Mini-review

Received: 12 March 2015 Revised: 4 August 2015 Accepted article published: 7 August 2015 Published online in Wiley Online Library:

(wileyonlinelibrary.com) DOI 10.1002/ps.4091

Host genetic resistance to root-knot nematodes, *Meloidogyne* spp., in Solanaceae: from genes to the field

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Abstract

Root-knot nematodes (RKNs) heavily damage most solanaceous crops worldwide. Fortunately, major resistance genes are available in a number of plant species, and their use provides a safe and economically relevant strategy for RKN control. From a structural point of view, these genes often harbour NBS–LRR motifs (i.e. a nucleotide binding site and a leucine rich repeat region near the carboxy terminus) and are organised in syntenic clusters in solanaceous genomes. Their introgression from wild to cultivated plants remains a challenge for breeders, although facilitated by marker-assisted selection. As shown with other pathosystems, the genetic background into which the resistance genes are introgressed is of prime importance to both the expression of the resistance and its durability, as exemplified by the recent discovery of quantitative trait loci conferring quantitative resistance to RKNs in pepper. The deployment of resistance genes at a large scale may result in the emergence and spread of virulent nematode populations able to overcome them, as already reported in tomato and pepper. Therefore, careful management of the resistance genes available in solanaceous crops is crucial to avoid significant reduction in the duration of RKN genetic control in the field. From that perspective, only rational management combining breeding and cultivation practices will allow the design and implementation of innovative, sustainable crop production systems that protect the resistance genes and maintain their durability.

Supporting information may be found in the online version of this article.

Keywords: cropping system; genetic background; plant resistance; pyramiding; quantitative trait loci; solanaceous crops

1 INTRODUCTION

The Solanaceae family comprises between 3000 and 4000 species in some 95 genera, the largest of which is *Solanum*, with 1500–2000 species, almost half the diversity of the family. Many of these species have considerable economic importance as crops, including tomato (*Solanum lycopersicum*), potato (*S. tuberosum*), pepper (*Capsicum annuum*), eggplant (*S. melongena*) and tobacco (*Nicotiana tabacum*). For example, potato represents more than 42% of the roots and tubers produced worldwide for food, while tomato, pepper and eggplant together account for more than 20% of the vegetables produced worldwide and more than 50% of the harvested area of vegetables (Table 1). These crops are cultivated in most tropical and temperate parts of the world, in open fields or under plastic tunnels and greenhouses, in the context of either sustainable agriculture or high-input commercial production.

Like other plants, solanaceous crops are the targets of a wide range of pathogens and pests, including nematodes. In particular, root-knot nematodes (RKNs) of the genus *Meloidogyne* are among the most damaging nematode species attacking these plants. The typical morphological response of compatible plants to infection by RKNs is root galling (Fig. 1), which alters water and nutrient uptake by the root system, resulting in a subsequent reduction in plant growth and yield.[1] In addition, the quality of the harvest may also be significantly altered in the case of root or tuber production, e.g. potato (Fig. 1). Because of the severity of the disease they cause on a broad range of plant hosts, RKNs have been ranked first among the top ten plant-parasitic nematodes,[2] and *M. incognita* has been regarded as possibly 'the single most damaging crop pathogen in the world'.[3]

Successful control of plant nematodes is often the result of the integrated use of various pest management strategies, e.g. chemical pesticides, resistant crop cultivars and cultural practices. However, some of these approaches are becoming increasingly unsatisfactory. Although widely practised, crop rotation is of limited value in the case of RKNs because of their extremely wide host

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Table 1. FAO world statistics for major Solanaceous crops for the year 2012[a]

	Production (10^6 t)	Percentage of world production	Area harvested (10^6 ha)	Percentage of world harvested area
Eggplant (*Solanum melongena*)	48.42	4.38[c]	1.85	3.23[e]
Pepper[b](*Capsicum annuum*)	34.52	3.12[c]	3.90	6.81[e]
Potato (*S. tuberosum*)	364.81	45.08[d]	19.20	34.69[f]
Tobacco (*Nicotiana tabacum*)	6.33	–	3.93	–
Tomato (*S. lycopersicum*)	161.79	14.63[c]	4.80	8.38[e]

[a] Information source: http://faostat3.fao.org/compare/E
[b] Including dry and green pepper.
[c,e] Percentage of world production and harvested area, respectively, of vegetables.
[d,f] Percentage of world production and harvested area, respectively, of roots and tubers.

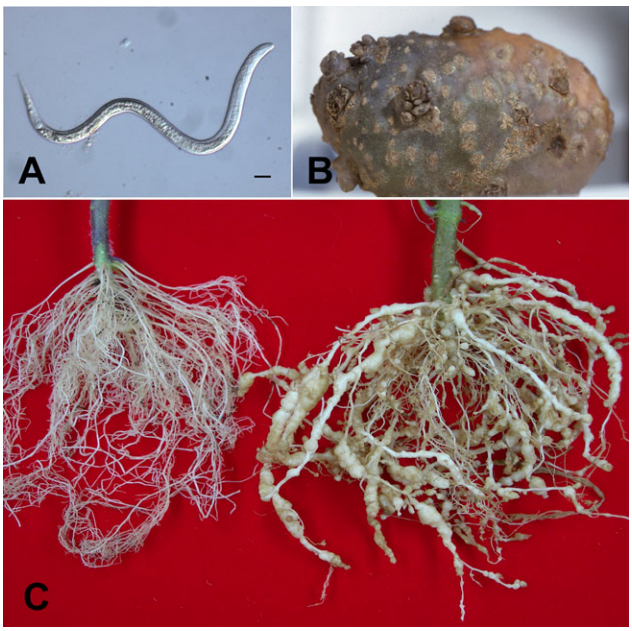

Figure 1. (A) Infective second-stage juvenile of *Meloidogyne incognita*. Bar = 15 µm. (B) Galls on a potato tuber infested with *M. chitwoodi*. (C) Root systems of a susceptible (right) versus resistant (left) tomato cultivar inoculated with *M. incognita*.

range encompassing the vast majority of the flowering plants.[3] The environmental and health concerns raised against nematicides and soil fumigants has led to the withdrawal of most of these chemicals in many locations, which further emphasises the need for alternative and durable control strategies. In this context, plant resistance appears to be the most attractive approach for controlling nematode populations from environmental, economic and practical points of view. Indeed, natural resistance (R) genes against some RKNs have been identified, mapped and cloned in a number of plant species, including Solanaceae.[4] Transgenic approaches for artificial RKN resistance have also been proposed, involving a battery of effectors active against the nematode or its feeding site within the root.[5–7]

Review articles have covered comprehensively the abundant literature devoted to the structure and function of R genes in solanaceous plants,[8] and the mechanisms of plant resistance to nematodes.[4,7] Here, our aim is to assess the diversity of the natural R genes against RKNs currently cloned or mapped in wild and cultivated Solanaceae, and to evaluate the prospects for their introgression into new cultivars using classical breeding or transgenic expression. Further information on the possible limitations in the use of these R genes under field conditions will also be provided, in order to give end-users (i.e. plant breeders and growers) objective elements and prospective comments about the development and implementation of natural R gene-based control strategies against RKNs in solanaceous crops.

