GENE ACTIONS AFFECTING SUNFLOWER RESISTANCE TO *SCLEROTINIA SCLEROTIORUM* MEASURED BY SCLEROTIA INFECTIONS OF ROOTS, STEMS, AND CAPITULA

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Abstract

Five sunflower inbred lines were crossed with one restorer line and F1, F2, BC1P1 and BC1P2 progenies were produced. Generation means analysis was done for all six generations, as well as the scaling test. The additive-dominance model was adequate for the analysis of the variation in three cases. Relative value of dominant gene effect (h) was higher than relative value of additive gene effect (d) and in two cases (h) was negative indicating that dominance had a negative effect on resistance to sclerotia tests on different organs. All other crosses showed significant deviation from zero for A, B, or C in all other tests indicating that the additive-dominance model was inadequate for those sets of data. In the cross PH-BC1-40A x RUS-RF-OL-168 in root and stem inoculation the dominance component (h) was positive and the dominance x dominance component (l) negative, indicating the presence of duplicate epistasis between dominant increasers. In the cross Ha-48A x RUS-RF-OL-168 in stem inoculation and crosses PR-ST-3A x RUS-RF-OL-168, CMS3-8A x RUS-RF-OL-168 and PH-BC1-40A x RUS-RF-OL-168 in capitulum inoculation, (h) was negative and l positive meaning that duplicate epistasis between dominant decreasers was present. The results of our study showed that epistasis has an effect on the inheritance of sunflower resistance to sclerotia tests, and that this gene effect should be taken in account by breeders working on *Sclerotinia* resistance.

Introduction

Diseases are the major limiting factor of sunflower (*Helianthus annuus* L.) production in most regions. Within the temperate regions of the world, white rot (caused by *Sclerotinia sclerotiorum* [Lib.] de Bary) is the most serious sunflower disease because it is widespread, persists many years in the soil, and has a wide host range. The severity of yield losses due to *Sclerotinia* wilt depends on the age of the plant at the onset of disease (Masirevic and Gulya, 1992). There are no suitable cultural control methods (Lumsden, 1979) and no immune genotypes of cultivated sunflower have yet been found or developed.

Inheritance of *Sclerotinia* resistance is generally polygenic and predominantly under the control of genes with additive effects (Robert et al., 1987; Tourvieille and Vear, 1990).

The scaling test described by Mather (1949) is one of the methods used to check the hypothesis that mean values of progeny depend only on additive and dominant genes. If the additive-dominance model is found to be inadequate, it is necessary to estimate epistatic gene effects (Mather and Jinks, 1982).

The aim of our work was to determine the effect of additive and dominant genes on the inheritance of resistance in sunflower capitula, stems and roots to *Sclerotinia sclerotiorum* measured by sclerotia infections, i.e., to check the adequacy of the additive-dominant model for these traits.

Materials and Methods

Five CMS inbred lines (PR-ST-3A, CMS3-8A, PH-BC1-40A, Ha-48A, and CMS1-50A) and restorer line RUS-RF-OL-168, all selected at the Institute of Field and Vegetable Crops, Novi Sad, Serbia and Montenegro, were used as parents in this study. CMS lines were crossed with the restorer line and F1 hybrids produced. The F1 was backcrossed with each parent to produce the first backcross generation. The F2 generation was produced by self-pollination of F1 plants. Three rows with 12 plants of each generation were sown in an irrigated field. All the plants in the plot were inoculated, and the percentage of resistant plants determined. Each row was treated as one replication.

For the sclerotia test on roots, two or three sclerotia were placed beside the seed during the sowing. Plants were screened two weeks after full flowering (stage M0), using a scale of 1-3.

Stems were inoculated by incorporation of sclerotia in the button stage (E4). Wounds with sclerotia were covered with wet cotton and aluminium foil. Screening was done two weeks after full flowering (M0), using a scale of 1 to 5. Resistance was determined as percentage of healthy plants.

