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Current Status of the Availability, Development, and Use of Host Plant Resistance to Nematodes [1]

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Abstract: Host plant resistance (HPR) to nematodes has been identified in many major crops and related wild germplasm. Most HPR is to the more specialized, sedentary endoparasitic genera and species, e.g., *Globodera, Heterodera, Meloidogyne, Nacobbus, Rotylenchulus,* and *Tylenchulus.* Some HPR has been developed or identified also to certain migratory endoparasites (*Aphelenchoides, Ditylenchus, Pratylenchus, Radopholus*) in a few hosts. Commercial use of HPR remains limited, despite its benefits to crop production when deployed appropriately. Restricted use and availability of HPR result from problems associated with transfer of resistance into acceptable cultivars. Difficulties occur in gene transfer to acceptable cultivars because of incompatibility barriers to hybridization or linkage to undesirable traits, for example in cucurbitaceous and solanaceous crops and sugarbeet. Specificity of HPR to only one species, or one or few pathotypes, as it relates to resistance durability and nematode virulence, and HPR response to abiotic factors such as high soil temperature, also limit availability and utility. A scheme for HPR development is presented to emphasize nematology research and information requirements for expanding HPR use in nematode control programs, for example in common bean, sugarbeet, and tomato. Nonbiological factors that influence HPR usage are discussed, including heavy reliance on nematicide programs, low priority of nematode HPR in many breeding programs, and insufficient breeder–nematologist collaboration.

Key words: breeding, durability, gene, inheritance, nematode, pathotype, resistance, screening, selection, tolerance, virulence.

Host plant resistance (HPR) is expected to contribute to the solution of many problems caused by nematodes. The potential of HPR is enormous because of increasing availability of and access to plant germplasm collections containing genes for resistance and because of rapid advances in plant science technologies. The need for progress in resistance development is critically important for two major reasons. First, in the less developed countries of tropical and subtropical regions, use of resistant cultivars may be the only economically practical management tactic around which other supportive strategies can be integrated. Second, in modern high-input production systems of developed countries, as the often singular reliance on chemical nematicides has been restricted or has ended, HPR must be developed further as a primary control option, whether alone or as a key element in an integrated management program.

RESISTANCE AVAILABILITY

Current HPR utilization has been summarized (4,14,19,20,58,68) and can be categorized according to crop, nematode (genus or species), geographical region, efficacy, and gaps in availability. These gaps can be subdivided into crops in which no HPR has been identified or made available and crops in which HPR is available for one or more target nematode species but not others. This type of summary should help in setting priorities for HPR research and for indicating which nematode–crop combinations are likely to have heritable HPR traits that can be exploited. Consideration of the critical factors that influence phenotypic expression of HPR is also help-

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ful; these include nematode virulence and its selection, heat sensitivity, disease complexes, and polyspecific nematode communities, races, or pathotypes.

The nematode genera for which most HPR have been identified and developed for use through breeding are primarily those with specialized host–parasite relationships for at least a portion of their life-cycles (14,58). These genera include the sedentary endoparasites *Globodera, Heterodera, Meloidogyne, Rotylenchulus,* and *Tylenchulus,* the migratory endoparasites *Aphelenchoides, Ditylenchus, Pratylenchus,* and *Radopholus,* and the ectoparasites *Criconemella* and *Xiphinema.* The sedentary endoparasites, especially cyst and root-knot nematodes, are the most specialized and compose the majority of important nematode pests for which HPR has been identified and developed (4,14,19,20,58, 68).

Migratory endoparasites: HPR has been identified against a few agriculturally important migratory endoparasites, particularly to *Ditylenchus dipsaci* with notable commercial success in red clover (*Trifolium pratense*), alfalfa (*Medicago sativa*), and oat (*Avena sativa*). Other HPR to migratory endoparasites includes that to *Radopholus citrophilus* in *Citrus* sp. with limited success, that to *Aphelenchoides besseyi* in rice (*Oryza sativa*) and to *A. ritzemabosi* in chrysanthemum (*Chrysanthemum* spp.), both in use, and that to *Pratylenchus brachyurus* in peanut (*Arachis hypogaea*) and to *P. penetrans* in potato (derived from *Solanum tuberosum* ssp. *andigena* and *S. vernei*) (6), identified but not developed. A vigorous search for resistance to *P. vulnus* in *Prunus* spp. germplasm is in progress (15). Given the numerous economically important genera and species of migratory endoparasitic nematodes worldwide, these are very few instances.

Ectoparasitic nematodes: Only isolated reports of HPR have been made, and these may be tolerance to nematode damage rather than true plant resistance to nematode infection. In grape (*Vitis* spp.), reports of HPR to *Xiphinema index* are tolerance in at least some cases (25,26,48), although population decline under some *Vitis* selections is indicative of HPR expression. Regardless, any combination of these traits would be beneficial commercially. Finding HPR to *Xiphinema* is not surprising because it has a more specialized parasitic relationship with its host than many other ectoparasites, as *Xiphinema* induces plant cellular modifications, including hypertrophy (26). Similarly, ring nematodes like *Criconemella xenoplax* (the focus of HPR or tolerance screening in *Prunus* spp. genotypes for improvement of almond, nectarine, peach, and plum plantings [52]) have a more specialized relationship with their host than most other ectoparasitic nematodes (32). Resistance screening procedures for these ectoparasitic forms are difficult, especially with perennial tree and vine crops (26), and commercial development of cultivars or rootstocks resistant to ectoparasitic nematodes has been insignificant.

The most frequently utilized resistant nematode–crop combinations (Table 1) include crop genotypes that are utilized on at least a moderate proportion of the infested crop acreage, that have sustained economic yields on nematode-infested land, and that in some cases have not selected for more aggressive species, populations, or pathotypes after many years of cultivation. The range of important crop types,

TABLE 1. Some highly effective host plant resistance programs.

Nematode	Crop
Globodera rostochiensis	Potato
Heterodera glycines	Soybean
Heterodera avenae	Barley, oat
Meloidogyne incognita	Cowpea, lima and common bean, soybean, tobacco
Meloidogyne spp.	Alfalfa, *Prunus* (Nemaguard rootstock), tomato, walnut (California Black)
Tylenchulus semipenetrans	Citrus (*Poncirus trifoliata*)
Ditylenchus dipsaci	Alfalfa, oat, red clover

including both annuals and perennials, suggests that most botanical groupings probably contain HPR traits to the major nematode parasites.

World survey: A recent world survey of the local availability of nematode-tolerant or resistant crops was summarized according to crop and global region (64), with responses from over 300 nematologists in 75 countries. Tolerance is defined as the ability of a plant to grow and yield despite injury from nematode attack, and it is independent of resistance or susceptibility, which refer to the ability (or lack thereof) of a plant to support nematode reproduction. The survey reveals that significant advances have been made in HPR development on a global scale, with resistance or tolerance locally available in at least some crops in every region of the world. The success in the breeding or simple selection of crop varieties with tolerance or resistance differs among regions but does not conform closely to any pattern of agricultural progressiveness. The modern distribution of commercial seed and other planting stock, such as root-knot nematode-resistant tomato (*Lycopersicon esculentum*) cultivars and lines, has no doubt contributed to the widespread availability and use of some important HPR germplasm.

