Association of Host Cytoplasm with Reaction to *Puccinia coronata* in Progeny of Crosses Between Wild and Cultivated Oats

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ABSTRACT

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Five strains of *Avena sterilis* were crossed reciprocally with two *A. sativa* cultivars to give 20 hybrids. The 20 hybrids were backcrossed twice to their respective *A. sativa* parents, and 20 lines derived from each original cross were field-tested for quantitatively expressed resistance to artificially initiated epidemics of *Puccinia coronata* in the F_5 and F_6 generations. Controls were maintained free of rust with a fungicide, and resistance data were recorded as yield and seed-weight indexes obtained by dividing values in the diseased test plots by corresponding values in the control plots. Although the absolute differences were very small, populations derived from the cultivar CI 9170 showed significantly higher mean indexes in favor of the cultivated cytoplasm. Populations from the cultivar Otee showed a similar but nonsignificant mean trend. In a few individual crosses, *A. sterilis* cytoplasm was superior. Certain individual lines in each of the 20 populations had significantly greater resistance than their respective cultivated parents.

The concept of the host cytoplasm genome affecting reaction to plant pathogens is not a new one. Before 1900, Eriksson (5) postulated that susceptibility of plants to several fungal diseases could be maternally inherited. This concept, however, was nearly dormant until 1961, when Mercado and Lantican (12) reported that maize (*Zea mays*) with Texas-type cytoplasmic male sterility was susceptible to *Helminthosporium maydis*. This association was confirmed in the Philippines by Villareal and Lantican (23). In 1970, *H. maydis* was responsible for one of the most destructive plant disease epidemics ever recorded (1,10,20). This susceptibility of maize in the United States was due to genes carried in the Texas male sterile cytoplasm, which was present in most maize grown in the United States in 1970.

Texas male sterile cytoplasm also conditions susceptibility to Phyllosticta leaf spot of maize (3,19), but not all male sterile lines are equally susceptible (2). Other types of male sterility in maize are not associated with susceptibility to the disease (15).

Maternal effects on plant disease other than those shown to be associated with cytoplasmically inherited male sterility have also been reported for maize. Reaction to seedling blight caused by *Fusarium moniliforme* was strongly influenced by the maternal parent (11). Fleming (6) demonstrated a significant male cytoplasmic effect on reaction of maize to seedling disease caused by *Fusarium* sp. in two of four comparisons. According to Singh (22), an interaction of nuclear genotype and cytoplasm of maize conditioned reaction to smut.

Cytoplasmic inheritance of reaction to disease has been found in other plant species also. Degree of susceptibility of strawberries (*Fragaria*) to mildew differed between reciprocal crosses, and the cytoplasmic effects persisted in the F_2 and in backcrosses (8). Symptoms of potato virus X in *Capsicum* were controlled by cytoplasm in interspecific hybrids (13). Rath and Padmanabhan (16), who studied F_2 populations from certain reciprocal crosses among seven rice (*Oryza sativa*) cultivars, found several cases of statistically significant maternal influence on rice blast (*Pyricularia oryzae*) reaction and its components, lesion number and type. Reaction to bacterial leaf blight of rice may also be under maternal control (14).

Sanchez-Monge et al (18) developed alloplasmic forms of 63 hexaploid (*Triticum aestivum*) and 54 tetraploid (*T. durum*) wheats and two triticales with cytoplasms of three species of *Aegilops*. In eight cases, alloplasmy induced resistance to stem rust (*Puccinia graminis*) in varieties that were susceptible with their own cytoplasms. In 13, alloplasmy induced susceptibility in varieties that were resistant with their own cytoplasms. In all others, the influence of alien cytoplasm was nil. Cytoplasmic control of reaction to wheat leaf rust (*P. recondita*) (26), powdery mildew (*Erysiphe graminis*) (24), and ergot (*Claviceps purpurea*) (4) has been reported also.

As a general assessment of the importance of cytoplasmic influence on

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plant disease, Hooker (9) averred that cytoplasmic effects on disease reaction of plants are uncommon.

The wild oat species *Avena sterilis* L. is a valuable source of resistance to crown rust (*P. coronata* Cda.) of cultivated oats (*A. sativa* L.) (25). This study was undertaken to determine whether this resistance might, in part, be conditioned by the *A. sterilis* cytoplasms.

