韓作誌(Korean J. Crop Sci.) 35(1) : 97~107(1990) 

# Epidemiological Concepts and Strategies in Breeding Soybeans for Disease Resistance

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The epidemiology of plant disease deals with the dynamic processes of host-pathogen interactions, which determine the prevalence and severity of the disease. Epidemic processes for most foliar diseases of plants follow a series of steps: arrival of pathogens on plant surfaces, initial infection, incubation period, latent period, sporulation, dissemination of secondary inoculum, and infectious period. These complex biological processes are influenced by the environment. Man also often interfers with these processes by altering the host and pathogen populations and the environment. Slowing or halting any of the epidemic processes can delay the development of the epidemic, so that serious losses in yield due to disease do not occur. It is generally recognized that the most effective and efficient method of minimizing disease damage is through the use of resistant cultivars, particularly when other methods such as fungicide applications are not economically feasible. Populations of plant pathogens are not genetically uniform nor are they necessarily stable. Cultivars bred for resistance to current populations of a pathogen may not be resistant in the future due to selection pressures placed on the pathogen populations. Understanding population development and genetic variability in the pathogen, and knowledge of the genetics of resistance in the plant should help in developing breeding strategies that will provide effective and stable disease control through genetic resistance.

In the United States, soybeans have ranked first in value of crops sold off the farm in recent years. Soybeans have been the leading U.S. agricultural export in dollars since 1962. The five north central states of Illinois, Iowa, Indiana, Minnesota and Ohio produced 58% of the soybeans harvested in 1970. Currently, Illionis is the largest soybean-producing state based on the acreage of harvested soybeans. A large number of foliar pathogens occur in this region. The diseases caused by these pathogens vary greatly in importance. Severe disease damage has occurred in isolated locations although most foliar diseases have not caused epidemics over large areas. Nonetheless, the virulence of pathogens of soybeans is dynamic and thus the pathogens have the genetic potential to cause severe epidemics.

The objective of this presentation is to discuss my research program at the University of Illinois on the epidemiology of foliar diseases of soybean and the genetics of disease resistance in soybeans. This program is being developed to maximize the potential use of genetic resistance to control serious soybean diseases based upon epidemiological concepts and strategies. Also, applications of plant tissue culture in developing disease resistance and molecular genetic techniques in characterizing pathogenicity genes in pathogen populations are included in the research program.

## BACKGROUND

Soybeans have ranked first in value of crops sold off the farm in recent years and have been the leading U.S. agricultural exports in dollars since 1962. The five north central states of Illinois, Iowa, Indiana, Minnesota and Ohio produce more than 60% of the soybeans harvested in the United States. Currently, Illinois leads in

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the acreage of harvested soybeans.

A large number of foliar pathogens occur in this region. The diseases caused by these pathogens vary greatly in importance. Most soybean foliar diseases are endemic in the Midwest and have not caused severe epidemics over large areas, although severe disease damage has occurred in isolated locations. Nonetheless, the virulence of these pathogens is dynamic and thus they have the genetic potential to cause severe epidemics. In addition, the current trends of increased minimum tillage, double-cropping, and higher plant population have the potential to increase the epidemic development of soybean disease and alter disease control strategies. Unless grown in a crop rotation, soybeans will suffer greater disease hazards witn minimum tillage as most foliar pathogens overwinter in crop residues left on the soil surface from the previous season. These pathogens are mostly destroyed by deep plowing because plant debris is decayed by soil micro-organisms. In double-cropped rotations where soybeans are planted late in the growing season, soybeans are exposed to pathogens at younger and more susceptible growth stages. Inoculum levels are higher due to the presence of spores from foliar pathogens that have infected and reproduced on early planted fields resulting in higher yield losses. At dense populations, a closed plant canopy results in a favorable microenvironment for the spread of foliar diseases.

Since these changes in soybean production intensify disease pressure from foliar pathogens, there is a need for research to identify immediate problems and future potential problems that result from changing cultural practices of soybean production. Since many pathogens are unpredictable in nature with regared to both temporal and spatial population dynamics and damage, a major priority should be developing methods to accurately predict epidemics.

