Physiological Specialization Between Races of Plasmopara halstedii in America and Europe

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ABSTRACT

Sunflowers, *Helianthus annuus*, resistant in the United States to downy mildew incited by *Plasmopara halstedii*, were, without exception, resistant to downy mildew in European trials. However, many lines resistant in trials in Europe were susceptible in the United States. These differences establish physiological specialization within the *P. halstedii* complex in Europe and America and suggest that appropriate measures be implemented to restrict the chance introduction of the more virulent North American race of *P. halstedii* into areas where it does not presently occur.

Resistance to both the North American and European races of downy mildew is traced to wild *H. annuus*. Exploration on a wide geographic scale of wild populations of *H. annuus* for additional genes for downy mildew resistance is suggested.

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Additional key words: disease resistance, genetics of resistance.

Downy mildew is a particularly destructive disease of cultivated sunflowers, *Helianthus annuus* L. Downy mildew occurs with regularity in most areas of intensive sunflower cultivation, and has been of serious economic consequence in Canada (1,11), France (7), Hungary (4), Romania (13), Russia (8), the United States (14), and Yugoslavia (T. Vrebalov, Institute of Agricultural Research, Novi Sad, Yugoslavia, *personal communication*). Although the binomial *Plasmopara halstedii* (Farl.) Berl. et de Toni has conventionally been used for this complex group of pathogens (6,9), Novotelnova (8), on the basis of pathogenic and morphologic assessments, proposed the establishment of *P. helianthi* Nov. and three biological forms, f. *helianthi*, f. *perennis*, and f. *patens*.

P. helianthi f. *helianthi* (8), not known to be pathogenic to other plants than annual sunflowers, has been the agent responsible for the serious outbreak of downy mildew in the major sunflower-growing regions of eastern Europe.

The intensification of sunflower cultivation in the Red River Valley area of North Dakota and Minnesota during the past decade has resulted in an increase in the incidence of downy mildew and has prompted the screening of diverse sunflower cultivars and lines for resistance (15). The sunflower breeding line, AD 66, which was reported to possess resistance to downy mildew in Romania (13), was found to be susceptible in trials in the U.S. (17). The lack of correlation between data from tests in the two countries suggested specialization for pathogenicity between the *P. halstedii* complex in North America and Europe, and prompted the following expanded study of pathogenic specialization.

MATERIALS AND METHODS.—Sunflower lines, reported to possess resistance to downy mildew in trials in Europe (13, and P. Leclercq, Center of Agronomic Research, Clermont-Ferrand, France, *personal communication*), or that were observed by the author to possess a high level of resistance in trials at the Institute for Agricultural Research, Novi Sad, Yugoslavia, were evaluated for resistance to North American collections of *P. halstedii*. About 50 seedlings of each of 25 lines and the downy mildew-susceptible cultivar 'Peredovik' were tested in the greenhouse for resistance to field collections of *P. halstedii*. Sporangia were collected in Minnesota, North Dakota, and South Dakota in July of 1971 and June of 1972 from the cultivars 'Commander', 'VNIIMK 8931', 'Peredovik', and 'Mingren'. Each collection was maintained and increased by continual culturing on the susceptible cultivars Mingren and Peredovik.

Inoculations were performed by dehulling germinated seedlings with radicle lengths of 10-20 mm and placing them in a 50-mm petri dish, which held 15 ml of distilled water containing more than 10^4 zoosporangia/ml, for 18 hours in the dark in a cabinet at a constant temperature of 20 C. After inoculation, the seedlings were removed, transplanted into 12.7-cm pots containing sterile soil, and maintained on greenhouse benches for 14 days at 20-25 C. Disease reaction was ascertained by placing the seedlings in a saturated-humidity chamber for 18 hours. Susceptibility was indicated by the sporulation on above ground parts. Resistant plants were assumed to be those that showed no sporulation. Damping-off resulting from heavy infection occasionally developed, and was ascribed to action by the downy mildew pathogen.

Most lines were also evaluated for resistance in the field. Two replications of single rows 6.1 m (20 ft) long of each line were grown in either 1972 or 1973 on land heavily infested with *P. halstedii*. The number of seedlings per row varied from 82 to 96. The percentage of plants of each entry that showed the typical symptoms of stunting and discoloration, were determined on June 25 in 1972 and July 1 in 1973.

RESULTS AND DISCUSSION.—Less than 12% of the plants of susceptible lines escaped systemic infection in the greenhouse trials. Under field conditions, however, the percentage of plants that escaped systemic infection averaged 51%. There was no indication of pathogenic

specialization among the various field collections tested in the greenhouse.

Lines CM 29-1, HA 61-1, HIR-34, 29-3, RHA 271, RHA 273, and RHA 274 were resistant to downy mildew showing 0-2% diseased plants in the greenhouse trials and 0-1% in field trials. The first four of these were resistant to downy mildew in Europe. The last three have not been tested there but are known to contain the Pl_2 gene (D. E. Zimmer, Unpublished) and should be resistant in Europe. The resistant lines, with the exception of HIR-34, have a common ancestor, 953-88, and are, therefore, assumed to carry the same gene(s) for resistance. The resistance gene in HA 61-1 which also traces to 953-88, has been designated Pl_2 (17). Line 953-88 was derived from a rust-resistant F_2 plant of a natural cross between the mildew susceptible variety Sunrise and wild *H. annuus* (10).

