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 F₁, F₂, and reciprocal backcross populations of crosses between these two varieties on the one hand and two susceptible varieties, 'LIVE' 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 (1961) devised 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. F₁ seeds and seeds from selfed plants from the late season harvest were planted in December 1979. F₁ and backcross pregenies were raised. Evaluation was done in April 1980. A total of 33 entries comprising F₁'s, backcrosses, F₁’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 K₂O/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...
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**