Interpretation of Mechanism for Nematode Resistance in Sugarbeet

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INTRODUCTION

The sugarbeet nematode, Heterodera schachtii Schm., is the most important nematode of sugarbeets, Beta vulgaris L., and is an important pest on some other crops. This nematode is responsible for major sugarbeet yield reductions in many parts of the world. Losses due to this pest range from partial decreases in production to total failure of a crop. In the United States the annual loss in sugarbeet yields caused by nematodes is estimated to be 10 percent (1, 4). When the viable H. schachtii cyst population exceeds 10 cysts per 750 g (one pint) of soil in Colorado (1), or 100 eggs per 100 g of soil in the Imperial Valley of California (2), economic losses can occur. A cyst usually contains a few to over 600 eggs (18, 22).

Resistance to H. schachtii is lacking in the primary gene pool of sugarbeet (3) and is one of the most sought traits to be incorporated in sugarbeet. By hybridization of B. vulgaris x B. procumbens Chr. Sm. and the succeeding selections and crosses, several diploid sugarbeet lines with nematode resistance have been developed (19, 25). Resistance in these nematode-resistant genotypes is not due to failure of nematode larvae to enter sugarbeet roots but is due to failure of the large majority of larvae to complete their life cycle in the roots (21, 26).

OBSERVATIONS AND DISCUSSION

Resistance from Wild Beta Species

Resistance to H. schachtii in sugarbeet was introduced
from *B. procumbens*, a remote species belonging to the section patellares of the *Beta* genus. Hjìner (6) considered that all the three species of wild beet in the section Patellares were immune to *H. schachtii*. Based upon our results, however, these wild *Beta* species should only be rated as highly resistant to *H. schachtii*. Among them, *B. procumbens* has expressed the best resistance.

A total of 301 seedlings from 13 different seed sources in *B. procumbens* were germinated and evaluated for nematode resistance utilizing greenhouse procedures that were used in sugarbeets (25). In the first test, no cysts were observed on 98.3% of the seedlings, but the remaining five seedlings (1.7%) had white cysts attached on the root system (Table 1). Two of the five seedlings were classified susceptible to *H. schachtii* because they had more than 10 cysts. None or less than 10 cysts per plant was our arbitrary criterion for resistance. These two plants contained less than seven cysts in the second test but had no cysts in the third test. *Heterodera schachtii* cysts may occasionally develop in sugarbeets with the resistant genotype.

**Table 1.** - Development of *Heterodera schachtii* cysts in *Beta procumbens*.

<table>
<thead>
<tr>
<th>No. of plants</th>
<th>1st Test</th>
<th>2nd Test</th>
<th>3rd Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>296</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>1-5</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>10</td>
<td>3-7</td>
<td>0</td>
</tr>
</tbody>
</table>

*Thirteen strains of *B. procumbens* were evaluated.

Resistant vs. Susceptible Genotype Reactions

*Heterodera schachtii* is most pathogenic on sugarbeets grown at soil temperatures of 21-27°C (2), and sugarbeets are particularly sensitive to *H. schachtii* attack during the seedling stage (5, 16). This sensitivity seems to apply to both the resistant and susceptible sugarbeets. Preliminary observations on a late June seeding at Salinas by myself and McFarlane et al. (11) showed that resistant sugarbeet selections also were partially stunted and their
yields reduced when they were grown in heavily nematode infested soil. This suggests that some resistant sugarbeet lines have as severe nematode damage as non-resistant lines when grown under a heavy infestation. This damage appeared to be expressed more readily on the resistant inbred sugarbeets, e.g., lines 3552 and 3584, than on the open-pollinated lines. Some seedlings from these inbred lines failed to emerge or emergence was delayed, and several stunted seedlings died within a few weeks after emergence.

Conceivably the hypersensitive host reaction induced by nematodes in radicles of the young resistant seedlings caused rapid necrosis of root cells (26), which severely affected normal growth of the sugarbeets. In contrast, in susceptible sugarbeets the infected cells usually do not become necrotic until after nematodes complete their life cycle (9,20). Hypersensitive reaction, which is characterized by necrosis of infected tissue together with inactivation of the attacking agent, is considered a defensive mechanism of the plant against potential parasites (12). In general, the severity to the host caused by hypersensitivity would depend upon the number of larvae invading the root, and the length of time larvae remain alive and feed. The few nematodes that finally develop to maturity in resistant plants sometimes form rather small cysts. They probably also contain fewer viable eggs.

Histopathology of Infected Sugarbeets

Histopathology of sugarbeet cultivars in response to the infection of H. schachtii has been reported by several researchers (9, 20, 23, 26). In suitable environmental conditions, the infective second-stage larvae hatch and emerge from the cysts then migrate to and enter roots of susceptible and resistant sugarbeets (Figure 1). Nematodes usually damage plants by altering host physiology which results in the formation of multinucleate syncytia (Figure 2).

Nematode damage is often increased by secondary pathogens. Sugarbeet seedlings are frequently damaged more
seriously by combined infections with *H. schachtii* and associated fungi or bacteria than by either pathogen alone (15, 24). The wounds from larval entrance may facilitate

![Figure 1. A nematode larva is migrating or feeding in the root cortex tissue of a resistant sugarbeet. (X ca. 390).](image)

![Figure 2. A multinucleate syncytium formed in the susceptible sugarbeet, in cross section. (X ca. 245). Note the partial dissolution of cell walls results in the coalescence of cytoplasm, which forms a continuous cytoplasmic unit.](image)

fungal penetration and establishment, and syncytia induced by nematode feeding are an even more suitable substrate for the fungi than are normal cells (15). The interacting effect of *H. schachtii* and fungi may be more than additive, i.e., synergistic, which causes increased reduction in sugarbeet yields (17).

