Symposium: Genetic Aspects of Breeding for Resistance Against Soilborne Plant Pathogens

Genetics of the Cyst Nematode-Soybean Symbiosis

V. D. Luedders

Research agronomist, USDA, ARS, and Department of Agronomy, University of Missouri, Columbia 65211. Journal Series Paper 9184. Accepted for publication 28 January 1983.

To many people, symbiosis means both symbionts or organisms benefit from the association. Although *Rhizobium japonicum*-soybean (*Glycine max*) associations are generally regarded as symbiotic, some do not fix nitrogen and the bacteria are parasitic. On the other hand, plant parasitic nematodes are usually not regarded as beneficial, but small numbers of nematodes may enhance seedling growth. DeBary's (cited in reference 9) original definition of symbiosis as the living together of unlike organisms covers all these variations.

The term resistance, which is frequently used (and misused), usually implies the host-oriented view of symbioses. Resistance generally refers to relative and variable categories of host response to specific pathogen isolates rather than to distinct phenotypes; thus, its meaning is usually vague. Consider cultivars X, Y, and Z; X is more resistant than Y, but less resistant than Z, to pathogen isolate W. The association is the functional unit, not the host or pathogen individually, and association phenotypes are determined by genotypes of both symbionts as they interact with each other and the abiotic environment. Whether a given host gene or allele acts as a gene either for or *not* for resistance or susceptibility, depends on the corresponding gene in the pathogen. For lack of better terms, I will use resistance and avirulence in the usual, vague sense.

The usual abbreviation for soybean cyst nematodes (*Heterodera glycines* Ichinohe) is SCN. In this presentation, only CN will be used to make reference to the association with soybeans (S) less confusing, eg, in the CN-S symbiosis. Hartwig (5) recently reviewed the history of CN, the inheritance of soybean resistance to CN, and his breeding program for resistance. As with any host-parasite association, the genetics of soybeans, CN, and their interaction must be considered at both the organismal and populational levels. The time and space allotted here are inadequate to cover the biology, genetics, host gene deployment strategies, etc. Therefore, this report will describe the state of the art and a cooperative research approach (with nematologist V. H. Dropkin) to learn how to live with this pest that cannot be eradicated.

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. § 1734 solely to indicate this fact.

Resistance and breeding. Resistance to CN is classified by the relative number of females or cysts supported by host lines. Lines may be considered to include cultivars, plant introductions (PIs), segregants from crosses, etc. A 0 to 4 scale (1) in which 0 = none, 1 = 1–5, 2 = 6–10, 3 = 11–30, and 4 = >30 white females per plant is frequently used. Lines are usually screened for resistance to CN in infested field soil in 8-cm-diameter pots; the root system is rated at ~30 days after planting. We have used this procedure to identify several good sources of resistance and to develop cultivars resistant to CN, recently including races 3 and/or 4. These cultivars have reduced damage from CN, but the resistance is not necessarily effective against all known or potential races of CN, so breeding programs are still being developed in reaction to changes in CN populations in the field. We still lack the knowledge necessary to greatly improve our breeding materials and methods or to plan long-range breeding or gene deployment strategies.

Many progeny from soybean crosses must be screened to provide enough resistant plants to evaluate for other characters. Based on results with mixtures of resistant and susceptible cultivars, Luedders and Duclos (11) suggested growing segregating soybean populations on CN-infested soil for several generations to increase the frequency of resistant plants. Hartwig et al (7) grew a segregating soybean population for four generations on land either free of or infested with CN. After the fourth generation, they tested the plants for resistance to CN in infested soil in the greenhouse and found that 5% of the plants from the population grown in the absence of CN were resistant, but 40% of the plants from the population exposed to CN were resistant to CN. Percentage changes in several backcross progenies were even larger. Thus, CN pressure can be used to change host gene frequencies.

Physiological races and population structure. The term "physiological" has frequently been used to "explain" phenomena that were not yet understood. The first "genetic" variation in CN was reported by Ross (17) in 1962. In 1970, Miller (14) differentiated 11 isolates of CN as races, while Golden et al (3) "officially" named four races. Riggs et al (16) arbitrarily separated

38 populations into 36 "variants" (races). Plants of PI 88788 and PI 90763 have been reported to be both resistant and susceptible to CN race 4 (22). This confusing situation was caused by vague definitions of resistance and races and a lack of statistical treatment of the data.

