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Review

Breeding for resistance to downy mildews and stalk rots in maize *

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Summary. The present review includes information on distribution, symptoms, inoculation techniques, disease rating, sources of resistance, genetics of resistance, breeding approaches for resistance, and the present status of resistance breeding with respect to *Sclerophthora* and *Peronosclerospora* downy mildews and *Erwinia, Cephalosporium* and *Fusarium* stalk rots. Some suggestions highlighting research gaps pertinent to future breeding strategies are mentioned.

Key words: *Zea mays* – Maize – Downy mildew – Stalk rot – Disease resistance

Introduction

In maize, chemical disease control is uneconomical except in connection with seed dressing. Even such a procedure takes a long time before being adopted by many cultivators who save their own seed. Breeding for disease resistance is the main approach for improving and stabilizing yield, and disease resistance is an essential facet of a total improvement programme.

Germplasm evaluation and resistance breeding in maize started in several Asian, African and Latin American countries in association with CIMMYT and the Inter-Asian Corn Programme (IACP). A wide variety of maize germplasm originating in Asia, Africa and the Western Hemisphere have been exposed to pathogens on a multi-location basis in most of the agroclimatic zones of the world, including India. A systematic population improvement programme, particularly for downy mildew and/or stalk rot resistance also began on a comprehensive basis in the early seventies, particularly at Pantnagar and Delhi (India), Farm Suwan (Thailand), Los Banõs (Philippines) and Texas A. and M. University, Texas (USA).

Disease spread, symptoms and rating systems

1 Sclerophthora downy mildew (= Brown stripe downy mildew; S. rayssiae var. 'zeae' Payak and Renfro)

This disease occurs in India, Nepal, Thailand, Pakistan and Bangladesh (Frederiksen and Renfro 1977). It has been most severe in areas that receive 100–200 cm of precipitation and has caused up to 63% yield loss (Payak et al. 1970b; Nene and Saxena 1970). Disease symptoms have been observed only on leaves (Fig. 1). In the initial stages, the lesions start developing on lower leaves as narrow chlorotic or yellowish stripes, 3–7 mm wide but variable in length. The stripes extend in parallel fashion, have well-defined margins and are limited by veins. The presence of a downy, whitish to creamy growth, usually as stripes on the ventral surface of the infected leaves, is the most characteristic symptom. Later, these chlorotic stripes turn brown and give a burnt appearance. Any sort of deformation, either vegetative or floral, has not been noticed in infected plants.

Artificial epiphytotic conditions can be created by using powdered infected maize leaves containing oospores, collected during the preceeding season and placing them in the furrows just before planting. Inoculum can also be prepared by collecting infected leaves supposed to be full of oospores from early plantings of maize of the same season, drying the leaves

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and making powder out of the debris. Inoculum should be placed in furrows in such a manner that seeds are in touch with the inoculum. Artificial epiphytotic conditions can also be created by putting 2–3 cm pieces of freshly infected leaves containing sporangia of the fungus in the whorls of the seedlings. This should be done in cloudy weather, between 5 and 7 p.m., two to three times and between 2–4 weeks after planting. In experimental plots, where disease occurs year after year, only this method is sufficient for creating epidemics. In areas of low disease incidence, more than one method of inoculation may be used to obtain better results (Lal 1982 b).

Scoring of individual maize germplasm can be done by evaluating/observing all the plants of a row(s) using a 1–5 index scale, where, 1 = no infection; 2 = light infection, a few scattered to moderate number of stripes on lower leaves; 3 = moderate infection, abundant stripes on lower leaves and few on middle leaves; 4 = heavy infection, stripes abundant on lower and middle leaves extending to upper leaves; and 5 = very heavy infection, stripes abundant on all leaves, no cob formation, plants may be killed prematurely. Based on this rating scale, plants with a 1 to 2 score can be considered highly resistant, with 2 to 3 moderately resistant, with 3 to 4 moderately susceptible, and with 4 to 5 highly susceptible.

