HERITABILITY OF RESISTANCE IN MAIZE TO THE CZECH ISOLATE OF *SUGARCANE MOSAIC VIRUS*

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Summary

The heritability of maize resistance to one Czech isolate of *Sugarcane mosaic virus* (SCMV) was determined. Parental maize lines with different degree of resistance or susceptibility, their F_1 and F_2 progeny were mechanically inoculated by this virus and symptom development were observed during 63 days. The analysis of resistance of F_1 hybrids, derived from resistant lines and lines with a different degree of susceptibility, suggests that there was no hybrid found which would not exhibit any symptom of infection induced by SCMV, but the symptoms appeared one or two weeks later, compared with the susceptible line, the onset of infection was very gradual and the percentage of infection never reached 100 percent. Heritability of this trait, determined on the basis of the variances of parents, F_1 hybrids, and the development of infection in F_2 populations was relatively high and ranged from 0,70 to 0,91. From these results we can assume that the resistance of tested lines to SCMV detected in the greenhouse is not inherited completely dominantly but it may be controlled partially dominantly or polygenically.

Keywords: maize, *Sugarcane mosaic virus*, resistance, heritability

Introduction

Sugarcane mosaic virus (SCMV) is a widespread pathogen of maize in the Czech Republic (POKORNÝ and PORUBOVÁ, 2000a) and also in other European countries (KRSTIC and TOSIC 1995, FUCHS *et al.* 1997, KOVÁCS *et al.* 1998). As there is no direct method of protection against viruses, and also protection against aphids, which transmit this virus, is often ineffective (ASÍNS and PONS 1999, PERRING *et al.* 1999), attention has been directed to breeding for resistance and testing genetic bases of resistance. The determination of genes controlling resistance to SCMV, or possibly their molecular markers, has been the object of study in several institutes. KOVÁCS *et al.* (1994) reported that maize resistance to SCMV was inherited dominantly or partly dominantly. Moreover, they recommended that resistance to this pathogen should be evaluated not only by the presence or absence of symptoms but also by development of infection e.g. using the index proposed by KUHN and SMITH (1977). LOUIE *et al.* (1991) reported that resistance to the American isolate SCMV-MDB was controlled by the *Mdm1* gene as well as resistance to other four strains of the closely related species *Maize dwarf mosaic virus* (MDMV). Using the segregation analysis of several F_2 and BC populations derived from crosses of lines resistant or susceptible to SCMV and tested on two field sites, MELCHINGER *et al.* (1998) found that resistance to SCMV was controlled by 1-3 genes, whereas one gene was detected in all the three resistant lines used. They used RFLP and SSR markers to map these dominant genes of resistance *Scmv1* on the short arm of

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chromosome 6 and *Scmv2* on the long arm of chromosome 3 (3L). These two genes, however, were not sufficient for complete resistance to SCMV in two lines. For this reason the authors assumed that for complete resistance one more gene or even more genes with major gene or minor gene effects were necessary.

Materials and methods

On the basis of previous resistance tests (POKORNÝ and PORUBOVÁ, 2001) four lines resistant to SCMV (R) were chosen. Then they were crossed with susceptible (S) lines (SxR crossing) or with one another (RxR). Crosses were also made between partly resistant and susceptible lines (SxPR) (Table 2). To assess heritability of maize resistance to SCMV, four F_1 populations derived from crossing the resistant line TR42 and four populations of the TR61 line were used to produce F_2 populations. In the greenhouse maize plants of individual parental lines and populations were inoculated with the SCMV isolate Tr190 (at the stage of three to four leaves) and the exhibition of symptoms was evaluated at weekly intervals on individual plants up till day 63 after inoculation (dpi), when most populations were at the stage of anthesis. By the rate of symptom development the plants were placed in different categories of resistance (1 – no symptoms of infection 63 dpi, 2 – symptoms of infection 63 dpi ..... 10 – symptoms of infection 7 dpi) and for individual populations the infection index (IR) was calculated. The experiment was carried out with three replications. In each replication 12 plants of parental lines, 24 plants of F_1 populations and 48 plants of F_2 populations were assessed.

