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GENE COMPLEMENTARITY OF RESISTANCE TO THE CASSAVA MOSAIC DISEASE AMONG AFRICAN CASSAVA ACCESSIONS

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ABSTRACT

The cassava mosaic virus disease (CMD) is the most important disease of cassava in Africa, causing severe economic losses. The genetic stock, clone 58308, has been extensively used in breeding for resistance to the disease, but recently, other sources of resistance to the disease have been identified among the landraces and could be used in breeding to diversify resistance to the disease. In this study, the progenies of 70 segregating F_1 crosses of some resistant and susceptible landraces, clone 58308 and its derivatives, were evaluated in 3 environments for their reaction to CMD to determine the mode of inheritance and allelic relationships among the various resistant accessions. The results indicated a polygenic mode of inheritance, with both resistant and susceptible accessions contributing effective factors towards CMD resistance in their progenies. Effective factors contributed by the susceptible parents were recessive. Among the resistant accessions, the results further showed that the genes for resistance are nonallelic and not linked. Positive transgressive segregants were also detected in several crosses. Significant differences in the mean distribution of F_1 progeny disease severity scores further revealed allelic differences among the various sources of resistance. These results imply that the resistant landraces are potential new sources of resistance, which could be used in a breeding programme, together with the resistant improved clones derived from clone 58308 to diversify resistance, while developing new genotypes with enhanced resistance to CMD.

Key Words: Allelism, environment, transgressive segregants

RÉSUMÉ

La maladie du virus de la mosa¿que de manioc (CMD) constitue la maladie du manioc la plus importante en Afrique causant des pertes économiques. Le stock génétique, clone 58308, a été utilisé de façon extensive en vue de la résistance à la maladie, mais récemment, d'autres sources de résistance à la maladie ont été identifiées au sein de races locales et pourraient être utilisées dans une optique de diversification de la résistance. Dans cette étude, les lignées de 70 croisements caractéristiques F_1 de certaines races locales susceptibles et résistances, clone 58308 et ses changements, étaient évaluées dans trois environnements pour leur réaction à CMD en vue de déterminer le mode d'héritage et les relations alléliques parmi les nouveautés résistantes. Les résultats ont indiqué un mode d'héritage polygénique avec et les nouveautés résistantes et susceptibles, toutes contribuant des facteurs fiables de résistance à CMD dans leurs lignées. Ces facteurs lorsque contribués par les parents susceptibles étaient récessifs. Au sein des nouveautés résistantes, les résultats montrent en plus que les gènes de résistance sont non alléliques et non liés. Des déviants transgressifs positifs étaient également détectés dans plusieurs croisements. Des différences significatives dans la distribution moyenne des impacts de sévérité de la maladie dans les lignées F_1 ont en plus révélé des différences alléliques parmi les diverses sources de résistance. Ces résultats impliquent

que les races locales résistantes sont nouvelles sources potentielles de résistance qui pourraient être utilisée dans un programme de culture de concert avec les clones résistants améliorés dérivés de 58308 pour pouvoir diversifier la résistance, tandis que l'on développerait de nouveaux génotypes avec résistance au CMD.

Mots Clés: Allélisme, environnement, déviants transgressifs

INTRODUCTION

The cassava mosaic disease (CMD) is regarded as the most prevalent and serious disease of the cassava crop (*Manihot esculenta*) in sub-Saharan Africa (Fargette *et al.*, 1985; Thresh *et al.*, 1994). Yield losses due to the disease, which could be as high as 95%, are estimated at US $2 billion per *annum* in Africa (Thresh *et al.*, 1997). Resistance breeding which started in the 1920s, deployed resistance from *Manihot glaziovii* into cassava (Jennings, 1994). Clone 58308, which was developed from this process, has been the main source of resistance in breeding for resistance to the disease (Hahn *et al.*, 1989). Cassava accessions with resistance derived from 58308 are widely cultivated in Africa and include TMS 30001, TMS 30572, TMS 4(2)1425, TMS 60142 and TMS 90257.

