

# Relationship Between Host Maturity and Reaction to the Eyespot Disease Caused by *Kabatiella zae* Narita and Hiratsuka on Maize<sup>1</sup>

## ABSTRACT

Twenty-two inbreds and the crosses on three tester lines (MS153, W64A, B84) with 14 inbreds were artificially inoculated with *K. zae*. Data on proportion of disease (X) for each genotype were collected periodically, transformed by the Gompertz equation ( $-1n - \ln(X)$ ), and regressed in time. The k values (regression coefficients) differed statistically among genotypes. Genotypes with the largest k values had the highest levels of disease. A regression analysis of the k values on days to tasseling yielded negative regression coefficients that were significant for the inbreds and for each group of crosses with the three testers. Early maturing genotypes were more susceptible, yet it was clear that factors other than maturity conditioned susceptibility. The reaction of the F<sub>1</sub> crosses to the disease varied according to the susceptibility of the tester lines, regardless of maturity.

## INTRODUCTION

The literature about *K. zae* and eyespot disease is very limited. Genotype reactions to the pathogens are usually contained in noncitable reports from maize pathologists and breeders. Some authors (1, 5, 14) have observed an apparent positive relationship between early maturity and susceptibility to the disease.

These observations, though only casual, should be substantiated, as variety selection is of major importance in those areas and circumstances that favor the development of the eyespot disease.

Eyespot disease was observed to develop slowly early in the season in some host genotypes, but then spread rapidly in the upper leaves later in the season (1, 3). The apparent sudden change in the level of disease was thought to be related to a change

## COMPENDIO

Se encontró una correlación entre la tasa de incremento de la enfermedad causada por *K. zae* y el tiempo a la floración en maíz. Esta respuesta se observó tanto en líneas puras como en sus híbridos; genotipos tempranos (precoces) fueron normalmente más susceptibles a la enfermedad. Sin embargo, algunos genotipos se mostraron más resistentes (o susceptibles) que lo esperado basándose exclusivamente en su precocidad, lo cual indica que existieron otros factores que condicionaron la respuesta del huésped al hongo. La reacción a la enfermedad de cruces de líneas puras con tres líneas de prueba varió de acuerdo a la susceptibilidad de la línea de prueba. Los cruces con MS153 (resistente) fueron más resistentes que los cruces con W64A (susceptible) a pesar de ser ambos grupos de cruces similares en cuanto a precocidad. Así mismo, los cruces con B84 (intermedio en resistencia) fueron más susceptibles y tardíos que los cruces análogos con MS153.

in the susceptibility of some genotypes (5), but further studies are needed to confirm this assumption.

The objectives of this study were to establish the relationship between early maturity and susceptibility to the eyespot disease, and to determine whether or not some host genotypes change in susceptibility to eyespot disease during the growing season.

## MATERIALS AND METHODS

Several experiments were conducted from 1982 to 1984 at three sites near Ames, Iowa.

A group of 23 maize inbreds was self-bred and crossed to three tester lines (Table 1). The tester lines were MS153, which was intermediate in maturity and resistant to the eyespot disease; W64A, which was near MS153 in maturity but susceptible; and B84, which was late in maturity but intermediate in its reaction to the disease.

### Inoculum production and inoculation

Cultures of *K. zae* were obtained by isolation of the fungus on potato dextrose agar from naturally infected leaves in the field. Inoculum was produced

<sup>1</sup> Received for publication 20 June 1986.

Portion of a thesis submitted by the author to the Graduate College of Iowa State University as partial fulfillment of the requirements for the Ph.D. degree. The author is grateful to Dr. C.A. Martinson for his help and advice in the realization of this work.

\* Laboratorio de Patología Vegetal, Facultad de Agronomía, Universidad de Costa Rica. San José, Costa Rica

on potato dextrose broth following a procedure developed by Dr C.A. Martinson (personal communication). *K. zeae* was grown on potato dextrose broth that consisted of 1 000 ml of potato infusion (200 g of diced potatoes autoclaved for 30 min in one liter of distilled water, then strained through several layers of cheese-cloth and readjusted to one liter), 20 g of glucose and about 10 mg of antifoam AF (Dow-Corning Inc.). The broth was poured into two 500 ml flasks (250 ml/flask) and autoclaved for 15 min. Each flask was seeded with a plug from a young culture (9-10 days old) of *K. zeae* and agitated on a new Brunswick RB-25 reciprocal shaker at room temperature (23-25°C).

