Importance of Secondary Inoculum in Strawbreaker Foot Rot of Winter Wheat

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ABSTRACT

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Inoculations with conidia of *Pseudocercosporella herpotrichoides* in November (1979) reduced yields of a susceptible wheat (Sel. 101) by 56%. Inoculations in February and March (1980) had little effect on yield or bushel weight, but severe lesions developed on many stems. The early infections, representing the effect of primary inoculum, contributed most to yield loss, but the late infections, representing the effect of secondary inoculum, added to the amount of inoculum for succeeding crops. Inoculations of the same wheat three times, once each in November, December, and February, did not increase disease appreciably over that resulting from a single heavy inoculation in November. A single, heavy fall inoculation is recommended for variety screening trials.

Strawbreaker foot rot (Cercosporella footrot or eye spot) is caused by a facultative saprophyte, *Pseudocercosporella herpotrichoides* (Fron) Deighton, that produces hyaline, septate conidia on the living plant and old lesions on the stubble of dead wheat (*Triticum aestivum* L.). The primary inoculum in cultivated fields comes from the residue of previously diseased plants. Sporulation on stubble usually begins in November and continues through April during cool, moist periods. Inoculum spread can, when the weather is cool and moist, begin in September and continue through May (7). Consequently, primary inoculum can be available to infect the winter wheat throughout fall, winter, and early spring. If primary inoculum is abundant, secondary inoculum would theoretically add little to disease development. However, when primary inoculum is limited, usually by previous crop rotation (2,3,8), secondary inoculum could be important. Secondary inoculum (13) consists of conidia produced on lesions on the current season crop.

The time required for the development of lesions with fungal stromata varies with temperature. Ponchet (12) in France observed stromata between 31 and 55 days after inoculation. The stromata formed between 20 October and 11 December during five different growing seasons. Sprague and Fellows (15) reported typical lesions 4 wk after 2-mo-old plants were inoculated in a greenhouse. Oort (11) in The Netherlands and Kerleroux (9) in France found lesions as early as November. In Washington identifiable lesions are rare earlier than February, but Chidambaram (1) found abundant conidia on winter wheat in November in 1975 at Pullman. We have found them (once) in November, in December in 1978 and 1979, and in January in 1980. Every time we examined a lesion with a stroma in late fall and early winter we found conidia. We assume that any lesion with a stroma is capable of sporulation until the end of spring. Some workers found conidia on hosts before the development of macroscopically visible lesions (6,7,14), so this assumption is probably valid.

Because Rowe and Powelson (13) reported that secondary inoculum is of little significance to the current season in eastern Oregon and because our observations and many of the reports in the literature indicated that this view may be only partially correct, we inoculated wheat in different months to simulate primary inoculum (early inoculations) and secondary inoculum (later inoculations).

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MATERIALS AND METHODS

Daws (CI 17419), susceptible, and Selection (Sel.) 101 (CI 13438), highly susceptible to *P. herpotrichoides*, were seeded 11 September 1979 in four-row plots on land free of *P. herpotrichoides* on the Dryland Experiment Station, Lind, WA. The site was irrigated with 10 cm of water before planting. The rows were 40 cm apart and 3 m long. Alleys were 1.3 m wide, and 0.6 m separated the outer rows of each plot. There were three replicates in experiment 1 and four in experiment 2.

Oat (*Avena sativa* L.) kernels infested with *P. herpotrichoides* (10,13) were washed to remove debris and then incubated outdoors on a plastic screen on soil at Puyallup, WA, in early October 1979. On 31 October, conidia were washed from the oat kernels and a suspension containing 250,000 conidia per milliliter was prepared. This suspension was sprayed onto wheat crowns until runoff on 1 November. The nozzle of the sprayer was held close to the ground, and all plants in each plot were inoculated. The wheat was in the 4- to 10-tiller stage. Other inoculations, with fresh batches of conidia (250,000 conidia per milliliter), were made on 4 December 1979 and on 6 February and 3 March 1980. One treatment consisted of wheat inoculated three times (once on each spray date in November, December, and February).

The plants in the center two rows (2.4 m^2) of each plot were harvested at maturity and the yields and test weights determined. Stubble was examined for lesions.

