GENETICS OF REACTION TO PEANUT MOTTLE VIRUS IN SOYBEANS

by

Emerson Russell Shipe

Dissertation submitted to the Graduate Faculty of the
Virginia Polytechnic Institute and State University
in partial fulfillment of the requirements for the degree of
DOCTOR OF PHILOSOPHY
in
Agronomy

APPROVED:

Dr. G. R. Buss, Chairman

Dr. C. W. Roane

Dr. T. M. Starling

Dr. S. A. Tolin

Dr. T. J. Smith

June, 1978

Blacksburg, Virginia

ACKNOWLEDGEMENTS

The author expresses gratitude to Dr. G. R. Buss for serving as chairman of his graduate committee, for advice and guidance in his research, and for many helpful comments during the preparation of this manuscript. Sincere appreciation is also expressed to Dr. Sue Tolin for supplying the virus strain and for making available the plant virology laboratory facilities. He is also grateful to Drs. C. W. Roane, T. M. Starling, and T. J. Smith for their counsel and constructive criticism of this manuscript.

Special acknowledgements go to , Superintendent, Eastern Virginia Research Station, and , technician, for their assistance with the field and greenhouse work. Gratitude is also expressed to for careful typing of the manuscript and to , and the Department of Agronomy for financial support during the period of graduate study.

Finally, special thanks and appreciation is expressed to my wife, , for her patience, understanding, and encouragement.

TABLE OF CONTENTS

Table of Contents, continued

LIST OF TABLES

List of Tables, continued

List of Tables, continued

LIST OF FIGURES

I. INTRODUCTION

The soybean, Glycine max (L.) Merrill, is grown widely throughout the world and is a vital crop in the U.S.A. It is used as a primary source of vegetable oil for human consumption and protein for animal feed. Currently there is great interest in its potential as a source of protein for humans. Despite a concentrated research effort over the past several years, the U.S. average yield has increased only 0.3 bushel per acre per year (Caldwell, 1973).

A contributing factor to such minimal average yield increases has been the increase in number and severity of diseases. It is known that over 100 pathogens affect soybeans and 35 of these pathogens are of economic importance. Twelve viruses have been found to occur naturally in soybeans (Sinclair and Shurtleff, 1975) and seven viral diseases are widespread and of major consequence (Dunleavy, 1973).

Peanut mottle virus (PMV) occurs naturally in peanuts (Arachis hypogaea L.), soybeans, and a few leguminous weed species (Kuhn, 1965; Kuhn et al., 1972). It is a widespread disease on soybeans in areas of the southeastern U.S. where peanut and soybean crops are grown in close proximity. The disease has also been reported occurring naturally on soybeans in Australia and East Africa (Demski and Kuhn, 1977). Researchers in Georgia and Virginia have found that PMV on soybeans significantly reduces yield (Demski and Kuhn, 1977; Roane et al., 1978).

An effective method for controlling virus diseases is the development of cultivars that are genetically resistant. In order to develop PMV-resistant soybean cultivars, sources of resistance must be identi-

fied. Secondly, an understanding of the genetic control of resistance must be gained so that plant breeders can manipulate the genetic factors and incorporate them into agronomically acceptable cultivars. Multiple and diverse resistance factors should be identified and utilized so as to obtain a broad base of genetic resistance rather than relying on only one source. Resistance based on multiple genes and/or alleles would not be as easily or quickly overcome by genetic changes in the virus pathogen.

The objectives of the present study were (1) to study the inheritance of reaction to PMV in soybeans; (2) to determine the allelic relationship of genes for resistance from various germplasm sources; and (3) to screen a part of the soybean Plant Introduction germplasm collection for resistance to PMV.

II. LITERATURE REVIEW

History and Importance of Peanut Mottle Virus

Occurrence on Peanuts

PMV was first described and named in 1965 by Kuhn. Over a four-year study period, he found the virus occurred quite commonly in Georgia peanut production areas. He also obtained PMV-infected peanut plants from Holland, Virginia. The name "peanut mottle virus" was proposed since this virus appeared to be unrelated to any previously described viruses. PMV has been observed in peanut plants in North Carolina for several years (Sun, 1974). A mild strain (PMV-M) is widespread almost every year but symptoms are not obvious. A severe strain (PMV-S) has a more visible effect on peanuts but does not occur as frequently as the mild strain. PMV has subsequently been reported from several other areas in the U.S.A. where peanuts are commercially grown (Demski et al., 1975).

Schmidt and Schmelzer (1966) reported a virus on peanuts in Bulgaria with physical characteristics similar to PMV. They concluded that it was PMV or a closely related virus. In 1969, Herold and Munz in Venezuela reported on a virus first observed in 1965 which causes leaf mottle in peanuts. A study of symptoms and physical properties showed the virus to be identical to PMV as described by Kuhn. PMV was reported from Australia in 1970 by Behncken who studied transmission, host range, and physical properties of the virus. A previously undescribed mosaic disease on peanuts was reported by researchers in West Malaysia (Ting et al., 1972). Although conceding that the virus is

very similar to PMV based on symptom expression, they maintained that it was a distinctly different virus due to its different host range and transmission characteristics.

Based on particle morphology, serology, host range and reaction, physical properties, and transmission characteristics, Bock (1973) concluded that virus isolates from both peanut and soybean plants in East Africa were PMV. The disease was reported to be common and widespread in Uganda, Tanzania, and Kenya. PMV has also been reported on peanuts and garden peas (Pisum sativum L.) in Japan (Inouye, 1969).

In the field, PMV causes a mild mottle on peanut leaves that is sometimes difficult to observe visually. Plant growth may be slightly reduced but the overall effect of the disease on peanuts is not sufficient to make it highly visible in the field (Kuhn, 1965). Kuhn and Demski (1975) presented a summary of research on yield loss in peanuts due to PMV. A mild strain of PMV caused an average reduction in seed yield of 25% in seven greenhouse tests and 22% in six field tests. A severe mosaic strain of PMV has been observed in North Carolina (Sun and Hebert, 1972) and a necrosis strain in Georgia (Paguio and Kuhn, 1973b). These strains occur much less frequently than the mild strains of PMV but yield losses of 70% and 68% have been recorded for the severe mosaic and necrosis strains, respectively (Kuhn and Demski, 1975). A Georgia field survey in 1973 (Paguio and Kuhn, 1974a) showed PMV to be present in all of the 117 fields observed in 45 counties and approximately one-half of the fields had significant economic losses (>$10/acre).

Occurrence on Soybeans

Although previously described as an experimental host of PMV (Kuhn, 1965), soybeans were first reported as a natural host in 1972 by workers in Georgia (Kuhn et al.). The PMV soybean isolate was identified by host range and serological tests and was similar to the mild strain of PMV previously described by Kuhn (1965). It was isolated from seven counties and appeared to be widespread in the Georgia soybean-peanut production area. Some soybean fields with 25-50% of the plants infected with PMV have been observed (Demski and Kuhn, 1977). The natural occurrence of PMV in soybeans has also been reported in East Africa (Bock, 1973) and Australia (Behncken and McCarthy, 1973). Ting et al. (1972) reported that the virus which was isolated from peanuts in West Malaysia also occurred naturally on soybean plants. However, they believed the isolate to be different from PMV based on host range and transmission studies.

In the U.S.A., PMV on soybeans has also been reported in Virginia (Tolin et al., 1974) and South Carolina (Demski and Kuhn, 1977). Of the eight viruses that have been found on soybeans in Virginia, PMV appears to be the most economically important (Roane and Tolin, 1977). Surveys conducted in 1973, 1974, and 1975 showed that PMV in soybeans is widespread in Southeastern Virginia where peanut and soybean crops are grown in close proximity.

Several studies have been conducted to determine the effect of PMV infection on soybean yields. It is difficult to estimate actual economic losses due to PMV because of factors such as time of infection and disease incidence. Roane and Tolin (1974) reported yield losses due

to PMV infection for four soybean cultivars; 'Essex', 'Lee', 'Wye', and 'York'. Mechanically inoculated and uninoculated plants were compared for symptomatology, growth, yield components, and total yield. Both a mild (PMV-M) and a severe (PMV-S) strain were used. The average yield reduction caused by PMV-M and PMV-S in all four cultivars was 4% and 22%, respectively. Since York is resistant to PMV, including its yield in the overall averages tended to reduce the average yield loss.

In a recent experiment conducted at Holland, Virginia, Roane et al. (1978) determined the effect of a severe strain of PMV on 25 adapted soybean cultivars. They reported a reduction in seed quality and a mean yield reduction of 44% for 21 PMV-susceptible cultivars. The reduction in yield appeared to be affected most by a reduction in seed number since seed weight was only reduced by 20%.

Workers in Georgia (Demski and Kuhn, 1977) found significant yield reductions on two soybean cultivars, 'Hampton 266 A' and 'Jackson', due to PMV infection. Yield reduction varied from 5 to 28% in the tests conducted from 1972 to 1975. Plant height was reduced an average of 6% and weight per 100 seed was significantly less in three of six experiments. For the same two cultivars it was found that PMV infection increased total seed protein content and decreased seed oil content. Demski and Jellum (1975) reported the following yield losses for soybean plants doubly infected with PMV and other viruses: PMV-cowpea chlorotic mottle virus, 46%; PMV-soybean mosaic virus, 78%; and PMV-tobacco ringspot virus, 80%. The negative effect of a double virus infection on yield was additive or less than additive for each of the

three virus combinations. Seed produced on PMV-infected soybean plants show streaking of hilum pigments, also called mottling, which may substantially reduce the market value of the crop (Roane and Tolin, 1974).

Characteristics of Peanut Mottle Virus

Transmission, Epidemiology, and Host Range

Over a three year period, peanut mottle virus was found to be one of the most prevalent virus diseases of soybeans in Georgia (Demski and Kuhn, 1974). Generally, it has been found in soybean in areas where both peanuts and soybeans are grown (Bock, 1973; Demski and Kuhn, 1974; Tolin and Roane, 1975). Incidence of PMV in soybean is high when soybeans are grown near infected peanut fields and/or when soybeans follow peanuts. PMV-infected volunteer peanut plants provide a source of inoculum for subsequent infection of soybean plants.

The source of primary inoculum of PMV is believed to be infected peanut seed (Paguio and Kuhn, 1974b). Several investigators have reported seed transmission of PMV in peanuts ranging from 0.001 to 20% (Behncken, 1970; Bock, 1973; Sun, 1974; Paguio and Kuhn, 1974b). It has also been shown to be seed transmitted in *Phaseolus vulgaris* L. (Behncken and McCarthy, 1973). Studies of seed transmission in ten soybean cultivars have shown that peanut mottle virus is not seed-borne (Demski and Harris, 1974).

PMV has been isolated in the field from the following weed species; *Cassia tora* L., *C. leptocarpa* L., and *C. occidentalis* L. However, field observations led to the conclusion that PMV moves from peanut to the *Cassia* spp. and not vice versa (Kuhn, 1965). Behncken and McCarthy

(1973) found PMV occurring naturally on peanuts, soybeans, garden peas, and navy beans (Phaseolus vulgaris) in Australia. An abbreviated form of the proposed PMV transmission cycle (Demski and Kuhn, 1977) is shown in Figure 1.

The virus can be transmitted mechanically by inoculation with sap from infected plants (Kuhn, 1965) and transmitted naturally by aphids (Paguio and Kuhn, 1976). It has been shown that the rate of natural spread of PMV is greater in peanuts than in soybeans, suggesting a vector preference for peanuts. It was also reported that soybeans become resistant with age to PMV infection by both natural and mechanical inoculation (Demski, 1974). It has been shown that Aphis craccivora (Koch.) and Myzus persicae (Sulz.) can both transmit PMV from peanuts to soybeans (Paguio and Kuhn, 1976). From several studies, it has been shown that the virus is transmitted in a stylet-borne, nonpersistent manner in peanuts and that the two previously mentioned species are the most common aphid vectors (Herold and Munz, 1969; Behncken, 1970; Bock, 1973; Paguio and Kuhn, 1976). Both species transmitted all known strains of PMV in peanuts except for the necrosis strain (Paguio and Kuhn, 1973b). Garden peas proved to be as adequate an acquisition host as peanuts (Paguio and Kuhn, 1976).

Kuhn (1965) reported that 16 species within the family Leguminosae were susceptible to PMV when mechanically inoculated. Phaseolus vulgaris L. 'Topcrop' was found to be a suitable assay host for PMV. Local lesions are produced on leaves of all P. vulgaris cultivars inoculated with PMV. Nicotiana clevelandii A. Gray, a non-legume, has also been reported as an artificial host (Behncken, 1970).

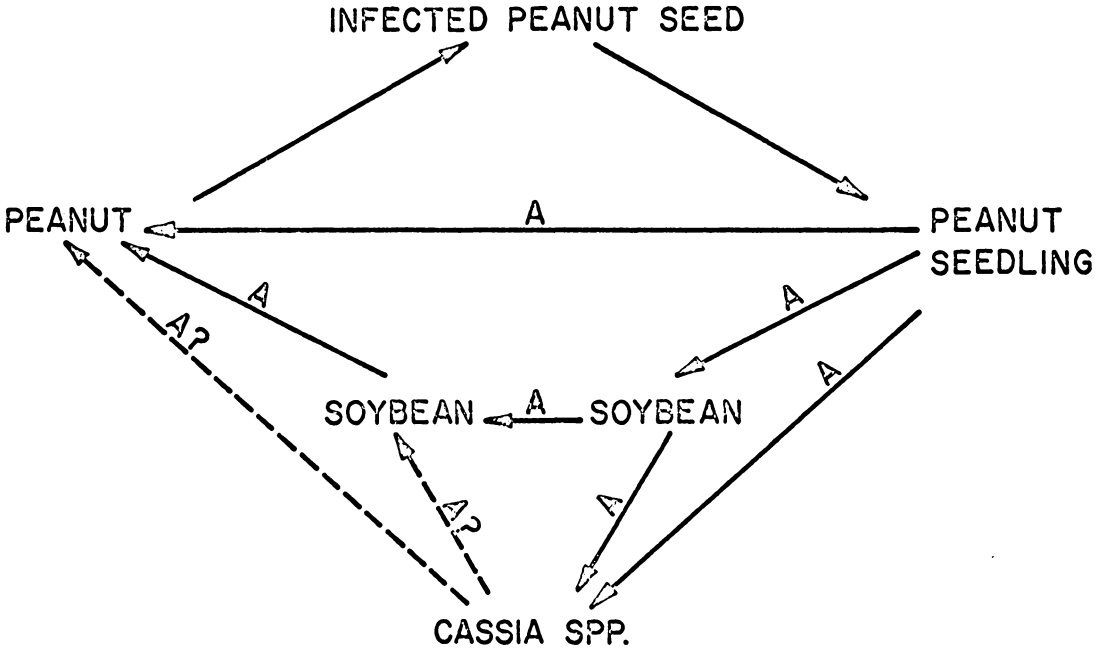

Figure 1. Proposed transmission cycle of peanut mottle virus (Demski and Kuhn, 1977).

Physical Properties

Peanut mottle virus is a member of the potyvirus group of viruses, contains single-stranded RNA, and is serologically distinct from all other potyviruses. The virus particles are flexous rods 740-750 nm long. About 6% of the particle weight is ribonucleic acid (Bock and Kuhn, 1975). The dilution end point of the virus was found to be between 10^{-3} and 10^{-4} and the thermal inactivation point between 60 and 65 C. Longevity *in vitro* at 25 C was approximately two days. Non-buffered sap from PMV-infected soybeans was infectious. However, mechanical inoculation was facilitated by the use of a phosphate buffer (0.01-0.05 M) at pH 8.0 (Demski and Kuhn, 1977). Five different strains of PMV have been found in peanuts based on symptom expression (Paguio and Kuhn, 1973b). A severe strain has been reported on soybeans in Virginia (Tolin et al., 1974). However, all PMV isolates from soybeans in Georgia have caused mild mottle symptoms in peanuts (Demski and Kuhn, 1977). Procedures for the purification of peanut mottle virus have been described by several investigators (Sun and Hebert, 1972; Paguio and Kuhn, 1973a; Bock, 1973; Tolin et al., 1974).

Disease Symptoms of Peanut Mottle Virus on Soybeans

In 1965, Kuhn reported the occurrence of a general mottle on soybean plants mechanically inoculated with sap from PMV-infected peanut plants. When PMV was observed in the field on naturally infected soybean plants (Kuhn et al., 1972), the most conspicuous symptom was a general mosaic, very similar to symptoms caused by other viruses on soybeans. When soybean seedlings were mechanically inoculated, small

chlorotic areas appeared on the first two trifoliolate leaves 6-9 days after inoculation. The chlorotic areas were discontinuous with small dark green islands occurring. Symptoms occurring on later developing trifoliolate leaves gave a general mosaic pattern. Kuhn et al. (1972) concluded that the soybean isolate was a mild strain of PMV (PMV-M), identical to the previously described peanut isolate (Kuhn, 1965). PMV may cause chlorotic or necrotic local lesions and mild to severe leaf curling on some soybean cultivars (Bock and Kuhn, 1975). Ringspots may sometimes form on the third and fourth trifoliolate leaves. As mentioned previously, agronomic characteristics such as plant height, number of pods per plant, weight per 100 seed, and seed quality may be affected unfavorably (Roane and Tolin, 1974; Demski and Kuhn, 1977).

Inheritance of Disease Resistance in Crops

Early Studies

One of the earliest recorded observations of crop varietal difference to disease susceptibility was made by Theophrastus (371-286 B.C.). Several investigators during the 1800s noted differences in disease between varieties of economic crops (Walker, 1957). The first report of Mendelian inheritance of disease resistance was made by Biffen in 1905. He showed that the resistance of 'Rivet' wheat (*Triticum turgidum* L.) to yellow rust (*Puccinia striiformis* West.) was determined by a single recessive gene.

