A Model Plant Pathogen from the Kingdom Animalia: *Heterodera glycines*, the Soybean Cyst Nematode

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Key Words

Glycine max, *Heterodera glycines*, nematode resistance, SCN, soybean, soybean cyst nematode

Abstract

The soybean cyst nematode, *Heterodera glycines*, adversely affects the production of soybean, *Glycine max*, in many areas of the world, particularly in the United States, where it is the most economically important soybean pathogen. Despite the availability of hundreds of *H. glycines*–resistant soybean cultivars, the nematode continues to be a major limiting factor in soybean production. The use of nonhost rotation and resistance are the primary means of reducing losses caused by the nematode, but each of these options has disadvantages. As a subject for study of nematode parasitism and virulence, *H. glycines* provides a useful model despite its obligately parasitic nature. Its obligately sexual reproduction and ready adaptation to resistant cultivars, formerly referred to as "race shift," presents an excellent opportunity for the study of virulence in nematodes. Recent advances in *H. glycines* genomics have helped identify putative nematode parasitism genes, which, in turn, will aid in the understanding of nematode pathogenicity and virulence and may provide new targets for engineering nematode resistance.

Juvenile: any one of four immature stages of a plant-parasitic nematode

J: juvenile

Cyst: the bodily remains of a dead female cyst nematode

CONTEXT

Heterodera glycines Ichinohe, the soybean cyst nematode, is one of the most economically important pathogens of soybean (*Glycine max* [L.] Merr.) worldwide (96). The nematode has shown itself able to adapt to most conditions under which soybean is produced, and its unique biology and ability to survive long periods under adverse conditions require that it remain one of the principal targets of soybean pest management strategies in many soybean production areas. The preponderance of research on *H. glycines* has been done in North America (65), so this paper will naturally reflect our knowledge of the nematode and its interaction with soybean as it is here; however, because the interaction is somewhat plastic, it is never surprising to find "real world" differences in the interaction among geographic areas. A recent monograph on the biology and management of *H. glycines* is a good source of further information on many of the topics covered in this review (70). Another recent monograph includes similar information on *H. glycines* and a number of other Heteroderidae (72).

LIFE HISTORY

H. glycines is an obligately endoparasitic pathogen. Like those of other nematodes, its life cycle comprises four juvenile stages and the adult. Development of the animal from one life stage to the next is punctuated by ecdysis (molting), the first of which occurs within the egg. The second-stage juvenile (J2) is the one that emerges from the egg and is the infective stage. Third- and fourth-stage (J3 and J4) juveniles develop within plant roots. Sexual dimorphism, in which males and females of the same species exhibit vastly different morphologies, becomes evident in the J3. The adult female, being too large to be contained within the root, is visible to the naked eye on the root surface as a pearly white spheroid (**Figure 1**). The brown cyst, to which the common name of the nematode refers, is the bodily remains of the dead female that contains viable eggs. Males, having regained a vermiform shape, exit the roots. Reproduction is sexual in this species. Under optimum conditions, the entire life cycle can take as little as 22 days (49) (**Figure 2**).

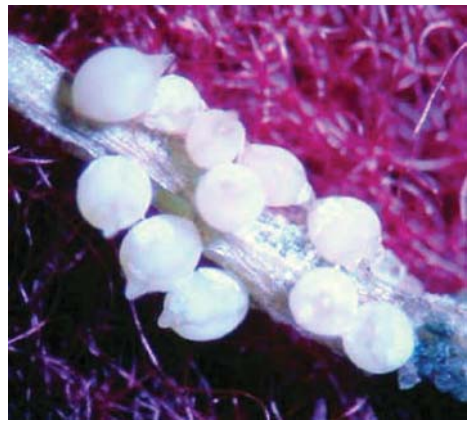

Figure 1

Heterodera glycines females on a soybean root. Photo courtesy of T. A. Jackson, University of Nebraska. *H. glycines* females are typically between 500 and 900 μm in length, depending on nutritional status or quality of the food source.

Eggs and Hatching

The egg is not only the reproductive unit but also the survival stage of *H. glycines*. The number of eggs per unit volume in a field can be regarded as the inoculum potential of the soil. Each *H. glycines* female is capable of producing up to 600 eggs (76). Some of the eggs, up to perhaps 200 (J.H. Yen & T.L. Niblack, unpublished data), are deposited into a gelatinous matrix produced by the female (**Figure 3**). The remainder is retained within the body of the female. Eggs in the gelatinous matrix, which is primarily carbohydrate, may be partially protected from predation by the presence of antimicrobial compounds such as chitinase and polyphenoloxidase (56). The protection need not be lengthy, however, because eggs deposited into the gelatinous matrix are more likely to hatch during the current season than are eggs retained within the cyst

(39, 79). Eggs within the cyst have the additional protection of the cyst wall. Individual eggs from the population contained within a single cyst may be observed to contain any stage, from one-celled to fully developed J2, depending on the age of the cyst and the conditions under which it was produced (83). The egg shell has not been well characterized (9), but is pliable and more or less permeable to certain compounds depending on the stage of the animal contained within (61).

The mechanisms involved in egg hatching in *H. glycines* are still a matter for study. Rupture of the shell to release the J2 may be a result of enzymatic activity, physical processes, or a combination of the two, if it is similar to the process employed by the potato cyst nematode, *Globodera rostochiensis* (18, 60). Hatching is the result of a complex interplay among external signals and the internal readiness of the J2 to emerge (73). Readiness to hatch, or rather the proportion of a population of eggs that will hatch readily, is probably under some degree of genetic control. Confirming this idea, we were able to select from an inbred *H. glycines* population subpopulations that were "slow-hatchers" and "fast-hatchers," but as yet the differences between these (other than the rate at which they hatch) have not been detailed thoroughly (77; A. M. Skantar & B. B. Burgwyn, unpublished data). Yen et al. (99), inspired by the work of Zheng & Ferris (101) on *H. schachtii*, the sugar beet cyst nematode, and others suggested that eggs within *H. glycines* cysts in the field exhibited three different types of dormancy: temperature-mediated, host-mediated, and time-mediated. This interplay of internal and external hatching controls ensures that infective J2 are present at any appropriate time to exploit whatever infection opportunities may exist, and that others are safely dormant until a host is present or sufficient time has passed, or both. Infective J2 are vulnerable to desiccation, predation, parasitism, and starvation; thus, an added survival benefit of the hatching behavior of *H. glycines* is that 100% hatch will not occur even when edaphic factors are favorable and a susceptible host is present, ensuring that a reserve of viable J2 will be present even if unfavorable conditions occur unexpectedly during a period of otherwise favorable conditions.