MATERIALS AND METHODS

The identification of cytoplasmic genes is relatively straightforward, with the initial criterion being the occurrence of differences in reactions of reciprocal crosses of a mating. For our study, each of five strains of *A. sterilis* was crossed reciprocally with each of two *A. sativa* cultivars (Table 1) to give 20 hybrids. The 20 hybrids were backcrossed twice to their respective *A. sativa* parents, and the BC_2 F_2 seeds were sown in the field. Twenty random nonshattering BC_2 F_2 plants were harvested from each reciprocal of each mating (17). The bulked seed from a single F_2 plant was used to establish a line. This gave 20 lines in each of 20 populations for a total of 400 lines to be tested. Seeds of the 400 lines were increased in the BC_2 F_3 and BC_2 F_4 generations. The BC_2 F_5 and BC_2 F_6 generations were field-tested in 1981 and 1982, respectively, in central Iowa in hill plots (7) with hills spaced 0.3 m apart in perpendicular directions and sown with 30 seeds per hill. The hills were planted in a randomized block design with eight replicates. Epidemics of *P. coronata* were initiated by using a hypodermic needle to inject an aqueous suspension of urediniospores of common races of the fungus into the culm of one plant per hill as soon as the culms had started to elongate. Trickle irrigation was applied as necessary to prevent drought damage and to ensure disease spread.

We anticipated that these lines would vary significantly in inherent yielding ability, seed weight, and other quantitative traits. Therefore, duplicate plantings, separated by a 2-m alley from the diseased plots, were maintained disease-free by a weekly application of maneb fungicide at 1 kg a.i./ha. Resistance data, expressed as indexes of yield and seed weight, were obtained by dividing the value from a diseased plot by the line mean from the eight rustfree replicates. Seed weight was estimated from samples of 200 seeds.

RESULTS

Performance in rustfree plots. Both 1980 and 1981 were satisfactory years for oat production in central Iowa. Yields in 1980, however, averaged about 15% higher than in 1981. There was significant ($P = 0.05$) variation among the 20 populations shown in Table 1 for both yield and seed weight in the rustfree environment in both years. Variation for these traits among lines within populations was also significant ($P = 0.05$) in both years.

Response to infection. Crown rust was severe in the rusted plots in 1980 and 1981, whereas occurrence of other diseases was negligible. All lines were visually rated susceptible to crown rust. Mean reductions in yield and seed weight attributable to disease were somewhat greater in 1980 than in 1981 (Table 1).

The primary objective of this investi-

Table 1. Yield and seed-weight indexes[a] of populations composed of lines derived from reciprocal crosses of *Avena sativa* cultivars Otee and CI 9170 with five strains of *A. sterilis*

Population from cross	Yield index						Seed-weight index					
	1980			1981			1980			1981		
	High[b]	Low[b]	$\bar{x}$	High	Low	$\bar{x}$	High	Low	$\bar{x}$	High	Low	$\bar{x}$
						***A. sterilis* cytoplasm**						
PI 324725 × Otee	0.71	0.34	0.50	0.85	0.42	0.64	0.74	0.56	0.66	0.77	0.55	0.67
PI 217512 × Otee	0.61	0.28	0.44	0.81	0.38	0.62	0.74	0.53	0.62	0.74	0.58	0.67
PI 317982 × Otee	0.96	0.46	0.61	1.10	0.57	0.74	0.81	0.62	0.70	0.82	0.66	0.71
PI 324819 × Otee	0.63	0.28	0.46	0.84	0.42	0.66	0.71	0.52	0.63	0.77	0.52	0.66
PI 317757 × Otee	0.80	0.35	0.55	1.00	0.50	0.72	0.75	0.55	0.67	0.78	0.58	0.69
$\bar{x}$ *A. sterilis* cytoplasm			0.51			0.68			0.66			0.68
						***A. sativa* cytoplasm**						
Otee × PI 324725	0.78	0.33	0.54	0.84	0.50	0.65	0.85	0.59	0.67	0.77	0.60	0.69
Otee × PI 217512	0.59	0.26	0.42	0.89	0.36	0.59	0.81	0.55	0.62	0.79	0.57	0.67
Otee × PI 317982	0.91	0.39	0.56	0.88	0.46	0.70	0.80	0.59	0.68	0.77	0.62	0.71
Otee × PI 324819	0.86	0.40	0.50	0.83	0.57	0.68	0.73	0.59	0.66	0.74	0.64	0.69
Otee × PI 317757	1.30	0.50	0.71	1.11	0.58	0.79	0.83	0.61	0.74	0.81	0.66	0.74
$\bar{x}$ Otee cytoplasm			0.55			0.68			0.67			0.70
						***A. sterilis* cytoplasm**						
PI 324725 × CI 9170	0.68	0.35	0.50	0.93	0.53	0.69	0.73	0.58	0.65	0.81	0.64	0.72
PI 217512 × CI 9170	0.73	0.23	0.51	1.00	0.41	0.66	0.74	0.48	0.64	0.76	0.59	0.68
PI 317982 × CI 9170	0.98	0.26	0.60	1.20	0.37	0.77	0.84	0.57	0.71	0.88	0.58	0.74
PI 324819 × CI 9170	0.91	0.42	0.56	1.02	0.53	0.76	0.76	0.60	0.66	0.84	0.60	0.71
PI 317757 × CI 9170	0.75	0.45	0.55	1.19	0.56	0.74	0.84	0.58	0.67	0.79	0.67	0.72
$\bar{x}$ *A. sterilis* cytoplasm			0.54			0.72			0.67			0.71
						***A. sativa* cytoplasm**						
CI 9170 × PI 324725	1.06	0.36	0.58	1.14	0.65	0.79	0.84	0.62	0.71	0.83	0.69	0.76
CI 9170 × PI 217512	0.91	0.34	0.56	0.98	0.45	0.73	0.84	0.60	0.69	0.82	0.65	0.72
CI 9170 × PI 317982	1.07	0.41	0.64	1.11	0.63	0.81	0.85	0.63	0.74	0.86	0.69	0.77
CI 9170 × PI 324819	0.80	0.36	0.50	0.98	0.48	0.70	0.74	0.59	0.66	0.79	0.63	0.72
CI 9170 × PI 317757	1.18	0.49	0.63	0.99	0.58	0.77	0.82	0.62	0.71	0.81	0.63	0.74
$\bar{x}$ CI 9170 cytoplasm			0.58			0.76			0.70			0.73
$\bar{x}$ Of original Otee parent			0.60			0.73			0.65			0.68
$\bar{x}$ Of original CI 9170 parent			0.51			0.74			0.64			0.71
LSD (0.05) For line vs. original parent			0.23			0.21			0.07			0.07
LSD (0.05) For population vs. population			0.09			0.08			0.03			0.03
LSD (0.05) For *A. sterilis* cytoplasm vs. *A. sativa* cytoplasm for each *A. sativa* parent			0.04			0.04			0.02			0.01

