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MECHANISMS CONFERRING PLANT INCOMPATIBILITY TO NEMATODES [1]

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SUMMARY

Incompatibility (resistance) of plants to nematode parasites is conferred by repulsive chemicals in a few cases but rarely, if at all, by penetration barriers or nutritional deficiencies in the host plant. The major incompatibility mechanism(s) appear(s) to be the post-infectional occurrence of « hypersensitive » reactions. Although the role of preformed simple phenols in this type of incompatibility is obscure, available information suggests that inducibly formed structural and chemical (*viz.* phytoalexins) barriers to nematode development may be responsible. No firm conclusions may be made at present on the validity of possible mechanisms since much of the early work is difficult to interpret and contains procedural inadequacies. Thus, appraisal of incompatibility mechanisms to nematodes requires additional testing of the hypotheses, especially as to the existence of such mechanisms at the localized site where and when incompatibility is expressed. An understanding of incompatibility may be attained more easily by utilizing those experimental approaches that have recently been used to study incompatibility systems to microbial parasites. Some of these are suggested which may be used in future nematode research.

RÉSUMÉ

Mécanismes conférant aux plantes l'incompatibilité aux nématodes

Une revue de la littérature a démontré que la réaction d'incompatibilité (la résistance) des plantes aux nématodes est due, dans certains cas, à des substances répulsives pour le parasite mais pratiquement jamais à l'existence de barrières s'opposant à sa pénétration ou au manque d'éléments nutritionnels dans la plante hôte. Le ou les mécanisme(s) majeur(s) conduisant à l'incompatibilité semble(nt) donc être l'apparition de réactions d' « hypersensibilité » après l'infection. Bien que dans ce type de réaction d'incompatibilité le rôle des phénols préformés soit encore obscur, l'information dont on dispose permet de penser que la formation, après l'infection, de barrières de nature chimique (par exemple les phytoalexines) ou structurale pourrait être responsable de la réaction d'incompatibilité. Cependant, à l'heure actuelle, on ne peut pas se prononcer en faveur d'un des mécanismes possibles car une expérimentation criticable rend une grande partie des travaux publiés difficile à interpréter. C'est pourquoi une étude plus fine est nécessaire avant qu'on puisse se prononcer sur la nature des mécanismes de la réaction d'incompatibilité aux nématodes. Il faudra notamment chercher à savoir si les mécanismes proposés apparaissent en même temps que la réaction d'incompatibilité, d'une part, et s'ils sont localisés aux sites mêmes où la réaction d'incompatibilité s'exprime, d'autre part. L'utilisation d'approches expérimentales variées, comme cela a été fait dans le cas des systèmes bactériens, viraux ou fongiques, devrait permettre d'atteindre cet objectif. Nous proposons quelques-unes de ces approches expérimentales qui pourraient être utiles dans le cas des systèmes à nématodes.

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Economics, availability, and government regulations portend reduced future reliance on chemical control of nematode-incited plant diseases. Greater emphasis will probably be placed on alternative methods of disease control and on the biology underlying their effectiveness. Because genetic resistance is and probably will remain a prime control measure of diseases caused by plant pathogenic nematodes, a review is presented of research published in the last few years on the mechanisms which limit nematode development and reproduction. Those papers will be emphasized which have appeared after the last reviews on the topic (Cook, 1974; Dropkin, 1976; Giebel, 1974; Levin, 1976; Rohde, 1972; Webster, 1975). Comparisons will also be made to selected research on mechanisms of incompatibility to bacterial and fungal plant pathogens in order to demonstrate similarities and differences and to suggest experimental approaches for future studies of nematode systems.

Because of its historical connotation with yield and not necessarily pathogen development, the term *resistance* will not be routinely used in this review; instead *incompatibility* will be used to denote the unsuitability of a plant for « normal » pathogen development relative to that occurring in a *compatible* interaction, where development and reproduction are considerable. *General incompatibility* is the ability of all members of a plant species to prevent reproduction of an entire nematode or microbe species. Thus, the nematode or microbe is a *nonpathogen* on the plant species in question, although it may be pathogenic on certain other plant species. *Specific incompatibility* is that exhibited by only certain cultivars or genotypes of a single plant species and is generally effective against only specific strains or races of a microorganism or nematode species. In its most refined form, the specificity of both partners is determined by alleles at single genetic loci, *viz. gene-for-gene* systems (Days, 1974; Price, Caviness & Riggs, 1978; Webster, 1975). Most of the nematology literature has been concerned with specific (*viz.* cultivar) incompatibility to certain nematode species.

Regardless of the host-parasite interaction considered, compatibility is dependent on the pathogen's ability to develop and escape barriers posed by the plant. Since failure to perform any of these would result in incompatibility, it is not surprising that examples of natural plant disease defense are known for most of them. Also, natural incompatibility in a single plant may result from the multiplier effect of two consecutive mechanisms, neither of which need be absolutely effective against the pathogen. Such sequences have been suggested for resistance to certain non-pathogenic fungi by Heath (1974), but no convincing evidence for them against nematodes exists.

Attraction and repulsion

Both ecto- and endoparasitic nematodes share a common environment during initial stages of the life cycle, and must successfully migrate through the rhizosphere to a root. Root exudates may be important as attractants or repellants, and substantial evidence exists indicating that roots exude attractant substances (Green, 1971). Certain plants may also be protected from nematode infection by releasing repellant materials into the rhizosphere. Several plants (*e.g.*, African marigold, onion, garlic, asparagus, and pangola grass) have been suspected of reducing soil populations of nematodes in this manner. Early work by Rohde and Jenkins (1958) suggested that release of a glycoside with nematicidal properties into soil by asparagus roots was responsible for resistance to *Paratrichodorus minor*. Concentrations of the glycoside in leachates collected from potted plants indicated that adequate concentrations were present in the rhizosphere to protect plants from infection by the ectoparasite. The compound was nematicidal *in vitro* and significantly reduced infection of tomato by *P. minor* when applied as a soil drench or foliar spray. The asparagus glycoside may therefore be a factor conferring disease incompatibility since the available evidence demonstrates that an effective concentration of the active compound is released into rhizosphere.

Intercropping of marigolds (*Tagetes patula* L.) reduces nematode infection of other plants (Giebel, 1974; Hackney & Dickerson, 1975; Motsinger, Moody & Gay, 1977; Rohde, 1972; Winoto, 1969). Marigolds contain α-terthienyls

which have *in vitro* nematicidal activity (Uhlenbroek & Bijloo, 1958, 1959). This has led to speculation concerning the possible protective role of terthienyls in soil, but their presence in the rhizosphere has not been demonstrated (Hackney & Dickerson, 1975). This and the fact that aqueous suspensions of terthienyls at concentrations up to 200 ppm applied as a soil drench did not control *Meloidogyne javanica* (Daulton & Curtis, 1963) question the validity of a terthienyl protective mechanism. Toxicity of terthienyls may, in fact, be less than believed. Gommers (1972), and Gommers and Geerligs (1974) have demonstrated that *in vitro* nematicidal activity of the terthienyls is greatly enhanced by near ultraviolet light and therefore they may be relatively innocuous in soils. Experimental results indicate that marigolds function as trap crops (Hackney & Dickerson, 1975), and they may reduce crop damage when planted in rotation or as an intercrop; however, the role of the terthienyls in this process is still unclear.