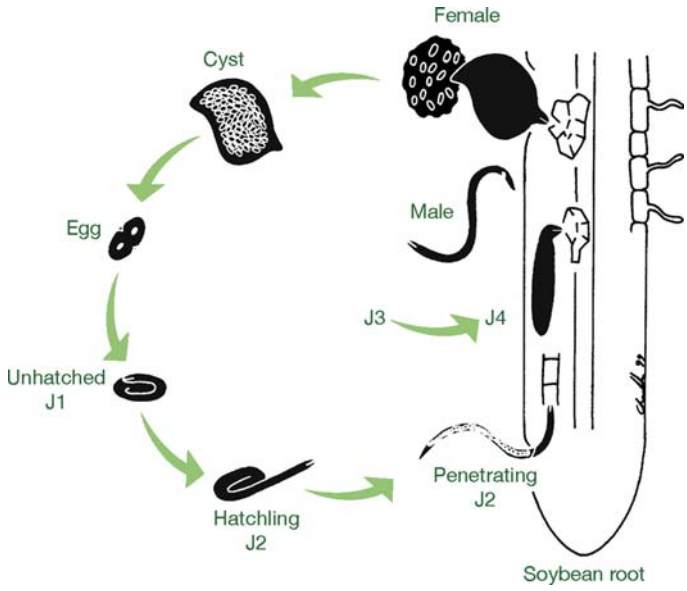

Figure 2

Life cycle of *Heterodera glycines* (all stages not drawn to the same scale) (D.V. Charlson, unpublished). A developed first-stage juvenile (J1) eventually forms in the egg. The J1 molts once within the egg shell, becoming a second-stage juvenile (J2) that hatches from the egg. The J2 penetrates the root and, in a host, develops through the third and fourth juvenile stages (J3 and J4, respectively). Vermiform, adult males fertilize lemon-shaped, adult females and the adult females produce eggs externally, in an egg mass, then fill up internally with eggs.

Figure 3

Gelatinous matrix (*translucent material*) produced by a virgin *Heterodera glycines* female (*opaque white sphere*).

Inagaki & Tsutsumi (38) showed that eggs within cysts of *H. glycines* could remain viable in a nonhatched condition for as many as 11 years. Anecdotal evidence suggests that *H. glycines* can survive in the field in numbers high enough to reduce yields of susceptible soybean for more than a decade (R.D. Riggs, personal communication).

Horizontal gene transfer: the hypothesis that genes for parasitism or virulence were transferred to nematodes from bacteria

Syncytium: the system of highly modified host cells from which the cyst nematode feeds within the plant root

Infection and Juvenile Development

Infectivity studies have shown that *H. glycines* J2 will enter the roots of resistant and susceptible hosts equally well, and will even enter the roots of nonhosts such as potato if introduced to those roots in a viable state (personal observation). Root penetration and migration is associated with production of cellulases and other enzymes (13), several of which are similar to those of microbial origin and not known to be produced by other animals. These interesting observations have led to speculation about the origin of plant parasitism through horizontal gene transfer (14).

Beyond penetration and migration, further development of the worm requires the successful induction of a specialized feeding site, or syncytium, within or near the vascular tissue depending on soil moisture conditions (41). Development of the syncytium is a host response to one or more elicitors produced by the nematodes, probably in the esophageal glands; identifying these elicitors is one of the most exciting areas of research on plant-parasitic nematodes today. It appears that nonhost cells simply do not recognize signals from the nematode, and consequently, formation of the syncytium is not initiated. Certain resistant hosts have a hypersensitive-like response that results in the death of the prospective syncytial cell (23, 43, 44). Other resistant hosts interfere with the development or function of the syncytium at later stages. Syncytial development is the crux of the *H. glycines*–host interaction, but there are numerous other morphological and physiological responses to infection, reviewed recently by Noel (57).

Following syncytium induction, the J2 swells to what is called a sausage stage and, through loss of most somatic musculature, loses its ability to move. Nematode feeding was elegantly described by von Mende et al. (89). Feeding continues for a minimum of 3 days after infection, at which time a molt event reveals the J3 stage. With some difficulty, male and female juveniles can be distinguished at this stage. Barring interference, third and fourth molts may follow in as little as 6 and 8 days, respectively.

Males and Females

Both male and female *H. glycines* are more or less in contact with the soil: males, because they exit the roots, having regained a vermiform shape and the necessary musculature for motility, and females, because their bodies are too swollen to be contained within the root. Some references mention the females' hindparts "breaking through" the root, suggesting that this is the result of physical forces, but observation of females developing shows apparent dissolution of the root cells surrounding the body (**Figure 4**) (V. H. Dropkin, unpublished data), suggesting that cellulases or other enzymes may be involved in the process. The presence of such enzymes in the gelatinous matrix (56) strengthens this hypothesis.

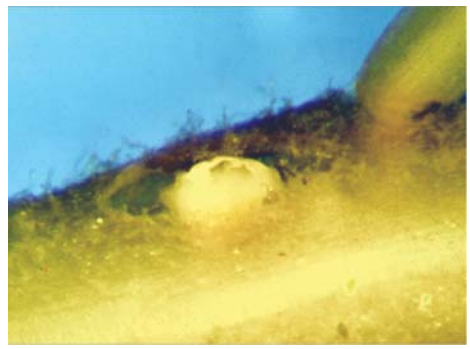

Figure 4

Maturing *Heterodera glycines* female erupting from soybean root surface. Photo courtesy of V.H. Dropkin, University of Missouri.

The role of males, beyond that of fertilization of the females, has been studied very little. However, the nature of males and females in *H. glycines* is very handy for biochemical and genetic studies. Virgin females can be produced in hydroponic culture (12, 21), because the males emerging from the roots settle to the bottom of the hydroponic vessel. The females will produce uncontaminated gelatinous matrices for analysis, or the nematodes can be carefully dissected from the roots and placed alive in "mating chambers." Males, harvested from the hydroponic vessels, can be introduced to the dissected females, and genetically controlled crosses can be completed thereby.

If infected roots are undisturbed, under optimum conditions, egg production will begin about 22 days after root infection (49). Maximum egg production is reached in about a week as the female becomes visibly yellowed. Death of the female is accompanied by darkening of the body wall, at which time the remains of the female body is properly referred to as a cyst.