Additional sources of resistance to CMD are, however, required to ensure that durable resistance is maintained, since extensive use of closely related cultivars could result in vulnerability to pests and diseases (Cui *et al.*, 2001). Hahn *et al.* (1977) noted that some levels of resistance exist among the landraces, which are the new cultivars obtained through farmers' selection of superior seedlings for vegetative propagation. These resistant landraces could serve as new sources of resistance to diversify resistance to the disease, since landraces are known to contain co-adapted gene complexes with tolerance to diseases or adaptation to specific ecological conditions (Harlan, 1975). To efficiently utilise resistance from the African landraces in breeding programmes, it is essential to compare the various resistant genotypes with one another to determine if the loci for resistance are similar, and how their effects complement one another to enhance resistance.

In crops such as oats, soybean and sorghum, gene complementarity studies have been used to examine the relationships among genes from different sources of resistance to diseases and pests (Dixon *et al.*, 1991; Fox *et al.*, 1997; Wang *et al.*, 1998). Hahn *et al.* (1980) deduced from the polygenic mode of inheritance that resistance to the disease must be attributed to the combined action of a number of loci which are linked on a chromosome or a set of chromosomes in the cassava genome. They further suggested that since cassava is genetically heterozygous and probably an alloteteraploid, a study on the genetic mechanism of resistance to CMD would be complicated and difficult. Establishing the genetic relationships between the different sources of resistance would facilitate the choice of parents for developing new resistant cultivars. By combining different genes that relate to different sources of resistance, epistatic interaction may be identified such that higher levels of resistance can be developed to protect the crop.

The objectives of this study were to (i) compare the genetic basis of CMD resistance in the African landraces with that of 58308; and (ii) examine relationships among different sources of resistance in order to determine whether the genes are allelic among themselves.

MATERIALS AND METHODS

Plant material. A total of 70 F_1 crosses derived from two mating designs were the genetic material used in the study, since cassava is a highly heterozygous species and the F_1 is genetically equivalent to an F_2 with respect to loci common to parents (Magoon and Krishnan, 1977; Kawano, 1978). The mating designs were, a diallel cross between the resistant genetic stock, clone 58308 and improved resistant accession TMS30001, TMS30572 and moderately susceptible improved accession TMS30555 in all possible crosses including reciprocals (16 crosses); a North Carolina Design II (NCD-II) with TMS30001, TMS30555 and TMS30572 as the female parents, improved resistant accessions TMS4(2)1425, TMS60142, TMS90257, 10 resistant landraces and four susceptible landraces as the male parents (54 crosses). Crosses were made by hand

pollination at Ubiaja, Edo State, Nigeria in 1996. Table 1 shows the pedigree, origin, CMD resistance status of the parental accessions and the cross combinations.

Experimental design and procedures. In the 1997 growing season, seeds from the 70 F_1 crosses were planted in a seedling nursery in Mokwa, Niger State, to produce woody cuttings for the study. Progenies ranging from 52 to 934 individual genotypes per cross were evaluated in Ibadan, Oyo State during the 1998 and 1999 growing seasons and in Mokwa during the 1998 growing season. To ensure the survival of each F_1 genotype, two cuttings each of a genotype were planted in each replicate. The second cutting was removed at 6 weeks after planting (WAP) when the plants were established. The trials in each environment was a randomised complete block design with two replications. Plants were spaced at 0.5 m by 1 m apart in rows (ridges 30 cm high and 10 m long) giving a plant population of 20,000 plants ha^{-1}. Twenty cuttings of each parental accession were also planted in each replicate with the same spacing as the crosses. The trials were evaluated under rain-fed conditions.

Individual plants in each F_1 cross and 10 parental stands were assessed for their reaction to CMD due to natural infection by whiteflies at 6, 12, and 20 WAP. Assessment was based on the standard 5 point scoring scale for CMD, where a score of 1 indicates no obvious symptom and a score of 5 indicates severe mosaic symptoms and stunting of the entire plant (IITA, 1990).

