Mi-1, but functions at greater temperatures than does Mi-1 [25]. RNA silencing experiments suggest this gene is a homolog of Mi-1 (I. Kaloshian, personal communication). The wheat Nem-R genes Cre1 and Cre2 co-localize with clusters of NBS-LRR genes [26], but functional confirmation has not been completed. Unfortunately, although Arabidopsis is a host of several nematode species [27], no nematode resistance genes have been described in this species, limiting the utility of this powerful system for characterization of nematode resistance.

Mi-1 also confers resistance against specific isolates of the potato aphid Macrosiphum euphorbiarum [28] and against the white fly, Bemisia tabaci [29]. Although nematodes and piercing-sucking insects such as aphids
Box 2. Resistance genes and the plant innate immune response

Plants in agricultural settings and in their native environments are attacked by a wide variety of pathogens and pests. Nevertheless, most plants are resistant to most pathogens owing to an array of active and passive defenses [70]. As a central component of their innate immune system, plants have a repertoire of resistance genes (R-genes) whose products serve as surveillance proteins to protect them from specific pathogens (or in some cases specific strains of a pathogen) including viruses, bacteria and fungi [21,22,71]. Following recognition of the presence of a specific pathogen-produced effector, the product of what is sometimes called an avirulence (Avr) gene, the cognate R-gene initiates an array of defense responses typically including a rapid, localized cell death, or HR at the site of infection. Because the response requires both the presence of a specific gene in the pathogen and of the corresponding gene in the host, this type of resistance has been dubbed ‘gene-for-gene’. More than 40 R-genes from various plant species have been cloned [21]. Most of these genes encode proteins having a domain structure that includes a central conserved region with a nucleotide-binding site (NBS) and a C-terminal leucine rich repeat (LRR) region (Figure I). For some NBS-LRR R-proteins the N-terminal region contains a Toll-interleukin receptor (TIR)-like domain. Other R-genes are characterized by a predicted coiled coil (CC) domain N-terminal to the NBS region. LRR motifs typically are involved in protein-protein interactions, and for R-genes there is evidence that they have roles in pathogen recognition and signal transduction [22]. The NBS is part of a larger conserved region of ~320 amino acids present in a large class of proteins that mediate recognition of specific molecules and undergo conformational changes leading to signaling of a response [22]. NBS-LRR proteins are predicted to be cytoplasmic or membrane-associated. A second broad class of R-genes encodes proteins with an extracellular LRR, a membrane-spanning region and, in some cases, a cytoplasmic kinase domain. These genes are thought to be receptors for extracellular elicitors.

Avr genes have been cloned in bacteria, viruses and fungi [22]. Although there are several examples in which the cognate R-gene and Avr genes are available, direct interaction has been detected in only a few cases [22]. This has led to a model in which Avr proteins modify host products, and these modifications are recognized by the R-genes that ‘guard’ the host products [71]. Several bacterial Avr genes have been shown to have roles in counteracting host defense [70]. We are just beginning to understand how R-gene products recognize pathogens and initiate defense signals and which downstream responses lead to resistance [70].

Figure I. A comparison of predicted protein structure of the cloned nematode resistance gene products. The five protein structure classes of plant Nem-R genes are shown in their predicted cellular location. The yellow line represents the cell membrane and the grey area represents the cytoplasm. The Nem-R genes Mi-1, Hero A, Gpa2 and Gro1-4 are predicted to be cytoplasmic, whereas Rhg1, Rhg4 and Hs1pro-1 are predicted to span the cell membrane. Protein motifs are shown in the key (upper left). Abbreviations: LR indicates a leucine rich region; TM, a transmembrane domain; LRR, a leucine rich repeat region; kinase, a protein kinase domain; NBS, a conserved motif containing a nucleotide binding site; TIR, toll-interleukin receptor-like domain; NT-1, amino terminal domain of NBS-LRR genes with long N-terminal regions; NT2, region immediately N-terminal to the NBS region of non-TIR NBS-LRR genes. The motif containing the letter N inside is at the amino terminus of the protein.

and whiteflies are evolutionarily distant, there are similarities in how they feed and in the defense responses that they induce in plants [30,31]. Although Mi-1 is the only cloned insect R-gene, there is evidence that similar genes, which recognize specific effectors to mediate resistance against piercing-sucking insects, do exist in a variety of plant species [30]. Recently an aphid resistance gene in *Medicago truncatula* was mapped to a single locus flanked by NBS-LRR resistance gene analogs [31].