Data collected from our soybean disease monitoring plots since 1977 have shown that brown spot, bacterial blight, bacterial pustule, downy mildew and soybean mosaic virus are endemic foliar diseases in Illinois. Of these diseases, brown spot is currently the most severe and is considered to be the most important foliar disease throughout the state. The lack of fundamental knowledge of epidemic development of these diseases in filed situations is a limiting factor in the development of efficient disease control strategies and in the prediction of future epidemic outbreaks. Hence, it is essential to develop a better understanding of the epidemic features of these diseases through analyses of epidemic progress, assessment of yield losses caused by individual or multiple diseases, and establishment of the relationship between disease severity and yield. The development of epidemic models is needed for the accurate assessment of disease problems, effective and efficient disease control strategies, and the prediction of future epidemics and yield losses.

In recent years, our work and that of others have shown that brown spot cause significant yield reductions and has the potential to be devastating. At present, there are no soybean genotypes available with resistance to the causal organism, *Septoria glycines*, that could be effectively incorporated into elite soybean cultivars. It is generally recognized that the most effective and efficient method of controlling diseases is the use of resistant cultivars, particularly when other methods such as fungicide applications are not economically feasible. Hence, there is a need to investigate the nature and expression of resistance in soybeans to *S. glycines* so that sources of resistance can be identified. Our work in this area has indicated that some cultivars exhibit tolerance in the field but results are inconsistent over years. The development of efficient and accurate methods for characterizing the expression of resistance and/or tolerance should be given a high priority in order to identify effective sources of resistance. Research designed to enhance the effectiveness and dependability of disease resistance in soybeans is of national importance and constitutes an essential part of our research program.

In contrast to the situation with *S. glycines*, sources of resistance to *Pseudomonas syringae* pv. *glycinea* (*PSG*), causal agent of bacterial blight, and *Xanthomonas campestris* pv. *glycines* (*XCG*), causal agent of bacterial pustule, are known. However, little information is available on the genetics of resistance. Polygenically inherited resistance to these two pathogens has never been investigated. Pathogenic variability of *PSG* has not been reported. Currently, most popular soybean cultivars are resistant to these two bacterial diseases. However, intensive cultural practices and the narrow germplasm base of soybean cultivars produce selection pressures which eventually will force the pathogen to genetically adapt to overcome resistance. There are many examples of gene-for-gene interactions between monogenically inherited resistance in plants and monogenically inherited virulence in pathogens. New races of the pathogen have suddenly developed, attacking crop which has a high level of resistance to the old races but has little or no defense against the new races. In these cases, adaptation in the pathogen required only the selection of a single gene for virulence to match a corresponding gene for resistance in the host cultivar. Thus, monogenic resistance is vulnerable to "breakdown". Resistance breakdown has occurred in soybeans. We found a new race of *Peronospora manshurica*, the causal agent of downy mildew, in the Illinois soybean disease -monitoring plots. This race was designated as Race 33 and was virulent on the soybean cultivar Union, which carries the gene *Rpm* for resistance to all other 32 races of *P. manshurica* previously described. Consequently, it is important to assess the pathogenic variability of pathogens.

The two bacterial organisms can be assessed by developing a set of suitable differential cultivars and by developing criteria by which various types and sources of resistance could be recognized. When appropriate, ways to enhance the durability of types of resistance that have a high probability of breaking down should be investigated.

Currently, a total of 33 races of *P. manshurica* have been indentified. The occurrence of *P. manshurica* over a wide geographic range throughout the world and diversity of *P. manshurica* races in the United States suggest that other physiological races may exist. Little is known about the genetic potential of this fungus to produce races or the ability of different races to survive and increase in nature. Therefore, it is important to monitor the pathogen population in the field and to identify resistance through the evaluation of the soybean germplasm.

Soybean mosaic virus (SMV) is one of the most prevalent viral diseases of soybeans in the world. Yield reductions up to 50% have been observed in SMV-susceptible soybeans. Several strains of SMV have been identified. Various sources of monogenic resistance to these SMV strains also have been found. Most of these sources were resistant to some, but not all of seven SMV strains tested. Recently, we have collected and identified several new sources of resistance from Korea that are effective against all previously known seven SMV strains. It is very important to continue our efforts to identify gene(s) conferring resistance in soybean collections and to determine inheritance of resistance. Breeding for resistant cultivars can be more effective and efficient when the genetic information on various sources of resistance is available.

