Journal of Plant Biology, October 2006, 49(5) : 392-397

Epidermal Cell Response to Rust Hyphae and the Resistance Mode of Tropical Maize to Southern Corn Rust (*Puccinia polysora* Underwood)

Hee Chung Ji[1,2]*

[1]Department of Tropical Plant and Soil Science, University of Hawaii at Manoa, Honolulu, HI 96822, USA
[2]Department of Crop Science, College of Agriculture and Life Sciences, Chungnam National University, Daejeon 305-764, Korea

Southern Corn Rust (SCR), caused by *Puccinia polysora* Underwood, is found primarily in the tropics, occurring especially during wet and cool winters in Hawaii. Whereas Hi38-71, a tropical Hawaiian super sweet corn, is resistant to SCR, the G24 inbred is susceptible. Anatomical traits are distinct in these two lines, with differences evident in their epidermal cell shapes, cell types, epidermal cell walls, hairs, and bulliform cells. Trichomes are found on the leaves of Hi38-71 plants and their epidermal surface cells are flat. In contrast, the epidermal surfaces of G24 leaves are circular, rough, and woolly, and are uniform in their size and pattern. These woolly epidermal leaf surfaces on the susceptible G24 provide access for disease infection while the trichomes and smooth leaves may confer more resistance in Hi38-71. Our genetic study utilized Generation Mean Analysis (GMA) of progenies derived from crosses of these two inbred lines. Resistance to SCR was scaled from 1 (resistant) to 9 (susceptible) in the winter and spring in Hawaii. Average scores for Hi38-71 and G24 were 3.1 and 6.9 respectively, while the F_1 and F_2 hybrids were both highly resistant to the rust. Both additive and dominant effects for SCR resistance were highly significant, as were their epistatic interactions [aa] and [dd]. However, the additive x dominance interaction was not. Therefore, a single dominant gene in inbred line Hi38-71 might be involved in determining its high resistance to SCR.

Keywords: epidermal cell, *Puccinia polysora* Underwood, southern corn rust, tropical maize, *Zea mays* L.

Southern corn rust (SCR), which arises from *Puccinia polysora* Underwood, occurs throughout the Americas, Africa, Asia, and Australia. The wind-blown spores of this fungus fall on the corn plant, then germinate. As the level of infection increases, the leaves become covered by pustules before dying. Since first being identified in Alabama (USA) in 1891 on eastern grama grass (*Tripsacum dactyloides* L.) (Underwood, 1897), this pest has shown enormous destructive potential in maize (*Zea mays* L.) (Melching, 1975; Chen et al., 2004). The host range for polysora rust is confined to maize and its relatives, including teosinte, which strongly resembles maize, and to *Tripsacum* species (Ullstrup, 1977). SCR thrives in an environment of high temperature (27°C) and relative humidity (Shurtleff, 1980), mainly occurring in the tropics at elevations below 1220 m. Although present throughout the southeastern portion of the United States, it also has been reported as far north as Wisconsin (Pavgi and Flangas, 1959).

SCR can be easily distinguished from common rust caused by *Puccinia sorghi* Schw. because of the size, shape, and color of its pustules (Ullstrup, 1977). Compared with rare mortality from common rust, its damage is considerably more devastating and deadly (Scott et al., 1984). Maize lines with small pustules surrounded by chlorosis or necrosis are rated as resistant while maize with well-developed, mature pustules are considered susceptible (Hulbert et al., 1991). Single-gene forms of resistance to *P. polysora* have been identified (Storey and Howland, 1957; Chen et al., 2004).

Epiphytotic levels caused by polysora rust have been observed in West Africa (Rhind et al., 1952; Cammack, 1954; Robinson, 1996) since 1949. During 1950-51, yield losses from polysora rust were estimated to be as high as 39 to 50% (Rhind et al., 1952; Rodriguez-Ardon et al., 1980; Raid et al., 1988). SCR epiphytotics in the Mississippi Valley of the southern US in 1972-1974 emphasized the potential for *P. polysora* to reduce yields (Futrell, 1975). The severity of the disease was attributed, in part, to the nearly uniform susceptibility of the US maize crop to SCR (Futrell and Atkins, 1957; Futrell, 1975).