2 MAJOR R GENES AGAINST RKNS IN WILD AND CULTIVATED SOLANACEAE

Although nematode resistance in general can result from (the combination of) several types of genetic determinant, including major/minor genes and quantitative trait loci (QTLs),[4,9] RKN resistance in solanaceous crops is mainly dominant and conferred by single major dominant genes (supporting information Table S1). In addition, one recessive gene has been hypothesised in the pepper cultivar 'Carolina Wonder',[10] associated with the dominant R gene named *N*.[11,12] Very recently, four QTLs have also been identified in pepper (see detailed discussion below).

Mapping studies indicate that genes conferring resistance to various pathogens, including RKNs, are often organised in clusters in Solanaceae. For example, the *N* and the *Me* genes (i.e. *Me1, Me3, Me4*, *Mech1* and *Mech2*) conferring resistance to RKNs, two QTLs conferring resistance to *Phytophthora capsici* and potyviruses PVY (0) and PVY (1, 2) and the *Bs2* gene conferring resistance to the bacterium *Xanthomonas campestris* pv. *vesicatoria* have been mapped to the same region of the pepper P9 chromosome.[13–16] Similarly, the *Mi-3* and *Mi-5* RKN R genes and the powdery mildew *Leveillula taurica* R gene *Lv* have also been mapped in a single cluster on the T12 chromosome of tomato.[17–19] The nematode resistance genes *Gpa2* and *MfaXII*, which control pathotype *Pa2* of the potato cyst nematode *Globodera pallida* and the RKN *M. fallax*[20,21] respectively, have been mapped to the distal end of potato chromosome XII, together with a R gene to potato virus *X (Rx1)*.[22,23] As the presence of transposable elements has been correlated both with large-scale genomic rearrangements[24,25] and with genomic clusters carrying R genes against several plant pathogens, including oomycetes and bacteria,[26,27] they may play a role in the creation and maintenance of such clusters in Solanaceae.[28] As an example, the sequencing of the P9 chromosome of pepper (carrying the *Me* gene cluster) highlighted how genome expansion due to transposable elements and duplication lead to the emergence of new genes and functions or 'neofunctionalisation'.[29] In this genomic region, 82 paralogues of the *Bs2* family of R genes were identified in

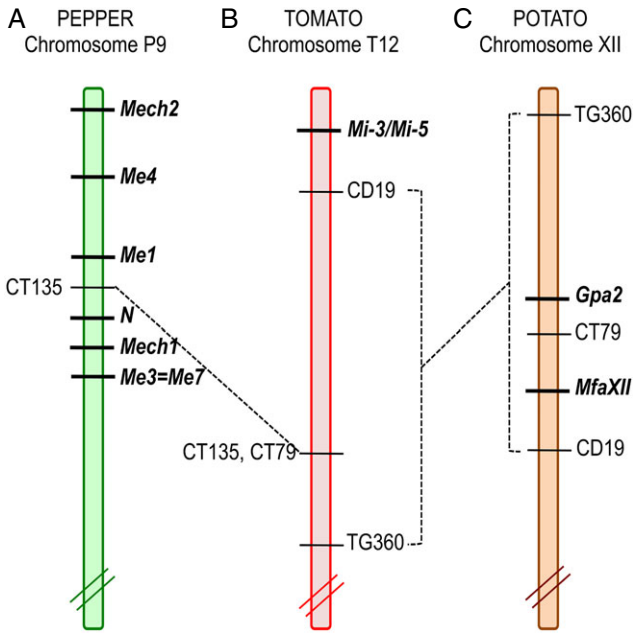

Figure 2. Schematic representation of the comparative mapping of nematode resistance loci in pepper, tomato and potato. Position of nematode R genes as determined from linkage to common markers on (A) an integrated map of the pepper chromosome P9,[103] (B) the tomato chromosome T12[17,18] and (C) the potato chromosome XII.[20,74] The putative alignment of markers between A, B and C is indicated by dotted lines. Distances are given in centimorgans (cM).

pepper, whereas in the corresponding genomic region only three paralogues where found in potato (namely the *Rx*, *Rx-2* and *Gpa-2* genes) and two paralogs with unknown function in tomato.[30] From an evolutionary point of view, the clustering of R genes may facilitate the coordination of plant defences against various pathogens and the generation of new specificities towards an ever-changing array of pathogens.[23,31,32]

Comparative studies have shown that homologues of cloned R genes map to syntenic positions in solanaceous genomes, suggesting that both the sequence and position of these genes are conserved.[28,33,34] For example, R genes in several *Solanum* species against alfalfa mosaic virus, Gemini viruses, bacterial pathogens, the oomycete *Phytophthora infestans* and the fungus *Oidium neolycopersici* map to the tomato *Mi-1* region of chromosome 6.[35–39] The *Me* and *N* genes of pepper have been assigned to an interval equivalent to that containing *Mi-3* and *Mi-5* in tomato in the vicinity of the RFLP marker CT135, and *Gpa2* and *MfaXII* in potato in the vicinity of CT79, which cosegregates with CT135 in tomato (Fig. 2).[16] These comparative mapping data suggest that the three clusters of R genes conferring resistance to nematodes are located in orthologous genomic regions of pepper, tomato and potato, and that these regions are conserved within and between species in these solanaceous crops. From a structural point of view, evidence is accumulating that NBS–LRR motifs [i.e. a nucleotide binding site (NBS) and a leucine rich repeat (LRR) region near the carboxy terminus] are common in R genes against nematodes, including R genes from solanaceous species, assuming that differential numbers of repeats and of TE sequences may disturb the colinearity in microsyntenic genomic regions. The R genes containing such NBS–LRR motifs may have evolved by divergent evolution from an individual ancestral gene in Solanaceae.[40]

3 PLANT GENETIC BACKGROUND, QTLS AND THE EXPRESSION OF R GENES

Even if the RKN resistance conferred by major R genes is theoretically regarded as complete, variation is regularly observed in the field, with some resistant plants/accessions exhibiting a low but varying number of egg masses on their root systems. In some studies, a dosage effect of the R gene has been proposed to explain these observations, with expression of the resistance being more effective in homozygous versus heterozygous plant genotypes. Although this hypothesis was raised for the tomato *Mi-1.2* R gene,[41,42] other experiments led to the opposite conclusion for both the tomato *Mi-1.2* and the pepper *Me3* R genes when the R gene was introgressed in homogeneous genetic backgrounds.[43–45]