Heritability in a wide sense (h^2) was determined as a result of comparison of variance of F_2 populations (σ^2_{F2}) with the geometric means of variance of both parents (σ^2_{P1},σ^2_{P2}) and F_1 populations (σ^2_{F1}) using the formula: $\sigma^2_{F2} - (\sigma^2_{P1} \times \sigma^2_{P2} \times \sigma^2_{F1})^{1/3} / \sigma^2_{F2}$ (BARBOSA *et al.*, 2001)

Results and discussion

As evident from Table 1, susceptible lines differed in development of infection induced by SCMV. Highly susceptible lines were TR8 and TR65 (IR=9,96, 9,88, respectively), among the susceptible lines with the slowest development of infection was TR 46 (IR=8,0). In the lines which were classified as resistant in this study, symptoms of infection did not appear until 42 dpi and only a small number of plants were infected. The results corresponded to some extent to the previous tests (POKORNÝ and PORUBOVÁ, 2001). An exception was the line TR20, which was classified as resistant in the previous experiments, but in these analyses it was assessed as partly resistant. The analysis of resistance of F_1 hybrids, derived from resistant lines and lines with a different degree of susceptibility, suggests that there was no hybrid found which would not exhibit any symptom of infection induced by SCMV (Table 2). On the hybrids the symptoms appeared one or two weeks later, compared with the susceptible line. In most cases, the onset of infection was very gradual and the percentage of infection never reached 100 percent, therefore, the determination of the degrees of resistance was based on the development of infection in time. The development of infection in individual hybrids was very often dependent on both the resistant and the susceptible line. For example, in the hybrid TR65 x TR61 the development of infection was relatively fast, as opposed to the hybrid TR65 x TR59 where the development of infection was much more gradual. Almost in all hybrids, however, the average of the index of infection in both parents was lower than the

Table 1 – Reaction of parental maize lines to inoculation with SCMV

Line	% of infection (dpi)									IR
	8	15	22	28	35	42	49	56	63	
Susceptible										
TR8	96,7	100,0	100,0	100,0	100,0	100,0	100,0	100,0	100,0	**9,97**
TR12	61,0	71,0	90,7	90,7	94,4	100,0	100,0	100,0	100,0	**9,08**
TR25	63,7	81,9	90,0	90,0	90,0	90,0	90,0	90,0	90,0	**8,76**
TR33	25,0	88,9	100,0	100,0	100,0	100,0	100,0	100,0	100,0	**9,14**
TR44	66,7	91,7	100,0	100,0	100,0	100,0	100,0	100,0	100,0	**9,58**
TR46	22,0	46,8	59,9	75,0	95,8	100,0	100,0	100,0	100,0	**8,00**
TR47	0,0	68,4	73,9	88,9	100,0	100,0	100,0	100,0	100,0	**8,31**
TR50	0,0	42,4	83,3	100,0	100,0	100,0	100,0	100,0	100,0	**8,48**
TR51	0,0	43,0	88,8	95,8	100,0	100,0	100,0	100,0	100,0	**8,28**
TR54	54,7	85,0	100,0	100,0	100,0	100,0	100,0	100,0	100,0	**9,40**
TR56	56,4	100,0	100,0	100,0	100,0	100,0	100,0	100,0	100,0	**9,56**
TR65	87,8	100,0	100,0	100,0	100,0	100,0	100,0	100,0	100,0	**9,88**
Partially resistant										
TR18	0,0	0,0	0,0	0,0	0,0	26,7	43,4	43,4	43,4	**2,40**
TR20	0,0	0,0	0,0	0,0	13,3	27,8	31,1	31,1	31,1	**2,34**
Resistant										
TR42	0,0	0,0	0,0	0,0	0,0	3,0	12,1	12,1	18,2	**1,46**
TR59	0,0	0,0	0,0	0,0	0,0	0,0	5,6	11,1	11,1	**1,28**
TR61	0,0	0,0	0,0	0,0	0,0	7,4	12,2	12,2	12,2	**1,44**
TR64	0,0	0,0	0,0	0,0	0,0	0,0	8,3	12,5	12,5	**1,33**