After two weeks of growth, the contents of the flasks (primarily stromatic hyphae with very few conidia) were centrifuged at 16 300 x g for 3 min and the supernatant discarded. The pellet from each flask was blended and mixed with 2 kg of fine quartz sand (Martin Marietta grade 37) and air-dried. The inoculum was poured into the whorl of the plants in the evening at a rate of about 1 g/plant.

#### Isolation plots

Each host genotype was planted in isolation plots that consisted of four rows that were 3 m long, with 76 cm between rows. The distance between plants in a row was about 25 cm. Plots were arranged in a completely randomized block design with three replications and were bordered by four rows of a hybrid which was tall and fairly resistant to the eyespot disease. The plants in the border rows adjacent to isolation plots of inbred lines were trimmed to the height of the inbred.

#### Disease assessment

Data on the progress of the disease was taken on two leaves, a middle (10th) and an upper (3rd-4th below the tassel) leaf in five plants per plot. The same leaves on the same plants were sampled throughout the experiment. The percentage of diseased tissue / leaf was estimated visually according to a scale of with 0.01, 0.1, 1, 5, 16, 31, 50 and 61 percent of diseased tissue/leaf. The lower values of the scale up to 5% were estimated assuming that under a severe attack about 10 000 lesions can develop on a leaf.

#### Field experiments (1982)

Two experiments were carried out in 1982. The first one was the initial screening of 24 inbreds (Table 1) that were planted in isolation plots on June 1 and June 3 at two different planting sites. Isolation

plots were bordered by the hybrid Mo17 x B73Ht. Inoculation was done when plants were at the 6-7th leaf stage on July 4 and July 12 at the two sites.

In a second experiment, the inbred lines were self-bred and crossed with three tester lines at one location (Table 1).

#### Field experiments (1983)

A selected group of inbreds and hybrids obtained in 1982 were planted in isolation plots on May 12 and May 27 at two different sites (Table 1). The isolation plots were surrounded by the hybrid Pioneer 3713. Plants were inoculated at the 5-6th leaf stage on July 20 and August 1 at the two sites.

#### Field experiments (1984)

Only the F1 crosses were planted at one site (Table 1) on May 15. The hybrid H99 x A632 was planted as the border variety. Inoculation was done on June 17 when plants were at the 6th leaf stage.

#### Statistical analysis

For each experiment in the different locations, the data on proportion of disease for each replication (mean of five plants/plot) were transformed using the Gompertz equation (2) and individual regression analyses were done by leaf position for each genotype.

The Gompertz equation (2) was chosen over the logistic equation (15) based on a previous study by the author (5) where the Gompertz transformation described the eyespot data on proportion of diseased tissue more accurately than the logistic transformation. During these experiments, the data on proportion of disease for randomly chosen replications for some genotypes were transformed using both logistic and the Gompertz transformations. The Gompertz transformation usually gave a better fit of the data to the regression line, particularly for the initial data points.

The k values (slopes) were statistically compared to determine differences among genotypes. Data (k values) coming from different locations and years were also put together to compare the overall performance of the genotypes when evaluated over different environments. The value of k was not calculated when only one reading on amount of disease was available.

The relationship between the reaction to the eyespot disease and maturity was evaluated by doing

Table 1. Maize inbred lines and F<sub>1</sub> crosses used to study the relationship between host maturity and reaction to the eyespot disease.

Code	Inbreds	Year of experiments				
		1982	1983	1983-1984		
		Inbreds		Crosses with		
			MS 153	W64A	B84 <sup>1</sup>	
A	MS153	*			*	*
B	W117	*	*	*	*	*
C	A662	*	*	*	*	*
D	CM7	*	*	*	*	*
E	CM105	*				
F	W153R	*	*	*	*	*
G	Co109	*	*	*	*	*
H	W64A	*	*	*		*
I	A661	*	*	*	*	*
J	H99	*				
K	Oh43	*	*	*	*	*
L	C123	*	*	*	*	*
M	A619	*	*	*	*	*
N	B70	*				
O	A632	*				
P	B73	*	*	*	*	*
Q	Mo17	*	*	*	*	*
R	B14A	*	*	*	*	*
S	B37	*	*	*	*	*
T	R117	*				
U	B84	*	*	*	*	*
V	Va35	*	*	*	*	*
X	H95	*				

1 In 1984 the crosses of B84 with W117 and B37 were not planted because of insufficient seed.

simple regression analyses of the k values on days to tasseling. An individual analysis was done for each planting site and also combined analyses over environments (sites and years).