In solanaceous crops, the genetic background associated with the R gene(s) [i.e. concomitant occurrence of genes (QTLs) with quantitative effects] is of prime importance for both the expression of the resistance and its durability, as shown for a wide range of pathogens, including viruses, oomycetes, fungi and nematodes.[45–51] For example, in a combination of field and greenhouse systems, the durability of resistance to the cyst nematode *Globodera pallida* was shown to be variable in different potato genotypes harbouring the same resistance factor but differing in their genetic background.[49] However, only a few QTLs involved in RKN resistance have been identified in a few diverse crops, e.g. sweet potato, cotton, soybean and peanut,[52–55] and none in the Solanaceae. Very recently, quantitative resistance to RKNs was detected in some pepper accessions.[45,56] A QTL analysis for resistance to the three main RKN species, *M. incognita*, *M. arenaria* and *M. javanica*, in a cross between a partially resistant and a susceptible pepper line yielded four new QTLs localised on two separate clusters: three QTLs clustered on chromosome P1 with each active against one of the three RKN species, and one QTL active against *M. javanica* on chromosome P9 (Barbary *et al.*, unpublished). Interestingly, this is the first time that RKN resistance factors have been identified on pepper chromosome P1. The favourable allele at these QTLs originated from the partially resistant pepper genotype. This same genotype was previously shown to contain a genetic background increasing the expression of the *Me1* or *Me3* major genes.[45] Thus, pyramiding such QTLs with the major R gene(s) into one cultivar is expected to provide a complete and durable resistance by taking simultaneous advantage of the resistance provided by major R genes and the reduction in the level of infestation by QTLs. Such genetic combinations in resistant cultivars should decrease the risk of resistance breakdown by RKNs; indeed, reducing the number of egg masses produced on the roots of resistant cultivars should reduce the risk of emergence and further selection of adapted variants and consequently increase the durability of the R genes, as demonstrated by several studies on different pathosystems.[46,57] Therefore, it is of crucial importance for breeders to take into account the genetic background into which they introgress major R genes, in order to increase their efficiency and likely improve the longevity of new elite varieties released on the market.

4 BIOTECHNOLOGICAL APPROACHES IN BREEDING PROGRAMMES

Although the availability of R genes against RKNs is rather good in the Solanaceae, most of these genes originate from wild relatives of the cultivated species, and their introgression into elite

cultivars via traditional breeding, along with the elimination of undesirable agronomic traits that may be tightly linked to them, is a laborious and time-consuming process that can take up to 10–15 years. For example, even though at least nine R genes for RKN resistance have been identified in wild tomato and more than ten in wild pepper accessions, only two of them are widely available in commercial varieties, i.e. *Mi-1.2* and *N* in tomato and pepper respectively.[11,12,57–63] In some instances, however, this process can be considerably accelerated by using molecular markers linked to the R gene of interest, and marker-assisted selection (MAS) has been estimated to reduce the time to market by 50–70%.[64] For example, several PCR-based markers (CAPS, RAPD and SCAR) linked to the *Mi-1.2* gene have been routinely used in tomato breeding programmes for selecting for RKN resistance (supporting information Table S1). Similarly, STS markers closely linked to the $R_{Mc1(blb)}$ gene encoding resistance to the Columbia RKN (*M. chitwoodi*) have provided an efficient alternative to greenhouse and field phenotypic screening to follow the introgression of $R_{Mc1(blb)}$ into advanced potato breeding lines.[65] Moreover, with the recent advances in genome sequencing, new and more informative PCR-based markers [e.g. single nucleotide polymorphisms (SNPs)] will further facilitate the use of MAS in plant breeding, including solanaceous crops. In this connection, the recent release of the reference genome sequences of potato, tomato and pepper (available at http://solgenomics.net/genomes/) provides pertinent information and tools to align genomic regions of interest and explore syntenic regions among the Solanaceae, thereby facilitating the establishment of more effective breeding programmes.[29,30,66,67]

Alternatively, in order to shorten the duration of classical introgression steps or to overcome the problems linked to interspecific crosses, the transfer and transgenic expression of natural R genes into related susceptible crops have been investigated in initial proof-of-concept studies employing the tomato *Mi-1.2* gene as a model system. Compared with induced translocation and introgression breeding, cisgenesis (i.e. transfer of a gene of interest from the same or a crossable botanical species) is considered as an improvement for gene transfer.[68] However, when this strategy was applied to tomato, a reduction in *Mi-1.2*-mediated RKN resistance was noted in the T2 transformed lines, and was more pronounced in the T3 generation. In addition, the variability of instability in resistance among clonally propagated cuttings indicated that resistance levels may be influenced by epigenetic effects.[69] Heterologous *Mi-1.2* transformation of other Solanaceae led to contrasting results, with RKN resistance conferred to transgenic eggplant,[70] but not to tobacco.[71] More recently, ectopic expression of *Mi-1.2* conferred resistance to RKNs in lettuce.[72] Overall, there has been limited success with transgenic expression of natural R genes from and in solanaceous crops. Alternative biotechnological approaches under investigation mostly concern (i) the overexpression of peptides or proteins that disrupt essential phases of the plant–nematode interaction (e.g. chemoreception, digestion) or (ii) the plant-delivered RNAi to silence nematode genes essential for the parasite to complete its life cycle.[5]

5 PRACTICAL LIMITATIONS OF THE USE OF NATURAL R GENES IN SOLANACEAE

Although the deployment of natural R genes may be the most attractive strategy for controlling RKN populations in solanaceous crops, a number of factors potentially limit their effective use. Firstly, prospecting for and evaluating new genetic resources are long processes, with no guarantee that resistance will be identified: presently, no major resistance against *M. enterolobii* has been found in Solanaceae. In several cases, resistance factors have been identified in wild relatives (supporting information Table S1) with poor cross-compatibility with the targeted cultivated species, limiting exploitation in breeding programmes. In potato, several wild species have been the source of RKN resistance, including, among others, *S. sparsipilum* for resistance to *M. incognita* and *M. fallax*[21,73] and *S. bulbocastanum* for resistance to *M. chitwoodi* and *M. hapla*.[74] In tomato, broad searches of wild germplasm identified several sources of RKN resistance, almost all in the heterogeneous *S. peruvianum* complex, which exhibited a high level of incompatibility with the cultivated species, *S. lycopersicum*.[71] The most commonly used resistance gene, *Mi-1.2*, was introgressed into *S. lycopersicum* through *in vitro* culture of immature hybrid embryos that permitted the recovery of one F1 interspecific hybrid,[75] which has long been considered as the sole source of all RKN resistance in currently available fresh-market and processing tomato cultivars.[71] Moreover, in addition to the difficulties encountered in successfully crossing wild and cultivated relative species, alleles with unfavourable horticultural traits linked to RKN resistance in the original resource (linkage drag) may slow down progress.

