Reaction of Sweet Corn Germ Plasm to Common Rust and an Evaluation of *Rp* Resistance in Illinois

JERALD K. PATAKY, Assistant Professor, Department of Plant Pathology, University of Illinois, Urbana 61801

ABSTRACT

Pataky, J. K. 1987. Reaction of sweet corn germ plasm to common rust and an evaluation of *Rp* resistance in Illinois. Plant Disease 71:824-828.

Several sources of rust resistance were identified in sweet corn (*Zea mays*) germ plasm. The Rp_1^d gene probably is carried in yellow (IL125b and IL791a) and white (IL18c) sweet corn inbreds. An *Rp* type of resistance that may be conditioned by Rp_1^d was observed on IL763a. Other rust-resistant reactions (type 1 and type 2) were identified on several lines. A purple pigmentation surrounding a resistant reaction was observed on IL677a and several lines derived from IL677a. Chlorotic infection types were observed on several other lines for which Country Gentleman was an ancestor. The Rp_1^d, Rp_1^e, Rp_1^f, Rp_1^g, Rp_1^i, and Rp_3^c genes conditioned type 0; resistant reactions when inbreds possessing those genes were infected with a mixture of *Puccinia sorghi* biotypes collected in Illinois in 1983 and 1985. In greenhouse trials, these genes were effective against a mixture of *P. sorghi* biotypes collected from California, South Dakota, Nebraska, Minnesota, Wisconsin, Ontario, Illinois, Ohio, New York, and Georgia. The Rp_1^k gene conditioned a type 1 reaction to all biotypes in field and greenhouse trials.

Common rust of corn (*Zea mays* L.) caused by *Puccinia sorghi* Schwein. can be economically damaging to sweet corn in environments favorable for rust development (3,15). Although specific and partial (i.e., generalized) rust resistance have been identified (2,4,6,9–12,18,21), many of the most popular sweet corn hybrids are susceptible (4,15), particularly hybrids with the high sugar endosperm mutation sh_2 (16). Consequently, commercial sweet corn breeders are developing rust-resistant genotypes by backcrossing *Rp* resistance genes into elite inbreds or selecting for high levels of partial resistance (22).

Specific rust resistance in corn is controlled by single genes that occur at six or more loci (7,8). At least two of these loci are a complex of very closely linked genes or pseudoalleles (19,20). More than 100 sources of specific resistance have been identified (2,4,6,9, 12,21). Infection types range from type 0; (small chlorotic flecks, no uredinia) to type 2 (small uredinia surrounded by chlorotic host tissue) (8).

Rp resistance genes from several sources were incorporated into the dent corn inbred line R168 by A. L. Hooker through backcrossing procedures (7). Eight of the dominant *Rp* genes in R168 conditioned resistance when inoculated and naturally infected with several *P. sorghi* biotypes in 1966 (7). A gene at one of these loci, Rp_1^d, conditioned a type 0; reaction to all of the known biotypes of *P. sorghi* in the continental United States. Since then, a virulent biotype was reported in 1984 (1). In Hawaii, races of *P. sorghi* that are virulent on Rp_1^d have been observed for several years (J. L. Brewbaker, *personal communication*).

Identification of rust resistance in sweet corn germ plasm would enhance breeding efforts for control of this disease. Because biotypes of *P. sorghi* exist that are virulent to most of the *Rp* genes (8), identification of sources of partial rust resistance in sweet corn germ plasm would be useful in conjunction with population improvement programs. Identification of sweet corn sources of specific rust resistance would be of value if resistance were expressed to the most common biotypes of *P. sorghi*. In the absence of resistance in adapted sweet corn germ plasm, resistance could be obtained from dent corn and exotic sources. This paper reports on an evaluation of sweet corn germ plasm for specific and partial rust resistance and an evaluation of the effectiveness of *Rp* genes to a collection of *P. sorghi* biotypes.

