農業科學技術硏究, 第48輯, 2014年 6月
Agricultural Science &
Technology Research, Vol. 48

Genetic variation of sweet corn inbreds on seedling emergence by *Fusarium moniliforme* infection

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요 약

마름병에 감염된 옥수수 종자는 마름병 발병율을 높이고 재배지에서 감염율을 높이게 된다. 식용 및 사료용 옥수수 계통 종자 역시 *F. moniliforme*에 감염된 경우가 있다. 일반적으로 *F. moniliforme*는 종자를 썩게 만들지만, 외면상으로 감염이 확인되지 않아 보이면서 건강하게 보이는 종자들이 종종 발견된다. 식용 단옥수수 계통들 중에서 *F. moniliforme*에 감염되었으나 저항성을 보이며 충실한 발아력을 가진 경우가 발견되었다. 본 연구에서는 *su1, se1, sh2* 육종 계통들을 대상으로 인위적으로 *F. moniliforme*에 감염시키고 발아력을 측정하여 유전적 다양성을 조사하였다. *F. moniliforme*에 감염 후 저항성 기작을 확인하였으며, 발아를 촉진하는 경우도 발견되었다.

Additional key words: *Fusarium moniliforme*, Germination, Germplasm, Mutation, Plant breeding

Introduction

On sweet corn *F. moniliforme* has been reported to cause stalk rot, leaf spot, ear and kernel rot, damping-off, and seedling blight (Christensen and Wilcoxson, 1966; Shurtleff, 1977). Planted infected seed may increase the incidence of seedling blight (Furtell and Kilgore, 1969) and contribute to systemic infection of plants (Foley, 1962). Although *F. moniliforme* causes a kernel rot, the pathogen is often found in healthy kernels that appear to be physically undamaged (Koehler, 1942; Thomas and Buddenhagen, 1980). Sixty inbreds of field or dent corn sampled in California, all had kernels infected with *F. moniliforme* (Smith and Madson, 1949).

Sweet corn inbreds have been identified that exhibit partial resistance to kernel infection by *F. moniliforme* and good emergence (Headrick and Pataky, 1989). Headrick et al. (1989) found genotypic variation for *F. moniliforme* susceptibility among *su1*, *sh2* and *se1* inbreds.

The *sugary enhancer* (*se1*) gene is a recessive modifier of the *su1* endosperm mutation (Ferguson et al., 1979). When homozygus, the *se1* allele increase total sugars in *su1* kernels to levels comparable to those found *sh2* kernels without a

reduction in phytoglycogen content (Gonzales et al., 1974; 1976). *Shrunken2* (*sh2*) hybrids have two to three times more sugar and one third less starch than *sugary1* (*su1*) hybrids from 16 to 28 days after pollination (DAP) (Creech, 1965). *Shrunken2* hybrids also have a higher moisture content and 10 to 20 % of phytoglycogen observed in *sugary1* hybrids (Garwood et al., 1976; Dickson et al., 1983).

Despite of the desirable attribute of the *se1* and *sh2* endosperm types, the significant reduction has hindered their commercial utilization (Douglass et al., 1993; Soberalske and Andrew, 1978; Wolf and Showalter, 1974). A number of kernel characters have been suggested as important in the poor emergence and seedling vigor associated with the *sh2* and *se1* endosperm mutations including; susceptibility of kernels in infection by fungal pathogens (Berger and Wolf, 1974; Futtrel and Kigore, 1969; Simon, 1978), especially *F. moniliforme* (Headrick and Pataky, 1989).

The work described in this study had been designed to evaluate a genetically diverse set of sweet corn inbreds for resistance to *F. moniliforme* infection with either *su1*, *se1* and *sh2* endosperm mutations. It will also identify promising parents on seedling emergence in breeding programs aimed to develop improved sweet corn germplasm.

Material and Methods

1. Plant Material

Seed of 25 sweet corn inbreds (8 *su1*, 8 *se1*, 9 *sh2*) were provided from the sweet corn breeding program in the Department of Horticulture at the University of Illinois (Table 1). Inoculation condition and procedure was detailed in Han (submitted).