More convincing evidence for a repulsion mechanism of incompatibility comes from genetic research with cucumbers. Haynes and Jones (1976) found that cucumber plants carrying a dominant allele at the *Bi* (bitter) locus attracted significantly fewer *Meloidogyne incognita* larvae to the roots than did the near-isogenic *bibi* (non-bitter) genotype. The *Bi* locus permits plants to accumulate curcurbitacins, toxic triterpenoids that are also important in resistance to other plant pests (Dacosta & Jones, 1971). The *bibi* genotype does not confer production of the compounds. When plants of the two cucumber genotypes were exposed to high numbers of *M. incognita* in greenhouse tests, plants having the *Bi* genotype attracted significantly less larvae than those with *bibi*; however, the root-knot indices and detrimental effects on plant growth were indistinguishable. Thus, although the *Bi* gene appears to reduce the number of nematodes attracted to cucumber roots, it does not provide an effective barrier to those nematodes that do reach and penetrate them.

Penetration

Preformed morphological (Royle, 1976) and chemical (Schönbeck & Schlosser, 1976) barriers have been reported which prevent penetration of plant tissues by bacterial and fungal non-pathogens. Such barriers do not appear to be effective against plant-parasitic nematodes. The action of stylet penetration combined with enzyme release by nematodes (Chitwood & Krusberg, 1977; Deubert & Rohde, 1971; Krusberg, 1960, 1964; Tracey, 1958) seem capable of overcoming mechanical barriers such as plant cell walls or cuticles. For instance, equal infection of resistant and susceptible cultivars has been reported for: *Meloidogyne incognita* and cotton (McClure, Ellis & Nigh, 1974*a*), or bush type snap beans (Fassuliotis, Deakin & Hoffman, 1970), *M. hapla* and alfalfa (Orion & Cohn, 1975), *Rotylenchulus reniformis* and soybean (Rebois, 1973) or cotton (Carter, 1974), *Heterodera schachtii* and radish (Müller, 1978), *M. incognita acrita* and cucumbers (Fassuliotis, 1970) or lucerne (Reynolds, Carter & O'Bannon, 1970), and *Ditylenchus dispaci* and pea (Muse, 1969). Griffin and Waite (1971) observed significant differences in penetration of resistant and susceptible alfalfa seedlings by *Ditylenchus dipsaci* at 20°, but not at other temperatures. Unlike *M. incognita* and *D. dipsaci*, *M. hapla* had a higher rate of infection in a susceptible alfalfa cultivar regardless of temperature (Griffin & Elgin, 1977). *D. dipsaci* infected but did not reproduce in sweet clover, onion, tomato, sugarbeet, and wheat (Griffin, 1975). From these studies one concludes that nematodes freely penetrate roots of hosts and nonhosts alike and that incompatibility at this stage may occur but is rare. This is very similar to the interaction of fungal pathogens with plant cultivars involved in gene-for-gene relationships in which only a few examples are known of cultivar or single gene resistance at the level of penetration (*e.g.*, Zimmer, Schaelling & Urie, 1968).

Nutrition

The nutrition hypothesis (Garber, 1956; Lewis, 1953) was devised to explain the incompatibility of plants to certain pathogens. In its simplest form, the hypothesis suggests that certain plants are incompatible to a pathogen because they lack one or more essential

nutrients. With few exceptions (*e.g.*, Strange, Smith & Majer, 1972), this idea has seldom been supported by studies with microbial pathogens and has been disproven in most. Although the idea has been applied to incompatibility to nematodes (Wallace, 1961), little or no direct evidence supports it.

It has been hypothesized that nutrition may be important in sexual differentiation in certain nematodes (Davide & Triantaphyllou, 1968) and this may reflect a mechanism of incompatibility. For instance, increased development of non-feeding males which are not required for reproduction (parthenogenic nematodes), would clearly reduce nematode reproduction by reducing the total number of egg-laying females as well as minimize immediate damage to the plant. Orion (1973) found that chlorofluorenol, a growth inhibitor of the morphactin group, inhibited giant cell formation in galls of tomato roots, retarded nematode development, and caused an increase in the percentage of males. Chlorofluorenol did not directly affect nematode metabolism *in vitro*, but inhibited synchronous division of nuclei within giant cells, thereby apparently limiting nutrient availability. A similar effect on sexual differentiation and development of nematodes occurred when maleic hydrazide, a plant growth inhibitor, was applied to the foliage of tobacco and tomato seedlings infected by *M. javanica* and *M. incognita* (Davide & Triantaphyllou, 1968). Translocation of maleic hydrazide from foliage to roots prevented establishment of normal host-parasite relationships by suppressing giant cell formation. Involvement of nutrition in sexual differentiation has been further substantiated in studies involving *M. incognita* (Trudgill, 1972) and *Meloidodera floridensis* (Triantaphyllou & Hirschmann, 1973), in which nematodes were physically removed from their nutritional source. Thus, the data suggests that altered sex expression attributed to changes in host nutrient levels may be a mechanism of incompatibility to certain nematodes.

Migration of larvae from roots shortly after infection may be nutritionally related. Larvae of *Pratylenchus scribneri* migrated out of roots of lima bean (Rich, 1976), and *M. hapla* and *M. incognita acrita* were noted to leave roots of incompatible alfalfa cultivars (Griffin & Elgin, 1977; Reynolds, Carter & O'Bannon, 1970). The actual mechanism(s) responsible for movement of larvae out of roots is unknown. Rather than being based on nutrition, it may reflect an active host response which creates conditions adverse to nematodes within the root.

Active incompatibility

Many studies have reported post-infection changes in the chemical composition of plants infected with nematodes to which they are incompatible, but proof is generally lacking that such alterations are causally related to the expression of incompatibility. As is true with incompatibility to many fungus parasites, limited colonization and development of nematodes in incompatible plants is frequently associated with the so-called *hypersensitive reaction* (HR). Classically, the HR has been considered to involve localized host cell necrosis and disorganization as well as restricted pathogen development at the infection site. In the case of plant pathogenic nematodes, the HR may cause immobilization of the nematode and inhibition of nematode development (Al Tait, 1974; Kaplan, 1978; Orion & Cohn, 1975; Ramana & Rao, 1977; Thomason, Rich & O'Melia, 1976; Van Gundy & Kirkpatrick, 1964). Although hypersensitivity is not completely understood, evidence from several fungus-plant studies indicates that necrosis or disorganization of host cells *per se* is not causally related to pathogen restriction (Kiraly, Barna & Ersek, 1972; Mayama, *et al.*, 1975; Sato & Tomiyama, 1977; Tani *et al.*, 1975), but may be a usual consequence of the incompatible response.

