Biochemical Mechanisms of Plant Resistance to Nematodes: A Review[1]

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In general, a plant resistant to nematodes resists attack or exhibits little damage and reduces nematode populations. The following four types of resistance may be distinguished: (i) The plant may produce toxins that kill the nematodes; e.g., *Asparagus officinalis* contains in its stems, leaves and roots a glycoside toxic to *Trichodorus christiei*, and populations of this nematode rapidly decline around the roots of these plants (57). The roots of *Tagetes patula* and *T. erecta* contain α-terthienyl and derivatives of bithienyl (68) which limit *Meloidogyne* and *Pratylenchus* populations (50, 77). *Eragrostis curvula* is resistant to four species of *Meloidogyne* (61) due to high concentration of pyrocatechol in its roots. (ii) The resistance depends upon the fact that not every plant contains substances necessary for the development and reproduction of a certain nematode species, or contains them in unsufficient amounts; e.g., Webster (73) stated that the indoleacetic acid and its precursor, tryptophan, are necessary for the reproduction of *Aphelenchoides ritzemabosi*. Szczygiel and Giebel (64) found systems of IAA-oxidase in strawberry varieties resistant to *A. fragariae* that were more active than those in susceptible varieties. Conceivably, indoleacetic acid or its derivatives represent some of the compounds that stimulate nematode development and reproduction. (iii) The resistance can be based on lack of attraction. Here, different chemical compounds secreted by plants may be neutral or act as attractants on different nematode species. (iv) Plant resistance is based on plant tissue hypersensitivity to nematode infection. The host-parasite interaction stimulates in the host definite biochemical reactions that cause histological changes; i.e., host cell necroses. These necroses form around the nematodes walling them off and either delaying development or causing them to die (8, 72).

With respect to susceptible host response to nematode invasion, Viglierchio (71) discerns three categories: (i) cell damage due to penetration is not conspicuous except for some cell separation; (ii) only some cells are changed, exhibiting conspicuous lesions, necroses or discolorations; (iii) galls, multinucleate syncytia or giant cells are formed, various degrees of hyperplasia and hypertrophy occur, and meristematic activity declines.

Plant response to the parasite depends not only upon the chemical composition of the plant or tissues attacked, but also upon the quantitative and qualitative composition of nematode secretion and excretion. These are related to nematode species or pathotype. The kind of parasitism (i.e., whether the nematode is sedentary or migratory, endo- or ectoparasitic) is also of significance. These features determine the extent of the feeding time on a given cell and the introduction of nematode secretions alone, or in combination with excretions.

Because the fourth kind of resistance is of greatest importance for plant protection, the following will relate mainly to it.

PRIMARY AND SECONDARY REASONS FOR CHANGES IN PLANT METABOLISM

Nematodes parasitizing plant tissues cause mechanical damage with their stylets. Presumably, they also secrete hydrolytic enzymes from their esophageal glands which dissolve cell walls enabling intracellular and intercellular movement of endoparasites, and they act as "digestive enzymes" digesting solid components of cells which can be imbibed by the parasite.

Many hydrolases have been found in nematodes (Table 1). The most common are cellulase, protease, and amylase. I believe hydrolases are the primary reason for metabolic changes caused by nematodes. Enzyme activities in nematode are probably influenced by parasitic activities. For example, in the migratory endoparasite *Pratylenchus penetrans* cellulase activity has been found to be seven times more active than in the sedentary endoparasite *Heterodera trifolii* (38). The stem nematode, *Ditylenchus dipsaci*, showed cellulase activity 28 times

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TABLE 1. Hydrolases identified by analysis of nematode secretion or nematode homogenates.

