Phytoalexins and Their Role in the Resistance of Plants to Nematodes[1]

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Abstract: Phytoalexins are antibiotic compounds synthesized in an infected plant in response to infection. Nematodes are capable of eliciting phytoalexins in resistant plants. Resistant lima bean (*Phaseolus lunatus*) infected by *Pratylenchus penetrans* produces the phytoalexin coumestrol; soybean (*Glycine max*) infected by *Meloidogyne incognita* produces glyceollin; cotton (*Gossypium hirsutum*) infected by *M. incognita* produces terpenoid aldehydes. *Key words:* review, physiology, host plant resistance.
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In their daily existence plants may be exposed to a multitude of organisms. Most of the organisms cause no apparent harm. Organisms constituting a biological threat are often thwarted successfully. Thus, it is axiomatic that plants are resistant to most of the organisms they encounter (for to be otherwise would be disasterous to the perpetuation of the plant species). Inherent in this concept are two general types of resistance: one predicated on constitutive factors that preclude infection—preinfection resistance, and the other on factors that unfold after infection—postinfection resistance.

Preinfection resistance is probably the most common type of resistance, and more often than not the plant involved in the relationship is considered a "nonhost" of the organism(s) it encounters. In postinfection resistance the plant becomes infected but it does not succumb to the advances of the hostile organism. This type of resistance may involve constitutive morphological or biochemical factors, or it may depend on the plant's response to infection. The plant's response may involve the production of morphological barriers that sequester the infecting organism or, it may involve the synthesis of certain biochemicals that interfere with the subsequent development of the pathogen. Among plant biochemical responses to infection are the synthesis of hydrolytic enzymes, protein inhibitors, and phytoalexins. The role of phytoalexins in the resistance of plants to nematodes is the specific subject of this paper.

PERSPECTIVES

The phytoalexin theory attempts to describe a mechanism of host plant resistance to pathogens. The theory is neither new nor static. From its introduction in 1940 (20) it has been subtly modified and adjusted to accommodate new developments (3,7,11,15, 16,17,19). I interpret the theory to say that resistance in plants may be manifested in the ability of the plant to respond to infection by producing antibiotics that limit the spread or development of the invading organism.

Cruickshank (7), Bell (3), and Kuc (17) present convincing arguments in favor of the role of phytoalexins in disease resistance; however, it is not certain that phytoalexins constitute a mechanism of resistance in all plants. Antibiotic compounds synthesized in response to infections (phytoalexins) have been isolated from a number of diverse plants infected by various organisms. Structurally, phytoalexins range from

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relatively simple straight chain compounds to complex heterocyclic compounds. Grisebach and Ebel (10) chemically classified phytoalexins into isoflavanoids, sesquiterpenes, furanoterpenoids, polyacetylenes, dihydrophenanthrenes, and miscellaneous compounds. Bell (3) classified phytoalexins into stilbenes, coumarins, polyenes, flavanoids, isocoumarins, terpenoids and furanoacetylenes. Phytoalexins representative of various classes of compounds are shown (Fig. 1). The biosynthesis of phytoalexins have been reviewed (3,10); most are derived from acetate condensed with cinnamic acid (for flavanoid and stilbene type phytoalexins) or mevalonic acid (for terpenoid type phytoalexins) or fatty acid metabolism (for polyacetylene type phytoalexins).

Many organisms and a multitude of abiotic substance stimulate phytoalexin synthesis. Substances that stimulate synthesis are called "elicitors" (16). Abiotic elicitors such as heavy metal salts (4), ultraviolet radiation (6), and low temperatures (9) have been reported. Besides whole microorganisms, fungal cell walls and fungal products in culture filtrates (2,8) also elicit phytoalexin synthesis. These are considered biotic elicitors. The fact that the synthesis of many phytoalexins can be turned on by a number of different elicitors is often argued as demeaning their importance. In fact, nonspecific elicitation may be a virtue for general or broad range mechanisms of resistance. The mechanism by which elicitors stimulate phytoalexin synthesis is not known. Bell (3) concluded from the literature that all elicitors, at effective doses, adversely affect membrane permeability, and that phytoalexins are consistently associated with a necrogenic response in resistant hosts. From these observations Bell builds a hypothesis for a mechanism of action: he suggests that elicitors bind to cell walls in a manner similar to wall binding of phytotoxins. That is, elicitors attach to oligosaccharide binding lectins (such as galactan- and glucosamine-binding lectins) on host membranes or walls. The binding of the elicitor then impairs the permeability of the membrane (as does phytotoxin) which in turn leads to phytoalexin production and subsequent cell death. This hypothesis is attractive but whether it will withstand kinetic analysis remains to be determined.