2 Peronosclerospora downy mildews (sorghum, Philippine and sugarcane downy mildews or P. sorghi (Weston and Uppal) Shaw, P. philippinensis (Weston) Shaw and P. sacchari (Miyake) Shirai and Hara)

This group of diseases is reported to be caused by seven species of *Peronosclerospora* in different parts of the world (Frederiksen and Renfro 1977). The most common species occurring in the tropics are *P. sorghi, P. philippinensis* and *P. sacchari.* Heavy losses in maize have been recorded from one or the other of the pathogens in the Philippines, Taiwan, Indonesia, Thailand, India, West Africa, Venezuela, Japan, Australia, Europe, North America and other parts of the world (Bonde 1982). The losses may be as high as 100% in some fields. All species of *Peronosclerospora* have a number of characters in common. They all induce systemic infection in the plant and some cause local lesions as well. They are incapable of systemically infecting plants older than approximately 4 weeks. The most important disease symptom is striping or chlorosis of leaves (Figs. 2 and 3). Other symptoms are stunting, narrowing of leaves, phyllody of floral parts and barrenness. Environment and host genotype also interact to modify symptoms. Therefore, the critical examination of these fungi is very important for their identification.

Artificial epiphytotic conditions can be created by planting spreader rows of a susceptible maize cultivar 20 days prior to planting around and at every fifth row of test material. For inoculation of the spreader rows as well as test material, diseased leaves should be collected from infected plants at 1 a.m. and the conidia washed off in a plastic bucket containing rain water to prepare the conidial suspension. This conidial suspension can be inoculated on maize seedlings by spraying with a sprayer at 2 a.m. The first inoculation should be made at the 2 leaf stage, followed by 2–3 more inoculations on alternate days (Lal et al. 1979). The plot can be diseased once or preferably reused year after year. In such a case, the infected plants should be chopped at harvest time to increase the level of the primary inoculum. About 40 days after planting, infected and healthy plants should be recorded and per cent infection may be calculated as indication of degree of resistance.

3 Erwinia stalk rot (E. chrysanthemi cv. 'zeae' Victoria, Arboleda and Munoj)

It is one of the most important diseases of maize in tropical countries. It occurs in India, Nepal, Bangladesh, Philippines, Thailand, Zimbabwe, Israel, Greece and South Africa (Saxena 1982). At first, the upper leaves show signs of wilting. The basal internodes become soft and discoloured, lower leaf sheaths and leaves covering these internodes also become discoloured and yellow and the rind becomes pale-straw losing its natural green colour (Figs. 4 and 5). With the advancement of the infection, the pith is completely destroyed leaving the bundles in a disorganised state and the stalk becomes extremely soft and pliable. The cobs may result in drooping and eventually such plants may collapse as the stalks topple down rapidly. A mild sweet but somewhat disagreable fermenting odour comes from the infected plants.

For creating good epidemics of *Erwinia* stalk rot, planting should be done early in the season, i.e., May–June, so that the flowering coincides with the period of frequent rains. This will result in good disease development. A virulent isolate of *E. chrysanthemi* cv 'zeae' should be selected for inoculation. To maintain the virulence of the bacterium, it should be inoculated on healthy plants and then reisolated every year before mass inoculation. The inoculum should be increased for mass inoculation on nutrient broth for 48 h at 30 °C. The inoculum may be diluted 10 times with sterile water to maintain a concentration of approximate $1 \times 10^{7-9}$ bacteria/ml. The inoculation may be carried out when the crop is at the pre-silking stage or until flowering has reached 75%. To inoculate the plants, a diagonal hole is made in the middle of second internode from the ground to the pith. One millilitre of bacterial suspension is injected into the plant through that hole by a hypodermic syringe. If necessary, a second inoculation may be done one week later in the third internode from the ground.

The inoculated plants in a plot may be rated individually for their disease reaction using a 1–5 index scale (for precise in-