## RESULTS

### Reaction of maize inbreds to *K. zea*

#### Disease severity on the middle leaves

The first visible symptoms of eyespot disease appeared in all inbreds eight and seven days after inoculation in 1982 and 1983 respectively. Differences among inbreds in the response to the disease were apparent in the first reading on disease severity. During 1982, the inbreds MS153, A661 and A632 began with less than 4% of diseased tissue in the middle leaves, in contrast to A662, W64A, CM7 and

A619, which had 8% or more of diseased tissue. In 1983, most genotypes began with less than 11% of diseased tissue on the middle leaves. During late August, the disease had increased to about 9% in MS153, in contrast to W117, A662, Co109 and CM7, where more than 50% of the middle leaves were affected by eyespot lesions.

The disease severity in B84 was always intermediate between that of W64A and MS153. In one experiment in 1982, W64A had six times more diseased tissue than MS153 and three times more than B84 during late August. These three inbreds were used as tester lines in the testing of the hybrid combinations in 1983 and 1984.

During 1983, a prolonged drought extended through most of the growing season. The lack of adequate rainfall caused a very slow increase of the

disease in all genotypes. The highest disease rating recorded for MS153 was about 1.4% of diseased tissue. Other inbreds –W117, CM7 and Co109– reached readings of 30% or more.

#### Disease severity in the upper leaves

The disease was not detected in the upper leaves of MS153 until early September, 1982 and was absent in these leaves in 1983. Symptoms also appeared comparatively late in the upper of B37, H95, B70, Mo17, B84 and Va 35. During middle August, 1983 the inbred MS153 was still free of disease and the percentage of diseased tissue in most genotypes was low (1% or less). The inbreds W117, A662, CM7 and Co109, however, all had 15% or more diseased tissue by that time. In 1983, only W117 and Co109 had more than 10% diseased tissue in the upper leaves.

In the majority of the inbreds, the disease severity was lower in the upper leaves and the final disease severity was often higher in the middle leaves.

#### Rate of disease progress

The rate of disease increase (k values) differed statistically among host genotypes in both the 1982 and 1983 experiments; however, the k values were normally lower in 1983 than in 1982.

An overall mean k value (including sites and years) for each leaf position was calculated for all 17 selected inbreds that were used in hybrid combinations in 1983 and 1984 (Table 2). The most susceptible genotypes (Co109, A662, W117, W153R, and CM7) had mean k values that were about three times or more greater than the mean k value for MS153, which was considered one of the most resistant inbreds. The mean value for W64A was always larger than that for B84 and MS153, and all were statistically different for both the middle and upper leaves (Table 2). The inbreds with the higher k values in both upper and middle leaves were the same ones that had the highest levels of diseased tissue throughout the growing cycle.

#### Inbred maturity and susceptibility to eyespot

The relationship between reaction to the eyespot disease and host maturity was evaluated by using simple regression analysis of the k values on days to tasseling. There was a highly significant tendency for k values to be negatively correlated with days to tasseling (Table 3). This response was observed for both the upper and middle leaves. A representation of this relationship is given for the 23 inbreds grown in 1982 (Fig. 1). The slope of the regression line was significant (Table 3), yet the k values for some

inbreds diverged greatly from the regression time. The regression equations for the upper and middle leaves were nearly identical (Table 3).

#### Reaction of maize hybrids to *K. zeae*

##### Disease severity in the middle leaves

Symptoms of eyespot appeared in all hybrids seven days after inoculation in 1983. The incubation period could not be determined precisely during 1984 because of inclement weather, but it was estimated to be between 8 and 9 days.

The first reading of disease severity reflected the response of the hybrids to the inoculation with *K. zeae*. The initial amount of disease was greater among hybrids with W64A than in crosses with MS153 and B84 in 1983. The crosses of W177, A662, and CM7 with any of the testers tended to have more initial disease than the other crosses in 1983. In 1984, the initial level of disease on the middle leaves was fairly uniform among all the crosses.

Table 2. Rate of eyespot disease increase (k values) in the middle and upper leaves of 17 inbreds grown in 1982 and 1983.

Inbreds	Middle leaf	Upper leaf
CO109	0.041747 a <sup>a</sup>	0.060723 a
A662	0.034095 b	0.032137 d
W117	0.034752 b	0.042174 b
W153R	0.030775 b	0.032309 d
CM7	0.033952 b	0.037994 c
W64A	0.025711 c	0.027862 e
A619	0.025069 cd	0.032942 d
C123	0.020660 de	0.010710 fg
B84	0.015249 f	0.014936 f
B37	0.016186 ef	0.009763 g
Va35	0.012848 fg	0.003324 h
Mo 17	0.012420 fg	0.010247 g
Oh43	0.014260 fg	0.010697 fg
B14A	0.011681 fg	0.12504 f
A661	0.014240 fg	0.011196 g
B73	0.01155 fg	0.009731 g
MS153	0.009642 g	0 h

a Mean of 12 values obtained for three replications at two planting sites in both 1982 and 1983. Means in a column followed by the same letter are not statistically different according to Duncan's Multiple Range Test ( $P=0.05$ ).