Enzyme	Nematode species	References
Pectinmethylesterase	*Ditylenchus triformis, D. myceliophagus, D. dipsaci*, alfalfa race.	13, 29
Non-specific esterases	*Meloidogyne javanica, M.hapla.*	6
Alkaline phosphatase	*Meloidogyne* spp.	7
Acid phosphatase	*D. triformis, Panagrellus redivivus, Meloidogyne* spp., *M. javanica, Tylenchulus semipenetrans.*	4, 69
Amylase	*Rhabditis terricola, Aphelenchoides sacchari, T. semipenetrans, D. dipsaci, D. destructor, D. triformis, Heterodera schachtii, H. rostochiensis, M. javanica.*	11, 42, 80
Cellulase	*A. avenae, A. sacchari, D. dipsaci, D. myceliophagus, D. triformis, Radopholus similis, Helicotylenchus nannus, Pratylenchus penetrans, P. zeae, H. trifolii, H. schachtii, M. arenaria, M. incognita, T. semipenetrans, Rhabditis*	3, 6, 13, 29, 38, 40, 42, 41, 66
Polygalacturonase	*A. avenae, P. zeae, D. dipsaci, D. myceliophagus, D. destructor.*	3, 13, 29, 30, 31, 40
Invertase	*A. sacchari, M. arenaria, R. similis, P. penetrans, P. redivivus*	42
β-glucosidase	*P. penetrans, Aphelenchoides fragariae H. rostochiensis.*	33, 39, 74
β-galactosidase	*H. rostochiensis*	74
Pectinase	*D. dipsaci, D. destructor, R. similis, P. penetrans, M. arenaria, M. hapla.*	38, 42, 66
Proteinase	*H. rostochiensis, H. schachtii, D. allii, D. destructor, D. triformis, D. dipsaci, A. ritzemabosi, P. redivivus.*	29, 36, 44, 45, 46, 80
Chitinase	*D. dipsaci, D. myceliophagus, D. destructor*	66

higher then did *D. myceliophagus* feeding on fungi (13).

The pectolytic and cellulolytic nematode enzymes that destroy host cells may activate plant endohydrolases. In this manner, the total hydrolytic activity at the feeding site can increase. Treatment of plant tissues with pectolytic and celluloytic enzymes involves the activity of oxidoreductases (63). Actually, in the area invaded by *M. incognita acrita* and *H. rostochiensis* hydrolase, oxidase and dehydrogenase activity was found to increase (15, 23, 70). The oxidoreductases seem to be connected with susceptible-resistant response of plant tissues; they are able to modify many physiologically active host cell components, such as phenols, auxins, and amino acids. It is conceivable that metabolic excretions of the parasites, such as organic acids, amines, amino acids, and indole compounds (43, 71) play an important role in initiating host plant reaction to nematode invasion.

ROLE OF AUXINS AND OTHER PLANT HORMONES IN HOST PLANT RESPONSE TO NEMATODES

Changes caused by some nematode species in cells of a susceptible host are similar to those caused by exogenous indoleacetic acid; i.e., hypertrophy, hyperplasia, adventitious roots, nuclear division without cytokinesis, and the breakdown of cell walls (16). Probably, the mechanism of auxin action depends upon the induction of RNA synthesis, the acid that controls cellulase synthesis and thus influences the plasticity of cell walls (1, 16, 49). Naylor et al. (47) showed that in excised tobacco pith tissues grown in vitro on a mineral nutrient with indoleacetic acid, intensive cell enlargement and mitotic divisions without cytokinesis occurs. Mitosis was not accompanied by the formation of cell walls and multinuclear cells were formed. The

addition of kinetin restored cytokinesis. Two substances, IAA and kinetin, are needed for normal cytokinesis.

In root galls of *Hibiscus esculentus* caused by *M. javanica,* paper chromatography revealed the presence of indole compounds (2). Bird (5), and Yu and Viglierchio (79) found indole-3-acetic acid (IAA), indole-3-acetonitrile (IAN), indole-3-acetic acid ethylester (IAE), or indole-3-butyric acid (IBA) in the extracts of galls incited on tomato roots by *M. hapla, M. javanica* and *M. incognita.*

The question now arises, what is the source of these auxins? There are several more-or-less documented hypotheses. The first possibility is that the nematodes secrete glycosidases or proteases into host tissues and release free auxins from complexes (24, 59). Also, the proteases breaking down protein may supply tryptophan, the IAA precursor, and amino acids such as phenylalanine, alanine, histidine, and serine which promote auxin synthesis (78).

