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Sugary1 and *Sugary Enhancer1* Sweet Corn Inbreds with Resistance to Northern Leaf Blight

John A. Juvik, M.A. Rouf Mian, and Andrea J. Faber
Department of Horticulture, University of Illinois, 307 Plant and Animal Biotechnology Laboratory, Urbana, IL 61801

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In Illinois, other regions of sweet corn production in the United States, and overseas, epidemics of northern leaf blight (NLB) in maize *(Zea mays* L.) are common. The fungal pathogen *Exserohilum turcicum* (Pass.) K.J. Leonard & E.G. Suggs is the causal agent of this foliar disease. NLB can significantly decrease yields and ear quality of susceptible and moderately susceptible sweet corn hybrids (Levy and Pataky, 1992). The gene introgression conferring NLB resistance into elite germplasm could be of significant value to the sweet corn industry, especially for late-season plantings when inoculum levels are favorable for rapid epidemic development. To address this issue, the Illinois Agricultural Expt. Station announces the release of one *sugary1 (su1)* and four *sugary enhancer1 (se1)* inbreds with qualitative (monogenic) and quantitative NLB resistance. The newly developed inbreds are designated IL 801a, IL 802a, IL 802b, IL 803a, and IL 804a.

Exserohilum turcicum infection causes large, cigar-shaped lesions to form on maize leaves (Hooker and Perkins, 1984). Individual lesions can enlarge, coalesce, and cover most of the leaf surface. Secondary spread of inoculum can result in a high proportion of necrotic leaf tissue. Pataky (1992) estimated that, in the upper 75% of the leaf canopy, for each 1% increase in leaf area infected by NLB there was a corresponding yield loss of 0.5%. The form of the pathogen that instigates infection is the conidium, which disseminates by wind. The most important environmental factor influencing the degree of plant infection is the dew period duration. Thus, cool, moist nights in late summer provide optimal conditions for NLB epidemics (Levy and Pataky, 1992).

To date, four genes (*Ht1, Ht2, Ht3,* and *HtN*) have been identified that qualitatively suppress lesion development (Hooker and Perkins, 1984). *Ht1, Ht2,* and *HtN* are nonallelic and map to chromosomes 2L, 8L, and 8L, respectively (Coe, 1993). The resistant phenotype of *Ht1, Ht2,* and *Ht3* displays a hypersensitive chlorotic zone surrounding the infection site, preventing lesion expansion. Resistance conditioned by *HtN* prolongs the latent period and incubation time of the inoculum (Hooker and Perkins, 1984). Four *E. turcicum* races have been identified in North America and have the following virulence formulas (effective/ineffective host genes): race 0 *(Ht1, Ht2, Ht3, HtN/*none), race 1 *(Ht2, Ht3, HtN/Ht1),* race 23 *(Ht1, HtN/Ht2, Ht3),* and race 23N *(Ht1/Ht2, Ht3, HtN)* (Leonard et al., 1989).

Recent increase in consumer demand and greater market value of the *se1* and *shrunken2* sweet corn hybrids makes NLB resistance in these new endosperm types of particular interest to growers (Marshall, 1987). Based on the sucrose and total sugar concentrations of fresh kernels harvested 20 days after self-pollination (DAP), IL 801a, IL 802a, IL 802b, and IL 803a seem to be homozygous for *se1,* a recessive modifier of the *su1* endosperm mutation (Ferguson et al., 1978). When homozygous, *se1* typically increases endosperm sucrose content in *su1* backgrounds by 50% to 100% while maintaining high levels of phytoglycogen—the water-soluble starch that gives a creamy texture to the kernels at typical harvest maturities (La Bonte and Juvik, 1990). Consumers prefer sweet corn with more kernel-sucrose content, and this type of corn retains higher sugar and moisture content for longer postharvest periods, providing the industry with more time to transport, process, and market a product with superior eating quality.

Origin

A.L. Hooker and W.L. Pedersen (Dept. of Plant Pathology, Univ. of Illinois, Urbana–Champaign) identified and introgressed the *Ht*-resistance alleles from different sources into several dent corn inbreds. Initially, the dent inbreds W22-*Ht1,* A619-*Ht2,* A619-*Ht3,* and *W22-HtN* each were hybridized with IL 677a, a high-quality, *su1* inbred homozygous for *se1*. Then, the F_1 was either backcrossed to IL 677a or crossed to another *su1 se1* inbred. *Sugary 1* seed from the BC_1 generation was planted on the Univ. of Illinois South Farm to generate ≈3000 seedlings.

