Influence of Genes for Resistance to *Puccinia coronata* from *Avena sterilis* on Yield and Rust Reaction of Cultivated Oats

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ABSTRACT

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A strain of the wild oat (*Avena sterilis*) known to carry a gene for resistance to *Puccinia coronata* was crossed with a susceptible cultivated oat (*A. sativa*). Lines started as F_3 plants were carried to F_5 in bulk. Two plants of cultivated type, one resistant and one susceptible, were selected from each of 11 F_5 lines. In addition, 33 resistant and 29 susceptible plants descended from different F_3's were selected in F_5. All lines were grown in F_7 and F_8, in replicated hill-plot field trials, and plants were exposed to artificially induced epidemics of crown rust. In duplicate trials, plants were maintained free of rust by use of a fungicide to measure any loss in yield or seed weight caused by crown rust. In the rust-free plots, the mean yield of the 11 resistant sister selections was 23% below the mean yield of their susceptible counterparts, and the mean yield of all 44 resistant lines was 10% below that of all 40 susceptible lines. Both the susceptible and the resistant lines varied significantly in response to infection, as measured by reductions in yield and seed weight caused by crown rust. Presumably, the variation was caused by minor genes for tolerance or by a low level of field resistance from the *A. sterilis* parent.

Additional key words: disease resistance, genetics.

The potential value of genes from the wild oat *Avena sterilis* L. for resistance to the fungus *Puccinia coronata* Cda. var. *avenae* Fraser and Led. that causes crown rust of cultivated oats (*Avena sativa L.*) is well known (11,12). While working with such genes in the development of isogenic lines for use as components of multiline oat cultivars, Frey and Browning (4,5) observed an association between certain resistance genes and yield. The isolines had been produced by crossing well-adapted cultivars with different strains of *A. sterilis*, followed by backcrossing with concurrent selection for the different resistance genes. The isolines, which were similar in height, maturity, and general appearance, were yield-tested with and without rust. Without rust, three isolines of an early cultivar and four of a midseason-maturing cultivar had grain yields that differed significantly from those of their respective recurrent parents. Two of these yielded less, and five yielded more, than the recurrent parents. Subsequent work (1) showed that the differences in yield could be explained by a tight linkage of "yield genes" to the rust-resistance genes. In some instances the isolines also differed from the parents in obscure growth traits, such as green leaf-area duration.

The purpose of this study was to investigate an alternative method of determining possible relationships between crown rust-resistance genes and yield, and to determine whether genes for high resistance would give equal protection to plants that differed in other genetic traits.

MATERIALS AND METHODS

A crown rust resistant strain of *A. sterilis* from Algeria (CW491-4) was crossed with a highly susceptible oat cultivar, Clinton. Plants in F_2 were selected for cultivated-plant type, and a single F_3 plant from each F_2 plant saved was further selected for type. Individual F_4 and F_5 lines from the F_3 plants were increased in bulk. One resistant and one susceptible plant was selected from each of 11 F_5 lines that were segregating for resistance to *P. coronata* but seemed uniform for all other traits. In addition, single plants were selected in F_5 from each of 33 true breeding resistant, and 29 true breeding susceptible, lines descended from different F_3 plants.

Seed was increased in F_6, and all lines were grown in F_7 and F_8 in eight replications each generation in hill-plot field trials in which they were exposed to artificially induced epidemics of crown rust (3,7). Because it was assumed that the lines would vary in inherent yielding ability, duplicate trials were conducted in which the plants were maintained free of disease by use of a fungicide. The effect of disease on yield and mean seed weight in individual lines, free of any effects of inherent yield differences among lines, was expressed as an index, for which the yield or mean seed weight for a plot of diseased plants was divided by the comparable value for the corresponding plot of disease-free plants.

RESULTS

Seedling inheritance studies. *Avena sterilis* strain CW491-4 was tested in growth chambers at 20 and 27 C in the seedling stage for reaction to many races of *P. coronata*. it was resistant to all races except race 202 at both temperatures. Currently, race 202 is a very rare race that was prevalent in the early 1950s. Resistance to race 264A, which is virulent at 27 C to an extremely wide range of oat cultivars is very rare. This combination of high-temperature resistance to race 264A and susceptibility to a usually innocuous race was somewhat surprising.

Seedlings of the F_2 populations derived from crosses between the susceptible cultivar Clinton and CW491-4 were tested in the greenhouse for reaction to races 264A and 264B; the latter is representative of the forms that currently make up most of the *P. coronata* population of the USA (6). Chi-square tests for 3:1 segregation of resistant and susceptible plants provided no evidence against the hypothesis that CW491-4 carried a single dominant gene for resistance. Under the conditions of these tests, there was no indication of minor modifying genes for reaction to the fungus. Fleischmann and McKenzie (2), working in Canada with other *P. coronata* races, also found the resistance in CW491-4 to be governed by a single dominant gene. This gene was designated Pc-38 (9).