Perhaps indole compounds are formed in nematodes as end-products of metabolism and are excreted by endoparasitic nematodes into the plant tissues or by ectoparasitic nematodes into the root region. Hence, probably great cytological changes occur in hosts invaded by such species as *Meloidogyne, Heterodera* and *Ditylenchus.* But in nematode secretions the presence of auxins have not been established (6), although in larvae and in egg masses of *Meloidogyne* spp. IAA, IAN, IAE, IBA (79), in *H. schachtii* larvae IAA (28), and in *D. dipsaci* indole-3-acetic acid methyl ester, IAM, (12) were found. It is not excluded, that indole compounds can be introduced into plants by nematodes similarly to the action of aphids and plant bugs where IAA synthesis occurs from tryptophan and the auxin is secreted via insect saliva (34, 35, 60). The IAA concn in insect saliva was correlated with the formation of plant galls incited by insect feeding.

No gibberellins nor cytokinins were found in nematode secretions (6, 58), but it is possible that these compounds as well as indole derivatives are excreted by nematodes into the environment. In that case, these compounds might be detected only during the process of nematode feeding. This would be of interest, considering the distinct role played by cytokinins in plant response to nematodes. Dropkin et al. (14) stated that the hypersensitivity reaction of resistance to *Meloidogyne* spp. can be reversed toward the susceptible reaction by exogenous cytokinins. IAA, NAA, and GA did not alter the resistant reaction.

ROLE OF PHENOLICS

The phenolic compounds are the best-known factors involved in susceptible-resistant response. There is a distinct correlation between the degree of plant resistance and the phenolics present in plant tissue (19, 54, 67). Most phenols occur in plant tissues in bound form as glycosides of low physiological and chemical activity. Activation requires their decomposition to free phenols (19, 26, 33, 39). Nematodes are able to do this by secreting β-glycosidases into the host tissue (33, 39, 74).

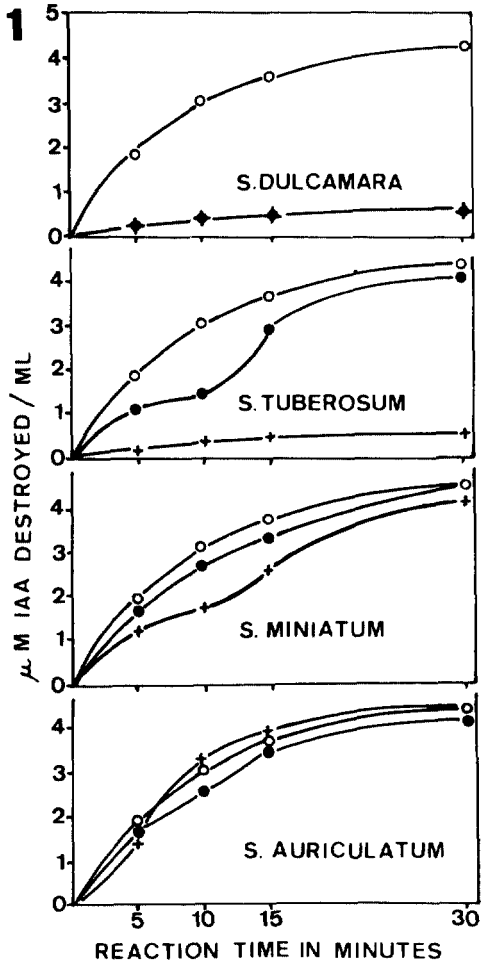
According to results of many authors, it is possible to distinguish four kinds of host response to nematode invasion in which phenolics play the main role: (i) Browning and slow formation of wide necroses in plants susceptible to the migratory nematodes. (ii) Quick browning and formation of non-expandable necroses in plants resistant to migratory parasites. (iii) IAA-oxidase inhibition which may favor auxin accumulation and, consequently, giant cell formation, galls, etc. in plants susceptible to sedentary endoparasites. (iv) IAA-oxidase stimulation which favors auxin decomposition and formation of necroses in plants resistant to sedentary endoparasites.

