GENE ACTIONS FOR SEED YIELD IN SUNFLOWER (*HELIANTHUS ANNUUS*)

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Abstract

Effects of additive and dominant genes and their interactions on the inheritance of seed yield in 10 sunflower hybrids developed by crossing five inbred lines derived from the synthetic NS-S-1 were analyzed in 2001 and 2002. Relationships among the expected mean values of the progenies were checked by the scaling method (Mather, 1949). Gene affects and mode of inheritance were estimated by generation mean analysis (Mather and Jinks, 1982). The additive-dominant model was not adequate for all crosses over the two years. Epistatic gene effects played a dominant role in the inheritance of seed yield in a large number of the crosses. Duplicate epistasis between dominant decreasers and complementary epistasis between dominant increasers were each expressed in a single cross while duplicate epistasis between dominant increasers was expressed in the other crosses. Thus it ensures that sunflower geneticists and breeders should take into account the existence of epistasis in order to facilitate the selection of the method to be used for improvement of a particular trait.

Introduction

Serbia and Montenegro is among the countries in which sunflower is the main crop for the production of edible oil. The national sunflower acreage ranged from 34,190 ha in 1983 to 208,000 ha in 1996. The average yield ranged between 1.4 t/ha in 1999 and 2.6 t/ha in 1986. Because of a limited area of arable land and the ever-increasing demand for edible oil, it is necessary to introduce into production more productive hybrids, i.e., hybrids possessing a higher genetic potential for seed yield per unit area than the ones which are currently grown. However, in sunflower just as in other crops, source material with high genetic variability is needed in order to be able to develop superior yielding hybrids. In addition, to be able to select the most suitable breeding method, it is necessary to obtain information on the mode of inheritance and the nature and magnitude of gene action for yield and its components.

Keeping the above in mind, we used generation mean analysis in this study (Mather and Jinks, 1982; Gamble, 1962). This method provides not only estimates of additive and dominant gene effects, but also estimates of the magnitude of all three types of digenic

epistatic gene effects, additive x additive, additive x dominant and dominant x dominant (Gangappa et al., 1997).

Materials and Methods

The inbred lines used in this study were derived from NS-S-1, a synthetic developed at the Institute of Field and Vegetable Crops in Novi Sad. The synthetic was made of eight lines crossed by the method of convergent crossing according to the principle of maximum recombination. Some of the source lines were the female parents of the best domestic sunflower hybrids, NS-H-26 RM, NS-H-27 RM, NS-H-45 and NS-H-43.

Out of a score of inbred lines derived from this synthetic in a 6-year cycle of self-pollination, five lines were selected for mutual crossing in 1998. The plants used as the female parent were manually emasculated. Ten crosses were made: c1 (ns-mr-1 x ns-mr-2), c2 (ns-mr-1 x ns-mr-3), c3 (ns-mr-1 x ns-mr-4), c4 (ns-mr-1 x ns-mr-5), c5 (ns-mr-2 x ns-mr-3), c6 (ns-mr- x ns-mr-4), c7 (ns-mr-2 x ns-mr-5), c8 (ns-mr-3 x ns-mr-4), c9 (ns-mr-3 x ns-mr-3), c10 (ns-mr-4 x ns-mr-5) and c11 (ns-mr-4 x ns-mr-5). The F_2 generation and backcrosses with both parents (BC1P1; BC1P2) were produced in the year 2000.

A trial that included the parent lines, F1 hybrids, F2 generation and the backcrosses as conducted at Rimski Šančevi experiment field in 2001 and 2002. The trial was established with randomized blocks with three replicates. The experimental material was planted into carefully prepared plots at the optimum date. The distance between rows was 70 cm, and the distance within the row 30 cm. Several seeds were placed in each hill and thinning was performed at the stage of 2-3 permanent leaves. The parent lines and the F1 hybrids were each planted in four rows, and the F2 generation and the backcrosses were each planted in eight rows. In the course of the growing season, the experimental plots were rototilled and hoed to eradicate the weeds that remained after herbicide treatment.

Seed yield was measured in the laboratory, after removal of seeds from individual heads. Sample size was 20 plants per replicate for the parent lines and the F_1 hybrids, or 60 plants for the entire experiment. In the case of the F2 generation and the backcrosses, the sample size was 60 plants per replicate or 180 plants for the entire experiment. Border rows and the first and the last plant in the inner rows were excluded from sampling.

Individual scaling tests and estimates of the effects of additive, dominant and epistatic genes were conducted according to the model of Mather (1949) and Mather and Jinks (1982).

Results and Discussion

The average seed yield per plant differed among the tested progenies and the test years. In the first year, the mean values of the F1 generation ranged from 60.60 g/plant in C2, to 82.58 g/plant in C4. In the second year, the values ranged from 76.11 g/plant in C3 to 87.51 g/plant in C1. In the first year, the mean value of the F1 generation differed significantly from the mean value of the better parents, indicating the expression of heterosis in the inheritance of this trait. In the second year, heterosis did not occur in only one cross (C5) in which it was replaced by the dominance of the better parent. In the first year, the lowest and the highest heterosis were registered in C5 and C10. In the second year, the lowest and the highest heterosis were registered in C1 and again in C10 (Table 1).