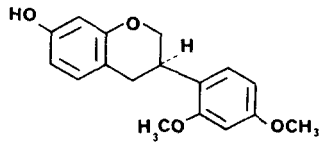
A number of host responses may occur upon elicitation. Differential accumulation of phytoalexins in susceptible and resistant hosts is often encountered and can form the basis of resistance. However, toxic concentrations of phytoalexins without the attendant resistant response may also be present. This absence of a resistant response in the presence of toxic concentrations of phytoalexin may be explained by the rate at which accumulation occurs (22) or by the sites at which phytoalexins accumulate. Additionally, oxidation of endogenous nontoxic derivatives to toxic phytoalexins during extraction and chemical work up may occur. If phytoalexin accumulation is not timely or if accumulation is not anatomically localized to contain the development or spread of the invading organism, toxic concentrations may accumulate but a susceptible host response will be observed. Both susceptible and resistant host cultivars will respond initially to elicitation, but the rate of accumulation is usually faster in resistant hosts. Additionally, phytoalexins may occasionally fail to be effective because some pathogens have a mechanism to metabolically detoxify phytoalexins (24). Although detoxification is not consistently associated with virulence (3), it can be considered a defense reaction of the pathogen to the host plant.

For phytoalexins to effect resistance, they must fulfill certain requirements of a time-space-effect (T-S-E) interrelationship (28). That is, they must be produced at the proper time (usually within 4–5 days after infection), localized in the proper cells or tissues (i.e., in close proximity to the pathogen), and have some type of antibiotic effect on the pathogen (induce death, inhibit development, or prevent spread). Histochemical or histological studies are often the best way of elucidating the T and S criteria of the relationship.

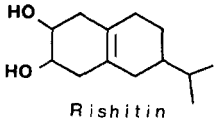
With this backround on phytoalexins, let me now turn to some specific examples of phytoalexins associated with plant resistance to nematodes.

SPECIFICS

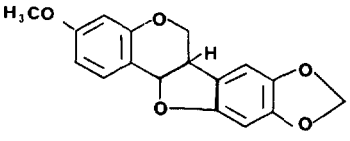
Considering the variety of pathogens



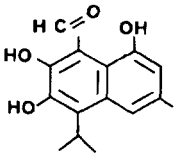
Sativan



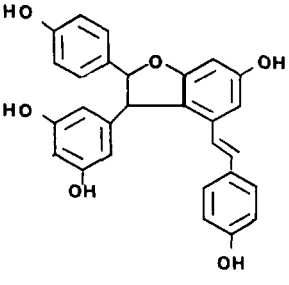
Rishitin



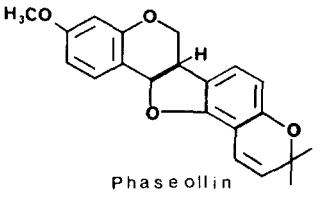
Pisatin



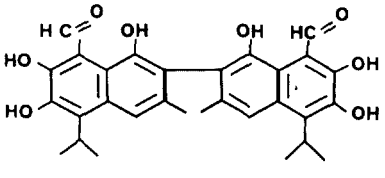
*Hemigossypol



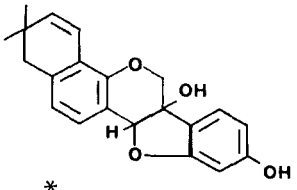
Viniferin



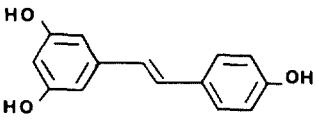
Phaseollin



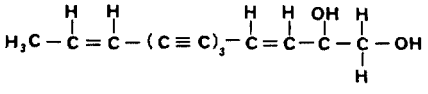
*Gossypol



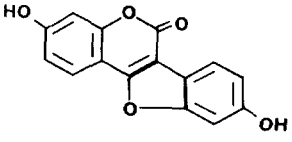
*Glyceollin



Resveratrol



Safynol



*Coumestrol

Fig. 1. Examples, from various classes of compounds, of phytoalexins, some of which (*) have been implicated in plant resistance to nematodes.

that elicit phytoalexin synthesis in plants, it is not surprising that nematodes can also induce such host responses. The first nematode-plant interaction study that specifically reported the induction of phytoalexin synthesis was that of Abawi et al. (1). They inoculated red kidney bean (*Phaseolus vulgaris*) with *Pratylenchus penetrans* and 5 days later extracted phaseolin (Fig. 1), a bean phytoalexin, from the nematode infected tissue. Since the beans were inoculated under sterile conditions with axenized nematodes and the noninoculated control plants did not produce detectable phaseolin, it is reasonable to infer that the infected plants produced phaseolin (59 μg/g root tissue) in response to infection by *P. penetrans*. Unfortunately, when *P. penetrans* larvae were exposed to 47 μg phaseolin/ml for 16 h they were not adversely affected. Thus, although phaseolin, a known phytoalexin, was synthesized by the host plant in response to infection by a nematode, it apparently did not constitute a resistance factor to *P. penetrans* in beans. Since phaseolin failed the effect aspect of T-S-E requirements, it is not necessary to consider the other two aspects. However, bean is known to produce several other phytoalexins, such as kievitone, and their antihelminthic activity should be investigated.