None of the currently known R genes in Solanaceae confers resistance to all RKN species, and thus the more or less narrow range of controlled species constitutes another practical limitation of resistant cultivars to manage these pests in infested fields. Interestingly, the most frequently used R genes in breeding, i.e. *Mi-1.2* in tomato and *N*, *Me1* and *Me3* in pepper, control the major RKN species *M. arenaria*, *M. incognita* and *M. javanica*.[71,76,77] However, other R genes are more specific and confer resistance to one single RKN species (e.g. *Mech1* or *Mech2* against *M. chitwoodi* in *C. annuum*),[16] or even to one or a few isolates from one species (e.g. *Me2*, *Me4* and *Me5* in *C. annuum*, which are active against a few isolates from only one species).[77,78] In addition, some major RKN species are not controlled by the R genes identified so far in solanaceous crops; for example, no resistance has been characterised in tomato against *M. hapla*.[71] Of particular concern is the case of *M. enterolobii*, a tropical, invasive RKN species able to develop and reproduce on most solanaceous crops, including resistant tomatoes (*Mi-1.2* gene), potatoes (*Mh* gene) and bell and sweet peppers (*N*, *Tabasco*, *Me*(s) gene).[79] Very recently, one *C. chinense* accession was considered to be resistant to *M. enterolobii* in experimental tests,[80] but this promising result still requires validation under agronomic conditions. Obviously, such variability in the specificity of the R genes available limits the use of resistant cultivars to manage RKNs, and should be taken into account when experimentally evaluating new plant genotypes for resistance, which requires an unambiguous identification of the nematodes used as inoculum source.

Although the expression of most R genes from solanaceous crops is not affected by high soil temperatures (e.g. *Me1* and *Me3* from pepper are still active at 42 °C),[77] there are a few notable exceptions. Probably the most documented case is that of the tomato *Mi-1.2* gene, which is inactive at constant soil temperatures above 28 °C,[81] a temperature common in tropical regions or greenhouses. Also, bell pepper cultivars harbouring the *N* gene exhibited a partial loss of resistance to RKNs at 28 and 32 °C in growth chamber experiments at constant soil temperatures.[60] However, resistance of the same cultivars did not break when tested in M. incognita-infested fields in Florida, where soil temperatures exceeded 30 °C,[63] thereby indicating that these cultivars represent viable options for managing *M. incognita* in bell pepper in subtropical environments.

Several R genes against RKNs have been routinely deployed in commercial cultivars of solanaceous crops, the most widely used being the tomato *Mi-1.2* gene.[71] For more than 70 years now, although some other R genes have been identified in the wild tomato *S. peruvianum*,[58] *Mi-1.2* has been the only source of resistance in tomato production against RKNs. Clearly, the extensive use of the same R gene at a large scale may result in the emergence and spread of virulent nematode populations able to overcome it, and *Mi-1.2*-resistance-breaking populations in tomato have been discovered, as have *N*- and *Me3*-resistance-breaking populations in pepper.[82–87] Such ability to overcome plant resistance may thus constitute a severe limitation for RKN control. However, it should be noted here that selection for virulence may not be successful with all RKN populations or against all resistance genes.[88] Indeed, in practice, *Mi-1.2* resistance remains efficient in most agronomical situations, in spite of its continuous use for decades, and should be considered as a very stable R gene in terms of durability at the worldwide scale. This stability may partly result from the fact that RKN species are soil organisms with limited active dissemination, and that the major species are asexual (parthenogenetic) organisms with poor capacity for gene flow and adaptive evolution.[89] However, recent advances in the genomics of RKNs suggested that mechanisms other than genetic recombination may be the source of phenotypic variability in these clonal organisms (e.g. gene duplications, epigenetic inheritance, etc.),[90,91] which could contribute to their ability to adapt to poor environmental conditions.

Major R genes are a rare resource in plant germplasm, and long-term ability to use them in management is essential. Although quantitative resistance occurs much more frequently than R-gene-based resistance in pepper germplasm collections,[92] exploitation of QTLs is much more complex in breeding programmes. Without careful management, the duration of commercial exploitation of most R genes available in solanaceous crops could be significantly reduced.

6 R GENE DEPLOYMENT IN AGROSYSTEMS: USE WITH CARE!

Integrated management strategies are required to avoid/reduce the negative effects associated with long-term use of such resistant cultivars, in order to preserve their durability. In the favourable but uncommon case where several R genes are available in one crop species, as in pepper, different spatiotemporal deployment strategies may be considered for utilisation, e.g. sequential use of the available R genes, mixtures, alternation or pyramiding. In that respect, we experimentally evaluated such strategies in a model system with the *Me1* and *Me3* R genes of pepper. Under field conditions over 3 years, the efficiency and the durability of resistance were assessed in a protected crop system with pepper as the summer crop and lettuce as the winter crop. Whatever the R gene(s) and the management strategy considered, resistant cultivars significantly reduced nematode infestations.[93] However, differences were observed when looking at three components of the cropping system (i.e. efficiency of resistance, durability of resistance and sustainability of crop rotation), which provided the same hierarchy of the tested strategies: pyramiding > alternation > mixtures > sequential use of a single R gene introgressed in a susceptible background.[93] In particular, pyramiding two major R genes that differ in their mechanisms (as is the case for *Me1* and *Me3* in pepper)[94,95] into a single cultivar seemed the most secure and durable strategy after 3 years of experimentation.[93] Interestingly, the recent use of a stochastic model of pathogen adaptation dynamics in response to quantitative resistance showed that the combination of QTLs affecting distinct pathogen traits was indeed durable, especially when the restoration process of repressed traits was antagonistic or independent.[96]

In cases where RKN-resistant elite cultivars are not commercially available, grafting plants on resistant rootstocks has been considered as a possible alternative, with the additional advantage that grafting may improve tolerance of vegetables to abiotic stresses.[97] For example, in south-eastern Spain, under greenhouse crop conditions, a close relationship was found between pepper rootstock resistance to *M. incognita* and yield, the more resistant accessions showing the better agronomic performance as rootstocks.[56] However, for some of the resistant genotypes tested, two successive years of growing grafted plants in a naturally *M. incognita*-infested greenhouse was sufficient to overcome resistance,[87] which again highlights the need for careful management of such genetic resources.