Table 3. Regression of rate of disease development (k values) on maturity; combined GLM<sup>1</sup> analysis for the 17 inbreds grown in 1982 and 1983.

Source	DF	Middle leaf			MS <sup>b</sup>	Upper leaf	
		MS <sup>2</sup>	F	R <sup>2</sup>		F	R <sup>2</sup>
Maturity	1	1 050	25***	0.62	2 279	17***	0.53
Error	15	42			134.5		
			I			T	
Intercept	0.791338		6.8***		0.106071	5.1***	
Maturity	-0.000920		-5.0***		-0.001355	-4.1***	

1 General linear model analysis for the means of 12 k values calculated for 3 replications at two sites in both 1982 and 1983.

2 X 10<sup>-6</sup>.

\*\*\* Significant (P = 0.001).

The progress of the disease, after the initial lesion development, varied greatly among genotypes. The severity of the disease varied according to the tester line and there were also differences among the hybrids with the same tester line. The crosses with the inbreds W117, A662 and CM7 normally had more diseased tissue on the middle leaves than any other cross at any reading date. The inbreds that supported the least amount of disease were those containing the inbreds B37, B73 and A661. The amount of disease was always highest in the crosses with W64A and lowest in crosses with MS153. During early August, 1984 the reading for the crosses of A662 with MS153, B84 and W64A respectively were 19.5%, 23% and 30% of diseased tissue in the middle leaves.

#### Disease severity in the upper leaves

The disease developed poorly in the upper canopy of the plants in 1983 due primarily to an extended drought during most of the growing season. Several of the crosses with MS153 never developed lesions in the upper leaves, and in other MS153 crosses the disease appeared very late in the season. The most susceptible crosses with MS153 were those with W117, A662 and CM7, and the proportion of diseased tissue in the upper leaves only reached 2% or less. The crosses of the two inbreds W117 and A662 with W64A had up to 13% and 19% respectively of the upper leaf tissue affected by eyespot by early September. All of the crosses with W64A and B84 eventually developed some eyespot disease. The severity, however, was greatly influenced by the inbred crossed onto these tester lines. The proportion of diseased tissue varied from 1% to nearly 30%.

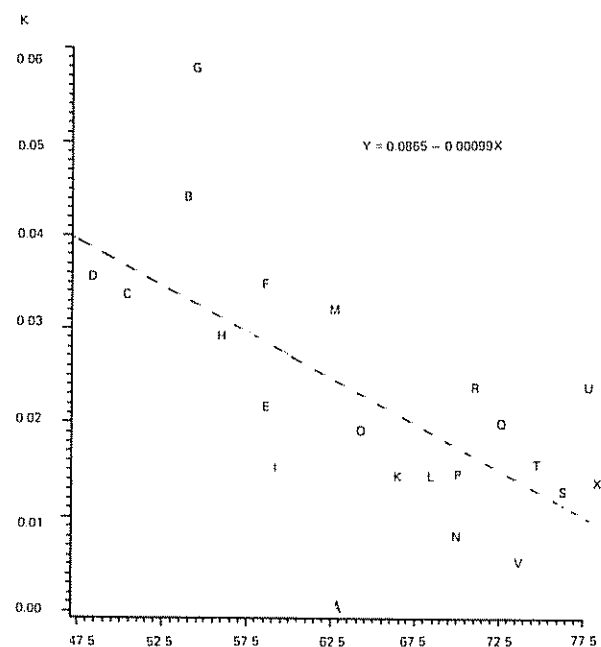


Fig. 1. Relationship between host maturity and the rate of eyespot disease increase (k) in the upper leaves of 22 corn inbreds grown at two locations in 1982 (code for inbreds in Table 1).

The growing season of 1984 was also characterized by a long dry period, but there was more frequent rainfall in July 1984 than in 1983. Overall, the weather was more conducive for eyespot development in 1984 than in 1983, but still the disease severity was very low in the upper leaves of most hybrids.

### Disease severity on crosses among tester lines

Data on proportion of disease was also taken for the crosses among the three tester lines (MS153 x W64A, MS153 x B84 and W64A x B84). The amount of diseased tissue in both the upper and lower leaves of diseased tissue in both the upper and lower leaves was always greatest for B84 x W64A at every year and site. The disease severity was least for MS153 x B84.