Mountain and Patrick (39) found that the glucoside amygdaline present in peach roots is hydrolyzed by β-glucosidase of *Pratylenchus penetrans* to hydrocyanic acid and benzaldehyde. These compounds were noxious to both the parasite and the host causing browning and death of the penetrated tissues. The rapidity of death was dependent on amygdaline concentration.

The presence of chlorogenic acid is thought to be the cause of browning and of the resistant reaction of chrysanthemums to *A. ritzemabosi* (72), and of *Nicotiana repanda* (37) and 'Nemared' tomato (52) to *M. incognita.*

HYPOTHESIS OF THE MECHANISM OF PLANT RESISTANCE TO *HETERODERA ROSTOCHIENSIS*

This hypothesis is based mainly on the



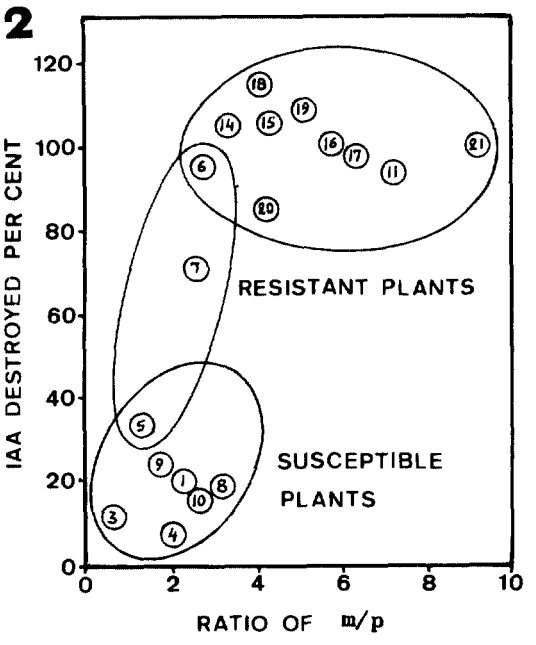


FIG. 1-2. 1. IAA destruction caused by peroxidase systems: o——o control, +——+ with potato root extracts hydrolysed with HCl, ●——● with potato root extracts not hydrolysed. [after Giebel (19)] 2. Chemical classification of some Solanaceae into those which are susceptible and resistant to *H. rostochiensis*, respectively, made on basis of the m/p ratio and the percentage of IAA decomposed in peroxidase systems with potato root extracts. [after Giebel (19)].

results of investigations carried out at the Institute for Plant Protection in Poznan, Poland.

For the development of this hypothesis, one must assume that the pathogenic changes which occur in susceptible plant tissues after nematode invasion are due to auxins secreted by the nematode or released from complexes by the action of nematode enzymes such as glycosidases and proteases. Admittedly there is little or no evidence for this assumption.

One of the first experiments has shown (24) that β-glucosidase introduced into potato roots by means of glass micro-capillaries caused necroses in resistant plants, and giant cells in susceptible ones (Fig. 4, 5). This indicated that factors which influence the susceptible-resistant reaction occur in plants in a nonactive form as glycosides. These compounds, decomposed by nematode β-glycosidases can form physiologically and biochemically active aglycones. Actually, in the invasive second stage larvae of *H. rostochiensis* pathotype A, a very active β-glucosidase and a less active β-galactosidase were found (74). In pathotype B (now *H. pallida*) no β-galactosidase was found, and the activity of β-glucosidase was five times less than in *H. rostochiensis*. It may be assumed that plants resistant to pathotype A are not resistant to pathotype B, because the latter is not able to release "the resistant factor" from the nonactive glycoside form.

