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Nematode Constraints of Chickpea and Pigeonpea Production in the Semiarid Tropics

Chickpea (*Cicer arietinum* L.) and pigeonpea (*Cajanus cajan* (L.) Millsp.) are the principal grain legumes (pulses) of subsistence farming systems in the Indian subcontinent and in other regions of the semiarid tropics. These crops are important both in management of soil fertility in the traditional farming systems and as sources of protein in the largely cereal-based diets of the people in the semiarid tropics.

Chickpea is grown in 33 countries, with about 5.6 million t produced on nearly 9.6 million ha (17). Asia accounts for 83% of the world production of chickpea, a major grain legume in Algeria, Ethiopia, India, Iran, Mexico, Morocco, Myanmar, Pakistan, Spain, Syria, Tanzania, Tunisia, and Turkey. Chickpea is grown as a post-rainy season "winter" crop in the Indian subcontinent. Cultivars adapted to peninsular India mature within 110 days, whereas those adapted to northern India require 170 days. The desi type (small seeds of various colors) is mostly grown in the Indian subcontinent and is consumed as flour, dhal (split pea), and whole seed; desi is also used to some extent as animal feed. The kabuli type (large beige seeds) is grown in the Mediterranean region, the Americas, and, to some extent, the Indian subcontinent; kabuli is usually consumed as whole seed.

Pigeonpea is grown in more than 25 tropical and subtropical countries, with 2.4 million t produced annually on more than 3.4 million ha (28). Reliable yield data are scarce in many regions, however, because pigeonpea is rarely grown as a field crop outside of South Asia and eastern and southern Africa.

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Pigeonpea is widely adapted to tropical conditions and, with its drought tolerance and deep root system, grows well in semiarid regions. In India, pigeonpea land races and cultivars sown in June or July when the rainy season begins develop into woody shrubs that flower and set pods in the post-rainy season. Cultivars are classified as early (3–5 months), medium (5–6 months), and late (6–9 months) maturing. Approximately 90% of world pigeonpea production is in the Indian subcontinent, where it is mainly consumed as dhal. In other countries in Asia, Africa, and the Americas, pigeonpea is grown for green or dry seed, usually in low-input systems with low yields.

Many fungi, bacteria, viruses, plant-parasitic nematodes, and mycoplasma-like organisms attack chickpea and pigeonpea. Although plant-parasitic nematodes have been associated with chickpea in 17 countries and with pigeonpea in 24 countries (29), only a few diseases caused by nematodes are recognized as important production constraints (Table 1). Yields are reduced when nematodes attack plant roots and *Rhizobium* nodules (cowpea miscellany group and *Cicer* group). Although usually not dramatic, yield losses tend to be cumulative, and production is reduced over extended periods (44). Unfortunately, very few chickpea and pigeonpea growers know how to recognize and manage nematode-caused diseases. The need for programs to educate growers about nematode problems and about the inexpensive, environmentally safe, effective controls for nematode diseases is especially acute in the tropics.

Nematode Diseases of Chickpea

Root knot. Root knot, caused by *Meloidogyne* spp. is the most serious nematode disease of chickpea (Fig. 1). *M. incognita* (Kofoid & White) Chitwood and *M. javanica* (Treub) Chitwood in the Indian subcontinent and *M. artiellia* Franklin in the Mediterranean region are the most damaging species (8,47). *M. incognita* and *M. javanica* are favored by warm weather and become serious problems in regions where winters are mild, as in peninsular India. However, severe crop damage also occurs in northern India, in the terai (submontane) region of Nepal, and in Pakistan, where minimum air temperature during the winter crop season is less than 15 C for many days. *M. incognita* and *M. javanica* infest various types of soils but cause the most plant damage in sandy and sandy loam soils. In northern India, Upadhyay and Dwivedi (57) reported a 40% increase in yield when plots infested with 4.6 *M. incognita* juveniles per cubic centimeter of soil were treated with carbofuran. *M. incognita* and *M. javanica* have a very wide host range and in India attack plant species in more than 232 and 141 genera, respectively (22).

Symptoms and disease cycle. Root galls (knots) are the most characteristic symptoms of nematode infection and are easily seen with the unaided eye. Gall size is influenced by soil temperature and susceptibility of the chickpea genotype. Galls produced at 25–30 C are 30–35% larger than those produced at 15–20 C. Galls are formed on the taproot and lateral roots but may be more numerous on the taproot. Aerial parts show no characteristic symptoms, but nematodes reduce plant vigor, delay flowering, and induce early senescence—symptoms that are often confused with decreasing soil fertility and deficiencies of nitrogen, iron, magnesium, sulfur, phosphorus, potassium, and other nutrients (Fig. 2).