Statistical analysis. Preliminary analysis of the data showed that in all three environments, CMD severity was highest at 12 WAP and the variance of the mean was also the highest. Subsequently, genetic analyses of the F_1 crosses were, therefore, based on CMD severity at 12 WAP, which give an overall impression of the symptom severity potential of a genotype.

Analysis of variance using the GLM procedure in SAS (SAS, 1999) were performed on the parents and progeny in each mating design. The analyses were based on mixed models with the genotypes considered fixed effects, replicate, environment and genotype by environment interaction (GXE) considered as random effects. Environment was tested with the replicates nested within environments, Env(Rep) mean square, and

TABLE 1. Cassava accessions, their pedigree/local name country of origin, CMD status

Clone	Pedigree information, local name and origin	Cross combination		Resistance status
TMS30001	Pedigree information lost	Parent 1	Female 1	R
TMS30555	58308 x Oyarugba dudu	Parent 2	Female 2	MS
TMS30572	58308 x Branca de Santa Caterina (OP)†	Parent 3	Female 3	MR
58308	*M. esculenta* x *M. glaziovii* (3 BC)	Parent 4		R
TMS60142	KR685 OP*		Male 1	MR
TMS90257	58308 x Oyarugba dudu		Male 2	R
TMS4(2)1425	58308 x Oyarugba fufun		Male 3	MR
TME1	Antiota (Ondo, Ondo State, Nigeria)		Male 4	R
TME2	Odungbo (Opeji, Ogun State, Nigeria)		Male 5	S
TME4	Atu (Iwo, Kwara State, Nigeria)		Male 6	R
TME5	Bagiwawa (New Busa, Niger State, Nigeria)		Male 7	R
TME6	Lapai-1 (Lapai, Niger State)		Male 8	R
TME7	Oko-Iyawo(New Lapai, Niger State Nigeria)		Male 9	R
TME8	Amala (Ireuekpen, Edo State, Nigeria)		Male 10	R
TME9	Olekanga (Ogbomosho, Oyo State, Nigeria)		Male 11	R
TME10	Orente (Ogbomosho, Oyo Nigeria)		Male 12	S
TME11	Igueeba (Warri Delta, Nigeria)-		Male 13	R
TME12	Tokunbo (Ibadan Oyo State, Nigeria)		Male 14	R
TME14	Abbey Ife (Abbey-Ife Osun, Nigeria)		Male 15	S
TME31	Bakince-Iri (Bahago, Sokoto, Nigeria)		Male 16	S
TME41	Danbusa (Kanji Niger, Nigeria)		Male 17	S
TME117	Isunikankiyan Ibadan, Oyo		Male 18	S

†OP=open pollinated; R=Resistant; MR=Moderately resistant; MS= Moderately Susceptible: S=Susceptible

the genotypic components were tested with their respective GXE interactions. The GXE mean squares were tested with the pooled error. The diallel cross was further analysed using Griffing's Method 1, Model 1 for fixed genotypes which partitions the crosses into the effects due to general and specific combining abilities and reciprocal effects (Griffing, 1956). Mean CMD severity scores of the F_1 crosses across environments were generated by least square means with the GLM analysis.

For each F_1 progeny in a cross, and in all three environments, the mean CMD severity score was determined with the means procedure in SAS. The means were then converted to the nearest integer to assign disease severity classes to the individual progenies in each cross. Frequency tables of progeny distribution in the five disease severity classes for each cross were constructed and the percentage of positive transgressive segregants (PTS) in the F_1 crosses were determined. A positive transgressive segregant was defined as an F_1 progeny with at least one disease severity score lower than the average disease severity score of the better parent in the cross. The Cochran-Mantel-Haenszel statistic, which tests the significance of differences between two variables in a contingency table (df=R-1), was used to test the mean distribution of the F_1 disease severity scores for the presence of significant relationships among the various sources of resistance, using the frequency procedure in SAS (SAS, 1999).