In hypersensitive reactions to fungi, plant cells appear to specifically recognize the pathogen very early after initial contact (Keen & Bruegger, 1977) and this initiates a series of events including *de novo* DNA transcription and protein biosynthesis, finally producing chemical (*viz.* phytoalexins) or physical (*e.g.*, induced lignification or suberization) barriers. If recognition for incompatibility does not occur, however, the plant will be compatible. Incompatibility is an active process and compatibility a passive failure of the plant to respond defensively. This is not to argue against the well

known occurrence of metabolic and anatomical changes in plants infected by compatible obligate parasites such as sedentary endoparasitic nematodes. However, it is now well established on genetic and biochemical grounds that specific recognition generally occurs only to the incompatible pathogen, and that compatibility is a passive failure of the host to recognize the pathogen and respond (Keen & Bruegger, 1977).

Consistent with gene-for-gene systems involving obligate fungal parasites (Ellingboe, 1972; Keen & Littlefield, 1979), the HR to nematodes may be invoked at different times following the initiation of a feeding site by a sedentary parasitic nematode. In the case of sedentary endoparasites such as species of *Meloidogyne*, the HR may be invoked relatively early (*e.g.*, Paulson & Webster, 1972; Van Gundy & Kirkpatrick, 1964) before appreciable giant cell or syncytium formation occurs, or later, after giant cell/syncytium formation has already progressed considerably (Endo, 1965; Powell, 1962; Cotten & Hayes, 1969). Perhaps the efficiency of host recognition of the pathogen is a variable character dependent on the host and nematode genotypes as in fungal systems (Keen & Littlefield, 1979).

Histopathological observations of incompatible reactions to nematodes have included changes in nuclear size and shape, density of cytoplasm, thickness of cell walls, and safranin staining (Al Tait, 1974; Carter, 1974; Dropkin & Nelson, 1960; Hung & Rohde, 1973; Kaplan, 1978; McClure, Ellis & Nigh, 1974*b*; Muse, 1969; Rebois, Madden & Eldridge, 1975; Rohde, 1972; Veech & Endo, 1970). Transmission electron microscopy has enabled us to take a closer look at these events and, despite the limited number of studies published, some conclusions may be drawn. The HR to sedentary endoparasitic nematodes is localized in host cells in the vicinity of the nematode and appears to involve host cell lysis and disorganization. The progression of ultrastructural changes involved in the hypersensitive response of tomato to *Meloidogyne incognita* (Endo & Wergin, 1971; Paulson & Webster, 1972) has been carefully studied. The HR included initial increases in electron density and increased affinity for certain stains by the cytoplasm. Then, a loss of cell membrane distinctness was accompanied by the disappearance of vacuolar inclusions throughout the affected cell. Membrane-bound organelles such as mitochondria and the Golgi apparatus disappeared. However, the endoplasmic reticulum increased in length and was observed as long-branched chains extending throughout the dense cytoplasm. These observations suggest that changes in the host cell may result from lysosome disruption and/or synthesis of proteins (enzymes) which affect disorganization of the cell. They reinforce the conclusion that the HR is a specific plant response to the pathogen, but as with all microscopy studies to date, they offer little insight into biochemical mechanisms.

Rohde (1972) previously summarized the possible role of preformed simple phenols in the incompatible host-parasite interaction. Phenol accumulation and products of phenolic oxidation have been associated with cellular browning in a variety of plants infected by several plant-parasitic nematode genera (Chang, 1969; Giebel, 1970; Giebel, Krenz & Wilski, 1970; Hung & Rhode, 1973). While phenols appear to have limited toxicity, certain oxidation products are more active. Quinones have been reported to be the most toxic forms and also the most reactive (Chang, 1969; Farkas & Kiraly, 1962; Giebel, 1970; Hung & Rohde, 1973).

The effect of phenolic compounds on nematode behavior and metabolism has also been studied. Chlorogenic acid adversely affected nematode coordination (Chang, 1969; Macaron, 1975). The effects of chlorogenic acid, oxidation products of chlorogenic acid, necrotic tissue, and crude extracts from necrotic tissue on migration and respiration of *Pratylenchus penetrans* have been studied *in vitro* (Chang, 1969). Chlorogenic acid acted as an attractant, but its oxidation products possessed nematode repellant properties. Similarly, chlorogenic acid did not affect nematode respiration, but oxidation products of the phenol caused significant reductions in oxygen uptake. Crude extracts of necrotic tissue also reduced respiration. Polymerization of oxidation products, however, destroyed their activity (Rohde, 1972).

Although levels of preformed phenols in roots have also been positively correlated with resistance of certain plant cultivars to nematodes (Cohn, 1974; Ponin *et al.*, 1977; Rohde, 1972;

Sidhu & Webster, 1973; Singh & Choudhury, 1974; Szczygiel & Giebel, 1970), their significance in incompatibility is not yet clear. The correlation of phenols with nematode incompatibility is not always correct (Feldman & Hanks 1971; Brueske & Dropkin, 1973), and critical comparisons of phenol levels in near-isogenic compatible and incompatible host lines have not been made. Thus, no convincing evidence exists indicating the involvement of simple, preformed phenolic compounds or their oxidation products in incompatibility of plants to fungi, bacteria, or nematodes. The majority of experiments studying the relationship of host phenol composition to nematode incompatibility have considered such changes long after the expression of incompatibility has occurred. Proof of phenol involvement requires demonstration that adequate concentrations of the active compound(s) are present in the immediate vicinity of the parasite at the time incompatibility is expressed.

Giebel (1974) regarded simple phenols as modifiers of IAA oxidase activity in a holistic hypothesis of incompatibility based on biochemical studies of the interaction of *Globodera rostochiensis* and potato *(Solanum tuberosum)*. It was hypothesized that hormone levels predicate compatible or incompatible reactions and that hydroxyproline rich glycoproteins and cell wall lignification are important events in incompatibility (Giebel & Krenz, 1975; Giebel & Stobiecka, 1974). We consider the hypothesis unproven because there is a conceptual problem in the design of several of the experiments presented in its support. The delicate timing and nature of the host-parasite interactions were not carefully considered, and chemical analyses of infected tissues were conducted long after incompatibility was expressed (Giebel, 1970, 1974; Giebel, Krenz & Wilski, 1970). Consequently, it is difficult to differentiate between changes in the host which were responsible for limiting parasite development and those which may have occurred secondarily.

