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**RESEARCH ON THE RESISTANCE OF SUNFLOWER INBRED LINES
AND HYBRIDS TO SCLEROTINIA LIBERTIANA FUCK.**

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White rot (*Sclerotinia libertiana* Fuck.) is a widely spread disease which has increasingly damaged the sunflower crop in Yugoslavia in the recent years. The only efficient way to further increase the sunflower productivity is to breed it for the resistance to white rot since there is not any other more efficient way of protection against this disease.

Presently, the mode of inheritance of sunflower resistance to white rot is not known. It is supposed to depend on several genes.

Tests of sunflower varieties indicated differences in resistance among the examined varieties (Naumov 1952, Borisenko 1962). The most resistant were top-crosses and intervarietal hybrids. Resistant male-sterile lines transmitted the resistance to their hybrids (Kukin 1968).

The basic sources of resistance genes are the natural plant forms which acquired the resistance through evolution (Vavilov 1935). This conclusion led us to cross various sunflower varieties which had resistance genes.

MATERIAL AND METHOD

Observations performed on the breeding material grown in field conditions showed differences in the degree of infestation among certain genotypes. The research material was of different genetic origin; we supposed that the differences in resistance among the genotypes were genetic in nature. In 1973, we tested a part of the breeding material for the resistance to white rot.

One test was run on foreign hybrids, another on domestic inbreds and hybrids. The standard variety VNIIMK 8981 was the control.

The test was conducted by a random block system in four replications. Ten plants were inoculated in each replication.

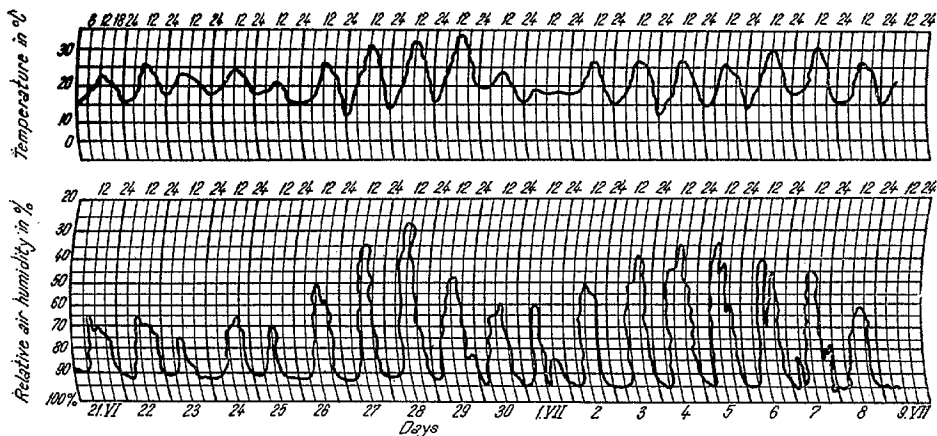


Fig. 1 — Temperature and relative humidity of air (21 June—9 July 1973).

The inoculation was performed with a five-day old culture of *Sclerotinia libertiana* Fuck. which had been grown in five generations on carrot agar (Kukin's method, 1968).

Conditions for the development of the disease were favourable (fig. 1).

The dynamics of the disease was followed after the occurrence of first symptoms. The number of infested and dead plants was recorded regularly. The plants which remained in the test plots were selfed.

The results were statistically processed.

RESULTS

A statistical analysis showed a large variability among the examined genotypes in the resistance to white rot. The hybrids $B_{11}a_3 \times HA\ 61$ (French) and $cms\ HA89 \times RHA266$ (American) (Table 1) had the highest resistance. These two hybrids were also resistant to *Plasmopara helianthi*, Novot. The resistance of these hybrids originates probably from the lines HA61 and RHA 266.

The inbreds CM 1/6—1 and H 2/1—1 (Table 2) had the highest resistance to white rot among the domestic lines and hybrids.

Differences in the resistance were noticed immediately after the occurrence of the disease. When the ecological conditions were favourable for the development of the disease, the infested plants died in a short period of time. The first symptoms were noticeable four days after the inoculation.

The number of infested plants increased rapidly. Twelve days after the inoculation the infestation slowed down.

Six days after the inoculation the control (VNIIMK 8931) had 90% of the plants infested; the hybrids $B_{11}a_3 \times HA\ 61$ and $cms\ HA\ 89 \times RHA$

Table 1

Development dynamics of *Sclerotinia libertiana* in sunflower after inoculation (observation : June 22, 1973)

No.	Material	Percentage of infested and dead plants													
		26.VI		27.VI		28.VI		30.VI		2.VII		4.VII		14.VII	
		Dise- ased	Dead	Dise- ased	Dead	Dise- ased	Dead	Dise- ased	Dead	Dise- ased	Dead	Dise- ased	Dead	Dise- ased	Dead
1	CPK31B ₃ -1	45	0	77	5	87	25	92	67	97	77	97	90	97	97
2	CV761BC23	25	0	37	0	47	5	60	17	60	37	60	37	73	73
3	E ₁₁ - ₃ HA61	10	0	27	2	27	2	35	12	40	20	40	25	50	50
4	E ₁₁ - ₃ V135	25	0	52	0	57	15	62	35	62	42	62	50	68	68
5	BFC13Y2	22	0	40	2	62	5	62	25	62	40	70	55	75	75
6	BK III	40	0	67	1	72	17	82	65	90	70	90	77	90	90
7	BK IV	20	0	57	0	70	12	72	55	72	57	72	65	75	75
8	H-52	32	2	35	5	77	25	82	60	85	72	85	72	97	97
9	H-53	27	0	67	0	72	22	72	62	80	77	80	80	85	85
10	Valley Carada	14	0	36	0	58	5	66	22	66	40	70	50	65	65
11	Sorex	32	0	47	0	72	10	72	30	72	37	72	47	72	72
12	/cmsHA89×HA 234/×RHA271	25	0	60	0	65	15	80	47	80	57	80	57	80	80
13	cmsHA89×RHA266	22	0	25	2	30	10	35	15	37	22	42	30	50	50
14	VNIIMK 8931	50	15	87	20	87	52	90	82	92	85	97	97	97	97