Advancing technology in the field of molecular biology provides an opportunity to develop research on the molecular genetics of soybean -pathogen interactions. Generally, the functions of genes that determine the virulence or avirulence of pathogens are not well understood. Single genes often determine whether a race or strain of the pathogen is virulent or avirulent and whether a particular host plant is susceptible or resistant. Genetic constitution between host lines or pathogen strains can be revealed as segregating characters in genetic studies. The epistatic properties of resistance genes and avirulence genes impose an additional difficulty in the recognition of genetic control of pathogenicity. Transposon mutagenesis in bacterial organisms is proving to

be a useful technique for studying complex phenomena such as pathogenicity. Since mutations induced by insertion of transposons are known to be associated with a selectable phenotype (e.g., antibiotic resistance), they may be easily studied by conventional culture techniques without the tedious and expensive testing of plants. The cloning of a transposon-mutagenized gene often is simple and yields a probe which in turn permits the isolation of the wild-type gene. Knowledge of the molecular genetics of pathogenic specificity in soybean-pathogen system is lacking and needs to be investigated. Some species of *X. campestris*, e.g., *X. campestris* pv. *oryzae* and *X. campestris* pv. *malvacearum*, have been studied extensively for their pathogenic specificity. Techniques for similar studies are available for *XCG*. Although this is a long term research project, the research will yield new initiatives in soybean protection which can be based on fundamental properties of host-pathogen interactions.

## OBJECTIVES

The long-range goal of this project is to develop epidemiological models that can be used to plan efficient disease control strategies that will reduce losses in soybeans from foliar diseases, and to assess the existence, nature, and heritability of genetic resistance in soybean to foliar diseases for the development of more durable and effective disease resistance. Specific objectives of the research include: 1) development of models to characterize epidemic progress of brown spot, bacterial blight and bacterial pustule resulting from individual and multiple infections, and to develop reliable methods of estimating yield losses in soybeans caused by these diseases; 2) determination of the nature, expression, and mechanism of resistance in soybeans to *S. glycines* (brown spot) and *X. campestris* pv. *glycines* (bacterial pustule), and determination of the heritability of genetic resistance to SMV and downy mildew; 3) application of plant tissue culture in soybean improvement for brown spot resistance; and 4) identification and characterization of pathogenicity genes of *X. campestris* pv. *glycines*.

## APPROACH

**Objective** 1: Our previous work with the monitoring of foliar diseases (see Literature Review) showed that brown spot is one of the most prevalent foliar diseases in Illinois. Hence our efforts on epidemic research have centered on brown spot. Portions of our results have been published (see Literature Review). For the analyses of epidemic progress and the development of epidemic models to describe and predict disease development, our research efforts are concentrated on two diseases, bacterial blight and bacterial pustule. Different epidemic levels of bacterial blight and bacterial pustule are established by inoculating soybeans at various growth stages in replicated field plots. Several growth function models are applied to fit disease progress data and to select the best fitting model providing biologically meaningful information about the analysis and development of models for predicting epidemics. This information is useful in developing the most efficient control strategies through the achievement of an adequate level of resistance in soybean cultivars. For example, very high levels of monogenic resistance is not necessary if the levels of the disease threshold which will influence yield are known to be higher than that level of resistance. Field experiments are performed in replicated field plots to assess the effect of individual and multiple disease infection on soybean yield using brown spot, bacterial blight and bacterial pustule. The relationships between disease severity and loss in yield are characterized. Negative production functions are developed to estimate the loss in yield associated with various levels of severity for these three diseases.

**Objective** 2: Reaction of soybeans to a pathogen is usually classified by the phenotypic expression

of soybean plants. The phenotypic expression can be both qualitative and quantitative. Qualitative reactions are characterized by symptom development: no symptom versus symptom, or lesion types(e.g., small flecks versus chlorotic lesions). Quantitative reactions are characterized by lesion size, lesion number, or percentage of leaf area infected. Since 1977, we have evaluated more than 8,000 entries of the USDA World Soybean Germplasm Collection maintained at Urbana, Illinois, for sources of resistance to *S. glycines*. However, no significant differences in either qualitative or quantitative reactions of soybeans to *S. glycines* were found. Hence, it is necessary to use other methods to identify sources of resistance. I have begun to evaluate levels of brown spot tolerance (the ability of a soybean cultivar to sustain less yield loss due to disease than equally susceptible cultivars when disease is severe). In the experiments, two treatments, benomyl protected and inoculated plots, are main plots and each of 20 soybean cultivars from the maturity groups II and III are subplots. Disease severities in each plot are rated at 2-week intervals after inoculation. Yield data are obtained from each plot. Infection rate and the area-under-the-disease-progress-curve are calculated to apply various epidemiological parameters in determining levels of tolerance in soybean cultivars. Various sources of monogenic resistance in soybeans to *P. manshurica* and SMV have been identified, an inheritance of resistance to these two pathogens have been determined. A series of $F_1$, $F_2$, $F_3$, $BC_1$ and $BC_2$ progenies from each cross between soybeans susceptible and resistant to different races of *P. manshurica* and to different strains of SMV are planted in the greenhouse. Plants are being evaluated for their reactions to the pathogen to determine inheritance and to identify gene(s) conferring resistance in soybeans to different races of *P. manshurica* and to different strains of SMV.