Symptom expression and tolerance to nematode population densities vary with genotype. In nematode-infested chickpea fields, patches of stunted plants usually appear earlier in infertile, moisture-deficient sandy soils with low pH. Tolerance limits (number of nematodes a plant can host without measurable damage) vary from 0.2 to 2.0 eggs and/or second-stage juveniles per cubic centimeter of

soil at the time of sowing.

The life cycle of root-knot nematodes is similar on chickpea and most other hosts. Second-stage juveniles of *Meloidogyne* spp. are infective. They invade roots, penetrate the cortex, and become established in the vascular cylinder, where they induce formation of the giant cells on which they feed. Galls are initiated within 48 hours after infection but are not essential for nematode growth and development. Juveniles feed and begin to grow slightly longer and much wider, undergoing three molts to become adults. Males are vermiform and females are pyriform. Females deposit 300–1,000 eggs in a gelatinous matrix, and infective second-stage juveniles hatch from the eggs. *M. javanica* and *M. incognita* have a life cycle of approximately 1 month at optimum temperatures of 25–30 C, and many generations are completed in a crop season.

Pathogen interactions. M. incognita and *M. javanica* interfere with nitrogen-fixation and suppress the formation of *Rhizobium* nodules in chickpea cultivars JG 62, K 850, and JG 74. Association of these nematodes with *Fusarium oxysporum* f. sp. *ciceri* advanced the onset of Fusarium wilt from 31 to 16 days after seedling emergence in chickpea genotypes and increased the disease incidence from 25 to 56% (27). However, host genotype influences the extent of interactions between nematode species and the wilt fungus. Vascular discoloration after fungus infection does not extend beyond the collar region in wilt-resistant genotypes. Coinfection with *M. javanica* does not modify the reaction of most such genotypes (e.g., ICC 11311, 11313, 12245), but in some (e.g., ICC 11319), discoloration extends beyond the collar region. Also, the nematode moderates wilt resistance in the cultivar ICC 12275. *M. javanica* modifies the resistance of chickpea cv. Avrodhi to *F. oxysporum* at Kanpur in northern India (58) but not to race 1 at Patancheru in southern India. Susceptible cultivars die earlier from wilt when coinfected with nematodes.

Root-knot nematodes also interact with other species of *Fusarium* and with species of *Glomus, Rhizoctonia*, and *Sclerotium*. These fungi reduce the population densities of the nematodes (4,6,7, 24,26,27,32,55).

Management options. Population densities of parasitic nematodes on chickpea are reduced by soil solarization during summer months (51) and by soil application of aldicarb, carbofuran, fenamiphos, and phorate (1–4 kg a.i./ha) (10,34). Seed treatment with these biocides (1–6%, w/w) is also effective (18,23). More than 250 chickpea genotypes have been reported as resistant to root-knot nematodes in India (43), but later evaluations have not confirmed such resistance. At the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), all of 1,000 chickpea genotypes and 35 accessions of wild species of *Cicer* evaluated for resistance to *M. javanica* were susceptible and the cultivars Bheema, N 31, and N 59 were tolerant. Sesame, mustard, and winter cereals are poor hosts of *M. javanica* and *M. incognita*, and 2- to 3-year rotations may be useful for disease management.

Root lesion. Root lesions caused by *Pratylenchus* spp. are widespread. *P. thornei* Sher & Allen damages chickpea in India and Syria, and *P. brachyurus* (Godfrey) Goodey is important in Brazil (8,47). The nematodes penetrate roots and move within the cortical parenchyma. While moving from cell to cell, the nematodes create large cavities and cause necrosis of tissues. Infection is characterized by dark brown to black lesions on the roots (Fig. 3). A population of 0.1 nematode per cubic centimeter of soil significantly reduced plant height, shoot mass, and number of *Rhizobium* nodules (59), and more than 8.0 nematodes per cubic centimeter of soil reduced seed germination by 34% and caused a 10% seedling mortality of chickpea cultivar BG 203 (60). Damage caused by root-lesion nematodes usually is less evident than that caused by root-knot nematodes.

Management options. Information is limited on the management of root-lesion disease of chickpea. Chemicals that control root knot are also effective against *Pratylenchus* spp. The very wide host range of *P. thornei* limits options for rotations (8). In preliminary tests in central India, chickpea genotypes ICC 11315, 11323, 12233, 12239, 12242, 12245, 12253, 12269, 12270, and 12275 showed resistance to *P. thornei* (1).