Seedlings at the four- to five-leaf stage were inoculated with *E. turcicum* race 0 and race 1 (formerly race 1 and 2, respectively). Races 0 and 1 originally were isolated from corn grown in central Illinois. J.K. Pataky (Dept. of Plant Pathology, Univ. of Illinois) provided the inoculum and produced it by culturing *E. turcicum* on lactose-casein hydrolysate agar at room temperature for 2 to 3 weeks. Cultures were flooded with tap water, ground in a blender, and filtered through several layers of cheesecloth (Meyer et al., 1991). Aliquots of the extracts then were mixed 1 : 1 and diluted with distilled water to generate a suspension of ≈103 conidia/ml. A backpack sprayer was used to deliver ≈3 to 5 ml of the inoculum into the whorls of each developing plant. Plants were reinoculated ≈7 to 10 days later to avoid escapes and to ensure adequate disease development.

About 10 to 20 days after the second inoculation, plants were evaluated for monogenic disease-resistance response, and those plants that did not display a hypersensitive response or a prolonged latent period were rogued. In addition to monogenic resistance, disease severity was assayed visually by determining the approximate percentage of total leaf area infected, using Elliott and Jenkins' (1946) standard diagram, modified to include additional classes. Plants carrying any of the *Ht* genes and expressing the fewest and smallest lesions were self-pollinated.

Ears from selfed plants were harvested 45 to 55 days after pollination and dried in a forced-air oven at 35C. A subset of ears was saved; based on shape, size, uniformity, and yield of good-quality seed. Visual selection for kernels homozygous for *se1* was conducted on mature, dry seed (La Bonte and Juvik, 1990). This inoculation process with pedigree selection was repeated for four additional generations to generate ≈110 BC_1S_5 lines. By the fifth cycle, lines did not seem to be segregating for NLB resistance. Plants within these lines were selfed without inoculation for two more cycles, with selection for ear and fresh quality (flavor, sweetness, and tenderness at 20 DAP) to yield 34 BC_1S_7 inbred lines.

These 34 lines were tested for disease resistance and fresh-market quality. Seed of each line was planted in the greenhouse in flats, with five rows of five seeds each. At the four- to five-leaf stage, the plants were inoculated twice with *E. turcicum* race 0 and race 1 as previously described. Fifteen days after inoculation, the leaves of each of the 25 seedlings of each inbred were assayed for qualitative *(Ht)* and quantitative disease response. Genotypes were scored resistant (when they carried lesions displaying hypersensitivity with a dark ridge surrounding the lesion) or susceptible. Plants also were assigned values for overall leaf area necrosis, ranging from 0 to 10 [0 = no necrotic tissue; higher degrees of leaf necrosis compared to that of the susceptible control (Ia5125 *sul*)].

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Table 1. Disease response, days from planting to 50% silk, number of kernel rows, kernel color, and sugar concentrations at 20 days after pollination for the five northern leaf blight (NLB) -resistant inbreds.

Inbreds	*Ht* gene	Monogenic disease response[z] Race 0	Monogenic disease response[z] Race 1	NLB disease severity[y] Race 0	NLB disease severity[y] Race 1	Days to 50% silk[x]	No. kernel rows	Yellow kernel color	Kernel sugar concn (mg/g dry wt) Fructose	Glucose	Sucrose	Total
Line												
IL 801a	*Htl*	R	S	1	2	52	12	Light	25	32	161	218
IL 802a	*Ht2*	R	R	2	4	60	18	Medium	18	19	173	210
IL 802b	*Ht2*	R	R	0	1	59	18	Medium	19	21	189	229
IL 803a	*Ht3*	R	R	1	1	56	12	Light	31	31	176	238
IL 804a	*HtN*	R	R	1	2	62	16	Medium	14	19	107	140
Reference inbreds[w]												
IL 677a		S	S	6	5	60	14	Light	20	22	201	243
Ia5125 *su1*		S	S	10	10	55	12	Dark	15	17	102	134
$LSD_{0.05}$									14	11	41	65

[z]A chlorotic hypersensitive zone surrounding the lesions was used to indicate presence of *Htl, Ht2,* and *Ht3* alleles, while the extension of lesion latent period was considered diagnostic of *HtN*.

[y]Overall leaf area necrosis scored visually from 0 to 10, where 0 = no necrotic tissue and 10 = severity comparable to Ia 5125 *sul*, the susceptible control.

[x]Days to 50% silking = days to when 50% of the ears of each inbred were silking from planting on 8 June 1993.

