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Loss of susceptibility as an alternative for nematode resistance

Janice de Almeida Engler, Bruno Favery, Gilbert Engler and Pierre Abad

Among plant pathogens, sedentary endoparasitic nematodes are one of the most damaging pests in global agriculture. These obligate parasites interact with their hosts in a quite unique and intriguing way. They induce the redifferentiation of root cells into specialized feeding cells essential for nematode growth and reproduction; thus, nematodes have evolved the ability to exploit plant genes and hijack host functions for their own requirements. Various approaches to engineer plants with resistance to parasitic nematodes have been pursued, most focusing on the introduction of resistance genes. An alternative strategy to achieve resistance is to exploit the susceptibility of plant disease. Better knowledge of the plant response during the compatible interaction should allow the identification of targets to engineer resistance to parasitic nematodes in crop species.

Addresses
INRA, UMR Interactions Plantes-Microorganismes et Santé Végétale, 400 Route des Chappes, 06903 Sophia-Antipolis, France

Corresponding author: de Almeida Engler, Janice (almeida@antibes.inra.fr)

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Introduction

Plants protect themselves from pathogen attack through the activation of a broad array of responses that lead to the production of defensive compounds [1]. The diverse resistance mechanisms are well described and often involve the induction of a hypersensitive reaction (HR) that results in host cell death, thus blocking colonization and reproduction of the pathogen. Many plant resistance (R) genes conferring resistance to pathogens, such as viruses, bacteria, fungi and nematodes, have been cloned, and research within the past 10 years has increased our knowledge of the genetics and the molecular mechanisms underlying plant disease resistance. One such example is the well-documented *Mi* gene of tomato, which confers resistance against root-knot nematodes [2,3].

In contrast to our knowledge of the plant genes involved in disease resistance, little is known about the essential plant components in compatible plant–pathogen interactions. The host plant was for a long time considered to be inactive during disease development, and studies focused mainly on pathogenicity factors and suppression of host defences. However, accumulating evidence from genetic and molecular studies indicates that plant genes are required for susceptibility to pathogens. Although it is unlikely that plants possess genes that function only for the benefit of pathogens, it is likely that pathogens have evolved the ability to make use of plant genes to manoeuvre host functions in their favour. The best-characterized example of a host susceptibility factor is the plant metabolite acetosyringone for *Agrobacteria*. This plant metabolite induces bacterial genes involved in pathogenesis in the same way that flavonoids exuded by plant roots will activate the expression of nodulation genes from *Rhizobia* during symbiosis [4,5].

This review focuses on two of the most economically damaging groups of plant parasitic nematodes, the cyst-forming and the root-knot nematodes, and highlights the exploitation of the so-called susceptibility genes as an alternative approach for plant resistance.

Genes required for pathogen susceptibility

Recently, two novel forms of disease resistance have been identified in plants. The first is based on the loss of function of genes that are active as negative regulators of plant defence or cell death. An example is the deletion of the Barley *Mlo* gene; mutation in this gene confers resistance to the pathogen *Blumeria graminis* f.sp. *hordei* by preventing fungal penetration [6]. A second type of resistance is based on the loss of a host susceptibility gene required by the pathogen for growth and development, rather than the activation of known host defence pathways [7,8•]. This form of resistance was identified through the characterisation of several *Arabidopsis* mutants that do not support the establishment of powdery mildew [8•,9,10]. Mutants that did not activate known defence mechanisms were shown to be affected in plant disease susceptibility factors required by these biotrophic pathogens. The recessive loss-of-function *pmr6* mutant of *Arabidopsis* was shown to be completely resistant to fungal infection and did not develop disease symptoms [7]. *PMR6* encodes a pectate-lyase-like protein and does not resemble previously described genes involved in host defence responses. Both artificial mutagenesis of the host plant [11,12] and the characterization of recessive natural resistance [13,14] have revealed an essential role for the eukaryotic translation factors eIF(iso)4E and eIF4E for potyvirus replication and infection. Notably,

although the eIF(iso)4E protein is relevant for susceptibility, its absence does not alter normal plant development [8•].

