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ROOT DISEASES OF BEANS AND THEIR CONTROL STRATEGIES

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The following is a brief overview of the root disease problems on beans and short summaries of the research results obtained during 1985 1986 at CIAT in cooperation with Dr. M. A. Pastor-Corrales.

A. Overview of root diseases of beans:

Root diseases of beans are numerous and several of them are of common occurrence throughout the bean-growing areas of the world. Continuous bean production, improper crop rotation, increased soil compaction and other factors contribute to the prevalence and severity of root diseases. Root rots have been observed causing considerable damage to beans in the northerst areas of Brazil, coastal areas of Peru the highland of Mexico, Nicaragua, the United States and many other countries. Detailed information on bean yield losses due to root disease in Latin America and other bean-growing regions are very limited. However, yield losses to these diseases can be considerable, although often variable among fields in the same area as well as in the same field from season to season. This variability is partly due to the effect of the prevaling environmental and soil conditions at planting time and partly due to the type and number of root pathogens present and active in initiating the disease under such conditions.

Generally, root diseases are most severe and causes the greatest damage to beans when environmental and soil conditions are favorable for disease initiation during the early part of the growing season and then followed by a severe stress factor (s) in mid season such as drought, excess moisture, severe insect injury, other soilborne or foliar plant pathogens, etc.

Root diseases also cause indirect effects to beans that include increased susceptibility to extreme ranges of temperatures, drought, many biological stress, and inefficient utilization of nutrients in soil.

Bean root diseases can be incited by species of several plant pathogenic fungi and nematodes. The main pathogenic fungi attacking beans include Fusarium, Rhizoctonia, Pythium, Thielaviopsis, Sclerotium and Macrophomina. Of the many nematodes that parasitize bean roots, only species of meloidogyne (Root-knot nematodes) and Pratylenchus (Root-lesion nematodes) are of significant importance. The above mentioned pathogens may infect beans singly, or in any possible combination resulting in diseases complexes. However, each pathogen causes a distinct disease on beans with many possibilities for interaction with other pathogens and with nonpathogenic organisms in soil.

Above-ground symptoms in a field with severe incidence of root diseases include poor seedling establishment, uneven growth, chlorosis and premature defoliation of severely infected plants. The poor seedling establishment and reduced plant density are the result of seed rot and damping-off diseases. The later occurs when germinating seeds and young seedlings are attacked during the first 2 to 3 weeks after planting. However, infection of older plants result in reduced vigor, discoloration and slow rotting of the stem and root tissues.

Roots of severely infected plants are reduced in size and exhibit different degree of decay. The tap root of severely infected plants often dies, however coarse adventitious roots are produced from the hypocotyl areas above the infected tissues. These roots also become infected later but their production continues during moist soil conditions. The shape and color of infections on the stem and root tissues are specific and characteristic of the attacking pathognes. For the proper examination of bean roots, the plants should be dug up carefully and the soil removed with little disturbance to the fibrous root system.

The use of highly resistant bean cultivars is the best and most efficient control strategy for root diseases and especially appropriate for small farmers with low inputs. However, until an adapted cultivar resistant to all pathogenic organisms in a region becomes available, there is a need to consider controlling root diseases with a combination of compatible and effective control measures. The lack of resistance in a cultivar to a component of the root disease complex may well be managed by a chemical (seed or soil treatment), cultural (crop rotation, organic mulches, adjusting planting time, fertilizers used, land preparation, etc) or biological (addition or inhancement of beneficial soilborne organisms) control measures.

B. Brief Summaries of Research Results Obtained Todate at CIAT:

1. ETIOLOGY OF ROOT DISEASES. Not surprisingly, field diagnosis and laboratory isolation on Agar media from infected bean tissues have suggested that the major root diseases differs greatly from one bean growing region to another. For example, the major root disease pathogens in Popayán, Colombia are Rhizoctonia solani and to a lesser extent Fusarium solani f.sp. phaseoli. However, the major pathogen at Quilichao, Colombia is Macrophomina phaseoli

whereas F. solani f.sp. phaseoli appear to be the most damaging in the Pasto-Ipiales area with Sclerotium rolfsii as second in importance. In North-East Brazil, the major pathogen is Fusarium oxysporium f.sp. phaseoli, However, in the coastal areas of Perú, the rootknot nematode (Meloidogyne spp), Rhizoctonia solani and F. solani f.sp. phaseoli appear to be the most prevalent pathogens. These information illustrate the importance of determining the etiology of bean root disease where adapted cultivar development is in progress. It is critical to know if the selected lines at a particular site are resistant to one, two or to several pathogens and their interacting disease complexes.

2. SCREENING PROCEDURE AND VIRULENCE OF ISOLATES OF Macrophomina phaseolina TO BEANS.

In greenhouse tests, dried sclerotia of Macrophomina phaseolina (Mp) were highly affective for determining the reaction of bean accessions and the virulence of isolates of Mp. Sclerotia were produced abundantly in liquid medium containing 10 g peptone, 15 g dextrose, 0.25 g MgSO4, 7H2O and 0.5 g K2HPO4 in L of water. After 2 wk at 30°C, the mycelial mats with sclerotia were homogenized in a mixter with distilled water, centrifuged, washed once and dried for 48 hr. Sclerotia were mixed with pasturized soil (2 g/kg) and bean seeds were covered with 2 to 3 cm layer of the infested soil. Most seedlings of susceptible lines such as A 464 failed to emerge, and those which did so, exhibited disease symptoms, often dying within 2 wk. Seedlings of resistant lines such as G 5059 exhibited very slight or no symptoms. Isolates of Mp differed significantly in their virulence to beans when evaluated at 0.5,1,2 or 4 g dry sclerotia/kg soil. The most virulent isolate -Mp 34- was obtained from the bean cv. Chileno grown near Palmira, Colombia, S. A.