On closer examination, the availability of HPR is substantially limited. Major gaps in HPR availability exist in nematode–crop combinations in which HPR has not yet been identified or introgressed or adequately developed (Table 2). In Africa only four crops were reported with tolerance or resistance, of which bean (*Phaseolus vulgaris*), cowpea (*Vigna unguiculata*), and tomato with resistance to *Meloidogyne* spp. are resistant to only some root-knot species or populations (64). Furthermore, the resistance in bean and tomato is heat-sensitive and thus unsuitable for the hot production areas in lower latitudes (1,53). Further limitations exist in bean, tomato, and other crops resistant to *Meloidogyne* spp. because HPR to other injurious nematodes is generally unavailable. The limited use of HPR in a huge continent like Africa with considerable nematode-induced crop losses underscores the enormity of the task facing nematologists and plant breeders in HPR research. A similar shortage in HPR was indicated for the region encompassing Mexico, Central America, and the Caribbean, for which only five crops with HPR were reported (64).

Among regions with more advanced agricultural systems, as in North America, gaps similar to those in Africa exist in crops such as tomato because of species, population, and heat sensitivity factors. Other crops have wider limitations. In *Pru-*

TABLE 2. Examples of important gaps in host plant resistance availability for nematode–crop combinations with no developed resistance and for crops with some developed nematode resistance.

Nematode	Crop
No developed resistance	
Heterodera schachtii	Sugarbeet, crucifers
Heterodera avenae	Wheat
Meloidogyne spp.	Barley, carrot, corn, cucurbits, eggplant, lettuce, peanut, sugarbeet, many others
Meloidogyne hapla	Most host crops
Rotylenchulus reniformis	Tomato, many host crops
Nacobbus aberrans	Pepper, sugarbeet, tomato
Some developed resistance	
Heterodera avenae	Barley, oat
Tylenchulus semipenetrans, *Radopholus citrophilus*	Citrus
Meloidogyne incognita, *Meloidogyne javanica*, *Meloidogyne arenaria*	Common bean, lima bean, cowpea
Meloidogyne incognita, *Rotylenchulus reniformis*	Cotton
Globodera rostochiensis, *Globodera pallida*, *Meloidogyne* spp., *Nacobbus aberrans*	Potato
Pratylenchus vulnus, *Xiphinema americanum*, *Criconemella xenoplax*	*Prunus* spp.
Heterodera glycines, *Meloidogyne* spp., *Rotylenchulus reniformis*	Soybean
Meloidogyne hapla, *Meloidogyne incognita*, *Meloidogyne javanica*, *Meloidogyne arenaria*	Tomato

nus, excellent resistance to *Meloidogyne* species based on a single dominant gene is widely used in Nemaguard rootstock. This rootstock, however, is susceptible to other injurious nematodes including ring (*Criconemella xenoplax*), pin (*Paratylenchus neoamblycephalus*), lesion (*Pratylenchus vulnus*), and dagger (*Xiphinema americanum,* a ringspot virus vector) nematodes (14,47). Other rootstocks with HPR or tolerance to one or more of these nematode pests is unavailable, unidentified, or in the screening and early selection phase (15,52).

A further limitation in the utilization of HPR is that it is incorporated commonly into only a small proportion of the most frequently grown cultivars or rootstocks. In fact resistant cultivars or rootstocks often have low yield or quality traits, undesirable maturation times, or other specific problems, e.g., the promotion of undesirable vegetative growth by *Meloidogyne*-resistant grape rootstocks (58). Thus, use of resistance may be restricted by limitations of the resistance itself, e.g., narrow specificity, as well as by the less desirable phenotypic characteristics of the germplasm in which it has been incorporated.

Durability of Resistance

Durable resistance is that which has remained effective in a cultivar during widespread cultivation for a long period of time, in an environment favorable to a disease or pest (35). Van der Plank (76) supported the general argument that disease resistance conferred by many genes (polygenic) is likely to be more durable than resistance conferred by a single gene (monogenic), with respect to resistance being horizontal (race-nonspecific) or vertical (race-specific). However, he provided examples of both monogenic and polygenic resistance to fungal pathogens that did not conform to this argument, and many other examples of exceptions have been reported (35). A review of existing HPR programs and some recent research findings suggests that durability of resistance to nematodes also cannot be predicted based on the genetic control of HPR.

Potato and Globodera: The resistance to *Globodera rostochiensis* in potato is conferred by the single dominant major gene *H1* derived from *Solanum tuberosum* ssp. *andigena,* and it has been transferred into several commercial cultivars, such as Maris Piper in the United Kingdom. These cultivars have been grown extensively on infested land since becoming available about 30 years ago (36). Despite intensive use, there is no reported evidence that gene *H1* has selected new virulent pathotypes. The presence in Northern Europe of pathotypes able to reproduce on plants with *H1* is apparently not a result of exposure to resistant potato cultivars, but rather is a consequence of the original introduction of these types from South America, where they were previously selected and where different pathotypes are present today (36). These observations suggest a useful level of durability of the major dominant gene *H1.* Paradoxically, with respect to the putative durability of polygenic resistance, there is now good evidence from independent studies (75,78) for selection of virulence over several generations of *G. pallida* on *S. vernei* clones, where resistance is conferred polygenically by genes with minor effects (36,75). Although these studies were made in pot and microplot tests that possibly could artificially promote fitness of the selected virulent populations, the rapid rate of selection in several distinct populations suggests that resistance breakdown might occur in *G. pallida*-infested fields routinely cropped with ex *vernei*-resistant potato cultivars. The extent of heterogeneity for virulence present within the nematode population and the potential for virulence to develop by mutation are important factors that determine resistance durability (61) and, therefore, its future utility.

Peach and Meloidogyne: The resistance to *Meloidogyne incognita* and *M. javanica* in the peach rootstock Nemaguard is conferred by a single dominant major gene (possibly

a separate single gene to each species) derived from a *Prunus persica* cross involving S.37 (C. Ledbetter, USDA, Fresno, CA, pers. comm.), a seedling that Chitwood et al. (13) reported resistant to both *M. incognita* and *M. javanica.* Nemaguard has been used in California in at least 85% of the almond, nectarine, peach, and plum plantings; and after about 35 years of use resistance-breaking, *Meloidogyne* populations have not appeared (M. V. McKenry, pers. comm.). This HPR durability is particularly interesting, as the continual presence of root-knot nematode populations is maintained by weeds in Nemaguard-planted fields, thus influencing the opportunity for selection.

Tomato and Meloidogyne: Resistance in tomato to *M. incognita, M. javanica,* and *M. arenaria* is conferred by a single dominant major gene, *Mi,* derived from one hybrid F_1 plant obtained through embryo rescue from a cross of resistant *Lycopersicon peruvianum* with *L. esculentum* (61,70,77). Gene *Mi* is the basis of all the root-knot resistance in worldwide commercial use today, including fresh market and processing types. Since its availability about 35 years ago, there has been little evidence for virulence selection under field conditions, although naturally virulent populations exist that have had little or no exposure to *Mi* gene-bearing plants (61,62). That virulence to *Mi* can be selected rapidly under artificial greenhouse conditions (16,34,61, 62) indicates that selection of virulence by frequent or continuous plantings of resistant tomato cultivars is a threat. However, the fitness under field conditions of selected, virulent populations has been studied to only a limited extent. Increase of *M. hapla* (able to reproduce on plants with *Mi*) populations on plantings of resistant tomato in fields containing polyspecific root-knot populations was reported (61). A full discussion of *Meloidogyne* resistance in tomato is available (61), as are recent analyses of selected *Mi*-virulent *Meloidogyne* populations (12,16,34,62).

