Site of Action of Factors for Resistance to *Fusarium moniliforme* in Maize

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ABSTRACT

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Fusarium moniliforme commonly infects maize (*Zea mays*) kernels. We investigated which kernel tissue was the site where resistance factor(s) were operative by evaluating the parents and reciprocal F_1, F_2, and backcross generations. Differences were conditioned by the genotype of the pericarp, and the genotype of the endosperm, embryo, or cytoplasm had little, if any, effect on the percentage of kernels infected by *F. moniliforme*. Thus, selection for resistance should be most efficient when the pericarp is homozygous, such as at the inbred level.

Fusarium moniliforme Sheld. infects a wide range of crops and causes seedling blight and root, stalk, and ear rots (1,2,7,8) in maize (*Zea mays* L.). It is frequently isolated from maize kernels, but infected kernels frequently appear undamaged (4–6,8,9).

Genotypic differences in maize for asymptomatic kernel infection by *F. moniliforme* (4,5,10) and to kernel rot (3,7) have been reported. King and Scott (5) showed that differences in percentage of healthy-appearing kernels infected with *F. moniliforme* was under genetic control.

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The objective of this study was to determine if the factor(s) for resistance to kernel infection by *F. moniliforme* is operative in the pericarp, endosperm, embryo, or cytoplasm.

MATERIALS AND METHODS

Two inbreds resistant to kernel infection by *F. moniliforme* (Mp317 and SC170) and two susceptible lines (Mp68:616 and Mp303) were chosen. The following populations were produced by hand pollination: P_1 (resistant), P_2 (susceptible), $P_1 \times P_2$, $P_2 \times P_1$, $P_1 \times (P_1 \times P_2)$, $P_2 \times (P_1 \times P_2)$, $(P_2 \times P_1) \times P_1$, $(P_2 \times P_1) \times P_2$, $(P_1 \times P_2)F_2$, and $(P_2 \times P_1)F_2$ in 1981 and 1982. Seed produced on a given generation is one generation advanced from the plant on which it was produced. Thus, the F_1 seed evaluated was produced on the inbred parent. Therefore, the pericarp of the F_1 seed is genetically identical to the female parent.

Hand-pollinated ears were harvested, dried, and shelled in normal procedures. One hundred kernels from each generation were surface-disinfected by being immersed in 70% ethyl alcohol, soaked for 3 min in 1.6% NaOCl, and rinsed in sterile distilled water. Kernels were then placed on Czapek solution agar (Difco) in petri plates, 10 kernels per plate. After 4 days at 28 C, kernels were examined for the presence or absence of *F. moniliforme*. Each test was repeated and the testing time considered as replicate. Data on the percentage of infected kernels were subjected to a standard analysis of variance.

For a separate test, seed of reciprocal F_1 crosses between some resistant and susceptible inbreds were planted in single-row plots in the field and plants were allowed to pollinate at random. A randomized complete block design with three replicates was used. About 10 ears from each plot were harvested, dried, and shelled. A random sample of 100 kernels from each field plot was evaluated for kernel infection by *F. moniliforme* using the procedure described.

RESULTS AND DISCUSSION

Resistance to kernel infection by a fungus could result from factors in the cytoplasm or nuclear factors operative in the pericarp, endosperm, or embryo. Assuming, for convenience, that resistance is conditioned by one gene (for illustration, A denotes allele for resistance) or by the cytoplasm (arbitrarily designated X and Y), we listed the genotype and cytoplasm for each generation (Table 1).

If the genotype of the pericarp affects the incidence of kernel infection by *F. moniliforme*, then the different generations divide into three groups. That is, when P_1 was used as a female, the P_1, $P_1 \times P_2$, and $P_1 \times (P_1 \times P_2)$ generations would all be expected to be equally resistant. Comparable generations involving P_2 as the female parent would all be expected to be equally susceptible.

