Effect of Detopping on Disease Incidence and Symptom Severity of African Cassava Mosaic Virus Disease (ACMD) on Some Newly Developed Cassava Cultivars from Landraces Introgression

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Twenty-five cassava genotypes were exposed to natural infection by African cassava mosaic disease (ACMD) in plots at the International Institute of Tropical Agriculture (IITA), Ibadan (forest-savanna transition zone), Nigeria. The effect of removing the shoot tips (detopping) of cassava plants on disease incidence and symptom severity was assessed fortnightly for 14 weeks, starting 8 weeks after planting (WAP). There were highly significant differences (P < 0.01) in disease incidence (DI) and symptom severity (ISS) among clones throughout the period of observation, indicating different levels of resistance to infection. Detopping produced a significant effect on disease incidence in clones 91/02322, 91/02324, 91/02327 and 92/0427. It also had a significant effect on symptom severity of clones 92/0342, M94/0177 and TMS 4(2) 1425. The interaction between the treatment (TRT) and clone was highly significant (P < 0.01) for DI and ISS throughout the period of observation, an indication that there are differential responses of the clones to detopping. Cassava genotypes M94/0121 and Isu were observed to be highly resistant and highly susceptible respectively to the disease, while plants of genotypes 82/00058 and 91/02322 showed moderate susceptibility. None of the genotypes was immune to the disease. There was also a highly significant and positive correlation between DI and ISS in both detopped and undetopped plants. A conclusion from this study is that removal of shoot tips from moderately resistant cassava clones for consumption should be discouraged as it increases the severity of ACMD infection in the regenerating shoots of these clones.

Keywords: Cassava genotypes, African cassava mosaic disease (ACMD), detopping, disease incidence, symptom severity.

Cassava (*Manihot esculenta* Crantz) is a major tropical root crop (Hahn et al., 1989). It is a perennial shrubby plant originating in South America, which was introduced to Africa and Asia in the 16th century by Portuguese travelers (Alaux and Fauquet, 1987; Guthrie, 1987; Beachy and Fauquet, 1989). Cassava production in sub-Saharan African countries was estimated at 83.1 million tonnes of fresh root in 1993, 50.98% of world cassava production and at 80.1 million tons in 1994, 50.03% see Ekanayake, 1996 and IITA, 1997.

Cassava is generally used directly for human consumption in the producer countries where it is processed on a small scale in a rather unsophisticated manner (Silvestre, 1989). International trade in dried cassava in the form of meal or more usually pellets accounts for 10 to 15% of world production on dry weight basis. The leaves contain up to 40% crude

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protein, depending on the variety (IITA, 1992) and are consumed as a vegetable in many countries. African cassava mosaic disease (ACMD) has been singled out as one of the major biological constraints. It is widespread throughout sub–Saharan Africa and its coastal islands (Rossel and Thottappilly, 1985). Resistance to this disease is one of the main attributes sought in the cassava-breeding program at IITA (Hahn et al., 1980). Many resistant cultivars available are either produced by breeding or selected under natural field conditions (Fauquet and Fargette, 1990) and their resistance is then rated in terms of phenotypic symptom expression using an index of symptom severity score (ISS) of 1 to 5 (IITA, 1990).

Brian et al. (1978) observed that the insect vector's feeding habit and occasional harvests by the farmers of the tender leaves and shoots for consumption may alter the growth pattern of the plant and its yield. These occasional harvests usually remove the apical part of the crop plant; a process called "detopping". This is common in the phenology of cassava plants, especially during harvesting of the leaves for use as a vegetable, as practiced in some parts of Africa (Hahn and Keyser, 1985; IITA, 1990; Ng, 1992). Thus, the study was initiated mainly to investigate the effect of detopping or apical defoliation on the incidence and symptom severity of ACMD on some newly developed cassava cultivars developed by introgression from landraces.