formation) or wilted and healthy plants in plot are counted after 15 days of inoculation. The observations may be recorded near the dry silk stage of the crop. During recording, the plants are cut from the ground in such a way that the first basal internode is intact. The plants are split open longitudinally from the first internode upward to clearly observe the spread of the disease in the internal tissues before rating (Lal 1982a). In rating, 1 = the infection limited to a very small spot in the pith at the site of inoculation; 2 = disease infection spread in the pith and cortical tissues, rind not infected; 3 = disease infection covered the entire length of the inoculated internode but not crossing the nodal plates; rind green and symptoms not visible externally, but plants showing sign of wilting; 4 = infection crosses nodal plates of inoculated internode and affects adjacent internodes; plant wilted; ear length and size considerably reduced as compared to healthy plants; and 5 = disease spread over three or more internodes; pith, cortical tissues and vascular bundles rotted and disorganised, rind discoloured, plant wilted and may be toppled down. Plants with disease rating 1 and 2 should not show any external symptom of disease and should be classified as resistant. Disease rating 3, 4 and 5 are to be classified as susceptible. The average rating of all inoculated plants in a plot can be used for comparative analysis (Singh 1979). For evaluation of germplasm, wilted and healthy looking plants should be counted 15 days after inoculation and percentage disease calculated. Germplasm taking less than 5 per cent infection may be considered highly resistant; 5–10% as resistant; 10–25% as moderately susceptible; and more than 25% as susceptible to highly susceptible.

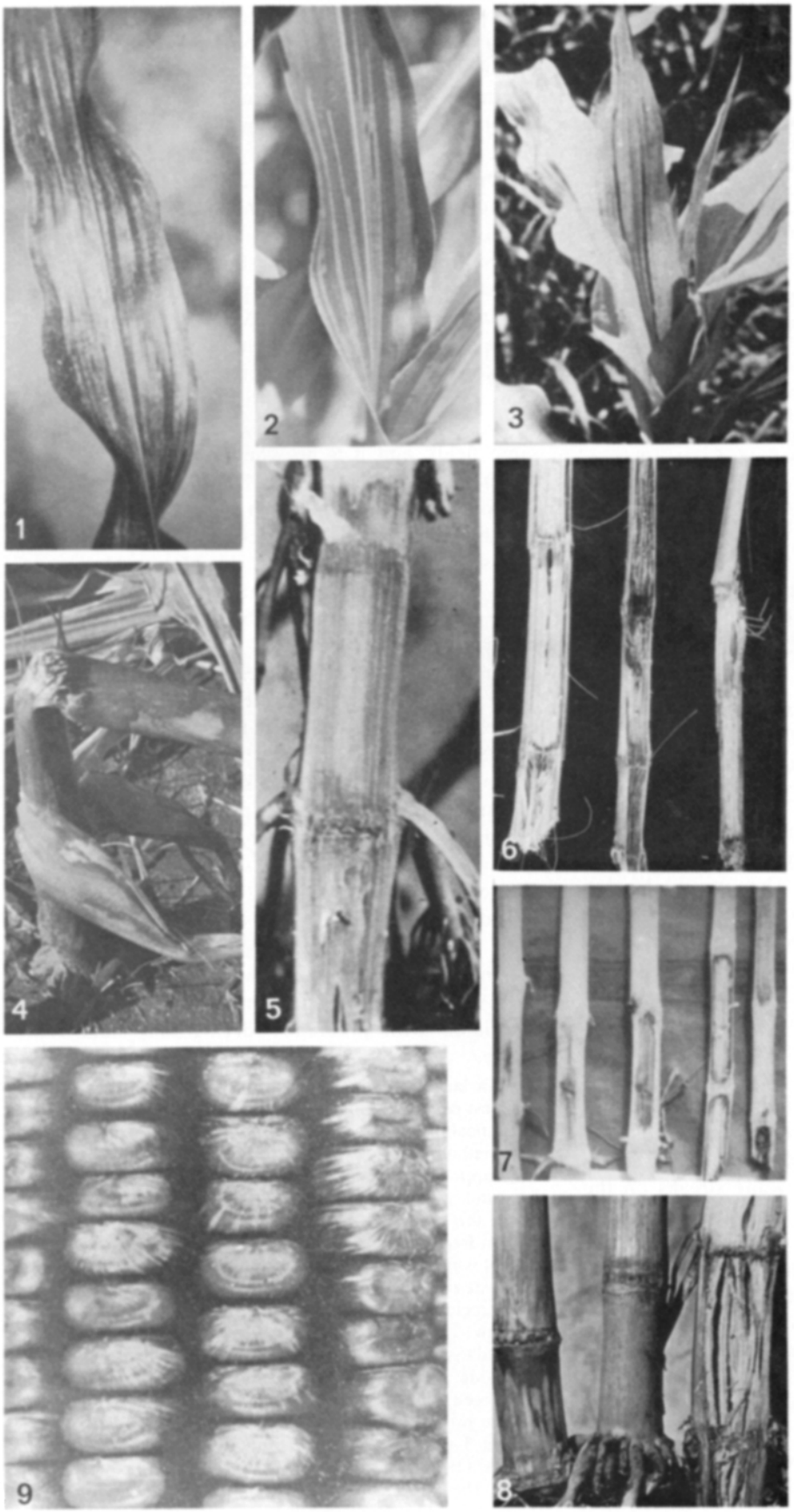
4 Cephalosporium and Fusarium stalk rots (black bundle, late wilt and dry stalk rot or Cephalosporium acremonium Corda, C. maydis Samra, Sabet and Hingorani and Fusarium spp.)