At the operational level, either for producers or plant breeders, the message here is that genetic resistance should be considered as one individual weapon only among the several available to fight RKNs, and that only the combination of genetic resistance with cultivation practices will allow the design and development of innovative, sustainable crop production systems that protect the R genes and maintain their durability. Among other options, the synergistic use of green manures, cover crops, solarisation, nematicidal plants, etc., in complement with plant resistance may become realistic.[98–101] The current challenge for pathogen control is to design new cropping systems that allow incorporation of alternative techniques with the use of R genes, in order to diversify the selection pressures on the nematode populations while satisfying the farmer constraints.[102] Obviously, this challenge will open new research questions at the crossroads of various disciplines from plant to agricultural sciences, as well as at the crossroads of experimental approaches varying from very controlled experiments to field surveys. A diversity of academic and non-academic partners will be necessary to provide the complementary expertise needed for elaborating such a new paradigm.

ACKNOWLEDGEMENTS

We thank the ANRT (Association Nationale de la Recherche Technologique) foundation and the private breeding companies associated with the CIFRE PhD scholarship to AB. Financial support from INRA (Métaprogramme SMaCH, Gedunem project) is also acknowledged.

SUPPORTING INFORMATION

Supporting information may be found in the online version of this article.

REFERENCES

1 Abad P, Favery B, Rosso MN and Castagnone-Sereno P, Root-knot nematode parasitism and host response: molecular basis of a sophisticated interaction. *Mol Plant Pathol* **4**:217–224 (2003).
2 Jones JT, Haegeman A, Danchin EGJ, Gaur HS, Helder J, Jones MGK *et al.*, Top 10 plant-parasitic nematodes in molecular plant pathology. *Mol Plant Pathol* **14**:946–961 (2013).
3 Trudgill DL and Blok VC, Apomictic, polyphagous root-knot nematodes: exceptionally successful and damaging biotrophic root pathogens. *Annu Rev Phytopathol* **39**:53–77 (2001).
4 Williamson VM and Kumar A, Nematode resistance in plants: the battle underground. *Trends Genet* **22**:396–403 (2006).