Materials and Methods

A field trial was established in April 1997 with 25 cassava genotypes (23 improved and 2 local cassava cultivars) at the research farm of the International Institute of International Agriculture (IITA), Ibadan, Nigeria (Table 1). IITA is located to the north of Ibadan at latitude 7° 31' N and longitude 3° 45' E and 210 m above sea level in the forestsavanna transition agroecological zone. The soil type is Ferric Luvisols. The mean annual rainfall is 1400-mm spread between April and October after which there is usually a 5month period of dry weather. The minimum temperature is 20–23 °C and the maximum 27-37 °C (Jagtap et al., 1994). The experimental layout was a randomized complete block design in a split-plot arrangement with four replications. Main plots consisted of clones and subplots of apical defoliation treatments (detopping). Virus-free stakes of the cassava genotypes measuring 25 cm were planted at the beginning of the rains (April 1997) and exposed to natural infection by viruliferous whitefly (Bemisia tabaci Genn.) vector in the field. Each main plot consisted of 40 plants of a clone in four rows (ridges of height 30 cm and length 10 m) spaced 1 m apart. Plant spacing was 1 m × 1 m giving a plant population of 10,000 plants per hectare. The genotypes were grown under rainfed conditions and no fertilizer or herbicide was applied during the course of the experiment. Plots were hand-weeded when necessary.

Plants of the outer rows of a plot were detopped for each clone (apical defoliation) at 8 weeks after planting (WAP). Detopping was effected by cutting off the growing tip at the fifth node position from the apex (Njukeng, 1994). The remaining rows were left undetopped.

Disease incidence (DI) and Index of symptom severity (ISS) were assessed on all cassava plants a day before detopping at 8 WAP. Scoring continued fortnightly for 14 additional weeks after detopping (WAT). In scoring for DI each plant was first tagged using ribbons of a different color for each of the lateral stems of a plant. The DI was scored for each leaf on a lateral stem. Scoring was done fortnightly for 14 WAT. The incidence per stem of a plant was calculated as:

Total number of diseased leaves per stem Total number of leaves per stem

This DI taken on each genotype was based on the number of leaves per plant that developed similar Cassava mosaic disease symptom, which is the characteristic disease symptom of infecting Cassava mosaic begomoviruses in the field. The symptom severity levels called "Index of Symptom Severity" (ISS) range from scale 1 (no symptom) to 5 (very severe symptom) (IITA, 1990) and was computed for each plant as follows (Fauquet et al., 1987; IITA, 1990):

 $ISS = \frac{Sum \text{ of severity scores for all leaves on a plant stem}}{Total number of leaves on the plant stem}$

The means of DI and ISS per plant were determined for all treatment combinations and replications.

Results

At 8 WAP DI was relatively low in some clones and increased gradually throughout the period of observation of detopped and undetopped plants (Table 1). There was no marked difference in the disease incidence between detopped and undetopped treatments in some clones such as 82/00058, 92B/00061, M94/0177 and M94/0461 at all observations. The standard error of difference (S.E.D) for detopped and undetopped plants of the four clones are ± 24.2 and 23.0(82/00058), ± 27.6 and 27.6(92B/00061), ± 27.7 and 27.2(M94/0177) and ±21.1 and 23.16(M94/0461) (Table 1). Both detopped and undetopped plants of clones TMS 30572 and ISU attained a higher DI (above 85%) before the last scoring date (Table 1). Detopped plants of these two clones had a higher DI than the undetopped plants at 22 WAP (94.8 detopped, 89.4 undetopped for TMS 30572, and 96.3 detopped, 80.6 undetopped, for ISU). The average DI for clone 91/02327 ranged from 0.8% to 79.5% (undetopped plants) and from 1.5% to 80.8% (detopped plants). For clone M94/0177 the range was from 1% to 78.4% (undetopped plants), from 20.6 to 92.4% (detopped). Detopped plants of these two clones recorded a higher DI than the undetopped plants at the end of the observation. At 22WAP, clone 92/0398 showed a marked difference in DI between detopped plants (96.2%) and undetopped plants (75.1%).