Thus in three different cases there is evidence for durability associated with simply inherited nematode HPR which, when used in an integrated management program, could continue to be effective in the future. The durability of these and other nematode HPR traits is unpredictable, and breakdown of resistance can occur at any time. Because the genetic basis of the durability is unknown (35), it cannot be prioritized with any certainty in breeding programs. The reports of selection for virulence in *G. pallida* on polygenically resistant potato cultivars underscore the unpredictability of nematode HPR durability and longterm usefulness. Therefore, identification of additional nematode resistance genes and their introgression into crop cultivars should be a continuing priority.

Resistance Development Scheme

A useful approach to prioritizing HPR development needs is close examination of these gaps (Table 2) in the context of the steps involved in the development of usable HPR. The scheme presented in Figure 1 attempts to summarize this process as a sequence of essential or highly desirable research components and information requirements for identifying HPR in plant genetic resources and then introgressing it to develop new acceptable commercial cultivars. The examples of successful HPR development in Table 1 were accomplished through programs that followed this general scheme for the most part. The scheme is based primarily on the perspective of the nematologist, and as such it highlights components in the process that the nematologist has a major responsibility for in working with the plant breeder. Breeder-specific concerns might include factors such as multiple disease and pest resistance, agronomic improvement requirements, resource limitations, and the fundamentals of the crossing, selection, and screening and testing cycles that make up the classical breeding program. These have been adequately discussed and reviewed (3,5,22,63).

Screen for HPR: In initiating an HPR

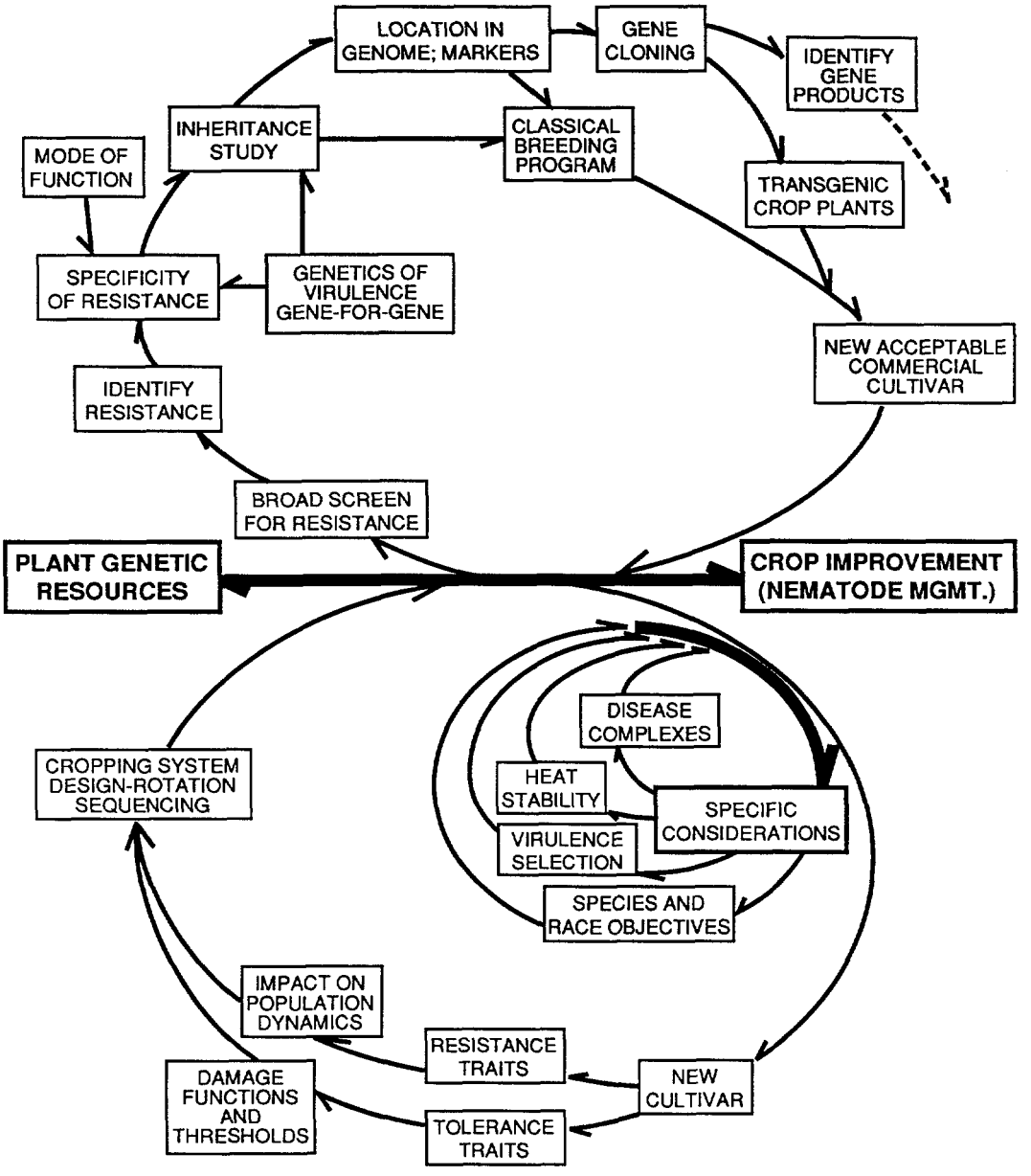

FIG. 1. Schematic presentation of the utilization of plant genetic resources in the development of nematode resistant (or tolerant) cultivars for crop improvement, emphasizing nematological research components and information requirements.

program (the upper sequence in Figure 1), a broad screen for HPR traits is required to identify potentially useful sources (59). In decreasing order of preference, HPR can be identified in current cultivars and breeding lines, older cultivars or breeding stocks, primitive cultivars or accessions likely to be found in gene pool centers, and finally, wild species progenitors or relatives (22,27,42). The obvious sequence to this search minimizes the genetic distance between the HPR source and the recipient crop cultivar type and the associated difficulties in efficient genetic transfer. Unfortunately, in most crop–nematode combinations with HPR needs (Table 2), HPR has

not been found in the domesticated genotypes of the crop plant species, but only in primitve relatives of the crop species (e.g., *Meloidogyne* HPR in common bean, lima bean, cowpea, carrot, pepper, sweet potato, and soybean) or, more commonly, in wild species relatives (e.g., *G. pallida* and potato; *H. avenae* and small grains; *H. schachtii* and sugarbeet and cruciferous crops; *Meloidogyne* spp. and cucurbits, eggplant, peanut, potato, small grains, tobacco, and tomato; *N. aberrans* and tomato, and *T. semipenetrans* and citrus crops).