### Rate of disease progression

The k values for both the upper and middle leaves of the crosses of the different inbreds with any one of the three tester lines were statistically different (Tables 4, 5). The middle leaf k values for the crosses of CM7, Co109, A619, W117 and A662 with a tester line were normally the highest among the crosses with that particular common parent (Table 4). However, there were often significant differences among these five susceptible lines, and frequently the k values were not significantly different from several other hybrids with lower k values. The hybrids with B73, B37, Va35, Mo 17 and Oh43 usually had comparatively low k values, particularly for the upper leaves of the crosses with MS153 (Table 5).

The k values for the middle leaves of crosses from one tester did not differ as much as those for the

upper leaves. In general, the most resistant hybrids, based on k values of the middle leaves, were the most resistant crosses based on the upper leaf k values. None of the hybrids seemed to change greatly in susceptibility based upon middle leaf versus upper leaf k values. When averaged over all the inbreds, the crosses with MS153 had the lowest k values for both middle and upper leaves. Conversely, the crosses with W64A normally had the greatest k values (Tables 4, 5).

### Hybrid maturity and susceptibility to *Kabatiella zae*

The regression analysis of the k values for each individual genotype on days to tasseling yielded negative regression coefficients that were significant for each group of crosses with the three tester lines (Tables 6, 7). The change in k per date of maturity was three times greater for the upper leaves than for the middle leaves. The regression coefficient was the highest for the W64A crosses with both leaves, and these crosses also had the highest correlation coefficients (Table 6). However, the differences among the regression coefficients for the three testers were non-significant. A graphic presentation of the relationship between the mean k values (1983 and 1984 experiments combined) and days to tasseling is presented for the upper leaves of the crosses with W64A (Fig. 2).

Table 4. Rate of eyespot disease increase (k values) in the upper leaves of the crosses of 14 inbreds with three tester lines planted in both 1983 and 1984.

Inbreds	MS153	W64A	B84
W117	0.024475 a <sup>1</sup>	0.039013 a	— <sup>2</sup>
CM7	0.023031 a	0.037781 ab	0.027864 b
A662	0.020565 a	0.040148 a	0.034479 a
A619	0.009966 b	0.027515 c	0.020722 c
CO109	0.009705 b	0.034997 ab	0.032139 ab
Oh43	0.005759 bc	0.018371 d	0.012990 de
B14A	0.003221 cd	0.018751 d	0.018279 cd
B37	0.003181 cd	0.014943 de	—
A661	0.002190 cd	0.016444 d	0.013883 de
C123	0.001844 cd	0.026073 c	0.013151 de
W153R	0	0.031990 bc	0.015050 de
B73	0	0.015484 de	0.004535 f
Mo17	0	0.009654 e	0.013139 de
Va35	0	0.002670 f	0.010342 e

1 Means of 9 values calculated for 3 replications in 3 different environments; means in a column with the same letter are not statistically different according to Duncan's Multiple Range Test (P=0.05).

2 Hybrid not planted because of insufficient seed.

Table 5. Rate of eyespot disease increase (k values) on the middle leaves of crosses of 14 inbreds with three tester lines planted in both 1983 and 1984.

Inbreds	MS153	W64A	B84
CM7	0.021618 a <sup>1</sup>	0.026273 a	0.022449 ab
CO109	0.019124 ab	0.026373 a	0.024716 a
W117	0.017682 bc	0.023653 ab	—
A662	0.017620 bc	0.022882 abc	0.022449 ab
A619	0.017318 bc	0.02201 bcd	0.022417 ab
C123	0.016279 bcd	0.017601 eb	0.018576 cc
W153R	0.016177 cd	0.023522 ab	0.019657 bc
A661	0.016063 cd	0.019878 cde	0.019192 bc
B37	0.015445 cd	0.018693 de	—
B14A	0.015402 cd	0.018991 de	0.018731 bc
Va35	0.015059 cd	0.018006 e	0.018221 c
Mo17	0.014181 d	0.019417 cde	0.019132 bc
Oh43	0.013598 d	0.018084 e	0.018784 bc
B73	0.013393 d	0.018693 de	0.018318 c

1 Mean of 9 values calculated from 3 replications in 3 different environments during 1983 and 1984. Means in a column followed by the same letter are not statistically different according to Duncan's Multiple Range Test ( $P=0.05$ ).

