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Short Communication

Sources of resistance to cassava anthracnose disease

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A total of 436 African landraces and 497 improved cassava genotypes were planted in 1996, 1997, 1998 and 1999 growing seasons.. These were evaluated for their reactions to cassava anthracnose disease (CAD) under natural infection conditions at Ibadan (a high infection zone). The severity of the disease was determined by counting the total number of canker/plants and measuring the diameter the cankers. Data were collected at 6, 9 and 12 months after planting. The four-year data were pooled and subjected to statistical analysis. Result showed that of the 436 improved germplasm evaluated, 10 were resistant, 64 moderately resistant, 328 were moderately susceptible, and 95 were highly susceptible. The results also showed that 45 of the landraces were resistant, 87 moderately resistant, 354 were moderately susceptible, whereas 60 were highly susceptible. Of the resistant landraces and the improved, TME 19, TME 53, TME113, TME 244, TME 475, and TME 523; I85/02015 and I8700028 were completely free of cankers. The resistant genotypes have been introgressed into broad-based breeding populations to diversify resistance to CAD in newly improved genotypes.

Key words: Anthracnose cassava resistance.

INTRODUCTION

In the early 1970's when extensive research began on cassava, it was observed that the crop was susceptible to at least thirty different diseases caused by fungal, bacterial, viral and mycoplasma pathogens (Theberge, 1985). Among these, the African Cassava Mosaic Virus (ACMV), Cassava Bacterial Blight (CBB), Cassava Anthracnose Disease (CAD) and root and tuber rots were the most important in Africa (Lozano and Booth, 1974; Ikotun, 1975; Smith, 1991). Of these diseases, CAD caused by *Colletotrichum gloeosporioides* Penz f.sp *manihotis* Chev is the most important fungal disease of cassava in the field (Hahn et al., 1989). The most outstanding effect of the disease is its ability to cause severe stem damage causing canker on stem, wilting of leaves and diebacks. Badly infected stems become brittle and break easily under strong winds.

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The overall effect of these is the reduction in yield and in the amount of healthy plantable stems available to the farmers that a search for resistant cultivars needed to be embarked upon. The frequency with which the disease is encountered in cassava has been a matter of concern to many workers. Muyolo (1984) and Makambila (1987) reported that between 80-90% of local cultivars were rated as severely infected in Zaire and Congo. ively. Fokunang (1995) also observed that the causal organism of CAD was found on diseased cassava stems sampled from some states in the humid and sub-humid agro-ecological zones of Nigeria.

Chemical and cultural controls of cassava diseases usually encounter some problems. It has been observed that chemical control of plant diseases and rouging of all infected plants are not feasible since major diseases such as ACMV, CBB and CAD are already widely spread and their methods of dissemination complicated.

Breeding for resistance to cassava anthracnose disease appears to be the most efficient means of control

of CAD (Hahn et al., 1989a). It aims at improving cultivars resistance in wide range of environmental conditions and for a long time. The considerable variability among local germplasm collection in Africa and the fact that some of these local cultivars compared favourably with improved ones in yield, and pest and disease resistance, indicate that there is the need to include these local germplasm in breeding programme (Omueti et al., 1995) .

Little work has been done on resistance of cassava to CAD and determination of sources of resistant genes. Studies in these areas will assist the breeder in formulating an efficient strategy for incorporating the resistant genes into high yielding improved and stable varieties. The objective of this study, therefore, is to identify resistant genotypes to CAD for broad based breeding populations.

MATERIALS AND METHODS

Field evaluation and Identification for Resistance to CAD

The field at IITA, Ibadan (high infection zone for CAD) was planted with 564 local cassava germplasm and 442 improved germplasm in a single row trial using an augmented design during the 1997, 1998, 1999 and 2000 planting seasons. Each row was planted to 10 cuttings/clone in single-row ridges which were 10 m long, 0.75 m wide and 0.60 m high. The ridges was spaced 1 m apart and cassava stem cuttings was planted 1 m apart within ridges. Weeding was done manually as necessary to keep plots weed-free. The plants were monitored for CAD symptoms under natural disease conditions. The severity of the disease was determined by counting the total number of canker/plants and by measuring the length and width of the spreading cankers. The data from the 4 years were pooled together and subjected to generalized linear model (GLM) of SAS analytical package. This quantitative severity scores was used to separate the genotypes into various levels of resistance using a modified rank-sum method of Kang (1988).