Phaseolus lunatus vs. Pratylenchus scribneri: The first specifically identified nematode-induced phytoalexin that appeared to be the active principle of a mechanism of resistance was identified in lima bean (*Phaseolus lunatus*). This plant produces phytoalexins in response to infection by various fungal and bacterial pathogens and is a poor host for *Pratylenchus scribneri;* hypersensitive lesions develope soon after infection by the nematodes. Rich et al. (21), therefore, hypothized that lima bean resistance to *P. scribneri* might be accounted for by nematode-induced synthesis of a phytoalexin.

To test their hypothesis, they isolated from nematode-infected lima bean roots four coumestans that accumulated concomitant with the hyper-sensitive (necrotic) response. One of the coumestans was identified as coumestrol (Fig. 1); a second was tentatively identified as psoralidin; the remaining two were not identified. By 1 day after inoculation, more than 40 μg coumestrol per g root tissue was extracted; by 4 days after inoculation, concentration exceeded 70 μg coumestrol/g tissue. Psoralidin accumulated more slowly but exceeded 40 μg/g tissue by 4 days after inoculation. During the same period, levels of coumestrol and psoralidin in healthy plants did not exceed 10 μg/g tissue. When lesions (necrotic tissue) were carefully dissected from adjacent nonnecrotic tissues, coumestrol and psoralidin levels in lesions were 89 and 39 μg/g, respectively. Neither coumestan exceeded 6 μg/g in adjacent nonnecrotic tissue. Thus, we see that phytoalexins accumulated at the sites of nematode attack.

To test the effect, coumestrol was bioassayed against the nematode species that induced its accumulation. Exposure of *P. scribneri* to coumestrol at 5 μg/ml significantly reduced motility compared to water-treated controls; the ED_{50} was 10–15 μg/ml. Exposure to 25 g coumestrol/ml for 48 h severely inhibited the motility of *P. scribneri,* but the effect was reversed when the nematode was removed from the phytoalexin. In similar bioassays, the phytoalexin had no adverse effect on *Meloidogyne javanica.*

In a complementary test, snap bean (*Phaseolus vulgaris*), which is a good host for *P. scribneri* and does not form a necrotic lesion in response to infection, was analyzed for phytoalexin production. Noninoculated root tissue accumulated coumestans in low levels comparable to noninoculated lima bean; this indicated an extant capacity to produce coumestans. However, additional accumulation in response to infection did not occur.

The fulfillment of the T-S-E requirements is difficult to assess for this plant-nematode interaction. The phytoalexin accumulates in substantial amounts within 4 days after inoculation and concomitant with the host hypersensitive response (time), it accumulates at the site of nematode attack (place), and it inhibits the motility of the nematode (effect). But, because of the vagrant nature of *P. scribneri,* the phytoalexin must quickly accumulate to sufficient levels to immobilize the nematode. It is conceivable that the nematode elicits phytoalexin

synthesis but that the phytoalexin accumulates to the observed levels after the nematode has migrated to nonelicited tissue. In which case it would be hard to imagine an effective mechanism of resistance.

Glycine max vs. Meloidogyne incognita. The most detailed and impressive study of a nematode-induced phytoalexin type mechanism of resistance is that reported by Kaplan et al. (13,14). These studies analyzed the responses of two soybean cultivars ('Centennial' and 'Pickett 71') to infection by two closely related nematodes (*M. incognita* and *M. javanica*). The host responses to these nematodes were as follows:

Host	*M. incognita*	*M. javanica*
Centennial	Resistant	Susceptible
Pickett 71	Susceptible	Susceptible

The phytoalexin hypothesis for this model requires, among other things, that phytoalexin accumulation occur with the host-nematode interaction that results in a resistant response but not with the susceptible responses.

