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Inheritance of resistance to *Helminthosporium maydis* blight in maize (*Zea mays* L.)

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Summary. The nature and mode of inheritance of resistance to *Helminthosporium maydis* blight was investigated in two maize varieties, 'RbU-W' and 'DIC'. The study of F_1, F_2, and reciprocal backcross populations of crosses between these two varieties on the one hand and two susceptible varieties, 'UVE' and 'ZPSc-58c' on the other, revealed that resistance in the two varieties is monogenic recessive. The genes for resistance in the two varieties are allelic. Resistance was shown to be a lesion-type and measurements revealed that it operated through reduced lesion size and lesion number.

Key words: Lesion-type resistance – Recessive – Allelic

Introduction

The first recorded work on the inheritance of disease reaction to *H. maydis* Nisik and Miyake is that of Ullstrup (1941) who reported that susceptibility is inherited as a monogenic recessive trait. Pate and Harvey (1954) observed a wide range of reactions which suggested polygenic inheritance. Similar conclusions were drawn by Jenkins and Robert (1952) for *H. turcicum* blight and Hooker et al. (1970) and Hooker (1972) for *H. maydis* blight. Van Eijnatten (1961) suggested that resistance to the Nigerian strains of *H. maydis* is recessive and governed by several genes. In another work van Eijnatten (1961a) deviced a scoring key based on the measurements of lesion size and lesion type. Craig and Daniel-Kalio (1968) described chlorotic lesion resistance which was shown to differ only in lesion size and not in number from the susceptible state. This kind of resistance is not expressed in the seedling stage (Orillo 1953; van Eijnatten 1961).

Craig and Fajemisin (1969) reported chlorotic lesion resistance in maize cultivar '024-2-4' which was controlled by two linked recessive genes. Smith and Hooker (1973) reported a single recessive gene for resistance in the same source.

Chlorotic lesion resistance to races O and T is expressed in reduced lesion number, lesion type (resistant lesions are small, brown and chlorotic while susceptible lesions are brown to tan, elongate and lack chlorosis), lesion size, depressed fungus sporulation, and delay in necrosis (Craig and Daniel-Kalio 1968; Hooker et al. 1970; Hilu and Hooker 1963). The most significant feature in chlorotic lesion resistance, therefore, is the long period required for spore development and the negligible number of spores formed in the chlorotic lesions.

The main objective of this work was to determine the nature and the mode of inheritance of resistance to *H. maydis* blight in 'RbU-W' and 'DIC'. The effect of the disease on grain yield was also monitored.

Materials and methods

The materials for this study and their characteristics are shown in Table 1. F_1 seeds and seeds from selfed plants from the late season harvest were planted in December 1979. F_2 and backcross pregenies were raised. Evaluation was done in April 1980. A total of 33 entries comprising F_1's, backcrosses, F_2's and the varietal inbreds were assessed in a Randomized Complete Block Design of three replicates. A complete fertilizer (80 kg N/ha as ammonium sulphate, P at the rate of 40 kg/ha as single super phosphate and K at the rate of 40 kg K_2O/ha as potassium chloride) was applied two weeks after planting, when half the amount of N and all the P and K were used, and the rest of the Ammonium sulphate was applied at tasseling.

Inoculation

Inoculum was raised on potato dextrose agar (PDA) from infected maize leaves. Inoculation was done 19 days after planting, between 4.30 p.m. and 6.30 p.m., by discharging

Table 1. Varieties used in the study and their general characteristics. *FARZ* = Federal Agric Research *Zea*; *'RbU-W'* = Rust and blight resistant, upright leaves, white grained; *'UVE'* = 'upper Volta early'; *'DIC'* = 'Diacol V153'; *NCRI* = National Cereals Research Institute, Ibadan, Nigeria

Variety	Origin	Disease reaction[a]	Grain type and colour	FARZ no.	Genetic make-up	(Days to 50% taselling)
'RbU-W'	NCRI	Highly resistant	Floury flint; white	–	Composite	53
'DIC'	NCRI	Resistant	Floury flint; white	FARZ 6	Synthetic	55
'UVE'	Upper Volta	Susceptible	Flint, yellow	–	Open-pollinated variety	42
'ZPSc-58c'	Yugoslavia	Very susceptible	Floury flint, yellow		Yugoslav hybrid	39

[a] According to Fajemisin (1978)

inoculum into the leaf whorl of every plant. Lesions started to develop five days after inoculation.