Capitula were inoculated with sclerotia as described above at the beginning of flowering, and screening was done at the end of vegetation (M4), using a scale of 1 to 6.

Percentage of resistant plants was arcsine transformed prior to the estimation of the genetic components. The data were analyzed using the Gen statistical program. ANOVA was done according to Steel and Torrie (1960); single scaling tests and estimation of the effects of additive, dominant and epistatic genes was done according to the models of Mather (1949) and Mather and Jinks (1982); and joint scaling test was done according to Cavalli (1952).

Results and Discussion

The data from the cross between PR-ST-3A and RUS-RF-OL-168 for resistance to inoculation of root and stem and from the cross between Ha-48A and RUS-RF-OL-168 for the resistance to inoculation of root gave insignificant values of A, B and C. Hence the additive-dominance model was perfectly adequate for the analysis of the variation in these single sets of data (Table 1). In these crosses the estimates of additive (d) and dominant (h) gene effects were not significantly different from zero (Table 2). Relative value of (h) was higher than (d), and in two cases (h) was negative indicating that dominance had a negative effect on resistance to sclerotia tests on different organs. This is not in agreement with the results of other authors concerning single *S. sclerotiorum* infections on roots (Tourvieille and

Table 1. Mean value of progeny and scaling tests for resistance to inoculation of root, capitulum, and stem with sclerotia in five sunflower crosses. C1: PR-ST-3B x RUS-RF-OL-168, C2: CMS3-8A x RUS-RF-OL-168, C3: PH-BC1-40A x RUS-RF-OL-168, C4: Ha-48A x RUS-RF-OL168, and C5: CMS1-50A x RUS-RF-OL-168.

Cross	Mean value of progeny						Scaling test		
	P1	BC1P1	F1	F2	BC1P2	P2	A	B	C
Root									
C1	90.00±0.00	83.51±5.29	76.11±5.88	85.69±3.52	84.42±4.56	90.00±0.00	0.92±12.12	2.73±10.84	10.56±18.33
C2	90.00±0.00	83.85±5.02	73.54±6.72	83.85±5.02	90.00±0.0	69.21±8.94	4.16±12.08	37.24*±11.18	29.11±25.76
C3	90.00±0.00	84.15±4.78	90.00±0.00	72.35±2.24	90.00±0.00	71.96±7.49	-11.71±9.56	18.04±7.49	-52.55**±11.68
C4	90.00±0.00	90.00±0.00	81.96±6.56	90.00±0.00	90.00±0.00	84.42±4.56	8.04±6.56	13.63±7.99	21.67±13.89
C5	90.00±0.00	90.00±0.00	81.96±6.56	90.00±0.00	78.29±4.78	65.37±11.74	8.04±6.56	9.26±16.49	40.71*±17.61
Stem									
C1	54.73±6.89	32.75±1.02	20.50±8.38	37.03±3.33	28.92±1.54	44.12±2.81	-9.74±11.01	-6.79±9.35	8.28±22.67
C2	44.12±2.81	22.25±9.12	17.01±0.22	24.13±4.09	39.12±2.44	27.30±5.49	-16.64±18.47	33.92**±7.36	-8.91±17.52
C3	44.12±2.81	31.00±0.41	25.50±3.11	17.63±2.69	37.37±2.02	37.31±2.37	-7.62±4.27	11.92±5.63	-61.93**±12.98
C4	44.12±2.81	29.34±4.17	39.31±1.68	40.88±1.28	36.68±7.44	34.98±2.19	-24.76*±8.95	-0.92±15.12	5.81±7.07
C5	44.12±2.81	32.98±1.27	25.41±6.05	45.75±0.79	40.42±1.36	36.40±7.14	-3.56±7.14	19.02±9.74	51.65**±14.68
Capitulum									
C1	67.53±2.41	37.33±5.67	59.84±2.36	41.88±2.27	42.20±1.36	43.96±2.03	-52.71**±11.83	-19.40**±4.13	-63.64**±10.71
C2	43.96±2.03	34.56±0.55	36.49±0.51	47.06±1.68	31.82±3.61	25.87±10.69	-11.32**±2.36	1.29±12.91	45.44**±12.84
C3	43.96±2.03	34.56±0.55	43.66±2.52	49.62±3.01	37.56±2.16	42.33±1.15	-18.49**±3.42	-10.87±5.14	24.88±13.26
C4	52.62±2.02	49.65±1.11	57.48±1.24	41.30±1.58	30.00±13.08	43.96±2.03	-10.79*±3.25	-41.43±26.27	-46.32**±7.35
C5	47.63±2.63	43.34±3.49	48.25±2.66	44.15±1.08	54.68±1.82	43.96±2.03	-9.20±7.92	17.14*±4.94	-11.49±7.61