**Objective** 3 : Our preliminary work has shown that most of the fungal culture media tested contain inhibitory substances to soybean tissue cultures and need to be modified to eliminate such inhibitory effects. Yeast extract, ammonium tartrate and mineral salts are some of the common substances found in the culture media which inhibit plant tissue in culture and require substitutions or modifications in media preparation. Pathotoxic substance(s) in the fungal culture filtrates can not be extracted by various organic solvents and can not be separated through dialysis. It is water soluble and pathotoxicity is not lost by heating. Some of these properties not commonly found in known pathotoxins, and suitable methods need to be developed to purify and identify the pathotoxin(s). The methods for initiation, maintenance, and subsequent regeneration of plants from soybean tissue cultures are available to investigate somaclonal variation for brown spot resistance. The efficiency of selecting resistant mutants could be greatly enhanced if plants can be regenerated through protoplast systems, but these cultures presently are incapable of plant regeneration. Attempts have been initiated to develop suitable media for protoplast cultures that would be more amenable to plant regeneration. The availability of non-pathogenic mutants of *S. glycines* provides on opportunity to investigate the molecular genetics of pathogenicity in this pathogen. Attempts are being made to develop a genetic transformation system of *S. glycines* for the molecular characterization of pathogenicity determinants.

**Objective** 4 : Our preliminary results using the cloned DNA fragments that complement NP1 as probes indicate that there are multiple complementation groups within the mutant populations that we have generated. These results support the contention that there are multiple factors involved and that their complete characterization require a long-term commitment of effort. Our intention, however, is to do a complete investigation of the pathogenicity genes we have in hand. The long term goal of the research is to determine the roles of the gene products in the disease. This involves

the characterization of plant factors that are responsible for susceptibility to *XCG*. Possible approaches to that problem can only be speculative until the above objectives are realized. At present, our only clue is the possible involvement of plant factors in the regulation of pathogenicity gene expression, and that avenue of research will be explored in this work. The regions responsible for restoration of pathogenicity have been defined and those regions are sequenced to provide putative amino acid sequences of the gene products. Comparison of the sequences to known DNA and amino acid sequences available in large data bases may provide a clue regarding functions of the products. Regulation of expression of the pathogenicity genes can be investigated by several approaches. A promotorless chloramphenicol acetyl transferase (CAT) gene can be inserted behind promoter regions in the pathogenicity genes, and expression of the CAT gene is investigated in a variety of culture conditions and *in planta*. DNA probes and antibodies to the gene products are used to study gene transcription and translation in infected plants and to localize translation products within the plant tissues.

If plant factors are found to be required for expression of pathogenicity genes, those plant factors are identified. Conservation of the pathogenicity genes isolated from *XCG* 8ra is investigated using the cloned genes as probes to detect DNA sequences in restriction digests of other *XCG* strains, other *Xanthomonas* pathovars, and in other bacterial pathogens of soybeans.

The achievement of these objectives would help to increase soybean production efficiency by reducing the deleterious effects of the major foliar disease through the accurate assessment of disease problems. The development of models to predict epidemics and yield losses and the development of improved methods of identifying, evaluating, transferring and utilizing different genetic sources of disease resistance are needed to more efficiently control soybean diseases. Results from epidemic analyses provide a better understanding of the development of brown spot, bacterial blight and bacterial pustule in the field, allow predictive measurement of epidemic development and serve as a guide for the direction of research efforts to minimize disease losses. Disease-loss appraisal represents an absolutely essential step because it is not possible to implement disease management strategies aimed at economic control unless economic loss can be measured. The most effective and efficient method of controlling soybean diseases is through the use of resistant cultivars. Therefore, information on the nature, expression and heritability of genetic resistance is important in utilizing various genetic sources of disease resistance for the efficient development of resistant soybean cultivars.

## LITERATURE REVIEW

Much work is underway in an effort to develop a better understanding of brown spot development in the field and to identify sources of resistance in soybeans to *S. glycines*. Brown spot of soybean, caused by *S. glycines* Hemmi (9), is one of the most common foliar diseases of soybeans in Illinois (34). The disease is present early in the growing season infecting the unifoliolates, progressing from lower to upper leaves, and prematurely defoliating plants (43). Brown spot causes yield losses in soybeans up to 8-12% in naturally infected plots (20, 36) and up to 8-34% in inoculated plots (20, 36, 37, 42, 45).