As the result of glycosidase activity in susceptible plant tissues damaged by nematodes, a system which favors IAA accumulation is stimulated. Conversely, in resistant plants a system which destroys IAA is formed (19, 26, 75, 76). The level of auxins in the plant depends on the activity of IAA-oxidase. This activity can be modified by free phenolic compounds (Fig. 1). Polyphenols

usually synergize IAA activity, while monophenols often antagonize IAA (27, 48, 65). Giebel (19) found that root extracts from good hosts of *H. rostochiensis* were characterized by a low ratio of monophenols to polyphenols (0.5-3.1), and they strongly inhibited the enzymatic destruction of IAA. Root extracts of plants resistant to this nematode showed a high ratio of mono- to polyphenols (3.4-9.3) and they did not inhibit, or very slightly inhibited, IAA destruction (Fig. 2). Besides, after root infection with *H. rostochiensis* larvae, the level of phenolics capable of inhibiting IAA action increased in resistant potato plants, whereas it decreased in roots of susceptible ones (76). One of the fractions of phenols obtained from resistant potato roots and introduced into susceptible potato plants shifted their reaction from susceptible to resistant (Fig. 8). Conversely, one of the fractions of phenols shifted the reaction of resistant potato root tissues toward a susceptible reaction (Fig. 7).

Histological tests of necroses formed in resistant potato after the invasion of *H. rostochiensis* larvae showed a positive reaction for lignin-like substances, but they did not show positive reaction for cellulose, callose or suberines (22). The strongest reactions for lignin were observed in cells adjacent to the head and to the excretory pore of the nematode (Fig. 6). These observations suggest that the processes of lignin formation are stimulated by substances excreted by the nematode.

Many authors (51, 62) are of the opinion that the processes of lignification cannot occur until IAA action stops. If this is true, the active systems of IAA-oxidase that occur in resistant plants favor lignin synthesis. In fact, in the region of necrotic cells (Fig. 9) an active peroxidase was found (23). In giant cells the activity of this enzyme has not been detected (Fig. 10). Veech and Endo (70) obtained rather different results. They observed a highly active peroxidase in the syncytia formed in soybean roots infected with *M. incognita acrita*. Maybe there are differences in the reaction of potato and soybean to different nematode species. Such great differences between susceptible and resistant potatoes in the activity of peroxidase indicate that there are phenolic compounds in potato roots that act as cofactors of peroxidase and hence influence the resistance of plants.

Phenol oxidase activity can be involved in

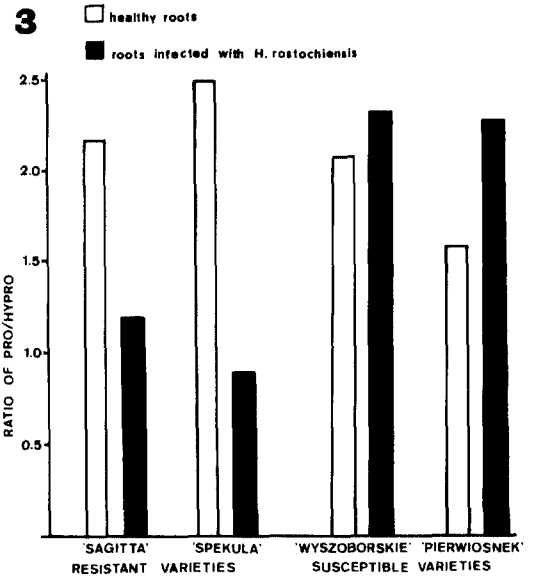


FIG. 3. Changes of the ratio of protein proline to hydroxyproline (PRO/HYPRO) in susceptible and resistant potato roots after their infection with *H. rostochiensis*. [after Giebel and Stobiecka (25)].

the modification of phenolics in plants. Activity increases both in the giant cells (Fig. 11) and in necrotic cells (Fig. 12), as compared with healthy tissue (22). This enzyme can oxidize some phenols to quinones perhaps formed in the injured tissue of susceptible plants. This would favor IAA synthesis from tryptophan. This amino acid can be released from plant protein by the proteolytic enzyme activity of *H. rostochiensis* larvae.

β-glucosidase was active in necrotic cells, whereas in giant cells its activity was low (22). In roots of susceptible plants the active enzyme was observed in cells adjacent to nematodes. The activity of β-glucosidase depends to a large degree on the pH. Thus, in necrotic cells where pH is low, this enzyme is more active than in giant cells where pH is higher (53). Undoubtedly pH also will influence the activity of peroxidase which is more active at a low pH.