Disease rating

Disease rating was done at the mid-silking stage. Individual plants were scored for disease reaction using three parameters: disease intensity, cover, and lesion size.

Disease intensity is an absolute measure of disease assessing the total aspect of the plant in a vertical manner. It uses a modified version of the 1 to 5 scale (Nelson 1973). In this modified system, the basal part of plant below the ear is taken as half of the maize plant. The two halves are visually divided into 'quarter parts'. The leaves are carefully examined in each 'quarter-part' and the area taken over by disease is estimated as a proportion of total leaf area. This value is multiplied by the corresponding number on the 1–5 scale and the value is summed stepwise on the plant to give the disease intensity scores.

Cover was measured on the flag leaf. A random sample of flag leaf measurements gave a mean length of 80 cm. Eight cards were numbered 1 to 8 to correspond to 8 segments, each 10 cm long, on the flag leaf. These cards were drawn with replacement, and for each plant a 5 cm^3 transparency with 10 random points was applied to the segment indicated by the card drawn. The number of points falling on lesions were read off and converted to area – 1 point represents a cover of 10%. Cover was entered as a mean of 3 random measurements.

Lesion size was entered as a mean of 4 random lesion measurements on the flag leaf. Lesion size and cover measurements were repeated 85 days after planting on the resistant varieties.

Results

Resistant lesions are small, brown and chlorotic while susceptible lesions are brown to tan, elongate and lack chlorosis. Table 2 shows the four varieties and their mean disease scores. The intensity scores are quite high for 'ZPSc-58c' and 'UVE'. Cover is correspondingly high – a cover of 82.7% to an intensity score of 4.7 in 'ZPSc-58c'. The scores for 'DIC' and 'RbU-W' are consistently low.

Mean intensity for 'ZPSc-58c' is significantly different from the mean intensity for 'UVE' at the 0.01 probability level. Mean lesion size, however, presents an opposite picture. There is no significant difference between the mean scores for each disease rating in 'RbU-W' and 'DIC'. No difference was detected when measurements were taken 85 days after planting. The resistant and susceptible varieties have quite distinct disease ratings as Table 2 shows. The trend persists even in segregating populations – lesion size was therefore made the criterion for grouping into disease reaction classes.

Inheritance studies

The F_1 hybrids of the 'RbU-W' × 'UVE', 'RbU-W' × ZPSc-58c', and 'DIC' × 'UVE', 'DIC' × ZPSc-58c'

Table 2. Varieties used in the study, their mean disease scores and yield

Variety	Mean intensity	Mean cover	Mean lesion size (mm)	Corrected grain wt (g)[a]
'RbU-W'	0.01 ±0.001	0.059±0.024	1.32±0.06	42.84±2.40
'DIC'	0.034±0.01	2.13 ±0.73	1.42±0.29	52.7 ±4.9
'ZPSc-58c'	4.7 ±0.52	82.7 ±1.49	17.40±0.40	2.49±1.27
'UVE'	3.02 ±0.03	36.30 ±0.02	10.76±0.54	43.7 ±4.7

[a] As a mean of 30 plants

crosses (Table 3) were all susceptible, indicating that resistance in 'RbU-W' and 'DIC' is recessive. The F_1 progenies of 'RbU-W' × 'DIC' were resistant while the F_1 progenies of 'UVE' × ZPSc-58c' were all susceptible.

The progenies of the backcrosses ('RbU-W' × 'UVE') × 'UVE', and ('RbU-W' × 'ZPSc-58c') × 'ZPSc-58c' were susceptible but the progenies of their reciprocal backcrosses i.e. ('RbU-W' × 'UVE') × 'RbU-W' and ('RbU-W' × 'ZPSc-58c') × 'RbU-W' segregated in a resistant : susceptible ratio of 1 : 1 (Table 4). The backcrosses with 'DIC' also segregated in similar ratios. As the data of Table 5 show the populations from the crosses of resistant and susceptible varieties segregated in a 1 : 3 ratio.

Tests of allelism

The F_1 hybrids of 'RbU-W' and 'DIC' were resistant (Table 3), and the F_2 population of this cross did not segregate for susceptibility. The backcrosses did not segregate either. These data indicate that the two resistant cultivars have the same recessive gene for resistance.