Zacheo *et al.* (1977) also noted increased hydroxyproline-rich protein content in tomato mitochondria after incompatibility to *Meloidogyne incognita* was expressed. They interpreted their data to indicate that cyanide insensitive respiration may be an important feature of the hypersensitive defense reaction to nematodes. This may be important because agents such as ethylene, which stimulate cyanide insensitive respiration, also increased the capacity for phytoalexin production in potatoes (Henfling, Lister & Kuc, 1978) and sweet potatoes (Haard, 1977), providing that the tissues were exposed to a suitable elicitor.

Post-infectional production of phytoalexins has often been associated with the hypersensitive response and is generally regarded as an important defense mechanism against certain fungal pathogens (Keen & Bruegger, 1977). Some efforts have been made to determine if phytoalexins are also associated with incompatibility to nematodes (Kaplan, 1978; Rich, Keen & Thomason, 1977; Veech, 1978*b*). *Pratylenchus scribneri* elicits a hypersensitive reaction in roots of lima bean *(Phaseolus lunatus)*, but not in those of snap bean *(Phaseolus vulgaris)* (Thomason, Tich & O'Melia, 1976). Rich, Keen and Thomason (1977) showed that coumestrol and psoralidin accumulated in lima bean roots beginning one day after inoculation, concomitant with the first appearance of hypersensitive symptoms; neither hypersensitive lesions nor significant accumulation of the two compounds occurred in the compatible, inoculated *P. vulgaris* roots. Incubation of *P. scribneri* in low concentrations of coumestrol (10-15 µg/ml) for 48 hours *in vitro* reduced nematode motility by 50 %. However, motility of *M. javanica*, a compatible nematode on lima bean, was not affected by this compound at concentrations of 25 µg/ml. This suggested that coumestrol and psoralidin represent phytoalexins that may be important in the hypersensitive resistance of lima bean roots to *P. scribneri*.

Veech (1977, 1978*b*), and Veech and McClure (1977) observed that the expression of incompatibility in cotton *(Gossypium hirsutum)* to *Meloidogyne incognita* was positively correlated with post-infectional increases in the concentration of methoxy-substituted terpenoid aldehydes The terpenoid aldehydes accumulated at the nematode infection site (Veech, 1979), and crude extracts of the terpenoid aldehydes were toxic to *M. incognita in vitro* (Veech, 1978*a*). The available evidence is thus consistent with the idea that the compounds may account for restricted nematode development.

We recently studied the interactions of two

soybean cultivars with two species of root-knot nematodes to further test the hypothesis that incompatibility may be dependent upon phytoalexin accumulation. The soybean phytoalexin glyceollin has been firmly associated with restriction of pathogen development in the soybean-*Phytophthora megasperma* var. *sojae* host-parasite system (Keen & Bruegger, 1977), and possible involvement of the compound in the nematode system was tested. The incompatible reaction of cv. Centennial to *M. incognita* was positively correlated with significant accumulation of glyceollin (Kaplan & Keen, 1977; Kaplan, 1978), while the related compatible cultivar Pickett 71 accumulated little. The highest concentration of phytoalexin was found in the stele of incompatible roots, the primary site of the incompatible response as deduced by microscopic studies (Kaplan, 1978). Glyceollin accumulated in this tissue when cytoplasm in host cells in the immediate vicinity of the nematode turned brown. Both soybean cultivars were compatible to *M. javanica*; giant cells developed in the stele, and significant glyceollin accumulation did not occur in either. Glyceollin had a nematistatic effect on *M. incognita in vitro* at low concentrations but did not affect *M. javanica*. These observations suggest but do not prove that accumulation of glyceollin may be responsible for the expression of incompatibility and that failure of the plant to accumulate the phytoalexin may account for a compatible reaction.

Induced structural barriers may also be involved in the HR of certain plant roots to nematodes (Al Tait, 1974; Giebel, Kranz & Wilski, 1970; Rebois, Madden & Eldridge, 1975). Thus far, however, no critical testing of the possible causal relationship of these barriers to incompatibility has been done. Thus, these may be wound healing responses that only occur after the expression of incompatibility.

Future considerations

Reversal experiments have been very useful to test critically the role of suggested mechanisms for the incompatibility of plants to certain fungus pathogens and should be used to study physiologically important defense mechanisms in nematode-plant systems. Normal incompatible reactions may be completely blocked by DNA transcription inhibitors or mRNA translation inhibitors (*e.g.*, Vance & Sherwood, 1976) when applied shortly after or immediately before inoculation. When inhibitors have little or no effect on the pathogen at concentrations used, normally incompatible plants will react in a completely compatible manner. Pre-inoculation heat treatments also frequently block the normal expression of incompatibility to fungal parasites (Chamberlain, 1972; Doke, Nakae & Tomiyama, 1976) and nematodes (Dropkin, 1969; Irizarry, Jenkins & Childers, 1971; Paulson & Webster, 1972). Actinomycin D and blasticidin S have been used (Yoshikawa, Yamauchi & Masago, 1978*a*, *b*) to demonstrate that reversal of normal incompatibility expression in soybeans to *Phylophthora megasperma* var. *sojae* also blocked glyceollin production. The experiments clearly supported a causal role for the phytoalexin in the expression of incompatibility. Using similar experiments with the oat-*Puccinia coronata* var. *avenae* host-parasite system, Tani and Yamamoto (1979) showed that blasticidin S also blocked the incompatibility of oat plants to the fungus. As in many host-parasite systems, incompatibility in oats to *Puccinia* normally involved activation of phenylalanine ammonia lyase, peroxidase, and polyphenol oxidase. Such increases in activity had been thought to be causally related to incompatibility, not only to fungi, but also to nematodes. However, when incompatibility was reversed with blasticidin S, the same increases in the three enzymes were observed (Tani & Yamamoto, 1979; Yamamoto *et al.*, 1977; Yamamoto, Hokin & Tani, 1978). These experiments thus preclude activation of PAL, peroxidase, and PPO as the direct cause of incompatibility. Similar approaches may be useful in testing other suggested incompatibility mechanisms. In addition to translation and transcription inhibitors, reversal experiments might involve pre-inoculation heat treatments and cytokinin application which block incompatibility in certain plant-nematode interactions (Dropkin, Helgeson & Upper, 1969; Kochba & Samish, 1971). Sawhney and Webster (1979) recently used cycloheximide to inhibit the visible HR of incompatible tomato roots to *Meloidogyne*

incognita. However, the nematodes still failed to form galls or multiply, even though cycloheximide applied to genetically compatible tomato plants did not interfere with gall formation. The reason for the failure of nematodes to multiply in the inhibitor-treated incompatible plants is unknown. Perhaps cycloheximide blocked the visible HR, but not the chemical mechanism for nematode inhibition.