Two terms used to describe the number of genetic factors conditioning disease reaction are oligogenic and polygenic. Oligogenic resistance is determined by one or a few genes which have easily

identifiable effects. Polygenic resistance is determined by many genes each of which has a small effect on overall resistance. Oligogenic resistance is usually determined by single dominant genes but there are several examples of resistance controlled by recessive genes (Day, 1974). A few reports have postulated that disease resistance is controlled by gene dosage in the host plant (Lupton and Macer, 1962; Dunn and Namm, 1970; Hooker and Saxena, 1971). The hypothesis is that disease resistance is governed by the level of "gene product" produced by the gene for resistance. Based on this hypothesis, one would expect an intermediate disease reaction in heterozygotes.

Allard (1960) states that the number of reports of disease resistance controlled by several genes is probably less than the number actually found due to the reluctance of researchers to publish data that do not fit a simple genetic ratio. A recent survey of all disease resistance studies published since 1912 showed that only 6.5% of the papers described polygenic resistance (Person and Sidhu, 1971). Day (1974) states that one reason for the predominance of oligogenic reports is the speed and convenience of screening seedlings in the greenhouse which is more likely to uncover resistance governed by one or a few genes.

In the past, many reports of disease resistance have identified only one source of resistance and then elucidated its inheritance based on F_2 and F_3 segregation ratios from crosses between resistant and susceptible cultivars or lines. Relatively few genetic studies in soybeans have attempted to identify more than one gene for disease resistance and to describe the relationships between such genes. Recent investiga-

tions have shown that currently grown cultivars of several major crops in the U.S.A. are derived from a relatively small number of ancestral cultivars and/or introductions. The result is undesirable genetic uniformity and the consequent reliance upon only one source of resistance to a given disease (Horsfall et al., 1972). Thus, it has become increasingly apparent that both new and multiple sources of resistance to a given disease should be identified and exploited.

Differentiating Resistance Genes

Day (1974) describes two methods for differentiating genes for resistance to a specific disease. The first method is the use of different races of the pathogen to "eliminate" previously recognized genes for resistance. Only "new genes" which are resistant against all known races of the pathogen will be selected. It is known that some resistance genes are closely linked or allelic (Hooker and Saxena, 1971) and can only be identified by the use of different races of a pathogen. The second method is a genetic test which involves the hybridization of appropriate lines and subsequent testing of the F_2 and F_3 generations for the segregation of new genes. Single chromosome substitution has also been used in wheat breeding as a method of mapping resistance genes (Riley and Macer, 1966).

There are many reports of investigations in which the hybridization method mentioned previously has been used to identify different genes for resistance. Briggs (1926) identified two dominant genes in 'Hussar' wheat (Triticum vulgare Vill.) for resistance to Tilletia tritici (Bjerk.) by analyzing F_2 and F_3 data from crosses between resistant

cultivars and between resistant and susceptible cultivars. Baker (1966) observed a 9 resistant (R):7 susceptible (S) F_2 genetic ratio when an oat cultivar 'Bond' (Avena sativa L.) which is resistant to crown rust (Puccinia coronata Cda.) was crossed to a susceptible cultivar. He isolated each of the dominant, complementary genes from Bond into two separate lines, crossed the two lines, and again observed a 9R:7S F_2 ratio. Roane (1962) identified four genes for resistance to race 4 of Puccinia hordei Otth. He made all possible crosses between nine resistant barley cultivars (Hordeum vulgare L.) and then crossed each resistant cultivar to a common susceptible cultivar. He observed 15R:1S and 63R:1S genetic ratios in the F_2 generations of some crosses between resistant cultivars indicating the presence of resistance genes at different loci in the parental cultivars.

Genetics of Resistance to Viral Diseases

Non-legume Crops

In tobacco (Nicotiana tabacum L.), partial resistance to tobacco mosaic virus (TMV) has been shown to be conditioned by two independent recessive genes in the cultivar 'Ambalema' (Nolla, 1938). Holmes (1938) identified a dominant gene for resistance in Nicotiana glutinosa L. and introduced it into cultivated tobacco. Later work (Holmes, 1960) showed that two independent, incompletely recessive genes isolated from a tobacco plant introduction, T.I. 245 (N. tabacum), also gave partial resistance to TMV. Holmes (1960) combined the three sources of resistance into a single line of tobacco which gave a very high order of resistance to TMV.

Cirulli and Alexander (1969) found that resistance in tomato (Lycopersicon peruvianum [L.] Mill.) against five TMV strains was controlled by a single dominant allele designated $Tm\text{-}2^a$. An interesting temperature effect on symptom expression in segregating populations was also reported. At 15-17 C in the greenhouse, homozygous and heterozygous resistant F_2 plants from a resistant X susceptible cross showed no disease symptoms resulting in a 3R:1S ratio. At 26-28 C, heterozygous F_2 plants exhibited a mild to severe necrosis (N) resulting in a 1R:2N:1S ratio. The occasional occurrence of necrotic plants in non-segregating, homozygous resistant progenies was also observed.

Koelle (1961) discovered a resistant mutant in a tobacco cultivar that was susceptible to potato virus Y (PVY). It was later reported (Silber et al., 1965) that the mutant was tolerant to PVY rather than resistant. Edmondson (1972) isolated two tobacco introductions that were tolerant to PVY. He found tolerance to be controlled by an incompletely dominant gene in T.I. 1406 and by a recessive gene in T.I. 1442. The two genes for tolerance were found to segregate independently.

Suneson (1955) identified a recessive gene in barley which conditioned an intermediate level of tolerance to barley yellow dwarf virus (BYDV). Resistance to BYDV was found to be controlled by one incompletely dominant gene in ten Ethiopian barley varieties based on tests of F_2 and F_3 generation material from resistant X susceptible and resistant X resistant crosses (Damsteegt and Bruehl, 1964). Schaller et al. (1964) confirmed the results of Damsteegt and Bruehl and identified the resistance gene in 16 barley cultivars of Ethiopian origin. Some cultivars have been found to be resistant in one area but susceptible

in other areas (Arny and Jedlinski, 1966; Bruehl, 1961).

Numerous studies have been conducted to determine the inheritance of resistance to maize dwarf mosaic virus (MDMV) in corn (Zea mays L.). Researchers have found resistance to MDMV to be partially dominant and controlled by relatively few major genes (Loesch and Zuber, 1967; Josephson et al., 1967; Dollinger et al., 1970). Experiments utilizing a diallel analysis to determine different types of genetic effects have shown that variation in resistance to MDMV is largely due to additive genetic effects (Johnson, 1971; Loesch and Zuber, 1972; Zuber et al., 1973). Significant nonadditive genetic effects have been found in crosses involving extremely susceptible inbred lines.

However, recent studies have shown that the disease called maize dwarf mosaic virus is actually a disease complex involving MDMV and maize chlorotic dwarf virus (MCDV) (Bradfute et al., 1972; Nault et al., 1973; Louie et al., 1974). Preliminary findings by Findley et al. (1976) indicate that resistance to five strains of the MDMV component of the MDM disease complex is controlled by a single dominant gene. Recognition and separation of the two specific diseases should facilitate genetic studies to determine the inheritance of resistance for each disease.

Legume Crops Other Than Soybeans

Pea common mosaic virus (PCMV) has long been recognized although it is not economically important on garden peas. Data from crosses between resistant and susceptible cultivars indicated that a single recessive gene controlled resistance to PCMV (Cousin, 1965). Resistance to pea

enation mosaic virus (PEMV) was found to be conditioned by a single dominant gene (Schroeder and Barton, 1958).

Bean yellow mosaic virus (BYMV) or bean virus 2 (BV2) is a common disease on peas. The virus is very similar to PCMV which is considered by some researchers to be a strain of BYMV (Hagedorn, 1973). Resistance to BYMV in peas is conditioned by a single recessive gene (Yen and Fry, 1956; Johnson and Hagedorn, 1958). It was noted that symptom expression was delayed in susceptible heterozygotes. Schroeder et al. (1966) found that symptom expression in F_2 heterozygotes from similar crosses was affected by temperature. In F_2 generation plants, resistance was dominant at air temperatures of 18 C or below and recessive at air temperatures of 27 C or above. Homozygous resistant and homozygous susceptible plant reactions were not affected by temperature changes. Heterozygotes could be separated from homozygotes by controlling the air temperature.

Zaumeyer and Meiners (1975) reviewed genetic studies on resistance of common bean (Phaseolus vulgaris) to bean yellow mosaic virus (BYMV). Both recessive and dominant genes have been reported to give resistance to BYMV. Phaseolus coccineus L. is generally considered to be the original source of resistance. The single, dominant gene for resistance derived from P. coccineus has been found to resist more BYMV strains than any of the resistance genes derived from P. vulgaris (Baggett and Frazier, 1957).

Bean common mosaic virus (BCMV) is almost nonexistent in the U.S.A. at present since most bean cultivars developed since 1940 are resistant (Silbernagel and Zaumeyer, 1973). Resistance in most U.S. snap bean cultivars is controlled by a single dominant gene derived from the

cultivar 'Corbett Refugee' (Zaumeyer and Meiners, 1975). Another cultivar, 'Robust', has been widely used as a source of resistance mainly in dry bean cultivars. Resistance to BCMV in Robust is conditioned by a recessive gene (Ali, 1950). Ali crossed Robust to cultivars carrying resistance derived from Corbett Refugee. He observed a 13R:3S genetic ratio in the F_2 generation. Ali explained the genetic results by proposing the presence of two gene pairs which control the resistant reaction and act with dominant and recessive epistasis.

Drijfhout (1975) has tentatively proposed a genetic model for resistance to BCMV which is based on the gene-for-gene concept (Flor, 1956). His complex genetic model is based on interactions of resistance genes at four loci and can account for 16 different BCMV strains, seven of which are currently recognized by Drijfhout. He has suggested standardization of all BCMV strains by the use of standardized differential bean cultivars.

Genetic resistance to peanut mottle virus (PMV) in peanut has not yet been found (Kuhn and Demski, 1975). A screening experiment (Kuhn et al., 1968) failed to uncover any resistant material in the 37 peanut cultivars and 428 plant introductions tested. However, some tolerant plant introductions have been identified (Kuhn and Demski, 1975).

Soybeans

Soybean mosaic virus (SMV) occurs on soybeans worldwide and is considered to be one of the most important soybean diseases in many areas. The virus is seed-borne and reduces seed yield as well as lowering seed quality (Sinclair and Shurtleff, 1975). Workers in Japan

(Koshimizu and Iizuka, 1963) reported that the inheritance of resistance to SMV in soybean was controlled by a single dominant allele based on segregation ratios observed in the F_2 generation of R X S crosses. However, one cross between resistant and susceptible cultivars produced F_1 plants which were susceptible. The F_2 progeny from the cross segregated 9S:7R.

Ross (1969) found that the cultivar 'Ogden', PI 96,983, and PI 170,893 were resistant to seven different isolates of SMV. Kiihl (1976) reported on the inheritance of resistance to SMV strains 'SMV-1' and 'SMV-1-B', a mutant strain of SMV-1. Based on field inoculation tests, he found that 35 of 71 F_1 plants derived from R X S crosses developed top necrosis after inoculation with SMV-1. Some heterozygous F_2 plants also developed top necrosis. He considered the necrotic response to be a hypersensitive reaction in resistant plants. Conover (1948) reported a similar hypersensitive necrotic reaction in 10-20% of Ogden plants inoculated with SMV.

Kiihl (1976) proposed the following multiple allelic series with the alleles listed in decreasing order of dominance: Rsv, carried by PI 96,983; rsv^1, carried by Ogden (and probably D71-9966 and D72-7842); rsv, carried by the susceptible types tested. Rsv gives resistance to both SMV-1 and SMV-1-B. The allele rsv^1 gives resistance to SMV-1 but Ogden shows necrosis when mechanically inoculated with SMV-1-B.

Boerma et al. (1975) made four crosses between soybean cultivars known to be resistant and susceptible to cowpea chlorotic mottle virus-soybean strain (CCMV-S). Based on greenhouse inoculation tests performed on the F_1, F_2, and F_3 generations, resistance was determined to

be controlled by a single dominant gene. Agreement in classification of the F_2 generations of reciprocal crosses indicated no cytoplasmic or maternal effect on the resistant reaction.

Seventy soybean cultivars and breeding lines were screened by Demski and Kuhn (1975) for resistance to peanut mottle virus (PMV) in the greenhouse. Fourteen genotypes, each of which had less than 2% susceptible plants, were rated as PMV resistant. Heat treatment (36 C) of three resistant cultivars inoculated with PMV resulted in a breakdown of resistance in one cultivar, 'Hardee'. A low concentration of PMV was isolated from inoculated primary leaves but no virus was found in the trifoliolate leaves.

A study of the inheritance of resistance to PMV in soybean was conducted by Boerma and Kuhn (1976). The methods used were similar to those used in the CCMV-S study previously described. Four R X S crosses were made involving two resistant (Dorman and CNS) and three susceptible cultivars (Ransom, Bragg, and Pickett). Plants in the F_1, F_2, and F_3 generations were inoculated and scored for reaction to PMV. A total of 24 F_1 plants from the four crosses were all resistant. An observed 3R:1S ratio in the F_2 generation indicated that a single dominant gene controlled the resistant reaction. Additional screening of F_3 lines from resistant F_2 plants confirmed the hypothesis. The cultivar 'Bragg' was used in both the CCMV-S and PMV studies. It is resistant to CCMV-S and susceptible to PMV indicating that resistance to the two viruses is not controlled by the same gene.

III. MATERIALS AND METHODS

Differentiation of Genes for Resistance to Peanut Mottle Virus

Propagation of the Virus and Inoculation Procedure

The PMV strain used throughout the study was isolated from soybeans grown at Holland, Virginia, 1974, and its identity confirmed by host range and serological tests (S. A. Tolin, personal communication). It was identified as a severe strain of PMV and labeled PMV-S/V74S. The strain was obtained from Dr. Tolin in November, 1975, as isolate number 74-80/81. For five months the virus was maintained in the greenhouse on the soybean cultivar 'Lee'. For the remainder of the study, one cultivar ('Hood') and six breeding lines (V73-166, V73-175, V73-178, V73-224, V73-228, and V73-741) were used as propagation hosts. These seven PMV-susceptible types are resistant to SMV (C. W. Roane, unpublished data). SMV is seed-transmitted in soybeans and produces plant symptoms that are very similar to those produced by PMV. Thus, SMV-resistant lines were used to propagate the PMV strain to insure that no SMV was introduced through infected seed.

Transfers to host soybean seedlings were made every 18-24 days. During 1977 and 1978 the virus was periodically transferred from soybean to the peanut cultivar 'Florigiant' and back to soybean. PMV-infected soybean tissue was tested serologically (Tolin et al., 1974) at irregular intervals throughout the study. These precautions were taken to detect any genetic changes in the virus strain.

The general inoculation procedure used in the greenhouse was described by Noordam (1973). Inoculum was prepared by grinding 18-24 day old PMV-infected soybean leaves in 0.01 M sodium phosphate buffer, pH

7.0. Approximately 1 g of leaf tissue per 10 ml buffer was ground with a sterilized mortar and pestle. The prepared inoculum was applied by rubbing the moistened pestle onto both of the primary leaves that had been previously dusted with 600-mesh carborundum. The inoculated leaves were then rinsed briefly with tap water to remove any inhibitors of infection. Normally seedlings were at the proper stage for inoculation 8-10 days after planting. First symptoms appeared on trifoliolate leaves 8-12 days after inoculation.

Preliminary Evaluation of Soybean Genetic Materials

The following cultivars and lines were classified as resistant to a mild strain of PMV by Demski and Kuhn (1975): 'Arksoy', 'CNS', 'Curtis', 'Davis', 'Dorman', Ga. 69-90, 'Haberlandt', 'Hale 3', 'Hale 7', 'Hardee', 'Peking', 'Pine Dell Perfection', 'Ralsoy', and 'Virginia'. Arksoy, CNS, Haberlandt, Peking, Pine Dell Perfection, and Virginia were chosen for use in this study because they appeared to be derived from different ancestors. The cultivars Davis, Dorman, Hardee, and Ralsoy contain at least one of the six chosen cultivars in their pedigrees and were not considered to be different sources of resistance. Pedigrees of the remaining four resistant types were not available.

During the winter of 1975-1976, 49 Plant Introductions (PI's), five cultivars, and one breeding line were tested in the greenhouse for reaction to PMV. Seeds of the six selected resistant cultivars and the breeding line were obtained from E. E. Hartwig, U.S. Regional Soybean Laboratory, Stoneville, Miss. Due to the limited number of Haberlandt seed received and their low percentage of germination, Haberlandt plants were not initially tested for reaction to our PMV strain. Seeds of the

PI's were obtained from the Virginia Polytechnic Institute and State University soybean breeding project. The PI strains were from Maturity Groups III and IV. Seed was originally obtained in 1965 from the U.S. Regional Soybean Laboratory in Illinois. The seeds used in this study were produced in 1975 at Warsaw, Virginia.

Plants were grown in 10 cm diameter plastic and clay pots. The potting mixture utilized was approximately 60% soil, 20% sand, and 20% peat moss. Seeding rate was 4-6 seeds per pot. Ten to fifteen plants per entry were inoculated in the manner previously described and scored at 2- and 4-week intervals. Due to low germination percentage, the number of plants inoculated was less than 10 for some entries. Twenty-four PI strains that were initially resistant to PMV were immediately replanted and tested again.

Also during the winter of 1975-1976, a preliminary experiment was conducted to determine the inheritance of resistance to PMV. F_2 seed from crosses between genotypes of known PMV reaction (C. W. Roane, unpublished data) were obtained from the V.P.I. and S.U. soybean breeding project. From 15 to 30 F_2 plants were evaluated from five crosses between PMV-resistant and susceptible types and from one cross between susceptible types.

Diallel Cross and Crosses Between Resistant Types

Four soybean cultivars and 16 PI strains that were found to be PMV-resistant in the preliminary greenhouse tests were planted in a crossing block at Warsaw, Virginia, in 1976 (Table 1). The resistant cultivars and PI's were selected based on diversity of origin in an attempt to increase the probability of identifying different genes controlling

Table 1. Soybean cultivars and plant introductions planted in crossing block at Warsaw, 1976.