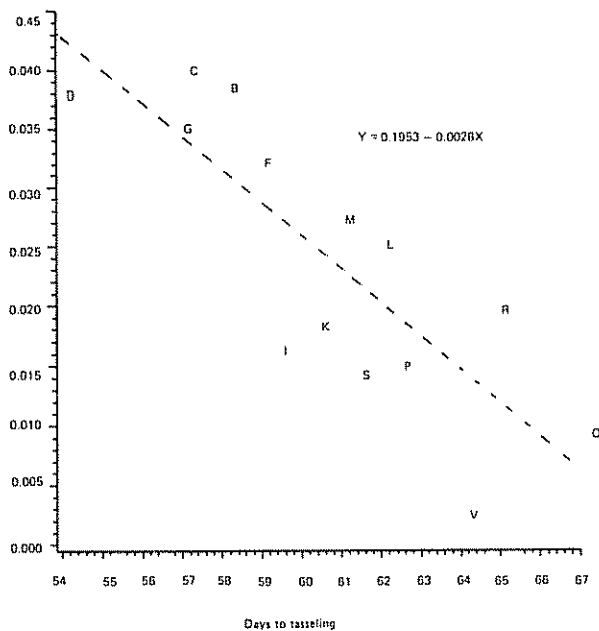


Fig. 2. Relationship between host maturity and the rate of eyespot disease increase (k) on the upper leaves of the crosses of 14 corn inbreds with W53A (code for hybrids in Table 1).

#### DISCUSSION

Arny *et al.* (1) observed that, although conditions late in the season seemed to favor the development of

the eyespot disease, resistance was more commonly present in late maturity host genotypes. In this work, a statistically significant relationship was observed between the reaction to the disease and host maturity expressed as days to tasseling. This response was found in both inbreds and hybrids; early maturing genotypes tended to be more susceptible to the disease. The relationship, however, was not strong, as illustrated by the low correlation coefficients. This was particularly true for the inbreds where some diverged greatly from the regression lines (Figs. 1, 2). This indicates that factors other than maturity condition susceptibility, and that the inherent genetic resistance is obviously the most important one. Additional evidence for the independence of maturity and reaction to the eyespot disease came from the study of the crosses of the inbred lines with the tester lines. The crosses reacted more in accordance with the susceptibility of the testers than their maturity. Crosses with the resistant inbred MS153 were always more resistant than crosses with the susceptible inbred W64A, in spite of a similar maturity. Crosses with B84 were more susceptible and later in maturity than the analogous crosses with MS153.

A late season eyespot epidemic in the upper canopy of the plants of some hybrids has been observed by some authors (1, 3, 5). Observations by Arny *et al.* (1) and Cassini (3) led them to suggest that the leaves may become more susceptible after they are fully expanded. No experimental proof has

Table 6. Regression<sup>1</sup> of disease reaction (k values) on maturity; combined analysis for the crosses of three tester lines with 14 inbreds planted in three environments in 1983 and 1984.

Crosses with	Leaf	DF		MS <sup>2</sup>		F	R <sup>2</sup>	Intercept	T	Regression coefficient	T
		Maturity	Error	Maturity	Error						
MS153	Upper	1	12	682.4	29.5	23 ***	0.66	0.146068	5.1***	-0.00228	-4.8 ***
MS153	Middle	1	12	41.4	1.9	21.7***	0.64	0.05023	6.9***	-0.0005567	-4.7 ***
W64A	Upper	1	12	1282.5	43.9	29 ***	0.71	0.195282	6.2***	-0.002824	-5.4 ***
W64A	Middle	1	12	78.5	3.2	24.3***	0.67	0.063547	7.4***	-0.0006984	-4.9 ***
B84 <sup>3</sup>	Upper	1	10	51.98	394.3	7.6***	0.43	0.133818	3.2***	-0.001786	-2.75*
B84 <sup>3</sup>	Middle	1	10	38.0	3.7	10.2***	0.50	0.056428	5.0***	-0.0005544	-3.2 ***

1 GLM analysis for the means of nine k values calculated for three replications at three planting sites.

2 X 10<sup>-6</sup>.

3c Only the 12 crosses planted in both 1983 and 1984 are included

\* Significant (P = 0.05)

\*\*\* Significant (P = 0.001)

been presented. Our observations of several eyespot epidemic have suggested a phenomenon that could be involved in the different responses to the disease by early and late genotypes.