Plant resistance can be affected by phenylalanine ammonia-lyase (PAL) and tyrosine ammonia-lyase (TAL) activity. Viruses and fungi, which cause necroses in plants, also increase the level of these two deaminases (17, 56). Higher activities of PAL and TAL were found in potato roots resistant to *H. rostochiensis* than in the roots of susceptible plants (20). Both cinnamic acid

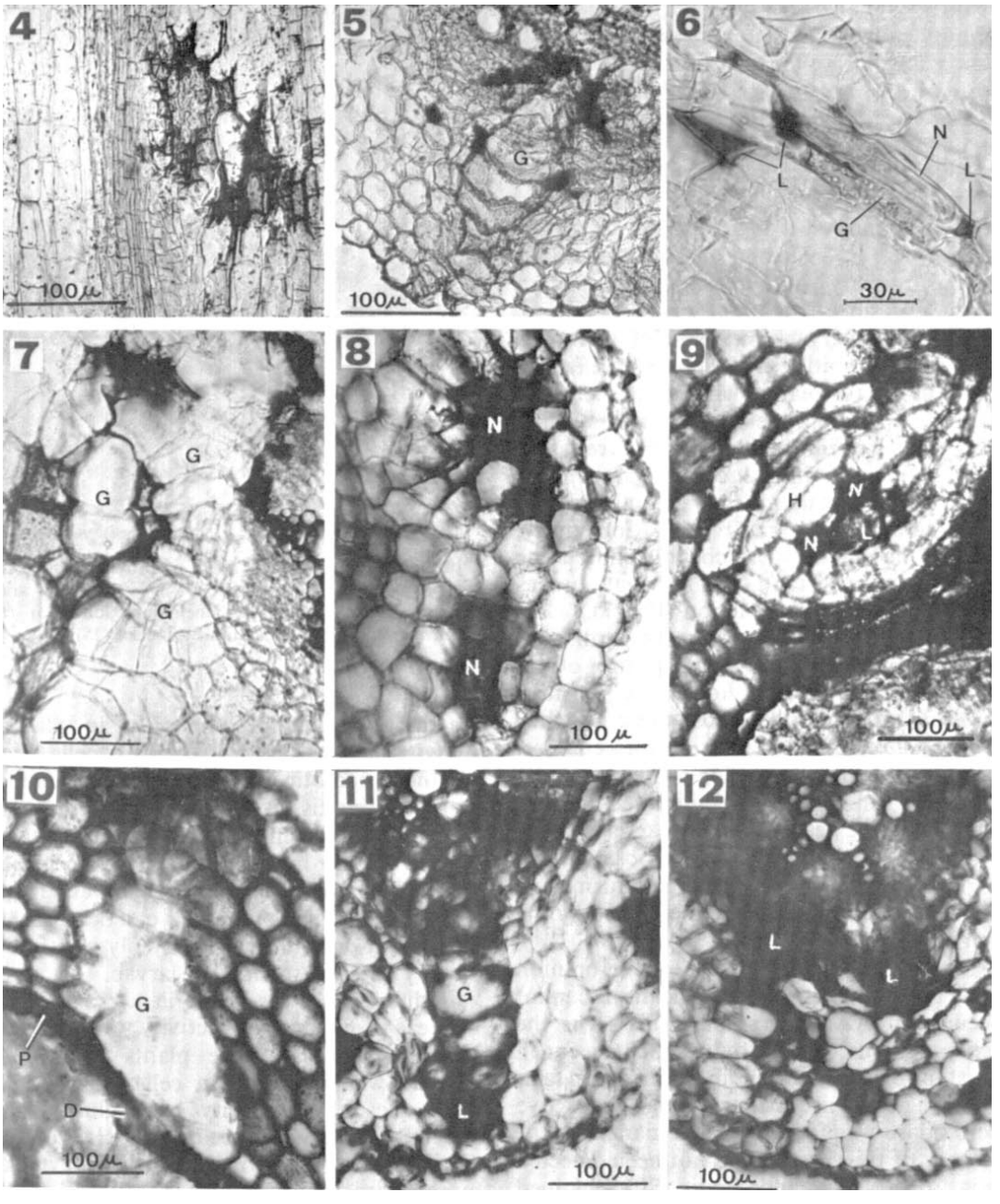


FIG. 4-12. **4-5**) Sections of resistant and susceptible potato root, respectively. Tissue necroses (dark areas) and thickened cell walls resulting from β-glucosidase insertion are seen. Giant cell (G) is forming near the point of β-glucosidase insertion. [after Giebel et al. (24)]. **6**) Root section of potato resistant to *H. rostochiensis* invaded by this nematode (N). Necrotic cells (L) and/or thickened cell wall giving positive reaction for lignin, and cell filled with granulation (G) [after Giebel et al. (22)]. **7-8**) Giant cells (G) in resistant potato root caused by both *H. rostochiensis* and fraction no. 5 from ethanol potato root extracts, and necrotic cells (N) in susceptible plant caused by both *H. rostochiensis* and fraction no. 3 from these extracts, respectively. [after Wilski and Giebel (76)]. **9-10**) Localization of the active peroxidase in root of resistant and susceptible potato infected with *H. rostochiensis*, respectively. The necroses (N) formed around the larvae (L) and containing the active enzyme are surrounded with cell (H) showing no enzyme activity. The giant cells (G) do not show an active peroxidase. Pericycle (P) shows the presence of the active enzyme which disappears near (D) the forming giant cell. [after Giebel et al. (23)]. **11-12**) Localization of the active tyrosinase in root of susceptible and resistant potato infected with *H. rostochiensis*, respectively. Larvae (L), the developing giant cell (G) and necroses (N) show the presence of an active enzyme. [after Giebel et al. (23)].

and p-coumaric acid, the products of PAL and TAL activities, respectively, are lignin precursors and cofactors of IAA-oxidase (51, 65). These acids, introduced into susceptible potato plants infected with *H. rostochiensis* larvae, prevented a susceptible response to nematode invasion. Typical giant cells were not formed; and, in many instances, typical necrotic cells were observed (20).

In roots of resistant solanaceous plants, on the average, 50% more hydroxyproline was found than in roots of susceptible ones (25).