Yield in plots free of rust. When the F_7 was field-tested, conditions were favorable for both host and fungus, and coefficients of variability were low. When the F_8 was field-tested the

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next year, conditions were relatively unfavorable for both host and fungus. Data from the F_7 were adequate for objectives of the experiment; analysis of data from the F_8 corroborated the conclusions drawn from data from the F_7 generation.

Data from the F_7 for the 11 pairs of sister lines that had been selected in F_5 are shown in Table 1. With the exception of a single pair, the heading dates (10) of members of pairs were virtually identical (author's data, *not shown*). In yield, however, the resistant member of the pair; ie, the one with the gene from *A. sterilis*, was significantly lower ($P = 0.05$) than its susceptible counterpart in six of the 11 pairs. Results from F_8 were in general agreement. In F_7 the mean yield of the resistant lines was 17.4 g/hill, and that of the susceptible lines, 22.6 g/hill. The 5.2-g difference (significant beyond $P = 0.001$) represents a mean yield reduction of 23% associated with the incorporation of the *P. coronata* resistance gene from *A. sterilis*.

Among the 44 resistant and 40 susceptible lines tested in the entire experiment, which included the 11 pairs above, heading dates varied considerably. The mean heading date of the resistant lines, however, differed by only a single day from that of the susceptible lines. Thus it is unlikely that heading date, or maturity, would have had a significant effect on mean differences in yield. Also, the yield of all individual lines was resonably high; none showed the extremely low yielding ability sometimes derived from *A. sterilis* crosses. The general level of yield can be gauged from that of the parental check cultivar, Clinton, which was 24 g/hill (Table 1).

The mean yield of the 44 resistant lines was 18.4 g/hill and that of the 40 susceptible lines, 20.4 g/hill. The 2.0-g difference, representing a mean 9.8% reduction in yield associated with the resistance gene, was significant beyond $P = 0.001$.

Quantitative response to *P. coronata* infection. The 11 susceptible lines listed in Table 1 showed a considerable range in responses to infection with *P. coronata*. Yield indexes ranged from 0.220 to 0.579, and seed weight indexes ranged from 0.524 to 0.720. Some of the differences were significant, $P = 0.05$. Variation was even more pronounced when all 40 susceptible lines were considered, with yield indexes ranging from 0.146 to 0.658, and seed weight indexes from 0.516 to 0.780.

Some of this variation probably was associated with differences in maturity among the individual lines (10). The correlation coefficient between heading date and reduction in yield was −0.55, and the corresponding value for heading date and reduction in seed weight was −0.25. Also, the lines with extremely low yield-index values were all late in maturity. I interpret the magnitude of these correlation coefficients to indicate a loose relationship between maturity and response to *P. coronata*, and also that the larger share of the variation in response was not associated with maturity. Thus, it seems highly likely that there is some real variation in rust response among these lines aside from that associated with differences in maturity. That variation could be explained by the presence of minor or modifying genes for tolerance or for a low degree of field resistance from the *A. sterilis* parent that were expressed in the field, but not in the greenhouse seedling tests. Interaction of minor genes from both parents also may have been involved.

TABLE 1. Yields without rust, and disease responses to crown rust infection, of 11 pairs of resistant and susceptible sister selections from a cross between *Avena sativa* and *A. sterilis* and of a highly susceptible *A. sativa* cultivar, Clinton

Pair no.	Yield without rust (g/hill)		Disease response[a]			
			Yield index		Seed wt. index	
	Res.	Susc.	Res.	Susc.	Res.	Susc.
1	21	22	0.865	0.392	0.872	0.642
2	14	23	0.869	0.477	0.913	0.675
3	16	21	1.040	0.272	0.947	0.581
4	19	25	0.939	0.488	0.907	0.598
5	17	29	0.957	0.262	0.945	0.592
6	15	15	1.026	0.579	0.912	0.616
7	10	27	0.778	0.290	0.841	0.542
8	17	20	1.103	0.372	0.940	0.605
9	19	21	0.759	0.535	0.947	0.674
10	17	22	0.992	0.541	0.923	0.720
11	26	23	0.841	0.220	0.879	0.524
Mean	17.4	22.6	0.922	0.402	0.912	0.615
LSD 0.05	3.9		0.148		0.072	
LSD 0.01	5.1		0.195		0.094	
Clinton	...	24	...	0.357	...	0.554

[a]Index = yield or seed weight for a plot of diseased plants divided by the comparable value for the corresponding plot of disease-free plants.