3. EVALUATION OF SELECTED BEAN ACCESSIONS FOR RESISTANCE TO Macrophomina phaseolina.

The reaction of 60 bean accessions to Macrophomina phaseolina (Mp) was evaluated in a field near Quilichao and a greenhouse near Palmira, Colombia, S. A. Field evaluations consisted of non-inoculated and Mp-inoculated treatments. Inoculations were made by pacing 4 g or whole rice grains colonized with Mp (2 wk) /2 m rows of beans (approx. 2-3 colinized rice grains/bean seed). Two nurseries were conducted, each with 3 replications. In greenhouse evaluations, bean seeds were covered with soil infested with dried sclerotia of Mp (2 g/kg soil). Based on emergence, Mp-incidence and severity ratings; 15 accessions were classified as highly resistant; 15 were considered intermediate and the rest as highly susceptible. A 300, BAT 332, BAT 447, BAT 1385, EMP 86, G 5059 and San Cristobal were among the highly resistant. In one test, San Cristobal 83 and G 5059 had average disease severities of 1.0, whereas A 294 (susceptible) had a score of 7.7 on a scale of 1-9 (1= no disease symptoms and 9= dead plant).

4. SEED TRANSMISSION AND EFFECT OF FUNGICIDE SEED TREATMENTS AGAINST Macrophomina phaseolina IN BEANS.

Surface-and internally-borne Macrophomina phaseolina (Mp) were detected in seeds of many bean accessions grown at Palmira and Quilichao, Colombia, S. A. The seedborne nature of Mp was confirmed by plating seeds on potato-dextrose agar and planting seeds in pasturized soil or sand. Seeds obtained from infected pods exhibited symptoms of Mp infection including discoloration, presence of pycnidia and sclerotia, and generally failed to germinate and/or emerge (e.g. 1/296 and 0/80). Seeds obtained from symptomless pods on infected plants showed as high as 28% infection. Surface disinfestation for 2 min in 0.6% NaOCl reduced the latter infection level to 3 to 5%. Six out of 62 lines grown in sand exhibited Mp

incidence ranging from 5 to 30%. Six fungicides were evaluated as slurry seed treatments at 2.5 g formulation/kg seeds in Mp-infested soil in the greenhouse. Benlate 50 wp was the most effective with Vitavax 75 Wp a close second.

5. FIELD AND GREENHOUSE REACTION OF BEAN GERMPLASM TO Rhizoctonia solani.

Two evaluations were conducted in an experimental field with a history of severe Rhizoctonia root rot near Popayán, Colombia, S. A. A total of 136 accessions were included in the first nursery with 3 replications. Each replicate consisted of two 1-m rows with 15 seeds/row. Seeds in one row were covered with 1 liter of Rhizoctonia-infested soil (2%, V:V; potato-soil inoculum: field soil) prior to closing the row. Emergence and disease severity rating were recorded at 2 and 5 weeks after planting. Stand counts and seed yield were recorded at harvest time. A total of 65 lines were re-evaluated in the same field. Greenhouse evaluation included 78 lines and was by planting seeds in 2% Rhizoctonia-infested soil. Emergence and disease severity ratings were recorded at 10 and 21 days after planting. A 300, BAT 1753, RIZ 21, RIZ 30, and EMP 81 were among the accessions considered resistant to R. solani in field evaluations.

6. REACTION OF BEAN GERMPLASM TO INFECTION BY Fusarium oxysporum F.sp. Phaseoli

An isolate of Fusarium oxysporum f.sp. phaseoli (FOp, F 5) obtained from severely infected plants near Belem do Sao Francisco, Brazil was used. The roots of 1 wk-old seedlings growing in sterilized sand were washed in running tap water and their tips were cut about 1-cm. Injured seedlings were then dipped for 2-5 minutes in a spore suspension of FOp and transplanted into pots filled with pasturized soil. Spore suspensions were prepared by adding about

5 cc of distilled water to 2 wk-old cultures of FOp growing on potato-dextrose agar plates at 25°C and scrapping the surface with a glass slide. The suspension was passed through 4 layers of cheese cloth, centrifuged at 5000 rpm, washed once, resuspended in water. Seedlings of the susceptible cv. IPA1$_5$ exhibited typical symptoms when dipped in suspension of 10^4, 10^5, 10^6 and 10^7 spores/cc. All seedlings dipped in the 10^6 spores/cc suspension exhibited symptoms 7-10 days after inoculation and died within 2 - 3 wks. Seedlings of the resistant line HF 465-63-1 exhibited slight symptoms only at the 10^7 spore conc. A total of 66 accessions were evaluated using the 10^6 spore conc. A total of 36 accessions were resistant to FOP among which where A 195, WAF 9, Cacahaute 72, AND 357, Mortiño, XAN 112, HF 465-63-1, and Ecuador 1056.