MATERIALS AND METHODS

Sweet corn germ plasm trials. Fifty sweet corn lines were evaluated in 1984, 1985, and 1986. An additional 71 lines were evaluated in 1986. The 1984 trial consisted of five replicates of three three-plant hill plots for each line arranged in a randomized complete block design. Hill plots were spaced about 22 cm within rows and 76 cm between rows. The 1985 trial consisted of three replicates of single rows of each line with about 20 plants per row. Rows were about 4.6 m long and 76 cm apart. The 1986 trial consisted of two replicates of single rows of each line. Trials were planted 1 June 1984, 7 May 1985, and 9 May 1986.

Plants were inoculated with suspensions of *P. sorghi* urediniospores (0.5 g of urediniospores suspended in 6 L of water and 1 ml of Tween 80) on 21 June 1984, 31 May and 3 June 1985, and 11 June 1986. Inoculum suspensions were sprayed directly into plant whorls with hand-held sprayers. Urediniospores had been collected from more than 20 locations in Illinois in 1983 and 1985 and increased in the greenhouse and field on several sweet and dent corn genotypes. Urediniospores were stored in a desiccator at −20 C for 3–36 mo.

Rust severity was rated as the relative percentage of the total leaf area covered by uredinia on a modified Peterson scale (17). Thus, when 37% of the total leaf area was covered by uredinia, rust severity was 100%. Infection types were rated on a scale of 0;–4 (8), where 0; = small chlorotic flecks, no uredinia; 1 = small uredinia surrounded by necrotic tissue; 2 = small uredinia surrounded by chlorotic tissue; 3 = medium-sized uredinia with or without chlorosis and necrosis; and 4 = large uredinia. Ratings were made 17 July and 8 August 1984, 9 July and 7 August 1985, and 7 and 23 July 1986.

Rust severity was analyzed by analysis of variance ($P = 0.05$). Lines were compared by Waller-Duncan Bayesian least significant difference values (BLSD) with $k = 100$. Lines also were grouped by infection type based on number of replicates for which the host-pathogen interaction was rated resistant (type 0;, 1, or 2).

Specific resistance trials. Dominant genes for rust resistance from 54 sources that were backcrossed into the susceptible dent corn inbred R168 by A. L. Hooker were evaluated (7). Most of the R168 lines were in at least the 10th backcross generation. These lines were grown in the rust nursery in 1984 and 1986 to identify *Rp* genes effective against biotypes of *P. sorghi* collected in Illinois. Both trials consisted of two replicates of single rows of about 20 plants of each genotype. Rows were about 4.6 m long and 76 cm apart.

In 1984, all plants were inoculated on 21 June as described previously. In 1986, all plants were naturally infected by urediniospores, which were disseminated primarily from the rust nursery located upwind from the specific resistance trial and had been inoculated as described previously. Rust severity and infection type were rated on 25 July 1984 and 7 July and 27 August 1986 as described.

Research supported by the Agricultural Experiment Station, University of Illinois, Urbana.

Accepted for publication 7 May 1987 (submitted for electronic processing).

In the greenhouse, the R168 inbreds carrying *Rp* genes were evaluated for reaction to a mixture of *P. sorghi* biotypes collected in 1986 from various locations in North America. These included isolates from Orange County, CA; Brookings, SD; York, NE; St. Paul, MN; Sun Prairie, WI; Ontario, Canada; Bloomington, IL; Forest City, IL; Painter Creek, OH; Kings Ferry, NY; Freeville, NY; and Athens, GA. Isolates from each location were increased in the greenhouse on the sweet corn inbred IL110g and the hybrid Sweet Sue. Urediniospores were collected and stored in a desiccator at −20 C. An equal mixture (w/w) of each isolate was used to prepare urediniospore suspensions (1 mg of urediniospores in 50 ml of water and one drop of Tween 80). Ten plants of each genotype were inoculated per replicate at the three- to four-leaf stage with an atomizer. Plants were placed in a mist chamber for 6 hr after inoculations, then moved to a greenhouse bench. Plants on the bench were subirrigated to prevent secondary infections. Reactions were evaluated 10 days after inoculation. There were two replicates in each of two trials.

