New Race of *Plasmopara halstedii* Virulent on Resistant Sunflowers in South Dakota

M. L. CARSON, Assistant Professor, Plant Science Department, South Dakota State University, Brookings 57007

ABSTRACT

Carson, M. L. 1981. New race of *Plasmopara halstedii* virulent on resistant sunflowers in South Dakota. Plant Disease 65:842-843.

A new race of *Plasmopara halstedii* capable of overcoming the PI_2 monogenic resistance in sunflower (*Helianthus annuus*) has been found in a hybrid demonstration plot near Brandt, SD. None of the known sunflower genes resistant to *P. halstedii* are effective against this race.

Additional key words: downy mildew, pathogen variability

The downy mildew of sunflower (*Helianthus annuus* L.) incited by *Plasmopara halstedii* (Farl.) Berl. & De T. is a potentially devastating disease. Yield losses were as great as 50% in some fields during an outbreak of the disease in the Red River Valley of North Dakota and Minnesota in 1970 (7). Systemic infection reduces achene size, weight, and oil percentage and increases hull percentage (11).

Genetically resistant hybrids are used to control downy mildew in sunflower. At least three dominant genes for resistance to *P. halstedii*, designated PI_1, PI_2, and PI_3, have been identified (5,6,10). All three confer resistance to European biotypes of *P. halstedii*, while only the PI_2 gene has been effective against North American biotypes (8). Hybrids with the PI_2 gene are now used throughout the north central sunflower growing region.

This article reports the occurrence of a *P. halstedii* isolate that is virulent to sunflower genotypes containing the PI_2 gene. The isolate was found in a commercial field of hybrid oilseed sunflower in east central South Dakota. About 50% of the plants in the field exhibited varying degrees of stunting and deformity. Six commercial hybrids were being grown in strips in the field for demonstration purposes. All the hybrids were purported to carry the PI_2 gene for resistance to *P. halstedii*. Diseased plants were found throughout the field and in all hybrids in an apparently random distribution.

MATERIALS AND METHODS

Source of isolates. I first observed the diseased field of oilseed sunflower near Brandt, SD, in early Octoer 1980 when all leaf tissue was either killed by frost or senescent. I thus obtained the Brandt isolate of *P. halstedii* by sowing USDA hybrid 894 in 15-cm-diameter clay pots filled with field soil from around the roots of the diseased plants. Two weeks later, the emerged seedlings in the pots were placed in a mist chamber overnight in a greenhouse (20–22 C) to obtain sporulation of *P. halstedii*. Systemically infected seedlings with visible sporulation on cotyledons and primary leaves were harvested for use as inoculum.

I obtained the Red River Valley isolate in the form of infected sunflower seedlings from Dr. T. Gulya, Agricultural Research Service, U.S. Department of Agriculture, Fargo, ND. The seedlings were placed in a mist chamber in the greenhouse to obtain sporulation of *P. halstedii* and inoculum of the Red River Valley isolate as described for the Brandt isolate.

Virulence tests. I used the Brandt and Red River Valley isolates in separate trials to infect a set of sunflower genotypes. Seeds of inbred sunflower lines RHA265, RHA271, RHA273, RHA274, HA61, HA89, and USDA hybrid 894 were germinated on moist filter paper in 100-mm-diameter petri dishes for 3 days and placed in suspensions of zoosporangia (10^4 – 10^5/ml) of *P. halstedii* overnight at 20 C to allow the zoospores to emerge and infect the seedlings. I then transplanted the seedlings into a potting soil mix of soil, sand, and peat moss (2:1:1, steamed overnight) in 15-cm-diameter clay pots in the green-house at 20–22 C. Ten to 14 days later, the plants were placed in a mist chamber in the greenhouse at 20–22 C to obtain sporulation of *P. halstedii*. I determined the number of systemically infected plants by the presence of sporulation on leaves and cotyledons.

RESULTS AND DISCUSSION

All sunflower genotypes tested were susceptible to the Brandt isolate of *P. halstedii* (Table 1). A genotype was considered susceptible if a high percentage of seedlings ($\geq$75%) exhibited sporulation of *P. halstedii* on leaves and cotyledons or resistant if a very low percentage of seedlings exhibited sporulation (6,8). All PI_2 genotypes were resistant to the common or Red River Valley race, but PI_1 genotypes were not resistant to either of the isolates. The inbred line HA61, which contains PI_2 and possibly PI_3, was susceptible to the Brandt isolate, indicating that the PI_3 gene (if it exists) was also not effective against the new biotype.