2. Germination Test Procedure

Kernels of both the inoculated and control (non-inoculated) ears from each inbred were partitioned visually into 3 groups: 1) kernels without visual sign of the pathogen, 2) kernels with visual sign and 3) kernels with broken pericarps or with signs of other diseases. The percentage of kernels with visual sign and without visual sign for each treatment and inbred was determined by dividing the number of kernels with and without signs of *F. moniliforme* by the total number of kernels. The occurrence of either white or pink mycelium was defined as sign of the pathogen. Kernels that were broken or showed signs of other disease were excluded from consideration in this experiment. Percentages of seeds in each group for each inbred were calculated to provide a unreplicated evaluation of ear susceptibility to inoculation.

A germination test was conducted with both the inoculated and non-inoculated seeds. Control and inoculated seeds were used to isolate the effect of *F. moniliforme* inoculation from other genetic and physiological influences on germination and seedling emergence. The effect of *F. moniliforme* infection was determined by the inoculated by the control treatment.

Three replications of 50 seeds from both treatment of each inbred were tested for germination by using a standard soil germination test (Jangulo, 1988). Fifty seeds were prepared in a ratio of kernels with and without visual sign of the pathogen observed in the bulked seed lot of each inbred and treatment. Ground bed soil [10 parts coarse quartz sand, (Best Sand Co. #620) and three parts silty clay loam soil by volume] uniformly mixed with the aid of a rotary cement mixer was sterilized before use and placed in plastic shoe boxes (Rubbermaid) measuring 31 cm × 165 cm × 9 cm. Fifty seeds were placed on top of 2.5 cm of sterilized soil using a plastic form, precut to the width and length

Table 1. The emergence percentages of each inbred and the influence of inoculation on seedling emergence. Seedling emergence values represent total emergence of both kernels with and without visual sign of *F. moniliforme.*

Inbred		% Seedling Emergence			Influence of inoculation on seedling emergence[y]	
		Control	Inoculated treatment	%△[x]		
IL101T	*sh2*	86	21	65	0.24	a
Ia453	*sh2*	84	37	47	0.45	ab
Oh43	*su1*	71	43	28	0.61	bc
IL784a	*sh2*	84	52	32	0.63	bcd
IL779a	*se1*	28	18	10	0.67	bcde
IL451b	*sh2*	50	33	17	0.68	bcde
IL451b	*se1*	89	60	29	0.69	bcde
C68	*sh2*	90	66	24	0.73	cdef
p39m94	*sh2*	84	63	21	0.75	cdefg
IL451b	*su1*	87	66	21	0.76	cdefgh
P39	*su1*	83	66	17	0.80	cdefgh
Ia453	*su1*	98	80	18	0.81	cdefgh
C68	*su1*	97	80	17	0.82	cdefgh
IL731a	*se1*	91	76	15	0.84	cdefgh
IL678a	*su1*	98	84	14	0.85	defgh
C23	*su1*	86	75	11	0.88	efghi
IL772b	*se1*	98	87	11	0.89	efghi
IL781a	*su1*	92	82	10	0.89	efghi
IL677a	*se1*	82	76	6	0.93	fghi
IL747b	*se1*	71	67	4	0.95	fghi
C40	*sh2*	86	83	3	0.97	fghi
IL775a	*se1*	72	68	4	0.97	ghi
IL678a	*se1*	99	99	0	1.00	hij
Oh43	*sh2*	73	81	-8	1.12	ij
IL442s	*sh2*	51	62	-11	1.24	j
Overall[z]	*su1*	89 b	72 b	17	0.81 a	
	se1	79 ab	69 b	10	0.87 a	
	sh2	77 a	56 a	21	0.76 a	

[x] The change in percent emergence of kernels with inoculation. This value was calculated by subtracting the mean percent germination of kernels of each inbred without sign of the pathogen in the inoculated treatment from the percent germination of kernels of the same inbred without visual sign of the pathogen in the control treatment.

[y] Influence of inoculation on seedling emergence. This value was calculated by dividing the mean percent germination of kernels of each inbred without sign of the pathogen in the inoculated treatment by the percent germination of the same inbreds without visual sign of the pathogen in the control treatment. Means for each inbred followed by dissimilar letters are significantly different at P=0.05 using Fisher's protected LSD test.