Nematode Diseases of Pigeonpea

Pearly root. Pearly root of pigeonpea caused by *Heterodera cajani* Koshy (Fig. 4) exists in the major pigeonpea-producing states of Andhra Pradesh, Bihar, Gujarat, Haryana, Karnataka, Maharashtra, Punjab, Rajasthan, Tamil Nadu, and Uttar Pradesh in India and in some areas of Egypt. The nematode is widespread in sandy loam soils in northern India and in black-cotton soils (Vertisols) in southern India. Its host range is limited and largely confined to species of the Leguminosae. Of 21 host plant species, only *Sesamum indicum* (Pedaliaceae) and *Phyllanthus maderaspatensis* (Euphorbiaceae) are non-legumes (20,53). Nematode infection reduces foliage production and grain yield (40). At sowing time, population densities of more than 2.0 eggs and juveniles per cubic centimeter of soil may

Table 1. Important nematode-caused diseases of chickpea and pigeonpea

Crop	Annual yield loss[a] %	Annual yield loss[a] U.S.$	Disease	Causal nematode	Distribution
Chickpea	13.7	328 million	Root knot	*Meloidogyne artiellia*[b]	Italy, Spain, Syria
				M. incognita	Bangladesh, Brazil, Ethiopia, India, Nepal, Pakistan
				M. javanica	Bangladesh, Brazil, India, Nepal, Pakistan, Zimbabwe
			Pearly root	*Heterodera ciceri*[b]	Syria
			Root lesion	*Pratylenchus brachyurus*	Brazil
				P. thornei	Australia, India, Syria
Pigeonpea	13.2	177 million	Pearly root	*Heterodera cajani*	Egypt, India
			Dirty root	*Rotylenchulus reniformis*	Fiji, India, Jamaica, Puerto Rico, Trinidad
			Root knot	*Meloidogyne incognita*	Australia, Bangladesh, Egypt, India, Malawi, Nepal, Trinidad Uganda, United States
				M. javanica	Australia, Bangladesh, Brazil, India, Kenya, Malawi, Nepal, Puerto Rico, Zambia, Zimbabwe

[a] Yield loss estimated on world basis (37); dollar loss calculated with 1989 crop prices.
[b] Not important in the tropics.

Fig. 1. Chickpea with root knots (galls) caused by *Meloidogyne javanica*.

Fig. 2. Low vigor and uneven growth of chickpea planted in sandy soil in Nepal infested with root-knot nematodes.

Fig. 3. Chickpea with root-lesion disease caused by *Pratylenchus* spp.

Fig. 4. Pigeonpea with pearly root caused by *Heterodera cajani*. The small, pearllike bodies are female nematodes.

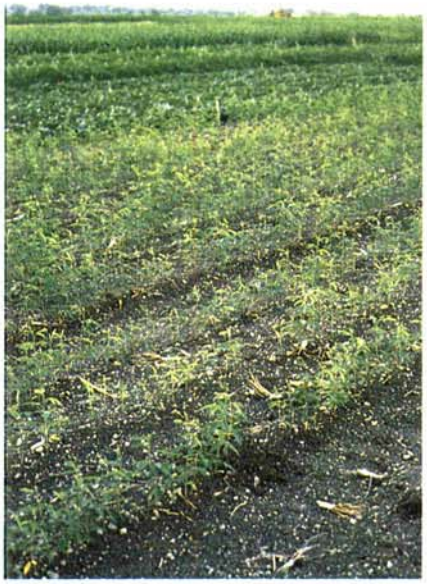

Fig. 5. Sparse, yellow foliage and stunted growth of pigeonpea planted in a field infested with *Heterodera cajani*.

Fig. 6. Pigeonpea genotype ICP 2376 (left) with no *Heterodera cajani* added to pot and with (center) 500 and (right) 5,000 juveniles added per pot.

Fig. 7. Flowering and podding of pigeonpea genotype ICPL 87 in soil (two rows at left) infested and (two rows at right) not infested with *Heterodera cajani*.

Fig. 8. Pigeonpea (left) with dirty root caused by *Rotylenchulus reniformis* compared with (right) healthy plant.