POPULATION DYNAMICS AND HOST RELATIONS

As a soilborne pathogen, *H. glycines* is subject to the direct and indirect effects by all manner of biotic and abiotic influences. Isolating these for separate study is fascinating, but often provides little insight into what goes on in an individual field situation. For example, early studies suggested that pH had little influence on *H. glycines*–soybean interaction, leading many to dismiss pH as a factor to consider in management strategies. Later, Tylka et al. (84) showed evidence for a strong positive correlation between pH and *H. glycines* population densities (**Figure 5**) that must be considered in management of infested soybean fields with high pH soils. Many such examples, taken together, make a compelling argument that there is very little one can say unequivocally about *H. glycines* population dynamics. Populations are subject to numerous

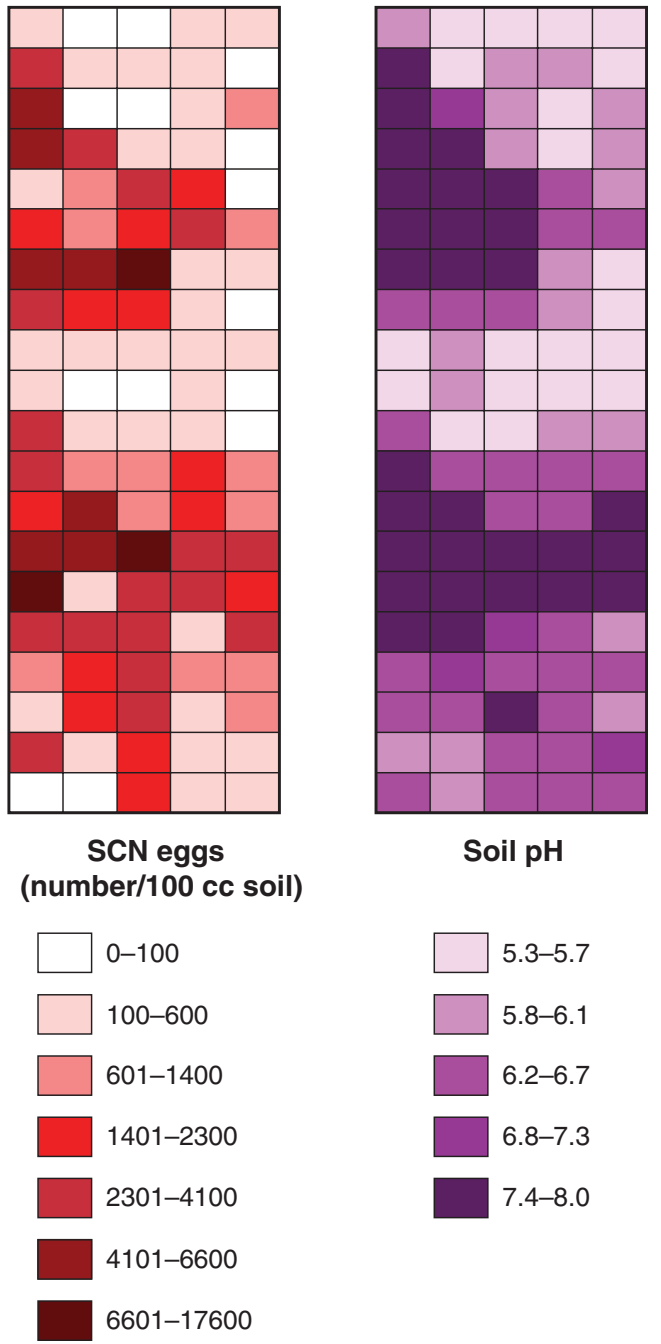

Figure 5

Heterodera glycines [soybean cyst nematodes (SCN)] population densities (*left*) and soil pH (*right*) in 100 0.2-hectare (0.5-acre) cells in a field in central Iowa (G.L. Tylka & A. P. Mallarino, unpublished).

Action threshold: the soil population density of nematodes at which some action should be taken to limit both yield losses and population density increases

QTL: quantitative trait locus (loci)

Virulence: for nematodes, this refers to the ability to develop on a resistant host plant, and does not include a concept of amount of disease induced

Race: designation of the virulence profile

PI: plant introduction

density-dependent and -independent factors that may lengthen generation time, reduce fecundity, influence the amount of host damage, and so forth (4). Nonetheless, some points can be made that will help elucidate the management strategies that have been proposed and put into practice for reducing yield losses associated with *H. glycines* infestations.

In the field, temperature, moisture, and host status have the greatest impact on nematode population density and development overall. Temperatures and moisture levels that are suitable for soybean growth are also suitable for nematode growth (45). References that state that the nematode can complete three to six generations per year fail to emphasize the adverb "theoretically." Even if true, generations following the first one or two may contribute little to final, or harvest, population densities. This can be seen from inspecting the numbers reported from field studies (4). Lawn & Noel (50) suggested that most of the population increase occurs during the first generation. A meta-analysis of data from 31 environments in Missouri and Illinois, studied for the effects of nematicides and cultural practices on soybean yield in *H. glycines*–infested fields, showed that *H. glycines* population growth curves leveled off after the first 60 days, near the first soybean reproductive growth stage (T. L. Niblack, unpublished data).

Numerous researchers have demonstrated that the principal component in yield loss due to *H. glycines* infection is the initial nematode population density (69). However, because of the complexity of the interactions among nematode, host, and environment, identification of damage thresholds (population densities at which yield loss can be expected) has lost favor in recent years because they are not consistent across environments, and because there are few, if any, therapeutic tactics that can be used within a growing season. A management strategy for *H. glycines* should be developed and implemented as soon as the nematode's presence in a field is confirmed; in other words, the action threshold is equal to the population density at the time the nematode is first found. Subsequently, sampling for population densities will give a good relative indication of the efficacy of the management strategy (4).

GENETIC DIVERSITY

The area of interest in the genetic diversity within and between populations of *H. glycines* with the greatest implications for management is, of course, its ability to adapt to resistant soybean cultivars. The first known instances of the nematode's adaptation to a resistant cultivar were reported within a decade of its first being found in the United States in 1954, and shortly after, work by soybean breeders and nematologists began to produce the first resistant cultivars (8, 67). Investigations into the nature of resistance to *H. glycines* have been ongoing ever since, and although a number of major and minor quantitative trait loci (QTL) associated with resistance have been mapped, to date none has been cloned and characterized. Complementary investigations into the nature of virulence in *H. glycines* have been mostly lacking until recently. A review of these investigations is included in the section entitled, "Molecular nematode-host interactions."

Races

Much of what is currently known about *H. glycines* virulence, or the ability to reproduce on resistant soybean lines, is based on a description of "races" published in 1970 (32). To that point in time, there were three known sources of resistance, a plant introduction (PI) named 'Peking,' PI 88788, and PI 90763. Until recently, several PI were called 'Peking,' and it is now thought that the line used to describe the race scheme was what is now numbered 548402 (R.L. Nelson, Curator of the USDA-ARS Soybean Germplasm Collection, personal communication). Believing that the *H. glycines*–soybean interaction was probably gene-for-gene, Golden et al. (32) proposed the race-determination system that was used

Table 1 Infraspecific designations[a] of the soybean cyst nematode according to Golden et al.[b]

Race	Pickett	Peking	PI88788	PI90763
1	No	No	Yes	No
2	Yes	Yes	Yes	No
3	No	No	No	No
4	Yes	Yes	Yes	Yes

[a]The infraspecific designation, "race," is determined by the pattern of "Yes" and "No" ratings for each race. A "Yes" rating is given if the number of females produced by an *H. glycines* population on each soybean differential is equal to or greater than 10% of the number produced on the standard susceptible cultivar Lee. If the number of females is less than 10%, a "No" rating is given.
[b]Reference (32).

for the next three decades, including the three PI and one resistant cultivar (**Table 1**).