RESULTS

Results presented in Table 1 show the variability among the local germplasm collection for resistance to CAD based on rank sum method. Of the 438 local evaluated in four growing seasons, 45 (10%) were found to be resistant, 87 (19.14%) were moderately resistant, 246 (54%) were moderately susceptible and 60 genotypes (13.2%) were highly susceptible (Figure 1). Five local genotypes in each of the categories were shown in Table 1. Among the resistant genotypes TME-113, TME-244, TME-475 and TME-89 were highly resistant i e completely free of cankers. Figure 1. also shows that of the 497 improved cassava genotypes evaluated, 10 (2.01%) were resistant. 64 (12.80%) were moderately resistant while 328 (54%) were moderately susceptible to CAD. 95 representing 19% of the improved germplasm evaluated were highly susceptible .TMS I85/02015 and I87/00028 among the resistant genotypes were highly resistant (Table 2). Using the size of cankers on stems as a parameter for screening, TME-475 had the smallest cankers and a mean size of whereas TME 22 and the check I30572 had the largest canker size and the mean size of 140.42 mm^3 and 160 mm^3 respectively (Table 1). Among the improved genotypes TMS M86/00086 had the smallest cankers and a mean of 15.12 mm^3 whereas TMS 91/00567 (the most susceptible genotype) had the largest canker size and a mean size of 257.23 mm^3 (Table 2).

Table 1. The field evaluation of African landraces for resistance to CAD.

Clones	Nc	*D*	Sc	*Dv*	*R*	Grd
TME-113	0.00	-15.17	0.00	-58.12	1	R
TME-244	0.00	-15.17	0.00	-58.12	1	R
TME-475	0.00	-15.17	0.00	-58.12	1	R
TME-89	0.00	-15.17	0.00	-58.12	1	R
TME-295	1.53	-13.64	I8.84	-19.28	8	R
TME-6	5.21	-9.96	27.22	-30.91	37	MR
TME-2	6.15	-9.02	46.36	-11.76	45	MR
TME-3	7.13	-8.04	30.25	-27.87	60	MR
TME-4	7.67	-7.02	37.24	-20.88	69	MR
TME-7	8.23	-6.94	15.02	-42.88	75	MR
TME-9	12.73	-2.44	54.03	-4.12	138	MS
TME-117	14.07	-1.01	69.12	11.01	154	MS
TME-11	22.00	6.83	93.32	35.21	238	MS
TME-40	24.07	8.91	73.49	15.37	262	MS
TME-180	24.87	9.72	56.46	-1.66	267	MS
TME-211	40.40	25.23	69.49	11.37	310	S
TME-159	45.33	30.16	100.18	42.06	311	S
TME-402	49.50	34.33	140.16	92.04	312	S
TME-1CHK	53.40	38.23	152.03	93.78	313	S
TME-22	78.60	63.43	140.42	82.28	314	S
I30572CHK	79.00	63.83	160.22	101.88	315	S
Grand mean	15.17		58.12			

nc = number of cankers /plant, *d* = deviation from grand mean of the total number of cankers/plant, sc = mean size of largest cankers (mm^3) *dv* = deviation from grand mean of the size of cankers, r = rank sum for each genotype, grp = susceptibility category using rank sum: R = resistant, MR = moderately resistant, MS = moderately susceptible, S = susceptible.