RESULTS

Sweet corn germ plasm trials. Early-season rust severity ranged from 0 to 60, 0 to 50, and 0 to 30% and averaged 21, 21, and 13% in 1984, 1985, and 1986, respectively. Late-season rust severity ranged from 0 to 5, 0 to 70, and 0 to 60% and averaged 43, 36, and 25% in 1984, 1985, and 1986, respectively. In general, genotype responses at early- and late-season rust evaluations were similar each year (r = 0.77, 0.68, and 0.79 for 1984, 1985, and 1986, respectively).

Four Illinois lines, IL18c, IL125b, IL763a, and IL791a, displayed a type 0; resistant reaction to *P. sorghi* (Tables 1 and 2; Fig. 1A). No uredinia were observed on these genotypes. Cuzco, a source of the Rp_1^d gene, was a common ancestor in the pedigrees of IL18c, IL125b, and IL791a, and therefore, these genotypes could all possess the Rp_1^d gene. IL763a was a selection from an accession of HS × Comp If obtained from J. L. Brewbaker, University of Hawaii. IL763a was crossed with R168 possessing effective *Rp* genes (Rp_1^d, Rp_1^e, Rp_1^f, Rp_1^g, Rp_1^i, and Rp_3^c). Reactions of segregating generations will determine if the resistant reaction of IL763a was conditioned by an identified *Rp* gene.

Several sweet corn lines displayed resistant reactions typified by smaller uredinia with necrotic, chlorotic, and/or purple borders (Tables 1 and 2, Fig. 1B–D). In general, rust severity was reduced on genotypes that displayed these reaction types compared with genotypes that displayed susceptible type 4 reactions. However, rust severity was high on some genotypes that displayed a resistant reaction type (i.e., I2123, IL776c, etc.).

Resistant reactions often were associated with common ancestors in the pedigrees of many genotypes. The open-pollinated sweet corn Country Gentleman, which was previously reported to be rust-resistant (13), is a common ancestor of a group of lines (IL21f, IL27a, IL31a, IL47a, IL200e, IL676a, and IL693a) that primarily displayed a chlorotic reaction (Fig. 1C). IL451b is a common ancestor to another group of lines (IL709a, IL710a, IL711a, and IL723a) that displayed a chlorotic reaction; however, these genotypes also may have obtained the chlorotic reaction from Country Gentleman because the pedigree of IL451b includes IL27a and IL31a.

IL677a is a common ancestor for a group of lines on which diverse and interesting resistant reactions were observed. IL677a, a selection from (IL44b × BOV1035) × IL442a, was relatively rust-resistant with a reaction type in which uredinia were very small and were bordered by necrotic and/or chlorotic tissue and by purple-pigmented host tissue (Fig. 1D). Genotypes with IL677a as an ancestor (IL744a, IL751a,

Table 1. Rust severity and infection type on sweet corn lines evaluated for reaction to *Puccinia sorghi* in 1984, 1985, and 1986