Triantaphyllou (20) proposed that different populations vary in gene frequencies and that frequencies of specific genes for parasitic ability determined the race designation of a population. He also cautioned against indiscriminate designation of new races. Conceptually, at least, variation in gene frequencies appears to be a sound basis for thinking about CN population structure, even though no individual genes have been identified thus far.

Gene frequencies can be changed by mutation, migration, and selection. Triantaphyllou (20) showed that selection increased a CN population's ability to reproduce (number of cysts) on resistant soybeans Pickett and PI 88788. Number of cysts formed by the base population on PI 88788 was only 5% of the number on the susceptible check Lee, but that increased to 40% of the number on Lee after five generations of selection. Changing a population's reproduction on one host line did not appear to change it on the other host line; thus, different genes must be active in the different CN-S associations. McCann et al (13) evaluated reproduction of the population selected on Pickett by Triantaphyllou (20) and several populations similarly selected from one field in Missouri on other soybean lines. Three different sets of soybean genes were postulated: those in Pickett-Pickett 71, in PI 89772, or in Cloud, PI 87631-1, PI 88788, and PI 209332. Young (24) selected populations from three different fields for ability to reproduce on several other soybean lines. He concluded that PI 89772, PI 90763, and Peking had genes for CN resistance in common but different from those in PI 88788 and Bedford. Since Pickett got some, but not all, of Peking's genes affecting CN reproduction, there may be only two distinct groups of genes.

Additional evidence for similarity of genes in Cloud, PI 87631-1, PI 88788, and PI 209332 was obtained by Luedders and Dropkin (*unpublished*). We selected CN for ability to reproduce on plants of seven resistant breeding lines developed from these four sources of resistance by R. L. Bernard (USDA, and Department of Agronomy, University of Illinois, Urbana). Selection on any plant line increased the resulting CN population's ability to reproduce on plants of all four sources of resistance and all seven lines. Like several cultivars released as resistant, several of the breeding lines were less resistant than the source from which they were developed.

Luedders and Dropkin (10) did a secondary selection test with two CN populations. The CN populations originally selected on PI 89772 or on Cloud could form few cysts on the other soybean line, so these were then selected for ability to do so. Four CN generations of secondary selection increased the populations' ability to reproduce on the second selecting soybean line, but decreased it on the first. The CN genes were not fixed by the primary selection (frequencies not 0 or 1) and did not appear to be independent. Results from selection experiments of others (20,24, and S. C. Anand and G. S. Brar, *personal communication*) support allelism more than linkage. Selection probably is more important than mutation or migration in changing CN gene frequencies in fields. CN are relatively immobile and relatively few would be moved from field to field passively. Reproduction is amphimictic and only on hosts, which includes a moderate range of plant species. Winter temperatures are usually too cool for much, if any, increase in numbers between growing seasons. Essentially nothing is known about genetic variation or its distribution within and among populations. Multiple matings are possible by both sexes of potato and sugar beet cyst nematodes (4). A heterozygous CN female inseminated by several heterozygous males could result in a cyst containing most of the species' genes. Triantaphyllou and Riggs (21) isolated a tetraploid population from a field in Indiana. Triploids resulting from crosses with diploids also were viable and competitive. Polyploidy increases the genetic variation available to individual nematodes and complicates the genetic considerations.

Eggs in cysts can remain viable in soil for many years. Eliminating them and the genes they contain may not be practical. Reproduction in the field probably is a continuum in time and space, with no discrete generations. Larvae invade only young, actively growing root tissue. Generation time varies with temperature, but several generations per soybean crop are possible. Eggs in external egg sacs can hatch immediately, whereas eggs in brown cysts may require low temperatures to break dormancy (8). Individual genes may be transmitted several times in one season and then be stored for several years. The methodology for following reproduction and gene flow is not simple.

Association phenotypes, genetics, and strategies. In symbiosis, the genotypes of two organisms determine the association phenotype and, for intraorganismal genetic studies, the genotype of one symbiont is held constant and becomes part of the other symbiont's environment. However, association phenotypes are determined by genes in both organisms. This basic relationship must be kept in mind even if vague host- or pathogen-oriented terms like resistance or avirulence are used. Results apply only to the particular genotypes used and not to lines, races, or the species broadly.