Although several monogenic resistance genes have been identified from a South African plant introduction (PI 186208; Ullstrup, 1965), these race-specific resistances are not durable, and have been overcome by racial variation in the pathogen (Robinson, 1996). Although the resistance gene, *Rpp9*, was introduced in South Africa, it broke down before any hybrids with that gene could be released commercially. However, a general resistance has been identified in field corn (Bailey, 1987; Zummo, 1988).

From Hawaii and the Philippines, Moon (1995) has reported a set of Recombinant Inbred Lines (RILs) that segregate approximately 50% tolerant and 50% susceptible. Ming (1995) has also identified restriction fragment length polymorphism (RFLP) markers linked to quantitative trait loci (QTLs) in that same set of RILs, and has found five QTLs on Chromosomes 2, 4, 6, 9, and 10, with emphasis on a possibly important role for the QTL on Chromosome 6.

Holland et al. (1998) have studied the inheritance of resistance to polysora rust in F_2 and F_3 populations, with broad-sense heritabilities estimated at 30% and 50%, respectively. A single locus on the short arm of Chromosome 10 has been identified as contributing 82 to 83% of the variation among field resistance scores in those two populations. QTLs on

*Corresponding author; fax +82-42-485-6738
e-mail cornhc@cnu.ac.kr

Chromosomes 3, 4, and 10 and their epistatic interactions can explain 96 to 99% of that variation.

This type of resistance is also present in sweet corn germplasm, and is of particular importance for breeding programs of that crop in tropical regions. Epiphytotics of SCR become most serious during the wet and cool winter season in Hawaii. Therefore, the aim of this study was to determined the epidermal cell response and the genetic resistance mode to SCR in inbred Hi38-71 (resistance) and G24 (susceptible) lines, using generation mean analysis (GMA) and a scanning electron microscope (SEM).

MATERIALS AND METHODS

Plant Materials

The F_1, F_2, and BC_1 and BC_2 populations were derived from the parental inbred Hi38-71 (resistant) and the semident maize inbred G24 (susceptible) lines of *Z. mays*. Hi38-71 is a sub line of Hi38 (commercial inbred *bt1* supersweet) bred from a *brittle1* (*bt*-1) conversion (6 backcrosses) of AA8sh2. The G24 inbred is one of the G set of Recombinant Inbred Lines (RILs) from the cross of Ki14 (a Thailand inbred) and B68 (Hawaii conversion, Hi31); it derives its rust susceptibility from the Corn Belt dent inbred B68. These experiments were designed as randomized complete blocks (RCB) with three replications in Winter 2002 and Spring 2004 at the Waimanalo Research Station, University of Hawaii on Oahu, USA. The trials were planted in ear to row plots 5 m long, with 0.75 m between-row spacing and about 25 plants per row. Plots consisted of two 5 m rows for the parents and the F_1 generation, four rows for the backcrosses, and six for the F_2 populations. Parents were planted in adjacent plots to minimize competition from hybrids. Two untreated seeds were planted per hill, then thinned to one per hill about three weeks afterward.

Measurements and Statistical Methods

About two to three weeks after mid-silking, 10 plants per row were visually rated according to a nine-point relative scale based on the levels of rust infection that appeared as chlorosis on the leaf surface and necrosis beyond the lesion. Natural inoculum was relied upon. Husk leaves were evaluated on a total of 120 plants for each parent, 120 plants for F_1, 360 for F_2, 240 for BC_1, and 240 for BC_2. The method for our rust rating scale was modified from that of Kim et al. (1980), where:

1 = Highly resistant; less than 1% of the area on the lower leaves being infected; considered monogenic resistant.

2-3 = Resistant; 2 to 20% of the area on the lower leaves and an ear leaf covered by pustules.

4 = Moderately resistant; 21 to 35% of the area on the lower leaves and an ear leaf covered by pustules.