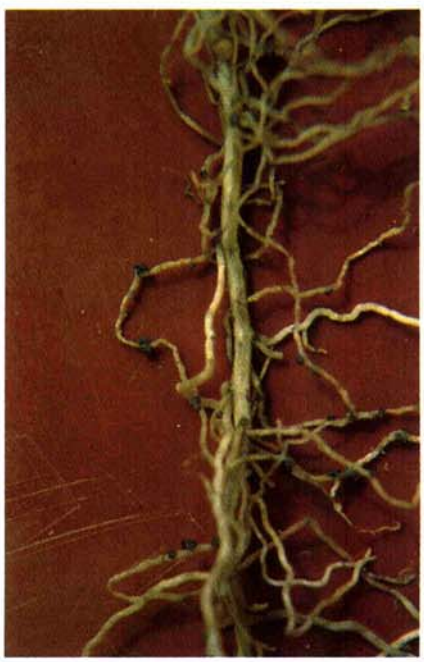

Fig. 9. Egg sacs of *Rotylenchulus reniformis* on pigeonpea roots stained with trypan blue.

Fig. 10. Growth of pigeonpea in plots infested with *Rotylenchulus reniformis*: (A) Untreated and (B) treated with carbofuran.

cause a 30% reduction in plant biomass and seed yield. Population densities of *H. cajani* reach very high levels on late-maturing pigeonpea cultivars, which are greatly affected because the nematode has multiple generations in each crop season (19).

Symptoms and disease cycle. The first symptoms of *H. cajani* infestation are the poor growth and stunted appearance of plants (Figs. 5 and 6), evident 30–45 days after sowing. The roots of 30- to 45-day-old infected seedlings bear many pearly white females of *H. cajani*; the females turn brown as they mature.

The nematode feeds endoparasitically in the stelar region (21). Females enlarge and damage the cortex and epidermis; widespread rupture and discontinuity of xylem vessels ensue. The life cycle is completed within 16 days at 29 C, and many generations are completed during the crop season, particularly on late-maturing pigeonpea genotypes. Nematode infestation delays flowering and pod formation for more than a week (Fig. 7).

Females of *H. cajani* lay eggs in egg sacs and within the body; the dead female body is referred to as a cyst. Infective second-stage juveniles emerge from egg sacs and cysts at 20–35 C. Emergence of juveniles from cysts is temperature-dependent, with 28 C the optimum; emergence from egg sacs is not as temperature-sensitive. More than 80% of juveniles emerge from egg sacs, 52% from white cysts, and 35% from brown cysts in 15 days at 28 C (53). Few juveniles undergo dormancy in the cyst; in the absence of a host, the nematode can survive for many years in the cyst. In Vertisols, summer fallow (February–June) reduces the number of eggs and juveniles by 18% at depths of 0–15 cm and by 11% at depths of 15–30 cm (52).

Pathogen interactions. H. cajani enhances the aggressiveness of *Fusarium udum* in wilt-susceptible pigeonpea (e.g., ICP 2376) but not in wilt-tolerant (e.g., BDN 1) or wilt-resistant (e.g., ICP 8863) genotypes. The fungus is antagonistic to the nematode population, however. Nematode infection suppresses *Rhizobium* nodulation (5,11,49).

Management options. Application of aldicarb, carbofuran, fensulfothion, and phorate to the soil (1.5–3.0 kg a.i./ha) and to seed (0.5–2.0%) reduces population densities of the nematode (9,16,61). Cereals are nonhosts of *H. cajani*. Sharma and Swarup (53) reported that *Echinochloa colona, Paspalum scrobiculatum, Setaria italica*, and *Zea mays* are nonhosts. Rotations with commonly grown cultivars of sorghum, pearl millet, cotton, groundnut, castor, maize, and rice for 2–3 years may suppress the deleterious effect of *H. cajani* on pigeonpea. Although host plant resistance in pigeonpea germ plasm has not been explored, accessions of *Cajanus scarabaeoides* are resistant to *H. cajani* and are easily crossed with pigeonpea (39). At ICRISAT, all of 400 pigeonpea genotypes evaluated for resistance to *H. cajani* were susceptible. Soil solarization reduces numbers of *H. cajani* and enhances yield of pigeonpea (51). *Pasteuria penetrans* infects *H. cajani*, and infected second-stage juveniles form a loosely woven "sticky swarm" in water (41). The bacterial infection completely disintegrates the internal tissues of developing nematodes, and infected nematodes do not produce eggs. Several other biocontrol agents, including *Allomyces anomalus, Catenaria auxiliaris, C. vermicola, Nematophthora* sp., *Olpidium* sp., and *Pythium* sp., reduce numbers of eggs and juveniles in soil (42).