Phenotypes observed in the CN-S symbiosis are usually quantitative cyst counts that have high coefficients of variation. Differences are rarely black and white. Thus, a sound experimental design and proper statistical analysis are necessary to determine which differences have a reasonable probability of reflecting real genetic differences. We probably do not know enough about the CN-S biological system to do any definitive genetic studies.

CN races and populations have been crossed to study the inheritance of parasitic ability, with the general conclusion that it is "nonrecessive." Price et al (15) stated ". . . data were not adequate to propose specific inheritance patterns," and Triantaphyllou (20) indicated that his data "are not easy to interpret." His parent populations on Pickett had indices of parasitism of 90 and 0 (0.90 and 0.00 of that on Lee) and the F_1 and F_2 were intermediate. What gene frequencies are required for "full reproduction" is not known, but may be considerably less than one.

It has been postulated that most soybean genes for CN resistance are recessive. Perhaps the best evidence for a dominant gene for resistance was for one in Peking linked with a gene for black seed coats (12). However, some of the ratios deviated considerably from 3:1, and Sugiyama and Katsumi (19) reported that Peking had a recessive gene for resistance to CN linked with black seed coats. Resistance usually is poorly defined and may refer to several different phenotypes. This problem is not unique to numbers of cysts since different qualitative infection types have been and still are used to indicate resistance or are lumped in resistant:susceptible categories.

The susceptible category may cover quite a range of phenotypes (and genotypes) when it includes everything with more cysts than the resistant parent or more than 30 cysts per plant (rating of 4 on a 0 to 4 scale). Distributions of cyst indices of parents and two F_2s are shown in Fig. 1 (D. R. Smith, *unpublished*). The cyst index of 100 was 216 cysts on Williams (average of 24 plants). The cyst index for each test host was calculated as $CI = 100\ (\text{ratio})^{1/2}$, in which the ratio is the number of cysts on the test host (F_2 or parent) divided by the 216 cysts on Williams. Hence, the equation, $\text{ratio} = (0.01\ CI)^2$, which gives distributions of ratios, would be somewhat different from those for cyst indices. Numbers of cysts produced by F_2 plants ranged from 12 to 276, or ratios of 0.06 to 1.25, which may be more realistic than CIs of 20 and 112.

Smith (*unpublished*) pooled the two crosses and considered nine of the 256 F_2 plants to be as resistant as PI 88788. He indicated that this ratio fit the expected for two dominant and two recessive genes exactly, but that it was also consistent with one dominant and two recessive genes for resistance to his CN population. A curve similar to that for the PI 88788 × Williams F_2 could be generated by equation 1 with four or more genes (*n* loci) dominant for producing cysts (recessive resistance), each locus with one or two dominant alleles adding some cysts. The equation can be

$$\left\{0.5\ (1 - 0.5^s)aa + [1 - 0.5\ (1 - 0.5^s)]\ A\_\right\}^n \qquad (1)$$

logically derived since heterozygosity changes by half (one-half or 0.5) each selfing(s) generation and taken from one (1.0), gives the

homozygotes, half of which are recessive like *aa*. The proportion $A\_$ is obtained by subtraction since $aa + A\_ = 1$. The binomial can be expanded in F_2 ($s = 1$) as $C(n,r)\ (0.25)^{n-r}(0.75)^r$ to get the theoretical frequencies (probabilities) of r loci with at least one dominant allele ($A\_$), where r varies from 0 to n. $C(n,r) = n!/[r!(n\text{-}r)!]$ is the number of combinations of n things taken r at a time.

Thus, the curves for the Williams crosses can be explained by four or more recessive genes for resistance. Smith (*unpublished*) also had F_2 curves of Custer and Williams × Bedford (genes from PI 88788), which were similar to those in Fig. 1. The mean and distribution of F_2 progeny of the Custer crosses differed significantly from those of the Williams crosses. Williams and Custer had similar phenotypes (Fig. 1), but probably different genotypes. Custer, like Pickett, supposedly got four genes (one dominant) for resistance to CN from Peking. Luedders and Dropkin (10) suggest that Williams may have at least one gene in common with Cloud and thus, probably also with PI 88788. One or more dominant genes for resistance could give the distribution of the Custer crosses, but they could also be explained by more recessive alleles with a lower average effect on numbers of cysts. Obviously, the genetics of the CN-S symbiosis are far from being resolved and may not conform to the usual single dominant genes for resistance-avirulence. Sidhu and Webster (18) suggested that the CN-S relationship also was in sharp contrast to that of other nematodes-hosts. A number of genes in both symbionts must be involved in basic compatibility. Variation in these genes could be manifested as apparent recessiveness for resistance-avirulence.