5 Atkinson HJ, Lilley CJ and Urwin PE, Strategies for transgenic nematode control in developed and developing world crops. *Curr Opin Biotechnol* **23**:251–256 (2012).
6 Bleve-Zacheo T, Melillo MT and Castagnone-Sereno P, The contribution of biotechnology to root-knot nematode control in tomato plants. *Pest Technol* **1**:1–16 (2007).
7 Fuller VL, Lilley CJ and Urwin PE, Nematode resistance. *New Phytol* **180**:27–44 (2008).
8 Van Ooijen G, van den Burg HA, Cornelissen BJC and Takken FLW, Structure and function of resistance proteins in solanaceous plants. *Annu Rev Phytopathol* **45**:43–72 (2007).
9 Roberts PA, Resistance in nematodes: definition, concepts and consequences, in *Methods for Evaluating Plant Species for Resistance to Plant Parasitic Nematodes,* ed. by Starr JL. Society of Nematologists, Hyattsville, MD, pp. 1–15 (1992).
10 Fery RL and Dukes PD, The inheritance of resistance to the southern root-knot nematode in 'Carolina Hot' cayenne pepper. *J Am Soc Hort Sci* **121**:1024–1027 (1996).
11 Fery RL, Dukes PD and Thies JA, 'Carolina Wonder' and 'Charleston Belle': southern root knot nematode-resistant bell peppers. *HortScience* **33**:900–902 (1998).
12 Thies JA and Fery RL, Characterization of resistance conferred by the *N* gene to *Meloidogyne arenaria* races 1 and 2, *M. hapla*, and *M. javanica* in two sets of isogenic lines of *Capsicum annuum*. *J Am Soc Hort Sci* **125**:71–75 (2000).
13 Thabuis A, Palloix A, Pflieger S, Daubèze AM, Caranta C and Lefebvre V, Comparative mapping of *Phytophthora* resistance loci in pepper germplasm: evidence for conserved resistance loci across Solanaceae and for a large genetic diversity. *Theor Appl Genet* **106**:1473–1485 (2003).
14 Caranta C, Lefebvre V and Palloix A, Polygenic resistance of pepper to potyviruses consists of a combination of isolate-specific and broad-spectrum quantitative trait loci. *Mol Plant–Microbe Interact* **10**:872–878 (1997).
15 Tai TH, Dahlbeck D, Clark ET, Gajiwala P, Pasion R, Whalen MC *et al.*, Expression of the *Bs2* pepper gene confers resistance to bacterial spot disease in tomato. *Proc Natl Acad Sci USA* **96**:14 153–14 158 (1999).
16 Djian-Caporalino C, Fazari A, Arguel MJ, Vernie T, VandeCasteele C, Faure I *et al.*, Root-knot nematode (*Meloidogyne* spp.) *Me* resistance genes in pepper (*Capsicum annuum* L.) are clustered on the P9 chromosome. *Theor Appl Genet* **114**:473–486 (2007).
17 Veremis JC and Roberts PA, Relationship between *Meloidogyne incognita* resistance gene in *Lycopersicon peruvianum* differentiated by heat sensitivity and nematode virulence. *Theor Appl Genet* **93**:950–959 (1996).
18 Yaghoobi J, Kaloshian I, Wen Y and Williamson VM, Mapping a new nematode resistance locus in *Lycopersicon peruvianum*. *Theor Appl Genet* **91**:457–464 (1995).
19 Chunwongse J, Doganlar S, Crossman C, Jiang J and Tanksley SD, High-resolution genetic map of the *Lv* resistance locus in tomato. *Theor Appl Genet* **95**:220–223 (1997).
20 Rouppe van der Voort J, Wolters P, Folkertsma R, Hutten R, van Zanvoort P, Vinke H *et al.*, Mapping of the cyst nematode resistance locus *Gpa2* in potato using a strategy based on co-migrating AFLP markers. *Theor Appl Genet* **95**:874–880 (1997).
21 Kouassi AB, Kerlan MC, Sobczak M, Dantec JP, Rouaux C, Ellisseche D *et al.*, Genetics and phenotypic characterisation of the hypersensitive resistance of *Solanum sparsipilum* to *Meloidogyne incognita*. *Nematology* **6**:389–400 (2005).
22 Ritter E, Debener T, Barone A, Salamini F and Gebhardt C, RFLP mapping on potato chromosomes of two genes controlling extreme resistance to potato virus X (*PVX*). *Mol Gen Genet* **227**:81–85 (1991).
23 Van der Vossen EAG, Rouppe van der Voort JNAM, Kanyuka K, Bendahmane A, Sandbrink H, Baulcombe DC *et al.*, Homologues of a single resistance-gene cluster in potato confer resistance to distinct pathogens: a virus and a nematode. *Plant J* **23**:567–576 (2000).
24 Robbins TP, Carpenter R and Coen ES, A chromosome rearrangement suggests that donor and recipient sites are associated during Tam3 transposition in *Antirrhinum majus*. *EMBO J* **8**:5–13 (1989).
25 Kim JM, Vanguri S, Boeke JD, Gabriel A and Voyta DF, Transposable elements and genome organization: a comparative survey of retrotransposons revealed by the complete *Saccharomyces cerevisiae* genome sequence. *Genome Res* **8**:464–478 (1998).
26 Meyers BC, Chin DB, Shen KA, Sivaramakrishnan S, Lavelle DO, Zhang Z *et al.*, The major resistance gene cluster in lettuce is highly duplicated and spans several megabases. *Plant Cell* **10**:1817–1832 (1998).
27 Song WY, Bureau TE and Ronald PC, Identification and characterization of 14 transposon-like elements in the noncoding regions of members of the *Xa21* family of disease resistance genes in rice. *Mol Gen Genet* **258**:449–456 (1998).
28 Grube RC, Radwanski ER and Jahn MK, Comparative genetics of disease resistance within the *Solanaceae*. *Genetics* **155**:873–887 (2000).
29 Qin C, Yu C, Shen Y, Fang X, Chen L, Min J *et al.*, Whole-genome sequencing of cultivated and wild peppers provides insights into *Capsicum* domestication and specialization. *Proc Natl Acad Sci USA* **111**:5135–5140 (2014).
30 Kim S, Park M, Yeom SI, Kim YM, Lee JM, Lee HA *et al.*, Genome sequence of the hot pepper provides insights into the evolution of pungency in *Capsicum* species. *Nat Genet* **46**:270–278 (2014).
31 de Jong W, Forsyth A, Leister D, Gebhardt C and Baulcombe DC, A potato hypersensitive resistance gene against potato virus *X* maps to a resistance gene cluster on chromosome 5. *Theor Appl Genet* **95**:246–252 (1997).
32 Hulbert SH, Craig AW, Shavannor MS and Qing S, Resistance gene complexes: evolution and utilization. *Annu Rev Phytopathol* **39**:285–312 (2001).
33 Pflieger S, Lefebvre V, Caranta C, Blattes A, Goffinet B and Palloix A, Disease resistance gene analogs as candidates for QTLs involved in pepper–pathogen interactions. *Genome* **42**:1100–1110 (1999).
34 Mazourek M, Cirulli ET, Collier SM, Landry LG, Kang BC, Quirin EA *et al.*, The fractionated orthology of *Bs2* and *Rx/Gpa2* supports shared synteny of disease resistance in the Solanaceae. *Genetics* **182**:1351–1364 (2009).
35 Zamir D, Ekstein-Michelson I, Zakay Y, Navot N, Zeidan M, Sarfatti M *et al.*, Mapping and introgression of a tomato yellow leaf curl virus tolerance gene, *Ty-1*. *Theor Appl Genet* **88**:141–146 (1994).
36 Thoquet P, Olivier J, Sperisen C, Rogowsky P, Prior P, Anais G *et al.*, Polygenic resistance of tomato plants to bacterial wilt in the French West Indies. *Mol Plant–Microbe Interact* **9**:837–842 (1996).
37 Gebhardt C and Valkonen JF, Organization of genes controlling disease resistance in the potato genome. *Annu Rev Phytopathol* **39**:79–102 (2001).
38 Van der Vossen EAG, Gros J, Sikkema A, Muskens M, Wouters D, Wolters P *et al.*, The *Rpi-blb* gene from *Solanum bulbocastanum* is an *Mi-1* gene homolog conferring broad-spectrum late blight resistance in potato. *Plant J* **44**:208–222 (2005).
39 Bai YL, van der Hulst R, Bonnema G, Marcel BC, Meijer-Dekens F, Niks RE *et al.*, Tomato defense to *Oidium neolycopersici* dominant *Ol* genes confers isolate-dependent resistance via a different mechanism than recessive *ol-2*. *Mol Plant–Microbe Interact* **18**:354–362 (2005).
40 Michelmore RW and Meyers BC, Clusters of resistance genes in plants evolve by divergent selection and a birth-and-death process. *Genome Res* **8**:1113–1130 (1998).
41 Jacquet M, Bongiovanni M, Martinez M, Verschave P, Wajnberg E and Castagnone-Sereno P, Variation in resistance to the root-knot nematode *Meloidogyne incognita* in tomato genotypes bearing the *Mi* gene. *Plant Pathol* **54**:93–99 (2005).
42 Maleita CM, Vieira dos Santos MC, Curtis RHC, Powers SJ and Abrantes IMO, Effect of the *Mi* gene on reproduction of *Meloidogyne hispanica* on tomato genotypes. *Nematology* **13**:939–949 (2011).
43 Cortada L, Sorribas JF, Ornat C, Andres MF and Verdejo-Lucas S, Response of tomato rootstocks carrying the *Mi*-resistance gene to populations of *Meloidogyne arenaria*, *M. incognita and M. javanica*. *Eur J Plant Pathol* **124**:337–343 (2009).
44 Thies JA and Fery RL, Heat stability of resistance to southern root-knot nematode in bell pepper genotypes homozygous and heterozygous for the *N* gene. *J Am Soc Hort Sci* **127**:371–375 (2002).
45 Barbary A, Palloix A, Fazari A, Marteu N, Castagnone-Sereno P and Djian-Caporalino C, The plant genetic background affects the efficiency of the pepper major nematode resistance genes *Me1* and *Me3*. *Theor Appl Genet* **127**:499–507 (2014).
46 Palloix A, Ayme V and Moury B, Durability of plant major resistance genes to pathogens depends on the genetic background, experimental evidence and consequences for breeding strategies. *New Phytol* **183**:190–199 (2009).
47 Brun H, Chevre AM, Fitt BD, Powers S, Besnard AL, Ermel M *et al.*, Quantitative resistance increases the durability of qualitative