Considering the yield reductions in soybeans associated with brown spot, workers (19, 44) have evaluated thousands of plant introduction lines and cultivars in search of brown spot resistance. Although no resistance was found, a difference in disease reaction between green and yellow seeded soybean lines was described (19, 22, 42). Symptoms of brown spot on leaves of plants grown from the common yellow seeds appeared chlorotic with angular brown spots surrounded by yellow halos. On the other hand, plants grown from the less common green seeds produced a nonchlorotic reaction, angular brown spots without the sur-

rounding halo.

The green and yellow seeded soybean lines were studied for their potential as sources of brown spot resistance (19, 22, 44). Lim (22) found that severity, defoliation, and apparent infection rates were similar for three isogenic lines derived from Clark soybeans (Clark-L-1 (yellow), L64-2545 G $d_1d_2$ (green), and L62-1027 *cyt*-G (green)). Although there was a difference in yield among the isolines, the yield reduction after inoculation for each isoline (when compared with benomyl protected treatments for an isoline) was not different. This indicated that resistance or tolerance could not be characterized by the nonchlorotic reaction.

Differing disease reactions among some cultivars were reported. Although there was no difference in disease severity or area-under-the-disease-progress-curve (AUDPC) between Williams, an indeterminate cultivar, and Elf, a determinate cultivar, there was some indication that brown spot was more severe on Elf than Williams during the late reproductive stages (37). Vertical progress ((maximum height in nodes that brown spot symptoms can be found divided by total nodes of the plant) x 100) was greater for Elf than Williams. Though brown spot progressed to the same height from the ground for both the determinate and indeterminate cultivars, Elf sustained the greater seed weight reduction probably because of premature defoliation in the upper canopy. Although differences in brown spot severity, vertical progress, and AUDPC among the narrow row (17 cm) and wide row (50 or 76 cm) soybeans were not consistent, there was a trend of greater severity in wide row treatments (37). Row width did not affect yield or seed weight reductions. In contrast, another study (27) reported that narrow row soybeans (18 cm) had higher disease severities than wide row soybeans in early August, but severities were similar later in the season. The 18 cm rows produced 17% more yield than the 76 cm rows.

Differences in brown spot severity on early- and late-maturing cultivars have been observed (20, 34). Moderate severity during early reproductive stages and severe infections later is common. Several studies (3, 41, 45) were done to determine if these effects were due to plant or environmental differences.

High disease indices, which accounted for severity and defoliation, were obtained in plants inoculated at the second trifoliolate or at the full pod stages and were significantly higher than plants inoculated at flowering or beginning pod fill (3, 45). When plants were inoculated simultaneously at the different growth stages, the older inoculated leaves had higher severities than the younger leaves (3). These studies may indicate that plants may be susceptible to *S. glycines* infections primarily because of plant and leaf age.

Studies of environmental influences on disease development late in the season also were done. Moist periods following inoculation were important for symptom development (41). Simulating daily dew when plants were not subjected to immediate post-inoculation moist periods also increased disease. With post-inoculation moist periods, simulated dews every 3-4 days increased disease.

Brown spot severity was significantly lower in soybeans grown in rotated and plowed plots in Illinois (39). Brown spot severity was reduced by benomyl applications at R3 or at both R1 and R6 growth stages of soybeans (36). Timing was critical because increased infection rates resulted after the fungicide lost its effectiveness.

Yield reductions occurred primarily by reducing seed weight (35). Seed weight reductions were 8, 11 and 18% for the upper, middle, and lower canopy, respectively. In general, other yield components, such as number of pods per plant or number of seeds per pod, were not significantly different among inoculated, sprayed, naturally infected, and inoculated and sprayed treatments. The loss of leaf dry matter through defoliation was suggested to cause reduction in dry matter accumulation in pods.

Yield loss models of brown spot were developed to identify critical growth stages for disease or

predict yield loss with disease severity at a critical growth stage (20). Yield and seed size were negatively correlated with percentage leaf area diseased, with defoliation and with a combination of both parameters (20, 37, 45). In Illinois, the highest correlation coefficient for the cultivars Williams and Wells were between yield reduction and AUDPC and between yield reduction and disease severity at R6 (20). Predicted yield reductions were calculated by regression equations with severity at R6 or AUDPC as independent variables. Another study showed that severity at R5, vertical progress at R6, and AUDPC correlated best with yield and seed weight reductions (37).