To identify the race of a population of *H. glycines*, a subsample of the population was introduced to each of the four soybean differentials and the susceptible check, 'Lee' soybean. After an unspecified period of time, the females and (or) cysts produced were extracted from each plant and counted. A percentage index was calculated from the number of females for each differential (N_r) and the susceptible check (N_s) as follows: $(N_r/N_s) \times 100$. The *H. glycines* population was designated virulent, i.e., given a "yes," for each differential for which the index was 10 or higher, based on the opinion that the population could not sustain itself or increase if the index were less than 10 (R.D. Riggs, personal communication). The population was rated "no" if the index was less than 10. The race designation for a population was determined by the pattern of "yes" and "no" it received in the chart. Four patterns had been observed by 1970, therefore four races were defined (note that 'Pickett' did not differentiate races). The system was supposed to have been open-ended so that new races would be added as they were found, but it was not until Riggs & Schmitt (66) expanded the original race scheme to its logical completion (16 races) that "new" races received a race designation (**Table 2**). The addition of new

Table 2 Races of the soybean cyst nematode *Heterodera glycines*, according to the race determination scheme[a] of Riggs & Schmitt[b]

Race	Pickett	Peking	PI88788	PI90763
1	–	–	+	–
2	+	+	+	–
3	–	–	–	–
4	+	+	+	+
5	+	–	+	–
6	+	–	–	–
7	–	–	+	+
8	–	–	–	+
9	+	+	–	–
10	+	–	–	+
11	–	+	+	–
12	–	+	–	+
13	–	+	–	–
14	+	+	–	+
15	+	–	+	+
16	–	+	+	+

[a]Race determination is made on the basis of the pattern of "+" and "–" ratings for each race. A "+" rating is given if the number of females produced by an *H. glycines* population on each soybean differential is equal to or greater than 10% of the number produced on the standard susceptible cultivar Lee. If the number of females is less than 10%, a "–" rating is given.
[b] Reference (66).

race differentials (soybean lines) to the race scheme would double the number of possible races with each addition; none was ever added, although new sources of resistance were identified (e.g., 1b).

Although the race scheme was subject to criticism for various reasons that have been reviewed elsewhere (52, 54), it remained the standard for characterizing *H. glycines* populations and was soon adopted by soybean breeders and seed companies as a means for conveying the resistance carried by soybean cultivars. Herein lie the biggest applied problems with the use of the race scheme: A listing in a seed company catalog or a label on a bag of soybean seed indicating that the cultivar is "resistant to soybean cyst nematode race 3" implies only that the cultivar has resistance to *H. glycines* populations with no capacity for

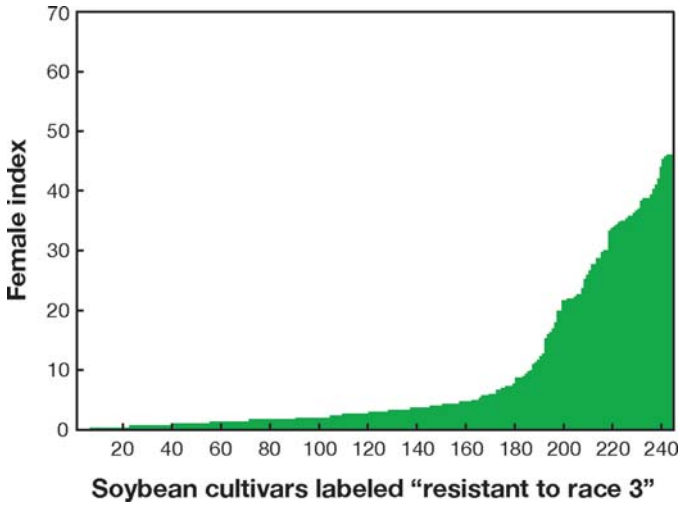

Figure 6

Female indices (FI) of 246 soybean cultivars labeled "resistant to race 3," in a greenhouse test with a race 3 population of *Heterodera glycines*, in 1999. Resistance is conventionally defined by FI values less than 10, calculated as follows: $(N_x/N_s) \times 100$, where N_x = average number of females on cultivar "*x*," and N_s = average number of females on the standard susceptible cultivar, Lee 74 [see TL Niblack & RD Heinz in (51*c*)].

HG Type: the designation, based on a bioassay, of the virulence profile of a *Heterodera glycines* population. The HG Type is a numerical designation referring to the sources of resistance in *Glycine max* (i.e., soybean germplasm lines) on which the nematode is able to develop

HG: *Heterodera glycines*

reproduction on a resistant cultivar (see **Tables 1, 2**), which is not very useful; furthermore, such labeling implies that all *H. glycines* populations designated as race 3 are the same and behave the same way (i.e., will reproduce to a similar extent) on all race 3-resistant cultivars. This is clearly not true, as one can see from the results of resistance screening programs, such as those conducted in Illinois (34–37) and Iowa (81, 82). For example, all of the cultivars tested in the Missouri Soybean Variety Performance trials were labeled "resistant to race 3," and were screened against an *H. glycines* isolate identified as race 3 (**Figure 6**). The cultivars that did not exhibit resistance in this test were probably not mislabeled by the seed companies, but they were screened during cultivar development to an *H. glycines* population that did not behave the same way as the race 3 population used for cultivar comparisons. A label that states "resistant to race 3" is not intentionally misleading, but clearly it is not informative or predictive either. Seed labels should state the source of resistance in cultivar development, not the "race resistance," to avoid giving unintentional misinformation to soybean growers.

A more serious problem for advancement of our understanding of virulence in *H. glycines* has been that a number of researchers have treated race designations as genotypes. For example, one can find citations for reports of attempts to identify genetic or other markers for *H. glycines* races. At least two characteristics of the race test make these efforts futile. First, race designations are based on average population phenotypes, and we have no idea how many genotypes may contribute to similar phenotypes. Obviously, one cannot identify markers in individuals for average characteristics of population phenotypes. Second, the definitions of races imply that virulence on one differential is linked to virulence on another. See, for example, the definition of race 4 (**Tables 1, 2**), which implies that each nematode in the population is able to reproduce on each differential, a situation that is impossible to test given the obligately parasitic nature of *H. glycines*.