5 = Intermediate; 36 to 50% of the area on the lower leaves and an ear leaf covered by pustules, making this classification ambiguous as to either resistant or susceptible.

6 = Moderately susceptible; 51 to 65% of the area on the lower leaves, an ear leaf, and the upper leaves covered by pustules. Intermediate infection on stalks.

7-8 = Susceptible; 66 to 80% of the area on the lower leaves, an ear leaf, and the upper leaves covered by pustules. Heavy infection on stalks.

9 = Highly susceptible; more than 80% of the entire plant covered by pustules, causing premature death.

The adopted genetic model was digenic, involving six parameters -- m, [a], [d], [aa], [ad], and [dd]. All data were assessed by Generation Mean Analysis (GMA), as modified from the methods of Gamble (1962a, 1962b) and Mather and Jinks (1977). Estimates for the six mean and variance parameters were obtained as follows:

$$
\begin{aligned}
m &= \bar{F}_2 \\
a &= \bar{B}_1 - \bar{B}_2 \\
d &= -1/2P_1 - 1/2P_2 + \bar{F}_1 - 4\bar{F}_2 + 2\bar{B}_1 + 2\bar{B}_2 \\
aa &= -4F_2 + 2\bar{B}_1 + 2\bar{B}_2 \\
ad &= -1/2P_1 + 1/2P_2 + \bar{B}_1 - \bar{B}_2 \\
dd &= \bar{P}_1 + \bar{P}_2 + 2F_1 + 4F_2 - 4\bar{B}_1 - 4\bar{B}_2
\end{aligned}
$$

In these formulas, 'm', 'a', 'd', 'aa', 'ad', 'dd' refer to the effects of the mean, additive, dominance, additive x additive, additive x dominance, and dominance x dominance, respectively. Narrow-sense heritability (h^2_{ns}) was estimated according to the method proposed by Warner (1952): $h^2_{ns} = [2V_{F2}-(V_{B1}+V_{B2})]/V_{F2}$, where V_{F2}, V_{B1}, and V_{B2} are the variances in the F_2, BC_1P_1, and BC_1P_2 generations. Broad-sense heritability (h^2_{bs}) was estimated as described by Burton (1951).

Sample Fixation for Scanning Electron Microscopy

Maize leaf samples were immediately fixed in 2.5% (w/v) glutaraldehyde and in 0.1 M sodium cacodylate buffer (pH 7.0). These plant materials were then dehydrated in a graded alcohol series and embedded in Spurr's resin (Spurr, 1969). Samples were dried in a Critical Point Dryer (CPD 030) after treatment in 50% isoamyl acetate inliquid (1:1 isoamyl acetate:alcohol) for 10 min and in 100% isoamyl acetate for 5 min. Samples were coated with gold or gold-palladium in a sputter coater (SCD 005), and observed using a scanning electron microscope (PHILIPS XL30 ESEM; FEI/Philips, USA) with an accelerating voltage of 15 kV.

RESULTS

Mixed Infection of *P. sorghi* and *P. polysora* on Maize Leaves, and Phenotypes of Infected Parent Lines

Unlike with infections by the common rust *P. sorghi* (Fig. 1B), those of SCR, caused by *P. polysora*, differ in maize, being aggressive enough to kill the host. The former is often observed at the Waimanalo Research Station following the polysora rust. Highly resistant genotypes have smaller uredosori than those that are moderately resistant or susceptible. Here, the epiphytotics of SCR began approximately two