A difficulty in testing suggested mechanisms of incompatibility to pathogens is determining whether the mechanism occurs at the time and cellular locale where incompatibility is expressed. Although determination of the concentration of toxicants or amounts of structural barriers within incompatible infection sites as small as those initiated by nematodes is difficult, recent progress with incompatible reactions to fungal pathogens suggests approaches to the question. Sato and Tomiyama (1977) made thin serial slices of potato petioles inoculated with incompatible *Phytophthora infestans* races and determined their levels of the phytoalexin rishitin. This allowed a relatively critical assessment of phytoalexin concentration soon after inoculation. Localization of the broad bean phytoalexins wyerone and wyerone acid were studied in epidermal strips (Mansfield, Hargreaves & Boyle, 1974) of leaves infected by the incompatible fungus *Botrytis cinerea* with fluorescence microscopy. Presence of the highly fluorescent phytoalexins was observed in host cells immediately surrounding hypersensitive lesions soon after inoculation. Since this was at or slightly before the time when pathogen development was restricted, it supported a causal role for the phytoalexins. Sherwood and Vance (1976) have performed similar experiments using phloroglucinol staining to demonstrate lignin deposition in incompatible infection sites in reed canary grass leaves. Application of techniques such as these, especially fluorescence microscopy with intact or freshly-sectioned roots infected with incompatible nematodes, might be useful in determining whether levels of phytoalexins or simple phenols were sufficiently high at the infection sites to account for restricted pathogen development.

Another impediment in research on nematode-plant incompatibility mechanisms is the unavailability of near-isogenic plant lines. Unlike the substantial number of such lines with and without dominant resistance genes to microbial pathogens, we are unaware of near-isogenic nematode resistant lines in which the desired 7-15 back-crosses have been made. Without such lines, it is more difficult to critically test the relevance of possible mechanisms of incompatibility. Consequently, it is hoped that plant breeders will incorporate several of the available single genes for nematode resistance into near-isogenic back-crossed lines.

A related approach that has not been well exploited in testing hypotheses of incompatibility mechanisms is the use of virulent and avirulent strains (races) of the same pathogen species on a single plant genotype. Since virulent and avirulent races or strains exist in several plant-nematode systems (for review, see Webster, 1975), they could be more widely used in incompatibility research.

There is no information on mechanisms of incompatibility to ectoparasitic nematodes. Although these nematodes have wide host ranges, cultivars that reduce field populations are known (Cohn, 1974; Schmitt, 1977). While greater difficulty is encountered in working with these nematodes, several laboratories have been successful. It would be of interest to understand the mechanisms by which non-host cultivars limit ectoparasitic nematode reproduction.

From this review it is obvious that more effort is needed to elucidate changes in host metabolism which are essential to incompatible responses. Such information might be used to devise novel disease control measures for diseases in which classical methods are ineffective or difficult to use. Whether this will materialize is unclear, but a certain prerequisite is elucidation of the actual mechanisms conferring natural incompatibility.

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References

Al Tait, B. (1974). Light and electron microscopy of resistant and susceptible alfalfa roots infected by *Meloidogyne hapla. Diss. Abstrs.*, 35B : 672.

Bergé, J. B., Faraut, J. & Ritter, M. (1976). Modifications d'activités enzymatiques sous l'influence de l'infection de plantes par des nématodes. *Ann. Phytopathol.*, 8 : 128-130.

Brueske, C. H. & Dropkin, V. H. (1973). Free phenols and root necrosis in Nematex tomato infected with the root knot nematode. *Phytopathology*, 63 : 329-334.

Carter, W. W. (1974). Histological responses of resistant and susceptible *Gossypium arboreum* to *Rotylenchulus reniformis. J. Nematol.*, 6 : 138.

Chamberlain, D. W. (1972). Heat-induced susceptibility to nonpathogens and cross-protection against *Phytophthora megasperma* var. *sojae* in soybean. *Phytopathology*, 62 : 645-646.

Chiang, L. M. (1969). The repellent effect of necrotic tissue on the nematode, *Pratylenchus penetrans* (Cobb, 1917), Filipjev & Schuurmans Stekhoven, 1941. *University of Massachusetts, M. S. Thesis.*

Chitwood, D. J. & Krusberg, L. R. (1977). Pectolytic enzymes in three populations of *Ditylenchus dipsaci. J. Nematol.*, 9 : 187-192.

Cohn, E. (1974). Relations between *Xiphinema* and *Longidorus* and their host plants. In : Lamberti, F., Taylor, C. E. Seinhorst, J. W. (Eds) *Nematode Vectors of Plant Viruses*, New York, Plenum Press : 365-386.

Cook, R. (1974). Nature and inheritance of nematode resistance in cereals. *J. Nematol.*, 6 : 155-174.

Cotten, J. & Hayes, J. D. (1969). Genetic resistance to the cereal cyst nematode *(Heterodera avenae)*. *Heredity*, 24 : 596-601.

Da Costa, C. P. & Jones, C. M. (1971). Cucumber beetle resistance and mite susceptibility controlled by the bitter gene in *Cucumis sativus* L. *Science*, 172 : 1145-1146.

Daulton, R. A. C. & Curtis, R. F. (1963). The effects of *Tagetes* spp. on *Meloidogyne javanica* in Southern Rhodesia. *Nematologica*, 9 : 357-362.

Davide, R. G. & Triantaphyllou, A. C. (1968). Influence of the environment on development and sex differentiation of root knot nematodes. III. Effect of foliar application of maleic hydrazide. *Nematologica*, 14 : 37-46.

Day, P. R. (1974). *Genetics of host-parasite interaction.* San Francisco, Freeman Press, 238 p.

Deubert, K. H. & Rohde, R. A. (1971). Nematode enzymes. In : Zuckerman, B. M., Mai, W. F., & Rohde, R. A. (Eds) *Plant Parasitic Nematodes* (Vol. II.), New York, Academic Press : 73-90.

Doke, N., Nakae, Y. & Tomiyama, K. (1976). Effect of blasticidin S on the production of rishitin in potato tuber tissue infected by an incompatible race of *Phytophthora infestans. Phytopath. Z.*, 87 : 337-344.

Dropkin, V. H. (1969). The necrotic reaction of tomato and other hosts resistant to *Meloidogyne :* reversal by temperature. *Phytopathology*, 59 : 1632-1637.

Dropkin, V. H. (1976). Nematode parasites of plants, their ecology and the process of infection. In : Heitefuss, R. & Williams, P. H. (Eds) *Physiological Plant Pathology*, Heidelberg, Springer-Verlag : 222-246.

Dropkin, V. H., Helgeson, J. P. & Upper, C. D. (1969). The hypersensitivity reaction of tomatoes resistant to *Meloidogyne incognita :* reversal by cytokinins. *J. Nematol.*, 1 : 55-61.