These stalk rots are of world-wide importance and are one of the most serious and destructive group of maize diseases (Lal and Dwivedi 1983). They mostly develop and predominate in the post-flowering stage of the crop. Black bundle has been reported to occur in the tropics of Tanzania, Pakistan and India (Christensen and Wilcoxson 1966). The main distinguishing character of the disease is the presence of blackened vascular bundles which may extend through several internodes and nodes (Fig. 6). Other important symptoms of this disease are a reddening or purpling of the leaves and stalks, lesions on basal portion of the stalk, multiple ear formation at nodes and excessive tillering. Sometimes, kernels show conspicuous white streaks on the pericarp (Fig. 9).

Late wilt has been observed in Egypt and India (Sabet et al. 1961; Samra et al. 1962, 1966; Payak et al. 1970a; Jain et al. 1975). This disease has not been reported so far from any other country. It is essentially a vascular disease of maize stalk occurring in the tropics (Renfro and Ullstrup 1976). The pathogen kills the plants prematurely at the flowering stage. Infected plants do not show symptoms until they reach the tasselling stage and then start wilting from the top leaves. The leaves are at first dull green, then turn yellow and eventually become dry. The vascular bundles in the stalk are reddish-brown and the nodes also turn to the same colour. In advanced stages, the lower internodes become dry, shrunken and hollow. Frequently, a wet rot develops in the lower part of stems (Fig. 7).

Dry stalk rot of maize caused by species of *Fusarium* is widely distributed throughout the world (Koehler 1960). *F. moniliforme* is able to cause the premature death of plants (Koehler and Boewe 1957). It has been proved to be a more virulent pathogen as compared to *F. oraminearum* (Koch and Murwin 1945). *F. moniliforme* frequently causes comparatively more damage in tropical as compared to temperate countries (Cappellini 1956; Wernham 1959; Christensen and Wilcoxson 1966). Sometimes, both pathogens affect plants in the same fields, and it is no wonder why the two or additional diseases have often been confused. This has led to several false reports in the past, and diagnoses are still confounded (Mace et al. 1981). In India, the disease was first reported in the Mount Abu area in 1957 and in recent years maize workers have been quite concerned with this problem, especially in Kharif (rainy) season (Payak 1982). The symptoms become apparent when the crop enters the senescence phase. They are similar for both pathogens and characterized by reddish discolouration in the interior of the stalks (Fig. 8). The disease causes a permanent wilting, leaves become flabby and basal stalk tissue obtains a pinkish to purple tinge. Some other species of *Fusarium* found associated with similar stalk rots in other countries are *F. oxysporum, F. culmorum, F. poae* and *F. solani* (Rintelen 1965; Zwatz 1969; Kommedahl et al. 1972; Karadzhova 1978; Lal and Dwivedi 1983). Though these fungi can cause extensive tissue disintegration in the internodes above the soil level, none of them has been confirmed so far to induce typical symptoms associated with premature drying and stalk rot of maize.