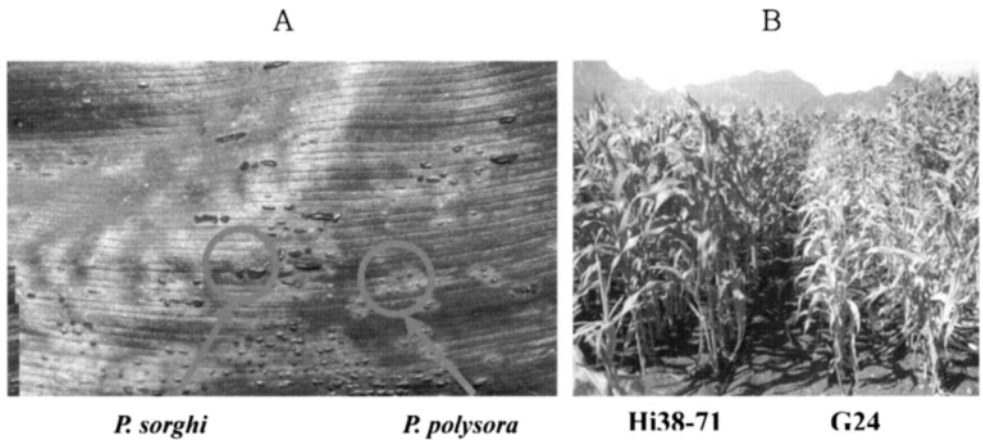

Figure 1. Mixed infection by *P. sorghi* and *P. polysora* on maize leaf, and phenotypes of infected parent lines. **A**, *P. sorghi* (left) and *P. polysora* (right). **B**, Parent lines, Hi38-71 (left) and G24 (right).

to three weeks before flowering, making it clearly distinguishable in the degree of resistance versus susceptibility. When mixed infections of *P. sorghi* and *P. polysora* occurred on maize leaves, the uredinia of the latter were generally smaller, more circular, and lighter in color (Fig. 1). Hi38-71, a sub line of Hi38 (commercially inbred *bt1* supersweet) was intermediate in its mature plant resistance (Fig. 1B) and also repeatedly exhibited no aphid development. Moreover, although many tropical inbreds generally show greater rust resistance in Hawaii, the G24 inbred (Fig. 1B) derived its susceptibility from the Corn Belt dent inbred B68.

Comparative Anatomy of Epidermal Cells between Hi38-71 and G24

Cell body structure is important for determining plant traits such as disease and insect resistances. Here, the epidermal cell shapes, cell types, epidermal cell walls, hairs, and bulliform cells differed anatomically between our Hi38-71 and G24 lines. For example, trichomes were found on the leaves of the former (Fig. 2A) but not the latter (Fig. 2B). The epidermal cells from G24 (Fig. 2B, H) were of relatively uniform size and pattern compared with the varying shapes of the Hi38-71 epidermal cells (Fig. 2A). Because the epidermes of G24 leaves (Fig. 2D, F, H) were rough and woolly, they provided a more suitable surface for contact with dust and diseases, possibly explaining the greater susceptibility to rust by that line. Furthermore, G24 surface cells were circular (Fig. 2B, E) while those of Hi38-71 were flat (Fig. 2G, I). Sizes of the cell walls with cuticle were 1.58 μm in G24 and 1.34 μm in Hi38-71 (Fig. 2I, J). The section shapes in Figure 2I and J differed between G24 and Hi38-71.

Genetic Analysis of Tropical Maize Resistance to Southern Corn Rust

Generation means and standard errors of visual score values for the resistability to SCR are summarized in Figure 3. Within a single line, the values did not differ between the Spring 2004 trials and the Winter 2002 trials. Average scores for the parents were 3.2 ± 0.17 (Winter) and 3.0 ± 0.36 (Spring) for inbred line Hi38-71 compared with 6.9 ± 0.43 (Winter) and 6.8 ± 0.83 (Spring) for inbred line G24. Values