HG Types

A new classification scheme was proposed in 2002 (54) to address some of the criticisms of the race scheme. Although it resolves the problem of there being more sources of resistance than are accounted for in the race scheme, it suffers from some of the same deficiencies. The HG Type test (HG simply stands for *Heterodera glycines*) currently involves seven sources of resistance—those known to have been used in developing resistant germplasm lines or cultivars in the United States—and decouples the resistance sources in nematode population descriptions (**Table 3**). This scheme is open-ended (additional soybean lines can be added) and easily adaptable to different geographic areas.

To identify the HG Type of an *H. glycines* population, a bioassay is conducted according to specific "rules," and the population is

Table 3 Indicator lines for HG Type classification of genetically diverse populations of *Heterodera glycines*[a]

Number	Indicator line
1	PI 548402 (Peking)
2	PI 88788
3	PI 90763
4	PI 437654
5	PI 209332
6	PI 89772
7	PI 548316 (Cloud)

[a]Reference (54).

named according to the number associated with soybean indicator lines (sources of resistance) on which it is virulent. Virulence is measured as a Female Index (FI), calculated exactly as described above for the race test index: $(N_r/N_s) \times 100$. HG Types should be reported not only by number, but also with the FI values for each number. For example, description of *H. glycines* field population #85 (**Table 4**) would be as follows: "the *H. glycines* population was Type 1.2-, with FI values of 23 and 42, respectively." The "-" following the HG Type designation indicates that the test was incomplete in that all seven HG Type indicator lines were not included in the test.

The HG Type system is useful for making cultivar recommendations. Results from an *H. glycines*–resistance screening program conducted at the University of Illinois (34–37) showed that the ability of an *H. glycines* population to reproduce on PI 88788 predicted its ability to reproduce on resistant cultivars carrying genes for resistance from that source (**Figure 7**). However, the level of resistance in soybean cultivars developed from the same source of resistance varies widely, so information like this must be combined with yield data (81, 82) and knowledge of the population densities in a particular field in order to optimize recommendations to soybean growers.

Table 4 HG Types of 16 *Heterodera glycines* populations collected in southern Illinois during 2004[a]

Sample number	Mean no. females on Lee 74	Female indices			
		PI 548402	PI 88788	PI 437654	HG Type
#14	163	7	32	0	2-
#52	403	3	30	0	2-
#57	124	7	8	0	0-
#59	140	19	13	0	1.2-
#60	223	0	25	0	2-
#63	279	0	7	0	0-
#66	139	0	8	0	0-
#67	127	11	26	0	1.2-
#70	189	31	25	0	1.2-
#71	327	6	19	0	2-
#73	291	1	31	0	2-
#80	113	5	24	0	2-
#85	208	23	42	0	1.2-
#89	232	9	9	0	0-
#92	209	2	15	0	2-
#112	258	7	27	0	2-

[a]J. Bond & T. L. Niblack, unpublished data.

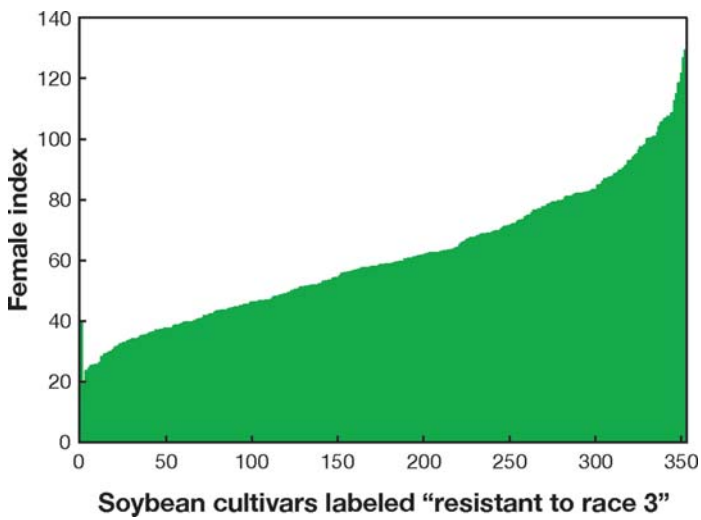

Figure 7

Female indices (FI) of 353 soybean cultivars with resistance derived from PI 88788, according to information provided by the seed companies, in a greenhouse test with an HG Type 2.5.7 population of *Heterodera glycines*, in 2003. Resistance is conventionally defined by FI values less than 10, where N_x = average number of females on cultivar "x," and N_s = average number of females on the standard susceptible cultivar, Lee 74.

INTERACTION BETWEEN *H. GLYCINES* AND OTHER DISEASES

In addition to causing yield loss through direct injury, the soybean cyst nematode also can affect the development of other soybean diseases.

For example, plants infected with *H. glycines* develop symptoms of sudden death of soybean (SDS), caused by the soil-inhabiting fungus *Fusarium solani* (Mart.) Sacc. f. sp. *glycines* Roy (= *F. virguliforme* Akoi, O'Donnell, Homma & Lattanzi), significantly earlier than uninfected plants, and disease severity is increased as well. The timing of appearance of disease symptoms and the severity of SDS are two major factors in the magnitude of yield loss caused by this disease.

Similarly, soybean brown stem rot (BSR) is caused by the soil-inhabiting fungus *Cadophora gregata* Harrington & McNew (*Phialophora gregata*) (Allington & D.W. Chamberlain) W. Gams (AC), and soybean plants infected with *H. glycines* are infected sooner with the fungus and disease incidence and severity are greater than in plants not infected with the nematode. In addition, plants with genetic resistance to BSR apparently lose expression of that resistance when infected with *H. glycines*.

Much is yet to be learned about the specific mechanisms whereby *H. glycines* affects SDS and BSR, as well as whether the nematode also interacts with other soybean pathogens or pests (See 1, 1*a*, 32*a*, 51*a*, 51*b*, 68*a*, 78*a*).

Management

Heterodera glycines is present throughout the soybean production regions of the United States (65), and may be found infesting a large percentage of the soybean fields in the Soybean Belt (95). For many years, soybean growers were urged to sample for the nematode if they observed areas in the field with stunted, chlorotic plants. However, *H. glycines* can cause significant yield loss in the absence of symptoms (58, 90, 100) (**Figure 8**), which makes it wise to recommend periodic sampling for nematode detection in any soybean field. An indirect symptom of an *H. glycines* infestation is early senescence of the soybean plants (**Figure 9**). In the midwestern United States, the symptoms expressed by soybean plants infected heavily with *H. glycines* are often those of a secondary problem in the field, for example, potassium deficiency (marginal chlorosis of leaves) or another pathogen (7).