Plant–nematode susceptible interactions

Plant parasitic nematodes are obligate biotrophs that can only feed on living cells. The two most harmful groups affecting crop species worldwide are the sedentary endoparasites root-knot nematodes (*Meloidogyne* spp.) and cyst nematodes (*Globodera* spp. and *Heterodera* spp.) and most studies have focused on these prime model species. Both types of nematode establish an intimate relationship with their host plants, inducing the redifferentiation of root cells into specialized feeding cells. The establishment of a feeding site within the root is essential to fulfil the nematode's nutritional demands for growth and reproduction. It is not yet understood how feeding sites are induced, but it is believed that pathogenicity factors secreted by the nematode play key roles during parasitism and might have direct effects on recipient host cells [15•,16].

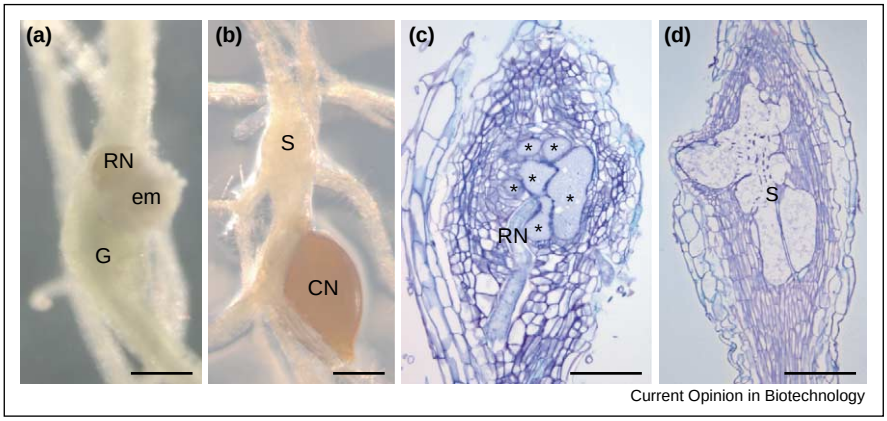
Upon infection, motile nematode larva present in the soil penetrate the roots, preferably at the elongation zone just behind the root tip. They migrate intercellularly (root-knot nematodes) or intracellularly (cyst nematodes) towards the vascular cylinder to select a competent root cell for the induction of enlarged and multinucleated feeding cells. These typical nematode feeding cells contain large volumes of cytoplasm and are converted into nutrient sinks that serve as the sole food source for the subsequent sedentary parasitic stages. During nematode development, juvenile nematodes undergo three moults before becoming adult. After the last moult, root-knot females will nearly always reproduce via parthenogenesis. Females will produce eggs (Figure 1a) that will hatch and complete the nematode life cycle between one and two months. For cyst nematodes, following the last moult, males fertilize females that subsequently die and become cysts filled with eggs (Figure 1b). Cysts resist drastic soil environments and eggs containing juvenile nematodes will hatch only under favourable conditions restarting a new life cycle.

Root-knot nematodes induce feeding sites containing several giant cells surrounded by dividing vascular cells. The asymmetric divisions of cells surrounding the giant cells result in the formation of typical swellings within the infected roots named 'galls' (Figures 1a,c). In giant cells the multinucleated status is reached through multiple nuclear divisions without cytokinesis alternated with additional events of DNA replication (endoreduplication) [17–19]. Conversely, in a syncytium induced by a cyst nematode the initial feeding cell expands within the vascular tissue by progressive cell-wall dissolution reaching a multinucleated state upon incorporation of dividing neighbouring cells [20] (Figures 1b,d). Although mitotic cycles have never been observed in syncytia, nuclear DNA synthesis has been shown to take place [21].