The minimum number of effective factor pairs responsible for resistance to CMD was estimated with the method described by Lawrence and Frey (1976), which incorporates the range of the segregating populations as the numerator in the estimation of effective factors and is valid when the parents do not represent the genotypic extremes. The number of effective factors in a cross was estimated as,

$$NE = \frac{\underline{R}^2}{8\sigma^2_g}$$

where R^2 was the range of the F_1 segregates in a cross and σ^2_g the genetic variance. Since cassava is vegetatively propagated, the genetic variance of a cross was estimated by subtracting the phenotypic variance of the F_1 mean from the variance of the parental means. Where little or no genetic variance was detected, the number of effective factors was not estimated. The numbers of factors contributed by the better and poorer parents in the crosses were then estimated using the method described by Dixon *et al.* (1991).

RESULTS

Variation among genotypes. The analysis of variance for CMD symptom severity in the 54 NCD-II crosses and their parents revealed significant variation ($P<0.01$) among the parents and crosses (Table 2). Significant variation among the parents was due to the both the resistant and susceptible male parents and significant variation among the crosses was attributed to the effects of the males ($P<0.05$). The genotype by environment effect was also significant for all components of the parents ($P<0.01$), crosses ($P<0.01$), the female ($P<0.05$) and male components of the crosses ($P<0.05$). In the diallel cross involving the resistant genetic stock clone 58308 and three improved cassava clones (TMS30001, TMS30555 and TMS30572) also used as females in the NCD-II, there was significant variation ($P<0.05$) among parents and crosses across (Table 2). The genotype by environment effect of the crosses was also significant ($P<0.01$) which was due to the GCA by environment effect ($P<0.01$).

Segregation of progenies and effective factors affecting resistance to CMD. The segregating F_1 crosses between susceptible parents (Table 3), resistant parents (Table 4), and between resistant and susceptible parents (Table 5) exhibited varying levels of resistance to CMD, suggesting polygenic inheritance.

Resistant phenotypes were detected in all the susceptible by susceptible crosses suggesting the presence of recessive resistance genes (Table 3). The highest frequencies of transgressive segregants were detected in all six susceptible by susceptible crosses, which ranged from 20.83% in the self, TMS30555 x TMS30555 to 72% in TMS30555 x TME41. The number of effective factors responsible for resistance to CMD across environments in the crosses involving susceptible parents ranged from 2 to 4. Both parents contributed equally to resistance, except for the

TABLE 2. Analysis of variance for CMD severity at 12 weeks after planting among all genotypes across environments

Source of variation	Diallel			NCD II		
		df	MS		df	MS
Environment (E)		2	9.00**		2	59.17**
Replicates within E		3	0.14		3	1.40**
Genotypes (G)		23	1.47**		78	1.39**
	Checks (Chk)	3	6.74**	Checks (Chk)	3	7.72**
	Parent (P)	3	1.08*	Parent (P)	20	3.17**
				Female (F)	2	1.64
				Male (M)	17	3.51**
				Susceptible (S)	5	1.08
				Resistant (R)	14	0.29
	Cross (C)	15	0.37*	Cross (C)	53	0.31**
	GCA	3	0.79	F (GCA)	2	1.40
	SCA	6	0.40	M (GCA)	17	0.57*
	Rec	6	0.13	F x M (SCA)	34	0.11*
G x E		43	0.21*		155	0.28**
	Chk x E	5	0.17	Chk x E	5	0.07
	P x E	4	0.11	P x E	40	0.26**
				F x E	4	0.26**
				M x E	34	0.28**
				S x E	10	0.36**
				R x E	28	0.36**
	C x E	30	0.16**	C x E	106	0.15**
	GCA x E	3	0.47**	F (GCA) x E	4	0.54*
	SCA x E	6	0.23	M (GCA) x E	34	0.27*
	R x E	6	0.21	F x M (SCA) x E	68	0.07
Error (genotypes)		66	0.11		233	0.07
Error (cross)		45	0.08		159	0.06

* Significantly different at the 0.05 probability level; ** Significantly different from zero at the 0.01 probability level