Root extracts from the four host-nematode combinations were made at various times after inoculation and analyzed for the presence of glyceollin (Fig. 1), a soybean phytoalexin. Constitutive low concentrations (15 μg/g root) of glyceollin were detected in the healthy roots of both soybean cultivars. After inoculation, however, additional accumulation occurred only in Centennial roots inoculated with *M. incognita* (the resistant host-nematode interaction). By 3 days after inoculation nearly 40 μg glyceollin/g root tissue was detected, and by 7 days after inoculation glyceollin exceeded 70 μg/g.

To determine more precisely the sites of localization of glyceollin, the roots of 5-day-old infections were mechanically decorticated and the cortex and stele were analyzed separately. The stele of Centennial infected by *M. incognita* contained almost 130 μg glyceollin/g stele tissue; the concentration in the stele from the three susceptible interactions was about 50 μg/g stele tissue. Glyceollin in cortex was highest in the resistant host-nematode interaction, but it did not exceed 30 μg/g cortex; glyceollin concentrations in the cortex of the susceptible host-nematode interactions did not exceed 20 μg/g cortex. Thus, it appeared that the bulk of the glyceollin, especially that synthesized in the resistant host in response to infection, accumulated in the stele.

The efficacy of glyceollin on inhibiting the motility of *M. incognita* and *M. javanica* larvae was determined. Exposure to 60 g/ml for 24 h had no apparent effect on *M. javanica*. However, about 70% of the *M. incognita* larvae were adversely affected by 15 μg/ml, and the ED_{50} was determined to be 11 μg/ml. Because affected nematodes regained motility upon removal of glyceollin from the test medium, Kaplan et al. (13) concluded glyceollin was nematistatic, but not nematicidal, to *M. incognita*.

In additional studies, Kaplan et al. (14) partially defined the mechanism by which glyceollin might function as a nematistatic phytoalexin. The rate of oxygen consumption by *M. incognita* larvae was reduced 50% by 48 μg glyceollin/ml; at the ED_{50} concentration for inhibition of *M. incognita* motility (11 μg/ml), oxygen consumption was reduced about 13%. Glyceollin did not inhibit nematode choline esterase activity. In soybean mitochondria the electron transport system was inhibited by 1 μg glyceollin/ml but mitochondrial membranes were not adversely affected and oxidative phosphorylation was not uncoupled. These effects on soybean mitochondria were assumed to apply to nematode mitochondria (based on commonality of mitochondria from all organisms), and the data were interpreted to indicate that the primary action of glyceollin is the inhibition of the electron transport system. This interpretation does not explain why glyceollin does not affect *M. javanica* larvae; the commonality between *M. incognita* and *M. javanica* mitochondria must be closer than between *M. incognita* and soybean mitochondria. Kaplan et al. (14) suggested the possibility of differential uptake of glyceollin by the nematodes; one might also suggest differential degradation of glyceollin by the nematodes.

This system has a number of attributes in addition to fulfilling the T-S-E requirements. The fact that the nematode becomes sedentary soon after establishing a feeding

site increases the probability that it remains associated with the cells that accumulate the phytoalexin. It is also interesting that infection by *M. javanica* did not result in phytoalexin accumulation in the *M. incognita* resistant plants. Were phytoalexins not elicited, or were they metabolized by *M. javanica* as they were formed?

Gossypium hirsutum vs. M. incognita: A phytoalexin-type mechanism of resistance of cotton to the root-knot nematode has been reported by Veech and McClure (29) and Veech (26,27). The mechanism is predicated on the accumulation, in response to infection, of nematicidal concentrations of terpenoid aldehydes.

Cotton constitutively synthesizes gossypol and related terpenoids. The rate and amount of constitutive terpenoid accumulation is a function of the cultivar and has little or no relationship to the level of host resistance or susceptibility. Terpenoid aldehydes accumulate in the epidermis in all but 3–4 cm of the tips of noninfected cotton roots. No accumulation occurs in the stele, and only occasional cortical cells accumulate terpenoid aldehydes. Thus, constitutive accumulation does not occur in the portion of the root where the nematode penetrates, or in root cells near sedentary nematode feeding sites. Hence, it is of little consequence that concentrations of constitutive terpenoid aldehydes are not correlated to susceptibility or resistance, because the preinfectional terpenoid aldehydes are not anatomically localized to be effective against nematodes.