There were highly significant differences (P < 0.01) between clones in terms of DI throughout the observations (*Table 2*) and mean DI values for genotypes such as, TME-1,

Table 1

Effect of detopping cassava clones on the incidence of ACMD

							Weel	Weeks after planting ^a	antinga									
Cassava		∞	Ī	10	1	12		14	1	16	18	~~	20		22	2	S. E	S. E. D.b
Clones	D	n	D	n	D	U	D	n	D	n	D	n	D	n	D	U	D	U
TMS 30572		59.8	50.1	76.5	78.5	79.0	90.2	86.3	92.7	88.4	94.3	88.7	95.3	6.88	94.8	89.4	±23.6	±10.3
4(2) 1425		11.4	39.9	47.4	76.8	56.4	83.4	61.9	91.9	53.9	92.3	54.4	96.4	53.3	97.0	55.9	±29.6	±15.8
82/00058		25.7	46.5	56.2	72.0	0.09	9.08	78.3	6.68	83.6	92.3	86.7	94.8	90.3	92.9	92.9	±24.2	± 23.0
91/02322		17.0	38.5	45.1	58.7	64.0	87.5	75.9	92.5	88.5	93.6	92.4	93.3	92.7	92.8	94.9	±21.2	±27.9
91/02324		2.1	8.6	23.6	28.3	38.4	41.8	51.0	64.1	61.0	72.6	72.8	78.4	84.8	86.3	93.4	±28.2	±31.1
91/02327		8.0	14.5	16.7	40.5	27.4	58.3	38.0	63.2	47.3	67.4	59.7	75.4	71.7	80.8	79.5	±32.3	±27.2
91B/00455		9.6	28.4	28.3	42.0	44.0	62.8	58.6	87.4	65.9	89.0	62.9	87.4	65.0	94.8	69.4	±28.9	± 21.8
92/0057		8.4	33.5	32.0	58.5	48.0	9.89	59.6	81.4	66.5	83.7	74.3	9.98	79.2	88.0	84.2	±32.6	±25.9
92/0325		19.1	59.8	48.9	73.0	61.5	81.8	69.2	87.2	71.5	86.5	74.4	88.0	75.7	89.5	76.7	±26.1	±19.7
92/0326		18.0	46.4	47.5	69.2	63.8	72.3	73.0	81.2	77.8	84.1	82.9	84.9	87.8	9.78	89.4	±20.7	±24.3
92/0342		13.0	56.3	32.6	73.1	46.0	83.9	57.9	87.4	64.9	8.98	73.1	85.9	76.0	89.4	75.9	±20.8	±22.9
92/0398		4.0	28.4	21.5	57.4	37.9	65.3	48.7	82.3	54.1	93.4	9.99	97.2	73.8	96.2	75.1	±23.9	±25.6
92/0427		9.3	33.8	38.5	58.0	26.7	65.7	0.89	77.5	6.62	81.4	85.0	86.0	89.4	9.68	9.88	±31.4	±28.4
92B/00061		16.8	50.9	35.1	72.3	54.2	85.4	68.4	87.1	77.5	88.7	84.7	84.9	91.3	6.68	92.7	±27.6	±27.6
92B/00068		12.1	27.5	26.9	50.2	42.9	65.2	61.9	72.6	69.2	81.4	78.5	87.4	8.98	93.3	93.2	±22.8	±29.1
94/0237		9.6	29.6	35.0	48.9	47.3	62.7	62.8	8.62	60.1	6.62	72.7	82.6	9.08	85.5	81.1	±28.4	±24.6
94/0239		9.6	12.5	16.5	33.4	27.2	42.2	38.6	49.4	48.5	58.8	56.7	67.4	65.7	72.8	73.6	±27.1	±24.0
94/0270		34.2	48.9	61.6	70.1	77.4	75.8	90.5	89.4	89.4	91.8	89.4	93.4	91.5	97.0	91.5	±24.8	±20.6
ISU		73.1	81.6	79.0	84.0	83.4	94.1	86.1	9.88	80.8	0.06	82.1	93.6	81.3	96.3	9.08	±24.6	±23.8
M94/0121		1.8	14.3	15.7	23.5	28.0	34.2	38.6	43.9	21.6	54.9	35.4	65.3	57.4	74.3	73.3	± 25.0	±22.9
M94/0177		1.0	39.5	35.1	60.3	51.2	75.0	62.2	6.06	71.2	92.0	9.92	91.1	78.2	92.4	78.4	±27.7	±27.2
M94/0192		12.1	36.7	34.6	59.7	45.5	74.3	57.3	83.5	63.7	97.6	68.9	95.3	75.7	93.1	77.9	±27.4	±22.6
M94/0461		14.3	26.4	25.6	43.4	39.1	53.5	49.3	57.8	57.4	65.0	66.5	71.0	75.0	77.3	9.87	±21.1	±23.16
M94/0583		4.0	26.8	15.1	37.4	25.8	46.0	38.1	53.5	48.5	53.6	60.2	53.5	66.2	57.2	0.69	±16.7	±24.3
TME-1	34.8	26.4	59.2	36.1	77.2	47.6	85.0	55.9	82.0	50.8	84.8	52.7	90.4	50.3	92.8	52.6	± 19.52	± 10.1