Heterotic effects in the inheritance of seed yield had been reported previously (Vranceanu and Stoenescu, 1969; Ge, 1981; Marinković, 1984; Joksimović, 1992). The calculated values

Table 1. Mean value of progeny and scaling tests for seed yield in sunflower crosses over two years.

2001.

Cross	Mean value of progeny						Scaling test		
	P_1	BC_1P_1	F_1	F_2	BC_1P_2	P_2	A	B	C
C_1	62,80±1,72	62,10±1,49	69,71±0,68	58,45±1,39	49,76±0,74	46,94±0,92	-8,30±3,51	-17,13±1,88**	-15,34±6,06*
C_2	63,84±1,22	68,71±1,26	74,08±0,50	60,94±1,24	64,77±1,90	46,94±0,92	-0,49±2,84	8,51±3,94	-15,18±5,30*
C_3	46,94±0,92	52,64±0,69	60,60±1,08	53,46±1,03	50,17±0,81	38,09±0,73	-2,27±1,98	1,64±2,11	7,62±4,83
C_4	46,94±0,92	63,24±1,49	82,58±1,72	53,09±1,33	63,90±0,62	40,04±0,31	-3,04±3,56	5,17±2,14	-39,79±6,39**
C_5	63,84±1,22	73,47±0,54	74,73±0,86	75,59±2,03	65,86±1,81	62,80±1,72	8,38±1,85**	-5,80±4,10	26,27±8,58*
C_6	62,80±1,72	60,82±0,71	78,08±2,44	46,72±0,74	48,47±1,70	40,04±0,31	-19,24±3,30**	-21,17±4,19**	-72,10±5,97**
C_7	62,80±1,72	63,15±0,46	72,64±0,46	54,77±1,46	45,69±1,78	38,09±0,79	-9,14±2,01**	-19,35±3,68**	-27,12±6,22**
C_8	63,84±1,22	74,44±1,47	72,51±1,19	54,53±0,47	57,46±0,62	38,09±0,79	12,54±3,39*	4,31±1,89	-28,83±3,36**
C_9	63,84±1,22	67,00±1,25	72,43±0,12	63,76±1,09	52,40±2,42	40,04±0,31	-2,27±2,79	-7,67±4,85	6,30±4,54
C_{10}	40,04±0,31	33,90±1,10	80,86±2,00	36,21±0,41	81,50±0,96	38,09±0,79	-53,10±2,99**	44,04±2,88**	-95,01±4,41**

2002.

Cross	Mean value of progeny						Scaling test		
	P_1	BC_1P_1	F_1	F_2	BC_1P_2	P_2	A	B	C
C_1	83,64±0,75	78,21±4,72	87,51±1,12	55,68±5,84	87,51±1,12	61,04±4,31	**-14,72±9,53**	**26,46±4,98****	**-96,96±23,85****
C_2	61,04±4,31	69,28±0,88	82,09±1,16	55,68±5,84	75,35±3,15	57,68±4,03	**-4,58±4,80**	**10,94±7,57**	**-60,17±24,19***
C_3	61,04±4,31	63,17±1,91	76,11±4,28	61,37±2,94	61,76±2,22	40,28±3,74	**-10,81±7,18**	**7,13±7,20**	**-8,07±15,61**
C_4	61,04±4,31	70,11±3,25	81,37±1,78	71,97±3,36	62,80±1,42	34,65±3,61	**-2,19±8,00**	**9,57±9,42**	**29,43±15,00**
C_5	83,64±4,63	80,55±1,40	85,34±5,40	61,72±0,54	71,57±1,21	57,68±4,03	**-7,87±7,64**	**0,13±7,16**	**-65,11±12,61****
C_6	83,64±4,63	70,13±3,53	87,10±1,66	67,53±1,82	66,99±1,73	40,28±3,74	**-30,49±8,60***	**6,60±5,35**	**-28,00±9,98***
C_7	83,64±4,63	73,14±0,33	78,65±1,34	62,07±4,82	63,90±3,18	34,65±3,61	**-16,02±4,86***	**14,49±7,43**	**-27,33±20,32**
C_8	57,68±4,03	76,34±2,84	86,12±3,30	71,24±3,14	68,99±0,93	40,28±3,74	**8,88±7,71**	**11,57±5,33**	**14,75±15,22**
C_9	57,68±4,03	74,41±2,95	87,02±3,32	59,27±2,90	68,47±2,38	34,65±3,62	**4,11±7,87**	**15,26±6,84**	**-29,31±14,40**
C_{10}	40,28±3,74	67,89±1,98	82,60±1,69	49,33±2,06	47,77±4,61	34,65±3,61	**12,90±2,26**	**-21,71±10,04**	**-42,81±10,32****

of the scaling tests (A, B and C) and their dispersion indicated that the null hypothesis on the effect of additive and dominant genes on the mean values of progenies was applicable in only two crosses from the first year (C3 and C9) and in four crosses from the second year (C3, C4, C8 and C9) (Table 1). In the other crosses, at least one test was significant or highly significant; indicating that in these crosses the inheritance of this trait was governed also by other parameters such as digenic epistatic gene effects.