Genotype	Rust severity (%)			Infection type
	1984	1985	1986	
AA12	51[a]	50	45	4[b]
A10579	31	23	...	...
B5870	38	34	...	...
B6129-2	29 C[c]	21 C	...	...
B6129-3	...	21	...	...
B6129-4	...	17 C	...	...
IL14h	36	36	28	3–4
ILT32	32	32	25	4
IL44b	33	28	23	4
ILT55	33	28	35	4
IL110g	71	60	50	4
IL125b	0	0	0	0;
IL304a	32	31	25 C	2–3
IL442a	23 CN	11 CN	15 CN	1–3
IL454a	21 NP	25 NP	22 CNP	1–3
IL459a	28	26	25	4
IL676a	26 CN	31 CN	25 C	1–4
IL677a	25 CNP	23 CNP	18 NP	1–2
IL678a	25 C	17 C	28 C	2–4
IL685d	17	...	18 C	2–3
IL729a	29	29	23	4
IL731a	24 CNP	22 CNP	16 CNP	1–2
IL747b	24	...	27 C	2–4
IL757b	20	20	28 C	2–4
IL758b	32	26	26	4
IL765a	25	23	18 C	2–3
IL767a	21 C	15 C	20 C	2–3
IL768a	24 CN	19 CN	25 NP	1–3
IL769a	32 NP	28 NP	20 NP	1–3
IL776a	38 CN	32 CN	28 P	1–2
IL781a	26 CN	28 CN	28	2–4
IL783a	54	35	43	4
IL784a	49	42	55	4
IL788a	43	50	38	4
IL789a	49	48	40	4
IL789b	45	33	35	4
IL790a	31 N	26 N	25 NP	1–3
I453	56	45	38	4
I2123	42 C	45 C	23 CN	2–3
I2256b	38	28	40	4
I5125	47	38	25	4
M6008	28	35	...	...
M6009	20	22	...	...
M6011	31	38	...	...
M6043	30	33	...	...
M6161	31	...	...	...
P51	39	43	40	4
R853	34	30	28	3–4
S5512	31	31	...	...
S6046	27 N	23 N	...	...
BLSD (k = 100)	4.7	5.5	7.0	...

[a] Rust severity = relative percentage of the total leaf area covered by uredinia based on the modifiefd Peterson scale (17).
[b] Infection type rated from 0; to 4 (8).
[c] Letters following severity values indicate chlorosis (C), necrosis (N), and/or purple pigmentation (P) associated with uredinia.

Table 2. Rust severity and infection type on sweet corn lines evaluated for reaction to *Puccinia sorghi* in 1986

Genotype	Severity (%)	Infection type	Genotype	Severity (%)	Infection type
C13	60[a]	4[b]	IL733a	28	4
C2A	17 CN[c]	1–4	IL740a	24	4
DSU146	25	4	IL744a	17 NP	1–3
IL5m	30	4	IL748a	15 CP	1–2
IL13c	35	3–4	IL751a	35 C	2–3
IL18b	43	4	IL752a	33	4
IL18c	0	0;	IL753a	28	4
IL21f	25 C	2–4	IL759a	35	3–4
IL27a	11 C	2–3	IL761a	25 NP	1–3
IL31a	16 C	2–3	IL763a	0	0;
IL47a	14 C	2–3	IL764a	15	4
IL103a	19 C	2–4	IL766a	16 CNP	1–3
IL11d	23	3–4	IL770a	23	4
IL112t	30	3–4	IL771a	25	4
IL126b	25	4	IL772a	25 C	2–3
IL190a	25	4	IL773a	30 CN	1–4
IL200e	28 C	2–3	IL774c	23 CN	1–3
IL201j	25	4	IL775a	40	3–4
IL318a	30	4	IL776c	20	4
IL323a	20 CNP	1–3	IL777a	19 CN	1–3
IL352a	15	3	IL778c	33 CP	2–3
IL370a	19	4	IL779a	19 CP	2–3
IL374b	38	4	IL780a	15 CP	2–3
IL552a	12	1–3	IL782a	38	4
IL567a	35	4	IL785a	25	4
IL671a	43	4	IL786a	20 C	3–4
IL685a	25	4	IL791a	0	0;
IL693a	17 NP	1–2	L1	40	4
IL707a	18	4	M6222a	20 CN	1–3
IL709a	25 C	2–4	M6222b	14	4
IL710a	13 C	2–3	M6222c	20	3
IL711a	25 C	2–3	M6222d	28	4
IL713a	50	4	M6222e	33	4
IL715a	20	4	M6223a	21	4
IL723a	23 C	2–3	IP39	35	3–4
IL724a	10 NP	1–3			

[a] Rust severity = relative percentage of the total leaf area covered by uredinia based on the modified Peterson scale (17).
[b] Infection type rated from 0; to 4 (8).
[c] Letters following severity values indicate chlorosis (C), necrosis (N), and/or purple pigmentation (P) associated with uredinia.