 a = disease incidence/plant b = standard error of the difference between means

Where:

D = detopped treatment U = undetopped treatment

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WAP	Clo	one	Treatme	ent (TRT)	TRT X	Clone
	Sum of squares	Pr > F	Sum of squares	Pr > F	Sum of squares	Pr > F
8	41.4319	0.0001**	0.5487	0.0001**	4.0986	0.0001**
10	53.9996	0.0001**	0.0123	0.5458 ns	5.6028	0.0001^{*}
12	51.6450	0.0001**	2.3975	0.0001**	4.5256	0.0001^{*}
14	45.2640	0.0001**	2.0718	0.0001**	6.0820	0.0001*
16	46.5785	0.0001**	6.6497	0.0001**	5.8858	0.0001*
18	32.8181	0.0001**	4.1118	0.0001**	6.1487	0.0065*
20	20.9080	0.0001**	2.4460	0.0001**	7.7116	0.0001*
22	14.9925	0.0001**	2.3382	0.0001**	6.8613	0.0001*

Table 2

Effect of detopping cassava clones on incidence of ACMD: Analysis of Variance

Where: ns = not significant

Pr > F = probability after comparing F tabulated and F calculated

X = interaction

92/0057, 92B/00068, 94/0237 with same letter are not significantly different as revealed by Duncan grouping (*Table 6*). The mean DI value of Isu was observed to be the highest (83.1), followed by TMS 30572 (80.5). But it was lowest for genotype M94/0121 (36.6). There was no significant difference in treatment effect at 10 WAP. But there was a highly significant (P < 0.01) interaction between detopping effect (TRT) and clone for the DI throughout the period of observation.

Plants of each of the two treatments showed a fluctuating pattern in index of symptom severity (ISS) (*Table 3*). The ISS rate increased slowly in both detopped and undetopped plants of clones 94/0239, 94/0237 and M94/0121. At the last record, detopped plants of these clones showed a higher ISS than undetopped plants. ISS increased throughout the period of observation at a faster rate in both detopped and undetopped plants of clone ISU (from 2.19 to 4.03 and 2.21 to 3.62, respectively, and the detopped plants showed a higher rating compared to undetopped plants of this clone.

In clone TMS 4(2) 1425 gradual increase in ISS over the same period was 1.94 to 3.68 (detopped plants) and 0.88 to 2.25 (undetopped plants), and the detopped plants had a generally higher score. Also the ISS of clones 92/0342 and M94/0177 ranged from 1.34 to 2.76 for (detopped plants) and from 0.96 to 1.91 for (undetopped plants). The ISS of the detopped plants of these two clones was significantly higher than that of the undetopped plants.

