Reaction of Winter Oat Germ Plasm to an Epidemic of Oat Powdery Mildew

S. LEATH, USDA-ARS, North Carolina State University, Raleigh 27695-7616, P. L. BRUCKNER, University of Georgia, Tifton 31793-0748, and J. P. WILSON, USDA-ARS, University of Georgia, Coastal Plain Experiment Station, Tifton 31793-0748

ABSTRACT

Leath, S., Bruckner, P. L., and Wilson, J. P. 1991. Reaction of winter oat germ plasm to an epidemic of oat powdery mildew. Plant Dis. 75:807-809.

In the spring of 1989, an epidemic of oat powdery mildew occurred naturally in four randomized blocks near Tifton, Georgia, in which 31 winter oat lines had been planted in the fall of 1988. Plots were rated on 14 and 18 April for percentage of foliage covered with mildew and for infection type (0–4 scale). Differences existed among cultivars on both assessment dates. Severity levels increased from a mean of 29.2 to 32.9% between assessment dates, and all cultivars showed symptoms; oat lines ranged from 7.5 to 59.4% in severity and from 1.4 to 4.0 in infection type. The same cultivars were tested in a growth chamber with a pure culture of *Blumeria graminis* f. sp. *avenae* recovered from oat cultivar Brooks to determine if they could be effectively screened as seedlings under controlled conditions. Seedling reaction means ranged from 2.3 to 7.0 on a 0–9 scale of powdery mildew severity and from 1.8 to 3.8 on a 0–4 scale for infection type. Severity and infection type data were correlated in both field ($r = 0.84$, $P < 0.01$) and controlled environments ($r = 0.50$–0.67, $P < 0.01$). Severities in the field and growth chamber also were correlated ($r = 0.37$–0.46, $P < 0.05$). Based on all data, the most resistant lines were AR-111-2, AR-02848, AR-820B-669, and GA-T81-1251. The lines PA 8014-608, PA 7915-1342, and TAMO 386 were the most susceptible. Several lines that may express adult plant resistance showed large differences between field and greenhouse evaluations; NK Coker 86-10, NK Coker 716, and Simpson showed high levels of resistance only in the field. This may be due to the existence of a mixture of pathogen phenotypes in the Southeast, despite the fact that no cleistothecia were observed in North Carolina or Georgia, making evaluation with a single isolate undesirable.

Powdery mildew of oats (*Avena sativa* L.) is caused by the ascomycete *Blumeria graminis* (DC.) E.O. Speer f. sp. *avenae* Ém. Marchal (= *Erysiphe graminis* DC. f. sp. *avenae* Ém. Marchal) and has occurred sporadically on winter oats in the southeastern United States. Powdery mildew is the most important disease of oats in the United Kingdom, however, causing estimated annual grain losses of 5–10% (9,11). The disease has the potential to severely limit oat production, and losses of approximately 40 and 20% have been reported by Lawes and Hayes (10) and Jones (6), respectively.

Powdery mildew of oats can be controlled through the use of host resistance or fungicides or by integration of the two approaches (9). Physiological specialization has been reported in *B. g. avenae* from the United Kingdom (3,4), but no race determinations have been completed in the United States. Emphasis in oat mildew control has been placed on adult plant resistance (1,5,7,8) in the hope that it will be durable and effective against a wide range of pathogen phenotypes (8).

The current winter oat germ plasm in use in the Southeast has not been characterized for its reaction to *B. g. avenae*. Over 4,000 oat cultivars and selections were evaluated for seedling reaction in the greenhouse in 1951 to an uncharacterized isolate of *B. g. avenae* from Iowa (2); 59 were classified as resistant. In previous work, levels of adult plant resistance and seedling resistance were not always correlated (4).

One of us (S. Leath) observed powdery mildew in a winter oat nursery during 1986 and at low levels in commercial and nursery fields during 1987 and 1989 in central North Carolina. We also observed powdery mildew during 1989 in nursery fields in southern Georgia. However, no cleistothecia of the fungus were observed in any of these instances. Because of the more regular and intense occurrence of oat powdery mildew coupled with its destructive potential, we believed the germ plasm grown in the southeastern United States should be evaluated. Our objectives were to evaluate current winter oat germ plasm for powdery mildew resistance and to determine the effectiveness of evaluating germ plasm as seedlings under controlled conditions.

MATERIALS AND METHODS

Thirty-one entries from combined 1988–1989 USDA-ARS Uniform Southern, Uniform Central, and Georgia oat performance nurseries were planted on the Gibbs Research Farm near Tifton, Georgia. Seed was planted 11 November 1988 in rows 30 cm apart. Plots were 1.25 m wide and 3 m long and were arranged in four randomized complete blocks. Before planting, all plots received 560 kg/ha of 5-10-15 (N-P_2O_5-K_2O). On 3 February, all plots were top-dressed with 67 kg/ha of nitrogen as ammonium nitrate.