Table 1. Genotype and cytoplasm of parents and reciprocal F_1, F_2, and backcross generations when the parents have different cytoplasms and also differ for a single nuclear gene

	Tissue					
	Pericarp		Endosperm		Embryo	
Generation	Genotype[y]	Cytoplasm[z]	Genotype[y]	Cytoplasm[z]	Genotype[y]	Cytoplasm[z]
P_1 (resistant)	AA	X	AAA	X	AA	X
P_2 (susceptible)	aa	Y	aaa	Y	aa	Y
$P_1 \times P_2$	AA	X	AAa	X	Aa	X
$P_2 \times P_1$	aa	Y	aaA	Y	Aa	Y
$P_1 \times F_1$	AA	X	AAA	X	AA	X
			AAa		Aa	
$P_2 \times F_1$	aa	Y	aaA	Y	Aa	Y
			aaa		aa	
$(P_1 \times P_2)F_2$	Aa	X	AAA	X	1 AA	X
			AAa		2 Aa	
			aaA		1 aa	
			aaa			
$(P_2 \times P_1)F_2$	Aa	Y	AAA	Y	1 AA	Y
			AAa		2 Aa	
			aaA		1 aa	
			aaa			
$(P_1 \times P_2) \times P_1$	Aa	X	AAA	X	AA	X
			aaA		Aa	
$(P_1 \times P_2) \times P_2$	Aa	X	AAa	X	Aa	
			aaa		aa	
$(P_2 \times P_1) \times P_1$	Aa	Y	AAA	Y	AA	Y
			aaA		Aa	
$(P_2 \times P_1) \times P_2$	Aa	Y	AAa	Y	Aa	Y
			aaa		aa	

[y]Denotes allele for resistance.
[z]Arbitrarily designated X and Y.

Table 2. Percentage of *F. moniliforme*-infected kernels of different generations involving a resistant and a susceptible parent

	Cross						
	Mp317 ×				SC170 ×		
	Mp68:616		Mp303		Mp68:616	Mp303	
Generation	1981	1982	1981	1982	1982	1982	Mean[y]
P_1 (Mp317 or SC170)	19	18	19	30	10	8	17 b
P_2 (Mp68:616 or Mp303)	59	91	92	95	84	95	85 a
$P_1 \times P_2$	19	9	13	44	9	6	17 b
$P_2 \times P_1$	78	84	86	78	69	85	80 a
$P_1 \times (P_1 \times P_2)$	15	19	32	22	92	8	16 b
$P_2 \times (P_1 \times P_2)$	87	96	99	97	64	29	79 a
$(P_2 \times P_1) \times P_1$	11	25	11	43	NT[z]	25	...
$(P_2 \times P_1) \times P_2$	15	9	26	15	8	17	15 b
$(P_1 \times P_2)$ F_2	3	30	24	29	19	38	25 b
$(P_2 \times P_1)$ F_2	NT	22	NT	35	37	36	...
LSD (0.05 level)	15	9	8	9	5	12	14
C.V. (%)	19	10	7	8	7	16	11

[y]Means not followed by the same letter differ significantly ($P = 0.05$) according to Duncan's multiple range test.
[z]NT = not tested.

Generations produced on the F_1 would be expected to be equal among themselves, but the relative level of kernel infection compared with the other two groups would vary depending on the type of gene action involved.

Our data (Table 2) indicate that the site of action of genes for resistance to *F. moniliforme* is the pericarp because generations produced on P_2 were significantly more susceptible than the other generations. The only exception was the Mp303 × (SC170 × Mp303) cross in 1982.

If genetic factor(s) for resistance are operative in the endosperm, we would also expect differences when the resistant parent was used as the female compared with when the susceptible parent was used. Additionally, the expected level of resistance would be: $(P_2 \times P_1) \times P_1 > F_2 > (P_2 \times P_1) \times P_2$.

Reciprocal F_1 crosses should not differ if the genetic factor(s) for resistance is operative in the embryo. We obtained large differences between reciprocal F_1 crosses.