There was a highly significant difference (P < 0.01) among clones in terms of (ISS) throughout the period of observation (*Table 4*). At 10 WAP, plants of clones 92B/00061, 92/0325, 91B/00455 showed a marked increase in ISS after detopping at 8 WAP, interaction between the treatment (TRT) and clone was highly significant (P < 0.01) throughout the period of observation.

^{* =} Significant (at 5% level of probability)

^{** =} Highly significant (at 1% level of probability)

Table 3

Effect of detopping cassava clones on the index of symptom severity of cassava mosaic disease

							=	Weeks after planting ^a	planting	8.								
Cassava	~	8		10		12		14		16	18		20		22	2	S. E	S. E. D.b
Clones	Q	n	Q	n	D	n	Q	n	D	n	D	n	Q	n	Q	n	۵	n
TMS 30572	1.64	1.77	1.89	2.05	2.38	2.33	2.73	2.48	2.85	2.59	2.92	2.72	3.02	2.85	3.12	2.98	±0.55	±0.41
4(2) 1425	1.94	0.88	1.73	1.22	2.16	1.62	2.73	1.90	3.16	1.77	3.33	1.95	3.51	2.10	3.68	2.25	±0.76	±0.46
82/00058	1.50	1.47	1.78	1.83	2.03	2.04	2.34	2.19	2.78	2.35	3.03	2.55	2.55	2.73	3.52	2.90	±0.71	±0.48
91/02322	1.33	1.17	1.54	1.57	1.77	1.77	1.96	1.93	2.21	2.10	2.38	2.25	3.28	4.5	2.67	2.60	±0.48	±0.47
91/02324	1.02	1.00	1.11	1.19	1.31	1.36	1.49	1.50	1.66	1.57	1.72	1.58	1.80	1.81	1.91	1.92	± 0.33	± 0.31
91/02327	1.02	0.99	1.19	1.13	1.41	1.24	1.61	1.34	1.63	1.44	1.70	1.56	1.78	1.69	1.90	1.81	±0.30	± 0.28
91B/00455	1.08	0.87	1.30	1.01	1.44	1.16	1.72	1.31	2.02	1.52	2.06	1.64	2.08	1.73	2.20	1.86	±0.42	±0.36
92/0057	1.22	1.10	1.45	1.32	1.61	1.47	1.74	1.60	1.81	1.64	1.84	1.74	1.87	1.83	1.92	1.93	±0.24	±0.27
92/0325	1.42	1.14	1.70	1.46	1.88	1.70	2.08	1.78	2.20	1.83	2.38	1.91	2.56	1.99	2.73	2.08	±0.4	± 0.31
92/0326	1.49	1.27	1.66	1.53	1.92	1.70	2.14	1.86	2.31	2.07	2.44	2.22	2.59	2.38	2.74	2.52	±0.4	±0.43