Resistance phenotypes

Resistance to nematodes in plants is generally characterized by failure of the nematodes to produce functional feeding sites in the host after invasion and to develop subsequently as reproducing females. The production of a localized hypersensitive response (HR) and the signals induced in the host following nematode infection in resistant interactions so far seem similar to those induced by other pathogens [32,33]. However, the timing and localization of the response varies with the particular Nem-R gene–nematode interaction. For example, *Mi-1* mediated resistance is characterized by a rapid localized cell death that occurs near the anterior end of the nematode in the region of the root where feeding site initiation occurs [6]. In nematode-resistant tomatoes, neither the feeding site nor the nematodes develop.
However, for other interactions such as *Hero A*-mediated resistance, the response seems to be initiated after feeding site induction and leads to atrophy or abnormal development of the feeding site [33]. The nematodes that do develop are mostly males, resulting in severe reduction in reproduction of the invading nematodes. Resistance coupled with a high ratio of males to females is commonly observed against cyst-forming nematodes (Ref. [33] and references therein). In another example, the pepper genes *Me1* and *Me3* each control the main root-knot nematode species that affect this plant, but the HR in response to nematode infection occurs much earlier in the interaction in the presence of *Me3* than for *Me1* [34]. Whether these timing differences reflect when the nematode elicitor is recognized or the timing or pathway of the response remains to be determined.

### Nem-R gene expression, recognition and signal transduction

Most *R*-genes are constitutively and ubiquitously expressed in the plant, but the level of expression is typically low. This constitutive expression is consistent with the hypothesis that *R*-genes encode surveillance proteins that detect effector molecules from pathogens directly or indirectly and initiate an effective defense response. *Mi-1* seems to be constitutively expressed [35,36], whereas *Hero A* is expressed constitutively in all tissues but is up-regulated in roots in response to potato cyst nematode infection [33]. Experiments with fusions of the *Hs1pro-1* promoter to a reporter gene indicate that expression of *Hs1pro-1* also increases after nematode infection, specifically in the nematode feeding sites [37]. Perhaps up-regulation of some Nem-*R* genes is required for maintaining or enhancing induction of the signal transduction pathways leading to resistance.

The role of a Nem-*R* protein in perceiving and signaling resistance has been most studied for *Mi-1*. A series of *in vitro* mutagenesis experiments exchanging sequences of *Mi-1.2*, the functional copy of *Mi-1*, with its closely linked, nonfunctional paralog *Mi-1.1* and transforming plants with the resulting constructs under the control of their endogenous promoters resulted in phenotypes ranging from loss of function to constitutive lethality [38,39]. Comparisons of phenotypes of mutants suggested a model in which the N-terminal 161 amino acids of *Mi-1.2* repress the defense response mediated by the other parts of the protein. In this model, the presence of a specific elicitor produced directly or indirectly by the nematode, causes a conformational change in the Nem-*R* protein that results in the signaling of a defense response.

Information about the defense-response signaling leading to nematode resistance is limited, and perhaps only a subset of the responses initiated by pathogen recognition in the presence of the nematode are responsible for resistance. For example, although HR is frequently observed, it is unknown whether this response is a cause or consequence of nematode resistance. In fact for some plant–pathogen resistance responses, HR and resistance have been decoupled [40]. Salicylic acid (SA) has been shown to be required for resistance mediated by several *R*-genes [21]. Expression of the bacterial gene *nahG*, which encodes an enzyme that degrades SA to catechol, in *Mi-1* tomato roots results in loss of resistance indicating that SA is required for signaling the resistance response [32]. The tomato homolog of an *Arabidopsis* gene *EDS1* has been shown to be required for resistance mediated by some resistance genes in tomato, but not for *Mi*-mediated nematode resistance [41]. This is consistent with previous findings that show different *R*-genes act through different signaling pathways [21]. Although differential expression studies have identified genes induced after nematode infection in resistant plants, a function for these genes in resistance has not been confirmed [42,43]. Recent successes in silencing gene expression in roots have provided a new tool to examine
the role of specific genes in the resistance response [44,45] (I. Kaloshian, personal communication).