Except for the cultivar Peredovik, all of the 18 entries that were highly susceptible in my trials had previously been reported (13, and P. Leclercq and T. Vrebalov, *personal communications*) or observed by the author to be resistant to downy mildew in field trials in Europe. Of these, 13 are known to have either 953-102-1-1-22 or 953-102-1-1-41 as parents, both of which, like 953-88, trace to outcrosses with wild populations of *H. annuus* in Texas (10). Based on the differential response to downy mildew in my trials and in Europe, the remaining four lines (CA 73, CV 4-2-3, CR 7-2-2, and P 1014) are suspected of having an ancestry similar to that of the other 13. Wild annual sunflowers have been observed to be highly variable for a number of morphological, physiological, and pathological characteristics (2,3); thus it is not surprising that some of them should contain sources of downy mildew resistance.

All 17 lines, susceptible in my trials but resistant in Europe, possess resistance to race 2 of *Puccinia helianthi* Schw. and are assumed to carry the R_1 gene for rust resistance. Vranceanu and Stoenescu (13) postulated linkage between the rust resistance gene (R_1) of AD 66 and the Pl gene. My observations, that additional lines such as RHA 265 and RHA 266 which carry the R_1 gene are also mildew-resistant in Europe, support that postulation.

Three genes have been discovered that condition resistance to the European race (12, 13). Only the H_1 gene, designated Pl_2 by Zimmer and Kinman (17), conditions resistance to the North American race. Gene Pl and gene H_2 (designated Pl_3 by Zimmer and Kinman), are ineffective against the North American race.

HIR 34, a French line developed from a cross of mildew-susceptible *H. annuus* var. 'Armavir 9343' × mildew-resistant *H. tuberosus* L. (Jerusalem artichoke), was highly resistant, both in Europe (5) and in my trials.

TABLE 1. Differential response of sunflower lines (*Helianthus annuus*) to downy mildew (*Plasmopara halstedii*) in European and U.S. trials

Country of origin	Line	Reported reaction in European trials[a]	Reaction in U. S. trials[b]	
			Greenhouse	Field
Canada	CM 29-1	R	0	0
	CM 90 RR	R	96	54
	S 37-388 RR	R	96	54
France	CA 73	R	98	...
	HIR-34	R	0	0
	P 1014	R	96	...
	29-3	R	2	...
	307-1	R	96	...
Romania	AD 66	R	100	52
Soviet Union	Peredovik (Control)	S	98	48
United States	HA 60	R	88	36
	HA 61-1	R	0	0
	RHA 265	R	98	62
	RHA 266	R	100	52
	RHA 271	R	2	1
	RHA 273	R	0	0
	RHA 274	R	0	0
Yugoslavia	CM 1-6-1	R	96	46
	CR 1-2-1	R	100	44
	CR 7-2-2	R	100	40
	CV 4-2-3	R	100	48
	NO 23-1	R	98	60
	SR 2-2-1	R	94	42
	SR 3-6-1	R	96	44
	SR 5-6-1	R	100	48
	SR 9-91	R	100	60

[a]R = resistant, S = susceptible.

[b]Percentage of plants with systemic symptoms based on 50 plants in the greenhouse trials, and about 200 plants in the field trials.

The origin of HIR 34 suggests that its resistance gene is unrelated to any of the other genes. Segregating ratios obtained in the F_2 and testcross populations from a cross involving an HA 61 derivative × HIR 34, however, suggests that HA 61 and HIR 34 share a common gene, which conditions resistance to the North American strain (16). This was unexpected, because Leclercq et al. (5) had reported that the gene or gene system conditioning mildew resistance of HIR 34 in Europe was carried by a chromosome from the At_1 or At_2 genome of *H. tuberosus*.

The North American *P. halstedii* closely parallels *P. helianthi* var. *helianthi* in that it also is restricted to annual sunflower. It failed to infect *H. grosseserratus* Martens, *H. maximilianii* Schral., *H. rigidus* (Cass). Desf., and *H. tuberosus* (Unpublished results of the author). Whether the North American *P. halstedii* restricted to annual sunflower is sufficiently distinct morphologically to be relegated to specific or varietal rank as *P. helianthi* f. *helianthi* is questionable.

The broadly virulent North American race of *P. halstedii* occurs throughtout the principal sunflower producing area of North America. The pathogenic specialization between the North American race and the European race, coupled with the probably long-term existence of the North American race based upon its distribution implies that the European *P. helianthi* var. *helianthi* may have evolved within the confines of the European continent, and then spread with seed movement within that continent. Regardless of the phylogenetic relationship of the North American race and the European race, it is readily evident that extreme care must be exercised to avoid the introduction of the more virulent North American race into areas of the world where it does not presently occur, especially into areas of Eastern Europe where sunflowers are the major oilseed crop.

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