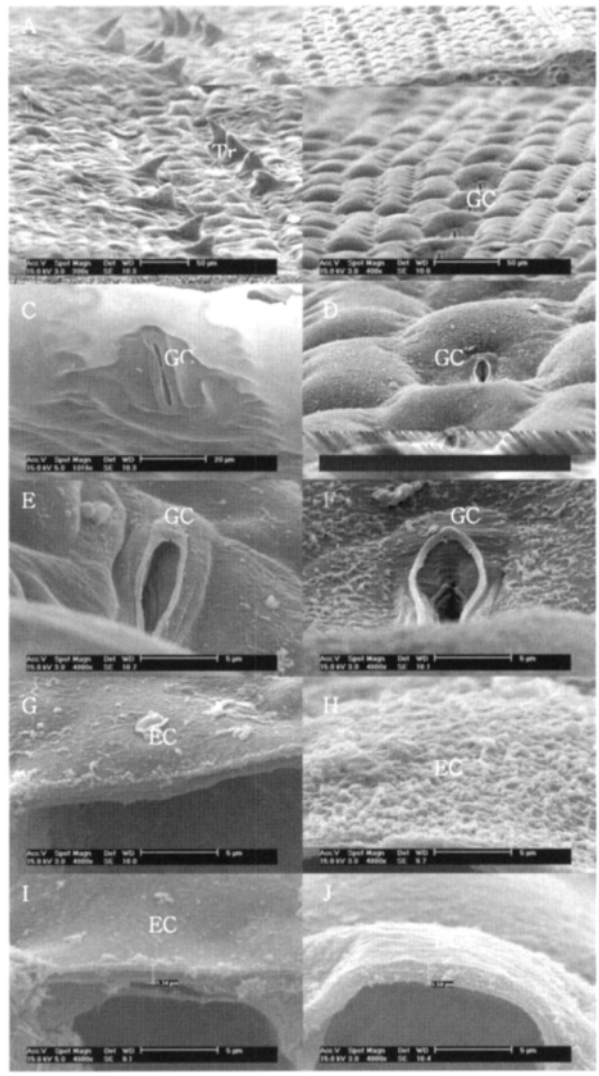

Figure 2. Scanning electron micrographs of leaf cell morphology in Hi38-71 and G24. **A**, Trichomes showing on Hi38-71. **B**, Absence of trichomes on G24. **C**, **D**, **E**, and **F**, Stomata. **G**, **H**, Leaf epidermal cell surfaces. **I**, **J**, Cell wall with cuticle of leaf epidermal cell. Imaged by Philips XL30 ESEM. Tr, trichome; GC, guard cells; EC, epidermal cells.

Table 1. Estimates for components of the generation means for the cross Hi38-71 x G24 fitting a six-parameter model by perfect fit estimation.

Six-parameter model	Rating score
m	2.71 ± 0.29**
a	-1.88 ± 0.04**
d	5.94 ± 0.71**
aa	2.27 ± 0.29**
ad	-0.19 ± 0.19ns
dd	-5.77 ± 0.44**

*, **Significant at the 0.05 and 0.01 probability levels, respectively; ns, not significant at $a=0.05$. m, main effect; a, additive effect; d, dominance effect; aa, additive x additive interaction; ad, additive x dominance interaction; dd, dominance x dominance interaction.