Dirty root. Dirty root of pigeonpea is caused by *Rotylenchulus reniformis* Linford & Oliveira. This nematode attacks many crops in 38 countries in subtropical and tropical regions (14), and its widespread distribution endangers pigeonpea wherever the crop is grown. The extensive host range includes fruits, vegetables, legumes, oilseeds, ornamentals, millets, and plantation crops. In Fiji, where pigeonpea is a major subsistence and cash crop, the nematode severely reduces yield (13). The nematode is associated with variable growth of pigeonpea in northern India and on sandy and red soils (Alfisols) in western and southern India. A preplant population density of 1.0 *R. reniformis* per cubic centimeter of soil can significantly reduce biomass of susceptible pigeonpea cultivars. Damage thresholds range from 1.0 to 4.0 nematodes per cubic centimeter of soil, depending on soil type and climatic factors (47,48).

Symptoms and disease cycle. Dirty root can be diagnosed by observing *R. reniformis* egg sacs on pigeonpea roots. Infected roots appear dirty because soil particles adhere to the mucilaginous egg sacs (Fig. 8) and are not easily dislodged by shaking the roots. Foliage of nematode-infected plants is light green, and young leaves of many infected genotypes become yellow. As with pearly root, aerial parts show no diagnostic symptoms of nematode attack. Patches of stunted plants indicate nematode infection, and the number of such patches increases under drought stress.

The life cycle of *R. reniformis* on pigeonpea and other hosts is completed within a month. Sedentary females lay eggs in sacs. The first molt occurs within the eggs, and second-stage juveniles hatch. Juveniles can survive in the absence of hosts for more than 300 days without losing infectivity (52). Preadult females are infective and penetrate the epidermal cells intercellularly and intracellularly, causing slight browning and necrosis of surrounding cortical cells as they feed in the phloem. Females begin to enlarge on the ventral side around the vulval region, continue to swell, and become reniform within 5 days after infection. Males do not feed.

Pathogen interactions. R. reniformis can feed on and reduce the number of *Rhizobium* nodules. Interactions between *R. reniformis* and *F. udum* have been observed in India and Fiji (47). Although *F. udum* reduces the population density of the nematode, Fusarium wilt–susceptible genotypes such as ICP 2376 die early when the nematode and the fungus are both present in the soil. Reactions of wilt-tolerant (BDN 1) and wilt-resistant (ICP 8863) genotypes are not modified by nematode parasitism (50).

Management options. Chloris gayana, Crotalaria spp., *Tagetes erecta*, and *T. patula* are poor hosts of *R. reniformis* (2). Rotations for 2–3 years with rice, maize, or groundnut and solarization may reduce the populations of the nematode. Pigeonpea genotypes with resistance to *R. reniformis* have been reported, but such resistance has not been confirmed (3,31,56). At ICRISAT, all of more than 500 pigeonpea genotypes and 40 accessions of related wild species evaluated for resistance to *R. reniformis* were susceptible when assayed by a simple technique (45) of counting egg sacs stained with 0.25% trypan blue (Fig. 9). Accessions of *Rhynchosia aurea, R. minima*, and *R. rothii* are resistant to the nematode, and two short-duration pigeonpea genotypes, ICPL 83045 and ICPL 85024, are tolerant. Application of carbofuran (6 kg a.i./ha) at the time of sowing in fields infested with above-threshold levels of *R. reniformis* has increased crop yield by 25% (Fig. 10).

Root knot. *M. incognita, M. javanica, M. arenaria* (Neal) Chitwood, *M. hapla* Chitwood, and *M. acronea* Coetzee attack pigeonpea (29). The first two species are widespread in pigeonpea-growing regions and thus are more important than the others. Pigeonpeas are highly susceptible to *M. arenaria* but not to *M. hapla* (38). *M. acronea* is reported only in Malawi, but there is little information on its pathogenicity. Susceptibility may increase when *M. javanica* and *M. incognita* feed together on pigeonpea (Table 2). Some resistant genotypes (ICP 11289, 11299, 8863, and 8860) are moderately to heavily galled when both species are present in the soil (30,54).

Symptoms and disease cycle. Nematode infection stunts susceptible pigeonpea genotypes, and roots are moderately to severely galled. When the galls are very small, nematode infection can be verified by examination of roots for egg sacs. Considerable pathogenic variation occurs in populations of *M. incognita* and *M. javanica* on pigeonpea. Some populations in India, Nepal, and Malawi do not induce galls on roots of pigeonpea

but do produce many egg sacs. Many genotypes on which only egg sacs are produced apparently are not stunted by nematode infection. The disease cycle is similar to that on chickpea.