resistance to *Leptosphaeria maculans* in *Brassica napus*. *New Phytol* **185**:285–299 (2010).
48 Tan MYA, Hutten RCB, Visser RGF and van Eck HJ, The effect of pyramiding *Phytophthora infestans* resistance genes $R_{Pi\text{-}mcd1}$ and $R_{Pi\text{-}ber}$ in potato. *Theor Appl Genet* **121**:117–125 (2010).
49 Fournet S, Kerlan MC, Renault L, Dantec JP, Rouaux C and Montarry J, Selection of nematodes by resistant plants has implications for local adaptation and cross-virulence. *Plant Pathol* **62**:184–193 (2012).
50 Paillard S, Trotoux-Verplancke G, Perretant M-R, Mohamadi F, Leconte M, Coedel S *et al.*, Durable resistance to stripe rust is due to three specific resistance genes in French bread wheat cultivar Apache. *Theor Appl Genet* **125**:955–965 (2012).
51 Quenouille J, Paulhiac E, Moury B and Palloix A, Quantitative trait loci from the host genetic background modulates the durability of a resistance gene: a rational basis for sustainable resistance breeding in plants. *Heredity* **112**:579–587 (2014).
52 Cervantes-Flores JC, Yencho GC, Pecota KV, Sosinski B and Mwanga ROM, Detection of quantitative trait loci and inheritance of root-knot nematode resistance in sweet potato. *J Am Soc Hort Sci* **133**:844–851 (2008).
53 Ulloa M, Wang C and Roberts PA, Gene action analysis by inheritance and quantitative trait loci mapping of resistance to root-knot nematodes in cotton. *Plant Breeding* **129**:541–550 (2010).
54 Xu X, Zeng L, Tao Y, Vuong T, Wan J, Boerma R *et al.*, Pinpointing genes underlying the quantitative trait loci for root-knot nematode resistance in palaeopolyploid soybean by whole genome resequencing. *Proc Natl Acad Sci USA* **110**:13 469–13 474 (2013).
55 Burow MD, Starr JL, Park CH, Simpson CE and Paterson AH, Introgression of homeologous quantitative trait loci (QTLs) for resistance to the root-knot nematode [*Meloidogyne arenaria* (Neal) Chitwood] in an advanced backcross-QTL population of peanut (*Arachis hypogaea* L). *Mol Breeding* **34**:393–406 (2014).
56 Sanchez-Solana F, Ros C, Guerrero MM, Lacasa CM, Sanchez-Lopez E and Lacasa A, New pepper accessions proved to be suitable as a genetic resource for use in breeding nematode-resistant rootstocks. *Plant Genet Resour* DOI: 10.1017/S1479262115000027 (2015).
57 Quenouille J, Montarry J, Palloix A and Moury B, Farther, slower, stronger: how the plant genetic background protects a major resistance gene from breakdown. *Mol Plant Pathol* **14**:109–118 (2013).
58 Veremis JC and Roberts PA, Differentiation of *Meloidogyne incognita* and *M. arenaria* novel resistance phenotypes in *Lycopersicon peruvianum* and derived bridge-lines. *Theor Appl Genet* **1993**:960–967 (1996).
59 Williamson VM and Roberts PA, Mechanisms and genetics of resistance, in *Root-knot Nematodes*, ed. by Perry RN, Moens M and Starr J. CABI Publishing, Wallingford, Oxon, UK, pp. 301–325 (2009).
60 Thies JA and Fery RL, Modified expression of the *N* gene for southern root-knot nematode resistance in pepper at high soil temperatures. *J Am Soc Hort Sci* **123**:1012–1015 (1998).
61 Thies JA and Fery RL, Heat stability of resistance to *Meloidogyne incognita* in Scotch Bonnet peppers (*Capsicum chinense* Jacq.). *J Nematol* **32**:356–361 (2000).
62 Thies JA and Fery RL, Characterization of *Capsicum chinense* cultigens for resistance to *Meloidogyne arenaria, M. hapla*, and *M. javanica*. *Plant Dis* **85**:267–270 (2001).
63 Thies JA, Dickson DW and Fery RL, Stability of resistance to root-knot nematodes in 'Charleston Belle' and 'Carolina Wonder' bell peppers in a sub-tropical environment. *HortScience* **43**:188–190 (2008).
64 Rommens CM and Kishore GM, Exploiting the full potential of disease-resistance genes for agricultural use. *Curr Opin Biotechnol* **11**:120–125 (2000).
65 Zhang LH, Mojtahedi H, Kuang H, Baker B and Brown CR, Marker-assisted selection of Columbia root-knot nematode resistance introgressed from *Solanum bulbocastanum*. *Crop Sci* **47**:2021–2026 (2007).
66 Potato Genome Sequencing Consortium, Genome sequence and analysis of the tuber crop potato. *Nature* **475**:189–195 (2011).
67 Tomato Genome Consortium, The tomato genome sequence provides insights into fleshy fruit evolution. *Nature* **485**:635–641 (2012).
68 Jacobsen E and Schouten HJ, Cisgenesis strongly improves introgression breeding and induced translocation breeding of plants. *Trends Biotechnol* **25**:219–223 (2007).
69 Goggin FL, Shah G, Williamson VM and Ullman DE, Instability of *Mi*-mediated nematode resistance in transgenic tomato plants. *Mol Breeding* **13**:357–364 (2004).
70 Goggin FL, Jia L, Shah G, Hebert S, Williamson VM and Ullman DE, Heterologous expression of the *Mi-12* gene from tomato confers resistance against nematodes but not aphids in eggplant. *Mol Plant–Microbe Interact* **19**:383–388 (2006).
71 Williamson VM, Root-knot nematode resistance genes in tomato and their potential for future use. *Annu Rev Phytopathol* **36**:277–293 (1998).
72 Zhang LY, Zhang YY, Chen RG, Zhang JH, Wang TT, Li HX *et al.*, Ectopic expression of the tomato *Mi-1* gene confers resistance to root knot nematodes in lettuce (*Lactuca sativa*). *Plant Mol Biol Repter* **28**:204–211 (2010).
73 Kouassi AB, Kerlan MC, Caromel B, Dantec JP, Fouville D, Manzanares-Dauleux M *et al.*, A major gene mapped on chromosome XII is the main factor of a quantitatively inherited resistance to *Meloidogyne fallax* in *Solanum sparsipilum*. *Theor Appl Genet* **112**:699–707 (2006).
74 Brown CR, Mojtahedi H and Santo GS, Introgression of resistance to Columbia and Northern root-knot nematodes from *Solanum bulbocastanum* into cultivated potato. *Euphytica* **83**:71–78 (1995).
75 Smith PG, Embryo culture of a tomato species hybrid. *Proc Am Soc Hort Sci* **44**:413–416 (1944).
76 Thies JA, Mueller JD and Fery RL, Effectiveness of resistance to southern root-knot nematode in 'Carolina Cayenne' pepper (*Capsicum annuum* L.) in greenhouse, microplot, and field tests. *J Am Soc Hort Sci* **122**:200–204 (1997).
77 Djian-Caporalino C, Pijarowski L, Januel A, Lefebvre V, Daubèze A, Palloix A *et al.*, Spectrum of resistance to root-knot nematodes and inheritance of heat-stable resistance in pepper (*Capsicum annuum* L). *Theor Appl Genet* **99**:496–502 (1999).
78 Hendy H, Pochard E and Dalmasso A, Transmission héréditaire de la résistance aux *Meloidogyne* portée par deux lignées de *Capsicum annuum*: études de descendances d'homozygotes issues d'androgénèse. *Agronomie* **5**:93–100 (1985).
79 Castagnone-Sereno P, *Meloidogyne enterolobii* (= *M. mayaguensis*): profile of an emerging, highly pathogenic root-knot nematode species. *Nematology* **14**:133–138 (2012).
80 Gonçalves LSA, Gomes VM, Robaina RR, Valim RH, Rodrigues R and Aranha FM, Resistance to root-knot nematode (*Meloidogyne enterolobii*) in *Capsicum* spp. accessions. *Rev Bras Ciencias Agrar* **9**:49–52 (2014).
81 Holzmann OV, Effects of soil temperature on resistance of tomato to root-knot nematode (*Meloidogyne incognita*). *Phytopathology* **55**:990–992 (1965).
82 Verdejo-Lucas S, Talavera M and Andres MF, Virulence response to the *Mi1* gene of *Meloidogyne* populations from tomato in greenhouses. *Crop Prot* **39**:97–105 (2012).
83 Iberkleid I, Ozalvo R, Feldman L, Elbaz M, Patricia B and Horowitz SB, Responses of tomato genotypes to avirulent and *Mi*-virulent *Meloidogyne javanica* isolates occurring in Israel. *Phytopathology* **104**:484–496 (2014).
84 Tzortzakakis EA, Conceicao I, Dias AM, Simoglou KB and Abrantes I, Occurrence of a new resistant breaking pathotype of *Meloidogyne incognita* on tomato in Greece. *J Plant Dis Prot* **121**:184–186 (2014).
85 Thies JA, Virulence of *Meloidogyne incognita* to expression of *N* gene in pepper. *J Nematol* **43**:90–94 (2011).
86 Djian-Caporalino C, Molinari S, Palloix A, Ciancio A, Fazari A, Marteu N *et al.*, The reproductive potential of the root-knot nematode *Meloidogyne incognita* is affected by selection for virulence against major resistance genes from tomato and pepper. *Eur J Plant Pathol* **131**:431–440 (2011).
87 Ros-Ibanez C, Robertson L, del Carmen Martinez-Lluch M, Cano-Garcia A and Lacasa-Plasencia A, Development of virulence to *Meloidogyne incognita* on resistant pepper rootstocks. *Spanish J Agr Res* **12**:225–232 (2014).
88 Bird DM, Williamson VM, Abad P, McCarter J, Danchin EGJ, Castagnone-Sereno P *et al.*, The genomes of root-knot nematodes. *Annu Rev Phytopathol* **47**:333–351 (2009).
89 McDonald BA and Linde C, Pathogen population genetics and the durability of resistance. *Euphytica* **124**:163–180 (2002).
90 Castagnone-Sereno P, Danchin EGJ, Perfus-Barbeoch L and Abad P, Diversity and evolution of root-knot nematodes, genus *Meloidogyne*: new insights from the genomic era. *Annu Rev Phytopathol* **51**:203–220 (2013).

