A STUDY OF THE INHERITANCE OF RESISTANCE TO LATE-WILT DISEASE IN MAIZE CAUSED BY CEPHALOSPORIUM MAYDIS

BY

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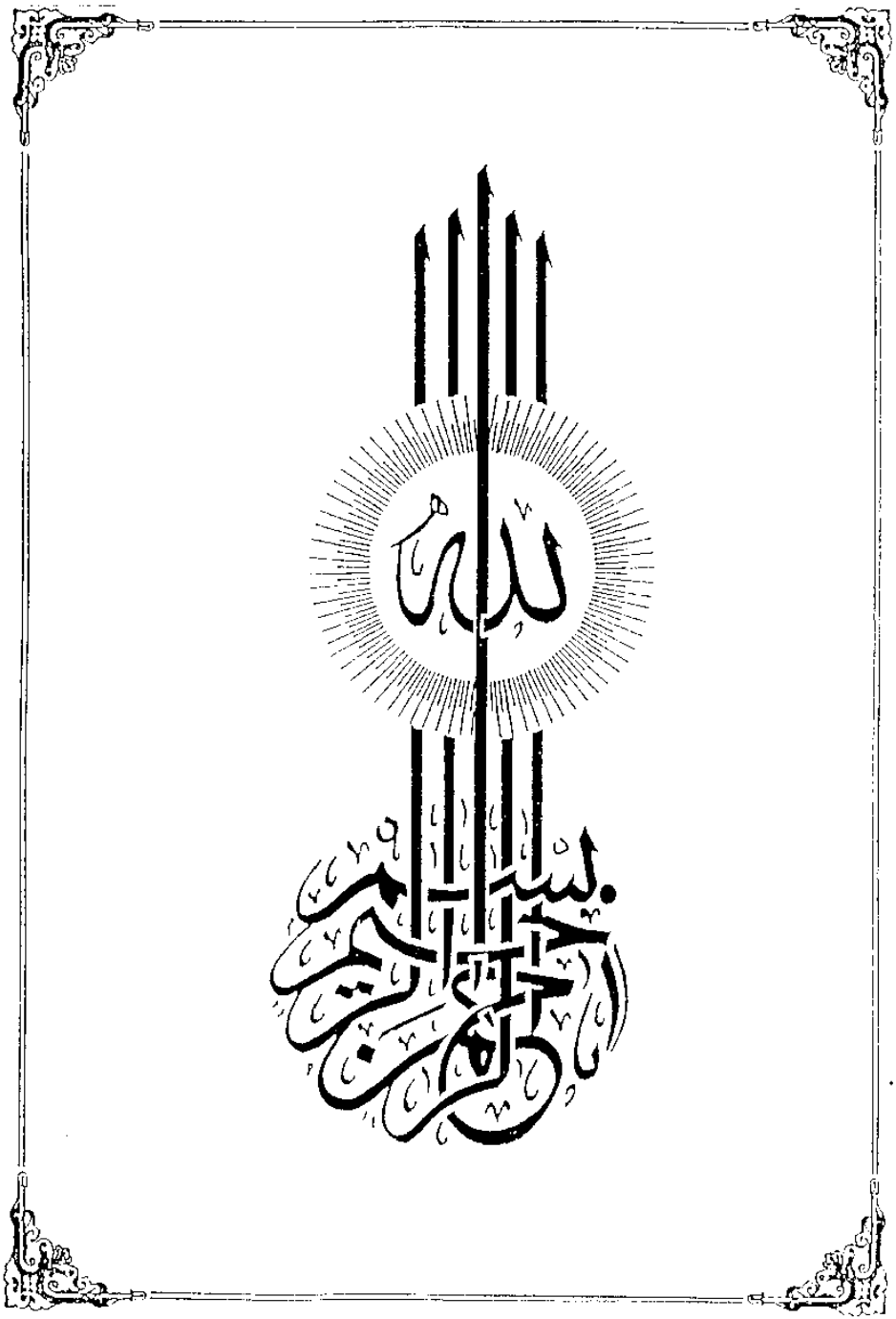

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I N T R O D U C T I O N

Maize is one of the more important grain crops grown in the Arab Repuplic of Egypt. Approximately 1.600.000 feddan have been devoted to maize production annually. (Statistics, Statistical Section of the Ministry of Agriculture, 1966).

The local hybrid maize breeding program, which was initiated in 1953 has resulted in the development of several adapted hybrids capable, under favourable conditions of yielding 20 to 30 percent more than the open - pollinated varieties. D.C. 186 and D.C. 17 S are the best hybrids now available.