[z] Means for each endorsperm group followed by dissimilar letters are significantly different at P=0.05 using Fisher's protected LSD.

dimensions of the containers with 50 holes drilled with even spacing for seed singulation. After placing the seed and removal of the form another 2.5 cm of soil mixture was spread over the seeds. The amount of water necessary to provide 70 % of soil saturation (280 ml) was carefully poured into each container. The boxes were covered with lids and placed in a Percival incubator (Boone, Iowa) at 25℃.

After seven days the boxes were removed and the number of emerged seedlings in each box counted and recorded. The emergence percentages of kernels with and without visual sign for each treatment and inbred were determined since the kernels of each type were separated in each box. The plants from each box were rinsed of soil, surface dried with paper towels and dissected into root, shoot, and kernel tissue and each portion weight. Seedling weight determine by summing the weight of the root and shoot tissue for each inbred and replicate and dividing by the number of seedling in each box.

Result and Discussion

The importance of the proportion of kernels displaying visual sign of the pathogen is related to the quality of the seed and its ability to successfully germinate and emerge from the soil. When averaged over all inbreds, seedling emergence of kernels displaying visual signs of the pathogen in the inoculated treatment was only 31% of the control (data not shown). This percentage was nearly identical for each endosperm class. Kernels of Oh43 *sh2*, IL678a *se1*, and C40 *sh2* with visual signs of the pathogens from the inoculated treatment had 90 to 100% seedling emergence. The seed of these genotypes can successfully germinate and emerge even with visual levels of infection. This represents a second mode of resistance distinct from that observed in previous study (Han, submitted).

Germination and seedling emergence of the inbreds were found to be differentially influenced by prior inoculation of silk with *F. moniliforme* (Table 1). The percent emergence of control seed lots averaged over all inbreds was 82% in comparison with 65% in the inoculated treatment, a 21% reduction. All but two of the inbreds (Oh43 *sh2*, IL442a *sh2*) displayed reduced mean seedling emergence with inoculation. Percent emergence was observed to range among inbreds from 28 to 99% in the control and from 18 to 99% in the inoculated treatment. To isolate only the influence of inoculation, inbred performance of the inoculated treatment was divided by that of the control for each inbred and statistically analyzed (Table 1, column 5). The ratio of inoculation divided by control emergence ranged from 0.24 in IL101T *sh2* to 1.24 in IL442a *sh2*. A t-test run between IL678a *se1*, which displayed no influence from inoculation on seedling emergence, and IL442a *sh2* indicated these lines were significantly different (t-statistic = 7.88, P = 0.048) in their performance. This suggests inoculation with *F. moniliforme* of IL442a *sh2* stimulated the germination and emergence of this seed compared to the control. *F. moniliforme* may serve as a deterrent to kernel invasion by other seed-infection fungi (Rheeder et al., 1990). Isolates of *Rhizopus arrihzus*, *F. oxysorum* and *Aspergillus niger* added to soil enhanced seed germination and growth in greenhouse pathogenicity trials in the presence of *F. moniliforme* (Baird, 1992). This could explain one possible mechanism of the IL442a *sh2* stimulation of germination. The variation in the response of the inbreds to inoculation suggests genetic difference are conditions their susceptibility to the influence of *F. moniliforme* on seedling emergence.

The LSD test (Table 1) also showed the emergence of IL101T *sh2*, Ia453 *sh2*, Oh43 *su1*, IL784a *sh2*, IL779a *se1*, IL451b *sh2*, IL451b *se1*,

C68 *sh2*, and p39m94 *sh2* inoculated seed was significantly reduced compared to their controls without inoculation. Although an LSD test comparing the averaged performance of the inbreds by endosperm classes uncovered no significant differences, six of the nine genotypes with significant reductions in emergence from inoculation were homozygous for *sh2*. Removal of the one *sh2* line (IL442a *sh2*) which displayed stimulation with inoculation from the comparison of endosperm classes resulted in the emergence of the *sh2* lines to be significantly lower than observed in the other endosperm classes. This tentatively suggests inbreds with the *sh2* mutation may be somewhat more susceptible to the effect of colonization by the pathogen on germination and seedling emergence. However, as evidenced by the comparative performance of IL101T *sh2* and IL442a *sh2*, there are other genetic factors aside from the type of endosperm mutation that can significantly affect inbred response to inoculation.