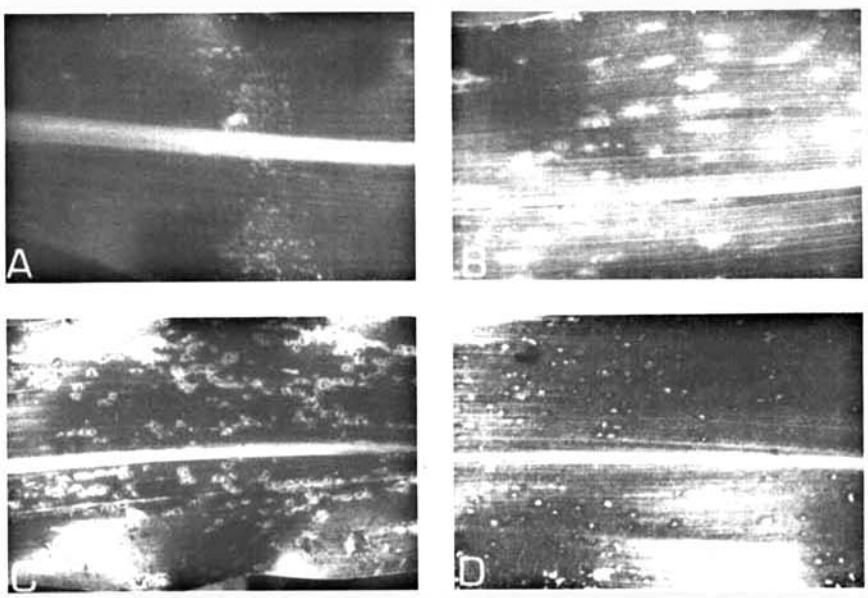

Fig. 1. Rust-resistant reactions observed in sweet corn germ plasm: **(A)** chlorotic flecks without uredinia conditioned by the Rp_1^d gene, **(B)** small uredinia surrounded by necrotic tissue, **(C)** small to medium-sized uredinia surrounded by chlorotic tissue, and **(D)** small uredinia surrounded by necrotic or chlorotic tissue and bordered by purple pigmentation.

IL766a, IL767a, IL768a, IL769a, IL772a, IL773a, IL774a, IL776a, IL777a, IL778a, IL779a, IL780a, IL781a, and IL790a) displayed chlorotic, necrotic, and/or purple-pigmentation reactions although none were as distinct and resistant as the reaction of IL677a. Several genotypes with IL677a as an ancestor displayed fully susceptible type 4 reactions (e.g., IL788 and IL789b). Sister lines derived from Il677a sometimes displayed different reactions (e.g., IL776a and IL776c). IL442a, a selection from [(W23 × IL104q) × IL104q) × IL104g] and one of the parents of IL677a, also displayed a chlorotic, necrotic reaction associated with low rust severity (Table 1).

The most susceptible genotypes that may serve as good susceptible testers were AA12, IL110g, IL783a, IL784a, IL789a, I453, and I2123. I2123 was severely rusted in spite of displaying a chlorotic reaction type. IL783a and IL784a are selections from sh_2 hybrids that are extremely susceptible to rust.

