In a separate study 135 eggs from nonmutant pairs and 178 eggs from high-insert pairs were placed on standard larval diet⁴ and observed on a continuing basis. Egg hatch percentages were 73 and 71 percent for the nonmutant and mutant eggs, respectively. The percent eggs that developed to adults was 69 percent (79) for nonmutants and 48 percent (85) for mutants. As reported above, the mutant progeny exhibited an inability to attain normal body configuration at eclosion with 44 percent (27) of the high-insert adults failing to contribute to the succeeding generation compared to the 5 percent failure of nonmutant adults. The 178 eggs from mutant pairs produced 85 progeny including 57 high-insert and 28 nonmutant (0:2:1).

Summary

An autosomal, dominant lethal mutant of the boll weevil, high-insert, is described. This mutation is characterized phenotypically by having the antennal insertion much nearer the eyes than normal. Matings between highinsert and nonmutants result in a 1:1 mutant-nonmutant ratio, and mutant/mutant matings yielded a 2:1 mutant-

Inheritance of resistance to plantago mottle virus in *Pisum sativum* L.

R. Provvidenti

T HE OCCURRENCE of plantago mottle virus (PIMV) in pea crops in New York state was recently reported by Provvidenti and Granett². The low incidence of field infection was attributed to the resistance to P1MV of many leading commercial cultivars, and to the limiting conditions for an efficient transmission of the virus by the vector(s)².

The purpose of this study was to elucidate the inheritance of resistance to PIMV in *Pisum sativum* L., and determine whether factors for resistance to P1MV and bean yellow mosaic virus (BYMV) are linked. Previously², a large number of P1MV-resistant cultivars were found to be also resistant to BYMV. Resistance to BYMV is controlled by a single recessive gene⁴ (mo), which in heterozygous condition (Molmo) is strongly influenced by temperature³.

Materials and Methods

Genetic populations were derived from crosses between Bonneville, a cultivar resistant to PIMV and

nonmutant ratio. In addition more than one-half of the mutant females failed to oviposit.

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BYMV, and Ranger, a cultivar susceptible to both viruses². Plants of these cultivars and F_1 , F_2 , and reciprocal backcross populations were mechanically inoculated with the type strain of P1MV originally recovered from *Plantago major*¹. For linkage detection, plants of the testcross (Bonneville × Ranger) F_1 × Ranger were first inoculated with PIMV and later with a strain of BYMV, used in a previous study². Inocula were derived from Ranger plants showing distinct symptoms of PIMV or BYMV. All plants were incubated at 18°C, using environmental chambers illuminated for 14 hours daily, with 45,-000 lux at the plant level. At this temperature, PIMV-infected plants exhibited prominent symptoms², and the *mo* gene behaved as dominant in the heterozygous plants³.

Results

Plants of Bonneville and Ranger, and the progenies of their crosses reacted to PIMV with distinct necrotic lesions on inoculated leaves. In Bonneville and other resistant genotypes this infection remained localized, whereas in Ranger and other susceptible genotypes the virus also caused systemic veinal chlorosis, mottle, necrosis, smaller leaves, and short internodes. At 18°C, this acute stage of infection usually involved 3 to 5 leaves and it was followed by a recovery stage during which plants resumed normal, symptomless growth.

As shown in Table I, all the F_1 plants were systemically resistant to PIMV and those of F_2 populations segregated in a ratio of nearly 3 resistant to 1 susceptible. The F_1 plants crossed to the susceptible parent segregated nearly to the ratio of 1 resistant to 1 susceptible, whereas those crossed to the resistant parent were all resistant.

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These data indicate that resistance to PIMV in *P. sativum* is monogenically dominant. The symbol Pmv (Plantago mottle virus) is proposed for this gene.

At 18°C, plants with the *Molmo* genotype failed to develop symptoms, since the *mo* gene behaved as a dominant character³. Consequently, the 108 plants of the testcross, which had been inoculated first with PIMV and later with BYMV (after PIMV-infected plants had recovered from symptoms) segregated as follows: 27 plants were resistant to both viruses; 29 were resistant to PIMV but susceptible to BYMV; 28 were resistant to BYMV but susceptible to PIMV; and 24 were susceptible to both viruses. This ratio close to the 1:1:1:1 segregation (P = 0.91) clearly indicated that *Pmv* and *mo* are inherited independently. Twenty plants of (Bonne-ville × Ranger) F₁ similarly inoculated with both viruses and included in the linkage tests, did not show symptoms.

Discussion

This study has determined that resistance to PIMV in P. sativum is conferred by the single dominant gene Pmv. It also has indicated that mo, the factor for resistance to BYMV, and Pmv are independently inherited. Thus, the presence of Pmv in many of the BYMV-resistant cultivars must be attributed to some cause other than linkage. Pmvis a newly recognized gene conferring resistance to a virus which, at present, causes no economic loss to the pea crops in New York state, and should not constitute a threat, unless circumstances change². Consequently, it is hoped that this gene will be preserved in order to maintain

 Table I. Response of Pisum sativum to systemic infection by plantago mottle virus

	No. plants			Good-
	resist- ant	suscep- tible	Expected ratio	ness- of-fit (P)
Bonneville	143	0		
Ranger	0	147		
(Bonneville \times Ranger)F ₁	48	0		
(Bonneville \times Ranger)F ₂ (Bonneville \times Ranger)F ₁	192	58	3:1	0.52
\times Bonneville (Bonneville \times Ranger)F ₁	85	0		
× Ranger	56	52	1:1	0.70

This study also offered the rare opportunity of using the testcross for linkage determination between a dominant and a recessive disease-resistant gene. Yen and Fry⁴ reported that resistance to BYMV in P. sativum appeared to be governed by a single recessive factor (mo). Later, Schroeder et al.³ demonstrated that the incubation temperature determines the presence or absence of symptoms on the heterozygous (Molmo) plants infected with BYMV. At 27°C, the heterozygote exhibits typical mosaic symptoms, whereas at 18°C the same genotype remains symptomless. Thus, by exploiting this temperature-dependent dominance of the mo gene, it is possible to use the testcross for linkage with the dominant Pmv. The complete recovery from symptoms of PIMV-infected plants and lack of cross protection between PIMV and BYMV are also key factors allowing the use of the same plants for two viruses. A previous study² had established that remission of PIMV-incited symptoms persisted as long as plants were kept at the original temperature, and recrudescence occurred only when the ambient temperature was lowered. Consequently, a strict control of the temperature during linkage tests was essential, and the absence of BYMV-caused symptoms in F₁ plants, used as control, validated the results.

Summary

Resistance to plantago mottle virus (PIMV) in pea (*Pisum sativum*) was determined to be conferred by a single dominant gene, designated Pmv. Although many PIMV-resistant pea cultivars are resistant also to bean yellow mosaic virus (BYMV), no linkage was detected between Pmv and mo, the factor conditioning resistance to BYMV.

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