Nevertheless, giant cells and syncytia share common traits. Within the dense cytoplasm of developing feeding cells, subcellular organelles proliferate, nuclei and nucleoli undergo hypertrophy, and small secondary vacuoles are formed. A characteristic feature of feeding cells is the development of cell wall ingrowths, typical of transfer cells, which most likely enhance solute uptake from the vascular system.

The complex morphological and physiological changes during feeding cell establishment are reflected by altered gene expression in the infected root cells. The genes identified are involved in diverse processes such as wound and defence response, the cell cycle, cytoskeleton reorganisation, cell-wall modification, physiology and

Figure 1



Giant cells and syncytia induced by plant parasitic nematodes. *Arabidopsis* roots infected with **(a)** the root-knot nematode *Meloidogyne incognita* and **(b)** the cyst nematode *Heterodera schachtii* 5 days after inoculation and **(c,d)** the respective longitudinal sections. Scale bars = 100 μm. CN, cyst nematode; G, gall; em, egg mass; *, giant cell; RN, root-knot nematode; S, syncytium.

water status, general metabolism, stress, transcription and hormone response [22••].

Genes and metabolic pathways important for feeding cell formation

Comparisons of host transcription patterns using a variety of techniques have identified many genes involved in feeding cell ontogeny. Only the more recently identified ones are mentioned in this review.

Hormone perception by the host seems to be an important feature for a successful interaction between plants and nematodes. A synthetic auxin-responsive promoter element DR5, derived from the soybean promoter GH3, confirmed the early perception of a localized increase of auxin concentration in feeding sites of both root-knot and cyst nematodes [23]. In addition, the induction in *Medicago truncatula* giant cells of the *PHAN* and *KNOX* genes, required for normal meristem function, and their involvement in changes in phytohormone levels implicates these genes in the regulation of auxin distribution during feeding cell development [24].

Not only auxin seems to play a role in feeding cell induction and maintenance. The observed upregulation of a cytokinin-responsive *ARR5* (*Arabidopsis* Response Regulator) promoter fused to the β-glucuronidase gene (*GUS*) in feeding cells suggests that a spike of cytokinin is also required during giant cell initiation [25,26]. In addition, downregulation of the RHD1 gene, encoding a UDP-glucose-4-epimerase, in *Heterodera schachtii*-infected roots seems to occur via an ethylene signal elicited by nematode infection [27].

The analysis of regulatory sequences and interacting transcription factors should provide information on the signal transduction pathways essential for feeding cell development. The possible role of heat-shock elements during giant cell development had been reported by Escobar *et al.* [28]. These authors showed that a short fragment of the promoter of the *Hahsp17*.7G4 gene, encoding a small heat-shock protein involved in embryogenesis and stress response, is specifically expressed in tobacco galls. In addition, point mutations in heat-shock transcription factor binding sites caused a significant decrease in nematode response. Mapping the nematode-responsive elements might bring us a step closer towards the transcriptional regulation and signalling cascade involved in feeding cell initiation [22••].

It appears that extensive cell-wall changes are necessary for giant cell and syncytium development. Plant hydrolases might play a fundamental role in feeding cell-wall architectural changes. Goellner *et al.* [29] have validated the idea that cell-wall-modifying enzymes of plant origin are implicated in feeding cell formation. Some of these cell-wall enzymes, such as endo-β-1,4-glucanases and pectin acetylesterase, are expressed in response to both root-knot and cyst nematode infection [29,30], although differential gene expression has also been observed. The *Arabidopsis* endo-1,4-β-glucanase *CEL1*, for example, showed activity only in giant cells and not in syncytia [31]. Therefore, the specific regulation of cell-wall-degrading enzymes is probably required for cell-wall modifications to build up feeding cells.