Specific resistance trials. Six *Rp* genes (Rp_1^d, Rp_1^e, Rp_1^f, Rp_1^g, Rp_1^i, and Rp_3^c) conditioned a type 0; reaction and were effective in 1984 and 1986 field trials (Table 3). An additional source of *Rp* resistance, M165sel, for which the locus and allele have not been identified, also conditioned a type 0; reaction. The Rp_1^k gene conditioned a type 1 reaction and was effective in both years. The Rp_1^a and Rp_1^b genes conditioned type 1 reactions in 1986 but did not appear to be effective in 1984 based on rust severity. The Rp_1^m gene appeared to be effective based on rust severity in 1984 but displayed a type 4 reaction even though severity was low in 1986. Possibly, virulent races occurred in 1986. In the greenhouse trials, inbreds carrying Rp_1^a, Rp_1^d, Rp_1^e, Rp_1^f, Rp_1^g, Rp_1^i, Rp_1^k, and Rp_3^c displayed resistant reactions.

DISCUSSION

Several sources of rust resistance are available in adapted sweet corn germ plasm. The Rp_1^d gene is available in yellow (IL125b and IL791a) and white inbreds. Also, an *Rp* type of resistance, which is probably Rp_1^d, has been incorporated into at least three commercial sweet corn hybrids, aRRestor, Excellency, and Prevailer (16). Other types of useful rust-resistant reactions also are available from Country Gentleman, IL677a, IL442a, or from horticulturally improved lines for which one of these genotypes is an ancestor. Also, a recessive gene for rust resistance, rp_2, was identified previously in IL13b (7).

The *Rp* genes may be used effectively in commercial sweet corn hybrids in the continental United States. Because sweet corn is not grown extensively throughout the corn belt, selection pressures on *P. sorghi* will not be intense in the corn belt unless these genes become more widely used in commercial dent corn hybrids.

Likewise, selection pressures on overwintering populations of *P. sorghi* will not be intense unless dent or sweet corn possessing *Rp* genes is widely grown in the southern United States and Mexico. Therefore, incorporation of the Rp_1^d gene into the most popular susceptible sweet corn hybrids seems prudent. However, hybrids possessing Rp_1^d probably should not be grown where *P. sorghi* overwinters. If extensive cultivation of corn with Rp_1^d in the southern United States or Mexico selects for biotypes of *P. sorghi* that are virulent on Rp_1^d, then these biotypes are likely to spread by the *Puccinia* pathway to the major sweet corn-producing areas of the northern United States, where rust is a more common problem.

Other effective *Rp* genes could be paired with the Rp_1^d gene in sweet corn hybrids because most of the *Rp* genes are dominant. To identify which *Rp* genes are widely effective and could be usefully paired with Rp_1^d, the *Rp* series should be grown at various locations in Minnesota, Wisconsin, northern Illinois, New York, California, and other areas where rust is a common problem on sweet corn. The genes that are effective could then be backcrossed into elite commercial inbreds for use in hybrid combination with Rp_1^d. The Rp_1^d, Rp_1^f, Rp_1^g, Rp_1^i, *and* Rp_1^k genes were effective in Illinois field trials in 1966 (7), 1984, and 1986 and in greenhouse trials in which the inoculum included a mixture of *P. sorghi* biotypes from various locations in North America.

By combining *Rp* resistance with partial resistance, commercial sweet corn breeders could greatly reduce the possibility of rust epidemics on sweet corn resulting from the occurrence of or selection for a new biotype of *P. sorghi* that is virulent on *Rp* genes. To do this, high levels of partial rust resistance should be selected before beginning to backcross for *Rp* resistance. Then, during the backcross incorporation of *Rp* genes, susceptible backcross progeny (*rprp*) could be used to identify and select for partial resistance. If a biotype of *P. sorghi* that is virulent to *Rp* genes should occur, genotypes would still possess adequate partial resistance. Also, resistance such as that observed in Country Gentleman and IL677a is distinguishable from and may be combined with *Rp* resistance, although the resistance of IL677a appears to be controlled by one or two recessive genes (S. K. Kim and J. L. Brewbaker, *personal communication*).