Both fungi and hosts have been crossed for intraorganismal genetic studies. Heterozygous F_1s of both organisms generally have given association phenotypes that were low infection types, definitive, or incompatible (host resistant, or pathogen avirulent). Incompatibility in the heterozygous state appears to favor hosts more than pathogens although these fungi are surviving quite well.

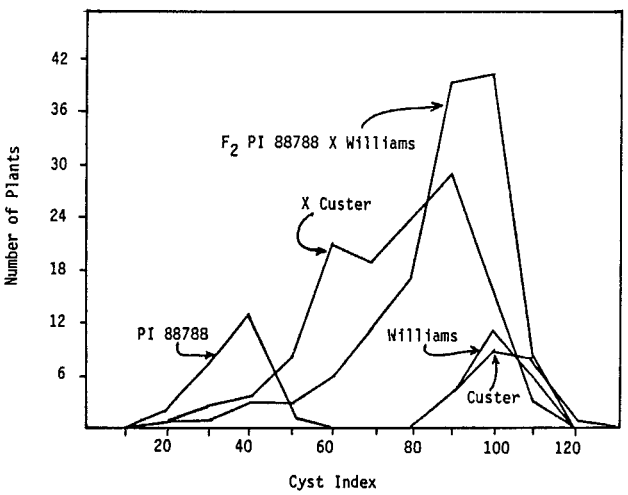

Fig. 1. Distributions of cyst indices on F_2 and parental generations of crosses of soybean cultivars Williams and Custer with PI 88788. Cyst index is the square root of the transformed cyst count, expressed as percentage of mean of cultivar Williams. (From D. R. Smith [*unpublished*]).

TABLE 1. Hypothetical results[a] from crosses of soybeans and CN

CN population	Soybean				
	PI 88788	PI 90763	F_1	Williams	L.S.D.[b]
Selected on					
PI 88788	1.00	0.10	0.95	350	0.20
PI 90763	0.15	1.10	1.05	95	0.25
F_1	0.87	0.95		230	0.25

[a] Values for cultivar Williams are numbers of cysts; others are proportion of Williams within CN populations.
[b] $P = 0.05$.

However, it would not be a particularly good strategy for survival of CN if heterozygotes were incapable of reproducing on several different soybean lines. Selection may be against specific genes for avirulence in heterozygous fungi, but for CN genes conditioning the ability to reproduce on certain soybeans.

If resistance to CN in soybeans is recessive and if the ability of CN to reproduce on resistant soybeans is dominant, then a study of the interorganismal genetics of CN-S could produce results resembling the general pattern illustrated in Table 1. The hypothetical results in Table 1 are based partly on Smith's (*unpublished*) data showing high CN reproduction on F_1s of crosses of resistant × susceptible soybeans and on data of Price et al (15) who reported a CN F_4 isolate with cyst indices of 84 on Peking, 70 on PI 88788, and 121 on PI 90763. Thus, a heterozygous CN may be capable of reproducing on genetically different sources of resistance.

Two selected populations are being intermated to determine if their progeny reproduce as postulated in Table 1. In presenting data in the form shown in Table 1, an analysis of variance should be performed on the numbers of cysts obtained with each CN population. Means and LSDs can then be expressed as a proportion of (or ratio to) Williams for easier comparisons among populations. Any line can be used in place of Williams as the check; a universal suscept might be best, but probably one does not exist. Actual numbers of cysts on Williams are more useful than a column of 1.00s. Transformation of data is not essential; it will sometimes detect additional significant differences in the lower end of the range while losing them in the upper end. Populations may perform differently for several reasons. The same CN-Ss give different numbers at different times, but relative values or ratios tend to be similar. The method could be used for CN, but not soybean F_2s. Materials and methods are not available to get discrete phenotypes with segregating soybeans, and specific genes cannot be identified by analyzing continuous distributions. The best phenotypic descriptor to use may be ratios, but even with homozygous CN-S, some variation about the true means must be expected.