Although different management tactics have been investigated (55), the only ones that consistently either increase yields in infested fields or reduce *H. glycines* population densities, or both, are rotation to nonhosts and the use of resistant cultivars. Unfortunately, much of the U.S. Soybean Belt alternates cropping of soybean with corn on an annual basis, so the use of nonhost crops in two or more consecutive years for nematode management purposes often may not be an option. There are hundreds of soybean cultivars available with resistance to *H. glycines* (34–37, 74, 85); most resistant cultivars contain resistance derived from PI 88788 and a few possess resistance from PI 548402 and PI 437654. Growers are advised to rotate sources of *H. glycines* resistance, if possible, in order to reduce selection pressure for an *H. glycines* population that can reproduce freely on the common, PI 88788 source of resistance (53).

Nematicides are available for management of *H. glycines*, and one or more are labeled for use in most states, but there is little evidence that they consistently increase soybean yields sufficiently to pay for their use. Often, the increase in *H. glycines* populations that can be measured in the fall following nematicide use at planting militates against their use unless other options have been exhausted. For the immediate future, rotation and resistance will probably remain the most viable options for management of *H. glycines*.

MOLECULAR NEMATODE-HOST INTERACTIONS

In recent years, a great deal of research has been conducted on the molecular biology of *H. glycines*. The focus has mainly been on understanding the molecular biology of *H. glycines*–soybean interactions; however,

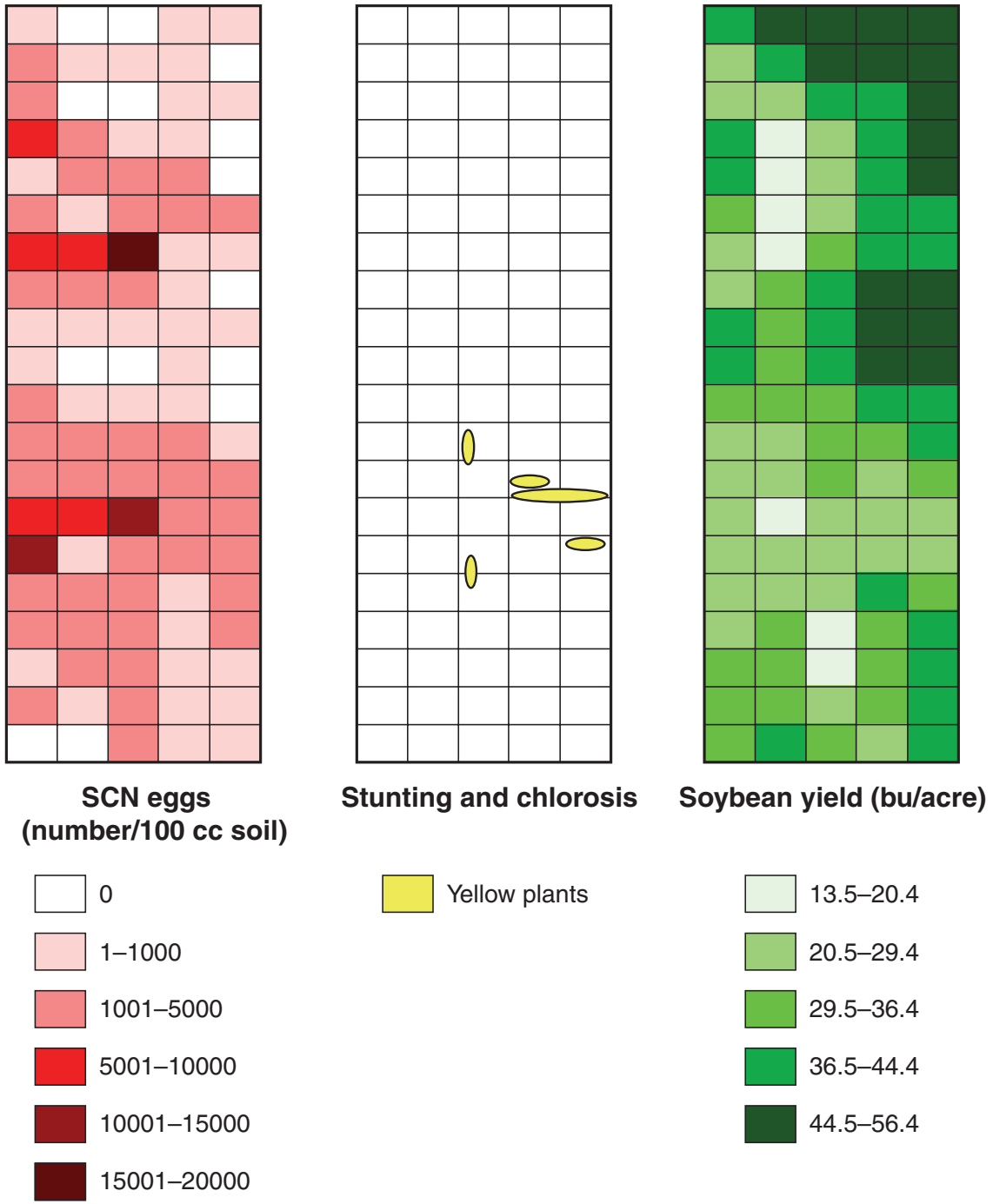

Figure 8

Heterodera glycines [soybean cyst nematodes (SCN)] population densities (*left*), visible stunting and chlorosis (*center*) and soybean yield (*right*) in 100 0.2-hectare (0.5-acre) cells in a field in central Iowa (G.L. Tylka & A.P. Mallarino, unpublished).