Pathogenic variation in 15 isolates of *S. glycines* from 12 different states in the United States were evaluated in soybean field plots (13). Quantification of brown spot development in the field did not detect differences in pathogenic variability among isolates of *S. glycines*. This explains, ir part, why sources of resistance to *S. glycine* were not found in more than 7,000 soybear germplasms evaluated. Our primary work (11) on the survival of *S. glycines* indicated that *S. glycines* associated with soybean leaf tissue could overwinter on the soil surface in central Illinois.

Because of similar environmental conditions influence the development of brown spot and bacterial blight, studies were done to determine the interaction of the two pathogens (1, 8, 42). One report showed that brown spot disease severities were higher on plants inoculated simultaneously with *S. glycines* and *PSG* than on plants where both pathogens were singly inoculated on different portions of the same leaf. The content of chlorophyll a and b was lower in the infected leaf tissues where *S. glycines* and *PSG* were inoculated than when the two pathogens were inoculated on separate leaves (8). Higher brown spot severities were obtained from plants infected with *S. glycines* alone than plants infected with *PSG* and *S. glycines* in field experiments (30, 42). There was no difference in yield if *S. glycines* was inoculated alone or in combination with *PSG*.

Soil temperature and moisture greatly influenced survival of *PSG* associated with soybean leaves (32). The effect of soil moisture was greater at 4 C than at 12 or -12C. Thus, *PSG* associated with soybean leaf tissue could overwinter on the soil surface in central Illinois if the weather is cold and dry (31).

Mathematical modeling of epidemic progress is necessary to quantify the effect of various factors influencing disease development. Serious defects in estimating a disease parameter common to several epidemic models used extensively by plant pathologists were identified using data on the development of soybean bacterial blight (33). New disease parameters are proposed to accurately describe and compare plant disease epidemics.

The spread of *P. manshurica* from an initial disease focus in a field plot was analyzed by using a regression model (18). The downy mildew severity gradients at 30 and 44 days after inoculation agreed with the slope of the theoretical line from a point source of inoculum. The level of disease spread throughout the plot was 21% at 58 days. A large number of spores trapped corresponded to periods of no precipitation. Frequent rainfalls, low wind speed (0-9 km/hr), and aging of soybean leaves all resulted in very low numbers of spores trapped despite favorable ranges of temperatures, relative humidity and wind direction.

In 1981, a new race of *P. manshurica* occurred in the Illinois soybean disease monitoring plots (23, 26). The new race was designated as race 33 and was virulent on the soyban cultivar Union, which carries the gene *Rpm* for resistance to the 32 previously described races of *P. manshurica*. Five cultivars (Pridesoy, Palmetto, Kabott, Ogden, Acadian) from a set of differential cultivars and three additional cultivars (Fayette, Tracy, PI 88788) were resistant to race 33. A preliminary study (25) indicated that the gene for resistance in Fayette to race 33 segregates independently from the *Rpm* gene of Union.

Various sources of SMV resistance have been identified in soybeans (5, 6). Most sources were

resistant to some, but not all seven, strains of the SMV. Resistance to some SMV strains that produce mosaic symptoms was shown to be conditioned by a single dominant gene (14, 15, 40), whereas resistance to a severe isolate, SMV-N (which produces necrotic symptoms on susceptible soybeans) was shown to be conditioned by a single recessive gene (16). Recently, several soybean lines collected from Korea were identified as resistant to all previously known seven SMV strains (21, 24). The soybean line OX670, a selection from a cross involving resistant cultivar Raiden (PI 360.844), was also shown to be resistant to all seven strains (4). Resistance in OX670 to strains G7 was conditioned by a dominant gene *Rsv2* (4). Resistance to strains G2 and G3 in PI 96983 was conditioned by a dominant gene *Rsv* (14). Resistance in PI 486.355 to all seven strains was conditioned by a dominant gene which was different from gene *Rsv2* (24).

The genetics of photopathogenic bacteria were reviewed in 1979 (17). Several chapters of a book (28) published in 1982, and a number of reviews and book chapters on the molecular biology of Ti plasmis (2, 7, 10, 38) are available. Also, the genetics of pathogenicity and selected aspects of the molecular biology of these organisms and their plasmids have been extensively reviewed (29). Techniques for the investigation of molecular genetics of pathogenic specificity in a soybean *XCG* system are available in the literature cited above.

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