Table 2. Gene effects for resistance to inoculation of root, capitulum, and stem with sclerotia in five sunflower crosses.

Cross	Gene effect						Type of epistasis
	m	*d*	*h*	*i*	*j*	*l*	
Root							
C1	96.91**	0.00	-24.06	-6.91	-1.82	3.26	-
C2	67.31*	10.39	59.93	12.29	-33.08*	-53.70	-
C3	22.10	9.02	133.11**	58.88**	-29.75*	-65.21*	Duplicate epistasis between dominant increasers.
C4	87.21**	2.79	16.42	0.00	-5.58	-21.67	-
C5	101.10**	12.32	-25.25	-23.41*	-1.22	6.11	-
Stem							
C1	74.23**	5.31	-95.07	-24.81	-2.95	41.33	-
C2	9.52	8.41	50.97	26.19	-50.56*	-43.47	-
C3	-25.51	3.40	121.54**	66.23**	-19.54**	-70.53**	Duplicate epistasis between dominant increasers.
C4	71.04**	4.57	-88.90	-31.49	-23.84	57.18	-
C5	76.45**	3.86	-71.76**	-36.19**	-22.58*	20.72	Duplicate epistasis between dominant decreasers.
Capitulum							
C1	64.22*	11.78**	-84.96	-8.47	-33.31*	80.58**	Duplicate epistasis between dominant decreasers.
C2	90.38**	9.04	-119.38**	-55.47**	-12.61	65.49**	Duplicate epistasis between dominant decreasers.
C3	97.39**	0.81	-137.34**	-54.25**	-7.62	83.61**	Duplicate epistasis between dominant decreasers.
C4	54.20	4.33*	-54.85	-5.91	30.64	58.13	-
C5	26.36*	1.84	49.27	19.43	-26.35*	-27.37	-

Vear, 1990), and leaves (Castano et al., 1993) where it was concluded that additive effects were more important than dominance effects in the inheritance of *S. sclerotiorum* resistance.

However, these and other crosses showed significant deviations from zero for A, B, or C in all other tests indicating that the additive-dominance model was inadequate for those sets of data, and that epistatic gene effects influenced the resistance to sclerotia tests on different organs (Table 1). According to Powers (1941), a model which is adequate for one cross may not be adequate for another which covers the same range of variation, and still less adequate for other crosses covering wider ranges of variation. Marinkovic et al. (2002) and Gangappa et al. (1997), who studied the mode of inheritance of some morphological and yield traits in cultivated sunflower, also found that the additive-dominance model was not adequate for all crosses.

In contrast to our results, Fick et al. (1983) concurred with the view that epistatic effects were less important compared to additive effects in Sclerotinia wilt resistance, as did Vear and Tourvieille (1988) for capitulum rot resistance. In several cases the values for epistatic gene effects were significant and the type of epistasis could be determined (Table 2). In the cross PH-BC1-40A x RUS-RF-OL-168 in root and stem inoculation, the dominance component (h) was positive and the dominance x dominance component (l) negative indicating the presence of duplicate epistasis between dominant increasers. In the cross Ha-48A x RUS-RF-OL-168 in stem inoculation and crosses PR-ST-3A x RUS-RF-OL-168, CMS3-8A x RUS-RF-OL-168 and PH-BC1-40A x RUS-RF-OL-168 in capitulum inoculation, (h) was negative and l positive meaning that duplicate epistasis between dominant decreasers was present.