For large scale field inoculation, the tooth-pick method is the most desirable, chiefly because of its ease of preparation of the inoculum and rapidity of the inoculation procedure. In this method, only 5–10 s are required per inoculation. The most appropriate plant stage for inoculation is between tasselling and pollination (Zschege 1969). The second internode above the ground is usually the first elongated internode and is most frequently used. Splitting the stalk open and observing the rot is the most reliable method of determining the amount and extent of stalk rot. The score may be done in different ways, including a numerical scale (Young 1943; Reece 1949; Andrew 1954; Koehler 1960). One of the most common methods is to estimate the percentage of tissue decayed in an internode. Whenever the rot extends beyond the internode inoculated, the total rating given is equal to the sum of the disease ratings of the internodes infected. DeVay et al. (1957) used a 1–4 index scale for estimating stalk rot. Khan and Paliwal (1979) used 0–5, while Gupta and Renfro (1972) used 1–10. The latter two scales are more general in use for the



Figs. 1–9. Downy mildew and stalk rot diseases of maize. **1** *Sclerophthora* downy mildew; **2, 3** *Peronosclerospora* downy mildews; **4, 5** *Erwinia* stalk rot; **6** black bundle disease; **7** late wilt; **8** dry stalk rot; **9** kernels showing white streaks due to *Cephalosporium acremonium*

stalk rots in question. The index scale 1–10 (Payak and Sharma 1975) is constructed as follows: 1 = 25% of the inoculated internode discolonized; 2 = 26–50% of the inoculated internode discolonized; 3 = 51–75% of the inoculated internode discolonized; 4 = 76–100% of the inoculated internode discolonized; 5 = discolouration of less than 50% of adjacent internode; 6 = discolouration of more than 50% of adjacent internode; 7 = discolouration of more than three internodes; 8 = discolouration of more than four internodes; 9 = discolouration of more than five internodes; and 10 = plants prematurely killed.

The index scale 1–5 (modified scale based on Khan and Paliwal 1979) is made up as follows: 1 = disease spreading up to half of the inoculated internode; 2 = disease spreading more than half to whole inoculated internode, but not crossing the nodal plate; 3 = disease crossing the inoculated internode but confined to the next internode on either side; 4 = disease spreading up to four internodes, plant might wilt; and 5 =

Sources of resistance

Diseases	Inbred lines	Composites/varieties	References
1. *Sclerophthora* downy mildew	P4 A (Y)-1-3-Y, Col 1 × 38-11, A Theo-21 (B), Peru-330, Cuba 342-2-2, Eto-190-4-2, G715-A1-2-2, CI 21E, ETO PL-13-1, Tenn 29, Venz 1-42, Cuba-24, Adec A-257, Eto-25A, Kmr-35, Pem, DA-1-5	Antigua Gr. 1, Antigua 2D, Carribean Flint Composite, Eto Amarillo, Dorado de Teq-u-sete, Puerto Rico Gr. 1, Mich 166 × Eto Blanco, Puerto Rico 22D × Puerto Rico 17 D, Phil. DMR 1, Phil. DMR 2, Phil. DMR 5, Suwan 1, Suwan 5, Jawahar, Kisan, TAD, Karim Nagar Local, Tarun, Navin, Sweta, Kanchan	Lal 1975; Payak and Sharma 1979;
2. *Peroposclerospora* downy mildew			
(a) Philippine downy mildew	Ph 9 DMR	Munies White Flint, MIT S-2, MIT Sel 2, A 206 DMR, Aroman White Flint, Tiniguib, Philippine DMR-1, 2, 3, 4, 5, 6, 8, and 10, Taiwan Composite No. 1 and 2; Carribean DMR Composite-2, Thai Opaque-2 Composite 1 and 2	Aday 1975; Exconde 1975; Carangal 1975; Payak and Sharma 1979; Matthews 1981;
(b) *Sorghum* downy mildew	T × 601 and Ph 9 DMR	Ph DMR 1, Ph DMR 2, Ph DMR 3, Ph DMR 5	Frederiksen *et al.* 1973 Frederiksen and Ullstrup 1975; Frederiksen and Renfro 1977; Senanarong 1975; Payak 1975; Payak and Sharma 1979
(c) Sugarcane downy mildew	Narino 330-6-6-2, DA-1-5-f-1, 2039-2-2, (PTR × K61)-1, Pr-1, T × 601	Ph DMR-2, Ph DMR-6	Lal 1975; Payak 1975
3. *Erwinia* stalk rot	Peru-330 A Theo-21 (B), C1 21 E-3-X-Bulk, T × 325 C-1-1-3- – -5-x-Bulk, Sona Syn 72 lines-553-B-1-2-x-Bulk, De Se.-2-229-1-1-f-f-4-x-Bulk	Rudrapur local	Lal *et al.* 1970; Payak 1975; Singh 1979
4. *Cephalosporium* and *Fusarium* stalk rots			
(a) Black bundle disease	Col. 1 × 38-11, Peru-330, Di × 18 A1-3-f, Eto PL-1, Eto-25, Eto 28 A, Venz 1-42, G 715-A 36, G 733-263, Cuba 2-2, CI 21E	B1 × Cuba 11 J, Do Eto Syn and Kenya yellow	Lal and Dwivedi 1983
(b) Late wilt	Col 1 × 38-11, A Theo-21 (B), Cuba 342-2-2, CI 21E, (Eto PL-13-1, Tenn. 29, Kmr-35	Selection Precoz, VL-D1, Amarillo theobromina, Antiguas such as Antigua Gr. 1, Antigua 2D, Antigua Gr. 2, Karim nagar local and Rudrapur local	Payak *et al.* 1971; Payak and Sharma 1979; Lal and Dwivedi 1983
(c) Dry stalk rot	–	–	–