Entry No.	Identity	PMV Reaction	Maturity Group	Origin	Date of U.S. Introduction
1	CNS	Resistant	VII	Selection from 'Clemson'. Clemson introduced under PI 71,569 from Nanking, China.	1927
2	Arksoy	Resistant	VI	Introduced under PI 37,335 from Pingyang, Korea.	1914
3	Haberlandt	Resistant	VI	Introduced under PI 6,396 from Pingyang, Korea.	1901
4	Peking	Resistant	IV,V	Selection PI 17,852-B from 'Meyer'. Meyer introduced under PI 17,852 from Peking, China.	1906
5	Virginia	Susceptible (mild)	IV,V	Selection PI 19,186-D from 'Morse'. Morse introduced under PI 19,186 from Manchuria.	1906
6	PI 54,613†	Resistant	III	Manchuria	1921
7	PI 59,849†	Resistant	IV+	Japan	1924
8	PI 80,837	Resistant	IV	Japan	1929
9	PI 89,784†	Resistant	III+	China	1930

† Identity not certain since flower color and/or pubescence color does not agree with that reported in RSLM 238, "Evaluation of Maturity Groups III and IV of the U.S.D.A. Soybean Collection," April, 1969.

Table 1, continued

Entry No.	Identity	PMV Reaction	Maturity Group	Origin	Date of U.S. Introduction
10	PI 181,555	Resistant	IV	Japan	1949
11	PI 200,463†	Resistant	IV	Japan	1952
12	PI 219,789	Resistant	V	Japan	1955
13	PI 229,359	Resistant	IV	Japan	1955
14	PI 227,355††	Resistant	--	---	--
15	PI 90,401	Resistant	IV	China	1930
16	PI 248,511†	Resistant	IV+	Japan	1958
17	PI 219,782†	Resistant	IV	Japan	1954
18	PI 224,271	Resistant	IV	Japan	1955
19	PI 246,367	Resistant	IV+	Japan	1958
20	PI 90,369†	Resistant	IV	---	--
21	PI 167,277†	Resistant	IV	---	--
22	PI 229,315	Susceptible (severe)	V	Japan	1956
23	York	Resistant	V	Dorman X Hood	--

† Identity not certain since flower color and/or pubescence color does not agree with that reported in RSLM 238, "Evaluation of Maturity Groups III and IV of the U.S.D.A. Soybean Collection," April 1969.

†† Not listed in U.S.D.A. Soybean Collection Evaluation Manuals, Maturity Groups 00-X.

resistance to PMV. Two susceptible lines (Virginia and PI 229,315) and a resistant "tester" ('York') were also included in the crossing block.

Entries were seeded separately in 3 m rows on May 13 and at weekly intervals thereafter until June 28 giving a total of seven planting dates. Malathion® was applied every other day during time of flowering to control pollen eating insects.

A diallel cross involving 13 entries (1 through 12 and 22, Table 1) was attempted which results in a potential total of 78 crosses $[\frac{n(n-1)}{2}]$. Eight crosses were also made between the remaining resistant entries and York, the resistant tester. Entry No. 14 (PI 227,355) was not used as a parent in any crosses since it was segregating for flower color and color of pubescence and was considered to be either heterozygous or a mixture.

Whenever possible, crosses were made in such a manner that genetic markers would be visually evident in the F_1 generation to distinguish actual F_1 plants from plants resulting from self-fertilization. Flower color, pubescence color, and hilum color were used as genetic markers. Both seed-parent and pollen-parent plants were identified and tagged for each cross attempted. Pods resulting from crosses were harvested and the seed(s) from each pod were stored separately in coin envelopes. Seed was also threshed and stored separately for each of the parent plants. A small quantity of bulked seed from each of the 23 entries was threshed and stored.

In 1977, nine PMV-resistant PI strains from Maturity Groups II and III were planted in a crossing block at the Agronomy Research Farm, Blacksburg (Table 2). Seeds were obtained from the 1976 PMV field

Table 2. Eleven PMV-resistant soybean lines used in crosses at Blacksburg, 1977.

Identity	Maturity Group	Origin	Date of U.S. Introduction
PI 84,673	II	Korea	1930
PI 86,031	II	Japan	1930
PI 153,280	II	Belgium	1946
PI 360,835	II	---	--
PI 86,490-2	III	Japan	1930
PI 92,686	III	Manchuria	1931
PI 181,553	III	Japan	1949
PI 235,339	III	Uruguay	1956
PI 281,850	III	Japan	1962
PI 89,784†	III+	China	1930
York	V	Dorman X Hood	--

† Identity not certain since flower color does not agree with that reported in RSLM 238, April, 1969.

screening experiment in which these nine PI's were classified as resistant. York and PI 89,784 were also planted in the crossing block to be used as resistant testers. The objective was to cross each of the nine resistant PI strains to one or possibly both of the resistant testers and screen the progenies of such crosses to determine if PMV resistance was controlled by the same gene in all nine genotypes. Both York and PI 89,784 were involved in the 1976 crosses at Warsaw (Table 1).

The 1977 crossing block consisted of 11 PMV-resistant lines planted on five dates 7-10 days apart. The first seeding date was May 18 and the last date was June 20, 1977. A pre-plant soil treatment with Treflan® was used to control weeds. Plants were periodically sprayed with Sevin® for control of foliage damaging insects. Crossing and seed handling procedures were similar to those used in 1976.

Growth of F_1 and F_2 Plants for Seed Production

Due to greenhouse space limitations, it was not feasible to grow out all F_1 seeds produced in the 1976 diallel cross. Three parental lines were chosen based on their origin and the number of successful crosses with them. Designated as "common" resistant parental types, the three lines chosen were Arksoy; PI 89,784; and PI 219,789. The F_1 seeds from 30 crosses out of the possible 33 between these three common parents and all other entries in the diallel crossing block were grown in the greenhouse in the winter and spring of 1976-77. The F_1 seeds from the 1976 crosses between York and each of eight PMV-resistant PI's were also grown at this time.

Up to three seeds from each cross were planted. Each seed was

planted in a 20 cm diameter plastic pot. Plants were maintained under 16 hour days for two weeks after emergence and then the day length was reduced to 13.5 hours. Malathion® and Temik® were used periodically to control common greenhouse insects.

Hypocotyl color, an early indicator of flower color, was noted and recorded for each seedling plant. Later the flower color and color of pubescence were recorded for each plant and compared to the "expected" colors so as to identify true F_1 plants. A total of 116 plants representing 30 crosses from the diallel block and eight crosses to York were grown to maturity. Four to five months after planting, mature seeds from each F_1 plant were threshed and stored separately in coin envelopes. Plants identified as arising from self-fertilized seed were discarded.

A second group of F_1 plants originating from the diallel cross was grown in the greenhouse in the fall and early winter of 1977. These plants were from crosses between the PMV-susceptible entry, PI 229,315, and the following resistant lines: Haberlandt; Peking; PI 54,613; PI 59,849; PI 181,555; and PI 200,463 (Table 1). Also, F_1 plants from the crosses Peking(R) X Virginia(S) and Virginia(S) X PI 229,315(S) were grown.

A third group of F_1 plants was grown in the spring of 1978 from the 1977 crosses made at Blacksburg (Table 2). Thirty-five plants representing 14 crosses were grown to maturity. Day length was maintained at 14 hours from planting to maturity of the plants. Cultural practices were the same as those described for the first group of F_1 plants.

Forty-eight F_2 seed from each of three selected crosses made in

1976 were planted in the greenhouse in the fall of 1977 to produce F_3 seed. The three crosses were as follows: PI 89,784(R) X PI 219,789(R); Peking(R) X PI 219,789(R); PI 229,315(S) X PI 219,789(R). Four seeds were planted in each of sixteen 20 cm plastic pots. The F_2 plants from the Peking X PI 219,789 cross set very few pods so another group of F_2 seed from the same cross were planted in January, 1978. Cultural practices used were the same as those described for growth of F_1 plants. None of the F_2 plants grown to produce F_3 seed were tested for reaction to PMV.

Evaluation of F_1, F_2, and F_3 Generation Plants

The first group of F_1 plants grown in the greenhouse were all tested for reaction to PMV with the exception of a few seedlings suspected to be carrying an unknown, seed-borne virus. These suspicions were based on the presence of distorted primary leaves but they were later shown to be unfounded. Four to six seedlings from each of the 22 parental lines were inoculated and scored at the same time. The second and third group of F_1 plants had only one plant per cross tested for PMV reaction. For crosses where only one seed was available, the resultant F_1 seedling was not inoculated.

Whenever possible, the F_2 seedlings tested for PMV reaction were progeny of F_1 plants that had also been tested. The F_2 seedlings were grown in metal flats in the greenhouse during 1977-78 and tested for reaction to PMV. Approximately 55 seeds were planted per flat. Plants were maintained under natural daylength from late spring to early fall. A constant daylength of 14 hours was used during winter months. Green-

house temperatures ranged from 24 to 32 C during daylight hours and from 15 to 21 C at night. Plants were inoculated by rubbing inoculum into both primary leaves as previously described.

F_3 generation plants derived from the three selected crosses mentioned in the previous section were tested in the spring of 1978. The F_3 progenies from a minimum of 24 F_2 plants were evaluated for each cross. Within each F_2 family, from 10 to 36 plants were inoculated and scored for reaction to PMV.

A known PMV-susceptible line, V73-178, was used as a check in all screening tests of F_1, F_2, and F_3 plants. Susceptible seedlings were grown in 10 cm pots with 3-5 plants per pot. One "check pot" for every 10 seedlings inoculated was used in the screening of F_1 plants. For F_2 and F_3 screenings, a pot of susceptible plants was inoculated after each flat of F_2 or F_3 plants was inoculated.

Counts of susceptible and resistant plants were recorded at 2- and 4-week intervals after inoculation. Specific notes on type of symptoms were also taken on both scoring dates. Reaction to PMV was divided into three categories: no symptoms; systemic necrosis; systemic mottling and mosaic symptoms.

The F_1, F_2, and F_3 data were summarized and analyzed. Chi-square tests were made on F_2 and F_3 data for goodness of fit to the ratios proposed. Chi-square values were calculated without correction for continuity since only unadjusted chi-square values are additive (Snedecor and Cochran, 1967).

Evaluation of Plant Introductions in Maturity Groups II, III, and IV for Reaction to Peanut Mottle Virus

Field Procedures

Seed of soybean PI and FC strains in Maturity Groups 00-IV were obtained in 1976 from Dr. R. L. Bernard, U.S. Regional Soybean Laboratory, Urbana, Ill.

In 1976 at Blacksburg all PI and FC strains in Groups II and III (Table 3) were planted at the Glade Road Plant Pathology and Physiology Research Center and at the Agronomy Research Farm, respectively. The soil at both locations was treated pre-planting with the herbicide Treflan®. Approximately 50 seeds per entry were planted in a 3 m row with rows spaced approximately 90 cm apart. A total of 556 Group II and 646 Group III PI and FC strains were planted. The Group II strains were planted May 28 and June 8. Group III entries were seeded June 7 and 8. Seedlings were thinned to 25-30 plants per row to facilitate inoculation and disease rating. Plants were periodically sprayed with Sevin® to control insect damage.

In 1977, 964 Group IV PI and FC strains were planted at the Agronomy Research Farm, Blacksburg (Table 3). Three "check" cultivars of known reaction to PMV were planted for each 50 FC/PI entries. These cultivars were as follows: York, resistant to PMV and soybean mosaic virus (SMV); Hood, susceptible to PMV and resistant to SMV; Dorman, resistant to PMV and susceptible to SMV. Dates of planting were as follows: entries 1-348, May 23; entries 349-614, May 24; entries 615-1021, June 10.

The plot where entries 1-348 were planted was pre-treated with

Table 3. FC/PI strains tested for reaction to PMV in 1976 and 1977.

Maturity Group	Range of FC/PI strains tested	PI strains not tested†
II	1,157 to 398,493	391,581
		391,585
III	2,108 to 399,080	68,710
		86,111
		297,533
		372,404A
IV	3,548 to 399,119	59,849
		80,837
		379,560
		379,562A
		379,562B
		398,489
		398,619
		398,666
		398,700
		398,753

† No seed available

Treflan®. The two areas where entries 349-1021 were planted were treated with the herbicide Preforan® immediately following planting. Germination percentage was lower in the 1977 test than in 1976. However, plant populations were thinned to the desired 25-30 plants per row where needed.

Inoculation Procedure

Inoculum was prepared by grinding PMV-infected soybean leaves in a Waring Blender containing 0.05 M sodium phosphate buffer, pH 8.0, plus 0.1% sodium sulfite (Na_2SO_3). The ratio of leaf tissue to buffer was approximately 1 g/10 ml. The resulting homogenate was then filtered through cheesecloth and 1% carborundum (600-mesh) added to the final volume. Inoculum was stored in a container on ice and used within three hours after preparation. The following PMV-susceptible types were used as sources of inoculum in 1976: Lee, V73-166, V73-175, V73-224, V73-228, and V73-741. Breeding line V73-178 was used as the sole inoculum source in 1977.

Plants were inoculated when the first trifoliolate leaf was fully expanded which was usually 3-4 weeks after planting, depending on weather conditions. Inoculations were made with an artist's airbrush (Model C, Thayer and Chandler, Inc., Chicago, Illinois). The airbrush was held 2-3 cm from the undersurface of the leaf to be inoculated. Inoculum was forced into the leaf by compressed air at a pressure of 4.2 kg/cm^2 (60 pounds per square inch). The appearance of a water soaked area on the leaf was considered to be the sign of a successful inoculation.

Disease Rating

One week following inoculation all plants showing virus symptoms were rogued since symptoms caused by the seed-borne SMV are difficult to distinguish from PMV symptoms. The number of virus infected plants rogued was recorded for each entry.

PMV symptoms did not appear until at least two weeks after inoculation. All entries were scored for percentage of PMV-infected plants and disease severity at 1- and 2-month intervals after inoculation. Disease severity was rated on a scale of 1 to 3 (Table 4).

In 1976, resistant and susceptible plants were identified and tagged for FC/PI strains that appeared to be segregating for reaction to PMV. Seed was harvested from the individual resistant and susceptible plants and stored separately. Also in 1976 seed was harvested in bulk from each strain with less than 10% virus infection. In 1977, seed was harvested only from FC/PI strains with 0% virus infection.

Greenhouse Evaluation of Selected Plant Introductions in Maturity Groups II and III

Progeny of selected FC/PI strains that showed less than 10% infected plants in the 1976 field test were later tested in the greenhouse for reaction to PMV. From three to ten progeny plants were evaluated for each strain. Also, self-progeny of individual plants of known PMV reaction in the 1976 field test were tested for PMV reaction in the greenhouse. Two to nine progeny plants were evaluated from each parent plant.

Table 4. The scale of PMV disease severity scores for field evaluation of soybean plant introduction strains.

Disease severity rating	Symptom
1	Light mottling
2	Moderate mottling, slight leaf distortion
3	Severe mottling, severe leaf distortion, stunting of plant

IV. RESULTS AND DISCUSSION

Differentiation of Genes for Resistance to Peanut Mottle Virus

Preliminary Evaluation of Soybean Genetic Materials

Results of two preliminary inoculation tests conducted in the greenhouse in 1975 to find cultivars and PI strains resistant to PMV-S/V74S are presented in Tables 5 and 6. Pine Dell Perfection and Virginia both appeared to be resistant two weeks after inoculation but nearly all ofthe plants of each cultivar showed typical PMV symptoms by the third week (Table 6). Infected plants had typical systemic mottle symptoms on trifoliolate leaves with very mild leaf distortion and little or no stunting. The causal agent of the symptoms on Pine Dell Perfection was positively verified as PMV by serology. PMV on Virginia was confirmed by transmission of virus to a known PMV-susceptible soybean line. Arksoy, Peking, and CNS were found to be resistant to PMV (Table 5). Demski and Kuhn (1975) reported that Virginia and Pine Dell Perfection were resistant to the Georgia PMV strain. Perhaps these two cultivars might be used as differential cultivars to distinguish the PMV-M (Georgia) strain from PMV-S/V74S.

A total of 49 Plant Introduction strains were inoculated with PMV. Twenty-four PI's were determined to be resistant to PMV (Table 5). Eighteen of the resistant PI's were from Japan, two from China, one from Manchuria and three of unknown origin. A majority (60%) of the 49 PI strains tested were from Japan which explains the preponderance of PMV-resistant strains from that country. Sixteen of the resistant PI's were selected to be used in the crosses made in 1976 at Warsaw (Table 1).

Table 5. Cultivars or introductions resistant to PMV from two inoculation tests in the greenhouse, 1975.

Identity†	Number of plants in first test		Number of plants in second test		Total number of plants	
	Resistant	Susceptible	Resistant	Susceptible	Resistant	Susceptible
Arksoy	7	0	-	-	7	0
Peking	18	0	-	-	18	0
CNS	9	0	-	-	9	0
PI 54,613	12	0	14	0	26	0
PI 59,849	16	0	16	1	32	1
PI 80,837	9	0	21	0	30	0
PI 81,777	1	0	4	0	5	0
PI 89,784	8	0	20	0	28	0
PI 90,369	8	0	13	0	21	0
PI 90,401	2	0	6	0	8	0
PI 167,277	7	0	19	0	26	0
PI 181,550	3	0	13	0	16	0
PI 181,554	4	0	17	0	21	0
PI 181,555	11	0	21	0	32	0
PI 181,557	2	0	11	0	13	0

† Due to different seed sources, some PI's shown here differ with regard to identifying phenotypic characteristics listed in the U.S.D.A. Soybean Collection Evaluation Manuals. Likewise, the PMV reaction shown here may differ from the reaction shown in Table 24 or Table 29.

Table 5, continued.