92/0342	1.34	96.0	1.60	1.26	1.85	1.43	2.11	1.59	2.35	1.69	2.47	1.79	2.60	1.89	2.76	1.99	±0.50	± 0.34
92/0398	1.19	1.01	1.41	1.85	1.58	1.32	1.74	1.45	1.86	1.48	1.94	1.60	2.05	1.70	2.17	1.79	±0.33	± 0.27
92/0427	1.14	1.12	1.37	1.35	1.64	1.51	1.81	1.64	1.81	1.86	1.90	1.98	2.04	2.08	2.17	2.19	±0.34	±0.38
92B/00061	1.42	1.15	1.69	1.41	1.90	1.60	2.12	1.74	2.30	1.83	2.45	2.01	2.60	2.17	2.74	2.34	±0.46	±0.39
92B/00068	1.21	1.13	4.1	1.13	1.58	1.45	1.74	1.63	1.82	1.75	1.93	1.93	2.03	2.10	2.19	2.27	±0.32	±0.40
94/0237	1.16	1.07	1.31	1.31	1.55	1.43	1.74	1.58	1.89	1.51	1.98	1.65	2.13	1.80	2.26	1.93	±0.39	± 0.27
94/0239	1.05	1.04	1.18	1.30	1.33	1.24	1.41	1.35	1.49	1.44	1.60	1.53	1.70	1.62	1.82	172	±0.26	± 0.22
94/0270	1.55	1.33	1.89	1.69	2.07	1.95	2.23	2.15	2.33	2.27	2.57	2.46	2.80	2.62	3.01	2.78	±0.48	±0.49
Isu	2.19	2.21	2.56	2.57	2.91	2.87	3.22	3.03	3.40	3.11	3.68	3.29	3.81	3.48	4.03	3.62	±0.64	±0.47
M94/0121	1.03	1.02	1.45	1.13	1.24	1.24	1.35	1.37	1.46	1.16	1.59	1.30	1.72	1.53	1.83	1.73	±0.26	± 0.23
M94/0177	1.31	1.08	1.52	1.36	1.70	1.45	1.94	1.59	2.10	1.64	2.29	1.76	2.47	1.87	2.61	1.99	±0.46	± 0.29
M94/0192	1.29	1.15	1.61	1.39	1.69	1.41	1.80	1.50	1.94	1.60	2.08	1.68	2.20	1.80	2.38	1.91	±0.35	± 0.25
M94/0461	1.22	1.06	1.36	1.19	1.49	1.34	1.56	1.46	1.58	1.55	1.66	1.66	1.75	1.78	1.82	1.93	±0.20	± 0.30
M94/0583	1.10	1.02	1.29	1.12	1.40	1.23	1.51	1.35	1.54	1.46	1.56	1.59	1.59	1.71	1.65	1.83	± 0.18	± 0.29
TME-1	1.53	0.01	1.76	1.08	2.02	1.21	2.34	1.35	2.57	1.25	2.76	1.36	2.93	1.45	3.09	1.55	±0.56	±0.18
W/here.	a I	a – disease incidence/nat	gueldence	/nlant				۲	detonne	D - detonned treatment	1							
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	II	standard	error of	the diffe	rence b	D = standard error of the difference between means	neans	□	undetop	U = undetopped treatment	ment							