disease spreading to more than four internodes, plant wilts and ear does not form.

Mean disease rating per plot can be computed for each line by averaging individual plant ratings. The entries showing an average disease rating of 2.0 may be considered as highly resistant; 2.0–3.0 resistant; 3.0–4.0 susceptible, and 4.0–5.0 highly susceptible. Koehler (1960) concluded that final data on stalk rot should be taken 3–4 weeks after inoculation, but before the plants die.

Genetics of resistance

1 Sclerophthora downy mildew

Handoo et al. (1970) detected in a cross Pem (R) × Venz (S) a single dominant gene for resistance, whereas in Pem (R) and PH3 (S) resistance was controlled by two dominant complementary genes. In all other crosses studied by them resistance was found to be polygenically determined.

Asnani and Bhushan (1970) reported the genetic control of resistance to this downy mildew to be under polygenic control. Cuba 24, one of the inbreds studied by them, showed resistance at natural infection but susceptibility under artificial epiphytotic conditions, suggesting that this particular inbred is resistant to foliar (natural) but susceptible to primary infection (artificial inoculation directly into the soil).

Singh and Asnani (1975 a, b) crossed four resistant and four susceptible inbred lines, analyzed their F_1's, F_2's and back crosses to both parents. They found that resistance was polygenically governed under natural as well as artificial infection. Additive genetic variance was reported to be predominant. However, in some crosses, dominant and epistatic gene effects also proved important.

2 Peronosclerospora downy mildews

Chang (1970) and Chang and Cheng (1968) showed that the resistance to sugarcane downy mildew is controlled by a single dominant gene DMR, which is located on the short arm of chromosome II. However, Chang (1972) did not rule out the possibility of a polygenic nature of inheritance. Gomez et al. (1963) suggested that resistance to Philippine downy mildew is partially dominant over susceptibility and governed by a few genes only.

The inheritance of resistance to Java downy mildew (*P. maydis* (Racib) Shaw) in maize was reported by Hakim and Dhalan (1973) who examined 10 resistant and 3 susceptible open-pollinated varieties and their F_1's, F_2's and back crosses. The frequency distribution, for downy mildew reaction indicated a quantitative inheritance pattern.

Jinahyon (1973) using open pollinated maize varieties found resistance to *Sorghum* downy mildew controlled by many genes. Frederiksen and Ullstrup (1975) studied the resistance to *Sorghum* downy mildew. Their results indicated resistance to be dominant in some crosses and recessive in others. Mochizuki et al. (1974); Yamada and Aday (1977) and Singburaudom and Renfro (1982) studied the mode of inheritance of resistance to Philippine downy mildew by means of diallel analyses. They all concluded that resistance was controlled by dominant gene(s) and that level of dominance was in the range of partial to over-dominance. Francis (1967) and Carangal et al. (1970) found that resistance to this downy mildew was quantitative in nature and followed an additive nature of inheritance.