Powdery mildew was first observed in late February and developed naturally. All entries had panicles visible by 12 April, and one line reached this stage by 1 April. Twenty-five entries reached heading between 4 and 8 April.

Plots were evaluated twice, first on 14 April and again 4 days later, approximately 13 and 17 days after initial head emergence. Plants in the center of each plot were rated on two scales. Disease severity was considered as the percentage of all emerged leaf tissue covered with colonies and was estimated visually at increments of 1, 3, 5, 10, 15, . . . 100%. The predominant infection type was scored on a 0–4 scale, where 0 = no visible symptoms, 1 = chlorotic flecking, 2 = sparse mycelial production, 3 = small colonies with sparse to moderate amounts of mycelium, and 4 = large colonies without a hypersensitive host response (12).

An isolate of *B. g. avenae* was collected from the oat cultivar Brooks by transferring three times from single colonies to establish purity. The isolate then was maintained on seedlings of Brooks oats grown at 17 C (13 hr days and approximately 75 $\mu E \cdot m^{-2} \cdot s^{-1}$ available light provided by cool-white fluorescent bulbs).

Seed of the 31 lines tested in the field were sown in 5.5-cm^2 plastic pots in a commercial potting mix (Metro-Mix, W. R. Grace & Co., Cambridge, MA) at 17 C under 13 hr days with 75 $\mu E \cdot m^{-2} \cdot s^{-1}$ available light in a growth chamber.

Plants 11 days old were inoculated in a settling tower 1.0 m high × 0.5 m wide × 0.5 m long. Sections of leaf 3 cm long and with numerous colonies were used for inoculations. Conidia were dislodged in a stream of air for 30 sec and allowed to settle. Conidia were deposited uniformly (based on direct counts of conidia on glass slides) and at a mean rate of 714/cm^2. Plants in the three ran-

The use of trade names in this publication does not imply endorsement by the North Carolina Agricultural Research Service or criticism of similar ones not mentioned.

Accepted for publication 28 January 1991 (submitted for electronic processing).

domized complete blocks were returned to the growth chamber until evaluation. At 11 and 18 days postinoculation, plants were evaluated on a 0–9 scale for disease severity and on a 0–4 scale for infection type (12). The 0–9 scale expressed the percentage of the second leaf covered with colonies, where 0 = no visible symptoms and 9 = greater than 90% of the second leaf covered with colonies. The 0–4 scale was identical to that described earlier. Two seedlings per pot were considered as subsamples, and one pot per oat line was considered an experimental unit. The experiment was repeated with a mean conidial deposition rate of 403 conidia per square centimeter.

RESULTS AND DISCUSSION

Natural conditions and levels of inoculum in 1989 at Tifton, Georgia, resulted in unusually high levels of oat powdery mildew, and all 31 lines under evaluation were symptomatic. Severity ranged from 7.5% on NK Coker 86-10 to 59.4% on PA 8014-608. Only two lines, NK Coker 86-10 and NK Coker 716, expressed a high level of resistance with a mean infection type < 2.0 (Table 1). The extreme variation in infection types among cultivars suggests that more than one physiologic race may have been present. Severity data and infection type data were highly correlated ($r = 0.84$, $P < 0.01$), and no large inconsistencies were observed (Table 2). Both severity and infection type data were consistent across the two dates, with $r = 0.86$ and 0.79 ($P < 0.001$), respectively. However, there was no association between powdery mildew severity or infection type in the field and grain yield or test weight (*data not shown*). This could be due in part to the high levels of crown rust (caused by *Puccinia coronata* Corda) present later in the season. The presence of crown rust did not corrupt powdery mildew data, however, because the rust developed after powdery mildew data had been recorded. On the first recording date (14 April), only eight of 96 (8.3%) of the plots had crown rust severities above 1%.

Growth chamber results were consistent across repetitions of the experiment, and data were similarly correlated to results from the field study. All lines showed symptoms under controlled conditions, with mean severities across both runs of the experiment ranging from 2.3 on AR-02848 to 7.0 on Florida 502 (Table 1). This range is similar to that observed in the field. Infection types also ranged similarly from 1.8 to 3.8. However, the correlation between disease severity and infection type was weaker under controlled conditions with the single isolate than under field conditions (Table 2).