Castano et al. (2001) found significant additive x additive and dominance x dominance gene effects for the level of resistance to mycelium tests on capitula. We obtained similar results in two crosses with sclerotia tests on capitula (Table 2). In accordance with the results of the same authors, the dominance x dominance effect had a positive value in four out of five crosses, indicating that this epistatic gene effect could reduce resistance to sclerotia tests.

Conclusions

The results of our study showed that epistasis has an effect on the inheritance of sunflower resistance to sclerotia tests, and that this gene effect should be taken into account by breeders working on *Sclerotinia* resistance.

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References

Castano, F., Vear, F., and Tourvieille, D. 1993. Resistance of sunflower inbred lines to various forms of attack by *Sclerotinia sclerotiorum* and relations with some morphological characters. Euphytica .68:85-98.

Castano, F., Vear, F., and Tourvieille, D. 2001. The genetics of resistance of sunflower capitula to *Sclerotinia sclerotiorum* measured by mycelium infections combined with ascospore tests. Euphytica. 122:373-380.

Cavalli, L.L. 1952. An analysis of linkage in quantitative inheritance. Quantitative Inheritance (E.C.R. Reeve and C.H. Waddington, eds.), HMSO, London, UK. p.135-144.

Fick, G., Gulya, T., and Auwater, G. 1983. Inheritance of *Sclerotinia* wilt resistance in sunflower. Proc 5th Sunflower Research Workshop, Minot, North Dakota, USA p. 21-22.

Gangappa, E., Channakrishnaiah, K.M., Chandan, T., and Ramesh, S. 1997. Genetic architecture of yield and its attributes in sunflower (*Helianthus annuus* L.). Helia. 20(27):85-94.

Lumsden, R.D. 1979. Histology and physiology of pathogenesis in plant diseases caused by *Sclerotinia* species. Phytopathology. 69:890-896.

Masirevic, S., and Gulya, T.J. 1992. *Sclerotinia* and *Phomopsis,* two devastating sunflower pathogens. Field Crops Res. 30:271-300.

Marinkovic, R., Skoric, D., Jovanovic, D., and Joksimovic, J. 2002. Expression of epistasis in the inheritance of some morphological characters in sunflower (*Helianthus annuus* L.). Zbornik radova Naucnog instituta za ratarstvo i povrtarstvo. 37:3-14. (In Serbian)

Mather, K. 1949. Biometrical Genetics. Dover Publications, New York, USA.

Mather, K., and Jinks, J.L. 1982. Biometrical Genetics. London, New York, Chapman and Hall.

Powers, L. 1941. Inheritance of quantitative characters in crosses involving two species of *Lycopersicum*. J. Agr. Res. 63:149-174.

Robert, N., Vear, F., and Tourvieille de Labrouhe, D. 1987. L'heredite de la resistance au *Sclerotinia sclerotiorum* (Lib.) de Bary chez le tournesol. Etude des reactions a deux test mycelins. Agronomie. 4:423-429.

Steel, R.G., and Torrie, J.H. 1960. Principles and procedures of statistics. McGraw Hill Book Company. London, UK.

Tourvieille, D., and Vear, F. 1990. Heredity of resistance to *Sclerotinia sclerotiorum* in sunflower. III. Study of reactions to artificial infections of roots and cotyledons. Agronomie. 10:323-330.

Vear, F., and Tourvieille, D. 1988. Heredity of resistance to *Sclerotinia sclerotiorum* in sunflowers. II. Study of capitulum resistance to natural and artificial ascospore infections. Agronomie. 8:503-508.