TABLE 3. Frequency distribution of F_1 disease severity scores, mean disease severity scores of progenies (MDSS), percentage of positive transgressive segregants (PTS), number of effective factors (NE) and number of factors contributed by better parent (NEBP) and by poor parent (NEPP) and mean number of progenies (N) across environments in crosses involving susceptible parents

Cross	CMD severity score					MDSS	PTS	NE	NEBP	NEPP	N
	1	2	3	4	5						
TMS 30555 ± TMS 30555	2	10	4	1	0	2.51±0.24	20.83	2	1	1	17
TMS 30555 ± TME2	24	3	10	18	1	2.57±0.30	42.50	2	1	1	56
TMS 30555 ± TME10	47	7	12	11	2	2.14±0.30	56.30	2	1	1	79
TMS 30555 ± TME31	20	4	4	8	0	2.06±0.30	57.8	2	1	1	36
TMS 30555± TME41	48	9	5	12	1	1.83±0.30	72.0	4	2	2	74
TMS 30555 ± TME117	10	2	3	3	1	2.17±0.30	59.80	3	2	1	19

±Standard error of mean

cross TMS30555 x TME117, where TMS30555, the better parent, contributed more (Table 3).

Susceptible progenies were detected in all the resistant by resistant crosses, which suggests that the genes for resistance to CMD in these accessions are non-allelic and not linked. The best resistant by resistant cross was TMS30572 x TMS90257, which had relatively few susceptible progenies (9%), while TMS30572 x TME12 followed by TMS30572 x TME9 were the worst resistant by resistant crosses with about 32% of their progenies being susceptible. The transgressive segregants detected in the resistant by resistant crosses ranged from 6.17% in the cross TMS30572 x TMS60142, to 39.17% in the cross TMS30572 x TMS90257. With the exception of crosses TMS30572 x TMS30001 and TMS30572 x TMS90257 where the number of effective factors estimated was 3, both parents in the resistant by resistant crosses each donated an effective factor to their progenies. A reciprocal effect in the differential number of effective factors contributed by the parents was

TABLE 4. Frequency distribution of F_1 disease severity scores, mean disease severity scores of progenies (MDSS), percentage of positive transgressive segregants (PTS), number of effective factors (NE) and number of factors contributed by better parent (NEBP) and by poor parent (NEPP) and mean number of progenies (N) across environments in crosses involving resistant parents