Severity data from the growth chamber and infection types from the field were both indicative of disease severity in the field (Table 2). There was little association between infection type in the growth chamber and infection type or severity in the field. This may be attributable in part to adult plant resistance, which would not be detected in seedlings, and to evaluation with only one isolate. Further, resistance in the field is often expressed quantitatively and may not be well associated with infection type. In addition, several lines showed large differences in disease severity between field and growth chamber environments. This could be due to a number of factors, including environment, adult plant resistance, and the particular isolate used in growth chamber testing. Hayes and Jones (4) indicated that resistance between adults and seedlings may not be closely associated for oat powdery mildew.

The fact that no cleistothecia have been observed at multiple sites and years in North Carolina and during a severe epidemic at Tifton, Georgia, indicates that sexual recombination may be relatively rare in *B. g. avenae* compared with that in other form species of *B. graminis* found in this region. Whether specificity is actually operating in this pathosystem is still unclear. Growth chamber testing with a single isolate collected off a moderately resistant line was not a good indicator of field resistance. Further, all lines showed enough disease to indicate no typical single gene resistance to powdery mildew was effectively operating against this isolate. If powdery mildew of oats becomes a problem in the southeastern United States, as it has in Europe, determining if specificity is operating and having resistant germ plasm available will be important. At least six lines (AR-111-2, AR-02848, AR-820B-669, NK Coker 716, NK Coker 86-10, and GA-T81-1251) showed high levels of resistance when all data were considered.

Table 1. Reactions of winter oat lines to a natural population of *Blumeria graminis* f. sp. *avenae* at Tifton, Georgia, in 1989 and to an isolate recovered from Brooks oats when evaluated under controlled conditions in a growth chamber

					Infection type[y]			
	Severity in field[w]	Severity in growth chamber[x]				Growth chamber		
Genotype	(%)	Expt. 1	Expt. 2	Comb.[z]	Field	Expt. 1	Expt. 2	Comb.
NK Coker 86-10	7.5	6.7	4.0	5.3	1.4	4.0	2.7	3.3
NK Coker 716	13.1	5.3	2.7	4.0	1.9	3.3	2.7	3.0
NK Coker 227	15.0	7.3	4.0	5.7	2.0	4.0	3.7	3.8
Simpson	16.1	4.7	4.3	4.5	2.0	4.0	3.3	3.7
AR-111-2	21.9	···	3.7	3.7	2.4	···	2.3	2.3
Brooks	21.9	3.3	5.7	4.5	2.5	2.6	3.0	2.8
AR-02848	22.5	2.3	2.3	2.3	2.6	2.3	2.0	2.2
NK Coker 87-10	23.8	4.7	2.7	4.5	2.4	2.7	2.7	2.7
AR-FOB-29	25.0	4.3	3.7	4.0	2.9	1.3	2.3	1.8
AR Co. Seed 833	25.1	6.0	5.7	5.8	2.5	3.7	3.7	3.7
AR-820B-669	25.6	4.3	2.0	3.5	2.1	1.6	2.0	1.8
GA-T81-1251	25.6	4.0	4.0	4.0	2.5	1.6	2.0	1.8
PA 8014-599	27.8	7.3	6.3	6.8	2.2	2.7	3.7	3.2
NK Coker 84-27	29.4	2.7	5.7	4.2	3.1	1.0	3.3	2.2
Conlee Blizzard	29.4	7.3	3.7	5.5	2.8	3.7	3.7	3.7
AR-02826	30.0	5.7	3.5	4.8	2.6	3.7	3.0	3.4
NK Coker 820	30.6	6.0	5.0	5.5	3.2	3.3	2.0	2.7
NK Coker 86-8	30.6	7.3	5.0	6.2	3.4	4.0	3.6	3.8
AR-FOB-30	32.5	4.0	4.3	4.2	3.1	3.0	3.0	3.0
Madison	33.1	5.3	4.3	4.8	2.6	2.3	3.0	2.7
Bob	33.8	2.5	3.6	3.2	3.0	1.5	2.0	1.8
AR-820B-965	38.1	5.0	3.7	4.3	3.4	3.0	3.3	3.2
Florida 502	39.4	8.0	6.0	7.0	3.2	3.0	2.3	2.7
Florida 501	40.0	3.3	4.3	3.8	3.2	2.0	2.7	2.3
GA-T81-1249	43.1	5.3	6.0	5.7	3.8	1.7	3.7	2.7
NK Coker 87-11	44.4	6.0	4.0	5.0	3.5	3.3	2.3	2.8
AR-102-5	45.0	7.5	3.0	4.8	3.6	3.5	2.6	3.0
PA 7915-1342	46.2	5.0	6.3	5.7	3.1	3.3	3.3	3.3
TAMO 386	46.2	6.7	6.7	6.7	3.8	3.3	3.7	3.5
AR-125-4A	46.9	5.7	1.3	3.5	3.4	3.3	3.0	3.2
PA 8014-608	59.4	5.7	7.0	6.3	4.0	3.7	3.0	3.3
LSD ($P = 0.05$)	9.0	1.9	4.1	2.0	0.7	1.2	2.0	0.8
CV × 100%	27.3	32.1	41.2	39.5	24.9	26.9	33.9	31.9

[w]Values are based on percentage of all emerged leaf tissue covered with colonies averaged over two dates approximately 13 and 17 days after head emergence and represent averages of four replications.