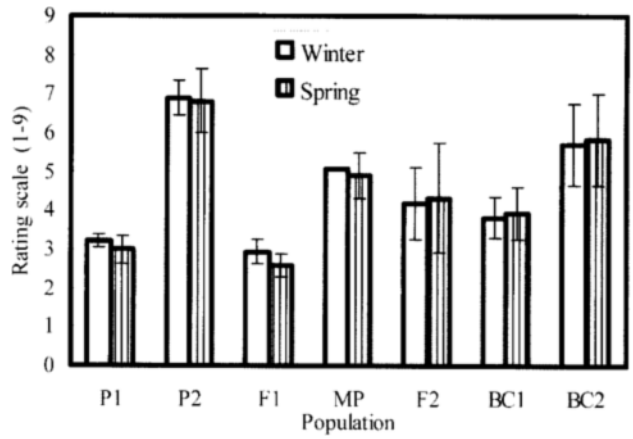

Figure 3. Visual scoring values for SCR in parents, F_1, F_2, and backcross (BC_1, BC_2) generations. F_1, F_2, and BC_1 and BC_2 populations were derived from parental inbred Hi38-71(resistant; P1) and semi-dent maize inbred G24 (susceptible; P2). P_1, Hi38-71; P_2, G24. 60 P_1, P_2, F_1: 60 plants each in Winter and Spring; F_2: 180 plants each in Winter and Spring; BC_1, BC_2: 120 plants each in Winter and Spring. Rating scale (1-9): 1, highly resistant: <1% of area showing appearance of rust on lower leaves; 2-3, resistant: 2-20% coverage on lower leaves and ear leaf; 4, moderately resistant: 21-35% coverage on lower leaves and ear leaf; 5, intermediate: 36-50%; 6, moderately susceptible: 51-65% coverage on lower leaves, ear leaf, and upper leaves; 7-8, susceptible: 66-80% coverage on lower leaves, ear leaf, and upper leaves; 9, highly susceptible: >80% coverage on entire plant.

for their F_1 and F_2 hybrids were 2.9 ± 0.05 and 4.2 ± 0.06, respectively. Resistance scores for the F_1 hybrid were the lowest among all trials, while those from the F_2 generation were higher than from P_1 and F_1. F_2 also showed a wide range of variation because of segregation. In the F_2 and backcross lines, values were higher than for P_1 and F_1. In back-crossed lines of the resistant and susceptible parents, ranges in variation were wider than with the parents and the F_1 population. These results demonstrate that the Hi38-71 inbred and the F_1 and F_2 hybrids are significantly resistant to SCR. Moreover, variations in the non-segregated offspring were generally fewer than for the segregated offspring, except for the susceptible parent. Therefore, heterosis might contribute to the conference of SCR resistance.

On a family basis, the broad-sense heritability of rust scores was 68.3%; the narrow-sense heritability was 53.3% (Table 1). The minimum number of effective gene loci, based on Castle and Wright formulas, was 0.99. Therefore, it can be concluded that resistance to SCR is controlled by a single dominant gene in this maize material.

Furthermore, all additive x dominance interactions, except for [ad], were highly significant in the six-parameter model (Table 1).

DISCUSSION

Southern Corn Rust, caused by *P. polysora*, is aggressive enough to kill its host plants. Unlike with *P. sorghi* (Fig. 1B), it is clearly distinguishable by its size, shape, and color, showing a different degree of resistance/susceptibility when a mixed infection occurs on maize leaves (Fig. 1A). In this study, the Hi38-71 inbred line proved resistant to SCR whereas the inbred G24 was susceptible (Fig. 1B).

Unique anatomical traits, e.g., epidermal cell shapes, cell types, epidermal cell walls, hairs, and bulliform cells, were important in their association with this rust resistance. For example, trichomes were found on Hi38-71 leaves (Fig. 2A) and surface cells were flat. In contrast, the epidermal surfaces of G24 leaves were rough and woolly, with cells being circular and uniform in their sizes and patterns (Fig. 3). This surface type may have enabled easier contact for dust and diseases by G24, making this line susceptible to SCR while Hi38-71 was resistant. The cell walls with cuticle were also thicker on the G24 leaves. Based on these characteristics, I suggest that Hi38-71 is useful for studies of plant development and physiology.

The parent inbred line Hi38-71 and the F_1 hybrids (Hi38-71 x G24) were highly resistant to SCR; the backcross line BC_1 also showed resistance. This indicates that Hi38-71 may be an important tool for breeding programs to improve SCR resistance. Such a trait would be even more important in field corn because such varieties require a longer growing season than does sweet corn. Levels of *polysora* resistance are generally greater in tropical field corn. The Hi38-71 inbred is a sub line of Hi38, which was bred from a *bt*-1 conversion (6 backcrosses) of AA8sh2. In previous studies of its resistance, *Rp*1-D has broken down because of the racial variation in the pathogen. This gene is located on the short arm of maize Chromosome 10, which carries a cluster of complex gene loci for resistance to common rust as well as to SCR (Holland et al., 1998; Hulbert et al., 2001). Chang (1976) also has observed a possible linkage between corn leaf aphid resistance and common rust resistance in AA8sh2. Although such aphid resistance in Hi38-71 probably originates from AA8sh2, it is still unclear whether that inbred line contains *Rp*1-D or other resistance gene(s). Therefore, in-depth molecular research will be required. The Hi38-71 inbred is valuable for sweet corn breeding in tropical regions because of its dual resistance to corn leaf aphids and SCR. Therefore, the development of molecular markers would help in determining the relationship between aphid and rust resistances in that line.