Cross	CMD severity score					MDSS	PTS	NE	NEBP	NEPP	N
	1	2	3	4	5						
TMS 30001 x TMS 30001	8	2	1	1	0	2.11±0.30	0	2	1	1	12
TMS 30001 x TMS 30572	29	9	1	0	0	1.76±0.30	0	2	1	1	39
TMS 30001 x 58308	6	3	0	0	0	1.69±0.30	0	2	1	1	9
TMS 30001x TMS 4(2)1425	5	5	2	1	1	2.09±0.30	0	2	1	1	14
TMS 30001 x TMS 60142	21	6	6	3	0	1.84±0.30	0	2	1	1	36
TMS 30001 x TMS 90257	6	4	2	1	0	1.96±0.30	0	2	1	1	13
TMS 30001 x TME1	3	3	2	1	0	1.95±0.30	0	2	1	1	9
TMS 30001 x TME4	12	3	4	5	0	2.20±0.30	0	2	1	1	24
TMS 30001 x TME5	15	9	6	4	1	2.09±0.30	0	2	1	1	35
TMS 30001 x TME6	9	4	4	2	0	2.09±0.30	0	2	1	1	19
TMS 30001 x TME7	15	7	6	7	0	2.20±0.30	0	2	1	1	35
TMS 30001 x TME8	6	8	1	1	0	2.00±0.30	0	2	1	1	16
TMS 30001 x TME9	30	8	9	9	1	2.04±0.30	0	2	1	1	57
TMS 30001 x TME11	8	4	2	3	0	1.92±0.30	0	2	1	1	17
TMS 30001 x TME12	8	2	1	2	1	1.91±0.30	0	2	1	1	14
TMS 30001 x TME14	4	2	1	2	0	2.28±0.30	0	2	1	1	9
TMS 30572 x TMS 30001	21	6	2	1	0	1.82±0.24	0	3	2	1	30
TMS 30572 x TMS 30572	10	2	1	0	0	1.75±0.24	36.5	2	1	1	13
TMS 30572 x 58308	35	20	7	2	0	2.07±0.24	0	2	1	1	64
TMS 30572 x TMS 4(2)1425	33	5	4	3	0	1.69±0.30	36.17	2	1	1	45
TMS 30572 x TMS 60142	15	3	3	3	0	1.79±0.30	6.17	2	1	1	24
TMS 30572 x TMS 90257	32	7	2	2	0	1.48±0.30	39.17	3	2	1	43
TMS 30572 x TME1	6	1	0	2	0	1.95±0.30	36.50	2	1	1	9
TMS 30572 x TME4	44	7	5	13	1	1.93±0.30	0	2	1	1	70
TMS 30572 x TME5	37	6	5	8	0	1.84±0.30	0	2	1	1	56
TMS 30572x TME6	32	5	4	6	0	1.73±0.30	0	2	1	1	47
TMS 30572 x TME7	20	3	3	5	0	1.86±0.30	0	2	1	1	31
TMS 30572 x TME8	47	10	3	11	0	1.86±0.30	9.83	2	1	1	71
TMS 30572 x TME9	157	21	37	44	2	1.96±0.30	0	2	1	1	261
TMS 30572 x TME11	52	20	3	10	0	1.74±0.30	23.83	+	+	+	85
TMS 30572 x TME12	45	12	10	17	1	2.08±0.30	0	2	1	1	85
TMS 30572 x TME14	41	15	7	12	1	1.91±0.30	8.83	+	+	+	76
58308 x TMS 30001	4	3	2	0	0	1.69±0.24	0	2	1	1	9
58308 x TMS 30572	18	16	6	2	0	2.15±0.24	0	2	1	1	42
58308 x 58308	2	2	1	0	0	1.93±0.24	0	2	1	1	5

+ = not estimated due to little or no genetic variance observed; ±Standard error of mean

seen in the crosses involving TMS30001 and TMS30572 (Table 4).

The crosses involving resistant and susceptible parents also had progenies falling into all classes (Table 5). The best cross between a susceptible and resistant parent was TMS30555 x TMS90527, with up to 96% of its progeny in the two resistant classes. The frequency of positive transgressive segregants was, however, generally low and ranged from 3% for the cross TMS30555 x TME14 to 33.33% for the cross TMS30572 x TME14. Two to five effective factors were responsible for resistance to CMD among the resistant by susceptible crosses. In most cases, especially where TMS30001 was the female parent, both the resistant and susceptible parents contributed an effective factor to their progenies. The crosses involving TMS30001 and TMS30555 exhibited a reciprocal effect in the differential number of effective factors that were contributed to their progenies.

Test for allelism. The CMH test on the mean distribution of F_1 progeny disease severity scores, revealed significant differences between improved accession TMS4(2)1425 and the resistant landraces TME12, TME14, and TME9 based on their crosses with the resistant accession TMS30572 (Table 6). Significant differences were also detected between TMS60142 and TME5 based on their crosses with TMS30555. The resistant improved accession TMS90257 exhibited

TABLE 5. Frequency distribution of F_1 disease severity scores, mean disease severity scores of progenies (MDSS), percentage of positive transgressive segregants (PTS), number of effective factors (NE) and number of factors contributed by better parent (NEBP) and by poor parent (NEPP) and mean number of progenies (N) across environments in crosses involving resistant and susceptible parents