index of the resultant hybrid. The exceptions were the following combinations TR65xTR61, TR47xTR61 and TR33xTR59. Also in the crosses between partly resistant and susceptible lines the development of infection in the hybrids was slower than in the respective susceptible parent, and the hybrids of resistant lines showed a high degree of resistance like their parents. Heritability of this trait, determined on the basis of the variances of parents, F_1 hybrids, and the development of infection in F_2 populations (Table 3), was relatively high. In the progenies of the line TR42 it ranged from 0,70 (TR46xTR42) to 0,91 (TR56xTR42), in the line TR61 it was also in the range of 0,71 (TR46xTR61) to 0,90 (TR65xTR41).

Assessment of resistance by the rate of infection development was applied because in F_1 hybrids there was none with a uniform reaction to inoculation with SCMV and also the intensity of symptoms on these populations was difficult to determine. This method is used, for example, in rusts infecting cereal crops. In some materials a longer latent period for urediospores maturation was found („slow rusting"), as e.g. in *Puccinia triticina* or *Puccinia striiformis* f.sp. *tritici* (DEGHANI and MOGHADDAM, 2004, XU *et al.*, 2005). Also, some resistance to viruses is based on slower development of infection. One of the best described pathosystems is rice (*Oryza sativa*) x *Rice yellow mottle virus* (RYMV). In the *Oryza sativa*

Table 2 – Reaction of parental maize F_1 hybrids to inoculation with SCMV

Hybrid	% of infection(dpi)									IR
	8	15	22	28	35	42	49	56	63	
SxR										
TR12 x TR42	0,0	5,6	12,5	27,8	32,9	48,9	51,5	71,5	71,5	**4,22**
TR47 x TR42	0,0	5,6	16,7	38,9	41,7	47,3	55,9	60,2	60,2	**4,27**
TR56 x TR42	0,0	0,0	2,8	9,7	18,1	26,4	38,9	54,2	54,2	**3,04**
TR46 x TR42	0,0	2,2	21,4	30,9	32,2	33,7	35,9	35,9	35,9	**3,28**
TR50 x TR42	0,0	0,0	0,0	1,4	7,0	9,9	12,7	12,7	12,7	**1,56**
TR33 x TR59	0,0	18,2	40,5	68,1	86,1	90,3	95,8	95,8	97,2	**6,92**
TR44 x TR59	0,0	0,0	0,0	2,8	7,1	12,6	21,1	25,2	25,2	**1,94**
TR65 x TR59	0,0	0,0	0,0	4,2	11,1	22,2	23,6	26,4	30,6	**2,18**
TR47 x TR61	0,0	20,2	59,5	77,6	84,5	84,5	85,9	89,7	92,7	**6,95**
TR50 x TR61	0,0	0,0	1,4	5,6	5,6	6,9	6,9	19,4	21,4	**1,70**
TR51 x TR61	0,0	0,0	0,0	4,2	8,3	11,1	14,2	17,0	21,3	**1,76**
TR65 x TR61	1,4	56,9	72,2	79,2	83,3	86,1	90,3	90,3	93,1	**7,52**
TR46 x TR61	0,0	3,3	13,6	26,7	35,0	42,5	42,5	42,5	42,5	**3,49**
TR50 x TR64	0,0	0,0	3,5	8,4	13,9	18,6	20,4	23,2	24,7	**2,13**
TR51 x TR64	0,0	0,0	0,0	0,0	15,0	20,0	20,0	21,4	21,4	**1,98**
SxPR										
TR8 x TR18	2,8	36,1	52,8	65,3	73,6	83,3	84,7	91,7	93,1	**6,83**
TR25 x TR18	0,0	0,0	0,0	0,0	0,0	6,9	8,3	11,1	15,3	**1,42**
TR44 x TR18	5,6	9,7	16,7	27,8	39,6	39,6	47,1	48,5	48,5	**3,83**
TR33 x TR20	1,4	29,2	44,4	65,3	75,0	76,4	78,9	80,3	81,8	**6,33**
TR54 x TR20	0,0	0,0	16,7	36,1	59,7	66,7	75,0	75,0	76,4	**5,06**
RxR										
TR42xTR59	0,0	0,0	0,0	0,0	0,0	1,4	1,4	1,4	1,4	**1,06**
TR59xTR61	0,0	0,0	0,0	0,0	0,0	0,0	0,0	2,1	2,1	**1,04**
TR61xTR64	0,0	0,0	0,0	0,0	0,0	1,6	1,6	1,6	1,6	**1,06**
TR64xTR59	0,0	0,0	0,0	0,0	0,0	0,0	4,2	4,2	5,6	**1,14**