Early genotypes produce most of their leaves early in the season when the environment is normally more favorable for an eyespot epidemic (cool, wet conditions (1, 3, 12)). The infected lower leaves provide an effective source of inoculum for the upper portion of the plant through the production of conidia that are dispersed by splashing rain (12). Late genotypes have a more extended vegetative stage and therefore

the upper leaves may escape infection when the environment becomes less conducive to the epidemic, as happened in 1983 and 1984 when a drought extended through most of July and August. In a year without extended periods of unfavorable weather, the disease may progress through continuous reinfections. The observed differences in susceptibility between some early and late hybrids might disappear under those conditions, and the disease could develop extensively in the upper leaves later in the season. The possibility of an actual change in resistance in some genotypes when they reach a certain growth stage cannot, however, be discarded.

Table 7. GLM analysis (1983 and 1984 experiments combined) of the k values calculated for the crosses of three tester lines with 14 corn inbreds.

Source	DF	Middle leaf		Upper leaf	
		MS <sup>1</sup>	F	MS <sup>1</sup>	F
Environment	2	4219.5	53***	32889.7	2115***
Rep.	2	64.8	0.81 <sup>ns</sup>	71.6	4.6*
Testers	2	8714.2	109***	836.2	53***
Maturity	1	2042.4	256***	1403.1	90***
Mat * testers	2	34.5	0.43 <sup>ns</sup>	9.1	0.58 <sup>ns</sup>
Error	350	79.6		15.6	

1 X 10<sup>-6</sup>.

\* Significant (P = 0.05)

\*\*\* Significant (P = 0.001).

ns: not significant.



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## Notas y Comentarios

### Una trampa para la mosca tsetse

Una nueva técnica para combatir la mosca tsetse (*Glossina morsitans*), las ha erradicado virtualmente en un sitio experimental localizado en Zimbabwe, Africa. La técnica podría conducir a abandonar métodos burdos de controlar poblaciones de insectos, tales como aspersiones masivas con plaguicidas desde el aire o el suelo.

Las moscas tsetse transmiten protozoarios parásitos que son una causa mayor de muertes y enfermedades en animales domésticos en Africa. En 1985, investigadores del Tsetse Research Laboratory, de la Universidad de Bristol, Inglaterra, y del Department of Veterinary Services, en Zimbabwe, desarrollaron una nueva técnica para matar moscas tsetse (*New Scientist*, 7 de noviembre de 1985, p. 40). La técnica involucra a científicos que colocaron un número de trampas, pedazos cuadrados de tela oscura, que, para las

tsetses, semejaban vacas y con la adición de sustancias atractivas, olían como vacas. Las moscas aterrizaban sobre los cuadrados de tela, sólo para absorber una dosis fatal de insecticida.

Desde entonces, los investigadores han establecido ensayos de campo en Zimbabwe en una área de sabana que alcanza a cubrir un territorio de 600 kilómetros cuadrados. En octubre de 1987, Tony Jordan, director del Laboratorio en Bristol, presentó los resultados de los ensayos en una reunión del tropical Research Institute en Londres (*New Scientist*, 116(1586):31).

Según Jordán, las moscas tsetse fueron virtualmente eliminadas colocando cuatro de estas "vacas" trampa por kilómetro cuadrado en el área en uno de los estudios. Los investigadores planean continuar los ensayos de campo en una zona más grande, de unos 2 000 kilómetros cuadrados. A.G.

### Causas de la popularidad de la fruta Kiwi en Europa

Como ya es notorio, en los últimos años se ha producido la introducción de una nueva fruta en el mercado europeo y ha recibido una gran aceptación. El "kiwi", una oscura planta, conocida sólo en China como planta de jardín, por sus hojas grandes, ha sido explotada en Nueva Zelandia en gran escala como fruta de exportación. La fruta, *Actinidia deliciosa* es verde hasta en el interior de sus frutos y llena hasta el tope de vitamina C. El nombre chino, que equivalía a grosella china, parecía a muchos inapropiado y sin atractivo, y fue cambiado por el de "kiwi", nombre de un pájaro corredor, *Apteryx oweni*, que es una especie de emblema nacional en Nueva Zelandia.

Nueva Zelandia es el principal exportador de la fruta, la cual se ha sembrado en pequeña escala en otras partes, principalmente en Estados Unidos. Países como España, Israel y Brasil, que luchan por exportar sus productos a Europa son los más activos. La exportación de Nueva Zelandia fue de 48 000 toneladas en 1984, principalmente a Alemania Occidental y Japón, los que toman una cuarta parte de la exportación neozelandesa. La cantidad comerciada es relativamente pequeña. Por ejemplo, la producción mundial de manzanas es de 37 millones de toneladas al año, la de bananas y cítricos mayor todavía, siendo todas superadas por la uva, de la que se produjeron 65 millones de toneladas en 1983. La producción planeada de kiwi en Nueva Zelandia es de 300 mil toneladas en 1991 (partiendo de 62 mil en 1984). Pero, lo importante no es el tamaño de la industria sino su ritmo de crecimiento. Desde cero como producto de exportación, la demanda ha crecido tanto que no se está abasteciendo satisfactoriamente.