Little is known about how the presence of specific nematodes is recognized by Nem-\(R\) genes. Whether a molecule from the nematode is recognized directly or by its effect on a host product is not known. There is specificity within each pest species for \(Mi-1\)-mediated resistance; that is, for each of the nematode species and the insect species against which \(Mi-1\) is effective, there are strains that can escape recognition. One explanation is that \(Mi-1\) is capable of recognizing two or more elicitors, each specific to a different pest or pest group. It is also possible that \(Mi-1\) recognizes a plant product modified by both pests. Screening a mutagenized tomato population identified a tomato gene, \(Rme-1\) that is unlinked to \(Mi-1\), but is required for nematode, aphid and whitefly resistance mediated by \(Mi-1\) [46]. \(Rme-1\) is not required for the function of other resistance genes tested and seems to act early in the signaling pathway, probably upstream of \(Mi-1\). These properties make the \(Rme-1\) product a good candidate for the plant product targeted by the nematode (aphid and whitefly) product and guarded by \(Mi-1\). Cloning of \(Rme-1\) will be necessary to test this hypothesis.

Function of Nem-\(R\)-genes in heterologous plants

A practical goal of isolating Nem-\(R\) genes is to transfer these genes to economically important crop plants where resistance is not available. Transgene-based resistance against severely damaging nematodes will have a clear benefit of reduced use of highly toxic nematicides [47]. So far intraspecific transfer of Nem-\(R\) genes by transgenic techniques has been successful, but there has been limited success in transferring these genes to new species. For example, \(Mi-1\) confers effective resistance when transferred into susceptible tomato, but it does not confer resistance against the same nematode when introduced into tobacco or \(Arabidopsis\) (V.M. Williamson et al., unpublished). Interestingly, introduction of \(Mi-1\) confers nematode resistance, but not aphid resistance, to eggplant [48]. Furthermore, transfer of the tomato \(Hera\) A gene (which confers resistance to potato cyst nematode) into potato did not result in resistance to potato cyst nematodes [33]. Perhaps in the foreign host the \(R\)-gene does not have available other gene products that are required for recognition of the pathogen or signaling the defense response. Even within cultivated tomato, genotype differences have an effect on the efficacy of \(Mi-1\) [49]. Understanding this phenomenon will be necessary for successful transfer of nematode resistance to new species and might also provide insights into host factors mediating specificity of recognition or signaling.

Nematode genes involved in Nem-\(R\) gene mediated resistance

Nematode strains that can reproduce (i.e. that are virulent) on hosts carrying specific Nem-\(R\) genes have been identified for many interactions [50]. Virulence could be due to lack of or modification to the nematode gene product whose presence alerts that plant by way of the Nem-\(R\) gene. Alternatively, virulence could be due to a gain of function enabling the nematode to circumvent the host resistance response of the host, for example, by producing antioxidants or altering the hormone balance to compromise the defense response [50,51]. Several strains of root-knot nematode that can reproduce on tomato with \(Mi-1\) have been identified in field isolates and after greenhouse selection [50]. The search for an avirulence gene corresponding to \(Mi-1\) has been hampered by the lack of sexual reproduction in nematodes that respond to \(Mi-1\). Candidate \(Ave\) genes have been identified by differential expression analyses with transcripts from closely related strains of nematodes that differ in virulence [52,53] (C. Gleason, PhD thesis, University of California, Davis, 2003). However, a role for these genes in the interaction has not been confirmed.

Many genes encoding products that are secreted from plant parasitic nematodes and thousands of nematode ESTs have been identified, providing a rich source of candidate avirulence and pathogenicity genes [2,51]. However, functional analysis of these genes has been limited in part by the small size and endoparasitic lifestyle of plant parasitic nematodes. Blocking gene expression by RNA interference (RNAi) has been widely used to investigate gene function in \(C. elegans\), and recent experiments indicate that targeted plant parasitic nematode genes can be silenced by soaking infective juveniles in double stranded RNA corresponding to that gene [54–56]. Because plant parasitic nematodes normally feed only on the cytoplasm of living plant cells, chemical stimuli are required for uptake of double stranded (ds) RNA from solution. Scoring the phenotype or assaying gene expression in individual worms is problematic because they are buried within the roots of their hosts. In addition, gene silencing seems to be short-lived, unlike the cycle of the nematode. Despite these limitations, RNAi is currently the most promising avenue for functional analysis. On the positive side, RNAi can be particularly useful for identification of an avirulence gene because silencing such a gene should enable the nematode to evade detection by the corresponding resistance gene and reproduce in resistant plants, providing a positive selection system. Although the silencing might be short-lived, for the \(Mi-1\) gene at least, resistance is determined within a few days of infection.