Cross	CMD severity score					MDSS	PTS	NE	NEBP	NEPP	N
	1	2	3	4	5						
TMS 30001 x TMS 30555	28	12	4	1	0	1.89±0.24	0	3	2	1	45
TMS 30001 x TME2	9	4	9	7	1	2.70±0.30	0	2	1	1	30
TMS 30001 x TME10	13	5	8	5	0	2.20±0.30	0	2	1	1	31
TMS 30001 x TME31	5	1	3	2	0	2.36±0.30	0	2	1	1	12
TMS 30001 x TME41	16	5	4	7	0	2.21±0.30	0	2	1	1	32
TMS 30001 x TME117	12	6	4	4	0	2.12±0.30	0	2	1	1	25
TMS 30572 x TMS 30555	19	9	5	1	0	1.79±0.30	15.20	2	1	1	34
TMS 30572 x TME2	18	3	7	8	0	2.34±0.30	18.80	3	2	1	36
TMS 30572 x TME10	37	5	9	6	0	1.77±0.30	34	4	3	1	57
TMS 30572 x TME31	37	7	9	14	1	2.15±0.30	23.70	4	3	1	68
TMS 30572 x TME41	49	3	4	14	1	1.97±0.30	33.33	2	1	1	71
TMS 30572 x TME117	53	15	14	25	1	2.19±0.30	21	5	4	1	108
TMS 30555 x TMS 30001	24	12	6	1	0	2.16±0.24	0	2	1	1	43
TMS 30555 x TMS 30572	21	10	3	0	0	1.79±0.24	32.33	2	1	1	34
TMS 30555 x TMS 58308	13	8	2	1	0	2.06±0.30	0	2	1	1	24
TMS 30555 x TMS 4(2)1425	6	4	1	2	0	1.69±0.30	33	4	2	2	13
TMS 30555 x TMS 60142	25	5	3	4	0	1.79±0.30	15.17	2	1	1	37
TMS 30555 x TMS 90257	15	8	1	1	0	1.48±0.30	18.83	2	1	1	25
TMS 30555 x TME1	25	10	5	4	0	1.80±0.30	23.33	3	2	1	44
TMS 30555 x TME4	34	7	3	7	1	1.78±0.30	0	2	1	1	52
TMS 30555 x TME5	19	6	7	8	1	2.23±0.30	0	2	1	1	41
TMS 30555 x TME6	53	9	8	14	2	1.93±0.30	0	3	2	1	86
TMS 30555 x TME7	49	11	5	10	2	1.84±0.30	0	4	3	1	77
TMS 30555 x TME8	43	16	4	7	0	1.75±0.30	8.50	3	2	1	70
TMS 30555 x TME9	27	3	4	7	0	1.83±0.30	0	2	1	1	41
TMS 30555 x TME11	29	13	5	9	1	2.04±0.30	16.33	3	2	1	57
TMS 30555 x TME12	40	5	4	6	0	1.69±0.30	0	3	2	1	55
TMS 30555 x TME14	29	7	3	5	0	1.97±0.30	3.0	3	2	1	44
58308 x TMS 30555	3	6	3	1	0	2.42±0.24	0	2	1	1	13

±Standard error of mean

TABLE 6. Chi-square values for differences in the mean distribution of disease severity scores of F_1 progenies in crosses involving resistant male parents when crossed with each of the female parents 30001 resistant to CMD, 30555 susceptible to CMD and 30572 resistant to CMD