Identity†	Number of plants in first test		Number of plants in second test		Total number of plants	
	Resistant	Susceptible	Resistant	Susceptible	Resistant	Susceptible
PI 181,559	16	0	19	2	35	2
PI 200,460	14	0	10	0	24	0
PI 200,463	2	0	16	0	18	0
PI 219,782	9	0	11	0	20	0
PI 219,787	3	0	10	0	13	0
PI 219,789	13	0	13	0	26	0
PI 224,271	3	0	13	0	16	0
PI 226,591	2	0	12	0	14	0
PI 227,355	12	0	13	0	25	0
PI 229,359	10	0	12	0	22	0
PI 246,367	3	0	6	0	9	0
PI 248,511	3	0	7	0	10	0

† Due to different seed sources, some PI's shown here differ with regard to identifying phenotypic characteristics listed in the U.S.D.A. Soybean Collection Evaluation Manuals. Likewise, the PMV reaction shown here may differ from the reaction shown in Table 24 or Table 29.

Table 6. Reaction of cultivars and introductions when inoculated with PMV in the greenhouse in 1975.

Identity[†]	Number of resistant plants	Number of susceptible plants	Total number of plants inoculated
Pine Dell Perfection	1	13	14
Virginia	3	23	26
D72-7139	0	8	8
PI 71,506	11	3	14
PI 82,302	1	15 N[††]	16
PI 85,666	6	4	10
PI 90,392	0	15 N	15
PI 90,490	1	4	5
PI 96,280	1	5 N	6
PI 96,983	13	1	14
PI 97,225	2	12 N	14
PI 154,470	0	5 N	5
PI 157,409	0	15 N	15
PI 157,457	0	10	10
PI 157,474	9	1	10
PI 158,765	0	7	7
PI 200,499	2	12 N	14
PI 200,518	0	10 N	10
PI 224,354	0	4	4
PI 227,555	10	1	11
PI 227,557	8	1	9
PI 229,315	0	2 N	2
PI 229,343	0	3	3

[†] Due to different seed sources, some PI's shown here differ with regard to identifying phenotypic characteristics listed in the U.S.D.A. Soybean Collection Evaluation Manuals. Likewise, the PMV reaction shown here may differ from the reaction shown in Table 24 or Table 29.

[††] N = necrotic flecking on systemically infected trifoliolate leaves in addition to mottle symptoms.

Table 6, continued.

Identity†	Number of resistant plants	Number of susceptible plants	Total number of plants inoculated
PI 229,352	0	5	5
PI 229,362	1	9	10
PI 246,366	0	4 N	4
PI 248,514	4	1	5
PI 261,461	2	13 N	15

† Due to different seed sources, some PI's shown here differ with regard to identifying phenotypic characteristics listed in the U.S.D.A Soybean Collection Evaluation Manuals. Likewise, the PMV reaction shown here may differ from the reaction shown in Table 24 or Table 29.

Eleven PI strains plus D72-7139, a breeding line, were susceptible to PMV while 14 PI's were mixed for reaction to PMV (Table 6). Variation in symptom expression among susceptible PI's was noted. Plants of some susceptible PI strains were stunted and showed yellowing and necrotic flecks on the oldest trifoliolate leaves. Other susceptible plants showed only the more typical systemic mottle and mosaic symptoms with no necrosis nor stunting. PI 229,315 exhibited a severely susceptible reaction to PMV and was retested to verify the results shown in Table 6. Virginia and PI 229,315 were used as PMV-susceptible parents in the 1976 crosses (Table 1).

Six of the PI strains (Table 6) with mixed reactions had a majority of resistant plants with only 1-3 susceptible plants. Seven PI strains had a majority of susceptible plants while PI 85,666 had an almost equal number of resistant and susceptible plants. For PI strains that show a majority of susceptible plants, one might tend to discount a few resistant plants as "escapes" from inoculation. However, results of 1976 and 1977 field evaluations of PI strains for reaction to PMV suggest that many PI's are either mixtures or are segregating for reaction to PMV.

It should be noted here that the reaction to PMV of certain PI strains in Maturity Groups III and IV (Tables 5 and 6) may differ from the reaction determined for the same numbered strains in subsequent field tests conducted in 1976 and 1977, respectively. As stated in the footnotes in Tables 5 and 6, discrepancies in certain phenotypic characteristics, such as flower color and pubescence color, as well as differences in reaction to PMV were noted between PI strains obtained from the V.P.I. and S.U. soybean breeding program and those obtained from the

U.S.D.A. Regional Soybean Laboratory, Urbana, Ill. Such discrepancies are possibly due to further selection within PI strains at the Soybean Laboratory after the original seed of the PI's listed in Tables 5 and 6 were obtained.

Table 7 presents results of the preliminary screening for reaction to PMV of F_2 generation plants derived from crosses between resistant and susceptible parents and between susceptible parents. The data agree with Boerma and Kuhn's report (1976) that PMV resistance is controlled by a single dominant gene and that there is no cytoplasmic or maternal effect on the inheritance of resistance. The five segregating F_2 populations were combined and a pooled chi-square value calculated. The heterogeniety chi-square value had an acceptable probability (Table 7-A) indicating that all families were segregating similarly.

Evaluation of F_1, F_2, and F_3 Generation Plants

Diallel cross. Seeds were obtained from 68 out of the possible 78 crosses in the diallel made a Warsaw in 1976. Advanced generation plants from 38 crosses (Table 8) were grown and tested for reaction to PMV. As shown in Table 8, four lines were used as "common" parents in crosses with all other lines in the diallel crossing block. The four common parents are as follows: Arksoy(R); PI 89,784(R); PI 219,789(R); and PI 229,315(S).

Advanced generation data obtained from the partial diallel cross (Table 8) with regard to inheritance of resistance to PMV will be presented and discussed in three phases in this section. First, results of PMV inoculation of nine F_2 populations derived from crosses among the

Table 7. Reaction of six F_2 populations to inoculation with PMV.

Cross	Number of plants inoculated			3:1 chi-square	
	Resistant	Susceptible	Total	X^2	P Value
Essex(S) X Dorman(R)†	8	7	15	3.7556	0.05 - 0.10
Williams(S) X V68-1242-1025(R)	18	11	29	2.5863	0.10 - 0.25
V69-1111-1851(R) X Williams(S)	21	9	30	0.4000	0.50 - 0.75
V68-1242-1025(R) X Essex(S)	26	7	33	0.2525	0.50 - 0.75
V69-1111-1851(R) X Essex(S)	47	19	66	0.5051	0.25 - 0.50
Total	120	53	173	2.9307	0.05 - 0.10
Essex(S) X Williams(S)	0	28	28	--	---

† R = resistant to PMV
S = susceptible to PMV

Table 7-A. Test of homogeniety of the 3:1 ratio for the five F_2 populations derived from crosses between resistant and susceptible lines.

Item	df	X^2	P Value
Sum of X^2 values	5	7.4995	0.10 - 0.25
Pooled X^2	1	2.9307	0.05 - 0.10
Heterogeneity	4	4.5688	0.25 - 0.50

Table 8. Thirty-eight crosses made at Warsaw, 1976, from which F_1 and F_2 generation plants tested for reaction to PMV were derived.

Entry Number	Identity	Entry number												
		1	2	3	4	5	6	7	8	9	10	11	12	22
1	CNS													
2	Arksoy	X												
3	Haberlandt	-	X											
4	Peking	-	X	-										
5	Virginia†	-	-	-	X									
6	PI 54,613	-	X	-	-	-								
7	PI 59,849	-	-	-	-	-	-							
8	PI 80,837	-	X	-	-	-	-	-						
9	PI 89,784	-	X	X	X	X	X	X	X					
10	PI 181,555	-	X	-	-	-	-	-	-	X				
11	PI 200,463	-	X	-	-	-	-	-	-	X	-			
12	PI 219,789	X	X	X	X††	X	X	X	X	X††	X	X		
22	PI 229,315†	-	X	X	X	X	X	X	-	X	X	X	X††	

† PMV susceptible lines

†† F_3 generation plants also tested for reaction to PMV

four common parents and Virginia(S) will be presented. Secondly, the allelic relationship of genes controlling reaction to PMV carried by the three resistant common parents and Peking(R) will be discussed. Finally, results of PMV inoculation of F_1 and F_2 plants derived from crosses between the four common parents and the remaining seven PMV-resistant lines (Table 8) will be presented and discussed.

Table 9 shows the reaction to inoculation with PMV of F_1 and F_2 plants from crosses between the three common resistant types and two susceptible types. No F_1 seeds were obtained from the attempted cross of Arksoy X Virginia. Some PMV inoculated F_2 plants from several crosses manifested a necrotic reaction in the apical bud. These plants were severely stunted and showed reddish-brown necrotic vein streaking in both primary and trifoliolate leaves. A few affected plants showed complete stem necrosis. The terminal bud necrosis was very similar to the "shepherd's crook" symptom that is observed in soybean plants infected with tobacco ringspot virus (TRSV). Apical bud necrosis was usually not evident until 2-4 weeks after inoculation although severe stunting was observable when the plants were scored two weeks post-inoculation. Top necrosis has previously been described in heterozygous F_1 soybean plants and F_2 populations segregating for reaction to SMV (Koshimizu and Iizuka, 1963; Lee and Ross, 1972; Kiihl, 1976).

Cirulli and Alexander (1969) reported a similar reaction in F_2 plants derived from TMV resistant X susceptible tomato crosses. By maintaining a constant temperature of 26-28 C in the greenhouse, they were able to identify all the heterozygous F_2 plants because of their necrotic response. Homozygous resistant plants produced no symptoms

Table 9. Reaction to PMV of F_1 and F_2 plants from crosses between three resistant and two susceptible lines.

Cross	Reaction of F_1 plants	Number of F_2 plants: Resistant — No symptoms	Resistant — Systemic necrosis	Resistant — Total	Susceptible — Typical PMV symptoms	Susceptible — Total	Expected ratio	X^2	P Value
Arksoy(R) X PI 229,315(S)	R	96	9	105	37	142	3:1	0.0845	0.75-0.90
PI 89,784(R) X PI 229,315(S)	R	66	3	69	21	90	3:1	0.1333	0.50-0.75
PI 229,315(S) X PI 219,789(R)	R	72	6	78	19	97	3:1	1.5155	0.10-0.25
PI 89,784(R) X Virginia(S)	R	78	-	78	17	95	3:1	2.5579	0.10-0.25
PI 219,789(R) X Virginia(S)	-	61	1	62	28	90	3:1	1.7926	0.10-0.25

while susceptible plants showed typical TMV symptoms. At a lower temperature (15-17 C) the heterozygous plants were symptomless and unable to be distinguished from the homozygous resistant plants.

For this study it was assumed that plants exhibiting the necrotic reaction were heterozygous and the PMV-resistant reaction classification was divided into two categories, no symptoms and systemic necrosis. The systemic necrosis category includes only those plants that exhibited complete necrosis of the terminal bud.

Segregation ratios observed in F_2 populations derived from crosses between resistant and susceptible types (Table 9) show that PMV resistance in the three lines is controlled by a dominant gene. However, it cannot be assumed that the gene for resistance is the same in the three resistant lines. Resistance to a mild strain of PMV in two other cultivars 'Dorman' and 'CNS' is known to be governed by a single dominant gene (Boerma and Kuhn, 1976).

As discussed in the previous section, the susceptible reactions of Virginia and PI 229,315 were noticeably different. Similarly, the susceptible F_2 progenies from R X S crosses involving these two susceptible types also differed in reaction to PMV. Susceptible F_2 plants from crosses between resistant types and PI 229,315(S) were stunted and showed necrotic spots on inoculated primary leaves and some necrosis on first and second trifoliolate leaves. Susceptible F_2 plants from crosses between resistant types and Virginia(S) exhibited a mild mottle and mosaic on trifoliolate leaves with only slight stunting and little or no necrosis on inoculated leaves. In general, the appearance of symptoms in plants of the two Virginia derived populations was later than in the

three PI 229,315 derived populations (Table 9). Only one F_2 plant from the two Virginia derived populations exhibited systemic necrosis.

Of the four F_1 plants from the Arksoy(R) X PI 229,315(S) cross that were inoculated with PMV only one was resistant (Table 9), one showed typical symptoms and was definitely susceptible (11C), and two showed mild mottle symptoms on the trifoliolate leaves (11B and 11E, Table 9). One F_1 plant, 11D, was not inoculated with PMV. The susceptible reactions of inoculated F_2 progeny plants from 11C and 11D indicate that the Arksoy parent plant was susceptible to PMV. Also, four self-progeny of the Arksoy parent of 11C and 11D were determined to be susceptible to PMV (Table 10). The virus on plant 11C was positively identified as PMV by serology. Furthermore, the virus was transmitted from 11C to the PMV-susceptible breeding line V73-178. The data presented in Table 10 involving the two F_1 plants, 11C and 11D, suggest that the Arksoy parent plant resulted from an outcross and was susceptible to PMV.

The F_2 progeny of 11B and 11E segregated in a 3R:1S ratio despite the mild mottle symptoms of the F_1 plants (Table 10). Attempts to mechanically transfer PMV from the two F_1 plants to V73-178 were not successful indicating that PMV was not present or present in amounts too small to be mechanically transmitted. Four self-progeny of the Arksoy parent plant were tested for reaction to PMV. Two of the plants showed no symptoms. Approximately two weeks after inoculation mild mottle symptoms developed on the third trifoliolate leaves of the other two progenies. Reactions of these four progeny indicate that the Arksoy parent plant was possibly heterozygous for the gene that conditions reaction to PMV. However, the F_2 populations derived from crosses

Table 10. Reaction to PMV of parental, F_1, and F_2 populations from Arksoy(R) X PI 229,315(S).

Self-progeny of Arksoy parent plant		F_1 plants		Number of F_2 plants							
				Resistant			Susceptible				
No symptoms	Typical PMV symptoms	Identity	PMV reaction	No symptoms	Systemic necrosis	Total	Typical PMV symptoms	Total	Expected ratio	X^2	P Value
2	2	11B	Mild mottle	30	2	32	12	44	3:1	0.1212	0.50-0.75
		11E†	Mild mottle	37	2	39	12	51	3:1	0.0588	0.75-0.90
0	4	11C	Suscep-tible	0	0	0	46	46	-	-	-
		11D††	Not inoc-ulated	0	0	0	47	47	-	-	-

† 11E has same parent plants as 11B.

†† 11D has same parent plants as 11C.

between Arksoy and PMV-resistant types segregated in a manner that indicated all other Arksoy parent plants were homozygous resistant.

The F_1 and F_2 plants derived from crosses among the three PMV-resistant types and between the two susceptible types were tested for reaction to PMV (Table 11). If the genes controlling resistance in the three resistant types were the same, a uniform resistant reaction would be expected in the F_2 generation. However, a few plants with systemic necrosis were observed in the three F_2 populations derived from crosses between the resistant types. A similar hypersensitive reaction to SMV was reported by Conover (1948). Ten to twenty percent of the resistant 'Ogden' plants inoculated with SMV developed top necrosis. All other Ogden plants were symptomless. At 26-28 C, Cirulli and Alexander (1969) observed a necrotic response in a small number of homozygous resistant tomato lines that had been inoculated with TMV. They postulated that the occurrence of necrotic plants in resistant lines might be influenced by inoculum dosage, incubation temperature, or a modifier gene.

In contrast, Kiihl (1976) reported a uniform symptomless, resistant reaction among F_2 progenies of nine crosses between SMV-resistant types. The F_3 lines from three of these crosses were also tested for reaction to SMV and uniform, symptomless resistance among all lines was again observed.

A few plants showing typical PMV symptoms were observed in the F_2 populations derived from crosses of Arksoy with the other two resistant types (Table 11). It was hypothesized that duplicate genes for resistance were present giving rise to a 15R:1S F_2 genetic ratio. The F_2 data from these two populations provide an acceptable fit to the proposed 15R:1S ratio confirming the hypothesis. There were no susceptible

Table 11. Reaction to PMV of F_1 and F_2 plants from crosses between three PMV-resistant lines and between two susceptible lines.

Cross	Reaction of F_1 plants	Number of F_2 plants: Resistant: No symptoms	Resistant: Systemic necrosis	Resistant: Total	Susceptible: Typical PMV symptoms	Susceptible: Total	Expected ratio	X^2	P Value
Arksoy(R) X PI 89,784(R)	R	123	5	128	7	135	15:1	0.2612	0.50-0.75
Arksoy(R) X PI 219,789(R)	R	100	2	102	4	106	15:1	1.1094	0.25-0.50
PI 89,784(R) X PI 219,789(R)	R	94	1	95	0	95	--	--	--
PI 229,315(S) X Virginia(S)	S†	--	--	0	46	46	--	--	--

† The F_2 plants tested are progeny of an uninoculated F_1 plant. The susceptible F_1 plant produced an insufficient number of seeds for an F_2 progeny test.

plants in the F_2 population derived from the cross PI 89,784 X PI 219,789. Thus, it appears that these two PI strains have the same dominant gene for resistance to PMV while Arksoy's resistance is controlled by a dominant gene at another locus. The relationship between these three resistant lines is discussed in more detail later in this section.

All F_1 and F_2 generation plants derived from the cross PI 229,315(S) X Virginia(S) were susceptible (Table 11). Although not shown in Table 11, the F_2 susceptible plants were divided into two groups based on the presence or absence of localized necrotic spots and vein streaking. Approximately 50% of the plants showed little or no localized necrosis while the remaining 50% had a severe necrosis on the inoculated primary leaves and some necrotic flecking of the two oldest trifoliolate leaves. Based on these differences of symptom expression in the F_2 plants, it appears that the susceptible reaction to PMV in PI 229,315 and Virginia is under different genetic control but the mode of inheritance is unclear.

Results of PMV inoculation of F_1 and F_2 generation plants from crosses between Peking(R) and two susceptible types and three resistant types are shown in Tables 12 and 13, respectively. Symptoms exhibited by susceptible F_2 plants derived from the cross Peking(R) X PI 229,315(S) (Table 12) were milder than those observed in susceptible F_2 plants derived from crosses between the three common resistant types and PI 229,315 (Table 9). Susceptible plants showed less stunting and little or no necrotic spotting on inoculated primary leaves.

One F_1 plant (Table 12) from the Peking X PI 229,315 cross was tested for reaction to PMV and found to be susceptible. The F_2 data were

Table 12. Reaction to PMV of F_1 and F_2 plants from crosses between Peking(R) and two PMV-susceptible lines.