91 Perfus-Barbeoch L, Castagnone-Sereno P, Reichelt M, Fneich S, Roquis D, Pratx L *et al.*, Elucidating the molecular bases of epigenetic inheritance in non-model invertebrates: the case of the root-knot nematode *Meloidogyne incognita*. *Front Physiol* **5**:211 (2014).

92 Sage-Palloix AM, Jourdan F, Phaly T, Nemouchi G, Lefebvre V and Palloix A, Structuring genetic diversity in pepper genetic resources: distribution of horticultural and resistance traits in the INRA pepper germplasm, in *Progress in Research on Capsicum and Eggplant*, ed. by Niemirowicz-Szczytt K. University of Life Sciences Press, Warsaw, Poland, pp. 33–42 (2007).

93 Djian-Caporalino C, Palloix A, Fazari A, Marteu N, Barbary A, Abad P *et al.*, Pyramiding, alternating or mixing: comparative performances of deployment strategies of nematode resistance genes to promote plant resistance efficiency and durability. *BMC Plant Biol* **14**:53 (2014).

94 Bleve-Zacheo T, Bongiovanni M, Melillo MT and Castagnone-Sereno P, The pepper resistance genes *Me1* and *Me3* induce differential penetration rates and temporal sequences of root cell ultrastructural changes upon nematode infection. *Plant Sci* **133**:79–90 (1998).

95 Pegard A, Brizzard G, Fazari A, Soucaze O, Abad P and Djian-Caporalino C, Histological characterization of resistance to different root-knot nematode species related to phenolics accumulation in *Capsicum annuum*. *Phytopathology* **95**:158–165 (2005).

96 Bourget R, Chaumont L, Durel CE and Sapoukhina N, Sustainable deployment of QTLs conferring quantitative resistance to crops: first lessons from a stochastic model. *New Phytol* **206**:1163–1171 (2015).

97 Schwarz D, Rouphael Y, Colla G and Venema JH, Grafting as a tool to improve tolerance of vegetables to abiotic stresses: thermal stress, water stress and organic pollutants. *Sci Hort* **127**:162–171 (2010).

98 Dallemole-Giaretta R, de Freitas LG, Lopes EA, Ferraz S, de Podestá GS and Agnes EL, Cover crops and *Pochonia chlamydosporia* for the control of *Meloidogyne javanica*. *Nematology* **13**:919–926 (2011).

99 Klein E, Katan J and Gamliel A, Soil suppressiveness to *Meloidogyne javanica* as induced by organic amendments and solarization in greenhouse crops. *Crop Prot* **39**:26–32 (2012).

100 Stirling GR, Integration of organic amendments, crop rotation, residue retention and minimum tillage into a subtropical vegetable farming system enhances suppressiveness to root-knot nematode (*Meloidogyne incognita*). *Aust Plant Pathol* **42**:625–637 (2013).

101 Ortiz AM, Sipes BS, Miyasaka SC and Arakaki AS, Green manure crops for management of *Meloidogyne javanica* and *Pythium aphanidermatum*. *Hortscience* **50**:90–98 (2015).

102 Collange B, Navarrete M, Montfort F, Mateille T, Tavoillot J, Martiny B *et al.*, Alternative cropping systems can have contrasting effects on various soil-borne diseases: relevance of a systemic analysis in vegetable cropping systems. *Crop Prot* **55**:7–15 (2014).

103 Fazari A, Palloix A, Wang LH, Hua MY, Sage-Palloix AM, Zhang BX *et al.*, The root-knot nematode resistance *N*-gene co-localizes in the *Me*-genes cluster on the pepper (*Capsicum annuum* L.) P9 chromosome. *Plant Breeding* **131**:665–673 (2012).