Obviously, if HPR is not found the program cannot advance, as occurs with *N. aberrans* and sugarbeet, *Meloidogyne* spp. and lettuce, and *M. naasi* and barley. In most cases, the lack of HPR probably reflects insufficient screening due to limited resources or lack of access to adequate germplasm sources. Screening of additional germplasm as it becomes available through collection and improved storage and access should help to identify HPR and fill in some important gaps. For example, recently, additional collections of eggplant (*Solanum melongena*) and closely related species from East Africa and India are being screened at Riverside for *Meloidogyne* resistance for the first time. These accessions could contain sources of HPR genetically much more compatible with eggplant than the *S. sisymbriifolium* resistance source currently used with considerable difficulty for possible transfer through tissue culture (21,24). Other recent screenings at Riverside of new germplasm collections have revealed HPR sources to the following: *M. incognita, M. javanica,* and *M. chitwoodi* in wild wheat (*Triticum tauschii*) (40,41); *M. javanica* and *M. arenaria* in common bean (54,55); *M. incognita* and *M. hapla* in *Lycopersicon peruvianum* (2,10); aggressive isolates of *M. incognita* in cowpea accessions (Roberts, unpubl.); and *N. aberrans* in other wild *Lycopersicon* spp. (9).

Specificity of HPR: Once HPR has been found, its potential utility should be determined to enable a prioritization for nematode management and for the breeding effort. Factors for consideration at this stage include resistance specificity with respect to pathotype, race, or species. Other specific factors, as indicated within the lower sequence of Figure 1, include temperature sensitivity and the utility of the resistance in controlling disease complexes that involve the target nematode, such as *Meloidogyne* and Fusarium wilt (67). Well-defined local requirements may need to be considered at this stage. Additional subjects of nematological research (Fig. 1) include the extent of hypersensitivity in the incompatible response and the degree of root galling, localized necrosis, or other effects on the host plant that can influence the tolerance to infection under field conditions. The broader the perceived utility of the HPR, the higher will be its likely priority for development if resources allow. The original decision to screen for HPR is indicative of a high research priority; however, unless a trained nematologist is involved, the most appropriate nematode screen may not be used. Such undesirable screens include those at inappropriate temperatures, those with single-species inoculum where mixed-species inoculum would be preferable, and those in which a less prevalent species or pathotype is used, for example with *Meloidogyne* spp. or *G. pallida* and *G. rostochiensis.*

Inheritance of HPR and nematode virulence: The inheritance of resistance also should be pursued by the nematologist, usually in conjunction with the plant breeder, to provide direction for the breeding and selection process. Although much of the identified HPR to nematodes is conferred by one or a few dominant genes with major effect (4,14,20,68), this is not always the case and should not be assumed for new resistance sources. For example, recessive resistance controlled by several genes with minor effects is involved in *G. pallida* resistance in potato (36), and recessive resistance to *H. glycines* occurs in soybean (43). Knowledge of inheritance will have a direct bearing on the breeding program best suited for transfer and selection of resistant genotypes in each generation,

whether by recurrent backcross, mass selection, or another approach. The potential for molecular characterization and isolation of resistance genes also will become clearer. Investigations can include genetic control of nematode virulence and parasitism as they relate to the genetics of resistance. This information can be helpful in determining the specificity of resistance with respect to pathotype and virulence. Limited studies undertaken thus far with the amphimictic potato cyst nematodes have revealed simple dominant monogenic control of avirulence that conforms to a gene-for-gene relationship with a single dominant potato gene, *H1* (33,37). Such studies may be difficult at best in apomictic nematodes such as the parthenogenetic *Meloidogyne* species, which require different approaches to study the nature of virulence (16).

Role of nematologist: At the point in the development sequence when the HPR trait is considered valuable and breeding is started (Fig. 1), the role of the nematologist should be one of support to the breeder. Nematologists should focus on provision of appropriate inoculum, guidance and possibly assistance in resistance screening and rating procedures, and perhaps ultimately in evaluation of field performance of advanced lines in infested sites (73). A strong collaborative relationship between breeder and nematologist is essential to an effective nematode HPR program. Boerma and Hussey (5) provide an excellent example of such a cooperative effort, focused on multiple nematode resistance breeding for soybean improvement. A lack of awareness in the breeding program of the importance to crop improvement of controlling nematode pests through HPR and a lack of technical knowledge or facility for the breeder to include the nematode HPR objective are likely unless there is input from the nematologist. Buddenhagen (7) outlined specific limitations of breeding programs to respond to objectives for pest and disease resistance, based on whether programs were private (commercial), public (state, federal, or foundation), or international (e.g., CIAT, CIMMYT, or IRRI). Although beyond the scope of this article, these nonscientific considerations will continue to have a major impact on progress in nematode HPR development throughout the world. The need to heighten awareness of nematode-induced crop losses and the potential for significant crop production improvement, professional reward, and commercial profitability by HPR development is a continuing responsibility for nematologists.

Introgression of HPR: The predominance of HPR traits in nondomesticated or wild plant germplasm obviously constrains attempts to breed for nematode resistance. These constraints are less difficult to overcome when HPR exists within the crop plant species, as exemplified by successful breeding of HPR to several types of nematodes in various leguminous crops; nevertheless, breeding can average 10–15 years (14). Where wild species donors must be used, the probability for successful transfer is often unpredictable. Only four examples of successful breeding with wild species as nematode HPR sources exist: resistance to *Meloidogyne* spp. in tomato from *L. peruvianum* using tissue culture (70), resistance to *M. incognita* in tobacco (*Nicotiana tabacum*) from *N. tomentosa* by traditional breeding (69), and resistance to *G. pallida* and *G. rostochiensis* in potato from *Solanum vernei* and *S. tuberosum* ssp. *andigena,* respectively, also by traditional breeding (14).

Other attempts to transfer HPR across interspecific boundaries have not been successful. Hawkes (27) summarized five broad categories representing increasing interspecific distance to be bridged. He used examples ranging from those in which some natural genetic exchange by hybridization and introgression occurs in the wild to those with low levels of hybrid fertility with incompatibility (e.g., from different chromosome number or genome formula) that require special transfer techniques, such as the embryo rescue used in *Mi* transfer (44,70). Other situa-

tions involve wide hybridization that may only be overcome by transgenic techniques or somatic hybridization (27). In breeding for nematode HPR, we must overcome this full range of interspecific incompatibility levels. Research on nematode HPR in sugarbeet, tomato, and common bean are discussed further to illustrate key points in the approach to development.

Resistance in Sugarbeet

Cyst nematode HPR: More than 50 years of research have not yet produced an acceptable sugarbeet (*Beta vulgaris*) resistant to *H. schachtii.* Initial hybridizations of different *Beta* species started in 1937 (66). Resistance to *H. schachtii* was reported for the first time in 1951 (31) in three wild species in the section *Patellares—B. patellaris, B. procumbens,* and *B. webbiana*—but no resistance was found then or since in *B. vulgaris* (section *Vulgares*). In 1958–60 Dr. Helen Savitsky (USDA, Salinas, CA) began to transfer resistance from these sources, especially *B. procumbens* (diploid), into commercial *B. vulgaris* (tetraploid) lines. Fifteen years later, she reported that a chromosome of *B. procumbens* carrying resistance gene(s) had been transferred to *B. vulgaris* to produce viable resistant monosomic addition lines. Four resistant trisomic plants were selected from 6,750 first backcross plants derived from triploid hybrids (65,66). This success was achieved through extensive backcrossing and screening, and two diploid plants with fixed resistance were selected from 8,834 backcross plants in the progenies of the trisomic individuals. Resistance was transferred from both plants to F_1 hybrids with susceptible diploid plants, and through crossing-over, a segment of the *B. procumbens* chromosome carrying resistance was transferred to a sugarbeet chromosome, thereby breaking the resistance linkage to early bolting (66). The difficulty of the transfer resulted from the rare pairing of the nonhomologous alien chromosomes in *Beta* hybrids, root necrosis and subsequent death of the hybrid seedlings (71). More recently, additional programs in the Netherlands (28–30,71,72) and in Germany (38,45) have further advanced Savitsky's and their own hybrid-derived resistant materials and generally complemented the continuing program at Salinas (46,81) in attempts to produce a resistant commercial sugarbeet.