A digenic model with additive x additive and dominance x dominance type epistasis adequately explains the genetic

variation observed here. The results demonstrate that epistasis is involved in the resistance to SCR. Therefore, higher-order interactions, such as trigenic epistasis and similar complex factors, do not make significant contributions to the differences in SCR resistance among the generations studied. This relationship between epistatic interactions and SCR resistance in a Tropical Corn Belt maize population has also been reported by Holland et al. (1998).

The negative sign for [dd] suggests interactions between increasing and decreasing alleles. Therefore, improving the resistance level in the Hi38-71 inbred is possible. Likewise, the significance of [a] and [aa] implies that a portion of the resistance can be fixed in inbred maize lines. Due to this simultaneous significance of [d] and [dd], a reciprocal recurrent selection scheme might be appropriate.

When epistasis is significant in such a study, some bias should be expected when estimating the additive and dominance effects (Hallauer and Miranda, 1981). This phenomenon provides a better understanding of the SCR association rather than when one obtains precise estimates of gene effects. Here, no information has been gained on gene effects in the different genetic backgrounds or environments. Further study is needed to evaluate any Genotype x Environment (G x E) interactions associated with SCR resistance.

Based on the results here, I can conclude that the Hi38-71 inbred line, a Tropical Hawaiian super sweet corn, is resistant to SCR while the G24 inbred is susceptible. Differences between their respective epidermal cell structures may sufficiently explain the relationship between such morphological traits and the incidence of disease infection. The woolly, rough surfaces on the G24 leaf epidermes may have enhanced the potential for contact with dust and diseases, making that line more susceptible to rust, while the trichomes and smooth epidermal cell surfaces on Hi38-71 leaves may have aided in the development of disease resistance in that line. SCR resistance was also examined via Generation Mean Analysis (GMA) of progenies derived from crosses of those inbred lines. The parent inbred line Hi38-71 and F_1 hybrids (Hi38-71 x G24) were highly resistant to SCR, and the F_2 and Backcross line BC_1 also showed some degree of rust resistance. Therefore, the Hi38-71 inbred is essential to breeding programs because of the possible existence of a single dominant gene. Finally, the genetic basis for this resistance was confirmed through the use of broad-sense and narrow-sense heritability scores and Castle and Wright formulas, which indicated the minimum number of effective gene loci. Both additive and dominant effects for SCR resistance, as well as Epistatic interactions [aa] and [dd], were highly significant, but the additive x dominance interaction was not.

Received August 1, 2006; accepted September 27, 2006.

LITERATURE CITED

Bailey BA (1987) Resistance to *Puccinia polysora* in *Zea mays* L.: Identification, genetic transmission and induced molecular responses. Ph.D. dissertation. Texas A and M University, College Station, pp 86

Burton WG (1951) Quantitative inheritance in pearl millet (*Pennisetum glaucum*). Agron J 43: 409-417

Cammack RH (1954) Observation in *Puccinia polysora* Underwood in West Africa. West Africa Maize Rust Res Unit Annu Rep 1: 16-31

Chang SH (1976) The genetics of resistance to the corn leaf aphid, *Rhopalosiphum maidis* (Fitch), in corn. Ph.D. thesis. University of Hawaii, Honolulu

Chen CX, Wang ZL, Yang DE, Ye CJ, Zhao YB, Jin DM, Weng ML, Wang B (2004) Molecular tagging and genetic mapping of the disease resistance gene *RppQ* to southern corn rust. Theor Appl Genet 108: 945-950

Futrell MC (1975) *Puccinia polysora* epidemics on maize associated with cropping practice and genetic homogeneity. Phytopathology 65: 1040-1042