Although classical genetic studies are not possible on some nematodes due to the lack of sexual reproduction, other nematodes do reproduce sexually (Table 2). Most cyst nematodes reproduce by obligate outcrossing, and there is generally great variation in host range and response to specific resistance genes between and within field populations [57,58]. Controlled crosses using inbred populations of the potato cyst nematode \(G. rostochiensis\) indicated that a single recessive gene determines the ability of the nematode to reproduce on potato with the \(H1\) resistance gene [59]. Crosses between inbred lines of the soybean cyst nematode have identified mendelian segregation of both dominant and recessive traits that control the ability to reproduce on nematode-resistant soybean [60]. Genetic linkage maps using amplified fragment length polymorphism (AFLP)-based DNA markers have been produced for potato cyst and soybean cyst nematodes [61,62]. However, the requirement of separate sexes to
produce progeny and the tiny size of the organisms limits the potential to test markers and virulence phenotype of individual progeny.

The unusual reproductive strategies of some root-knot nematode species provide the potential for both sexual and asexual reproduction. Many strains of *Meloidogyne hapla* can produce progeny by both asexual reproduction (meiotic parthenogenesis) and sexual reproduction [63]. This situation enables production of both segregating populations and inbred lines. DNA polymorphisms are abundant between strains of this species and molecular markers have been used to monitor genetic crosses and to produce F2 lines [64] (Q.L. Liu and V.M. Williamson, unpublished). Isolates of *M. hapla* also differ in ability to reproduce on specific plant species and cultivars. For example, some isolates are virulent and others are avirulent on the common bean (*Phaseolus vulgaris*) cultivar NemaS-nap, which carries a single, dominant gene for *M. hapla* resistance [65]. Analysis of segregation of virulence in progeny of a controlled cross indicates that virulence in the nematode is inherited as a single recessive trait, and that the nematode-bean interaction might be classified as a gene-for-gene type interaction. A genetic map with molecular and phenotypic markers is under construction and sequence analysis of the genome of *M. hapla* is in progress [3] (Q.L. Liu and V.A. Williamson, unpublished). Together these efforts have made *M. hapla* an excellent candidate as a model plant parasitic nematode for functional genomic analyses.

Plant parasitic nematodes have small genomes (~100 Mb), similar in size to that of the model nematode *C. elegans*. In addition to the ongoing genetic and genomic analysis of *M. hapla*, programs to sequence the genomes of *H. glycines*, *G. pallida* and *M. incognita* have been initiated or are planned (K. Lambert, J. Jones, P. Abad, personal communication). When these genomes are sequenced, it should soon be possible to map and clone nematode genes that have roles in virulence and pathogenicity.

**Concluding remarks**

During the past decade there has been significant progress in the molecular and genetic characterization of plant-nematode interactions. Identification of several nematode resistance genes has provided insights into possible mechanisms for achieving a resistance phenotype. RNAi technology is a particularly promising tool for dissecting virulence traits in the nematode and the resistance pathway in the host. Rapid progress in genomics and genetics of both plants and nematodes will improve our understanding of plant-nematode interactions. In addition, identification of both plant and nematode genes involved in key stages of incompatible plant-nematode interactions will provide the opportunity for developing novel resistance strategies for durable nematode resistance in crop plants.

**Acknowledgements**

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**Table 2. Genetics and genomics of plant parasitic nematodes**

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<th>Reproductive mode</th>
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<td>Not available</td>
<td>23 117</td>
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*Data from the NCBI-EST database, December 2005.*


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4. Place the cursor over the image and click to engage the ‘Enlarge Image’ option
5. On a PC, right-click over the expanded image and select ‘Copy’ from pull-down menu (Mac users: hold left button down and then select the ‘Copy image’ option)
6. Open a blank slide in PowerPoint or other image-display program
7. Right-click over the slide and select ‘paste’ (Mac users hit ‘Apple-V’ or select the ‘Edit-Paste’ pull-down option).

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