The testing of repeated backcross selections to produce resistant sugarbeets has yielded considerable frustration. At Salinas, M. H. Yu (pers. comm.) has indicated that the more advanced resistant material does not meet acceptable yield and quality standards and may require another 5 to 10 years of breeding and selection. This process is complicated by an apparent paracentric inversion of the *B. procumbens*-derived resistance-carrying chromosome fragment (17), which will require correction by the rare event of simultaneous crossing-over at each end of the transferred fragment. Heijbroek et al. (28,30) have selected resistant lines that show high rates of resistance transmission to offspring but lack the requisite commercial standards of sugar and juice content and uniformity.

Sugarbeet tolerance and cyst nematode virulence: A major additional problem with the *B. procumbens*-derived resistance is that it is based on a strong, localized hypersensitivity (82) that causes root necrosis and renders the plant intolerant to nematode infection. Consequently, resistant plants in *H. schachtii*-infested soil showed root weight losses of 50–60% (46). Unless the intolerance to initial infection and subsequent water stress can be overcome by further breeding, the management value of resistant sugarbeets may be limited to shortening rotations through restricting *H. schachtii* reproduction (46). The utility of this strategy may be limited further, as indicated by recent reports of wide differences among *H. schachtii* populations in reproduction on resistant breeding lines (49) and the existence of populations of *H. trifolii* able to parasitize and injure resistant (ex *procumbens*) sugarbeet (74). Wilt-tolerant pollinators derived from *B. vul-*

garis and *B. maritima* may facilitate the introduction of tolerance to hypersensitivity into resistant sugarbeet lines (30). The apparent differences in specificity of the *H. schachtii* resistance genes from section *Patellares* (50) suggest that an array of resistance genes will be required in *B. vulgaris* to manage the pathotypes of *H. schachtii* that will emerge as resistant sugarbeets are brought into commercial use. The difficulty of resistant sugarbeet development through traditional breeding efforts would justify the rigorous application of molecular biotechnology to expedite resistance gene transfer, such as that initiated by Jung et al. (39).

Root-knot nematode HPR: The reduced availability of nematicides for controlling *Meloidogyne* spp. infestations makes the identification and transfer of HPR to root-knot a high priority for sugarbeet improvement (Table 2). *Meloidogyne* spp. resistance has been reported in *B. procumbens* (18), and one can anticipate many of the same or similar problems for introgression as those encountered in developing resistance to *H. schachtii.* Hypersensitivity occurs in *Meloidogyne*-resistance responses and, if present in sugarbeet, its impact on tolerance to root-knot nematode infection must be assessed.

Resistance in Tomato and Bean

Tomato: Transfer of gene *Mi* into tomato is a good example of successful introduction of HPR from a wild species, although many gaps in HPR availability exist (Table 2), some of which were not apparent until recently. The resistance was detected in 1941, and in 1944 one resistant F_1 hybrid plant with *L. esculentum* was recovered using embryo rescue (70). A few initial backcrosses were obtained with cuttings of this plant (77), and the backcrosses were used in the California and Hawaii breeding programs to develop resistant commercial tomato cultivars. However, 15 years were required to break the tight linkage between *Mi* and undesirable fruiting characters (23). All *Meloidogyne*-resistant tomato cultivars available today are derived from this single source, and they have had a major impact worldwide in reducing tomato yield losses due to root-knot nematodes. In 1979 the first machine-harvestable processing tomato cultivar with *Mi* was released. Since then, many other processing cultivars with *Mi* have been developed that possess various yield and fruit quality and maturity traits, enabling widespread use in the processing tomato industry (60,61).

Despite the wide utility of HPR conferred by *Mi,* significant gaps remain to be filled in expanding nematode HPR use in tomato. For example, the incorporation of the recently identified gene in *L. peruvianum* that confers heat-stable resistance to *M. incognita* (1,2,10,11) should greatly extend resistance use into hotter regions, where breakdown of *Mi*-conferred resistance occurs. This new resistance also appears to be effective against some isolates of *M. incognita* selected for virulence to *Mi*-bearing plants (62), a finding that has important implications for broadening the narrow genetic base of resistance in tomato. The heat-stable resistance gene has been transferred with embryo callus and embryo cloning techniques into F_1 hybrids with *L. esculentum* that express heat-stable resistance, as a preliminary step toward introgression (11). Resistance to *M. hapla,* which is not controlled by *Mi,* has been identified in some *L. peruvianum* accessions (2,9) and should be transferred to tomato.

The false root-knot nematode, *Nacobbus aberrans,* is an important pest of tomato and other vegetables in Central and South America, where significant yield losses occur (8). This nematode often occurs in the same field as root-knot nematodes, making identification and diagnosis difficult and control of only one pest ineffective in protecting the crop. The recent identification in our program (9) of putative resistance to *N. aberrans* in accessions of *L. chmielewskii* and *L. peruvianum,* the former of which is compatible with *L. esculentum,* presents the opportunity to develop tomato cultivars with HPR to *N. aberrans* alone or with *Meloidogyne* spp.

In an analogous case, a single dominant gene for resistance to the reniform nematode, *Rotylenchulus reniformis,* was identified some years ago in the currant tomato, *L. pimpinellifolium* (56,57). Reniform nematode is widespread and damaging in subtropical and tropical tomato production areas, often concomitantly with root-knot nematodes (51). The currant tomato is compatible with *L. esculentum* and preliminary attempts at transferring the resistance by hybridization produced resistant F_3 plants (57). Complete introgression and cultivar development should be possible. The different nematode resistance genes available for tomato make breeding for multiple nematode resistance a real possibility, similar to the soybean and potato programs for resistance to cyst, reniform, and root-knot nematodes (5,20).