An increased metabolic activity has been observed in giant cells and syncytia throughout nematode parasitism. The idea that feeding sites are large nutrient sinks is supported by data on the expression of a sucrose transporter gene (*AtSUC2*) observed in syncytia [32]. The lack of expression of this gene in giant cells, which are also carbon sinks, suggests the involvement of other genes. In a recent study, transcripts isolated from giant cell cytoplasm in the later stages of infection were shown to have significant homology to mitogen-activated protein kinases, *S*-adenosylmethionine decarboxylases, cysteine synthases, cytochrome *c* reductase subunits, and ribosomal proteins. These results confirm the high metabolic turnover in mature giant cells [33].

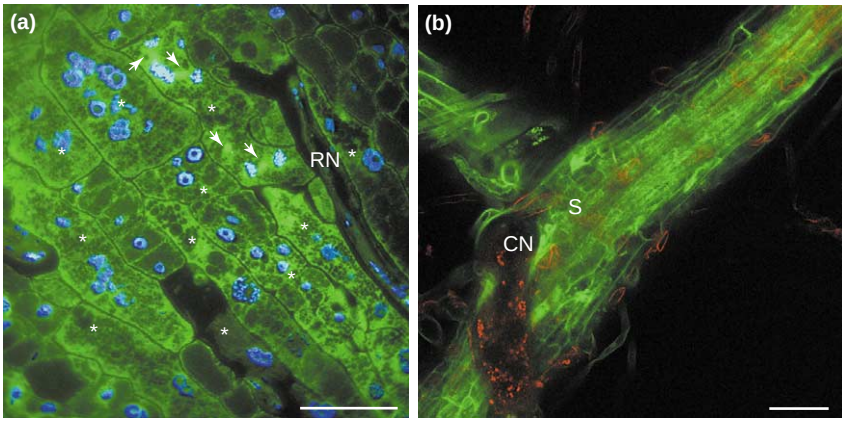
Extensive gene analysis must be coupled with a more detailed cellular expression pattern analysis, the characterization of mutants and biochemical investigations, to more accurately dissect gene function during feeding cell development.

The first demonstration of the effect of inactivating a gene function essential for giant cell formation has been provided by knockout of the *rpe* gene [34]. Analysis of *rpe* mutants showed that *RPE* is essential during the early steps of giant cell formation, but not during syncytia development. This first plant gene required for nematode susceptibility has been identified in a screen for genes upregulated in giant cells. It encodes D-ribulose 5-phosphate 3-epimerase, a key enzyme in the pentose phosphate pathway. This pathway has a crucial role in actively growing cells by generating the NADPH required in numerous biosynthetic reactions and by generating carbohydrate intermediates for the synthesis of nucleotides and cell-wall polymers. These results strongly support the hypothesis that biochemical functions operating in normal, non-infected plants have been recruited to play key roles in nematode development [34,35].

The cell cycle and the cytoskeleton are involved in feeding cell formation

The complex morphological changes during feeding cell formation might be the result of strategies used by the nematode to manipulate processes such as cell-cycle progression and cytoskeleton organization in the plant host. Chemical inhibitors can be used to interfere with cellular mechanisms, such as the cell cycle and the overall organization of the cytoskeleton. Chemical blocking of

Figure 2



Microtubular cytoskeleton reorganization in nematode feeding sites. **(a)** Immunolocalization of tubulins (green) and DNA staining (blue) in giant cells reveal phragmoplasts (arrows), homogeneous cytoplasmic microtubular staining and multiple mitotic nuclei. **(b)** Green fluorescent protein-decorated microtubules in an infected root showing perturbed and partially depolymerised microtubules in a syncytium mainly close to the nematode head. Background fluorescence is red. Scale bars = 100 μm. CN, cyst nematode; *, giant cell; RN, root-knot nematode; S, syncytium.

the cell cycle and cytoskeleton dynamics results in the arrest of proper root-knot and cyst nematode feeding site development [21,36••]. These results point to the relevance of these processes and their genes for feeding cell initiation and maintenance.