Partial resistance by itself should be effective in most field situations. Even though some rust develops on partially resistant genotypes, rust severity remains below levels that warrant fungicide control (15). Several sugary enhancer (*se*) sweet corn hybrids (e.g., Miracle), which apparently possess the IL677a type of resistance, have sustained very little damage from rust in the Illinois sweet corn disease nursery (16). Reproduction-related components of the infection cycle are effected by this resistance (14). Also, the spatial and temporal development of rust has been slowed by partial resistance (J. M. Headrick and J. K. Pataky, *unpublished*).

Table 3. Rust severity and infection type of R168 dent corn inbreds possessing *Rp* genes for resistance to *Puccinia sorghi*

Rp gene	Source	Severity (%) 1984	Severity (%) 1986	Infection type
Rp_1^a	GG208R, Golden Glow, Golden King, PI 213777	38[a]	3	1[b]
Rp_1^b	B38	40	3	1
Rp_1^c	K148, B.Y. Dent, Syn A	35	7	4
Rp_1^d	Cuzco, Kitale, Njoro	0	0	0;
Rp_1^e	B49	0	0	0;
Rp_1^f	PI 172332	0	0	0;
Rp_1^g	PI 163558	0	0	0;
Rp_1^h	Guanajuato 29-157-A	70	7	4
Rp_1^i	PI 63558	0	0	0
Rp_1^j	Queretaro VI366	40	10	3
Rp_1^k	Queretaro V231-5-2-1	3	1	1
Rp_1^l	PI 163558N	80	5	4
Rp_1^m	PI 163563	10	5	4
Rp_1^n	BZU-20	60	10	4
Rp_3^a	25	40	10	3
Rp_3^b	M16, E697	80	10	4
Rp_3^c	NN14	0	0	0;
Rp_3^d	Leon I 27-4-1	40	7	4
Rp_3^e	Hidalgo 3-5-1-1-1	70	10	4
Rp_3^f	PI 251653	85	15	4
Rp_4^a	Queretaro V260-1-2-1	30	10	4
Rp_4^b	PI 193906	85	12	4
Rp_5	PI 196191	80	15	4

[a] Rust severity = relative percentage of the total leaf area covered by uredinia based on the modified Peterson scale (17).
[b] Infection type rated from 0; to 4 (8).

The purple pigmentation (anthocyanin accumulation) associated with the resistant reactions of IL677a and several lines derived from IL677a appeared to be a trait of those genotypes rather than a type of resistant response. I also have observed purple pigmentation on IL677a*Ht1* infected by race 1 of *Exserohilum turcicum* (Pass.) Leonard & Suggs in a disease nursery in Illinois. Northern leaf blight-resistant reactions, which are typically chlorotic, were atypically purple on IL677a*Ht1*. Anthocyanin accumulation also has been observed around *Bipolaris zeicola* (Stout) Shoem. (syn. *Helminthosporium carbonum* Ullstrup) and *B. maydis* (Nisik.) Shoem. (syn. *H. maydis* Nisikado & Miyake) infections of B73Ht × Va26Ht mesocotyls (5). In all cases, however, the anthocyanin accumulation is not considered to be responsible for the resistant response.

ACKNOWLEDGMENTS

I wish to thank John Headrick and John Gantz for technical assistance; Jack Juvik, Don Elliott, and Jim Pope for seed and information about pedigrees of Illinois sweet corn lines; and Jim Groth and Dave Fisher for discussions about the use of *Rp* and partial resistance.