At present, the genetics of CN probably can be determined only rather grossly. However, it is necessary to plan a strategy of soybean gene deployment. If rotating genes from PI 88788 and PI 90763 results in field CN populations that damage all cultivars with any of these genes, then what? Only these two different groups of genes have been postulated. Doom may not necessarily be immediate or inevitable; Hartwig et al (6) reported "no significant change" after 5 yr of continuous resistant soybeans. Plots in one field may not represent all CN-S or their interactions with environment.

The genetics of resistance of soybeans to CN are far from definitive. Apparently, a number of genes are involved. Four or more is consistent with the number of resistant segregates recovered in a breeding program that has some true-breeding lines with intermediate CN resistance (R. L. Bernard, *personal communication*). Reactions of soybean progeny can be used to postulate genetic differences, but results depend on the corresponding CN genes. CN populations are heterogeneous. Since individuals may be heterozygous and multiple matings may occur, starting with single cysts may not result in more homogeneous populations or conclusive results. Also, observed phenotypes are quantitative, and such traits generally are affected more by unexplainable variation than are qualitative characters.

Research, models, and reality. It is necessary to know the genetic composition of pest populations to deal with them effectively. A CN gene pool was made to sample and recombine the CN genes. We obtained about 20 CN samples from several states and mixed them into a microplot (0.91 m in diameter). The populations were allowed to intermate on three CN-susceptible soybean cultivars, with CN-soil mixed prior to replanting each time.

Relatively few soybean lines previously screened against field populations had sufficiently low numbers of cysts to be considered useful in breeding. It seemed unlikely that all the genes limiting CN development were located in only a few lines and that most lines had no useful genes. Therefore, we tested some "useless" lines that had intermediate numbers of cysts, against the CN gene pool.

Reproduction ratios ranged from less than 0.1 of, to not different from the Williams standard, which could reflect different soybean genes and varying frequencies of corresponding CN genes. Part of the variation may be due to variable CN gene frequencies, but soybeans with intermediate numbers of cysts might have fewer and/or different alleles for resistance than those with few cysts. Ratios of numbers of cysts on the "good, useful" sources of resistance to those on the Williams standard were less than 0.1, but the genetic complements of "useless" soybeans with ratios significantly less than 1.0 are also of particular interest.

Second-stage larvae must hatch, find and invade roots, induce syncytia or giant feeding cells, and develop into fifth-stage adult females before soybean resistance to them is assayed. Thus, there are a number of steps at which CN development can be blocked. Changing a soybean allele interacting with a CN gene at a given step may not change the probability of CN success from 1 to 0. Rather, the change in probability may be much more moderate, neither 1 nor 0, with changes at several loci required to decrease number of cysts from many to none. The overall probability would be the product of probabilities at all steps. The genetic system might, however, appear to be additive: each gene change (from dominant to recessive in either symbiont) eliminating some cysts. If 10 genes were involved, the probability of CN success might be $0.95^{10} = 0.599$. Ten recessive alleles for resistance might each reduce the probabilities of success by 30 or 40%; for all 10 together $0.7^{10} = 0.028$ and $0.6^{10} = 0.006$. Different alleles at the same or different loci may give different probabilities of CN success, which would also vary with the alleles at corresponding loci in the other symbiont. For example, one can also get 0.028 from $0.95^8 \times 0.21^2$, and 0.006 from $0.9^7 \times 0.23^3$ or $0.9^4 \times 0.6^3 \times 0.35^3$. Innumerable combinations of probabilities from alleles and loci can be imagined. Dominance for resistance-avirulence cannot be ignored, because of its effects on phenotypic distributions in segregating generations. Blocks at different steps are not as readily observed as qualitative infection types in rusts and mildews, but they may be analogous. Since phenotypes observed with CN-S are quantitative numbers of cysts, perhaps no comparisons should be made.

Phenotypes (numbers of cysts) are determined by the genotypes of both organisms. The mating system of CN encourages heterozygosity of individuals in heterogeneous populations. Consistent phenotypes cannot be expected from variable genotypes; thus, CN genetic variability must be reduced before definitive genetic studies can be conducted.