Because producers of hybrid sunflower seed in the principal sunflower growing region of the United States rely on the PI_2 gene for control of downy mildew, this new biotype of *P. halstedii* is of concern. The frequency and distribution of biotypes virulent on PI_2 genotypes in the north central region is not yet known. Fick (2) recently reported a similar isolate virulent upon PI_2, but it was not known whether this isolate was present in the field or arose through mutation or recombination during greenhouse testing (2). The report is evidence, however, that

Table 1. Incidence of downy mildew in seedlings of a hybrid and six inbred lines of sunflower inoculated with two isolates of *Plasmopara halstedii*

Hybrid or line	Resistant gene	Incidence of downy mildew[a]	
		Brandt isolate	Red River Valley isolate
RHA265	PI_1	21/21	8/9
RHA271	PI_2	17/18	0/33
RHA273	PI_2	12/12	0/27
RHA274	PI_2	18/18	0/32
HA61	PI_2, PI_3	13/17	0/21
HA89	None	...[b]	36/44
USDA hybrid 894	PI_2	56/56	...

[a] Number of seedlings evaluated/number exhibiting sporulation of *P. halstedii*. Numbers of seedlings evaluated in each genotype varied because of seed availability, germination, and seedling emergence.

[b] The genotype was not evaluated against this isolate.

Accepted for publication 28 July 1981.

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. § 1734 solely to indicate this fact.

0191-2917/81/10084202/$03.00/0

the mutation or selection for virulence to the PI_2 gene is not an isolated event and may occur again. Disease surveys during the 1981 growing season will allow plant pathologists to determine the distribution and the potential impact of new biotypes virulent on PI_2 genotypes.

Until a source of resistance to this new race of *P. halstedii* is discovered and incorporated into commercially used inbred lines, a systemic fungicidal seed treatment can be used in affected fields to protect seedlings during critical growth stages (1,3,4,9).

Because the pathogen undergoes a sexual cycle every season and has exhibited physiologic specialization, new races of *P. halstedii* can be expected to arise as resistant genes are developed. New genes that are resistant to *P. halstedii* should be found, and hybrids with more than one effective gene for resistance to the pathogen should be developed. Researchers should also explore the possibility of lines with nonspecific, horizontal resistance to *P. halstedii,* as reported by Vear (5).

LITERATURE CITED

1. Cohen, Y., and Sackston, W. E. 1973. Factors affecting infection of sunflowers by *Plasmopara halstedii.* Can. J. Bot. 51:15-22.
2. Fick, G. N. 1981. A new race of downy mildew. In Proc. Sunflower Forum, 4th Sunflower Assoc. Am. Fargo, ND. (In press).
3. Sackston, W. E. 1979. Treatment with Ridomil to control downy mildew of sunflowers. Sunflower Newsl. 3(4):7-8. Int. Sunflower Assoc. Zevenaar, Netherlands.
4. Sackston, W. E. 1980. Control of downy mildew of sunflower by treatment with Ridomil. (Abstr.) Phytopathology 70:468.
5. Vear, F. 1974. Studies on resistance to downy mildew in sunflowers (*Helianthus annuus* L.). Pages 297-302 in: Proc. Int. Sunflower Conf., 6th. Bucharest, Romania.
6. Vear, F., and Leclercq, P. 1971. Deux nouveaux genes de resistance au mildiou du tournesol. Ann. Amelior. Plant. 21:251-255.
7. Zimmer, D. E. 1971. A serious outbreak of downy mildew in the principal sunflower production area of the United States. Plant Dis. Rep. 55:11-12.
8. Zimmer, D. E. 1974. Physiological specialization between races of *Plasmopara halstedii* in America and Europe. Phytopathology 64:1465-1467.
9. Zimmer, D. E. 1975. Some biotic and climatic factors influencing sporadic occurrence of sunflower downy mildew. Phytopathology 65:751-754.
10. Zimmer, D. E., and Kinmann, M. C. 1972. Downy mildew resistance in cultivated sunflower and its inheritance. Crop Sci. 12:749-751.
11. Zimmer, D. E., and Zimmerman, D. C. 1972. Influence of some diseases on achene and oil quality of sunflower. Crop Sci. 12:859-861.