Approximately equal numbers of straws of Sel. 101 and Daws were graded for lesion severity, soaked overnight in water at 10 C, and then incubated outdoors starting 17 November 1980 at Pullman. The straws were washed in 100–300 ml of water, the amount varying with the number of straws, and the number of conidia per straw present in the wash water was counted with the aid of a hemacytometer.

RESULTS

Yields and test weights were reduced (Tables 1 and 2) by the November and December inoculations that simulated heavy early primary inoculum. Yield and test weight were little affected by the February and March inoculations that simulated heavy secondary inoculum (Tables 1 and 2). The yield losses resulted

Table 1. Yield and test weight of Selection 101 winter wheat inoculated with conidia of *Pseudocercosporella herpotrichoides* at Lind, WA, 1979–1980, plot 1

Inoculation date	Yield (g/plot)	Test weight	
		(lb/bu)	(kg/hl)
1 November	393[a]	55.5	71.5
4 December	573	57.5	74.1
6 February	880	60.9	78.4
3 March	892	61.5	79.2
Control	948	61.1	78.7
Nov. + Dec. + Feb.	424	55.8	71.9

[a] LSD_{05} = 171 g.

from empty heads, light grain, and dead tillers. Neither Sel. 101 nor Daws, both semidwarf winter wheats, lodged appreciably in these plots. The triple inoculations (November + December + February) did no more damage than the single heavy November inoculation, indicating that a single inoculation early in the fall achieved maximum disease prevalence and severity.

The data for Sel. 101 of plots 1 and 2 (Tables 1 and 2) were combined, making seven replicates. When this was done, the average yields for the November inoculation (375 g) and for the repeated inoculations (November + December + February) were still not significantly different (384 g). The November inoculation resulted in a yield reduction ($P = 0.01$) below the December inoculation (375 vs. 509 g). The February inoculation (818 g) did not reduce yield significantly below that of the controls (845 g).

Even though the late inoculations had little effect on the yield and test weight of Sel. 101 and had no effect on Daws, extensive infection and lesion development occurred (Table 3).

Wheat inoculated in November and December was heavily diseased by February, so lesions resulting from the February and March inoculations represent those that could result from secondary spread. The infected controls resulted from the natural spread of conidia from adjacent plots inoculated at earlier dates. Lesions from the late inoculations were well developed with abundant stromata and conidia (Table 4). The straws were not materially weakened, however, and the yields and test weights were not reduced.

Table 2. Yields and test weights of winter wheat inoculated with conidia of *Pseudocercosporella herpotrichoides* at Lind, WA, 1979–1980, plot 2.

	Selection 101			Daws		
	Yield	Test weight		Yield	Test weight	
Inoculation date	(g/plot)	(lb/bu)	(kg/hl)	(g/plot)	(lb/bu)	(kg/hl)
1 November	361[a]	57.8	74.4	683[a]	60.1	77.4
4 December	461	58.0	74.7	787	60.2	77.5
6 February	771	61.1	78.7	883	61.5	79.2
Control	783	61.8	79.6	894	61.5	79.2
Nov. + Dec. + Feb.	354	56.1	72.3	659	59.2	76.2

[a] LSD_{05} = 131 g.

Table 3. Foot rot lesions on stems of winter wheat inoculated in February or March with conidia of *Pseudocercosporella herpotrichoides* at Lind, WA, 1979–1980, plot 1

	Lesion rating[a]				
Inoculation date	0	1	2	3	4
		Selection 101			
6 February	22	53	121	113	33
3 March	2	49	123	116	18
Control	174	80	72	38	0
		Daws			
6 February	0	44	210	189	15
Control	101	106	212	67	0

[a] 0 = healthy stem, 4 = very extensive lesions with heavy stroma.