Because there are cases (Powers, 1941) when a model adequate for one cross is inadequate for another cross although the latter has either the same range of variability or even a higher range of variability, we decided to apply the joint scaling test in this study.

Involvement of both additive and dominant gene effects was found to be responsible for the inheritance of seed yield in crosses C2, C4, C6, C8 and C10 in the first year and in crosses C1, C2, C5, C9 and C10 in the second year. However, the size of dominant gene effects was larger than the size of additive gene effects in all these crosses, indicating the predominance of the former in the inheritance of seed yield (Table 2).

Additive gene effects were predominant in crosses C1, C3, C7 and C9 in the first year and in crosses C3, C4, C6, C7 and C8 in the second year.

Predominance of additive gene effects in the inheritance of seed yield has been reported by Sindagi et al. (1979). Conversely, Marinković (1984), Joksimović et al. (1994) and Gangappa et al. (1997) reported the predominant role of the nonadditive genetic variance in the inheritance of this trait.

Epistatic gene effects (*i*, *j* and *l*) were not equally important for the inheritance of seed yield in the two years of study. Besides the principal gene effects, the epistatic gene effects additive x additive and dominant x dominant played significant roles in crosses C2, C4 and C6 in the first year and in crosses C1, C5 and C9 in the second year. All three epistatic gene effects were significant only in crosses C4 and C8 in the first year and in cross C1 in the second year.

Two crosses from the first year (C3 and C5) and five crosses from the second year (C3, C4, C6, C7 and C10) could not be classified for epistasis expression because neither dominant (*h*) nor epistatic gene effect dominant x dominant (*l*) was significant. In the first year of study, duplicate epistasis between dominant decreasers was expressed in crosses C1, C7 and C9, and duplicate epistasis between dominant increasers was expressed in crosses C2, C4, C8 and C10, while complementary epistasis between dominant increasers occurred in cross C6. Only one type of epistasis was expressed in the second year–duplicate epistasis between dominant increasers. It was registered in crosses C1, C2, C5, C9 and C10.

Gangappa et al. (1997) registered the expression of duplicate epistasis between dominant increasers as well as the expression of complementary epistasis between dominant decreasers.

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Table 2. Gene effects for seed yield in ten crosses of sunflower in two years.

2001.

Cross	Gene effect						Type of epistasis
	m	*d*	*h*	*i*	*j*	*l*	
C_1	64,97**	7,93**	-30,79	-10,09	8,83	35,53**	Duplicate epistasis between dominant decreasers
C_2	32,19**	8,45**	73,11**	23,20*	-9,00	-31,22*	Duplicate epistasis between dominant increasers
C_3	50,76**	4,42**	0,96	-8,25	-3,91	8,87	-
C_4	1,58	3,45**	125,05**	41,91**	-8,21*	-44,04**	Duplicate epistasis between dominant increasers
C_5	87,01**	0,52	-33,41	-23,69*	14,18*	21,12	-
C_6	19,73**	11,38**	49,63**	31,69**	1,93	8,72	Complementary epistasis between dominant increasers
C_7	51,82**	12,35**	-9,04	-1,37	10,21*	29,86**	Duplicate epistasis between dominant decreasers
C_8	5,29	12,87**	129,75**	45,68**	8,23*	-62,53**	Duplicate epistasis between dominant increasers
C_9	68,17**	11,90**	-21,91	-16,23	5,40	26,17*	Duplicate epistasis between dominant decreasers
C_{10}	-46,89**	0,97	204,65**	85,95**	-97,14**	-76,90**	Duplicate epistasis between dominant increasers

2002.

Cross	Gene effect						Type of epistasis
	m	*d*	*h*	*i*	*j*	*l*	
C_1	-36,36	11,30**	244,31**	108,70**	-41,19**	-120,04**	Duplicate epistasis between dominant increasers
C_2	-7,17	1,68	162,14**	66,53*	-15,52	-72,89*	Duplicate epistasis between dominant increasers
C_3	46,27**	10,38*	30,54	4,39	-17,94	-0,70	-
C_4	69,90**	13,20**	-3,19	-22,05	-11,76**	14,66	-
C_5	13,30*	12,98*	121,65**	57,36**	-8,00	-49,61**	Duplicate epistasis between dominant increasers
C_6	57,84**	21,68**	9,49	4,11	-37,09**	19,77	-
C_7	33,35	24,49**	69,58	25,80	-30,51**	-24,27	-
C_8	43,27*	8,70*	69,01	5,71	-2,69	-26,16	-
C_9	-2,52	11,51*	157,61**	48,69*	-11,15	-68,06*	Duplicate epistasis between dominant increasers
C_{10}	3,47	2,81	104,32*	34,00*	34,60*	-25,19	Duplicate epistasis between dominant increasers

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