Selection changes gene frequencies, with some increase in homozygosity of the genes involved. Inbreeding increases homozygosity, which may give genotypes with low survival, especially in species that normally have little or no inbreeding. CN may be subjected to some inbreeding due to their relative immobility, especially at low population levels. In experimentally controlled inbreeding, single sister-brother matings are not very practical. It is relatively easy to transfer single gravid females before they mature into cysts and fall from the roots. The rate of inbreeding depends mainly on the rarer sex (2), so multiple matings of the single females are not a deterrent to using this easy method for inbreeding CN. The method was tested with cysts from a field soil. Duplicate inbred females were transferred to reduce loss of inbreds due to dead plants, delayed or no hatching, etc. In nine generations of inbreeding some inbreds were lost, which may or may not have been due to lethals. Ten generations of single-cyst transfers should have decreased within-inbreds genetic variability considerably (increased homozygosity). Apparently, CN will tolerate inbreeding as well as selection.

If the base population used for inbreeding has low frequencies of genes interacting with specific soybean genes, then only a few inbreds may be useful for extensive genetic studies. It is probably much easier to generate 100 inbreds than to test them adequately. Simultaneous selection-inbreeding should increase the proportion of inbreds with genes interacting with the selecting genotypes. We grew PI 89772 and PI 209332 in soil with the CN gene pool and recovered relatively few cysts on them. These cysts had selection pressure, but no inbreeding and were transferred singly for selection-inbreeding on the same soybean line. Subsequent single female transfers were duplicates; a few were lost in nine generations of selection-inbreeding.

The selected-inbred (SI) CN populations were developed with two main questions in mind. Are CN genes for ability to reproduce on PI 89772 and PI 209332 alleles? Do the useless soybean genes appear to be similar to those in PI 89772, in PI 209332, in neither, or in both? Secondary selection indicated that some CN genes may not be independent. If all SIs can reproduce only on the soybeans they were selected on (only PI 89772 and PI 209332 are considered here), then some CN genes are alleles. However, SI reproduction on both PIs could indicate linkage. Linkage would be most obvious with the level of reproduction on their selecting host equal to or greater than that on the check cultivar. Lower levels could be due to less than the full complement of genes required. Crossing such SIs should increase the reproduction of some CN F_1s. For example, SIs that produced 0.3 and 0.4 times as many cysts as the check on PI 89772 could produce an F_1 that produced 0.9 as many cysts as the check. This would be interpreted as evidence that at least two genes were involved and some alleles were fixed by inbreeding before selection could increase the frequency of other alleles (or they were not present in the original selected cysts). Some SIs will be subjected to additional selection to test homozygosity or fixation of genes.

Cyst ratios of other soybean lines relative to the Williams standard with gene pool CN are benchmarks; deviations of ratios obtained with 40 SIs from benchmarks will be used to postulate soybean genetic differences and/or similarities to PI 89772 and PI 209332. Since soybean genes can be identified only by their interaction with CN genes, corresponding CN genetic differences also can be postulated. What appears to be complex with heterogeneous CN populations should become somewhat simpler (and perhaps different genetically) with reduced genetic variation in the SIs.

The genetic model that was the basis for this research may appear to be a combination of assumptions, conclusions based on vague data, and misuse of sound principles. However, it seems that: the CN-S genetic system may not be the same as that used to develop the gene-for-gene concept and interorganismal genetics; interactions among loci might be additive rather than epistatic; and selection could be for, rather than against, specific CN genes.

Genetic models or hypotheses are useful and essential for planning experiments. One danger with models is that they may be confused with reality. Our hypothetical models should be grasped tenuously, so we can alter or discard them readily, as indicated by results of sound experiments. We probably do not know all the mechanisms or rules of symbiosis. Some CN-S genes may follow Wheeler's (23) rule of definition (specific interactions for host susceptibility) rather than Flor's rule (specific interactions for host resistance). Wheeler (23) indicated that genetic analysis of pathogenesis (symbiosis) might appear to be a game that anyone could play by their own rules. Actually symbionts are playing the game by their rules; we are merely trying to determine what their genetic rules are. We became involved in the game when we started managing hosts, but their environments are not gnotobiotic. To manage symbioses to our advantage, we must learn their rules.