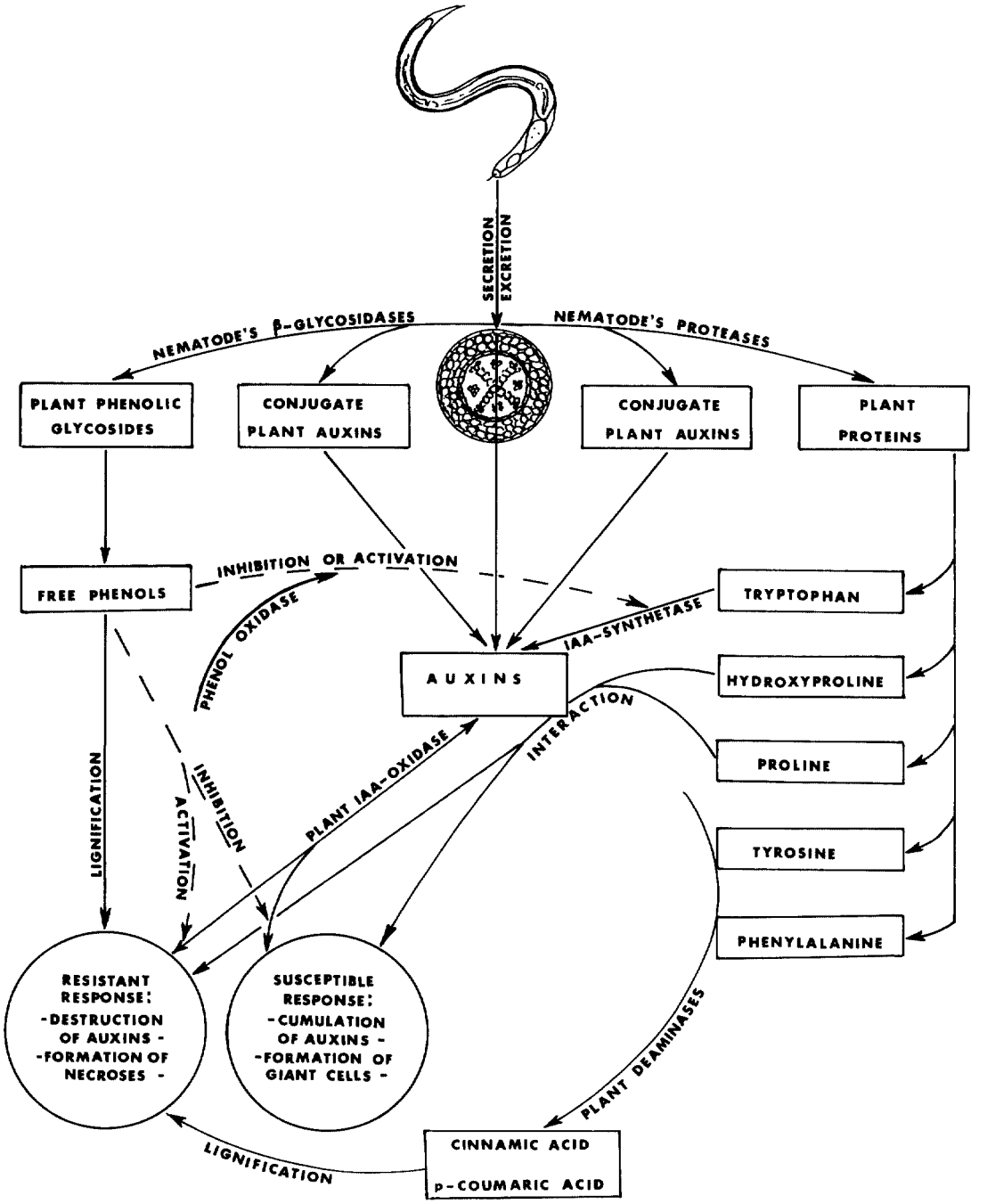


FIG. 13. Hypothetical reactions involved in biochemical mechanism of plant resistance and susceptibility to *H. rostochiensis*.

Following nematode invasion of resistant potato plants, the ratio of protein proline to hydroxyproline (PRO/HYPRO) decreased, while it increased in susceptible ones (Fig. 3). What role can these two amino acids play in the mechanism of plant resistance? Hydroxyproline introduced into solutions strongly inhibits the elongation of immersed *Avena* coleoptile sections (9, 10). Also, it strongly inhibits the growth of carrot callus tissue (55). This inhibitory effect of hydroxyproline is reversed by proline. In plants, hydroxyproline is present mainly in cell wall glycoproteins (32). The hypothesis may be proposed that hydroxyproline-rich protein which inhibits cell extensibility can suppress the hypertrophy. Besides, enzymes of *H. rostochiensis* can decompose glycoproteins to free hydroxyproline. In this way, this amino acid may inhibit the action of IAA. But, this action of hydroxyproline may be reversed by proline which is either free in plants or is released from proteins.

One would expect that a low ratio of PRO/HYPRO in cell wall protein after nematode infection will favor resistance. This suggestion was confirmed experimentally (21) when susceptible potato watered for a few days with hydroxyproline solution showed nearly the same reaction as resistant plants; i.e., giant cells were formed only occasionally, but often necroses were seen. This response was not observed when plants were watered with a mixture of both hydroxyproline and proline solutions.

On the basis of results presented above, it is possible to propose a hypothesis of biochemical mechanism of plant resistance and susceptibility to *H. rostochiensis*. The hypothesis which I presented in Fig. 13 is, no doubt, more complicated and, probably, involves many systems of plant metabolism. Although, this hypothesis relates to the potato/*H. rostochiensis* relationship, it is possible that some mechanism of plant resistance to other nematode species can be explained by it.

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