Cross	Reaction of F_1 plants	Number of F_2 plants: Resistant: No symptoms	Resistant: Systemic necrosis	Resistant: Total	Susceptible: Typical PMV symptoms	Susceptible: Total	Expected ratio	X^2	P Value
Peking X PI 229,315	S[†]	14	1	15	44	59	1:3	0.0057	0.90-0.95
		19	-	19	48	67	1:3	0.4030	0.50-0.75
	Pooled data from 2 F_1 plants	33	1	34	92	126	1:3	0.2646	0.50-0.75
Peking X Virginia	R	29	-	29	5	34	3:1	1.9216	0.10-0.25
		16	-	16	4	20	3:1	0.2667	0.50-0.75
	Pooled data from 2 F_1 plants	45	-	45	9	54	3:1	2.0000	0.10-0.25

[†] The F_2 plants tested are progenies of two uninoculated F_1 plants. The susceptible F_1 plant produced an insufficient number of seeds for an F_2 progeny test.

Table 13. Reaction to PMV of F_1 and F_2 plants from crosses between Peking(R) and three PMV-resistant lines.

Cross	Reaction of F_1 plants	Number of F_2 plants: Resistant: No symptoms	Resistant: Systemic necrosis	Resistant: Total	Susceptible: Typical PMV symptoms	Susceptible: Total	Expected ratio	X^2	P Value
Arksoy X Peking	R	85	-	85	18	103	13:3	0.1098	0.50-0.75
		90	1	91	24	115	13:3	0.3391	0.50-0.75
	Pooled data from 2 F_1 plants	175	1	176	42	218	13:3	0.0381	0.75-0.90
Peking X PI 89,784	R	77	-	77	21	98	13:3	0.4615	0.25-0.50
Peking X PI 219,789	R	108	-	108	21	129	13:3	0.5170	0.25-0.50

tested against a 1R:3S genetic ratio which provided an acceptable fit. A susceptible F_1 reaction and a 1R:3S F_2 ratio are expected if Peking is homozygous recessive for PMV resistance and PI 229,315 is homozygous dominant for susceptibility at the same gene locus. These data indicate that resistance in Peking is controlled by a recessive gene rather than a dominant gene as in the three common resistant types discussed previously (Table 9).

The reactions of PMV inoculated F_1 and F_2 plants from the Peking(R) X Virginia(S) cross (Table 12) do not fit the pattern of the previous cross. The inoculated F_1 plant was resistant. Susceptible F_2 plants did not show any symptoms until 3-4 weeks after inoculation, similar to Virginia. The systemic symptoms were mild with no localized necrosis. The different ratios observed in the two F_2 populations derived from crosses between Peking and the two susceptible types substantiate the hypothesis that the susceptible reactions of PI 229,315 and Virginia are under different genetic control.

It is postulated that the reaction to PMV in Virginia is controlled by a different allele at the same locus as the PI 229,315 allele. The Virginia allele conditions a milder reaction to PMV. Because of the mild reaction of Virginia, the heterozygous plants in the Peking X Virginia F_2 population appeared to be resistant but actually carried the susceptible genotype. Only the homozygous dominant plants gave a visible susceptible reaction. Thus, a 3R:1S F_2 ratio was observed (Table 12) rather than the actual 1R:3S ratio due to the mild susceptible reaction of the Virginia allele which failed to express itself phenotypically in the heterozygous F_2 plants.

The data presented in Table 13 support the prior evidence (Table 12) that suggests the presence of a recessive gene for PMV resistance in Peking. All F_1 plants from the crosses between Peking and the three common resistant types were resistant to PMV inoculation (Table 13). If the gene controlling resistance in Peking were the same as in the three common resistant genotypes, then one would expect no segregation in any F_2 populations from crosses among these types. However, segregation was observed in all three F_2 populations from crosses of Peking with the other resistant parents. Assuming a recessive gene for resistance in Peking, the observed F_2 data were tested against an expected 13R:3S ratio and found to provide an acceptable fit.

Thus, the presence of susceptible plants in each of the F_2 populations derived from crosses between Peking and the three common resistant parents (Table 13) indicates that the genes controlling resistance in the parental types are different. None of the observed F_2 data from the three crosses fit a 15R:1S ratio which would be expected if duplicate, dominant genes were present. The F_1 and F_2 data derived from specific crosses among the three common resistant parents, Peking, and PI 229,315 (Tables 9, 12, and 13) very strongly indicate that a recessive gene conditions resistance to PMV in Peking.

There have been other reports of recessive genes conditioning resistance to viral diseases in legume crops (Yen and Fry, 1956; Johnson and Hagedorn, 1958; Cousin, 1965). Ali (1950) reported on the inheritance of resistance to bean common mosaic virus (BCMV) in field beans. He found that resistance was controlled by a dominant gene in the cultivar Corbett Refugee and by a recessive gene in the cultivar Robust.

Both cultivars showed complete resistance to BCMV when inoculum was rubbed into the primary leaves. When inoculum was applied by the "approach-graft" inoculation technique, Corbett Refugee plants showed a hypersensitive necrotic response but the Robust plants showed no symptoms.

A 13R:3S ratio was observed in the F_2 generation from a cross between these two cultivars when the F_2 plants were rub inoculated. When F_2 plants were inoculated by the approach-graft method, a ratio of 9 (top necrosis):4 (no symptoms):3 (typical BCMV symptoms) was observed. He suggested the presence of two genes that acted with dominant and recessive epistasis in the following manner. A dominant gene "A" is required for virus infection. Another dominant gene "I", when present with "A" inhibits symptom expression after rub inoculation or natural field infection. However, the "I" gene conditions top necrosis when there is a continuous supply of virus inoculum, as with the approach-graft technique. The recessive allele "a" acts as a recessive epistatic and conditions resistance to typical BCMV symptoms as well as top necrosis.

Symptoms exhibited by susceptible F_2 plants from crosses between Peking and the three resistant types were typical of PMV-infected plants. There was little or no stunting of infected plants and there was no localized necrotic reaction on inoculated leaves. Some susceptible F_2 plants did not show disease symptoms until 2-3 weeks post-inoculation.

Non-inoculated F_2 plants from three selected crosses were grown to maturity and their progeny tested for reaction to inoculation with

PMV (Table 14). The F_3 lines tested from the PI 229,315(S) X PI 219,789(R) cross verified the F_2 segregation data (Table 9) for this cross and confirmed the presence of a dominant gene for PMV resistance in PI 219,789. A uniform resistant reaction was observed for all F_3 lines from the PI 89,784(R) X PI 219,789(R) cross confirming the F_2 data shown in Table 11. Three plants exhibiting top necrosis were observed in three of the lines. As mentioned previously, other investigators have observed a hypersensitive necrotic reaction to virus inoculation in homozygous resistant soybean and tomato plants (Conover, 1948; Cirulli and Alexander, 1969).

Thirty-five F_3 lines from the Peking(R) X PI 219,789(R) cross were tested for reaction to inoculation with PMV. The reactions of the F_3 lines to inoculation with PMV (Table 14) confirm the presence of a recessive gene for resistance in Peking as first made evident by the reactions of F_1 and F_2 plants from selected crosses (Tables 12 and 13).

Reactions to PMV of F_1 and F_2 populations of crosses between a susceptible type, PI 229,315, and five other resistant lines in the diallel experiment were determined (Table 15). The five resistant lines each appear to have a single dominant gene for resistance based on the F_2 segregation data.

In conjunction with these F_2 results, the reactions to PMV of F_1 and F_2 plants from crosses between three resistant parents, Arksoy, PI 89,784, and PI 219,789, and all other resistant entries in the diallel cross were determined (Tables 16, 17, and 18). Symptoms of susceptible F_2 plants in the populations derived from crosses between resistant types were usually very mild. Generally, a mild, systemic

Table 14. Reaction to PMV of F_3 lines from three selected crosses.

Cross	Number of F_3 lines				Expected ratio	X^2	P Value
	Resistant	Segregating	Susceptible	Total			
PI 229,315(S) X PI 219,789(R)	6	15(3R:1S)	3	24	1:2:1	2.2500	0.25-0.50
PI 89,784(R) X PI 219,789(R)	30	0	0	30	-	-	-
Peking(R) X PI 219,789(R)	12	16(13R:3S,3R:1S) 5(1R:3S)	2	35	7:6:2:1†	1.4518	0.50-0.75

† 7R:6(13R:3S and 3R:1S):2(1R:3S):1S

Table 15. Reaction to PMV of F_1 and F_2 plants from crosses between a common susceptible parent, PI 229,315 (designated CP), and five PMV-resistant lines.

Cross	Reaction of F_1 plants	Number of F_2 plants: Resistant: No symptoms	Resistant: Systemic necrosis	Resistant: Total	Susceptible: Typical PMV symptoms	Susceptible: Total	Expected ratio	X^2	P Value
Haberlandt X CP†	R	27	6	33	9	42	3:1	0.2857	0.50-0.75
CP X PI 54,613	-	40	6	46	9	55	3:1	2.1879	0.10-0.25
PI 59,849 X CP	R	36	4	40	9	49	3:1	1.1497	0.25-0.50
CP X PI 181,555	R	28	4	32	14	46	3:1	0.7246	0.25-0.50
CP X PI 200,463	R	30	5	35	10	45	3:1	0.1852	0.50-0.75

† No distinguishing genetic marker to verify the cross

Table 16. Reaction to PMV of F_1 and F_2 plants from crosses between a common resistant parent, Arksoy (designated CP), and six PMV-resistant lines.

Cross	Reaction of F_1 plants	Number of F_2 plants: Resistant: No symptoms	Resistant: Systemic necrosis	Resistant: Total	Susceptible: Typical PMV symptoms	Susceptible: Total	Expected ratio	X^2	P Value
CP X CNS	R	82	-	82	6	88	15:1	0.0485	0.75-0.90
CP X Haberlandt	R	89	2	91	4	95	15:1	0.6744	0.25-0.50
CP X PI 54,613	R	78	3	81	5	86	15:1	0.0279	0.75-0.90
CP X PI 80,837	R	86	-	86	3	89	15:1	1.2592	0.25-0.50
CP X PI 181,555	R	71	1	72	2	74	15:1	1.5892	0.10-0.25
CP X PI 200,463	R	69	2	71	2	73	15:1	1.5352	0.10-0.25

Table 17. Reaction to PMV of F_1 and F_2 plants from crosses between a common resistant parent, PI 89,784 (designated CP), and six PMV-resistant lines.

Cross	Reaction of F_1 plants	Number of F_2 plants: Resistant: No symptoms	Resistant: Systemic necrosis	Resistant: Total	Susceptible: Typical PMV symptoms	Susceptible: Total	Expected ratio	X^2	P Value
Haberlandt X CP	R	93	1	94	0	94	-	-	-
PI 54,613 X CP†	R	91	2	93	5	98	15:1	0.2204	0.50-0.75
CP X PI 59,849	R	93	1	94	0	94	-	-	-
CP X PI 80,837	R	87	1	88	0	88	-	-	-
CP X PI 181,555	R	100	2	102	0	102	-	-	-
PI 200,463 X CP	R	95	-	95	0	95	-	-	-

† No distinguishing genetic marker to verify the cross

Table 18. Reaction to PMV of F_1 and F_2 plants from crosses between a common resistant parent, PI 219,789 (designated CP), and seven PMV-resistant lines.

Cross	Reaction of F_1 plants	Number of F_2 plants: Resistant: No symptoms	Resistant: Systemic necrosis	Resistant: Total	Susceptible: Typical PMV symptoms	Susceptible: Total	Expected ratio	X^2	P Value
CP X CNS	R	97	-	97	5	102	15:1	0.3163	0.50-0.75
Haberlandt X CP†	R	95	1	96	0	96	-	-	-
CP X PI 54,613	R	90	3	93	4	97	15:1	0.7484	0.25-0.50
PI 59,849 X CP	R	82	-	82	0	82	-	-	-
PI 80,837 X CP	R	92	1	93	0	93	-	-	-
CP X PI 181,555	R	105	-	105	0	105	-	-	-
CP X PI 200,463†	R	93	-	93	0	93	-	-	-

† No distinguishing genetic marker to verify the cross

mottle occurred on the third or fourth trifoliolate leaf with a slight curling of the affected leaves. No plant stunting was observed. Very often, susceptible plants did not manifest any symptoms until 2-4 weeks after inoculation. The systemic necrosis reaction observed in most crosses was the same as described previously.

Based on the F_2 segregation data presented in Table 11, it was established that the dominant gene for resistance to PMV in Arksoy differed from the dominant gene present in PI 89,784 and PI 219,789. The presence of susceptible plants in F_2 populations derived from the crosses between Arksoy and the six resistant types (Table 16) indicates that the Arksoy gene for PMV resistance is not allelic to any of the genes carried by the six resistant types.

The 15R:1S genetic ratio observed in the F_2 generation of the PI 54,613 X PI 89,784 cross indicates that a different dominant gene controls the reaction to PMV in these two resistant types (Table 17). A 15R:1S ratio also was observed in the F_2 population of the cross PI 219,789 X PI 54,613 (Table 18). The absence of susceptible plants in F_2 progenies of crosses between five resistant types and two of the resistant testers, PI 89,784 and PI 219,789 (Tables 17 and 18), confirms the uniform resistant reaction of F_2 plants from the cross between these two testers (Table 11). Thus, additional evidence is provided for the presence of the same or allelic dominant genes controlling resistance to PMV in PI strains 89,784 and 219,789.

A summary of F_2 genetic ratios observed in crosses between the three common resistant types and all other resistant types in the diallel cross, except Peking, is presented in Table 19. The 15R:1S and

Table 19. Summary of genetic ratios observed in F_2 populations derived from crosses between the three "common" resistant types and all other resistant types in the diallel block.

	Arksoy	PI 89,784	PI 219,789
CNS	15R:1S	-	15R:1S
Haberlandt	15R:1S	All R	All R
PI 54,613	15R:1S	15R:1S	15R:1S
PI 59,849	-	All R	All R
PI 80,837	15R:1S	All R	All R
PI 181,555	15R:1S	All R	All R
PI 200,463	15R:1S	All R	All R

3R:1S F_2 segregation ratios observed in populations derived from crosses between resistant types (Tables 11 and 19) and from crosses between resistant and susceptible types (Tables 9 and 15), respectively, provide evidence for the presence of three different dominant genes conditioning resistance to PMV (Table 20).

Arksoy is a parent of Dorman (Weiss and Stevenson, 1955). Therefore, it is most probable that the dominant gene Rpv for resistance to PMV in Dorman (Boerma and Kuhn, 1976) is derived from Arksoy. Thus the gene controlling resistance in Arksoy should now be designated Rpv_1 (Table 20) since it was the first PMV resistance gene to be isolated and identified.

The evidence presented for a recessive gene controlling resistance in Peking is quite adequate (Tables 12, 13, and 14). It is designated rpv_2 (Table 20) since it has been shown to act in a recessive manner in combination with Rpv_1 and also with the dominant gene for resistance in PI 89,784 and PI 219,789 (Table 13). It is assumed that the Peking gene will also act as a recessive in combination with the seven other resistant lines carrying dominant genes for resistance. However, no F_2 data are presented from crosses between Peking and these other resistant lines to confirm this assumption.

The gene controlling resistance to PMV is designated Rpv_3 for the following resistant types: Haberlandt; PI 59,849; PI 80,837; PI 89,784; PI 181,555; PI 200,463; and PI 219,789 (Table 20). That this dominant gene is different from the dominant gene for resistance in Arksoy, CNS, and PI 54,613 is demonstrated by 15R:1S segregation ratios from crosses with these resistant types (Tables 11 and 19). The F_2 genetic ratios

Table 20. Proposed genotypes of the 11 resistant cultivars and PI strains in the diallel cross and their genetic relationships.

Identity	Origin	Gene conditioning resistance to PMV
Arksoy	Korea	Rpv_1
Peking	China	rpv_2rpv_2
Haberlandt	Korea	Rpv_3
PI 59,849	Japan	Rpv_3
PI 80,837	Japan	Rpv_3
PI 89,784	China	Rpv_3
PI 181,555	Japan	Rpv_3
PI 200,463	Japan	Rpv_3
PI 219,789	Japan	Rpv_3
CNS	China	Rpv_4
PI 54,613	Manchuria	$Rpv_?$

presented in Table 19 also illustrate that CNS and PI 54,613 carry different dominant gene(s) (Rpv_4) than either Arksoy or PI 89,784 and PI 219,789. Based on available F_2 data (Table 19), we cannot determine if the genes for resistance in CNS and PI 54,613 are allelic. Segregation data from advanced generations from the cross CNS X PI 54,613 would be necessary to establish the genic relationship between these two resistant lines. Based on the results, the presence of at least three different dominant genes for resistance to PMV (Rpv_1, Rpv_3, Rpv_4) is indicated (Table 20). Additional testing of larger numbers of F_2 plants and testing of F_3 lines from each of the crosses between resistant types (Tables 11 and 19) would be desirable to more firmly establish the relationship of these genes.

Crosses between resistant types and two resistant testers. Table 21 shows reactions of F_1 and F_2 plants derived from crosses between seven resistant PI strains and the resistant cultivar 'York' to inoculation with PMV. A relatively small number of F_2 plants was tested for each cross. As in crosses between resistant types in the diallel experiment, some F_2 plants exhibited systemic necrosis. Summer greenhouse temperatures (27-32 C) seemed to favor the development of top necrosis. These F_2 plants were inoculated and scored in August and September. A similar effect of temperature on expression of a hypersensitive necrotic reaction to virus infection has been reported for heterozygous plants (Schroeder et al., 1966; Cirulli and Alexander, 1969) and suggested for homozygous resistant plants (Cirulli and Alexander, 1969).

It is assumed that York carries a dominant gene for resistance to PMV since it is resistant and one of its parents, Dorman, has been shown

Table 21. Reaction to PMV of F_1 and F_2 plants from crosses between a resistant tester, York (T), and seven PMV-resistant PI strains.