LITERATURE CITED

1. Epps, J. M., and Hartwig, E. E. 1972. Reaction of soybean varieties and strains to race 4 of the soybean cyst nematode. (Abstr.) J. Nematol. 4:222.
2. Falconer, D. S. 1960. Introduction to Quantitative Genetics. The Ronald Press Company, New York. 365 pp.
3. Golden, A. M., Epps, J. M., Riggs, R. D., Duclos, L. A., Fox, J. A., and Bernard, R. L. 1970. Terminology and identity of intraspecific forms of the soybean cyst nematode (*Heterodera glycines*). Plant Dis. Rep. 54:544-546.
4. Green, C. D., Greet, D. N., and Jones, F. G. W. 1970. The influence of multiple mating on the reproduction and genetics of *Heterodera rostochiensis* and *H. schachtii*. Nematologica 16:309-326.
5. Hartwig, E. E. 1981. Breeding productive soybean cultivars resistant to the soybean cyst nematode for the southern United States. Plant Dis. 65:303-307.
6. Hartwig, E. E., Epps, J. M., and Buehring, N. 1982. Response of

resistant and susceptible soybean cultivars to continuous cropping in area infested with cyst nematode. Plant Dis. 66:18-20.
7. Hartwig, E. E., Kilen, T. C., Young, L. D., and Edwards, C. J., Jr. 1982. Natural selection in heterozygous soybean populations exposed to Phytophthora rot or soybean cyst nematodes. Crop Sci. 22:588-590.
8. Ishabashi, N., Kondo, E., Muraoka, M., and Yooko, T. 1973. Ecological significance of dormancy in plant parasitic nematodes. I. Ecological differences between eggs in gelatinous matrix and cyst of soybean cyst nematode. Appl. Entomol. Zool. 8:53-63.
9. Loegering, W. Q. 1978. Current concepts in interorganismal genetics. Annu. Rev. Phytopathol. 16:309-320.
10. Luedders, V. D., and Dropkin, V. H. 1983. Effect of secondary selection on cyst nematode (*Heterodera glycines*) reproduction on soybeans. Crop Sci. 22:(in press).
11. Luedders, V. D., and Duclos, L. A. 1978. Reproductive advantage associated with resistance to soybean cyst nematode. Crop Sci. 18:821-823.
12. Matson, A. L., and Williams, L. F. 1965. Evidence of a fourth gene for resistance to the soybean cyst nematode. Crop Sci. 5:447.
13. McCann, J., Luedders, V. D., and Dropkin, V. H. 1982. Selection and reproduction of soybean cyst nematodes on resistant soybeans. Crop Sci. 22:78-80.
14. Miller, L. I. 1970. Differentiation of eleven isolates as races of soybean cyst nematode. (Abstr.) Phytopathology 60:1016.
15. Price, M., Caviness, C. E., and Riggs, R. D. 1978. Hybridization of *Heterodera glycines*. J. Nematol. 10:114-118.
16. Riggs, R. D., Hamblen, M. L., and Rakes, L. 1981. Intra-species variations in reaction to hosts in *Heterodera glycines* populations. J. Nematol. 13:171-179.
17. Ross, J. P. 1962. Physiological strains of *Heterodera glycines*. Plant Dis. Rep. 46:766-769.
18. Sidhu, G. S., and Webster, J. M. 1981. The genetics of plant-nematode parasitic systems. Bot. Rev. 47:387-419.
19. Sugiyama, S., and Katsumi, H. 1966. A resistant gene to the soybean cyst nematode observed from cross between Peking and Japanese varieties. Jpn. J. Breed. 16:83-86.
20. Triantaphyllou, A. C. 1975. Genetic structure of races of *Heterodera glycines* and inheritance of ability to reproduce on resistant soybeans. J. Nematol. 3:356-364.
21. Triantaphyllou, A. C., and Riggs, R. D. 1979. Polyploidy in an amphimictic population of *Heterodera glycines*. J. Nematol. 11:371-376.
22. Tisselli, O., Sinclair, J. B., and Hymowitz, T. 1980. Sources of resistance to selected fungal, bacterial, viral and nematode diseases of soybeans. Int. Agric. Pub., INTSOY Series 18.
23. Wheeler, H. 1975. Plant Pathogenesis. Springer-Verlag, New York.
24. Young, L. D. 1982. Reproduction of differentially selected soybean cyst nematode populations on soybeans. Crop Sci. 22:385-388.