What is of consequence, however, is the accumulation of terpenoid aldehydes in host plants in response to infection. To demonstrate this putative mechanism of resistance, five cotton cultivars with different levels of resistance to *M. incognita* were selected for study; in order of decreasing resistance, the cultivars were 'Auburn 623,' 'N6072,' 'Clevewilt,' 'Deltapine 16,' and 'M-8.' The concentrations of five terpenoid aldehydes—hemigossypol, methoxyhemigossypol, gossypol, methoxygossypol, and dimethoxygossypol—were determined in the roots of each cultivar 5 days after inoculation with *M. incognita;* the concentrations in comparably aged noninoculated roots were also determined. The concentrations of each terpenoid aldehyde increased in response to infection, compared to the noninoculated controls, in the roots of the three most resistant cultivars, but it decreased in the two least resistant cultivars. Coefficients of correlation between infection-induced concentrations of methoxygossypol and the level of host resistance based on root-knot index, egg masses/g root tissue, and eggs/g root tissue, were .91, .96, and .97 ($P = .01$), respectively. This indicated that methoxygossypol accumulation in response to infection was directly proportional to the level of host resistance.

Because constitutive terpenoid aldehydes accumulated in roots at sites not likely to be effective against the sedentary nematode, new sites of accumulation had to be formed in response to infection. A histochemical study of infected roots demonstrated that within 4 days after inoculation, infection-induced terpenoid aldehydes accumulated in the resistant host in histochemically detectable amounts in the endodermis and stele at, or very near, the feeding site of the nematodes. Infection-induced terpenoid aldehyde accumulation was occasionally observed in the susceptible host, but it did not accumulate as rapidly as in the resistant host, nor did it encompass as many cells.

The effect of terpenoid aldehydes on the motility of *M. incognita* was determined by exposing larvae to a mixture of terpenoid aldehydes at various concentrations (based on gossypol equivalents) for various times. Exposure to 10 ppm for 24 h had little effect on larvae motility. Exposure to 50 ppm immotilized about 70% of the larvae, but the effect was reversed by a 24-h recovery period in the absence of terpenoid aldehydes. Exposure to 125 ppm for 5 h immotilized all the larvae, but 88% regained motility with a 24-h recovery period; only 17% regained motility after a 24-h exposure.

Considering the rate at which infection-induced terpenoid aldehydes accumulate in response to infection, and the highly localized nature of that accumulation, antibiotic levels of terpenoid aldehydes probably accumulate in the resistant plant concommitant with the nematode becoming sedentary.

CONCLUSIONS

The role of phytoalexins in the resistance of plants to nematodes has not been extensively explored, but the findings to date indicate that this is a fertile area of research. Fortunately, phytopathologists have already established much on which nematologists can capitalize. Nematologists interested in phytoalexin production need not proceed stochastically at isolating and identifying such antibiotic compounds. Although not absolute, a relationship seems to exist between the plant taxonomic family and the type of phytoalexins produced (11). The Leguminosae generally produce iso-flavanoid-type phytoalexins, Compositae usually produce polyacetylene phytoalexins. Malvaceae and Solanaceae generally produce terpenoid type phytoalexins. These generalities, together with the established literature on specific phytoalexins, constitute excellent starting points in searching for phytoalexins synthesized in response to nematode infection.

Other challenges in the area of phytoalexins and plant resistance to nematodes await our attention. Van Staden and Dimella (25) correlated constitutive cytokinin levels in the root to levels of susceptibility to *M. javanica,* and Bird and Loveys (5) reported increased cytokinin levels associated with nematode infection. Since Sziraki et al. (23) reported that exogenously applied cytokinin inhibits necrosis caused by mercuric chloride, and since mercuric chloride is a good elicitor of phytoalexins, one may speculate that cytokinins are responsible for the reduced or inhibited accumulation of phytoalexins in nematode susceptible plants. Or, if Bell's (3) hypothesis is correct and phytoalexin elicitors do bind to walls or membranes, we may speculate that the Concanavalin A binding sites on the head of *M. incognita* (18) represent a mechanism whereby some nematodes inhibit elicitation by binding the elicitors.

A slight delay in the elicitation of phytoalexins by sedentary endoparasites would seem to be ideal. Too rapid accumulation of phytoalexins could be energy inefficient, because the transitory nematode might detect sub-effective levels of the phytoalexin and migrate to nonelicited cells. New sites of infection-induced synthesis would then have to be established at additional energy expense to the plant. Ideally, phytoalexin accumulation should begin about the time the nematode becomes sedentary and progress fast enough to adversely effect its development.

I am reasonably convinced, and Kaplan and Keen (12) seem to concur, that phytoalexins can serve as effective mechanisms of resistance of plants to nematodes, expecially sedentary nematodes. We have only begun to catalog the phytoalexins that are synthesized in response to nematode infection and to understand how these compounds function in resistance. There is ample evidence to indicate that phytoalexin synthesis is ammenable to qualitative and quantitative genetic manipulation. Thus, the exploitation of phytoalexins can develop into a powerful tool for protecting plants from nematodes and thereby increase agricultural productivity.

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