Common bean: Resistance to *Meloidogyne* spp. in common bean, *Phaseolus vulgaris,* exists (19,53) but has been exploited on a limited scale. Resistance to *M. incognita* in the old cultivar Alabama No. 1 that was used to develop a resistant pole bean cultivar, Manoa Wonder, is conferred recessively by at least two genes and is heat-unstable above 26 C (53). Resistance to *M. incognita* in the accession PI 165426 from Mexico is controlled by one dominant and one recessive gene; the dominant gene is completely dominant up to 26 C but incompletely dominant at 28 C and above, whereas the recessive gene is ineffective above 26 C (53). This resistance was used to develop the resistant bush snapbean cultivar Nemasnap, which has been used on a limited basis in the southeastern United States (19,20,80). A single dominant gene for resistance to *M. javanica* and *M. arenaria* and to some *M. incognita* isolates, which is also incompletely dominant at or above 28 C, was identified recently in lines A315 and A445 derived from Mexican accessions G2618 and G1805 (53,54). The latter resistance has not been transferred into commercial cultivars. This genetic analysis, in relation to temperature of root-knot resistance in *P. vulgaris* (53), demonstrates the importance to breeding of the screening, specificity, and inheritance components in the HPR development scheme (Fig. 1, upper sequence), together with specific factors such as temperature sensitivity (Fig. 1, lower sequence). Definition of the nature and expression of resistance should facilitate and expedite the development of commercial cultivars of the many types of fresh and dry edible beans for use in particular climatic regions.

Resistance Application

The utility of nematode HPR should be considered from two general perspectives: i) the value of HPR in crop or cultivar self-protection, based on the level of tolerance to the injury caused by nematode initial infection, and ii) the rotational value of HPR in cropping systems for protecting subsequent crops, based on the ability to reduce nematode population densities in soil by restricting nematode reproduction.

Later aspects of the HPR scheme (Fig. 1, lower sequence) include the goal of producing resistant or tolerant cultivars and the goal of feedback of information on cultivar performance. The assessment of tolerance and resistance traits under field conditions can be done usually only later in the breeding process when yield measurements become meaningful. However, earlier testing in the program may avoid subsequent problems, such as the severe intolerance to *H. schachtii* in sugarbeet. The utility of the resistance will be anticipated at the start of the HPR program, but the true extent of its value for nematode management will probably not be realized until implementation occurs. At that time, the role of the resistant cultivar in the rotation sequence of a cropping system can be maximized.

Because more planning is involved in cropping system design now than in the past, it is important to consider the placement of resistance in terms of which and how many crops should carry resistance genes. The resistance to Columbia root-knot nematode, *M. chitwoodi,* in wheat (40) probably has little value for wheat im-

provement but could be valuable in reducing nematode populations in rotations of wheat with potato, for which resistance is not available. The resistance to *M. javanica* and *M. incognita* in wheat (41) could be exploited in various annual crop rotations to manage these species. A similar scenario can be suggested for incorporating *Meloidogyne* HPR into corn, a root-knot tolerant crop commonly rotated with many susceptible crops on infested ground. In rotations that include several crops for which HPR to a common nematode pest is available but which may require further breeding, decisions could be made on which crops should be prioritized for receiving resistance, as not all susceptible crops in the system may require HPR. Such decisions will maximize the allocation of critical HPR breeding resources for nematodes with typically wide host ranges.

Feedback on resistant cultivar performance (Fig. 1, lower sequence) can help to determine effectiveness of resistance for regions with different environmental characteristics and specific nematode problems. This feedback should facilitate a rational reprioritization of breeding objectives, for example whether or not the resistance already developed should be incorporated into additional cultivars or whether other types of resistance should be bred into the system. Resistance performance should be assessed for factors such as 1) reducing nematode population densities, 2) self-protection in terms of tolerance to nematode injury, 3) the presence or selection of virulent pathotypes of the target species or shifts in the prevalence of other nematode pest species, 4) response to disease complexes that include nematodes, 5) response to temperature regimes or other abiotic factors, and 6) system compatibility including any undesirable linkages with other pest, disease or agronomic traits.

The status of HPR for nematode control will improve rapidly because the decreasing availability and use of nematicides emphasize the need for HPR, and because molecular biology techniques will provide more direct methods of resistance gene selection and transfer. This aspect of HPR development is indicated in the upper sequence of Figure 1. Two practical considerations for molecular techniques applied to crop improvement with nematode HPR can be summarized. First, the difficulties encountered in traditional breeding for resistance, like those described for *H. schachtii* resistance in sugarbeet, should be overcome in many cases through application of molecular markers for selection or by transfer of cloned genes. Second, the potential is offered for transferring cloned resistance genes between unrelated crops to control a nematode pest common to both. The attempts underway to isolate and clone *Mi* (79) raise the question of the extent to which this gene can or should be transferred to other root-knot susceptible crops without threatening the durability of *Mi*. Cloning, transfer, and expression of resistance genes in plants may become simple and routine; but, if not, the wide utilization of key nematode resistance genes or active resistance gene products must be planned carefully. Application of biotechnology to resistance breeding will almost certainly still require traditional breeding methods to develop primary transgenic plants established in tissue culture into acceptable commercial cultivars or rootstocks (5).

Literature Cited

1. Ammati, M., I. J. Thomason, and H. E. McKinney. 1986. Retention of resistance to *Meloidogyne incognita* in *Lycopersicon* genotypes at high soil temperature. Journal of Nematology 18:491–495.

2. Ammati, M., I. J. Thomason, and P. A. Roberts. 1985. Screening *Lycopersicon* spp. for new genes imparting resistance to root-knot nematodes (*Meloidogyne* spp.). Plant Disease 69:112–115.

3. Bassett, M. J. 1986. Breeding vegetable crops. Westport, CT: AVI Publishing.

4. Bingefors, S. 1982. Nature of inherited nematode resistance in plants. Pp. 188–219 *in* K. F. Harris and K. Maramorosch, eds. Pathogens, vectors and plant disease: Approaches to control. New York: Academic Press.

5. Boerma, H. R., and R. S. Hussey. 1992. Breeding plants for resistance to nematodes. Journal of Nematology 24:242–252.

6. Brodie, B. B., and R. L. Plaisted. 1991. Resis-

tance in potato to *Pratylenchus penetrans*. Journal of Nematology 23:522 (Abstr.).

7. Buddenhagen, I. W. 1983. Plant breeding or pesticides to narrow the yield gap? Proceedings of the Tenth International Congress of Plant Protection, Brighton, England 2:803–809.

8. Canto-Saenz, M. 1992. Life cycle and pathogenicity of *Nacobbus aberrans*. *In* Proceedings of the Second International Nematology Congress, 11–17 August 1990, Veldhoven, The Netherlands, in press.

9. Cap, G. B. 1991. Inheritance and phenotypic expression of heat stable resistance to *Meloidogyne* spp. in *Lycopersicon peruvianum* and its transfer to edible tomato. Ph.D. dissertation, University of California, Riverside.

10. Cap, G. B., P. A. Roberts, and I. J. Thomason. 1991. Inheritance of heat stable resistance to *Meloidogyne incognita* in *Lycopersicon peruvianum*. Journal of Nematology 23:522–523 (Abstr.).

11. Cap, G. B., P. A. Roberts, I. J. Thomason, and T. Murashige. 1991. Embryo culture of *Lycopersicon esculentum* × *L. peruvianum* hybrid genotypes possessing heat-stable resistance to *Meloidogyne incognita*. Journal of the American Society for Horticultural Science 116:1082–1088.

12. Castagnone-Sereno, P., C. Piotte, P. Abad, M. Bongiovanni, and A. Dalmasso. 1991. Isolation of a repeated DNA probe showing polymorphism among *Meloidogyne incognita* populations. Journal of Nematology 23:316–320.

13. Chitwood, B. G., A. W. Sprecht, and A. L. Havis. 1951. Reactions of peach seedlings to nematode infections. Phytopathology 41:559 (Abstr.).