Table 4

Effect of detopping cassava clones on incidence of ACMD: Analysis of Variance

Weeks	Clo	ne	Treatme	ent (TRT)	TRT X	Clone
after planting	Sum of squares	Pr > F	Sum of squares	Pr > F	Sum of squares	Pr > F
8	137.36100	0.0001**	10.6154	0.0001**	9.6640	0.0001**
10	199.3087	0.0001**	9.8256	0.0001**	14.5420	0.0001**
12	275.4886	0.0001**	15.9534	0.0001**	14.9569	0.0001**
14	335.4249	0.0001**	29.9459	0.0001**	23.8816	0.0001**
16	398.7665	0.0001**	51.5286	0.0001**	47.7778	0.0001**
18	450.3260	0.0065**	47.9692	0.0065**	54.0553	0.0065**
20	500.7372	0.0001**	46.7681	0.0001**	64.1844	0.0001**
22	541.5846	0.0001**	49.4046	0.0001**	72.4746	0.0001**

Where: ns = not significant

* = Significant (at 5% level of probability)

** = Highly significant (at 1% level of probability)

Pr > F = probability after comparing F tabulated and F calculated

X = interaction

Table 5

Correlation between CMD incidence and symptom severity on detopped and undetopped cassava clones for all periods of observation

			1	Disease inc	idence (we	eks after p	lanting)		
Disease parameter	Treatment	8	10	12	14	16	18	20	22
Symptom severity	$\mathrm{D^{r}}$	0.97	0.92	0.89	0.87	0.74	0.69	0.69	0.66
	Sig.	**	**	**	**	**	**	**	**
	$\mathbf{U}^{\mathbf{r}}$	0.92	0.89	0.87	0.84	0.79	0.73	0.60	0.50
	Sig.	**	**	**	**	**	**	**	*

D = detopped treatment

U = undetopped treatment

r = correlation coefficient

Sig. = significance

* = Significant correlation at 95% confidence level

** = Significant correlation at 99% confidence level

There was a positive correlation (at 95% confidence level) between DI and ISS 22WAP in the detopped clones, while for other dates of observation it was significantly positively correlated at 99% confidence level (*Table 5*). There was a positive correlation (at 99% confidence level) between DI and ISS in both detopped and undetopped plants of all the clones throughout the period of observation and no clone was observed to be immuned to the disease.

Table 6

Evaluation of resistance status of Cassava genotypes to African cassava mosaic virus disease (ACMD)

Cassava clones	Mean DI values (%)	Resistance status
Isu	83.1a	HS
TMS 30572	80.5b	S
94/0270	76.2b	S
82/00058	73.3c	MS
91/02322	72.0d	MS
92B/00061	69.2e	MR
92/0326	68.5e	MR
92/0325	68.3e	MR
92/0342	63.9e	MR
92/0427	63.7e	MR
M94/0177	63.5e	MR
M94/0192	62.1f	MR
4(2)1425	61.9f	MR
TME-1	61.2g	R
92/0057	60.7g	R
92B/00068	60.3g	R
94/0237	58.2g	R
92/0398	57.4g	R
91B/00455	56.7g	R
M94/0461	51.1h	R
91/02324	50.6i	R
91/02327	46.4j	HR
94/0239	42.1k	HR
M94/0583	41.5k	HR
M94/0121	36.61	HR

Based on the number of plants that developed cassava mosaic disease symptoms in the field. Means with the same letter are not significantly different based on Duncan Multiple range test at P=0.05 where, HS=Highly susceptible, S=S susceptible, S=S moderately susceptible, S=S moderately resistant, S=S mean of disease incidence in percentage

Discussion

There was a highly significant difference (P < 0.01) in the DI among clones, indicating that the 25 clones differ in resistance. But genotypes with mean DI values having the same letter have similar resistance status. For instance, genotypes TME-1, 92/0057, 92B/00068 and 94/0237 were grouped as resistant "R". Also genotype Isu showed the highest mean DI value (83.1) while genotype M94/0121 showed the lowest (36.6). This indicates further that genotype Isu is the most susceptible of all the clones to the virus infection while clone M94/0121 is the least susceptible. There was also a marked difference in the DI between the two treatments for some clones, including 91/02324, 91/02327 and 92/0427. Therefore detopping may have influenced ACMD incidence.

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The interaction between the treatment and clone was highly significant (P < 0.01) for the DI, an indication that the clones differed in responses to detopping. Detopped plants of clone TMS 4(2) 1425 showed a markedly higher ISS than the undetopped plants. Similarly with clones 92/0342 and M94/0177, there was a highly significant interaction (P < 0.01) between treatment and clone for ISS. This suggests that there are different responses of the clones to the treatments.

There was a highly significant positive correlation between DI and ISS in both treatments that confirmed the observation of Njukeng (1994). It implies that the higher the DI, the higher the ISS. There was a significant difference among clones in terms of DI and ISS. This suggests that there are different levels of resistance to CMD among the clones screened (Hahn et al., 1980; Fauquet et al., 1987).

Detopping (apical defoliation of cassava plants) for vegetable purposes by farmers aids the buildup of vector whitefly (*Bemisia tabaci* Gennadius) and this invariably increases the level of disease incidence as well as symptom severity. The resultant effect is usually a considerable yield loss. Therefore, local farmers may be discouraged from this practice so as to reduce the possible predisposing stages of the cassava plant to incidence of infection of ACMD.

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