

PEA SEEDBORNE MOSAIC SYMPTOM VARIATION AMONG PISUM PLANT INTRODUCTION  
ACCESSIONS: EXPRESSIONS AND VARIATION IN SYMPTOM EXPRESSION AMONG DIFFERENT  
GENOTYPES OF PISUM AND THE PATHOLOGICAL IMPLICATIONS

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In early efforts to identify Pisum germplasm immune to pea seedborne mosaic virus (PSbMV) (1), I noted considerable diversity in symptoms expressed by the 1,326 Plant Introduction accessions inoculated with this virus. The range of PSbMV-induced symptoms made possible the selection of Pisum differentials for standardized comparisons of PSbMV-like viruses and strains. Nineteen such differentials, each responding to PSbMV inoculation with specific symptoms, were used recently for definitive comparisons among PSbMV strains first reported in Czechoslovakia, Japan, the U.S.A., and the Netherlands between 1966 and 1970 (2).

Symptoms induced in these differentials by PSbMV ranged from very rapid development (5 to 9 days after inoculation) of whole-plant necrosis on one extreme to very slight leaf rolling and/or vein clearing on the other. Symptoms induced in differentials of intermediate reactions consisted of plant stunting, vein swelling, tendril kinking, and downward rolling of leaves, most commonly reported for this virus. At least some plants of three differentials were immune to PSbMV.

This range of symptoms poses some interesting genetic implications, because it is generally accepted that immunity to PSbMV is conferred by a single recessive gene pair, sbm (3). Such a range of responses to PSbMV infection would therefore appear to result from modification of susceptibility or sensitivity, by genetic and/or environmental means. Accumulated evidence indicates that the genetic influence is significant. Accordingly, whole-plant necrosis would presumably be caused by the action of modifier genes than enhance sensitivity to the virus (decrease tolerance), whereas tendencies toward infection without symptoms (4) would presumably be caused by modifiers that reduce sensitivity (increase tolerance).

Because it appears that such a modifier-gene system exists in peas, it may also be appropriate to discuss the implications of this phenomenon for breeders. Specifically, there is a possibility that in efforts to develop PSbMV-immune cultivars, breeders might mistake (PSbMV) tolerance or resistance to PSbMV for immunity. This mistake would not necessarily result from failure to assay for the presence of PSbMV in inoculated plants, but from an underestimation of the extent to which gene Sbm can be modified, including simulation of immunity.

If immunity, indeed, specifies that the pathogen cannot multiply in the host at all or beyond the point of inoculation, then the term immunity is purely qualitative and precludes systemic host infection. Therefore, the difference between resistant and immune plants is the presence or absence, respectively, of the systemic infection process. In the case of PSbMV, immune pea plants would contain no detectable virus beyond the virus inoculated tissues. Anything less than immunity becomes a quantitative phenomenon, representing degrees of resistance with correspondingly lesser or greater deleterious effect on plant performance.

Indirect evidence from early efforts to breed for PSbMV-immunity, as well as direct experimental evidence, suggest the existence of a "threshold effect" in the identification of PSbMV-immune breeding progenies. That is, progenies from certain PSbMV-immune parents may appear to be immune when inoculated with PSbMV once or twice, but in fact become infected after three or four inoculations. Such pseudo-immune plants, then, are able to transmit PSbMV through their seeds and would therefore have the potential of transmitting virus from inoculated progenies (screened for immunity) into succeeding generations. It is hoped, on the basis of this evidence, that immunity would be the only acceptable breeding objective, an objective warranting precise virus-detection methodology.

1. Baggett, J. R. and R. O. Hampton. 1972. *Plant Dis. Rept.* 56:131-132.
2. Hampton, R., G. Mink, L. Bos, M. Inouye and D. Hagedorn. 1980. *Netherlands Journal of Plant Pathology* (In press).
3. Hagedorn, D. J. and E. T. Gritton. 1973. *Phytopathology* 63:1130-1133.
4. Hampton, R. O. 1972. *Phytopathology* 62:268-272.

#### THE SEED PRODUCTION OF RECOMBINANTS IN COMPARISON WITH THEIR PARENTS

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The recombinant R 46C of our collection, homozygous for two mutated genes (efr/efr, bif-1/bif-1), shows early flowering and ripening and reduced penetrance of gene bif-1 for dichotomous stem bifurcation. This recombinant was crossed with several mutants in order to study the influence of the mutant genes on seed production.

The main traits of the mutants used are: mutant 122, narrow leaflets; mutant 423, waxless stipules and pods; mutant 250, stem fasciation, late ripening, small seeds; mutant 107D, stem fasciation, late ripening, small seeds.

The resulting recombinants were compared with their parental genotypes and in relation to our initial line 'Dippes gelbe Viktoria' = 100% (Fig. 1).

All recombinants are early flowering and ripening; thus gene efr for earliness is not influenced by the mutant genes tested.

Recombinant R 833, homozygous for earliness, stem bifurcation (from R 46C) and narrow leaflets (from 122) did not differ much from R 46C nor from 122 with respect to the number of seeds per plant. Since the influence of mutant 122 is to reduce the thousand seed weight, the seed weight per plant of R 833 lies between that of R 46C and 122.

Recombinant R 837, homozygous for earliness, stem bifurcation, and waxlessness (from mutant 423), showed a slight increase in the number of seeds per plant as compared with the parental genotypes, almost reaching the value of our initial line. But because of its reduced seed size the seed weight per plant was not much higher than in recombinant R 833 but not as reduced as in mutant 423.