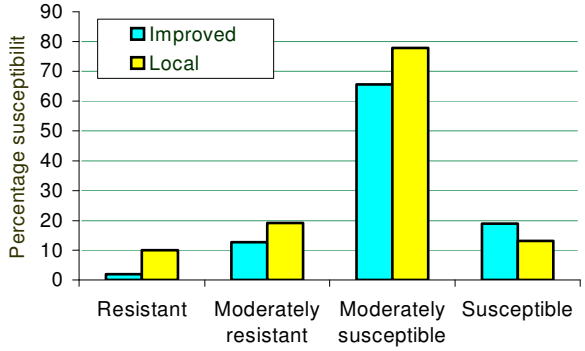

Figure 1. Resistance variability among local and improved cassava germplasm.

Table 2. The field evaluation improved cassava clones fo resistance to CAD

Clones	Nc	*D*	Sc	*Dv*	*R*	Grd
I85/02015	0.00	-18.99	0.00	-114.27	1	R
I87/00028	0.00	-18.99	0.00	-114.27	1	R
I84776	0.80	-18.19	23.5	-90.77	2	R
M86/00086	2.33	-16.66	15.12	-99.17	3	R
I91/00747	2.50	-16.44	28.15	-86.12	4	R
O88/01454	7.80	-11.19	16.51	-97.77	30	MR
I88/02268	8.86	-10.13	46.46	-67.81	41	MR
M86/0083	9.00	-9.99	17.42	-96.87	43	MR
O84/00009	10.00	-8.99	48.22	-66.05	56	MR
I4(2)1425PUB	10.40	-8.59	65.23	-49.07	61	MR
O85/00345	24.50	5.51	145.72	31.43	259	MS
I83/02561	25.52	6.53	179.54	65.27	261	MS
W5814	25.40	6.41	146.75	32.43	264	MS
I82/00959	26.65	7.66	189.56	75.23	270	MS
I92/0509	26.67	7.68	195.92	81.85	271	MS
I30572CHK	26.74	7.75	181.44	67.17	272	MS
I89/00854	56.87	37.88	195.98	81.71	350	S
91/00426	59.25	40.26	240.11	125.73	351	S
91/00590	61.67	42.68	350.87	236.6	352	S
91/01216	64.00	45.01	337.12	222.83	353	S
91/00567	65.33	46.34	257.23	142.93	354	S
Grand mean	18.99		114.27			

nc = number of cankers /plant, *d*= deviation from grand mean of the total number of cankers/plant, sc= mean size of largest cankers (mm^3), *dv* = deviation from grand mean of the size of cankers, r= rank sum for each genotype, grp= susceptibility category using rank sum: R = resistant, MR = moderately resistant, MS= moderately susceptible, S = susceptible.

DISCUSSION

The result of the field evaluation of local (African landraces) and the IITA improved genotypes for resistance to CAD showed that significant differences existed in both the number of cankers per plant and the size of the cankers among the genotypes 12 MAP. CAD infection is characterized by deeper cankers, causing stems to become brittle and easy to break by wind action.. The deeper cankers can block translocation of vital nutrients to active growing regions (IITA, 1990). It is therefore advantageous to screen for clones that are very low in canker number and small-sized cankers. This because the lower in the number of cankers on stems the fewer the number of disruptions in timely delivery of vital minerals and supply of nutrients to the plant. Moreover, the fewer the number of cankers, the lower the risk of stem breakage, thus more planting materials for the farmers. This study has shown that considerable variation in susceptibility exists among the local and improved genotypes suggesting a large range of genetic diversity within the germplasm and the possibility of breeding for resistance to CAD. Variation in resistance to CAD in cassava genotypes has been reported by Fokunang (1995) who observed that some improved genotypes had very high level of resistance to the disease than others. However, this study has shown that some of these local cassava genotypes are highly resistant to CAD and have higher sources of resistance than the IITA improved cassava genotypes, and could serve as new sources of resistance to the disease. These local genotypes referred to as landraces provide a wealth of genetic traits and form part of crop genetic resources (Hershey, 1993a).The resistant genotypes however, have been introgressed into a broad-based breeding populations at IITA.

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