[a] Indexes are a measure of relative host damage caused by *P. coronata*, obtained by dividing the value from a diseased plot by the line mean from eight rustfree replicates. Seed weight was estimated from samples of 200 seeds.

[b] High and low refer to the means of the highest and lowest performing lines, respectively, of the 20 individual lines making up each population.

gation was to determine whether there was an association of host cytoplasm with reaction to *P. coronata*. Mean values for matings involving the cultivar Otee (Table 1) showed no striking differences in yield and seed-weight resistance indexes between Otee and *A. sterilis* cytoplasms. If a trend existed, it was for the Otee cytoplasm to give higher resistance indexes.

In the CI 9170 matings, higher resistance indexes were associated with the *A. sativa* cytoplasm. In fact, mean yield and seed-weight indexes of populations with CI 9170 cytoplasm were significantly greater than those with *A. sterilis* cytoplasm in both years.

A. sterilis cytoplasm, although generally inferior to *A. sativa* cytoplasm for resistance indexes, did show several cases of superiority (Table 1). For example, in the mating involving Otee and PI 317982, the *A. sterilis* cytoplasm was consistently superior to the Otee cytoplasm. The *A. sterilis* cytoplasm had a similar advantage in the mating involving CI 9170 and PI 324819.

The original *A. sterilis* parents used in this study were not tested for reaction to *P. coronata*, but seedling tests of the derived lines showed no evidence of seedling resistance. Previous studies (21), however, have shown that it is likely that all the *A. sterilis* parents carried at least some degree of field resistance or tolerance. As shown by the ranges for yield and seed-weight indexes, all 20 populations contained considerable variation for resistance when measured by either trait in both years (Table 1). This was reflected by the significant mean squares for lines within populations.

The seed-weight index of the highest individual line in each population significantly ($P = 0.05$) exceeded the performance of its respective cultivated parent in nearly every population. The same general pattern was shown by the yield index data, except fewer of the differences were significant, probably because of the high experimental error associated with yield index. In general, the poorest performing line of each population was significantly below its respective cultivated parent in seed-weight index. This was corroborated by the yield indexes of the lowest performing lines, but here again, the conclusion was clouded by high experimental error. There was no discernible trend for any parents, wild or cultivated, to consistently transmit greater or lesser resistance than any of the others.

DISCUSSION

This study was undertaken with the hope that potentially useful resistance to *P. coronata* would be found to be associated with cytoplasm of strains of wild *A. sterilis*. The data suggested that such effects exist for *A. sterilis* and provided reasonably convincing evidence of resistance being associated with the cytoplasm of the two cultivars of *A. sativa* that were used. Because the amount of germ plasm sampled (two cultivars and five wild strains) was only a minuscule fraction of the total available, it is quite possible that investigation of larger numbers of either cultivated or wild strains would reveal some in which the degree of resistance associated with the cytoplasm would be much higher. On the other hand, the small absolute size of these differences may indicate that the cytoplasm plays a negligible role in resistance to *P. coronata*.

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