[w]IL 677a is a high-quality inbred displaying kernel sugar concentrations typical of a line homozygous for *sel.* Ia5125 is NLB susceptible and has kernel sugar concentrations typical of an inbred homozygous for *su1*.

Kernel sugar concentrations were quantified from ears of the 34 inbreds grown in the field in 1993. Four fresh ears were harvested from each inbred at 20 DAP. Harvested ears were husked, frozen in liquid N, and stored at –80C for subsequent analysis. One hundred frozen kernels (25 each of the four ears) were separated from cobs of each inbred and bulked to from a composite sample. Kernel samples were weighed, freeze-dried, and reweighed to determine dry weights and moisture content and then were ground into a fine powder with a coffee grinder. Four 250-mg samples of kernel powder from each inbred sample then were extracted for soluble sugar concentration using procedures described by Juvik and La Bonte (1988). Kernel sucrose, fructose, and glucose concentrations were quantified using high pressure liquid chromatography (Waters Associates, Milford, Mass.) as described by Azanza et al. (1994). Samples of IL677a (an inbred homozygous for *se1*) and Ia5125m (a *su1* inbred) were included as controls.

Based on NLB resistance from the greenhouse evaluation, inbred field performance in 1993, and kernel sugar concentration at 20 DAP, a subset of five inbreds was chosen for public release. IL 801a is an S_7 from the cross of IL 766a (*su1 se1*) to the F_1, IL677a (*su1 se1*) x w22 *Ht1* (*Su1 Se1*); IL 802a and IL 802b are S_7s from the backcross, IL 677a (*su1 se1*) x [IL 677a (*su1 se1*) x A619 *Ht2* (*Su1 Se1*)]; IL 803a and S_7 from the cross of IL774a (*su1 se1*) to the F_1, IL 677 (*su1se1*) x A619 *Ht3* (*Su1 Se1*); and IL 804a and S_7 from the backcross, IL 677a (*su1 se1*) x [Il 677a (*su1 se1*) x W22 *HtN* (*Su1 Se1*)]. Information is available on the parentage of IL 677a, IL 766a, and IL774a (Rhodes et al., 1982).

Description

IL801a, IL 802a, IL 802b, IL 803a, and IL 804a are homozygous for their respective *Ht* resistance genes. In addition to monogenic resistance, the lines display fewer and smaller lesions compared to the susceptible control (Ia 5125 sul) and IL 677a (Table 1). Disease evaluations were conducted during Summer 1993—a wet and humid summer, extremely conducive to NLB and other leaf diseases in corn. Even without artificial inoculation, Ia 5125 *su1* was severely infected with NLB, Stewart's wilt, and rust. The five inbreds were almost free of these foliar diseases, suggesting that the quantitative NLB resistance observed in these inbreds may be associated with resistance to other maize foliar pathogens, as previously reported in sweet corn lines developed from IL 677a (Meyer et al., 1991).

Kernel sugar concentrations at 20 DAP (Table 1), rates of kernel moisture loss, and appearance of mature, dry kernels suggest that IL 801a, IL 802a, IL 802b, and IL 803a are homozygous for *se1*. Sugar levels of these inbreds were roughly comparable to that of IL 677a. In contrast, kernel sugar concentration of IL 804a was in the range of that of Ia 5125 *sul*, suggesting that this inbred does not carry *se1*. Analysis of variance was used to generate least significant difference values at $P \leq 0.05$. Kernel sucrose (which accounted for 79% of total kernel sugars) and total sugar concentrations of IL 801a, IL 802a, IL 802b, and IL 803a were significantly higher than those of Ia 5125 *su1* and IL 804a. Sucrose and total sugar concentrations of these four inbreds were not significantly different from those of IL 677a *su1se1* (Table 1). Informal taste tests of raw kernels of these inbreds in the field at 20 DAP indicated that all five had tender, sweet-tasting kernels with good to excellent husk protection. Each of the five lines showed excellent resistance to lodging and produced a high yield of good-quality, disease-free seed. The ear appearance, size, and seed yield of IL 802a and IL 802b are particularly notable.

IL 801a, IL 802a, IL 802b, IL 803a, and IL 804a can be used directly for the development of *su1* and *su1 se1* commercial, single-cross hybrids or as germplasm sources for backcrossing NLB resistance and the *se1* allele into elite inbreds. The lines have not been tested for combining ability; thus, their potential for direct use as parents of commercial hybrids is unknown.

Availability

A limited quantity of seed (50 kernels per release) is available for distribution on written request to J.A.J., Dept. of Horticulture, 307 Plant and Animal Biotechnology Laboratory, Univ. of Illinois, Urbana, IL 62801.

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