14. Cook, R., and K. Evans. 1987. Resistance and tolerance. Pp. 179–231 *in* R. H. Brown and B. R. Kerry, eds. Principles and practice of nematode control in crops. Orlando: Academic Press.

15. Culver, D. J., D. W. Ramming, and M. V. McKenry. 1989. Procedures for field and greenhouse screening of *Prunus* genotypes for resistance and tolerance to root lesion nematode. Journal of the American Society for Horticultural Science 114:30–35.

16. Dalmasso, A., P. Castagnone-Sereno, M. Bongiovanni, and A. de Jong. 1991. Acquired virulence in the plant parasitic nematode *Meloidogyne incognita*. 2. Two-dimensional analysis of isogenic isolates. Revue de Nématologie 14:305–308.

17. de Jong, J. H., W. Heijbroek, and B. Mans. 1981. Identification of the alien chromosome in cyst nematode resistant trisomics of *Beta vulgaris* × *B. procumbens*. Pp. 49–54 *in* J. H. de Jong. Investigation into chromosome morphology of sugarbeet and related wild species. Ph.D. thesis, University of Amsterdam, The Netherlands.

18. Di Vito, M. 1983. Reaction of *Beta* spp. to root-knot nematodes. Journal of Nematology 15:144–145.

19. Fassuliotis, G. 1982. Plant resistance to root-knot nematodes. Pp. 33–49 *in* R. D. Riggs, ed. Nematology in the southern region of the United States. Southern Cooperative Series Bulletin 276, Arkansas Agricultural Experiment Station, Fayetteville.

20. Fassuliotis, G. 1987. Genetic basis of plant resistance to nematodes. Pp. 364–371 *in* J. A. Veech and D. W. Dickson, eds. Vistas on nematology. Society of Nematologists.

21. Fassuliotis, G., and D. P. Bhatt. 1982. Potential of tissue culture for breeding root-knot nematode resistance into vegetables. Journal of Nematology 14:10–14.

22. Fehr, W. R. 1987. Principles of cultivar development, vol. 1. Theory and technique. New York: Macmillan.

23. Gilbert, J. C., and D. C. McGuire. 1956. Inheritance of resistance to severe root knot from *Meloidogyne incognita* in commercial type tomatoes. Proceedings of the American Society for Horticultural Science 68:437–442.

24. Gleddie, S. W., W. A. Keller, and G. Setterfield. 1986. Production and characterization of somatic hybrids between *Solanum melongena* L. and *S. sisymbriifolium* Lam. Theoretical and Applied Genetics 71:613–621.

25. Harris, A. R. 1983. Resistance of some *Vitis* rootstocks to *Xiphinema index*. Journal of Nematology 15:405–409.

26. Harris, A. R. 1990. Evaluating resistance to ectoparasitic nematodes. Pp. 67–86 *in* J. L. Starr, ed. Methods for evaluating plant species for resistance to plant-parasitic nematodes. Society of Nematologists.

27. Hawkes, J. G. 1977. The importance of wild germplasm in plant breeding. Euphytica 26:615–621.

28. Heijbroek, W. 1990. The effect of homozygous and heterozygous nematode resistant sugarbeet hybrids on the population development of *Heterodera schachtii* and *Heterodera trifolii* f.sp. *betae*. Nematologica 36:358 (Abstr.).

29. Heijbroek, W., A. J. Roelands, and J. H. de Jong. 1983. Transfer of resistance to beet cyst nematode from *Beta patellaris* to sugar beet. Euphytica 32:103–108.

30. Heijbroek, W., A. J. Roelands, J. H. de Jong, C. Van Hulst, A. H. L. Schoone, and R. G. Munning. 1988. Sugar beets homozygous for resistance to beet cyst nematode (*Heterodera schachtii* Schm.), developed from monosomic additions of *B. procumbens* to *B. vulgaris*. Euphytica 38:121–131.

31. Hijner, J. A. 1951. De gevoeligheid van wilde bieten voor het bietecystenaaltje (*Heterodera schachtii*). Mededelingen Instituut voor Rationale Suikerproductie 21:1–13.

32. Hussey, R. S., C. W. Mims, and S. W. Westcott. 1991. Ultrastructure of food cells in roots parasitized by *Criconemella xenoplax*. Journal of Nematology 23:533–534 (Abstr.).

33. Janssen, R., J. Bakker, and F. J. Gommers. 1990. Selection of virulent and avirulent lines of *Globodera rostochiensis* for the H1 resistance gene in *Solanum tuberosum* ssp. *andigena* CP1673. Revue de Nématologie 13:265–268.

34. Jarquin-Barberena, H., A. Dalmasso, G. de Guiran, and M.-C. Cardin. 1991. Acquired virulence in the plant parasitic nematode *Meloidogyne incognita*. 1. Biological analysis of the phenomenon. Revue de Nématologie 14:299–303.

35. Johnson, R. 1983. Genetic background of durable resistance. Pp. 5–26 *in* F. Lamberti, J. M.

Waller, and N. A. Van der Graaff, eds. Durable resistance in crops. New York: Plenum.

36. Jones, F. G. W. 1985. Modelling multigenic resistance to potato cyst nematodes. OEPP/EPPO Bulletin 15:155–166.

37. Jones, F. G. W., D. M. Parrott, and J. N. Perry. 1981. The gene-for-gene relationship and its significance for potato cyst nematodes and their solanaceous hosts. Pp. 23–36 *in* B. M. Zuckerman and R. A. Rohde, eds. Plant parasitic nematodes, vol. 3. New York: Academic Press.

38. Jung, C., and G. Wricke. 1987. Selection of diploid nematode-resistant sugar beet from monosomic addition lines. Plant Breeding 98:205–214.

39. Jung, C., M. Kleine, F. Fischer, and R. G. Herrmann. 1990. Analysis of DNA from a *Beta procumbens* chromosome fragment in sugar beet carrying a gene for nematode resistance. Theoretical and Applied Genetics 79:663–672.

40. Kaloshian, I., P. A. Roberts, and I. J. Thomason. 1989. Resistance in *Triticum* and *Aegilops* spp. to *Meloidogyne chitwoodi*. Supplement to Journal of Nematology 21:632–634.

41. Kaloshian, I., P. A. Roberts, J. G. Waines, and I. J. Thomason. 1990. Inheritance of resistance to root-knot nematodes in *Aegilops squarrosa* L. Journal of Heredity 81:170–172.

42. Knott, D. R., and J. Dvorak. 1976. Alien germ plasm as a source of resistance to disease. Annual Review of Phytopathology 14:211–235.

43. Leudders, V. D. 1983. Genetics of the cyst nematode–soybean symbiosis. Phytopathology 73:944–948.

44. Litz, R. E. 1982. Germplasm modification and its potential for finding new sources of resistance to diseases. Journal of Nematology 18:18–22.

45. Löptien, H. 1984. Breeding nematode resistant lines. 1. Development of resistant alien additions by crosses between *Beta vulgaris* L. and wild species of section *Patellares*. Zeitschrift für Pflanzenzüchtung 92:208–220.

46. McFarlane, J. S., H. Savitsky, and A. E. Steele. 1982. Breeding for resistance to the sugarbeet nematode. Journal of the American Society of Sugarbeet Technologists 21:311–323.