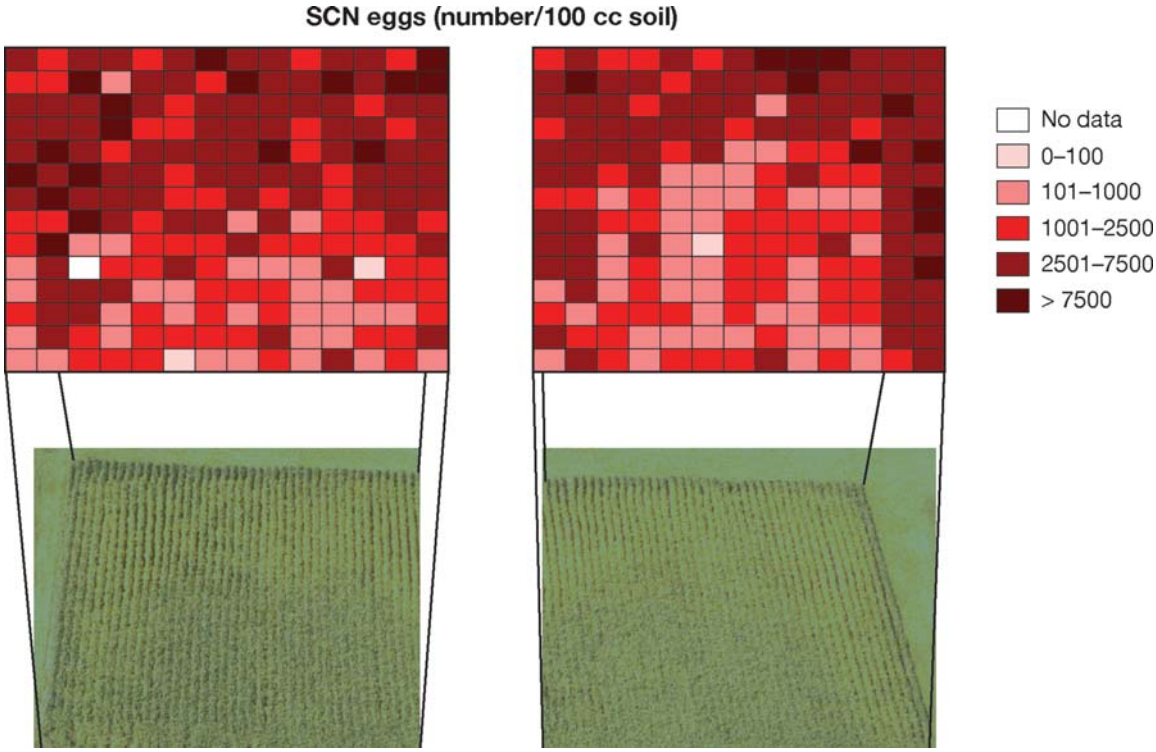

Figure 9

Heterodera glycines population densities at planting in May (*top*) corresponding to appearance of maturing soybeans on September 10 (*bottom*) in a field in north central Iowa (G.L. Tylka, unpublished).

important research has also been conducted on *H. glycines* target genes that may be of use in engineering soybean for resistance to *H. glycines*. In this review we focus on recent progress in the understanding of the molecular biology of *H. glycines* parasitism [for a recent review on engineering plants for resistance to *H. glycines* see (3)].

H. glycines parasitism of soybean is a complex process involving the interchange of information between the nematode and the soybean host. *H. glycines* is thought to parasitize soybean via the secretion of disease-causing substances, such as enzymes, peptides, and small metabolites. Although any cell in the nematode that emits an extracellular secretion could potentially play a role in nematode parasitism, most recent work has focused on the analysis of genes encoding *H. glycines* esophageal gland proteins.

A major contribution to the understanding of cyst nematode parasitism occurred when cellulose-degrading enzymes (β-1,4-endoglucanases) from *H. glycines* and *Globodera rostochiensis*, the golden potato cyst nematode, were identified (78). That nematodes contained plant cell-wall–degrading enzymes had been previously reported (6), but this discovery was notable since it was the first esophageal gland gene cloned and also because the β-1, 4-endoglucanases had homology to bacterial enzymes, suggesting that nematodes had acquired parasitism genes via horizontal gene transfer from bacteria. In *H. glycines*, β-1,4-endoglucanases have been shown to be secreted from the nematode (92) and are postulated to play a key role in *H. glycines* invasion of the root but not in syncytium formation, where plant β-1,4-endoglucanases play a key role (31). The

idea that nematode β-1,4-endoglucanases are essential for root parasitism has been supported recently by the finding that using RNA interference (RNAi) to inhibit β-1,4-endoglucanase expression in the potato cyst nematode lowers nematode infectivity (10). β-1,4-endoglucanase in *H. glycines* is not a single enzyme in the nematode but is a member of a gene family (26, 97, 98). These family members have differences in gene expression and substrate specificity, suggesting that the degradation of the plant cell wall during *H. glycines* invasion is a complex, highly regulated process (17, 27).

Other plant cell-wall–degrading enzymes have been discovered in plant-parasitic nematodes including pectate lyases, cellulose-binding proteins, and expansins. Pectate lyases were first discovered in potato cyst nematodes (62), but have also been identified in *H. glycines* (15). Enzymatic activity is difficult to measure for these enzymes and has not been demonstrated for *H. glycines* pectate lyases; still, this enzyme is assumed to be critical for aiding the nematode to break down plant cell walls during initial root invasion. The role of cellulose-binding proteins in nematode invasion of the root is not clear (24), but reports of bacterial cellulose-binding proteins altering plant cell expansion have been reported (51, 75). Thus, the *H. glycines* cellulose-binding protein may play a similar role. Along the same theme, nematode expansins have been reported in cyst nematodes, including *H. glycines* and have been shown to expand plant cell walls (46, 63). Nematode expansins are unusual because they have homology to plant and bacterial expansins and may represent further evidence of horizontal gene transfer from bacteria to phytoparasitic nematodes (46). Although dramatic progress has been made in understanding the molecular mechanisms of nematode invasion of the host root, how *H. glycines* alters the metabolism and development of plant cells is still unclear.

Nematode feeding cells, such as syncytia, are large, metabolically active plant cells, and it has long been assumed that *H. glycines* and other nematodes alter basic metabolic and developmental pathways. The first evidence for how a plant nematode may alter plant metabolism came with the discovery of a nematode chorismate mutase (CM) in the root-knot nematode, *Meloidogyne javanica* (48), and the subsequent discovery of CMs in *G. pallida* (42) and in *H. glycines* (5, 29). CMs are enzymes found in the shikimate pathway, a primary metabolic route in plants and microorganisms. Neither CMs nor the shikimate pathway are found in animals or nematodes other than root-knot and cyst nematode species. The shikimate pathway in plants produces the aromatic amino acids (phenylalanine, tyrosine, and tryptophan) and numerous secondary metabolites that play key roles in plant cell development, structure, and defense against biotic and abiotic stress. CM is a key branch point regulatory protein controlling the production of phenylalanine and tyrosine. Like other animals, phytoparasitic nematodes do not have a shikimate pathway; thus it is thought the nematode CM plays a role in altering the plant's shikimate pathway to assist the nematode in parasitizing the plant. Nematode CMs have signal peptides that predict they are secreted, and, in fact, a root-knot nematode CM has been shown to be secreted into the plant tissue as feeding cells are being formed (22); however, cyst nematode CMs have not been experimentally shown to be secreted. The enzymatic activity of *H. glycines* CM has been confirmed, but exactly how this enzyme might modulate plant metabolism is unclear due to a lack of understanding about the subcellular organization of the plant's shikimate pathway. It has been hypothesized that *H. glycines* CMs suppress chorismate-derived compounds produced in the plastid, while elevating cytoplasmic chorismate-derived compounds. This redirection of a plant metabolic pathway could drastically alter the types and levels of phenolic compounds produced by the plant and could greatly alter plant cell form and function (5).