¿Cuál es la razón de este éxito? El kiwi no es la más vistosa de las frutas. Se le ha comparado como "un verdusco y veloso huevo de gallina" y una "bola de tenis vieja", lo que puede dar una idea de su aspecto exterior. No tiene los colores vivos y atractivos de las manzanas, naranjas o fresas. Pero lo que le falta a la fruta en apariencia exterior, lo recupera en su interior con una espectacular combinación de color y sabor. El *Wall Street Journal* la ha descrito así: "...córtese el kiwi y adquiere una vida propia. Subitamente, todo es verde eléctrico y fragante, con una explosión de un sol amarillo rodeado de anillos violeta. El kiwi partido es una fruta encantadora".

El kiwi debe parte de su demanda, y sus precios altos, a su único sabor, el que depende de la influencia olfatoria de más de 25 compuestos. Pero hay algo más. Un conjunto de tres armas químicas han ayudado a la fruta en el campo de batalla dietético. Una le da su atractiva fisonomía verde, la segunda le confiere una reputación saludable, y la tercera, la hace útil en la cocina.

El color esmeralda es debido a la clorofila. Es raro para las frutas retener la clorofila en su madurez

(piénsese en las bananas, naranjas y manzanas). Otra fruta verde en su madurez es el aguacate.

La imagen saludable del kiwi depende mucho de su masivo contenido de vitamina C. Las cifras varían, pero un contenido usualmente dado al cultivar Hayward es de 105 miligramos por 100 gramos de pulpa comestible (la fruta pesa unos 100 gramos. Esto es el doble del contenido de vitamina C de frutas no tropicales de gran consumo, como los cítricos y las fresas. Es superado, sin embargo, por el cas de Costa Rica (*Psidium friedrichstahlianum*), la guayaba (*Psidium guajaba*), que tienen alrededor de 200 miligramos por 100 gramos, y por la sorprendente acerola (*Malpighia glabra*), que ha rendido en análisis de la fruta madura hasta 1700 mg/100 g. (INCAP 1969).

La tercera arma química del kiwi es una proteasa, una enzima que digiere proteínas. La enzima, llamada actinidina, es similar a la papaína, que viene de la papaya, a la bromelina, que proviene de las piñas, y la ficina, que se extrae del latex de la higuera. La función de estas sustancias en la naturaleza es algo oscura; quizás estén destinadas a evitar ataques de hongos y de insectos. Pero no hay tal incertidumbre sobre su papel en la cocina, donde las frutas que contienen proteasas son muy apropiadas para la laboriosa tarea de suavizar la carne.

Un último, y no menos importante, atractivo del kiwi es que ha abierto el campo para nuevas frutas exóticas en Europa, Canadá y Japón. Aunque el aguacate y el mango son bastante conocidos, aunque no al nivel de los grandes supermercados como ha ocurrido con el kiwi, hay otras frutas a las que han dirigido sus miradas los importadores (y, en consecuencia, los exportadores). Con la destreza de los neozelandeses para promover nuevos productos exportables, ahora parece que están prestando su atención al tamarillo o tomate de árbol (*Cyphomandra betacea*), introducida hace tiempo de Iberoamérica a ese país, y que se reporta que se ha vendido ya en algunos lugares de Inglaterra para probar su aceptación.

Mientras tanto, muchas otras frutas de América del Sur y Central están sometidas en Nueva Zelandia al escrutinio científico del DSIR (Departamento de Investigaciones Científicas e Industriales). Entre estas plantas está la ampliamente cultivada y deliciosa chirimoya (*Annona cherimola*); el "babaco", un híbrido natural de dos miembros del grupo de la papaya (*Carica* spp.), la "feijoa" (*Feijoa sellowiana*), una pariente de la guayaba; el pepino dulce (*Solanum muricatum*), que se cultiva en América del Sur; el suave y dulce zapote blanco (*Casimiroa edulis*), cultivado ya en Florida e Italia; y, por último, la lúcuma (*Lucuma biferá*), esa sapotácea que ha demostrado en los países andinos que es casi insuperable en la confección de helados.

Sólo el tiempo dirá cuáles de estas candidatas al estrellato repetirán el notable éxito del kiwi. A.G.