The development of adapted maize hybrids offers farmers a readily available means of increasing their maize yields. In 1953 a project was set up, aiming at covering the major part of the maize area with hybrids, but unfortunately an out-break of stalk-rots set in, so that the entire project was seriously shaken, as maize stalk-rots caused more damage in 1960 than any other year since 1953 (Samra and Sabet 1966). Since that time the distribution of hybrid seed decreased sharply.

Different hybothesis have been put forth as to the cause of so called " Shalal" phenomenon of maize, i.e., physiological disorders, e.g., excess nitrogen, excess soil moisture, lack of oxygen in the soil, or infection with certain organisms. Among the organisms suspected are Erwinia carotovora f. zeae, Pectobacterium carotovorum f. zeae, and species of Fusarium. They are supposed to infect maize plants indepently, or in association with nematodes. (Report submitted to the Central Minister of Agriculture, Maize Stalk-rot Committee August 1960).

Sabet, Samra, and Hingorani (1963) subsequently identified the main pathogen responsible for the "Shalal" phenomenon as a distinct species of Cephalosporium which they named C. maydis. They described the symptoms caused by that organism as a "late-wilt" of maize which is a major contributor to the "Shalal" condition.

Since then special attention has been given to the isolation of inbred lines of maize resistant to late-wilt as a means of combating shalal by way of incorporating them into the constitution of hybrids whether double or otherwise.

The investigation of inheritance of reaction to the "Shalal" condition in maize is beset with serious difficulties. There is a multiplicity of organisms, as previously indicated, which share in the production of symptoms. The manifestation of symptoms also seem to be influenced by environmental conditions. Still, disease reaction of genetic stocks, whether pure bred, or hybrid, is unstable, which adds to the confusion.

In this study an attempt is made to study the inheritance of late-wilt disease of maize caused by C.maydis, which to the author's knowledge has not been attempted before. The effect of incorporating 0,1,2,3 and 4 resistant inbreds, in the constitution of hybrids, on the extent to which this would affect the performance of the hybrids, single or double, is also studied. It is hoped that the results obtained would be of value in producing hybrids, or synthetics more resistant to the "Shalal" condition.

REVIEW OF LITERATURE

Stalk Rots Abroad:-

Stalk rots of maize have been known for a long-time abroad. An early report of the infection of maize with a species of Cephalosporium was made by Adams and Manns (1921) who studied the black-bundle condition in sweet corn and reported it as due to the invasion of Cephalosporium sacchari Butler and Khan. Reddy and Holbert (1924) found that this condition was due to invasion of C. acremonium, which was apparently mistaken by previous workers for C. sacchari.

Valleau (1921), found that Fusarium moniliforme was carried between layers of the grain covers and might extend to the aleurone layer. Lobick (1933) showed that several species of Fusarium (e.g. F. moniliforme) and Gibberella saubinetii were carried in the seed and were present in the embryo. Edgerton (1939), examined samples of seed, and found a high percentage of internal seed-borne infections by Gibberilla fujikuroi var subglutinans(7.9%), Gibberella moniliformis (4.9%), Penicillium spp. (6.8%),

and Cephalosporium acremonium (2.5 %). Koppe (1940), reported that F. moniliforme and G. saubinetii were the most dominant causual organisms of ear-rot of maize in different locations in the U.S.A. Christensen and Gordon(1948) found F. moniliforme, nigrospora spharica, Diplodia zeae, C. acermonium, Penicillium spp, Aspergillus spp., and Mucor spp., on all samples of seed collected from commercial lots of maize. Roane (1950) reported that F.moniliforme was a major cause of corn stalk-rot disease in Virginia. Taylor (1952) found F. moniliforme in 86 percent of the rotted stalks in Iowa.

Koppe (1923), concluded that the germination period is a critical one in the life of a maize plant. Maize seedlings normally are resistant to most parasitic disease under conditions which favor germination and early growth. Ullstrup (1952), reported that the pathogen which may be present in the soil, or seeds enters the young plant by invading the mesocotyl, or crown. He added that such seedlings do not die, once they have established adventitious roots. They remain parasitized during the remainder of the growing season. The progress of the fungus is limited until sometime after pollination. But, as soon as the physiological activity that accompanies the

approach to maturity, is lessened, the quiescent infection becomes active, and the lower internodes of the stalk are invaded. Local infections at the lower internodes may also occur. It seemed that the fungus activity accompanied the maturation of the host.