Cross	Reaction of F_1 plants	Number of F_2 plants: Resistant: No symptoms	Resistant: Systemic necrosis	Resistant: Total	Susceptible: Typical PMV symptoms	Susceptible: Total	Expected ratio	X^2	P Value
PI 90,401 X T	R	27	4	31	2	33	15:1 13:3	0.0020 3.4880	0.95-0.975 0.05-0.10
T X PI 248,511	Mild mottle	30	2	32	7	39	13:3	0.0164	0.75-0.90
T X PI 219,782	R	37	2	39	0	39	-	-	-
T X PI 224,271	R	27	3	30	10	40	13:3	1.0256	0.25-0.50
T X PI 246,367†	R	26	7	33	0	33	-	-	-
T X PI 90,369	R	27	2	29	4	33	15:1 13:3	1.9414 0.9518	0.10-0.25 0.25-0.50
PI 167,277 X T	R	38	6	44	1	45	15:1	1.2459	0.25-0.50

† No distinguishing genetic marker to verify the cross

to carry a dominant gene for resistance (Boerma and Kuhn, 1976) and the other parent, Hood, is susceptible to PMV. PMV resistance in York can be traced back to the Arksoy gene, Rpv_1, if one accepts the hypothesis that resistance in Dorman came through the Arksoy parent.

Susceptible F_2 plants were observed in five of the seven populations (Table 21). Two PI strains, PI 219,782 and PI 246,367, appear to carry the same dominant gene for PMV resistance as does York. Segregating F_2 populations were tested for an acceptable fit to two genetic ratios, 15R:1S and 13R:3S. Resistance to PMV in PI 90,401 and PI 167,277 appears to be controlled by a dominant gene at a different locus than the York gene based on the 15R:1S F_2 genetic ratios. The 13R:3S ratio provided the best fit for three F_2 populations suggesting the possibility of recessive genes for resistance to PMV in the following PI strains: PI 248,511; PI 224,271; and PI 90,369.

A mild mottle was observed on the two F_1 plants from the York X PI 248,511 cross. The symptoms were very similar to those observed on two F_1 plants from the Arksoy(R) X PI 229,315(S) cross (Table 10). The plants were not stunted and seed production did not appear to be adversely affected. An attempt to mechanically transfer the "virus" from the F_1 plants to the PMV-susceptible soybean line V73-178 was not successful. Apparently, the mottle symptoms either were caused by a pathogen other than PMV or the concentration of PMV in the infected plants was too low to allow mechanical transmission. Additional F_2 and F_3 data are needed before any definitive statements can be made concerning allelic relationships between York and these seven resistant PI strains.

The F_1 plants derived from 1977 crosses of new PMV-resistant PI's X York and/or PI 89,784 were grown in the greenhouse in 1978. One F_1 plant from each cross was inoculated with PMV (Table 22) except when only one was available. The F_1 plant from the PI 84,673 X PI 89,784 cross developed mild PMV symptoms as described in the previous paragraph. The seed parent, PI 84,673, was classified as resistant in the 1976 field test but developed a mild mottle symptom when inoculated by rubbing in the greenhouse (Table 22). Three other PI strains in the 1977 crossing block when retested under greenhouse conditions were found to exhibit mild mottle symptoms that appeared 2 to 4 weeks after inoculation.

Kuhn and Smith (1977), working with MDMV, reported that the length of time between inoculation and appearance of symptoms in the greenhouse was correlated to field resistance in hybrid corn. A disease index based on the percentage of infected plants and length of the virus incubation period in greenhouse tests was used to predict reactions of hybrids to MDMV in the field.

The crosses shown in Table 22 provide a starting point for a continued search for different alleles and/or genes for PMV resistance in soybeans. PI 89,784 was used in both the 1976 diallel cross and in the 1977 crosses. Thus, it could be used as a common parent to establish allelic relationships between the resistant types used in the 1976 and 1977 crosses. Likewise, the F_2 data obtained from the 1976 and 1977 crosses involving York could be used to determine allelic relationships between all resistant PI strains crossed to York. Thus, F_2 and F_3 segregation data obtained from the 1977 crosses could be related back to the 1976 findings (Tables 19 and 20) and other genes for resistance

Table 22. Summary of 1977 crosses between two resistant testers, PI 89,784 (T-1) and York (T-2), and nine PMV-resistant PI strains and the reaction of F_1 plants to inoculation with PMV.

Cross	Number of F_1 plants grown	PMV reaction of one F_1 plant
PI 84,673† X T-1	2	Mild mottle
PI 86,031† X T-1	1	Not inoculated
PI 86,490-2 X T-1	3	Resistant
PI 92,686† X T-1	3	Resistant
T-1 X PI 153,280†	3	Resistant
T-1 X PI 181,553	3	Resistant
T-1 X PI 235,339	1	Not inoculated
PI 281,850 X T-1	3††	Resistant
PI 360,835 X T-1	3	Resistant
PI 86,490-2 X T-2	1	Not inoculated
PI 89,784 X T-2	3††	Resistant
T-2 X PI 235,339	2	Resistant
PI 281,850 X T-2	3††	Resistant
PI 360,835 X T-2	1	Not inoculated

† Parental genotype was resistant in 1976 field test but exhibited mild mottle symptoms when re-evaluated under greenhouse conditions.

†† No distinguishing genetic marker to verify the cross

to PMV might possibly be discovered.

Evaluation of Plant Introduction Strains in Maturity Groups II, III, and IV for Reaction to Peanut Mottle Virus

Tables 23 and 24 list the FC and PI strains in Maturity Groups II and III, respectively, that showed fewer than 10% PMV-infected plants in the 1976 field inoculation test. The ten per cent figure was chosen arbitrarily. However, it is not implied that all FC/PI strains scored as having less than 10% infected plants are resistant to PMV and vice versa. The lack of a clear-cut, uniform reaction to PMV within FC and PI strains was generally observed in both 1976 and 1977 field inoculation tests. The ten per cent level was chosen so as not to delete any PMV-resistant strains which might have had a few plants infected with a seed-borne virus such as SMV. Even though seedling plants showing seed-transmitted virus symptoms were rogued, it is possible that some plants did not manifest virus symptoms until after roguing occurred.

The appearance of both resistant and susceptible plants within an FC/PI strain might be due to one or more of the following possibilities: some plants within a strain infected with a virus other than PMV; differences in reaction due to different stages of maturity of plants when inoculated; inconsistency of inoculation technique; previous accidental seed mixtures; and actual genetic differences for reaction to PMV within FC/PI strains. Progeny of selected FC/PI strains showing 0-10% PMV infection in the field test were grown and retested in the greenhouse for reaction to PMV (Tables 23 and 24). Inoculations in the greenhouse were made using the "mortar and pestle" technique described previously.

Table 23. Maturity Group II soybean PI strains having 0-10% PMV-infected plants from field inoculation test of 556 strains and the reaction of selected strains to PMV inoculation in the greenhouse.

PI number	Field evaluation		Greenhouse evaluation		
	Percentage of infected plants	Disease severity score	Number of resistant plants	Number of susceptible plants	Total number of plants inoculated
68,410	3	1	0	6M†	6
68,521	0	-	0	4M	4
68,522	9	1	0	9M	9
68,609LB	6	1	6	1M	7
68,676††	3	1	8	0	8
68,706	0	-	0	4	4
68,708	0	-	0	11	11
70,078††	0	-	4	0	4
79,609	0	-	0	5N†††	5
79,756	6	1	-	-	-
81,029N	6	1	0	3N	3
84,673	0	-	0	9M	9
86,031	0	-	0	7M	7
86,069	3	1	1	7N	8
87,628	0	-	0	2N	2
88,803	9	1	-	-	-
89,003-1††	9	1	14	0	14
89,008††	0	-	2	0	2
91,107	0	-	0	2	2
91,117††	0	-	5	0	5
92,563	0	-	0	4	4
92,596	0	-	0	5M	5

†M = mild mottle on trifoliolate leaves that developed 2-4 weeks after inoculation.

††Resistant reaction in both field and greenhouse inoculation tests

†††N = veinal necrosis on trifoliolate leaves

Table 23, continued.

PI number	Field evaluation		Greenhouse evaluation		
	Percentage of infected plants	Disease severity score	Number of resistant plants	Number of susceptible plants	Total number of plants inoculated
92,630	0	-	0	5	5
92,683	0	-	0	4	4
92,705	6	1	-	-	-
92,733	0	-	0	5N	5
96,188	0	-	1	1N	2
153,280	0	-	0	7M	7
153,289††	0	-	2	0	2
189,929	3	1	-	-	-
189,930	9	2	-	-	-
200,596	0	-	0	11M	11
227,321	9	1	-	-	-
227,324	3	1	-	-	-
232,993	0	-	0	5N	5
238,922	3	1	0	6M	6
266,806B	6	1	-	-	-
266,807B	0	-	0	4	4
290,130	0	-	0	11	11
291,291	9	1	-	-	-
291,298	6	1	-	-	-
291,315	3	1	-	-	-
297,510	9	2	-	-	-
360,835††	0	-	7	0	7
361,062B††	0	-	2	0	2
361,065B††	0	-	4	0	4
361,074	3	2	-	-	-
391,577	6	1	-	-	-

††Resistant reaction in both field and greenhouse inoculation tests

Table 24. Maturity Group III soybean FC/PI strains having 0-10% PMV-infected plants from field inoculation test of 646 strains and the reaction of selected strains to PMV inoculation in the greenhouse.

FC/PI number	Field evaluation		Greenhouse evaluation		
	Percentage of infected plants	Disease severity score	Number of resistant plants	Number of susceptible plants	Total number of plants inoculated
4,002N	6	1	-	-	-
31,678	6	1	-	-	-
54,583	3	1	1	9	10
54,608-3	6	1	-	-	-
54,610-1	9	2	-	-	-
54,620-2	9	1	-	-	-
68,398	6	3	-	-	-
68,479	0	-	0	5	5
68,479-1	6	2	-	-	-
68,494	9	2	-	-	-
68,523	9	1	-	-	-
68,759	6	1	-	-	-
70,199	1/6[†]	2	-	-	-
70,528	9	2	-	-	-
71,850-1	6	1	-	-	-
79,691	0	-	0	1	1
79,693	6	2	-	-	-
79,726	3	2	0	9M[††]	9
79,835	6	2	-	-	-
80,459[†††]	6	1	1	0	1
80,466-1	6	1	-	-	-

[†] In rows with less than 10 plants, the number of susceptible plants/total number of plants was recorded.

[††] M = mild mottle on trifoliolate leaves that developed 2-4 weeks after inoculation.

[†††] Resistant reaction in both field and greenhouse inoculation tests

Table 24, continued.

FC/PI number	Field evaluation: Percentage of infected plants	Field evaluation: Disease severity score	Greenhouse evaluation: Number of resistant plants	Greenhouse evaluation: Number of susceptible plants	Greenhouse evaluation: Total number of plants inoculated
80,831	6	2	-	-	-
80,847-1	6	1	1	6N††††	7
81,041-1	0	-	2	5	7
83,945-1	6	1	-	-	-
84,611	9	1	-	-	-
85,559	3	2	4	8	12
85,630	6	1	5	2N	7
86,073	3	1	1	10M	11
86,144†††	0	-	3	0	3
86,490-2†††	0	-	13	0	13
88,287†††	0	-	3	0	3
88,289	6	1	-	-	-
88,349	6	1	-	-	-
88,350	0	-	0	8	8
88,782	0	-	0	1M	1
89,146†††	6	1	9	0	9
90,499-1†††	1/9	1	7	0	7
90,573	3	2	7	1	8
91,108N	6	1	-	-	-
91,113	6	1	-	-	-
91,121-1	9	1	-	-	-
91,750†††	0	-	2	0	0
92,605	9	1	-	-	-
92,659	6	1	-	-	-
92,686	0	-	0	7M	7

†††Resistant reaction in both field and greenhouse inoculation tests

††††N = veinal necrosis on trifoliolate leaves

Table 24, continued.

FC/PI number	Field evaluation: Percentage of infected plants	Field evaluation: Disease severity score	Greenhouse evaluation: Number of resistant plants	Greenhouse evaluation: Number of susceptible plants	Greenhouse evaluation: Total number of plants inoculated
92,718-2†††	3	3	11	0	11
92,720	6	1	-	-	-
93,563	9	1	-	-	-
96,194-3	9	2	-	-	-
153,292	0	-	1	6M	7
157,416	6	1	0	8N	8
157,421	9	1	-	-	-
181,553†††	0	-	11	0	11
181,554†††	0	-	2	0	2
200,457†††	0	-	1	0	1
224,272†††	0	-	1	0	1
235,339†††	0	-	10	0	10
261,468†††	0	-	1	0	1
273,483B†††	0	-	2	0	2
281,850†††	0	-	5	0	5
339,995	0	-	2	1M	3
360,841†††	0	-	5	0	5
360,844†††	0	-	3	0	3
361,063†††	0	-	2	0	2
361,064	6	1	-	-	-
398,312	6	1	3	3M	6
398,395	0	-	-	-	-
398,494	0	-	-	-	-
398,697	6	1	-	-	-
398,702†††	9	1	10	0	10
398,726†††	3	1	2	0	2

†††Resistant reaction in both field and greenhouse inoculation tests

Table 24, continued.

FC/PI number	Field evaluation		Greenhouse evaluation		
	Percentage of infected plants	Disease severity score	Number of resistant plants	Number of susceptible plants	Total number of plants inoculated
398,751	9	2	-	-	-
398,752	6	1	-	-	-
398,755	3	2	-	-	-
398,763	9	2	-	-	-
398,813	9	1	-	-	-
398,841	0	-	-	-	-
398,930†††	9	1	5	0	5
398,955†††	9	2	5	0	5
398,965†††	9	1	16	0	16
399,008†††	0	-	2	0	2

†††Resistant reaction in both field and greenhouse inoculation tests

Tables 25 and 26 allow comparisons between the field and greenhouse inoculation tests of selected strains in Maturity Groups II and III, respectively. In Maturity Group II (Table 25), 18 of 26 PI strains that showed 0% PMV infection in the field were found to be susceptible in the greenhouse. For Maturity Group III (Table 26) the trend was not so pronounced. Again, however, some PI strains that showed no PMV-infected plants in the field were determined to be susceptible in the greenhouse test.

Data presented in these two tables suggest that the mortar and pestle inoculation technique is a more severe test for resistance than the artist's airbrush inoculation technique. These findings are in agreement with a previous report (Leong, 1976) of greater efficiency of the mortar and pestle inoculation technique as compared to the airbrush technique. Environmental differences between the field and greenhouse may also be involved in these differences in reaction to PMV.

Another important factor involved in explaining the differences in reactions to PMV between field and greenhouse tests is the stage of plant maturity at inoculation. Demski and Kuhn (1975) reported a marked decrease in percentage of susceptible plants under both greenhouse and field conditions when mechanical inoculation was delayed longer than 20 and 30 days after planting, respectively. The average time from seeding until field inoculation was 32 days for the FC/PI strains in Maturity Groups II, III, and IV. It is recognized that weather conditions have an effect on rate of plant growth. Thus, time between seeding date and date of inoculation is dependent on the rate of plant growth and not on a specified number of days.

Table 25. Reaction of selected PI strains in Maturity Group II to PMV inoculation in the greenhouse in comparison to their reaction to PMV inoculation in the field.

Percentage of infected plants in field test†	Number of PI strains	Number of PI strains tested in greenhouse	Classification of PI strains after greenhouse inoculation		
			All resistant	Resistant and susceptible	All susceptible
0	26	26	7	1	18
3	8	4	1	1	2
6	7	2	-	1	1
9	7	2	1	-	1
Total	48	34	9	3	22

† Percentage based on PMV reaction of approximately 30 plants

Table 26. Reaction of selected PI strains in Maturity Group III to PMV inoculation in the greenhouse in comparison to their reaction to PMV inoculation in the field.

Percentage of infected plants in field test†	Number of PI strains	Number of PI strains tested in greenhouse	Classification of PI strains after greenhouse inoculation		
			All resistant	Resistant and susceptible	All susceptible
0	27	24	16	3	5
3	8	7	2	4	1
6	27	6	2	3	1
9	20	5	5	-	-
Total	82	42	25	10	7

† Percentage based on PMV reaction of approximately 30 plants

In several of the field resistant PI strains retested in the greenhouse (Tables 23 and 24) symptoms did not appear until 2-3 weeks after inoculation and even then they were very mild mottle symptoms. This apparent relationship between plant resistance and length of incubation period for symptom expression was also found in a previously mentioned study of MDMV on corn (Kuhn and Smith, 1977). Perhaps, these PI strains have some type of "field tolerance" to PMV that is broken down only under the more extreme conditions of the greenhouse inoculation test. Demski and Kuhn (1975) reported a breakdown in PMV resistance in the soybean cultivar Hardee when subjected to an extremely high temperature (36 C) for three days prior to and eight days after inoculation. Maturity Group II and III PI strains that were resistant in both field and greenhouse inoculation tests are indicated by footnotes in Tables 23 and 24, respectively.

Seed was saved from one resistant and one susceptible plant in each of 25 selected PI strains that appeared to be segregating for reaction to PMV in the 1976 field test (Maturity Groups II and III). The percentage of infected plants observed in the field in the 25 strains ranged from 15 to 88% with a mean of 49%. Progeny plants were subsequently tested for reaction to PMV in the greenhouse (Table 27). Only four of the field tested plants had progeny that were resistant to PMV and in each instance it was only one progeny plant. Each of the two field susceptible plants from PI strains 86,741 and 88,508 had one progeny plant that was resistant in the greenhouse test. Such an occurrence is possible if the parent plant was infected with a virus other than PMV in the field and was misclassified as susceptible when it was

Table 27. PMV reaction of greenhouse inoculated progenies of one resistant and one susceptible plant selected from each of 25 field tested PI strains from Maturity Groups II and III.