47. McKenry, M. V., and P. A. Roberts. 1985. Phytonematology study guide. Publication 4045, Division of Agriculture and Natural Resources, University of California, Oakland.

48. Meredith, C. P., L. A. Lider, D. J. Raski, and N. L. Ferrari. 1982. Inheritance of tolerance to *Xiphinema index* in *Vitis* species. American Journal of Enology and Viticulture 33:154–158.

49. Müller, J. 1990. Investigations on the occurrence of pathotypes in *Heterodera schachtii*. Nematologica 36:373 (Abstr.).

50. Müller, J. 1990. The effect of nematode-resistant sugar-beet hybrids on the population dynamics of *Heterodera schachtii* under field conditions. Nematologica 36:373 (Abstr.).

51. Netscher, C., and R. A. Sikora. 1990. Nematode parasites of vegetables. Pp. 273–283 *in* M.Luc, R. A. Sikora, and J. Bridge, eds. Plant parasitic nematodes in subtropical and tropical agriculture. Wallingford, UK: CAB International.

52. Okie, W. R., A. P. Nyczepir, and C. C. Reilly. 1987. Screening of peach and other *Prunus* species for resistance to ring nematode in the greenhouse. Journal of the American Society for Horticultural Science 112:67–70.

53. Omwega, C. O., and P. A. Roberts. 1992. Inheritance of resistance to *Meloidogyne* spp. in common bean and the genetic basis of its sensitivity to temperature. Theoretical and Applied Genetics 83, in press.

54. Omwega, C. O., I. J. Thomason, and P. A. Roberts. 1990. A single dominant gene in common bean conferring resistance to three root-knot nematode species. Phytopathology 80:745–748.

55. Omwega, C. O., I. J. Thomason, P. A. Roberts, and J. G. Waines. 1989. Identification of new genes for resistance to root-knot nematodes in *Phaseolus*. Crop Science 29:1463–1468.

56. Rebois, R. V., B. J. Eldridge, J. M. Good, and A. K. Stoner. 1973. Tomato resistance and susceptibility to the reniform nematode. Plant Disease Reporter 57:169–172.

57. Rebois, R. V., A. E. Steele, A. K. Stoner, and B. J. Eldridge. 1977. A gene for resistance to *Rotylenchulus reniformis* in tomato, and a possible correlation with resistance to *Heterodera schachtii*. Journal of Nematology 9:280–281 (Abstr.).

58. Roberts, P. A. 1982. Plant resistance in nematode pest management. Journal of Nematology 14:24–33.

59. Roberts, P. A. 1990. Resistance to nematodes: Definitions, concepts and consequences. Pp. 1–15 *in* J. L. Starr, ed. Methods for evaluating plant species for resistance to plant-parasitic nematodes. Society of Nematologists.

60. Roberts, P. A., and D. M. May. 1986. *Meloidogyne incognita* resistance characteristics in tomato genotypes developed for processing. Journal of Nematology 18:353–359.

61. Roberts, P. A., and I. J. Thomason. 1989. A review of variability in four *Meloidogyne* spp. measured by reproduction on several hosts including *Lycopersicon*. Agricultural Zoology Reviews 3:225–252.

62. Roberts, P. A., A. Dalmasso, G. B. Cap, and P. Castagnone-Sereno. 1990. Resistance in *Lycopersicon peruvianum* to isolates of *Mi* gene-compatible *Meloidogyne* populations. Journal of Nematology 22:585–589.

63. Russell, G. E. 1978. Plant breeding for pest and disease resistance. London: Butterworths.

64. Sasser, J. N., and D. W. Freckman. 1987. A world perspective on nematology: The role of the society. Pp. 7–14 *in* J. A. Veech and D. W. Dickson, eds. Vistas on nematology. Society of Nematologists.

65. Savitsky, H. 1973. Meiosis in hybrids between *Beta vulgaris* L. and *B. procumbens* Chr. Sm. and transmission of sugarbeet nematode resistance. Genetics (Supplement) 74:241.

66. Savitsky, H. 1975. Hybridization between *Beta vulgaris* and *B. procumbens* and transmission of nematode (*Heterodera schachtii*) resistance to sugarbeet. Canadian Journal of Genetics and Cytology 17:197–209.

67. Shepherd, R. L. 1982. Genetic resistance and

its residual effects for control of the root-knot nematode-Fusarium wilt complex in cotton. Crop Science 22:1151–1155.

68. Sidhu, G. S., and J. M. Webster. 1981. The genetics of plant-nematode parasitic systems. Botanical Review 47:387–419.

69. Slana, L. J., and J. R. Stavely, 1981. Identification of the chromosome carrying the factor for resistance to *Meloidogyne incognita* in tobacco. Journal of Nematology 13:61–66.

70. Smith, P. G. 1944. Embryo culture of a tomato species hybrid. Proceedings of the American Society for Horticultural Science 44:413–416.

71. Speckmann, G. J., and T. S. M. de Bock. 1982. The production of alien monosomic additions in *Beta vulgaris* as a source for the introgression of resistance to the beet root nematode (*Heterodera schachtii*) from *Beta* species of the section *Patellares*. Euphytica 31:313–323.

72. Speckmann, G. J., T. S. M. de Bock, and J. H. de Jong. 1985. Monosomic additions with resistance to beet cyst nematode obtained from hybrids of *Beta vulgaris* and wild *Beta* species of the section *Patellares*. 1. Morphology, transmission and level of resistance. Zeitschrift für Pflanzenzüchtung 95:74–83.

73. Starr, J. L. 1990. Methods for evaluating plant species for resistance to plant-parasitic nematodes. Society of Nematologists.

74. Steele, A. E., H. Toxopeus, and W. Heijbroek. 1983. Susceptibility of plant selections to *Heterodera schachtii* and a race of *H. trifolii* parasitic on sugarbeet in The Netherlands. Journal of Nematology 15:281–288.

75. Turner, S. J. 1990. The identification and fitness of virulent potato cyst nematode populations (*Globodera pallida*) selected on resistant *Solanum vernei* hybrids for up to eleven generations. Annals of Applied Biology 117:385–397.

76. Van der Plank, J. E. 1968. Disease resistance in plants. New York: Academic Press.

77. Watts, V. M. 1947. The use of *Lycopersicon peruvianum* as a source of nematode resistance in tomatoes. Proceedings of the American Society for Horticultural Science 49:233–234.

78. Whitehead, A. G. 1991. Selection for virulence in the potato cyst-nematode, *Globodera pallida*. Annals of Applied Biology 118:395–402.

79. Williamson, V. M., J.-Y. Ho, and H. M. Ma. 1992. Molecular transfer of resistance genes. Journal of Nematology 24:234–241.

80. Wyatt, J. E., G. Fassuliotis, J. C. Hoffman, and J. R. Deakin. 1983. 'Nemasnap' snap bean. HortScience 18:776.

81. Yu, M. H. 1984. Transmission of nematode resistance in the pedigree of homozygous resistant sugarbeet. Crop Science 24:88–91.

82. Yu, M. H., and A. E. Steele. 1981. Host–parasite interaction of resistant sugarbeet and *Heterodera schachtii*. Journal of Nematology 13:206–212.