Dropkin, V. H. & Nelson, P. E. (1960). The histopathology of root-knot nematode infections in soybeans. *Phytopathology*, 50 : 442-447.

Ellingboe, A. H. (1972). Genetics and physiology of primary infection by *Erysiphe graminis. Phytopathology*, 62 : 401-406.

Endo, B. Y. (1965). Histological responses of resistant and susceptible soybean varieties, and backcross progeny to entry and development of *Heterodera glycines. Phytopathology*, 55 : 375-381.

Endo, B. Y. & Wergin, W. P. (1971). Fine-structural changes in red clover *(Trifolium pratense)* roots during penetration by the root-knot nematode, *Meloidogyne incognita. J. Nematol.*, 3 : 309 (Abstr.).

Farkas, G. L. & Kiraly, Z. (1962). Role of phenolic compounds in the physiology of plant diseases and disease resistance. *Phytopath. Z.*, 44 : 105-150.

Fassuliotis, G. (1970). Resistance of *Cucumis* spp. to the root-knot nematode, *Meloidogyne incognita acrita. J. Nematol.*, 2 : 174-177.

Fassuliotis, G., Deakin, J. R. & Hoffman, J. C. (1970). Root-knot nematode resistance in snap beans : breeding and nature of resistance. *J. am. Soc. hort. Sci.*, 95 : 640-645.

Feldman, A. W. & Hanks, R. W. (1971). Attempts to increase tolerance of grapefruit seedlings to the burrowing nematode *(Radopholus similis)* by application of phenolics. *Phytochemistry*, 10 : 701-709.

Garber, E. D. (1956). A nutrition-inhibition hypothesis of pathogenicity. *Am. Nat.*, 90 : 183-194.

Giebel, J. (1970). Phenolic content in roots of some solanaceae and its influence on IAA-oxidase activity as an indicator of resistance to *Heterodera rostochiensis. Nematologica*, 16 : 22-32.

Giebel, J. (1974). Biochemical mechanisms of plant reistance to nematodes : a review. *J. Nematol.*, 6 : 175-184.

Giebel, J. & Krenz, J. (1975). Role of amino acids in plant tissue response to *Heterodera rostochiensis.* II. Effect of proline and hydroxyproline. *Nematol. medit.*, 3 : 49-53.

Giebel, J. & Stobiecka, M. (1974). The role of amino acids in plant tissue response to *Heterodera rostochiensis.* I. Proteinproline and hydroxyproline content in roots of susceptible and resistant solanaceous plants. *Nematologica* 20 : 407-414.

GIEBEL, J., KRENZ, J. & WILSKI, A. (1970). The formation of ligninlike substances in roots of resistant potatoes under the influence of *Heterodera rostochiensis* larvae. *Nematologica*, 16 : 601.

GOMMERS, F. J. (1972). Increase of the nematicidal activity of A-terthienyl and related compounds by light. *Nematologica*, 18 : 458-462.

GOMMERS, F. J. & GEERLIGS, J. W. G. (1973). Lethal effect of near ultraviolet light on *Pratylenchus penetrans* from roots of *Tagetes*. *Nematologica*, 19 : 389-393.

GREEN, C. D. (1971). Mating and host finding behavior of plant nematodes. In : Zuckerman, B. M., Mai, W. F., & Rohde, R. A. (Eds) *Plant Parasitic Nematodes* (Vol. II), New York, Academic Press : 247-266.

GRIFFIN, G. D. (1975). Parasitism of nonhost cultivars by *Ditylenchus dipsaci*. *J. Nematol.*, 7 : 236-238.

GRIFFIN, G. D. & ELGIN, J. H., JR. (1977). Penetration and development of *Meloidogyne hapla* in resistant and susceptible alfalfa under differing temperatures. *J. Nematol.*, 9 : 51-56.

GRIFFIN, G. D. & WAITE, W. W. (1971). Attraction of *Ditylenchus dipsaci* and *Meloidogyne hapla* by resistant and susceptible alfalfa seedlings. *J. Nematol.*, 3 : 215-219.

HAARD, N. F. (1977). Potentiation of wound induced formation of ipomeamarone by cyanide insensitive respiration in sweet potato *(Ipomoea batatas)*. *Z. Pflanzenphysiol.*, 81 : 364-368.

HACKNEY, R. W. & DICKERSON, O. J. (1975). Marigold, castor bean, and chrysanthemum as controls of *Meloidogyne incognita* and *Pratylenchus alleni*. *J. Nematol.*, 7 : 84-90.

HAYNES, R. L. & JONES, C. M. (1976). Effects of the *Bi* locus in cucumber on reproduction, attraction, and response of the plant to infection by the southern root-knot nematode. *J. amer. Soc. hort. Sci.*, 101 : 422-424.

HEATH, M. C. (1974). Light and electron microscope studies of the interactions of host and non-host plants with cowpea rust *Uromyces phaseoli* var. *vignae*. *Physiol. Pl. Pathol.*, 4 : 403-414.

HENFLING, J. W. D. M., LISTER, N. & KUC, J. (1978). Effect of ethylene on phytuberin and phytuberol accumulation in potato tuber slices. *Phytopathology*, 68 : 857-862.

HUNG, C. L. & ROHDE, R. A. (1973). Phenol accumulation related to resistance in tomato to infection by root-knot and lesion nematodes. *J. Nematol.*, 5 : 253-258.

IRIZARRY, H., JENKINS, W. R. & CHILDERS, N. F. (1971). Interaction of soil temperature and *Meloidogyne* spp. on resistance of the common bean, *Phaseolus vulgaris* L., to the root-knot disease. *Nematropica*, 1 : 41-42.

KAPLAN, D. T. (1978). Characterization of soybean incompatibility to *Meloidogyne incognita* and its association to glyceollin accumulation in infected root tissue. *Ph. D. Dissert., Univ. California, Riverside*, 115 p.

KAPLAN, D. T. & KEEN, N. T. (1977). *De novo* synthesis of an isoflavonoid compound by soybeans in response to *Meloidogyne incognita* infection. *J. Nematol.*, 9 : 274.

KEEN, N. T. & BRUEGGER, B. B. (1977). Phytoalexins and chemicals that elicit their production in plants. In : Hedin, P. (Ed.) *Host Plant Resistance to Pests*, American Chemical Society Symposium, Series 62 : 1-26.

KEEN, N. T. & LITTLEFIELD, L. H. (1979). The possible association of phytoalexins with resistance gene expression in flax to *Melampsora lini*. *Physiol. Pl. Pathol.*, 14 : 265-280.

KIRALY, Z., BARNA, B. & ERSEK, T. (1972). Hypersensitivity as a consequence, not the cause, of plant resistance to infection. *Nature, Lond.* 239 : 456-458.