Fresh seedling weight (root plus shoot fresh weight) for each inbred in the control and inoculated treatments and their ratios are calculated (Table 2). Averaged over all the genotypes seedling weights after seven days at 25℃ in the incubator were 8.5% less than the control. Inoculation with *F. moniliforme* did not influence seedling weight as dramatically as it affected the proportion of kernels with visual sign of the pathogen or seedling emergence. Only two genotypes, IL779a *se1* and IL451b *se1* displayed seedling weights significantly influenced by inoculation. The significantly increased seedling weight of only those two lines is difficult to explain and may be due to experimental error and random chance.

Styer and Cantliffe (1984) tested a set of inbreds and commercial hybrids for the incidence of kernel asymptomatic infection by *F. moniliforme*. Using fungal specific media they observed 90% of the genotypes contained *F. moniliforme* and suggested visual undetectable infection by the pathogen may reduce field germination and emergence. To ascertain in this study the importance of asymptomatic kernel infection by *F. moniliforme*, emergence of visually uninfected kernels in the inoculated treatment was divided by the emergence of visually uninfected kernels in the control (Table 3). In this study since the seed lots were not cultured on *F. moniliforme* specific media to evaluate for the presence of the pathogen in kernels without visual sign, significant differences are presumed to be due to asymptomatic kernel infection reduced emergence by only 11.3%. No differences were observed between endosperm classes. The emergence of only three of the inbreds (Oh43 *sh2*, IL747b *se1*, IL442a *sh2*) was significantly influenced by asymptomatic infection. The enhanced emergence of IL442a *sh2* described above and displayed in table 3 appeared to be largely the result of stimulated emergence among asymptomatic kernels. Asymptomatic infection was also responsible for a significant reduction in the emergence of the IL101T *sh2* inoculated seed.

In conclusion, there are second resistance mode after *F. moniliforme* infection. In some cases, *F. moniliforme* even stimulated the germination. Endosperm mutation of sweet corn was not the only factor affecting germination rate, which means there are big variation in it. *F. moniliforme* infection influenced on seedling weight in a minor way. This genetic variation can be adopted in the breeding program in future.

Acknowledgements

This research was supported by Bio-industry Technology Development Program, Ministry for Food, Agriculture, Forestry and Fisheries, and by the LMO environment risk assessment of assign the RDA, Republic of Korea.

Table 2. The fresh seedling weights of each inbred and the influence of inoculation on seedling weight.

Inbred		Seedling weight (mg/plant)[w] Control	Inoculated treatment	%△[x]	Influence of inoculation on seedling weight[y]
IL772b	*se1*	84	55	29	0.66 a
Ia453	*su1*	112	74	38	0.68 ab
C68	*sh2*	61	42	19	0.69 ab
IL678a	*su1*	87	60	27	0.69 abc
Ia453	*sh2*	57	40	17	0.71 acd
IL101T	*sh2*	68	53	15	0.78 abcd
p39m94	*sh2*	38	30	8	0.79 abcd
IL747b	*se1*	71	56	15	0.79 abcd
IL451b	*sh2*	38	29	9	0.80 abcd
IL781a	*su1*	75	63	12	0.83 abcd
IL451b	*su1*	78	69	9	0.89 abcd
IL784a	*sh2*	73	66	7	0.90 abcd
IL775a	*se1*	53	48	5	0.92 abcd
P39	*su1*	88	82	6	0.93 abcd
Oh43	*su1*	63	60	3	0.96 abcd
Oh43	*sh2*	55	53	2	0.96 abcd
C40	*sh2*	59	59	0	1.01 abcd
IL678a	*se1*	79	81	-2	1.04 abcd
C23	*su1*	77	80	-3	1.05 bcd
IL731a	*se1*	61	64	-3	1.07 cde
IL677a	*se1*	68	74	-6	1.10 de
C68	*su1*	64	71	-7	1.11 de
IL442a	*sh2*	41	47	-6	1.16 e
IL779a	*se1*	36	51	-15	1.44 e
IL451b	*se1*	31	68	-37	2.31 f
Overall[z]	*su1*	81 b	70 c	11	0.89 a
	se1	60 a	62 b	-2	1.16 b
	sh2	54 a	47 a	8	0.86 a

[w] Seedling weight consisted of shoot plus root fresh weight.

