In a separate study 135 eggs from nonmutant pairs and 178 eggs from high-insert pairs were placed on standard larval diet[4] 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 high-insert and nonmutants result in a 1:1 mutant-nonmutant ratio, and mutant/mutant matings yielded a 2:1 mutant-nonmutant ratio. In addition more than one-half of the mutant females failed to oviposit.

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

R. PROVVIDENTI

THE OCCURRENCE of plantago mottle virus (PlMV) in pea crops in New York state was recently reported by Provvidenti and Granett[2]. The low incidence of field infection was attributed to the resistance to PlMV 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 PlMV in *Pisum sativum* L., and determine whether factors for resistance to PlMV and bean yellow mosaic virus (BYMV) are linked. Previously[2], a large number of PlMV-resistant cultivars were found to be also resistant to BYMV. Resistance to BYMV is controlled by a single recessive gene[4] (*mo*), which in heterozygous condition (*Mo/mo*) is strongly influenced by temperature[3].

Materials and Methods

Genetic populations were derived from crosses between Bonneville, a cultivar resistant to PlMV and BYMV, and Ranger, a cultivar susceptible to both viruses[2]. Plants of these cultivars and F_1, F_2, and reciprocal backcross populations were mechanically inoculated with the type strain of PlMV originally recovered from *Plantago major*[1]. For linkage detection, plants of the test-cross (Bonneville × Ranger) F_1 × Ranger were first inoculated with PlMV and later with a strain of BYMV, used in a previous study[2]. Inocula were derived from Ranger plants showing distinct symptoms of PlMV 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, PlMV-infected plants exhibited prominent symptoms[2], and the *mo* gene behaved as dominant in the heterozygous plants[3].

Results

Plants of Bonneville and Ranger, and the progenies of their crosses reacted to PlMV 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 1, all the F_1 plants were systemically resistant to PlMV 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 PlMV in *P. sativum* is monogenically dominant. The symbol *Pmv* (Plantago mottle virus) is proposed for this gene.

At 18°C, plants with the *Mo/mo* genotype failed to develop symptoms, since the *mo* gene behaved as a dominant character[3]. Consequently, the 108 plants of the testcross, which had been inoculated first with PlMV and later with BYMV (after PlMV-infected plants had recovered from symptoms) segregated as follows: 27 plants were resistant to both viruses; 29 were resistant to PlMV but susceptible to BYMV; 28 were resistant to BYMV but susceptible to PlMV; 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 (Bonneville × Ranger) F_1 similarly inoculated with both viruses and included in the linkage tests, did not show symptoms.

Discussion

This study has determined that resistance to PlMV 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. *Pmv* is 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 change[2]. Consequently, it is hoped that this gene will be preserved in order to maintain a broad genetic base of resistance in leading cultivars, which are usually grown in many areas of the United States and abroad.

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 Fry[4] 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 (*Mo/mo*) 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 PlMV-infected plants and lack of cross protection between PlMV and BYMV are also key factors allowing the use of the same plants for two viruses. A previous study[2] had established that remission of PlMV-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 F_1 plants, used as control, validated the results.

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

	No. plants			
	resistant	susceptible	Expected ratio	Goodness-of-fit (*P*)
Bonneville	143	0		
Ranger	0	147		
(Bonneville × Ranger)F_1	48	0		
(Bonneville × Ranger)F_2	192	58	3:1	0.52
(Bonneville × Ranger)F_1 × Bonneville	85	0		
(Bonneville × Ranger)F_1 × Ranger	56	52	1:1	0.70

Summary

Resistance to plantago mottle virus (PlMV) in pea (*Pisum sativum*) was determined to be conferred by a single dominant gene, designated *Pmv*. Although many PlMV-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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