japonica var. Azucena, resistance based on the slower spread of the virus in the system, lower concentrations and lower intensities of symptoms was described (PRESSOIR *et al.* 1998, IOANNIDOU *et al.*, 2003). From the results of the development of progeny infection we can assume that the resistance of tested lines to SCMV detected in the greenhouse is not inherited completely dominantly but it may be controlled partially dominantly or polygenically. Our results are different from the findings of MELCHINGER *et al.* (1998), who described two genes of resistance. But it is necessary to realise that these authors tested resistance under field

Table 3 – Reaction of parental maize F_2 hybrids to inoculation with SCMV

Hybrid	% of infection									IR
	8	15	22	28	35	42	49	56	63	
TR12xTR42	14,1	22,6	27,2	37,8	52,6	58,9	62,6	67,0	67,7	**5,11**
TR47xTR42	6,9	27,1	48,7	56,9	61,3	68,6	70,1	71,7	72,4	**5,84**
TR56xTR42	16,2	21,1	23,9	26,9	31,0	37,9	42,9	47,6	48,5	**3,96**
TR46xTR42	6,0	11,2	20,2	29,9	36,7	48,2	53,0	55,6	55,6	**4,16**
TR50xTR61	0,0	0,0	3,2	9,6	13,4	28,3	36,1	43,6	45,4	**2,80**
TR51xTR61	0,0	1,4	2,8	8,5	15,4	22,9	30,9	35,2	36,0	**2,53**
TR65xTR61	27,8	43,2	49,1	60,0	67,2	72,9	76,5	79,8	80,5	**6,57**
TR46xTR61	7,7	11,3	21,7	33,9	45,1	50,8	54,5	56,4	57,1	**4,39**

conditions after artificial inoculation. A similar manner of heritability like in our experiments was described by KOVÁCS *et al.* (1994) who made assessments relatively shortly after inoculation (20 days). These authors also discussed the reasons for the discrepancy of results of particular studies – different inoculation methods, different genotypes, testing under different conditions and non-uniform classification of maize reactions to infection by this virus. The reason why we decided to carry out a greenhouse test was, as found in the previous experiments, that inoculation in the greenhouse is most effective (POKORNÝ and PORUBOVÁ, 2000b). Also, SCOTT and LOUIE (1996) showed in the closely related virus MDMV that the greenhouse test is much better compared to the field test. Another explanation of different results may be using different isolates of the same strain of the virus. FRASER (1992) reported a number of cases, in which different isolates of the same strains of virus pathogens overcame genes of plant resistance to other isolates. Nevertheless, relatively high heritability of resistance, which was detected in our experiments, suggests that selection and successive breeding for this trait is feasible.

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