Development dynamics of *Sclerotinia libertiana* in sunflower after inoculation (observation : June 21, 1973)

No.	Material	Percentage of infested and dead plants													
		25.VI		26.VI		27.VI		28.VI		30.VI		2.VII		14.VII	
		Dise- ased	Dead	Dise- ased	Dead	Dise- ased	Dead	Dise- ased	Dead	Dise- ased	Dead	Dise- ased	Dead	Dise- ased	Dead
1	NS 4/2-1	90	20	100	57	100	65	100	85	100	97	100	100	100	100
2	SA 4/1-1	50	0	67	15	67	25	67	42	70	67	70	70	70	83
3	P 5/2-3	22	0	65	2	67	17	67	32	72	52	75	63	85	85
4	CM 1/6-1	45	0	57	2	65	15	70	37	72	67	73	75	75	75
5	9343.1/3-3	23	2	61	12	74	33	74	41	74	58	74	62	80	80
6	1646 4/6-1	21	0	65	10	76	21	76	34	76	60	79	66	83	83
7	SR 1/7-1	19	2	51	10	56	17	63	26	66	46	71	59	80	80
8	S 3/5-1	12	2	50	15	65	22	65	32	67	62	80	75	81	83
9	H 2/1-1	33	0	61	19	61	27	61	50	66	55	67	63	75	75
10	VNIIMK 8931	57	17	82	52	87	75	90	75	95	92	98	98	100	100
11	PEREDOVIK	38	13	89	48	94	66	97	89	100	94	100	100	100	100
12	NS-GMS-A9345	43	2	95	17	95	44	95	68	95	85	95	95	98	98
13	NS-GMS-A9345 X XNS 4/2-1	65	2	90	32	90	65	92	72	92	75	98	98	98	98
14	NS-GMS-A9345 X SA 4/1-1	17	2	47	15	60	17	70	32	75	55	78	70	85	85
15	NS-GMS-A9345 X P 5/2-3	55	0	85	15	87	27	90	52	97	85	98	90	98	98
16	NS-GMS-A9345 X X 1646 4/6-1	20	2	51	13	56	33	64	43	72	66	75	67	89	89
17	NS-GMS-A9345 X SR 1/7	43	0	68	22	68	29	75	49	80	68	81	76	85	85
18	NS-GMS-A9345 X S 3/5-1	37	5	75	27	87	50	87	55	97	87	98	93	98	98
19	NS-GMS-A9345 X VNIIMK 8931	70	15	90	57	95	62	95	72	95	92	95	95	95	95
20	NS-GMS-A9345 X PEREDOVIK	62	5	85	30	90	45	92	65	92	90	95	95	100	100

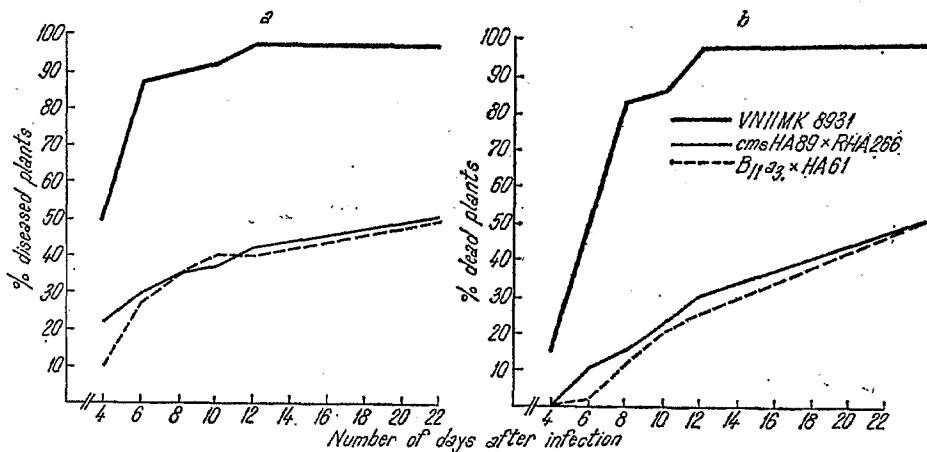


Fig. 2 — Dynamics of disease development of *Sclerotinia libertiana* on sunflower (1973).

266 had 10 to 25% of the plants infested (fig. 2). After the occurrence of the first symptoms, the differences in resistance among the examined genotypes were significant. However, these differences were not statistically justifiable at the end of the test. The differences in resistance of sunflower to white rot were harder to be noticed during the first test. After this test and the selection of resistant plants, the differences were larger (K u k i n 1968). The differences in resistance of the test material are even more significant and will probably be more noticeable in further tests.

CONCLUSIONS

Significant differences in the resistance to white rot were found among the examined sunflower genotypes. These differences were particularly significant immediately after the inoculation (4—8 days).

The disease symptoms occur later in more resistant genotypes. Also, the disease spreads slower. An evaluation of the resistance of the breeding material can be made 12 days after the inoculation.

The resistance of sunflower to white rot is genetically conditioned; the sunflower breeding for the resistance to white rot should be continued.