Peppelis and Smith (1960) and Koehler (1960), tested different numbers of inbred lines, single crosses, and hybrids of maize over different years by applying different pathogens, and reported positive correlation showing uniformity between artificial inoculation and natural infections.

Otto and Everett (1955), stated that the disease complex, known as stalk-rot of maize may be caused by anyone, or a combination of several micro organisms, among which, Gibberella zeae, and Gibberella fujikuroi, as the chief pathogens in the form state. They pointed out that Stalk-rot, a disease of maturing plants, is not manifest by [illegible] of maize prior to pollination, and that in susceptible lines, resistance decreases as the plant matures. Koehler (1960), stated that the distribution of these pathogens varies from year to year and from one locality to another.

Foley (1962) found, that F. moniliforme increased in frequency with plant age, and was not a secondary invader. Wilcoxon (1963), showed that reduction in yield of 14 commercial maize hybrids reached 17 % following infection by Diplodia zeae (Diplodia maydis) or Fusarium graminearum (Gibberella zeae). Yield was reduced more by several lesions per stalk than by only one lesion per stalk, and when rotting began 9 - 10 weeks before harvest than at 3 - 6 weeks before harvest. Kamara (1965), reported that 36 percent of all pathogens causing stalk rotting belong to Fusarium spp.

Stalk-Rots at Home:

Some 16 years back, hybrid maize was introduced into Egyptian agriculture, and a project was set up aiming at covering the major part of the maize cultivated areas with hybrids. It was hoped that home production of this crop would be sufficiently raised to meet the increasing demand on maize. Unfortunately the project had failed far behind expectation (Kamra and Sabet 1966).

At the very beginning of the project, an outbreak of the bacterial stalk-rot was which was atributed to

Erwinia carotovora f. zeae (Sabet 1954). It was particularly severe and devastating on introduced inbred lines, Single and double crosses, so that the entire project was shaken.

Between 1956 and 1960, the bacterial stalk-rot seemed to be subsiding giving way to a new condition, the so called "Shallal". This condition rapidly became widespread and serious, causing in some fields up to 100 % infection. It was soon realized, that the released hybrids were more susceptible than the local open-pollinated varieties. The farmers became, as a result, reluctant to grow hybrid maize and in many areas, rejected any change from American Early and other Balady varieties.

Conflicting openions were expressed at to the cause of "Shallal" and accordingly there was a state of confusion as to the way the problem should be attacked. The "Shallal" condition was found to be a stalk-rot problem of complex nature. Several fungi and bacteria seem to be involved. However the most important condition was found to be the "Late-wilt", hitherto unrecorded in Egypt and caused by a new species of Cephalosporium, named, C. maydis Samra, Sabet and Hingorani (1962-1963).

Several surveys of the stalk-rot conditions in the maize growing areas were made over the period extending from 1960 to 1963 inclusive. Samra and Sabet (1966) classified the stalk-rots observed as follows:-

1- The late-wilt disease caused by C.maydis. The maximum infection was observed in Qualubiya and Munufia governates in 1960, but in subsequent years, the disease was more severe and widespread all over the country. The rate of infection varied from 0 to 80 % in fields chosen at random with an average of 15 %.

2- The black-bundle condition caused by C.acremonium corda was frequently associated with the late-wilt disease in the same fields. The disease was very common in the provinces where borer infestation was generally common.

3- Charcoal-rot caused by Sclerotium batatticola Taub. It was found to occur late in the growing season especially where high atmospheric temperature and high soil moisture prevailed.

4- [illegible] moniliforme Sheldon has been frequently observed through many conditions. The fungus is better known as a cause of ear-rot, but the role it plays in the stalk-rot complex is rather controversial.

— ... stalk-rot caused by Erwinia carotovora f. zeae, is not at present as devastating as it used to be some ten years back. Certain areas, however, still suffer from heavy losses due to the disease.

Sabet, Samra and Mansour (1956) stated that the symptoms of the late-wilt disease start as a moderately rapid wilting of plant at about tasseling time, the first indication of which is streaking of the leaves. The chlorotic streaks become translucent, and the whole leaf rolls inwardly starting from the tip as though suffering from loss of water. Eventually the leaves lose their colour and dry up. Reddish brown streaks appear on the basal internodes of the stalk, extending up to the fifth internode or above. Long yellowish to brownish streaks appear on the internal tissue which gradually became disjointed as the stalk dries up and shrinks. Stalk symptoms may be modified depending on the extent and type of invasion by secondary organisms. In severe cases of infection no cob formation takes place. If any grains are formed at all, they are shrunken and poorly developed.

Other organisms, indicated above, are found associated with the disease, and produce specific modifications