[x] The change in fresh seedling weight with inoculation. This value was calculated by subtracting mean seedling weights of each inbred in the inoculated treatment from the mean seedling weights of the same inbred without inoculation.

[y] Influence of inoculation on fresh seedling weight. This value was calculated by dividing the mean seedling weight of each inbred in the inoculated treatment by the mean seedling weight of the same inbred without inoculation. Means for each inbred followed by dissimilar letters are significantly different at P=0.05 using Fischer's protected LSD test.

[z] Means for each endosperm group followed by dissimilar letters are significantly different at P=0.05 using Fisher's Protected LSD.

Table 3. The emergence percentages of inoculated and control kernels without visual sign of *F. moniliforme* for each inbred and the influence of inoculation on seedling emergence for kernels without visual sign of the pathogen.

Inbred		% Seedling Emergence (Kernels without sign)			
		Control	Inoculated treatment	%△[x]	Influence of inoculation on seedling emergence[y]
IL101T	*sh2*	87	36	51	0.41 a
Oh43	*su1*	70	50	20	0.72 b
IL451b	*se1*	92	70	22	0.76 bc
p39m94	*sh2*	91	70	21	0.76 bc
C68	*sh2*	91	70	21	0.77 bcd
IL779a	*se1*	27	21	6	0.78 bcd
Ia453	*sh2*	84	65	19	0.78 bcd
IL451b	*su1*	90	71	19	0.80 bcd
IL451b	*sh2*	49	38	11	0.80 bcd
IL784a	*sh2*	88	70	18	0.80 bcd
Ia453	*su1*	99	83	16	0.84 bcde
C68	*su1*	97	83	14	0.86 bcde
C23	*su1*	90	78	12	0.86 bcde
IL731a	*se1*	91	80	11	0.87 bcde
IL678a	*su1*	99	89	10	0.91 bcde
IL775a	*se1*	79	72	7	0.93 bcde
IL781a	*su1*	92	86	6	0.93 bcde
P39	*su1*	89	85	4	0.96 cdef
IL772b	*se1*	98	95	3	0.97 cdefg
C40	*sh2*	86	83	3	0.97 cdefg
IL678a	*se1*	99	100	1	1.01 defg
IL677a	*se1*	82	85	-3	1.04 efgh
Oh43	*sh2*	74	86	-12	1.18 fgh
IL747b	*se1*	73	88	-15	1.21 gh
IL442a	*sh2*	54	67	-13	1.26 h
Overall[z]	*su1*	91 b	78 b	13	0.95 a
	se1	80 a	76 b	4	0.86 a
	sh2	76 a	65 a	13	0.86 a

[x] The change in percent emergence of kernels with inoculation. This value was calculated by subtracting the mean percent germination of kernels of each inbred without sign of the pathogen and the inoculated treatment from the percent germination of kernels of the same inbred without visual sign of the pathogen in the control treatment.

[y] Influence of inoculation on seedling emergence. This value was calculated by dividing the mean percent germination of kernels of each inbred without sign of the pathogen in the inoculated treatment by the percent germination of the same inbreds without visual sign of the pathogen in the control treatment. Means for each inbred followed by dissimilar letters are significantly different at P=0.05 using Fischer's protected LSD test.

[z] Means for each endosperm group followed by dissimilar letters are significantly different at P=0.05 using Fisher's Protected LSD.

Summary

Infected sweet corn seed may increase the incidence of seedling blight and contribute to systemic infection of plants. Inbreds of field or dent corn also had kernels infected with *F. moniliforme*. Although *F. moniliforme* causes a kernel rot, the pathogen is often found in healthy kernels that appear to be physically undamaged. Sweet corn inbreds have been identified that exhibit partial resistance to kernel infection by *F. moniliforme* and good emergence. The work described a genetically diverse set of sweet corn inbreds for seedling emergence to *F. moniliforme* infection with either *su1*, *se1* and *sh2* endorsperm mutations. There were second resistance mode after *F. moniliforme* infection, in some cases, *F. moniliforme* even stimulated the germination.

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