Studying plants at different stages of development, Kaneko and Aday (1980) found that mode of inheritance of resistance to Philippine downy mildew varied with epiphytotic conditions. Inheritance pattern was found to change from complete to partial dominance for resistance as the infection changed from very slight to severe. At about 50% level of infection there was no dominance for either resistance or susceptibility. Thereafter there was a shift to susceptibility showing partial to complete dominance. Such findings suggest that downy mildew resistance is governed by a polygenic system with a threshold reaction.

3 Erwinia stalk rot

The first attempt to work out the nature of inheritance of *Erwinia* stalk rot indicated that one or two genes govern the resistance (Payak et al. 1971). In a later study including parental inbreds, F_1's, F_2's and back crosses, F_1's exhibited a tendency towards resistance. In F_2, resistant and susceptible plants showed a 9 : 7, 15 : 1, 13 : 3 or 3 : 1 type of segregation. In several combinations the back cross data did not, however, confirm the ratios obtained in F_2. From a recombination program comprising four resistant and four susceptible inbreds and all their F_1's, F_2's and back cross generations, Singh (1979) came to the same conclusion that resistance was dominant over susceptibility and that number of major genes determining resistance was one or two dependent on inbred parents used. The two studies thus confirm each other.

4 Cephalosporium and Fusarium stalk rots

a) Black bundle. Khan and Paliwal (1979) analyzed 10 inbred lines (5 resistant and 5 susceptible), their F_1's, F_2's and back crosses in order to determine inheritance of resistance to *Cephalosporium acremonium.* Their results showed resistance to be incompletely dominant over susceptibility. On an average, F_2 plants were more diseased than those in F_1. The disease reaction after a back cross tended to go towards the recurrent parent.

Additive effects and additive-dominance interactions proved more important than other gene effects.

b) Late wilt. Sehata and Salem (1971) analyzed the genetics of resistance to late wilt of maize in the field. They utilized six generations resulting from two resistant and two susceptible inbred lines. They found additive gene effect to be significant in two crosses and dominance gene effect in one cross. Evidence for epistasis varied in a non-significant way from cross to cross.

c) Fusarium stalk rot. In connection with resistance to *F. moniliforme,* Russel (1961) found additive gene effects to be more important than non-additive gene effects. In a similar study, Younis et al. (1969) used highly resistant and susceptible inbred lines, F_1's, F_2's and back cross generations. They found two gene pairs controlling the disease reaction and complete dominance of resistance over susceptibility in F_1. Widakas et al. (1980) reported, however, incomplete dominance in F_1 for resistance to *F. moniliforme.* They concluded also few genes to be involved.

Breeding approaches for resistance

1 Downy mildews

Inheritance studies conducted so far have indicated resistance to *Sclerophthora* downy mildew to be polygenic. However, Handoo et al. (1970) found two complementary dominant genes governing resistance in some combinations. If substantial additive genetic variance exists, host resistance can be built up gradually by accumulation of genes for resistance. Such an approach could go via mass selection, S_1 selection, full-sib family selection or recurrent selection under optimum epiphytotic conditions. Whenever resistance proves to be dominant, recurrent selection for specific combining ability could be practiced. Even simple back crossing may prove useful.

The resistance to *Peronosclerospora* is polygenically governed, but the phenotypic expression varies with level of infection with some kind of threshold reaction. Kaneko and Aday (1980) suggested that selection should be done under severe epiphytotic conditions, provided inbreeding depression caused by a narrowed genetic variability is kept low.

2 Stalk rots

Singh (1979) found only one to two dominant genes to determine resistance to *Erwinia* stalk rot. Such an uncomplicated pattern will allow resistant genes to be incorporated into any susceptible background by backcrossing accompanied by artificial inoculation and detection of resistant plants. Transfer of resistance is not expected to pose any problem for developing improved populations or hybrids. A very similar situation is encountered in connection with resistance to *Fusarium* stalk rot allowing the same breeding approach. A preponderance of additive genetic variance in case of resistance to *Cephalosporium acremonium* as well as to *C. maydis* calls for application of mass selection, recurrent selection or full-sib family selection with the aim of building up an adequate resistance.