Reciprocal F_2s should differ when factor(s) for resistance are operative in the cytoplasm. Except for the SC170 × Mp68:616 reciprocal F_2s, we did not obtain evidence for cytoplasmic effects in 1982 (Table 2). Unfortunately, we did not have the reciprocal F_2s in 1981; however,

Table 3. Percentage of *F. moniliforme*-infected kernels on reciprocal F_1 crosses grown in the field and allowed to pollinate at random

	Female parent	
Pedigree	Resistant	Susceptible
SC170 × CI90C (Res. check)	20	...
Mp440 × Mp303 (Susc. check)	...	75
CI90C × Mp68:616	14	13
CI90C × Mp440	12	19
Mp317 × Mp440	18	30
Mp317 × Mp303	31	44
SC170 × Mp68:616	21	23
SC170 × Mp440	19	22
SC170 × Mp303	42	28
Mean[a]	23	25
LSD (0.05 level) = 15.6		
C.V. (%) = 34.6		

[a]Does not include values of checks.

we evaluated reciprocal F_2s (open pollination) for seven crosses (Table 3) and found no evidence that the cytoplasm affects kernel infection by *F. moniliforme*.

We have found that the genotype of the pericarp conditions resistance to kernel infection to *F. moniliforme*. We acknowledge, however, that these results would also be expected if the site of action of genes for resistance occurs outside of the pericarp. For instance, if the mode of entry of *F. moniliforme* is through the silk, the actual site of gene action could be in the silk. Until the mode of entry of this fungus into the corn kernel has been determined, however, the conclusion that the site of action of genes for resistance is in the pericarp seems appropriate.

We could speculate that resistance to kernel infection is dominant because the generations produced on the F_1 parent were equal to the generations produced on the resistant parent, but more data are needed before such a conclusion would be substantiated.

These findings that the genotype of the pericarp conditions response to kernel infection by this fungus dictate that a selection and breeding program for resistance must be handled differently than with other diseases that affect the plant rather than the seed. That is, when we evaluate the F_1 for response to a disease that affects the plant itself, we plant the F_1 seed that was produced on the inbred parent and evaluate that plant for response to the disease. To evaluate the F_1 for pathogens that invade the seed, however, we have to evaluate directly the seed that was produced on that inbred parent. Thus, it appears that selection for resistance would be most efficient on inbreds rather than F_1s. If two resistant inbreds have the same genes for resistance, then the seed produced on the F_1 plant (F_2 seed) would be resistant. If the genes for resistance differed, however, the level of resistance in the seed produced on the F_1 plants would be dependent on the type of gene action involved.

LITERATURE CITED

1. Christensen, J. J., and Wilcoxson, R. D. 1966. Stalk rot of corn. Monogr. 3. Am. Phytopathol. Soc., St. Paul, MN. 59 pp.
2. Futrell, M. C., and Kilgore, M. 1969. Poor stands of corn and reduction of root growth by *Fusarium moniliforme*. Plant Dis. Rep. 53:213-215.
3. Gulya, T. J., Jr., Martinson, C. A., and Loesch, P. J., Jr. 1980. Evaluation of inoculation techniques and rating dates for Fusarium ear rot of opaque-2 maize. Phytopathology 70:1116-1118.
4. Ikenberry, R. W. 1961. The isolation of *Fusarium moniliforme* Sheld. from corn kernels. Proc. Iowa Acad. Sci. 68:100-102.
5. King, S. B., and Scott, G. E. 1981. Genotypic differences in maize to kernel infection by *Fusarium moniliforme*. Phytopathology 71:1245-1247.
6. Koehler, B. 1942. Natural mode of entrance of fungi into corn ears and some symptoms that indicate infection. J. Agric. Res. 64:421-422.
7. Smith, F. L., and Madsen, C. B. 1949. Susceptibility of inbred lines of corn to Fusarium ear rot. Agron. J. 49:347-348.
8. Thomas, M. D., and Buddenhagen, I. W. 1980. Incidence and persistence of *Fusarium moniliforme* in symptomless maize kernels and seedlings in Nigeria. Mycologia 72:882-887.
9. Tuite, J. 1961. Fungi isolated from unstored corn seed in Indiana in 1956-1958. Plant Dis. Rep. 45:212-215.
10. Warren, H. L. 1978. Comparison of normal and high-lysine maize inbreds for resistance to kernel rot caused by *Fusarium moniliforme*. Phytopathology 68:1331-1335.