Early transcriptional activation of cell-cycle markers such as cyclin-dependent kinases (*CDC2a* and *CDC2b*) and mitotic cyclins (*CYC1At* and *CYCA2;1*) has been observed in feeding cells [21]. In addition, genes involved in endoreduplication, such as *CCS52*, have been shown to be induced in giant cells [37] and syncytia. Indeed, preventing DNA synthesis or blocking the cell cycle after nematode infection significantly inhibited feeding cell progression.

The involvement of the cytoskeleton has been reported in various plant–pathogen interactions [37]. Nematodes induce long-term rearrangements of the cytoskeleton during the infection process [38] (Figure 2). Although tubulin and actin genes appear to be upregulated in nematode feeding sites, only cortical microtubules and not cytoplasmic microtubules are seen within giant cells and syncytia. The appearance of a complex network of actin filaments and bundles within the cytoplasm of giant cells suggests the involvement of the actin cytoskeleton in feeding cell development. The recent observation of the activation of a membrane-anchored formin gene (*AtFH6*) in giant cells [39•], involved in actin nucleation, supports the idea that the reorganization of the actin cytoskeleton takes place during feeding cell development.

Recently, the involvement of the actin cytoskeleton in plant–fungal interactions has been confirmed by actin array analyses in mutants and knockdowns of two plant susceptibility genes, *Mlo* and *Rac*, respectively [40]. MLO and RACB proteins are most likely to be involved in the modulation and reorganisation of actin filaments and in cell polarity during the interaction of barley and *B. graminis*.

Within multinucleated giant cells multiple phragmoplasts develop during late anaphase (Figure 2a). A phragmoplast is a double ring-like structure that gradually expands outwards, along with the formation of a new cell plate. This centrifugal growth is crucial for the cell plate development. In giant cells some phragmoplasts are composed of two bundles of antiparallel microtubules and actin filaments, as observed in typical plant cells, while others seem malformed. An arrest in phragmoplast expansion seems to occur in giant cells, often leading to the formation of cell-wall stubs as a result of aborted cell division. The initial shape of a phragmoplast is a cylinder, which later changes into a ring-like structure that centrifugally expands. This change in shape is crucial for lateral growth of the cell plate, and is affected or absent in giant cells. Thus, the screening of genes implicated in cytokinesis [41,42] will be a useful approach to identify the ones involved in cell division arrest in giant cells. As such, interfering with feeding site formation or development via the cytoskeleton sounds a plausible approach to identify new targets during this susceptible interaction and might encourage the development of novel approaches to engineer nematode-resistant plants.

Conclusions

Developing strategies to combat nematode parasitism becomes an important issue when considering constraints

on the use of chemical pesticides. Among them, nematicides are the most toxic for the environment. Although studies have been carried out to introduce natural resistance genes into crop species, this approach is limited to species containing the proper resistance loci and by the emergence of new virulent populations of nematodes [43]. In a compatible interaction, host 'susceptibility' factors are required for the establishment and maintenance of the infection process. Consequently, the loss of function of such host genes is predicted to result in resistance against the pathogen. Given that the involvement of genes implicated in cytoskeleton rearrangements during nematode infection indicates their participation in the establishment of a functional feeding cell, it seems promising to exploit these genes to impede nematode propagation. In addition, the application of high-throughput methods such as microarray technologies will provide large-scale information about patterns of gene expression during plant–nematode interactions [44•] (A Haeger *et al.*, Abstract 140; Jammes *et al.*, Abstract 4, XXVII ESN International Symposium, Rome, 14–18 June 2004). A more comprehensive view of the molecular mechanisms underlying the formation of feeding cells will permit the identification of targets for the development of new approaches to engineer plant resistance. For example, targets like translation factor alleles responsible for potyvirus replication and infection [11] are promising candidates. This class of genes belongs to multigene families and harbour resistant alleles without any pleiotropic effect. Therefore, it will be essential to search in natural populations and cultivars for the appropriate allelic forms of those genes that are central for a compatible plant–nematode interaction. Alternatively, suitable alleles can be induced by mutagenesis to create durable resistance against parasitic nematodes.

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