3 General picture

Recently a comprehensive resistance evaluation program for maize germplasm has been undertaken (Payak and Sharma 1979). During the last 15 years, a wide collection of maize has been screened at several research stations covering most of the disease agroclimatic regions in India. In addition, the evaluation has improved through applying artificial infections in the field.

Hybrid Ganga-5, a double top cross hybrid released in 1968, possesses good resistance to foliar diseases, especially against *Sclerophthora* downy mildew, but not to diseases as stalk rots and *Peronosclerospora* downy mildews. Hybrid Ganga Safed-2 has a high resistance to stalk rots such as those caused by *Pythium,* and *Erwinia* but shows susceptibility to foliar diseases. Among the composites developed by the All India Coordinated Maize Improvement Programme in the sixties, Kisan and Jawahar have high resistances to foliar diseases and Vikram has a tolerance to stalk rots. The early maturing composites developed in the seventies and early eighthies at Pantnagar have shown high degrees of resistance to both downy mildews and stalk rots. Steady progress has been possible through reliable methods of creating disease epidemics in the fields. Four populations, viz. 'Tarun', 'Navin', 'Shweta' and 'Kanchan' are examples of this systematic approach.

In general, local varieties have exhibited high degrees of susceptibility to foliar diseases. However, there is one exception in the inbred line 'Kmr-35' which was developed from a local variety of the Karimnagar district of Andhra Pradesh (India). It has been found quite promising against *C. acremonium* and *C. maydis.* 'Rudrapur local' has contributed resistance against *Erwinia* and *Pythium* stalk rots to the hybrid Ganga Safed-2.

In the cases of *Sclerophthora* and *Peronasclerospora* downy mildews, most of the resistant sources are yellow flint varieties from the Philippines ('Ph DMR-1' and 'Ph DMR-5'). The Philippine downy mildew resistance (DMR) programme started in 1953 and six Philippine resistant synthetics were developed (Aday 1975). Between 1953 and 1963 resistant inbreds were selected

and later between 1964 and 1968, local varieties and introduced germplasms were utilized to produce high yielding open pollinated varieties. The local varieties 'Munies White Flint' and 'MIT Sel. 2' have been found highly resistant. Between 1967 and 1973, local varieties 'MIT Sel. 2', 'Ph 9 DMR', 'A206 DMR', 'Aroman White Flint' and 'Tiniguib' were hybridized with introduced high yielding yellow flints and composites. Eight populations (Philippine DMR 1, 2, 3, 4, 5, 6, 8 and 10) were selected for improvement. Emphasis is now being laid on intra-population improvement of composite populations with the aim to combine high downy mildew resistance with high yields.

The Inter-Asian Corn Improvement Programme (IACP) has been responsible for the cooperative development of 'Taiwan composites No. 1 and No. 2' (Carangal 1975). Cooperative breeding started in 1972 using a Caribbean composite as a base population in crosses with resistant Philippine inbreds. Advanced generations of such crosses were exposed to infection in downy mildew nurseries. Survivors were selfed and equal amounts of seed from each ear bulked to produce 'Caribbean DMR composite'.

In Thailand the aim has been to develop downy mildew resistant opaque-2 varieties (Sriwatanapongse 1975). Resistance was introduced from 'Ph DMR 4' and 'Ph DMR 5' in crosses with seven elite populations. These were crossed with 'Thai Opaque-2 composite 1', advanced to F_2 and opaque types were selected. In F_4 full-hard-endosperm (semi-Opaque) families were selected in each population and bulked to form 'Thai Opaque-2 composite No. 3'. This was exposed to downy mildew in a nursery and reciprocal full-sibs taken from mildew-free plants with good hard endosperm were saved.

Conclusion

It is obvious from the foregoing account that no single material either released or experimental has shown resistance to stalk rots and downy mildews as well as to some other major disease constraints that attack maize in one region or the other. Efforts are now being made to build up multiple disease resistance (Payak and Sharma 1979). Experimental improvements are still needed such as (1) inoculation techniques particularly for some of the stalk rots in order to distinguish resistant plants from escapes, (2) studies pertaining to appropriate inoculum load for individual pathogens and (3) more genetic studies on resistance against different pathogens, since the success of a resistance breeding programme depends upon the understanding of the inheritance pattern.

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