LITERATURE CITED

1. Bergquist, R. R., and Pryor, A. J. 1984. Virulence and isozyme differences for establishing racial identity in rusts of maize. Plant Dis. 68:281-283.
2. Groth, J. V., Davis, D. W., Zeyen, R. J., and Mogen, B. D. 1983. Ranking of partial resistance to common rust (*Puccinia sorghi* Schw.) in 30 sweet corn (*Zea mays*) hybrids. Crop Prot. 2:219-223.
3. Groth, J. V., Zeyen, R. J., Davis, D. W., and Christ, B. J. 1983. Yield and quality losses caused by common rust (*Puccinia sorghi* Schw.) in sweet corn (*Zea mays*) hybrids. Crop Prot. 2:105-111.
4. Hagan, W. L., and Hooker, A. L. 1965. Genetics of reaction to *Puccinia sorghi* in eleven corn inbred lines from Central and South America. Phytopathology 55:193-197.
5. Heim, D., Nicholson, R. L., Pascholati, S. F., Hagerman, A. E., and Billett, W. 1983. Etiolated maize mesocotyls: A tool for investigating disease interactions. Phytopathology 73:424-428.
6. Hooker, A. L. 1962. Additional sources of resistance to *Puccinia sorghi* in the United States. Plant Dis. Rep. 46:14-16.
7. Hooker, A. L. 1969. Widely based resistance to rust in corn. Pages 28-34 in: Disease Consequences of Intensive and Extensive Culture of Field Crops. J. A. Browning, ed. Iowa Agric. Home Econ. Exp. Stn. Spec. Rep. 64. 56 pp.
8. Hooker, A. L. 1985. Corn and sorghum rusts. Pages 207-236 in: The Cereal Rusts. Vol. 2. Diseases, Distribution, Epidemiology, and Control. A. P. Roelfs and W. R. Bushnell, eds. Academic Press, Orlando, FL. 606 pp.
9. Hooker, A. L, and LeRoux, P. M. 1957. Sources of protoplasmic resistance to *Puccinia sorghi* in corn. Phytopathology 47:187-191.
10. Kim, S. K., and Brewbaker, J. L. 1976. Sources of general resistance to *Puccinia sorghi* on maize in Hawaii. Plant Dis. Rep. 60:551-555.
11. Kim, S. K., and Brewbaker, J. L. 1977. Inheritance of general resistance in maize to *Puccinia sorghi* Schw. Crop Sci. 17:456-461.
12. Mains, E. B. 1931. Inheritance of resistance to rust, *Puccinia sorghi*, in maize. J. Agric. Res. 43:419-430.
13. Mains, E. B., Trost, F. J., and Smith, G. M. 1924. Corn resistant to rust, *Puccini sorghi*. (Abstr) Phytopathology 14:47-48.
14. Pataky, J. K. 1986. Partial rust resistance in sweet corn hybrid seedlings. Phytopathology

76:702-707.
15. Pataky, J. K. 1987. Quantitative relationships between sweet corn yield and common rust, *Puccinia sorghi*. Phytopathology 77:1066-1071.
16. Pataky, J. K., Headrick, J. M., and Suparyono. 1985. 1985 Sweet corn disease nursery. Ill. Veg. Res. Rep. Hortic. Ser. 56:10-14.
17. Peterson, R. F., Campbell, A. B., and Hannah, A. E. 1948. A diagrammatic scale for estimating rust intensity of leaves and stems of cereals. Can. J. Res. (Sect. C.) 26:496-500.
18. Randle, W. M., Davis, D. W., and Groth, J. V. 1984. Improvement and genetic control of partial resistance in sweet corn to corn leaf rust. J. Am. Soc. Hortic. Sci. 109:777-781.
19. Saxena, K. M. S., and Hooker, A. L. 1968. On the structure of a gene for disease resistance in maize. Proc. Nat. Acad. Sci. 61:1300-1305.
20. Saxena, K. M. S., and Hooker, A. L. 1974. A study on the structure of gene Rp_3 for rust resistance in *Zea mays*. Can. J. Genet. Cytol. 16:857-860.
21. Wilkinson, D. R., and Hooker, A. L. 1968. Genetics of reaction to *Puccinia sorghi* in ten corn inbred lines from Africa and Europe. Phytopathology 58:605-609.
22. Zeyen, R. J., Groth, J. V., and Davis, D. W. 1984. Understanding rust resistance in sweet corn. 1984 Veg. Crops Res. Rep. Dep. Plant Pathol. Hortic. Univ. Minn. 94 pp.