Table 3. Reaction to *Helminthosporium maydis* of F_1 progenies of crosses of maize cultivars

	No. of plants		
	Resistant	Susceptible	Total
'RbU-W' × 'UVE'	0	80	80
'UVE' × 'RbU-W'	0	73	73
'RbU-W' × 'ZPSc-58c'	0	65	65
'ZPSc-58c' × 'RbU-W'	0	59	59
'DIC × UVE'	0	66	66
'UVE' × 'DIC'	0	63	63
'DIC' × 'ZPSc-58c'	0	52	52
'ZPSc-58c' × 'DIC'	0	40	40
'DIC' × 'RbU-W'	64	0	64
'RbU-W' × 'DIC'	61	0	61
'UVE' × 'ZPSc-58c'	0	75	75
'ZPSc-58c' × 'UVE'	0	62	62

Table 4. Reaction to *Helminthosporium maydis* of progenies from reciprocal backcrosses of maize cultivars

Cross	No. of plants		
	Resistant	Susceptible	χ^2 1 : 1
('RbU-W' × 'DIC') × 'DIC'	101	0	–
('RbU-W' × 'DIC') × 'RbU-W'	107	0	–
('RbU-W' × 'UVE') × 'RbU-W'	65	58	0.398
('RbU-W' × "UVE') × 'UVE'	0	123	–
('RbU-W' × 'ZPSc-58c') × 'ZPSc-58c'	0	123	–
('RbU-W' × "ZPSc-58c') × 'RbU-W'	64	55	0.680
('DIC' × 'UVE') × 'UVE'	0	110	–
('DIC' × 'UVE') × 'DIC'	61	52	0.716
('DIC' × 'ZPSc-58c') × 'ZPSc-58c'	0	107	–
('DIC' × 'ZPSc-58c') × 'DIC'	42	49	0.538
('UVE' × 'ZPSc-58c') × 'ZPSc-58c'	0	102	–
('UVE' × 'ZPSc-58c') × 'UVE'	0	116	–

Table 5. Classification of F_2 plants from the crosses of different maize cultivars for their reaction to *Helminthosporium maydis*

Cross	No. of plants			
	Resistant	Susceptible	Total	χ_2 1 : 3
'RbU-W' × 'ZPSc-58c'	25	96	121	1.215
'RbU-W' × 'UVE'	27	91	118	0.273
'DIC' × 'UVE'	30	69	99	1.485
'DIC' × 'ZPSc-58c'	26	96	122	0.885
'RbU-W' × 'DIC'	102	0	102	–
'ZPSc-58c' × 'UVE'	0	116	116	–

Discussion

'RbU-W' and 'DIC' have been shown by statistical analysis to be equally resistant. Intensity is probably a more realistic measure of disease on 'ZPSc-58c' because of the difficulty in obtaining typical and isolated lesions. 'ZPSc-58c' is therefore judged more susceptible than 'UVE'.

The susceptibility of all the crosses between resistant and susceptible cultivars indicates that resistance in 'RbU-W' and 'DIC' is recessive. Results from the backcrosses and F_2 populations show that resistance in these two cultivars is governed by a single recessive gene. Reactions of F_1 and F_2 populations from the crosses between resistant parents also indicate that the genes conditioning resistance in the two resistant cultivars are allelic. The non-significance of reciprocal differences in disease scores in F_1's indicates a lack of cytoplasmic effects. The observable increase in scores of reciprocals may be due to modifying factors which are equally effective in 'ZPSc-58c' and 'UVE'.

The results of this work show that resistance in 'DIC' and 'RbU-W' is the lesion-type. The results agree with the work of Craig and Daniel-Kalio (1968). There is also agreement with the work of Smith and Hooker (1973). It has also been shown that lesion size and cover measured at 60 and 85 days after planting did not differ appreciably. For chlorotic lesions of the order of 1.2 mm, the method of assessing cover is actually a good estimate of the number of lesions in an area of 5 cm^2. This work therefore provides nonpathological evidence that chlorotic lesion resistance operates by reducing lesion number and consequently the photosynthetic area taken over by disease. From the practical standpoint, selection for homozygous plants will be easy in a backcross programme.

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