[x]Values are based on a 0–9 scale, where 0 = no visible symptoms and 9 = greater than 90% of the second leaf covered with colonies, and represent means from three replications in two experiments.

[y]Values are based on a 0–4 scale, where 0 = no visible symptoms and 4 = large colonies without a hypersensitive host response (12).

[z]Data are from combined analysis of the two repetitions of the growth chamber experiments.

Table 2. Spearman rank correlation coefficients between field evaluations at Tifton, Georgia, in 1989 and growth chamber evaluations of 31 winter oat lines for resistance to powdery mildew caused by *Blumeria graminis* f. sp. *avenae*

	Infection type[w]			Severity in field[x] (%)	Severity in growth chamber[y]	
		Growth chamber				
	Field	Expt. 1	Expt. 2		Expt. 1	Expt. 2
Infection type field	1.00	...	...	...	...	...
Infection type growth chamber, expt. 1	0.05	1.00	...	...	...	...
Infection type growth chamber, expt. 2	0.02	0.41*[z]	1.00	...	...	...
Severity field	0.84**	0.00	0.22	1.00	...	...
Severity growth chamber, expt. 1	0.28	0.67**	0.42*	0.37*	1.00	...
Severity growth chamber, expt. 2	0.30+	0.07	0.50**	0.46**	0.24	1.00

[w]Values are based on a 0–4 scale, where 0 = no visible symptoms and 4 = large colonies without a hypersensitive host response (12).

[x]Values are based on percentage of all emerged leaf tissue covered with colonies averaged over two dates approximately 13 and 17 days after head emergence and represent averages of four replications.

[y]Values are based on a 0–9 scale, where 0 = no visible symptoms and 9 = greater than 90% of the second leaf covered with colonies, and represent means from three replications in two experiments.

[z]** = $P < 0.01$, * = $P < 0.05$, and + = $P < 0.10$.

ACKNOWLEDGMENT

We thank L. C. Whitcher for excellent technical assistance during the growth chamber studies.

LITERATURE CITED

1. Carver, T. L. W., and Carr, A. J. H. 1977. Race non-specific resistance of oats to primary infection by mildew. Ann. Appl. Biol. 86:29-36.
2. Finkner, R. E., Murphy, H. C., and Atkins, R. E. 1953. Reaction of oat varieties to powdery mildew. Agron. J. 45:92-95.
3. Hayes, J. D., and Catling, W. S. 1963. Physiological specialization in *Erysiphe graminis* DC. in oats. Nature 199:1111-1112.
4. Hayes, J. D., and Jones, I. T. 1966. Variation in the pathogenicity of *Erysiphe graminis* D.C. f. sp. *avenae*, and its relation to the development of mildew-resistant oat cultivars. Euphytica 15:80-86.
5. Jones, I. T. 1975. The preconditioning effect of day-length and light intensity on adult plant resistance to powdery mildew in oats. Ann. Appl. Biol. 80:301-309.
6. Jones, I. T. 1977. The effect on grain yield of adult plant resistance to mildew in oats. Ann. Appl. Biol. 86:267-277.
7. Jones, I. T. 1978. Components of adult plant resistance to powdery mildew (*Erysiphe graminis* f. sp. *avenae*) in oats. Ann. Appl. Biol. 90:233-239.
8. Jones, I. T., and Hayes, J. D. 1971. The effect of sowing date on adult plant resistance to *Erysiphe graminis avenae* in oats. Ann. Appl. Biol. 68:31-39.
9. Jones, I. T., Roderick, H. W., and Clifford, B. C. 1987. The integration of host resistance with fungicides in the control of oat powdery mildew. Ann. Appl. Biol. 110:591-602.
10. Lawes, D. A., and Hayes, J. D. 1965. The effect of mildew (*Erysiphe graminis* f. sp. *avenae*) on spring oats. Plant Pathol. 14:125-128.
11. Lawes, D. A., Valentine, J., and Jones, J. E. 1983. Oat breeding at Aberystwyth. Pages 198-217 in: Rep. Welsh Plant Breed. Stn. 1982.
12. Leath, S., and Heun, M. 1990. Identification of powdery mildew resistance genes in cultivars of soft red winter wheat. Plant Dis. 74:747-752.