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

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ABSTRACT

A new race of Plasmopara halstedii capable of overcoming the PI1 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 (Farr.) 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 PI1, PI2, and PI3, have been identified (5,6,10). All three confer resistance to European biotypes of P. halstedii, while only the PI1 gene has been effective against North American biotypes (8). Hybrids with the PI1 gene are now used throughout the northern center sunflower growing region.

This article reports the occurrence of a P. halstedii isolate that is virulent to sunflower genotypes containing the PI1 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 purposed to carry the PI1 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 observed the diseased field of oilseed sunflower near Brandt, SD, in early October 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 - 10^7/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 greenhouse 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 (>75%) exhibited sporulation of P. halstedii on leaves and cotyledons or resistant if a very low percentage of seedlings exhibited sporulation (6,8). All PI1 genotypes were resistant to the common or Red River Valley race, but PI1 genotypes were not resistant to either of the isolates. The inbred line HA61, which contains PI1 and possibly PI3, was susceptible to the Brandt isolate, indicating that the PI1 gene (if it exists) was also not effective against the new isolate.

Because producers of hybrid sunflower seed in the principal sunflower growing region of the United States rely on the PI1 gene for control of downy mildew, this new isolate of P. halstedii is of concern. The frequency and distribution of biotypes virulent on PI1 genotypes in the north central region is not yet known. Fick (2) recently reported a similar isolate virulent upon PI1, 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

<table>
<thead>
<tr>
<th>Hybrid or line</th>
<th>Resistant gene</th>
<th>Incidence of downy mildew*</th>
</tr>
</thead>
<tbody>
<tr>
<td>RHA265</td>
<td>PI1</td>
<td>21/21</td>
</tr>
<tr>
<td>RHA271</td>
<td>PI1</td>
<td>17/18</td>
</tr>
<tr>
<td>RHA273</td>
<td>PI1</td>
<td>12/12</td>
</tr>
<tr>
<td>RHA274</td>
<td>PI1</td>
<td>18/18</td>
</tr>
<tr>
<td>HA61</td>
<td>PI1, PI3</td>
<td>13/17</td>
</tr>
<tr>
<td>HA89</td>
<td>None</td>
<td>...^b</td>
</tr>
<tr>
<td>USDA hybrid 894</td>
<td>PI1</td>
<td>56/56</td>
</tr>
</tbody>
</table>

*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.

^bThe genotype was not evaluated against this isolate.
the mutation or selection for virulence to the Pl 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 Pl 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**