Variation in response of yield and seed weight to infection also was observed among the resistant members of the pairs (Table 1). That variation was unexpected; it had been assumed that the resistance gene from *A. sterilis* would give complete protection and thereby preclude the expression of any other genes for resistance. The possibility of differences in disease response being associated with differences in maturity was considered, but the correlation coefficients for heading data versus both yield index and seed weight index were nonsignificant. Thus, it is likely that real differences in response to infection did exist among these resistant lines, and that they were conditioned by minor genes from the *A. sterilis* parent or from an interaction of minor additive genes from both parents.

The possibility of a relationship between these minor genes and the inherent yielding ability of the lines also was investigated. Examination of the data showed no clear indication of such a relationship (Table 1). This observation was substantiated by data from all 40 lines that lacked the major gene; nonsignificant correlation coefficients of 0.07 and −0.16 were calculated for yield in plots without rust versus yield index, and for yield in plots without rust versus seed weight index, respectively. The corresponding correlation coefficients for the 44 lines carrying the major gene were −0.35 and 0.01, respectively.

DISCUSSION

Frey and Browning (5) detected yield differences associated with different crown rust resistance genes by comparing yields of isolines of oats that differed by the presence or absence of single resistance genes. Discovery of these yield differences was, of course, incidental to developing the isolines as components of multiline cultivars. The cost of deliberately developing isolines simply to determine such differences would ordinarily be considered prohibitive. The present study indicated that similar information on crown rust resistance genes may be obtained by an alternative approach. It must be admitted, however, that this approach, while perhaps less laborious than that of Frey and Browining (5), still requires considerable effort and expense.

The question of whether the resistance gene from the strain of *A. sterilis* used in this study was consistently associated with either increased or decreased yield when it was transferred to cultivated oats was not answered. Data from the pairs of sister lines selected in F_5 showed that a generalization could not be made. For most of the pairs, the line with the resistance gene yielded less than its susceptible counterpart. Members of some pairs, however, yielded alike, and it seems quite possible that a larger sample would have revealed sister lines in which the resistant member outyielded the susceptible member.

The variation in response of yield and seed weight to infection (free of inherent differences in these traits, as explained in Materials and Methods) among the susceptible members of the pairs was not surprising because it is known that strains of *A. sterilis* commonly carry minor genes for tolerance, resistance, or both (8). The variation may have been due to random distribution of such genes in the absence of selection for them. This assumption holds for the resistant members of the pairs, but it is surprising that minor genes could be expressed in the presence of the major gene for resistance. Under commercial field conditions, or even nursery conditions with only a moderate amount of rust, the major gene no doubt would have prevented measurable damage from rust. But artificially induced infection in these experiments was severe enough to partly overcome the influence of the major gene, and thus allow expression of the minor genes.

LITERATURE CITED

1. BRINKMAN, M. A., and K. J. FREY. 1977. Growth analysis of isoline-recurrent parent grain yield differences in oats. Crop Sci. 17:426-430.
2. FLEISCHMANN, G., and R. I. H. McKENZIE. 1968. Inheritance of crown rust resistance in Avena sterilis. Crop Sci. 8:710-713.
3. FREY, K. J. 1965. The utility of hill plots in oat research. Euphytica 14:196-208.
4. FREY, K. J. 1972. Stability indexes for isolines of oats (Avena sativa L.). Crop Sci. 12:809-812.
5. FREY, K. J., and J. A. BROWNING. 1971. Association between genetic factors for crown rust resistance and yield in oats. Crop Sci. 11:757-760.
6. MICHEL, L. J., and M. D. SIMONS. 1977. Aggressiveness and virulence of Puccinia coronata avenae isolates, 1971-1975. Plant Dis. Rep. 61:621-625.
7. SIMONS, M. D. 1966. Relative tolerance of oat varieties to the crown rust fungus. Phytopathology 56:36-40.
8. SIMONS, M.D. 1972. Crown rust tolerance of Avena sativa-type oats derived from wild Avena sterilis. Phytopathology 62:1444-1446.
9. SIMONS, M. D., J. W. MARTENS, R. I. H. McKENZIE, I. NISHIYAMA, K. SADANAGA, J. SEBESTA, and H. THOMAS. 1978. Oats: A standardized system of nomenclature for genes and chromosomes and catalog of genes governing characters. U.S. Dep. Agric. Handb. 509. 40 p.
10. SIMONS, M. D., and L. J. MICHEL. 1968. Oat maturity and crown rest response. Crop Sci. 8:254-256.
11. WAHL, I. 1970. Prevalence and geographic distribution of resistance to crown rust in Avena sterilis. Phytopathology 60:746-749.
12. ZILLINSKY, F. J., and H. C. MURPHY. 1967. Wild oat species as sources of disease resistance for the improvement of cultivated oats. Plant Dis. Rep. 51:391-395.