RNAi: ribonucleic acid interference

CM: chorismate mutase

H. glycines CM comprises a small gene family with several alleles (5, 47). Genes encoding particular CM alleles are selected in *H. glycines* depending on the type of soybean on which they are grown. A recent population genetic study demonstrated that the *H. glycines* CM allele, *Hg-cm-1A*, is preferentially selected for on the resistant soybean PI88788 (47), suggesting the *H. glycines* CM may play a role in assisting the nematode to develop on resistant soybean or on that type of soybean background. *Hg-cm-1* is postulated to act as a virulence gene by a general suppression of shikimate chorismate-derived compounds that play a role in host plant defense (5, 47).

Another cyst nematode protein that is postulated to play a role inside the plant cell is an ubiquitin extension protein (86). This protein is secreted from the nematode and contains a nuclear localization signal that directs the protein, or part of it, to the plant cell nucleus. This is the first example of a nematode protein being targeted to the nuclei; however, its functional role in plant-nematode interactions remains to be determined. An *H. glycines* homologue of this protein has been identified along with other putative *H. glycines* proteins that may be targeted to the plant nucleus, but none has been functionally tested (29).

An interesting *H. glycines* gene that alters plant development is *HgCLE*, which encodes a small protein with a structural similarity to the extracellular plant peptide CLAVATA3 (CLV3) (59, 64). This small nematode peptide may play a key role in *H. glycines* feeding cell development, as suggested by the central role that CLAVATA-like plant signaling peptides play in plant meristem homeostasis. In plants, CLV3 interacts with a transmembrane receptor kinase (CLV1) to limit meristem size. *HgCLE* has been shown experimentally to complement *clv3–1* mutants in *Arabidopsis* and to alter plant development when overexpressed in wild-type *Arabidopsis* plants (93). However, the observation that distantly related *CLV3*-like genes (CLV40) can also complement clv3 mutant plants and that *CLV3* is part of a gene family with 24 members suggests unraveling the exact role of *HgCLE* will be an interesting challenge (11, 33). *HgCLE* is also unique in that it has been postulated to have been acquired by *H. glycines* not by horizontal gene transfer but by convergent evolution (59), which may represent another avenue for how phytoparasitic nematodes have evolved their parasitic ability.

Many other *H. glycines* genes that are expressed in the nematode esophageal glands and appear to encode secreted proteins have been identified via a variety of creative techniques (28, 29, 91). Some encode proteins with homology to known genes, but for most, their function remains obscure. Such genes include *H. glycines* homologs to Mi-SXP/RAL (87) and *H. glycines* venom allergens (25), *H. glycines* chitinase (26, 30), and proteins with recognizable plant-like domains (29).

Future high-resolution protein localizations and functional studies are needed to verify the secretion and subcellular location of the currently identified putative *H. glycines* parasitism proteins. The use of microarrays for large-scale *H. glycines* gene expression analysis (16) and the use of RNAi as a method to silence *H. glycines* genes (88) will be helpful in the functional analysis of genes expressed in nematode glands.

Although much work has been expended in cloning nematode esophageal gland genes, a lesser focus has been on the analysis of metabolites produced by plant-parasitic nematodes that may be critical for nematode parasitism. An elegant bioinformatics/genetic approach has determined that root-knot nematodes secrete nod factor–like metabolites that can alter plant development (71, 94). To date, genes that synthesize nod factors have not been discovered in cyst nematodes; however, the application of similar methods to *H. glycines* may identify new metabolites that play critical roles in parasitism.

Genetic analysis of *H. glycines* parasitism is an emerging approach that shows great promise. Classical genetic studies conducted on *H. glycines* over the years have been hampered by a lack of genetically homogeneous,

inbred *H. glycines* lines, molecular markers, and reliable methods to perform controlled crosses (80). Despite these difficulties, several papers have been published in recent years proving the feasibility of conducting genetic analysis of cyst nematode interactions with the plant. These studies have indicated that *G. rostochiensis* has a gene-for-gene relationship with corresponding resistance genes in the potato host, *Solanum tuberosum* ssp. *andigena* (40), and that several *H. glycines* genes, termed *ror* genes (19), some of which are dominant and others recessive, are involved with controlling *H. glycines* parasitism of resistant soybean cultivars (20, 21).

Preliminary genetic maps for *Globodera rostochiensis* (68) and *H. glycines* (2) have been developed; thus the tools for map-based cloning in *H. glycines* are now available. As further genetic/genomic resources are developed for *H. glycines*, the genetic analysis of phytoparasitic nematode parasitism will add to our understanding of how the nematode breaks down host plant resistance, alters it host range, and triggers host plant resistance mechanisms. The convergence of reverse and forward genetic approaches will allow detailed functional analysis of important phenotypes in *H. glycines*. In the future, information gained from the molecular analysis of *H. glycines*–soybean interactions will enable new methods to be developed to control these devastating plant pathogens.

SUMMARY LIST

1. *Heterodera glycines* continues to be an economically important pathogen of soybean because the primary means of managing it are limited by economics or adaptation by the nematode.
2. The life history and pathogenic characteristics of *H. glycines* make it an excellent model to study parasitism and virulence in plant-parasitic nematodes.
3. Two ideas should be integrated into management recommendations for fields infested with *H. glycines*: the action threshold and the HG Type concepts. These ideas, newly applied to *H. glycines*, are more useful than the widely used damage threshold and race concepts.
4. Recent advances in *H. glycines* genomics has helped identify many putative nematode parasitism genes, which in turn will aid in the understanding of nematode pathogenicity and virulence and will provide new targets for engineering nematode resistance.

FUTURE DIRECTIONS/UNRESOLVED ISSUES

Future genomic analysis will focus on building a genomic infrastructure for the detailed molecular genetic investigations of *H. glycines* parasitism, virulence, or other traits of interest. The sequencing of the *H. glycines* genome(s) will allow the identification of the entire genes space; this in turn will allow whole-genome analysis and a systematic functional analysis of the genome. By focusing on the virulence and other key traits relevant to managing *H. glycines*, significant progress will be made in enhancing the durability of *H. glycines* resistance in soybean and in the development of new, "engineered" nematode-resistant soybean cultivars. Specific items:

1. Completion of the sequencing of the *H. glycines* genome
2. Development of a genetic approach to the analysis of SCN
3. Development of whole-genome methods of analysis of SCN (microarray)
4. Development of proteomic methods of analysis
5. Development of metabolomic methods of analysis
6. Development of high-throughput methods of functional analysis (RNAi)
7. Development of high-throughput methods for subcellular localization.

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