PI number	Percentage of field infected plants	Resistant parent plant		Susceptible parent plant	
		Number of progeny plants inoculated	Reaction to PMV†	Number of progeny plants inoculated	Reaction to PMV
(Maturity Group II)					
68,661	24	-	-	4	S
68,704	33	4	S	3	S
68,729	42	1	S	3	S(M)††
68,778	33	7	S	5	S
69,992	66	5	S	5	S
70,077	36	2	S	3	S(M)
70,197	15	2	S	2	S
72,342	76	3	S	3	S(M)
73,583	88	-	-	4	S
73,587	82	4	S(M)	5	S
86,050	54	6	S	5	S
86,741	33	6	S(M)	5	1R,4S(M)
88,508	27	5	1R,4S(M)	6	1R,5S(M)
91,138	51	-	-	4	S

† S = susceptible to PMV; R = resistant to PMV.

†† M = mild mottle on trifoliolate leaves that developed 2-4 weeks after inoculation.

Table 27, continued.

PI number	Percentage of field infected plants	Resistant parent plant		Susceptible parent plant	
		Number of progeny plants inoculated	Reaction to PMV†	Number of progeny plants inoculated	Reaction to PMV
92,595	24	2	1R,1S	1	S(M)
92,748	45	-	-	5	S
291,293A	33	4	S	3	S
	Total	51	2R,49S	66	2R,64S
(Maturity Group III)					
70,466-4	51	5	S(M)	5	S(M)
79,874	54	4	S	3	S
80,844-2	54	1	S	2	S
86,456	36	2	S	2	S
86,502	51	5	S	6	S
89,784	51	1	S	3	S
196,157	75	4	S(M)	1	S
229,345	82	2	S	2	S
	Total	24	24S	24	24S

† S = susceptible to PMV; R = resistant to PMV.

actually resistant to PMV but heterozygous. It could also be due to an inoculation escape. The greenhouse test data (Table 27) suggest that non-infected plants observed in strains that showed a sizeable percentage of infected plants probably were escapes rather than resistant plants. Such escapes are most likely the result of uneven germination and emergence which results in delayed inoculation for the earliest and fastest growing plants. The larger, more mature plants showed no symptoms but those plants inoculated at a younger stage of growth were susceptible as pointed out by Demski and Kuhn (1975).

Progenies of 106 individual field resistant plants from FC and PI strains of Maturity Group III were also inoculated with PMV in the greenhouse (Table 28). The airbrush inoculation technique was used rather than the mortar and pestle technique. A relatively high percentage (63%) of parent plants from PI strains showing 0-10% infection in the field produced progeny plants that were all resistant to PMV. The uniform resistant reaction of the progeny plants indicates genetic homozygosity for reaction to PMV within the parental plants. Conversely, it was noted that the percentage of field escapes tended to increase with an increase in the percentage of field infected plants (Table 28). Progenies of resistant plants from strains that showed more than 10% infection in the field had an approximate 60% level of segregating progenies for reaction to PMV. This level of segregation suggests that some plants in the FC/PI strains are heterozygous for reaction to PMV. These results confirm the segregation observed within PI strains in the preliminary greenhouse evaluation test (Table 6). Due to the relatively small average number (5) of progenies per plant, it was not

Table 28. Reaction of progenies of individual PMV field resistant plants of Maturity Group III FC and PI strains to inoculation with PMV in the greenhouse.

Percentage of infected plants in field test	Number of field resistant parental plants†		Classification of reactions of progenies to PMV inoculation in greenhouse		
			All resistant	Resistant and susceptible	All susceptible
0-10	27	Number	17	9	1
		Percentage	63	33	4
11-20	34	Number	10	21	3
		Percentage	29	62	9
21-30	21	Number	5	13	3
		Percentage	24	62	14
31-50	15	Number	4	9	2
		Percentage	27	60	13
Over 50	9	Number	1	6	2
		Percentage	11	67	22
Total	106	Number	37	58	11
		Percentage	35	55	10

† Each plant represents a different FC or PI strain.

feasible to draw any conclusions about inheritance patterns in the segregating progenies.

Table 29 lists the FC and PI strains in Maturity Group IV that showed 10% or less PMV-infected plants in the 1977 field test. There was a higher percentage of Maturity Group IV strains in this category than there was in Maturity Groups II and III (Table 30). Table 30 also shows that the mean percentage of infected plants for all Maturity Group IV strains showing 11 to 100% infected plants was lower than for Maturity Groups II and III.

Two explanations are advanced to explain the lower mean percentage value in the 1977 field test and the higher percentage of Maturity Group IV strains in the 0-10% category. Due to a dry period after planting germination and emergence was very uneven in 1977, especially for Entries 1-614 planted May 23-24. Thus, at the time of inoculation some plants were past the optimum stage for inoculation. These plants appeared to be resistant while the plants inoculated at an earlier stage became infected with PMV. The cultivar Hood used as a susceptible check in the 1977 test had a mean value of 29% infected plants. The low value is also attributed to uneven germination and emergence.

A second hypothesis is also suggested. Maturity Group IV strains originate from latitudes where they are more likely to have been previously exposed to a virus that also infects peanut plants. Thus one might expect a higher percentage of Group IV strains to be resistant to PMV due to the prior co-evolution of the two organisms. Whereas, Maturity Group II and III strains would tend to be more susceptible to PMV due to no prior exposure to the disease.

Table 29. Maturity Group IV soybean FC/PI strains having 0-10% PMV-infected plants from field inoculation test of 959 strains at Blacksburg, 1977.

FC/PI number	Percentage of infected plants	Disease severity
19,976-1	0	-
19,976-2	0	-
19,979-1	0	-
19,979-6	0	-
31,702	10	1
19,986	0	-
54,606-1	0	-
54,608-4	0/4[†]	-
62,202-2	0	-
68,692	0	-
70,013	10	2
70,242-2	1/10	1
71,506	0	-
79,743	0	-
80,466-2	10	1
80,479	5	1
80,847-2	10	3
81,764	0	-
81,777	0	-
82,210	0	-
82,218	0	-
82,264	0	-
82,315	0	-
82,325	5	2
82,554	10	1
82,555	0	-
83,858	0	-
84,594	0	-
84,639	0	-
84,671	10	1

[†] In rows with less than 11 plants, the number of susceptible plants/total number of plants was recorded.

Table 29, continued.

FC/PI number	Percentage of infected plants	Disease severity
84,679	10	2
84,724	1/10	2
84,751	0	-
85,420	1/7	2
85,469	0	-
85,505	1/6	1
85,658	1/6	1
86,062	0/9	-
86,103	5	1
86,109B	0	-
86,134-4	10	1
86,136	1/10	1
86,740	0	-
86,908	0	-
87,011	0/5	-
87,013	0	-
87,029	1/6	1
87,561	0	-
87,606-1	10	2
87,620-1	5	1
87,629	0	-
87,631-3	5	2
87,632	5	2
88,302-1	10	1
88,490-2	0	-
88,491	10	1
89,769	0	-
89,772	0	-
90,256	0	-
90,401	0	-
90,499-2	10	2
90,763	0	-
91,082	10	2
91,100-4	10	1
91,103	0/10	-
91,108-1	0	-
91,108-2	10	2
91,133	10	1

Table 29, continued.

FC/PI number	Percentage of infected plants	Disease severity
91,346	5	1
91,679	0	-
91,731-1	5	2
91,733-1	0	-
92,604	0	-
92,641B	10	2
95,801	0	-
95,853	10	1
96,333	5	1
96,783	10	2
96,984	0	-
103,091	10	2
157,398	5	2
157,404	0	-
157,405	5	1
157,409	0	-
157,410	0	-
157,428	0	-
157,431	5	1
157,435	0	-
157,436	1/8	2
157,439	5	2
157,447	10	1
157,449	0	-
157,454	0	-
157,458	5	1
157,459	0	-
157,462	10	2
157,468	10	1
157,483	10	1
157,485	0	-
157,492	0	-
158,765	10	1
159,923-1	0	-
171,427	0	-
181,550	0	-
181,551	0	-
181,555	0	-

Table 29, continued.

FC/PI number	Percentage of infected plants	Disease severity
181,557	0	-
200,460	0	-
200,463	10	1
200,504	0/9	-
200,519	0	-
200,536	10	2
200,541	0	-
205,089	0	-
209,332	0	-
219,782	0	-
219,787	0	-
224,271	0	-
226,591	0	-
229,313	0	-
229,314	0	-
229,319	0	-
229,325	0	-
229,327	0	-
229,341	0	-
229,342	0	-
229,343	0/9	-
229,349	0	-
229,352	0	-
229,353	0	-
229,359	0	-
229,361	0	-
229,362	0	-
235,335	0	-
235,340	0	-
235,344	0	-
243,520	0/3	-
243,521	0	-
243,523	0/5	-
243,530	0/2	-
243,533	0/6	-
243,535	1/6	1
243,541	10	2
243,544	0	-

Table 29, continued.

FC/PI number	Percentage of infected plants	Disease severity
243,545	0	-
243,548	0	-
246,367	0	-
246,368	0	-
246,369	0/9	-
248,514	0/8	-
262,181	0	-
264,555	0	-
273,483D	0	-
273,484	10	3
274,205	5	2
274,209	0	-
274,212	0	-
274,420	0	-
274,423	0	-
339,864A	0	-
339,868B	0	-
339,868C	0	-
339,981	0/4	-
339,984	10	1
339,997	10	2
340,017	5	1
340,038	5	1
340,046	5	2
342,004	0/2	-
342,005	0/6	-
360,842	1/6	2
360,845	0/9	-
360,847	0	-
360,848	0	-
385,942	0	-
398,223	0	-
398,225	10	1
398,226	5	2
398,229	10	1
398,242	0	-
398,243	0	-
398,244	10	1

Table 29, continued.

FC/PI number	Percentage of infected plants	Disease severity
398,268	0	-
398,274	5	1
398,276	10	1
398,278	10	2
398,289	0	-
398,299	10	2
398,306	0	-
398,317	5	1
398,321	5	2
398,322	0/9	-
398,356	0	-
398,357	0	-
398,374	5	3
398,379	5	3
398,386	0	-
398,392	5	1
398,404	0	-
398,429	5	1
398,472	0	-
398,495	0/7	-
398,499	1/10	1
398,519	10	2
398,621	5	1
398,636	1/5	1
398,657	10	1
398,701	10	1
398,739	10	2
398,816	10	2
398,857	5	1
398,858	0	-
398,871	10	2
398,873	10	1
398,878	5	1
398,880	5	1
398,884	0	-
398,887	0	-
398,890	10	2
398,914	5	2
398,919	0	-
398,939	0	-

Table 29, continued.

FC/PI number	Percentage of infected plants	Disease severity	FC/PI number	Percentage of infected plants	Disease severity
398,944	0	-	398,996	0	-
398,953	10	2	399,005	5	1
398,964	5	2	399,022	5	2
398,970	5	1	399,023	0	-
398,985	0	-	399,039	5	2
398,988	5	2	399,051	0	-
398,990	0	-	399,119	5	3

Table 30. Summary of PMV field inoculation tests on soybean FC/PI strains at Blacksburg.

Maturity Group	Year of test	Number and percentage of FC/PI strains				Total inoculated	Mean percentage of infection in strains with >10% infected plants
		0% PMV infected plants		1-10% PMV infected plants			
II	1976	26	(5%)	22	(4%)	556	66%
III	1976	27	(4%)	55	(8%)	646	65%
IV	1977	140	(15%)	96	(10%)	959	50%
	Total	193	(9%)	173	(8%)	2161	

As has been shown for Maturity Groups II and III, field inoculation using the airbrush technique is not as efficient as the mortar and pestle inoculation technique under greenhouse conditions (Tables 25, 26, and 27). Thus, the Maturity Group IV strains shown in Table 29 should be tested under greenhouse conditions to verify field results and to identify those strains that have a high level of resistance to PMV. However, it is most probable that a field test using the airbrush inoculation technique to inoculate plants is more similar to natural transmission by aphid vectors than is inoculation by rubbing (mortar and pestle).

V. SUMMARY AND CONCLUSIONS

Genetic studies were conducted to determine the inheritance of reaction and to identify different genes and/or alleles for resistance to PMV-S/V74S in soybeans. The same PMV strain was used in field inoculation tests in 1976 and 1977 to identify new sources of resistance in Maturity Groups II, III, and IV of the soybean Plant Introduction germplasm collection.

The cultivars Virginia and Pine Dell Perfection were reported to be resistant to a Georgia PMV isolate (PMV-M) but were found to be susceptible to PMV-S/V74S. These findings suggest their possible use as differential cultivars to distinguish different PMV strains isolated in the field.

A partial diallel cross involving 11 resistant and two susceptible lines was made in 1976. Advanced generations from selected crosses were evaluated in the greenhouse to determine their reaction to PMV. The F_2 and F_3 genetic ratios observed in populations derived from crosses between resistant and susceptible types indicate that resistance in each of the 11 lines is monogenically controlled. However, different genes for resistance to PMV were identified in some of the resistant parental types by testing F_2 and F_3 generation plants derived from crosses among these resistant types.

Reaction to inoculation with PMV of advanced generation plants derived from crosses between Peking and both susceptible and resistant types provides evidence that resistance in Peking is controlled by a recessive gene, designated rpv_2. This is the first report of a

recessive gene for resistance to PMV in soybeans. The F_2 population from the cross between Peking and the susceptible type, PI 229,315, segregated 1R:3S whereas F_2 populations from crosses between eight other resistant types and PI 229,315 segregated 3R:1S. A 1R:3S F_2 ratio is expected if the gene conditioning resistance in Peking is recessive and the corresponding gene controlling the susceptible reaction in PI 229,315 is dominant. A 13R:3S ratio was observed in F_2 populations derived from crosses of Peking with the three resistant types, Arksoy, PI 89,784, and PI 219,789. Thirty-five F_3 lines from the Peking X PI 219,789 cross segregated in a manner that demonstrated conclusively the presence of a recessive gene for resistance to PMV in Peking.

Three different dominant genes for resistance to PMV-S/V74S are suggested by the 15R:1S F_2 genetic ratios observed in crosses between resistant types. The dominant gene for resistance (Rpv_1) carried by Arksoy was shown to differ from the gene for resistance (Rpv_3) in PI 89,784, PI 219,789, and five other resistant types. The F_2 data from crosses between CNS and susceptible and resistant types suggests that CNS has a dominant resistance gene (Rpv_4) that differs from Rpv_1 and Rpv_3. In a similar manner, PI 54,613 was shown to carry a dominant gene for resistance that is unlike Rpv_1 and Rpv_3. However, the relationship between PMV resistance genes in CNS (Rpv_4) and PI 54,613 ($Rpv_?$) is unknown.

The susceptible lines in the partial diallel cross, Virginia and PI 229,315, differed in their reaction to PMV. Virginia showed mild, systemic mottle symptoms while PI 229,315 exhibited severe stunting, leaf yellowing, and formation of necrotic spots when inoculated

with PMV. All F_2 plants derived from a cross between these two types were found to be susceptible but symptom differences were noted. The presence of different alleles or modifying genes conditioning the susceptible reaction in the two susceptible types appears likely but their genetic relationship is unclear.

Preliminary data were obtained from F_1 and F_2 generation plants derived from crosses between PMV-resistant PI strains and two resistant testers, York and PI 89,784. Observed ratios in five F_2 populations derived from 1976 crosses between resistant PIs and York indicate the possibility of three recessive genes and two dominant genes for resistance that are non-allelic to the York gene. The absence of susceptible plants in two F_2 populations suggest that PI 219,782 and PI 246,367 carry the same dominant gene for resistance as does York. Testing of larger numbers of F_2 plants from each of these seven crosses as well as testing of F_3 generation plants is suggested before any conclusive statements are made.

The reaction to inoculation with PMV was determined for 10 F_1 plants derived from the 1977 crosses between resistant PI strains and the two resistant testers. Nine of the plants were resistant while one plant exhibited mild mottle symptoms. Future testing of F_2 and F_3 populations derived from these crosses may lead to the identification of other new genes for resistance to PMV. Since York and PI 89,784 were used in 1976 and 1977 crosses, they can be used to interrelate F_2 and F_3 genetic ratios observed in populations from crosses made in both years.

A total of 2161 FC and PI strains in Maturity Groups II, III, and IV were inoculated with PMV-S/V74S in the field during 1976 and 1977.

In the two field tests 366 strains showed less than 10% infection. Retesting in the greenhouse showed that a sizeable percentage of Maturity Group II field resistant PI's were susceptible. Differences in field and greenhouse results were attributed to three factors. Due to non-uniform germination and emergence in the field, older, more mature plants gave a resistant reaction to PMV while other plants within the same PI strain that were inoculated at an earlier stage of growth gave a susceptible reaction. In contrast, all seedlings were inoculated at an earlier, nearly identical stage of growth in the greenhouse. Secondly, different environmental conditions may have affected the different reactions in the field and greenhouse. Higher temperatures in the greenhouse could have been a factor in breaking down the previously observed field resistance. Finally, rub inoculation using the mortar and pestle appears to be a more efficient inoculation technique than the artist's airbrush technique. However, the airbrush technique may be more similar to the natural, aphid-transmission of PMV and thus a better tool to identify field resistant genotypes.

Progenies of individual field "resistant" plants from 106 PI strains in Maturity Group III were inoculated in the greenhouse using the airbrush technique. As shown by the progeny evaluation test, the number of field escapes tended to increase as the percentage of field infected plants in the PI strain increased.

A large number of progenies segregated for reaction to PMV indicating that the parent plants were heterozygous. Although segregation in the greenhouse was also observed in plants that were inoculated by rubbing, it appears that escapes occur more frequently when the airbrush

technique is used. It is suggested that many of the "resistant" plants in segregating progenies were inoculation escapes.

VI. LITERATURE CITED

Ali, Mohamed A. 1950. Genetics of resistance to the common bean mosaic virus (Bean Virus 1) in the bean (Phaseolus vulgaris L.). Phytopathology 40:69-79.

Allard, R. W. 1960. Principles of plant breeding. John Wiley and Sons, Inc., New York.

Arny, D. C., and H. Jedlinski. 1966. Resistance to the yellow dwarf virus in selected barley varieties. Plant Dis. Rep. 50:380-381.

Baggett, J. R., and W. A. Frazier. 1957. The inheritance of resistance to bean yellow mosaic virus in Phaseolus vulgaris. J. Am. Soc. Hort. Sci. 70:325-333.