Futrell MC, Atkins IM (1957) Trends in disease in cereal crops in Texas in recent years. Plant Dis Rep 41: 42-46

Gamble EE (1962a) Gene effects in corn (*Zea mays* L.): I. Separation and relative importance of gene effects for yield. Can J Plant Sci 42: 339-348

Gamble EE (1962b) Gene effects in corn (*Zea mays* L.): II. Relative importance of gene effects for plant height and certain component attributes of yield. Can J Plant Sci 42: 349-358

Hallauer AR, Miranda JB (1981) Quantitative Genetics in Maize Breeding. Iowa State University Press, Ames

Holland JB, Uhr DV, Jeffers D, Goodman MM (1998) Inheritance of resistance to southern corn rust in tropical-by-corn-belt maize populations. Theor Appl Genet 96: 232-241

Hulbert SH, Lyons PC, Bennetzen JL (1991) Reactions of maize lines carrying *RP* resistance genes to isolate of common rust pathogen *Puccinia sorghi*. Plant Dis 75: 1130-1133

Hulbert SH, Webb CA, Smith SM, Sun Q (2001) Resistance gene complexes: Evolution and utilization. Annu Rev Phytopathol 39: 285-312

Kim SK, Buddenhagen IW, Brewbaker JL (1980) Changes in the apparent resistance of maize to *Puccinia sorghi* Schw. during development. SABRAO J 12: 139-147

Mather K, Jinks JL (1977) Introduction to Biometrical Genetics. Chapman and Hall, London

Melching JS (1975) Corn rust: Type, races and destructive potential. The American Seed Trade Association, Washington DC, pp 90-115

Ming RG (1995) Restriction fragment length polymorphism analysis of host-plant resistance to four maize pathogens. Ph.D. thesis. University of Hawaii, Honolulu

Moon HG (1995) Quantitative genetic analysis of recombinant inbred lines (RILs) from tropical maize single crosses. Ph.D. thesis. University of Hawaii, Honolulu

Pavgi MS, Flangas AL (1959) Occurrence of southern corn rust in Wisconsin. Plant Dis Rep 43: 1239-1240

Raid RN, Pennypacker SP, Stevernson RE (1988) Characterization of *Puccinia polysora* epidemics in Pennsylvania and Maryland. Phytopathology 78: 579-585

Rhind D, Waterston JM, Deighton FC (1952) Occurrence of *Puccinia polysora* Underw. in West Africa. Nature 169: 631-632

Robinson RA (1996) Return to Resistance: Breeding Crops to Reduce Pesticide Resistance. AgAccess, Davis, pp 173-199

Rodriguez-Ardon R, Scott GE, King SB (1980) Maize yield losses caused by southern corn rust. Crop Sci 20: 812-814

Scott GE, King SB, Armour JW (1984) Inheritance of resistance to southern corn rust in maize populations. Crop Sci 24: 265-267

Shurtleff MC (1980) Compendium of Corn Diseases. Ed 2, APS Press, St. Paul

Spurr AR (1969) A low-viscosity epoxy resin embedding medium for electron microscopy. J Ultrastr Res 26: 31-43

Storey HH, Howland AK (1957) Resistance in maize to the tropical American rust fungus, *Puccinia polysora* Underw.: I. Genes Rpp_1 and Rpp_2. Heredity 11: 289-301

Ullstrup AJ (1965) Inheritance and linkage of a gene determining resistance in maize American race of *Puccinia polysora*. Phytopathology 55: 425-430

Ullstrup AJ (1977) Diseases of corn, *In* GF Sprague, ed, Corn and Corn Improvement. American Society of Agronomy, Madison, pp 391-500

Underwood LM (1897) Some new fungi chiefly from Alabama. Bull Torrey Bot Club 24: 81-86

Warner JN (1952) A method for estimating heritability. Agron J 44: 427-430

Zummo N (1988) Components contributing to partial resistance in maize to *Puccinia polysora*. Plant Dis 72: 157-160