In susceptible sugarbeets, cessation of feeding by the matured nematode is followed by deterioration and collapse of syncytia. At this time, the milky-white, lemon-shaped cysts increase in size and break out of root tissues but remain clinging to the beet roots by their heads. They
are visible with the unaided eyes (Figure 3). It should be noted that although the majority of the larvae hatch from eggs when conditions are favorable, not all hatch the same year (18,22). The number of viable eggs held in brown cysts decreases gradually over a period of years, particularly in the absence of a host-plant.

Figure 3. The white nematode cysts, adult females of *Heterodera schachtii*, attached on roots of a susceptible sugar beet visible after the foil cylinder is removed. (ca. 3/4 size).

Figure 4. A deteriorated nematode (H) with cuticular infolding that contains no recognizable organs in a transverse section of a resistant sugar beet root. (X ca. 245). Note the necrotized syncytium (Nec) occupies only a small sector of the root.

Initial stages of syncytial formation in the resistant and susceptible plants appears to be similar. How-
ever, the hypersensitive reaction of the resistant host causes the formation of cytoplasmic granules in the cells fed upon by nematodes within four days, and necrotic lesions within 10 days after larval penetration (26). Thereafter, necrosis becomes progressively more severe, resulting in the collapse of syncytia and the death of nematodes. Syncytia formed in the resistant sugarbeets are generally smaller than those in susceptible plants. If there are not massive multiple infections, hence syncytial complexes, those local necroses (Figure 4) in the resistant plants involve only limited sectors of the root which generally can be repaired or compensated and cause little effect on the host-plant physiology.

**Classification of Resistance to *Heterodera schachtii***

Host-plant responses to the parasite infection have a wide range of variations. Classification of resistant phenomena may be expressed by the fate of parasites feeding on the host-plant or by degree of host-plant damage resulting from infection. Presently the criteria of resistance classification in the field of nematology have yet to be established. The standards used in entomological literature are, therefore, tentatively adopted here to describe the phenomenon of resistance to *H. schachtii* in sugarbeet.

Painter (14) classifies intensities of host resistance into five categories, viz., immunity, high resistance, low resistance, susceptibility, and high susceptibility. An immune plant is a non-host for a given parasite; the parasite will never consume or injure the plant under any known conditions. A highly susceptible plant, on the other hand suffers more than average damage from a specific parasite. Based on these standards the resistance to *H. schachtii* in sugarbeet can only be classified as high resistance, not immunity.

Classification of host-plant resistance based on the causative factor is a better criterion for both basic and applied research. The three fundamental mechanisms of host-plant resistance (14) that have gained wide
acceptance and popularity because of their simplicity and accuracy in describing parasite and plant responses are:

1. Antixenosis (10), or nonpreference. The plant is an undesirable host; it is avoided by the parasite for food, oviposition, or shelter.

2. Antibiosis (13). The plant used for food exerts adverse effects on the parasite such as reduced fecundity, decreased size, subnormal length of life, or increased mortality.

3. Tolerance. An adaptive mechanism of a plant to withstand infection and to support parasite populations that would otherwise severely damage susceptible plants.

These three categories of resistance are nonexclusive. They may interact, complement, or compensate each other in intensifying resistance expression (7). Among these mechanisms, antibiosis and antixenosis exert selection pressure on the parasite, but tolerance does not.

The high mortality of nematode larvae, subnormal length of life, smaller cyst size, and reduced fecundity suggest that the resistance mechanism involved here is antibiosis. In other words, the host-plant resistance found in B. procumbens that was introduced to sugarbeet is resistance to survival and reproduction of H. schachtii. This nematode resistance is not antixenosis because nematodes do enter and feed on the resistant plant and use it for food. Nor is it tolerance, since the invading nematodes encounter severe adverse effects exerted by the resistant host.

Development and Utilization Potential of Resistant Sugarbeets

Because of the hypersensitive host reaction in resistant sugarbeets to nematode parasitism, which is usually expressed to a greater degree at the seedling stage, the growth and hence the yields of some greenhouse-selected resistant sugarbeet lines may be affected when they are grown in a heavily infested field. A similar phenomenon, a nematode-resistant cultivar that suffers considerable damage due to the high larval invasion, also has been observed in other crops, e.g., potatoes (8). Nonetheless, a
wide range of variation seems to exist among resistant sugarbeet lines in response to nematode infection under field conditions. Selection and development of favorable true-breeding nematode-resistant genotypes should be achievable in future breeding programs, especially when additional resistant lines from different accessions are recovered.

It is expected that resistant sugarbeet plants would be suppressive to nematode larvae and ultimately reduce the population density of *H. schachtii* in the soil, at the same time preventing nematode buildup from lower levels. However, until a tolerable "threshold level" of the nematode population is reached in heavily infested fields, integrated means of control, such as crop rotation, chemicals, or drilling seed when the ground temperature is low may be needed in order to produce high yields from resistant sugarbeet cultivars.

**SUMMARY**

The resistance to *Heterodera schachtii* Schm. in sugarbeet *Beta vulgaris* L., that was introgressed from *B. pro­cumbens* Chr. Sm. conditions high resistance, not immunity. A hypersensitive reaction of the resistant sugarbeet cells not only causes the death of nematode larvae but also hampers certain normal vascular functions of the host-plants. Germinating seedlings of the resistant sugarbeets are more sensitive to the multiple nematode infections than the larger plants. The mechanism of resistance to *H. schachtii* in sugarbeet was determined to be antibiosis. Antibiosis is expressed as resistance to the survival and reproduction of *H. schachtii*.

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**LITERATURE CITED**


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