KOCHBA, J. & SAMISH, R. M. (1971). Effect of kinetin and 1-naphthyl-acetic acid on root-knot nematodes in resistant and susceptible peach rootstocks. *J. amer. Soc. hort. Sci.*, 96 : 458-461.

KRUSBERG, L. R. (1960). Hydrolytic and respiratory enzymes of species of *Ditylenchus* and *Pratylenchus*. *Phytopathology*, 50 : 9-22.

KRUSBERG, L. R. (1964). Investigations on *Ditylenchus dipsaci* polygalacturonase and its relation to parasitism of this nematode on alfalfa *(Medicago sativa)*. *Nematologica*, 10 : 72 (Abstr.).

LEVIN, D. A. (1976). The chemical defenses of plants to pathogens and herbivores. *A. Rev. Ecol. Syst.*, 7 : 121-159.

LEWIS, R. W. (1953). An outline of the balance hypothesis of parasitism. *Am. Nat.*, 37 : 273-281.

MACARON, J. (1975). Étude de la résistance de deux variétés de tomate aux *Meloidogyne* spp. et au *Phytophthora parasitica*. *Nematol. medit.*, 3 : 35-41.

MANSFIELD, J. W., HARGREAVES, J. A. & BOYLE, F. C (1974). Phytoalexin production by live cells in broad bean leaves infected with *Botrytis cinerea*. *Nature Lond.*, 252 : 316-317.

MAYAMA, S., DALY, J. M., REHFELD, D. W. & DALY, C. R. (1975). Hypersensitive response of near-isogneic wheat carrying the temperature-sensitive Sr6 allele for resistance to stem rust. *Physiol. Plant Pathol.*, 7 : 35-47.

MCCLURE, M. A. ELLIS, K. C. & NIGH, E. L. (1974*a*). Resistance of cotton to the root-knot nematode, *Meloidogyne incognita*. *J. Nematol.*, 6 : 17-20.

MCCLURE, M. A. ELLIS, K. C. & NIGH, E. L. (1974*b*). Post-infection development and histopathology of *Meloidogyne incognita* in resistant cotton. *J. Nematol.*, 6 : 21-26.

MOTSINGER, R. E., MOODY, E. H. & GAY, C. M. (1977). Reaction of certain French marigold *(Tagetes patula)* cultivars to three *Meloidogyne* spp. *J. Nematol.*, 9 : 278.

MÜLLER, J. (1978). L'élevage monoxénique d'*Heterodera schachtii* sur crucifères et son application pour la sélection des plantes résistantes. *Revue Nématol.*, 1 : 47-52.

MUSE, B. D. (1969). Histopathology of susceptible and resistant reactions in « Wando » pea seedlings to two populations of *Ditylenchus dipsaci*. *J. Nematol.*, 1 : 299 (Abstr.).

ORION, D. (1973). Studies on plant root-knot nematode interrelationships. *OEPP/EPPO Bull.*, 9 : 67-71.

ORION, D. & COHN, E. (1975). A resistant response of citrus roots to the root-knot nematode, *Meloidogyne javanica*. *Marcellia*, 38 : 327-328.

PAULSON, R. E. & WEBSTER, J. M. (1972). Ultrastructure of the hypersensitive reaction in roots of tomato, *Lycopersicon esculentum* L., to infection by the root-knot nematode, *Meloidogyne incognita*. *Physiol. Pl. Pathol.*, 2 : 227-234.

PONIN, I. Y., VOINILO, V. A., GLADKAYA, R. M. & TIMOFEEV, N. N. (1977). Nematode-resistant potato varieties and ways of using them. *Pl. Breed. Abstr.*, 47 : 7545.

POWELL, N. T. (1962). Histological basis of resistance to root-knot nematodes in flue-cured tobacco. *Phytopathology*, 52 : 25 (Abstr.).

PRICE, M., CAVINESS, C. E., & RIGGS, R. D. (1978). Hybridization of races of *Heterodera glycines*. *J. Nematol.*, 10 : 114-118.

RAMANA, K. V. & RAO, Y. S. (1977). Histopathogenesis in rice roots infested by *Hoplolaimus indicus*. *Indian J. Nematol.*, 5 : 232-234.

REBOIS, R. V. (1973). Effect of soil temperature on infectivity and development of *Rotylenchulus reniformis* on resistant and susceptible soybeans, *Glycine max*. *J. Nematol.*, 5 : 10-13.

REBOIS, R. V., MADDEN, P. A. & ELDRIDGE, B. J. (1975). Some ultra-structural changes induced in resistant and susceptible soybean roots following infection by *Rotylenchulus reniformis*. *J. Nematol.*, 7 : 122-139.

REYNOLDS, H. W., CARTER, W. W. & O'BANNON, J. H. (1970). Symptomless resistance of alfalfa to *Meloidogyne incognita acrita*. *J. Nematol.*, 2 : 131-134.

RICH, J. R. (1976). Association of coumestans with the resistance of lima bean *(Phaseolus lunatus)* to *Pratylenchus scribneri* and some other host-parasite relations of this nematode. *Ph. D. Dissert., Univ. California, Riverside*, 72 p.

RICH, J. R., KEEN, N. T. & THOMASON, I. J. (1977). Association of coumestans with the hypersensitivity of lima bean roots to *Pratylenchus scribneri*. *Physiol. Pl. Pathol.*, 10 : 105-116.

ROHDE, R. A. (1972). Expression of resistance in plants to nematodes. *A. Rev. Phytopath.*, 10 : 233-252.

ROHDE, R. A. & JENKINS, W. R. (1958). Basis for resistance of *Asparagus officinalis* var. *altilia* L. to the stubby root nematode *Trichodorus christiei* (Allen 1957). *Univ. Maryland Agric. Expt. Sta. Bull.*, A-97 : 1-20.

ROYLE, D. J. (1976). Structural features of resistance to plant diseases. In : Friend, J. & Threlfall, D. R. (Eds) *Biochemical Aspects of Plant-Parasite Relationships*, London, Academic Press : 161-193.

SATO, N. & TOMIYAMA, K. (1977). Relation between inhibition of intracellular hyphal growth of *Phytophthora infestans* and rishitin concentrations in infected potato cells. *Ann. phyto-path. Soc. Japan*, 43 : 598-600.

SAWHNEY, R. & WEBSTER, J. M. (1979). The influence of some metabolic inhibitors on the response of susceptible/resistant cultivars of tomato to *Meloidogyne incognita*. *Nematologica*, 25 : 86-93.

SCHMITT, D. P. (1977). Suitability of soybean cultivars to *Xiphinema americanum* and *Macroposthonia ornata*. *J. Nematol.*, 9 : 284 (Abstr.).