Pathogen interactions. The nematode species increase the severity of Fusarium wilt (36). *M. incognita* and *M. javanica* occurring with *F. udum* moderate wilt resistance in the cultivar ICP 8863 (50).

Management options. Resistance to *Meloidogyne* spp. is available in pigeonpea germ plasm. Among more than 200 genotypes evaluated at ICRISAT for resistance to *M. javanica*, only 13 were highly susceptible. Of the resistant genotypes, ICP 11289, 11299, and ICPL 151 are very promising. ICP 11289 and 11299 have resistance to nematode populations in India, Malawi, and the United States, and ICPL 151 is an early-maturing cultivar. Cereals are poor hosts of *M. incognita* and *M. javanica*. Rotations with sorghum, pearl millet, *Setaria* spp., maize, rice, and sesame can reduce the nematode population densities in 2–3 years. A pigeonpea/wheat cropping system also suppresses the nematode population (47). Seed treatment with 2.0% carbofuran and benfuracarb is effective (25).

Difficulties and Future Needs

Considerable progress has been made in identification of nematode-caused diseases and assessment of their damage potential. Future attention should focus on identification of cost-effective and practical management tactics for the most important diseases. The greatest obstacle to effective management is the lack of recognition that nematodes seriously limit crop yields. An urgent need is to educate growers that nematode problems exist and to train farmers and extension workers to detect, diagnose, and control these problems on chickpea and pigeonpea. Availability of trained personnel in the tropics is crucial for completion of this daunting task.

Farmers in the semiarid tropics traditionally manage important pests and diseases with cultural control. Chemical control is rarely an instrument of pest and disease management in subsistence crops. For example, *Helicoverpa armigera* is a highly destructive foliar insect pest of pigeonpea, but surveys of 10 states of India revealed that only 6% of the fields were treated with pesticides to control this pest (15). Several expensive pesticides have been tested for control of nematode diseases, but farmers are not expected to use them.

Any nematode management option must be compatible with low-input crop production strategies. Use of solar heat for nematode control is environmentally safe and effective, and coupling solarization with other control measures, such as application of neem cake and other soil amendments, may give long-lasting control of nematode-caused diseases. This management option may be useful in regions where multiple pests and diseases must be controlled, but the cost of the polyethylene sheet required for solarization may keep it beyond reach of resource-poor farmers in developing countries. Use of nematode-resistant cultivars is practical, and useful sources of resistance are available for root-knot nematodes. These promising sources of resistance should be tested extensively and utilized in breeding programs. Techniques for evaluation of resistance should be standardized, and plant breeders should be involved from an early stage in this research. At ICRISAT, screening techniques for evaluating pigeonpea genotypes for resistance and tolerance to *H. cajani, R. reniformis*, and *M. javanica* and chickpea genotypes for resistance to *M. javanica* have been standardized (45,46). We need to search for promising sources of resistance and tolerance to nematode diseases so that durable disease-resistant chickpeas and pigeonpeas can be identified.

Cropping systems, rotations, and intensity affect severity of nematode problems. Research at ICRISAT has shown that population densities of nematodes parasitic to pigeonpea in peninsular India can be reduced by removing plant hosts of the nematodes for extended periods. However, weed hosts enable nematode reproduction in the absence of cultivated plant hosts. Although an uncommon practice in pigeonpea and chickpea production, weed control is an essential part of crop rotation for successful management of nematode diseases.

Rotations that include a fallow period are slowly vanishing in parts of the tropics with explosive population growth and declining land productivity. Tropical agriculture needs adequate nematode control with minimum environmental disturbance and little capital input. The objective should be to reduce nematode-caused losses rather than to kill nematodes. A population planning program may permit nematodes to subsist on chickpea and pigeonpea but not in damaging proportions. Research and extension nematologists should provide technology for improved management of nematode diseases and innovative education of farmers. This will benefit the science of nematology and farmers in developed countries as well as resource-poor farmers in the semiarid tropics.

Table 2. Number of pigeonpea genotypes resistant to root-knot nematodes

Meloidogyne species	No. of genotypes screened/no. resistant	References
M. incognita	163/146	12,33,38
M. javanica	47/42	35,38
M. incognita + *M. javanica*	91/13	30,54
M. arenaria		
Race 1	47/2	35
Race 2	47/21	35

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