Table 4. Conidia produced by *Pseudocercosporella herpotrichoides* per winter wheat stem incubated outdoors, November 1980 to May 1981[a]

	Conidia (×1000) at stem lesion rating[b] of				
Sample date	0	1	2	4	Total
		Daws			
17/12/80	0	80	113	1,060	1,253
26/1/81	662	1,128	2,021	8,278	12,539
5/3/81	172	471	874	2,047	3,564
3/4/81	23	113	83	373	592
12/5/81	27	40	107	170	344
Total	884	1,832	3,198	12,378	
		Selection 101			
17/12/80	63	50	88	150	351
26/1/81	202	1,025	707	6,500	8,434
5/3/81	76	600	727	750	2,153
3/4/81	23	80	150	397	650
12/5/81	10	60	77	227	374
Total	374	1,815	1,749	8,024	

[a] The winter was mild and wet and ideal for sporulation.
[b] The number of straws in each sample varied from 48 to 94. 0 = healthy stem, 4 = very extensive lesions with heavy stroma.

DISCUSSION

The effect of date of inoculation of wheat with *P. herpotrichoides* has been studied by many workers, but the results have varied considerably. Foex (6) in France inoculated wheat on 15 January 1935, and the wheat was severely diseased by harvest. Disease was less severe from inoculations made between mid-February and April. Inoculation on 15 May had little effect on the yield. In contrast, Sprague and Fellows (15) reported severe disease following a February 1934 inoculation at Corvallis, OR, a humid site. Chidambaram (1) inoculated wheat at Lind, WA, a dry site, in March 1975, and severe lesions developed on 8–20% of the stems, with some white heads. In the same season, an April inoculation of late-seeded, sprinkler-irrigated wheat at Lind resulted in severe lodging and white heads. Weather and stage of host development probably influence the effect of date of inoculation on disease development.

Our results and those of Rowe and Powelson (13) were obtained under relatively dry conditions. Under dry spring conditions, primary inoculum accounts for most of the yield loss. Our results, like those of Rowe and Powelson (13), indicate that under dry conditions secondary inoculum is important mostly because it increases the inoculum level for succeeding crops.

The February and March inoculation (Table 3) resulted in severe, extensive lesions on more than 40% of the stems, but the effect on the crop was slight. In these late infections, lignification of the hypodermis and vascular bundles must have preceded fungal attack so that the stem remained standing and the vascular tissue continued to function. The parenchyma was ramified by mycelium, supporting abundant fungal stromata on the lesions.

We concluded from these trials that a single heavy inoculation with conidia in autumn is a reliable way to test breeding materials for resistance to *P. herpotrichoides*. Chidambaram (1) reported that little disease developed in the 1975–1976 season at Pullman from oat-kernel inoculum because the warm, dry fall and cold winter suppressed adequate sporulation, spread, and disease development. Oat inoculum broadcast in a breeding nursery in late October at Pullman in the 1978–1979 season also failed to produce severe, significant disease. The fall of that season was dry until winter, and the winter was constantly cold. Disease developed too little and too late for meaningful results in a breeding test. Use of conidial suspensions in early November should prevent this type of failure in a breeding program.

Evidence of the potential importance of secondary inoculum is provided from humid sites in New York (3) and Oregon in the United States (15) and in England (2,8), France (12), Germany (4), and Norway (10). The experiments of Fehrmann and Schrödter (5) in Germany in particular illustrate the role of secondary inoculum in the development of foot rot after another crop in the rotation. The ability to sporulate on the host during the season of crop growth is a valuable attribute of the pathogen in many situations, and it is unsafe to extend results from one location to another.

Foex (6) and Glynne (7) found conidia on coleoptiles before visible lesions had developed. We found slight sporulation (Table 4) on mature straws with no distinct lesion symptoms. This may contribute to the survival of the fungus under minimal conditions.

The straws (Table 4) are all from the February inoculation, representing those that could result from secondary spread. Two things are particularly noteworthy: conidia were obtained from straws on which identifiable symptoms were not observed (the 0 or healthy rating), and a very great number of conidia were washed from the straws rated as severely diseased. Even 12,000,000 conidia from one straw during a sporulating season is not the total number. How many conidia washed off into the soil between sampling dates? Most of the straws had an undetermined number of lesions per straw: the data are conidia per straw, not conidia per lesion. Late infections (if the February inoculation can be called late) result in lesions that add materially to the conidia for future crops even though they may have little or no effect on the current season crop.

The severely diseased stems produced many more conidia than the lightly diseased stems. A cultivar resistant to *P. herpotrichoides* with few severely diseased culms could reduce the inoculum level for subsequent crops.

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