SCHÖNBECK, F. & SCHLÖSSER, E. (1976). Preformed substances as potential protectants. In : Heitefuss, R. & Williams, P. H. (Eds) *Physiological Plant Pathology, Encyclopedia of Plant Physiology*, Berlin, Springer-Verlag : 653-678.

SHERWOOD, R. T. & VANCE, C. P. (1976). Histochemistry of papillae formed in reed canarygrass leaves in response to noninfecting pathogenic fungi. *Phytopathology*, 66 : 503-510.

SIDHU, G. & WEBSTER, J. M. (1973). Genetic control of resistance in tomato. I. Identification of genes for host resistance to *Meloidogyne incognita*. *Nematologica*, 19 : 546-550.

SINGH, B. & CHOUDHURY, B. (1973). The chemical characteristics of tomato cultivars resistant to root-knot nematodes *(Meloidogyne* spp.). *Nematologica*, 19 : 443-448.

STRANGE, R. N., SMITH, H. & MAJER, J. R. (1972). Choline, one of two fungal growth stimulants in anthers responsible for the susceptibility of wheat to *Fusarium graminearum*. *Nature, Lond.*, 238 : 103-104.

SZCZYGIEL, A. & GIEBEL, J. (1970). Phenols in the leaf buds of two strawberry varieties resistant and susceptible to *Aphelenchoides fragariae*. *Proc. Int. Nematol. Symposium, Warsaw :* 247-253.

TANI, T., & YAMAMOTO, H. RNA and protein syntheses and enzyme changes during infection. In : Daly, J. M. (Ed.) *Recognition and Specificity in Host-Parasite Interactions* (In press).

TANI, T., YAMAMOTO, H., ONOE, T. & NAITO, N. (1975). Initiation of resistance and host cell collapse in the hypersensitive reaction of oat leaves against *Puccinia coronata avenae*. *Physiol. Pl. Pathol.* 7 : 231-242.

THOMASON, I. J., RICH, J. R. & O'MELIA, F. O. (1976), Pathology and histopathology of *Pratylenchus scribneri* infection of snap bean and lima bean. *J. Nematol.*, 8 : 347-352.

TRACEY, M. V. (1958). Cellulase and chitinase in plant nematodes. *Nematologica*, 3 : 179-183.

TRIANTAPHYLLOU, A. C. & HIRSCHMANN, H. (1973). Environmentally controlled sex expression in *Meloidodera floridensis*. *J. Nematol.*, 5 : 181-185.

TRUDGILL, D. L. (1972). Influence of feeding duration on moulting and sex determination of *Meloidogyne incognita*. *Nematologica*, 18 : 476-481.

UHLENBRECK, J. H. & BIJLOO, J. D. (1958). Investigations on nematicides. I. Isolation and structure of a nematicidal principle occurring in *Tagetes* roots. *Rec. Trav. Chim. Pays-Bas Belg.*, 77 : 1004-1009.

UHLENBRECK, J. H. & BIJLOO, J. D. (1959). Investigations on nematicides. II. Structure of a second nematicidal principle isolated from *Tagetes* roots. *Rec. Trav. Chim. Pays-Bas Belg.*, 78 : 382-390.

VANCE, C. P. & SHERWOOD, R. T. (1976). Cycloheximide treatments implicate papilla formation in resistance of reed canarygrass to fungi. *Phytopathology*, 66 : 498-502.

VAN GUNDY, S. D. & KIRKPATRICK, J. D. (1964). Nature of resistance in certain citrus rootstocks to citrus nematode. *Phytopathology*, 54 : 419-427.

VEECH, J. A. (1977). The relationship between terpenoid aldehydes and the resistance of cotton to *Meloidogyne incognita*. *J. Nematol.*, 9 : 287-288 (Abstr.).

VEECH, J. A. (1978*a*). The toxicity of terpenoid aldehydes to nematodes. *J. Nematol.*, 10 : 301 (Abstr.).

VEECH, J. A. (1978*b*). An apparent relationship between methoxy-substituted terpenoid aldehydes and the resistance of cotton to *Meloidogyne incognita*. *Nematologica*, 24 : 81-87.

VEECH, J. A. (1979). Histochemical localization and nematotoxicity of terpenoid aldehydes in cotton. *J. Nematol.*, 11 : 240-246.

VEECH, J. A. & ENDO, B. Y. (1970). Comparative morphology and enzyme histochemistry in root knot resistant and susceptible soybeans. *Phytopathology*, 60 : 896-902.

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VEECH, J. A. & MCCLURE, M. A. (1977). Terpenoid aldehydes in cotton roots susceptible and resistant to the root-knot nematode, *Meloidogyne incognita*. *J. Nematol.*, 9 : 225-229.

WALLACE, H. R. (1961). The nature of resistance in chrysanthemum varieties to *Aphelenchoides ritzemabosi*. *Nematologica*, 6 : 49-58.

WEBSTER, J. M. (1975). Aspects of the host-parasite relationship of plant-parasitic nematodes. In : Dawes, B. (Ed.) *Advances in Parasitology* (Vol. 13), London and New York, Academic Press : 225-250.

WINOTO, R. S. (1969). *Studies on the effect of* Tagetes *species on plant parasitic nematodes*. Wageningen, Veenman & Zonen, 134 p.

YAMAMOTO, H., HOKIN, H. & TANI, T. (1978). Peroxidase and polyphenoloxidase in relation to the crown rust resistance of oat leaves. *Phytopath., Z.*, 91 : 193-202.

YAMAMOTO, H., HOKIN, H., TANI, T. & KADOTA, G. (1977). Phenylalanine ammonia-lyase in relation to the crown rust resistance of oat leaves. *Phytopathol. Z.*, 90 : 203-211.

YOSHIKAWA, M., YAMAUCHI, K. & MASAGO, H. (1978*a*). *De novo* messenger RNA and protein synthesis are required for phytoalexin-mediated disease resistance in soybean hypocotyls. *Plant Physiol.*, 61 : 314-317.

YOSHIKAWA, M., YAMAUCHI, K. & MASAGO, H. (1978*b*). Glyceollin : its role in restricting fungal growth in resistant soybean hypocotyls infected with *Phytophthora megasperma* var. *sojae*. *Physiol. Plant Pathol.*, 12 : 73-82.

ZACHEO, G., LAMBERTI, F., ARRIGONI-LISO, R. & ARRIGANI, O. (1977). Mitochondrial protein-hydroxyproline content of susceptible and resistant tomatoes infected by *Meloidogyne incognita*. *Nematologica*, 23 : 471-476.

ZIMMER, D. E., SCHAELLING, P. & URIE, A. L. (1968). The nature and inheritance of seedling rust resistance of Nebraska 1-1-5 safflower. *Phytopathology*, 58 : 1451-1456.