Baker, E. P. 1966. Isolation of complementary genes conditioning crown rust resistance in the oat variety Bond. Euphytica 15:313-318.

Behncken, G. M. 1970. The occurrence of peanut mottle virus in Queensland. Aust. J. Agric. Res. 21:465-472.

Behncken, G. M., and G. J. P. McCarthy. 1973. Peanut mottle virus in peanuts, navy beans, and soybeans. Queensland Agric. J. 99:635-637.

Bernard, R. L., and C. R. Cremeens. 1969. Evaluation of Maturity Groups III and IV of the U.S.D.A. soybean collection. RSLM 238. U.S. Regional Soybean Laboratory. ARS-USDA. Urbana, Illinois.

Biffin, R. H. 1905. Mendel's laws of inheritance and wheat breeding. J. Agric. Sci. 1:4-48.

Bock, K. R. 1973. Peanut mottle virus in East Africa. Ann. Appl. Biol. 74:171-179.

Bock, K. R., and C. W. Kuhn. 1975. Peanut mottle virus. Descriptions of plant viruses. No. 141. Commonwealth Mycological Institute and Association of Applied Biologists. Surrey, England.

Boerma, H. R., and C. W. Kuhn. 1976. Inheritance of resistance to peanut mottle virus in soybeans. Crop Sci. 16:533-534.

Boerma, H. R., C. W. Kuhn, and H. B. Harris. 1975. Inheritance of resistance to cowpea chlorotic mottle virus (soybean strain) in soybeans. Crop Sci. 15:849-850.

Bradfute, O. E., R. Louie, and J. K. Knoke. 1972. Isometric virus-like particles in maize with stunt symptoms. Phytopathology 62:748 (Abstr.).

Briggs, F. N. 1926. Inheritance of resistance to bunt Tilletia tritici (Bjerk.) Winter, in wheat. J. Agric. Res. 32:973-990.

Bruehl, G. W. 1961. Barley yellow dwarf. Am. Phytopathol. Soc. Monogr. No. 1.

Caldwell, B. E. 1973. Soybeans: Improvement, production, and uses. Agronomy 16 (Preface). Am. Soc. of Agron., Madison, Wis.

Cirulli, M., and L. J. Alexander. 1969. Influence of temperature and strain of tobacco mosaic virus on resistance in a tomato breeding line derived from Lycopersicon peruvianum. Phytopathology 59: 1287-1297.

Conover, R. A. 1948. Studies of two viruses causing mosaic diseases of soybean. Phytopathology 38:724-735.

Cousin, R. 1965. Etude de la sensibilite des varietes de pois au virus de la mosaique commune du pois. Etude genetique de la resistance. Ann. Amelior. Plantes 15:23-36.

Damsteegt, V. D., and G. W. Bruehl. 1964. Inheritance of resistance in barley to barley yellow dwarf. Phytopathology 54:219-222.

Day, P. R. 1974. Genetics of host-parasite interaction. W. H. Freeman and Company, San Francisco.

Demski, J. W. 1974. Spread of peanut mottle virus in soybeans. Proc. Am. Phytopathol. Soc. 1:37.

Demski, J. W., and H. B. Harris. 1974. Seed transmission of viruses in soybeans. Crop Sci. 14:888-890.

Demski, J. W., and M. D. Jellum. 1975. Single and double virus infection of soybean: Plant characteristics and chemical composition. Phytopathology 65:1154-1156.

Demski, J. W., and C. W. Kuhn. 1974. Source of peanut mottle virus in soybean and peanut. Proc. Am. Phytopathol. Soc. 1:158.

Demski, J. W., and C. W. Kuhn. 1975. Resistant and susceptible reaction of soybeans to peanut mottle virus. Phytopathology 65:95-99.

Demski, J. W., and C. W. Kuhn. 1977. A soybean disease caused by peanut mottle virus. Georgia Agric. Exp. Stn. Res. Bull. 196.

Demski, J. W., D. H. Smith, and C. W. Kuhn. 1975. Incidence and distribution of peanut mottle virus in peanut in the United States. Peanut Sci. 2(2):91-93.

Dollinger, E. J., W. R. Findley, and L. E. Williams. 1970. Inheritance of resistance to maize dwarf mosaic virus in maize (Zea mays L.).

Crop Sci. 10:412-415.

Drijfhout, E. 1975. Classification of strains of bean common mosaic virus and genetic interaction between these strains and Phaseolus vulgaris L. In Proceedings of bean plant protection workshop. Dec. 1-3, 1975. Centro Internacional de Agricultura Tropical (CIAT). Cali, Colombia.

Dunleavy, J. M. 1973. Viral diseases. In B. E. Caldwell (ed.) Soybeans: Improvement, production, and uses. Agronomy 16:505-526. Am. Soc. of Agron., Madison, Wis.

Dunn, G. M., and T. Namm. 1970. Gene dosage effects on monogenic resistance to northern corn leaf blight. Crop Sci. 10:352-354.

Edmondson, E. R. 1972. Studies of potato virus Y on tobacco: Inheritance of resistance and interaction with other viruses. Ph.D. Thesis. Virginia Polytechnic Institute and State University, Blacksburg.

Findley, W. R., R. Louie, J. K. Knoke, and E. J. Dollinger. 1976. Breeding corn for resistance to virus in Ohio. p. 123-128. In L. E. Williams, D. T. Gordon, and L. R. Nault (ed.) Proceedings international maize virus disease colloquium and workshop. Aug. 16-19, 1976. Ohio Agric. Res. and Dev. Ctr., Wooster.

Flor, H. H. 1956. The complementary genic systems in flax and flax rust. Adv. Genet. 8:29-54.

Hagedorn, D. J. 1973. Peas. p. 326-343. In R. R. Nelson (ed.) Breeding plants for disease resistance. The Pennsylvania State University Press, University Park.

Herold, F., and K. Munz. 1969. Peanut mottle virus. Phytopathology 59: 663-666.

Holmes, F. O. 1938. Inheritance of resistance to tobacco mosaic disease in tobacco. Phytopathology 28:553-561.

Holmes, F. O. 1960. Inheritance in tobacco of an improved resistance to infection by tobacco mosaic virus. Virology 12:59-67.

Hooker, A. L., and K. M. S. Saxena. 1971. Genetics of resistance in plants. Annu. Rev. Genet. 5:407-424.

Horsfall, J. G., et al. 1972. Genetic vulnerability of major crops. Report to National Academy of Sciences. Washington, D.C.

Inouye, T. 1969. Peanut mottle virus from peanuts and peas. Ber. Ohara Inst. Landwirtsch Biol. Okayoma Univ. 52:159-164.

Johnson, G. R. 1971. Analysis of genetic resistance to maize dwarf

mosaic disease. Crop Sci. 11:23-24.

Johnson, K. W., and D. J. Hagedorn. 1958. The inheritance of resistance to bean virus 2 in Pisum sativum. Phytopathology 48:451-453.

Josephson, L. M., J. W. Hilty, and Jerry Arnold. 1967. Inheritance of tolerance of corn inbreds to the maize dwarf mosaic virus. Agron. Abstr. p. 13.

Kiihl, R. A. de Souza. 1976. Inheritance studies of two characteristics in soybeans (Glycine max [L.] Merrill): I. Resistance to soybean mosaic virus II. Late flowering under short-day conditions. Ph.D. Thesis. Mississippi State University, Starkville.

Koelle, G. 1961. Genetische analyse einer Y-virus (Rippenbraune) resistenten mutante der tabaksorte Virgin A. Der Zuchter 31:71-72.

Koshimizu, Y., and N. Iizuka. 1963. Studies on soybean virus diseases in Japan. Tohoku Natl. Agric. Exp. Stn. Bull. 27:1-103.

Kuhn, C. W. 1965. Symptomatology, host range, and effect on yield of a seed-transmitted peanut virus. Phytopathology 55:880-884.

Kuhn, C. W., and J. W. Demski. 1975. The relationship of peanut mottle virus to peanut production. Georgia Agric. Exp. Stn. Res. Rep. 213.

Kuhn, C. W., J. W. Demski, and H. B. Harris. 1972. Peanut mottle virus in soybeans. Plant Dis. Rep. 56:146-147.

Kuhn, C. W., and T. H. Smith. 1977. Effectiveness of a disease index system in evaluating corn for resistance to maize dwarf mosaic virus. Phytopathology 67:288-291.

Kuhn, C. W., G. Sowell, Jr., J. H. Chalkley, and H. F. Stubbs. 1968. Screening for immunity to peanut mottle virus. Plant Dis. Rep. 52:467-468.

Lee, Y. S., and J. P. Ross. 1972. Top necrosis and cellular changes in soybean doubly infected by soybean mosaic and bean pod mottle viruses. Phytopathology 62:839-845.

Leong, J. L. 1976. The interaction of peanut mottle virus and soybean mosaic virus on soybean (Glycine max [L.] Merrill). M.S. Thesis. Virginia Polytechnic Institute and State University, Blacksburg.

Loesch, P. J., and M. S. Zuber. 1967. An inheritance study of resistance to maize dwarf mosaic virus in corn (Zea mays L.). Agron. J. 59: 423-426.

Loesch, P. J., and M. S. Zuber. 1972. Inheritance of resistance to maize dwarf mosaic virus. Crop Sci. 12:350-352.

Louie, R., J. K. Knoke, and D. T. Gordon. 1974. Epiphytotics of maize dwarf mosaic and maize chlorotic dwarf diseases in Ohio. Phytopathology 64:1455-1459.

Lupton, F. G. H., and R. C. F. Macer. 1962. Inheritance of resistance to yellow rust (Puccinia glumarum Erikss. and Henn.) in seven varieties of wheat. Trans. Br. Mycol. Soc. 45:21-45.

Nault, L. R., W. E. Styer, J. K. Knoke, and H. N. Pitre. 1973. Semipersistent transmission of leafhopper-borne maize chlorotic dwarf virus. J. Econ. Entomol. 66:1271-1273.

Nolla, J. A. B. 1938. Inheritance in Nicotiana. III. A study of the character for mosaic resistance in Nicotiana tabacum L. J. Hered. 29:43-48.

Noordam, D. 1973. Identification of plant viruses: Methods and experiments. Centre Agric. Pub. Doc., Wageningen.

Paguio, O. R., and C. W. Kuhn. 1973a. Purification of a mild mottle strain of peanut mottle virus. Phytopathology 63:720-724.

Paguio, O. R., and C. W. Kuhn. 1973b. Strains of peanut mottle virus. Phytopathology 63:976-980.

Paguio, O. R., and C. W. Kuhn. 1974a. Survey for peanut mottle virus in peanut in Georgia. Plant Dis. Rep. 58:107-110.

Paguio, O. R., and C. W. Kuhn. 1974b. Incidence and source of inoculum of peanut mottle virus and its effect on peanut. Phytopathology 64:60-64.

Paguio, O. R., and C. W. Kuhn. 1976. Aphid transmission of peanut mottle virus. Phytopathology 66:473-476.

Person, C., and G. Sidu. 1971. Genetics of host-parasite interrelationships. p. 31-38. In Mutation breeding for disease resistance. Panel Proceedings Series. Oct. 12-16, 1970. IAEA and FAO, Vienna.

Riley, R., and R. C. F. Macer. 1966. The chromosomal distribution of the genetic resistance of rye to wheat pathogens. Can. J. Genet. Cytol. 8:640-653.

Roane, C. W. 1962. Inheritance of reaction to Puccinia hordei in barley. I. Genes for resistance among North American race differentiating varieties. Phytopathology 52:1288-1295.

Roane, C. W., and S. A. Tolin. 1974. Effects of four viruses upon four soybean cultivars. Proc. Am. Phytopathol. Soc. 1:36-37.

Roane, C. W., and S. A. Tolin. 1977. Virus diseases and brown stem rot

of soybeans in Virginia. Research Report to Virginia Soybean Commission. Virginia Polytechnic Institute and State University, Blacksburg.

Roane, C. W., S. A. Tolin, and P. H. Reid. 1978. Effects of peanut mottle virus (PMV) on twenty-five soybean cultivars. Potomac Division, Am. Phytopathol. Soc., 35th Annual Meeting. (Abstr.).

Ross, J. P. 1969. Pathogenic variation among isolates of soybean mosaic virus. Phytopathology 59:829-832.

Schaller, C. W., C. O. Qualset, and J. N. Rutger. 1964. Inheritance and linkage of the Yd2 gene conditioning resistance to the barley yellow dwarf virus disease in barley. Crop Sci. 4:544-548.

Schmidt, H. B., and K. Schmelzer. 1966. Elektronemikroskopische Darstellung und Vermessung eines Saftübertragbaren Virus aus der ErdnuB (Arachis hypogaea L.). Phytopathol. Z. 55:92.

Schroeder, W. T., and D. W. Barton. 1958. The nature and inheritance of resistance to the pea enation mosaic virus in garden pea, Pisum sativum L. Phytopathology 48:628-632.

Schroeder, W. T., D. W. Barton, and W. Mishanec. 1966. Temperature differentiation of genotypes for BV^2 resistance in Pisum sativum. Phytopathology 56:113-117.

Silber, G., H. E. Heggestad, and R. P. Kuhn. 1965. A tobacco with tolerance to potato virus Y. Phytopathology 55:1076 (Abstr.).

Silbernagel, M. J., and W. J. Zaumeyer. 1973. Beans. p. 253-269. In R. R. Nelson (ed.) Breeding plants for disease resistance. The Pennsylvania State University Press, University Park.

Sinclair, J. B., and M. C. Shurtleff. 1975. Compendium of soybean diseases. Am. Phytopathol. Soc., St. Paul, Minn.

Snedecor, G. W., and W. G. Cochran. 1967. 6th ed. Statistical methods. The Iowa State University Press, Ames.

Sun, M. K. C. 1974. Epidemiology of peanut mottle virus in North Carolina. Proc. Am. Phytopathol. Soc. 1:49.

Sun, M. K. C., and T. T. Hebert. 1972. Purification and properties of a severe strain of peanut mottle virus. Phytopathology 62:832-839.

Suneson, C. A. 1955. Breeding for resistance to yellow dwarf virus in barley. Agron. J. 47:283.

Ting, W. P., S. L. Geh, and Y. C. Lim. 1972. Studies on groundnut mosaic virus of Arachis hypogaea L. in West Malaysia. Exp. Agric. 8:355-368.

Tolin, S. A., R. H. Ford, and C. W. Roane. 1974. Purification and serology of peanut mottle virus from soybean and peanut. Proc. Am. Phytopathol. Soc. 1:114.

Tolin, S. A., and C. W. Roane. 1975. Identification and distribution of soybean viruses in Virginia. Proc. Am. Phytopathol. Soc. 2:129.

Walker, J. C. 1957. Plant pathology. McGraw-Hill Book Company, Inc., New York.

Weiss, M. G., and T. M. Stevenson. 1955. Registration of soybean varieties, V. Agron. J. 47:541.

Yen, D. E., and P. R. Fry. 1956. The inheritance of immunity to pea mosaic virus. Aust. J. Agric. Res. 7:272-280.

Zaumeyer, W. J., and J. P. Meiners. 1975. Disease resistance in beans. Annu. Rev. Phytopathology 13:313-334.

Zuber, M. S., E. S. Hilderbrand, P. J. Loesch, and A. J. Keaster. 1973. Prediction of reactions to maize dwarf mosaic virus in double-cross hybrids based upon single-cross reaction. Crop Sci. 13:172-175.

The vita has been removed from the scanned document

GENETICS OF REACTION TO PEANUT MOTTLE VIRUS IN SOYBEANS

by

Emerson Russell Shipe

(ABSTRACT)

Experiments were conducted at Blacksburg from 1975 to 1978 on soybean, Glycine max (L.) Merrill. The objectives were: (1) to study the inheritance of reaction to peanut mottle virus (PMV); (2) to determine the allelic relationships between genes for resistance from various germplasm sources; and (3) to screen a part of the soybean Plant Introduction germplasm collection and identify PMV-resistant strains.

Soybean cultivars, Plant Introduction (PI) strains, and advanced generation progenies derived from selected crosses were artificially inoculated with PMV-S/V74S (a Virginia isolate) and evaluated for their reaction to PMV in the field and greenhouse. Two cultivars, 'Virginia' and 'Pine Dell Perfection', that were previously reported as resistant to a mild PMV strain were found to be susceptible to PMV-S/V74S.

Crosses between resistant and susceptible lines and among resistant lines were made in the field in 1976 and 1977. The F_1, F_2, and F_3 generation seedlings derived from selected crosses were tested for reaction to PMV in the greenhouse. It was shown that resistance in the cultivar 'Peking' is conditioned by a single recessive gene designated rpv_2. Evidence based on segregation in F_2 populations was also presented that indicates the presence of three other dominant genes for resistance to PMV-S/V74S. The three dominant genes are designated Rpv_1 (first

reported by workers in Georgia), Rpv_3, and Rpv_4.

Preliminary F_1 and F_2 data were obtained from crosses between 15 resistant PI strains and two resistant "testers," 'York' and PI 89,784. The F_2 data obtained from five crosses indicate the possibility of still other genes for resistance to PMV. The two susceptible lines used in the study, Virginia and PI 229,315, differed markedly in their reactions to PMV. The presence of different alleles or modifying genes controlling the susceptible reactions in the two lines is suggested.

A total of 2161 FC and PI strains in Maturity Groups II, III, and IV were inoculated with PMV-S/V74S in the field during 1976 and 1977. Three hundred sixty-six strains that showed 10% or less virus infection were identified. These "resistant" strains provide a pool from which other genes for PMV resistance perhaps can be isolated. Differences in PMV disease reactions of plants from the same strain were noted when plants were tested in both the field and greenhouse. The differences were attributed to the following three factors: (1) differences in stage of plant growth at time of inoculation (field grown plants were generally larger at time of inoculation than plants inoculated in the greenhouse); (2) differences in environmental conditions between the field and greenhouse; and (3) the artist's airbrush inoculation technique was used in the field while the rub inoculation technique (mortar and pestle) was used in the greenhouse.