Resistant male	Female parent			
	58308	30001	30555	30572
TMS 30001 vs 58308	0.00	0.49	0.00	1.18
TMS 30001 vs TMS 30572	0.01	1.92	0.83	0.27
TMS 30572 vs 58308	0.00	0.08	0.63	1.76
TMS 4(2)1425 vs TMS 60142		1.31	0.79	1.09
TMS 4(2)1425 vs TMS 90257		0.48	1.68	0.26
TMS 4(2)1425 vs TME1		0.00	0.37	0.65
TMS 4(2)1425 vs TME4		0.02	0.29	2.83
TMS 4(2)1425 vs TME5		0.05	0.39	1.17
TMS 4(2)1425 vs TME6		0.24	0.02	0.66
TMS 4(2)1425 vs TME7		0.00	0.20	1.40
TMS 4(2)1425 vs TME8		0.76	0.86	1.02
TMS 4(2)1425 vs TME9		0.15	0.15	4.62*
TMS 4(2)1425 vs TME11		0.11	0.00	0.89
TMS 4(2)1425 vs TME12		0.08	1.23	6.11*
TMS 4(2)1425 vs TME14		0.00	0.75	4.06*
TMS 60142 vs TMS 90257		0.09	0.18	2.25
TMS 60142 vs TME4		1.27	0.21	0.14
TMS 60142 vs TME5		1.39	4.09*	0.02
TMS 60142 vs TME6		0.45	1.14	0.11
TMS 60142 vs TME7		2.18	0.40	0.01
TMS 60142 vs TME8		0.05	0.01	0.05
TMS 60142 vs TME9		1.04	0.39	0.34
TMS 60142 vs TME1		0.89	0.22	0.00
TMS 60142 vs TME11		0.63	1.85	0.15
TMS 60142 vs TME12		0.49	0.07	0.93
TMS 60142 vs TME14		0.82	0.00	0.34
TMS 90257 vs TME1		0.37	0.81	1.36
TMS 90257 vs TME4		0.36	0.67	4.53*
TMS 90257 vs TME5		0.34	4.93*	2.47
TMS 90257 vs TME6		0.07	1.76	1.69
TMS 90257 vs TME7		0.64	0.94	2.69
TMS 90257 vs TME8		0.01	0.32	2.27
TMS 90257 vs TME9		0.18	0.95	6.78**
TMS 90257 vs TME11		0.15	2.67	2.22
TMS 90257 vs TME12		0.11	0.04	8.44**
TMS 90257 vs TME14		0.31	0.24	6.18*
TME1 vs TME4		0.00	0.00	0.03
TME1 vs TME5		0.02	3.11	0.02
TME1 vs TME6		0.15	0.45	0.09
TME1 vs TME7		0.01	0.03	0.00
TME1 vs TME8		0.62	0.20	0.05
TME1 vs TME9		0.07	0.05	0.09
TME1 vs TME11		0.06	0.98	0.11
TME1 vs TME12		0.04	0.64	0.31
TME1 vs TME14		0.00	0.18	0.10
TME4 vs TME5		0.01	2.91	0.44
TME4 vs TME6		0.14	0.43	0.78
TME4 vs TME7		0.03	0.03	0.10
TME4 vs TME8		0.58	0.20	0.70
TME4 vs TME9		0.08	0.04	0.07
TME5 vs TME6		0.12	1.55	0.06
TME5 vs TME7		0.09	2.88	0.06
TME5 vs TME8		0.58	5.70*	0.01
TME5 vs TME9		0.05	2.01	1.11

TABLE 6. Contd.

Resistant male	Female parent			
	58308	30001	30555	30572
TME6 vs TME7		0.36	0.30	0.20
TME6 vs TME8		0.17	1.56	0.02
TME6 vs TME9		0.03	0.15	1.60
TME7 vs TME8		1.00	0.47	0.12
TME7 vs TME9		0.30	0.00	0.30
TME8 vs TME9		0.33	0.44	1.72
TME11 vs TME4		0.05	0.92	1.19
TME11 vs TME5		0.03	0.79	0.09
TME11 vs TME6		0.02	0.13	0.00
TME11 vs TME7		0.17	0.76	0.27
TME11 vs TME8		0.28	2.48	0.03
TME11 vs TME9		0.00	0.47	2.72
TME11 vs TME12		0.00	3.26	4.29*
TME11 vs TME14		0.05	1.88	2.07
TME12 vs TME4		0.04	0.62	0.68
TME12 vs TME5		0.02	6.23*	2.21
TME12 vs TME6		0.02	2.30	2.76
TME12 vs TME7		0.13	1.04	0.93
TME12 vs TME8		0.21	0.19	2.98
TME12 vs TME9		0.00	0.91	0.65
TME12 vs TME14		0.04	0.12	0.36
TME14 vs TME4		0.00	0.17	0.06
TME14 vs TME5		0.02	4.31*	0.90
TME14 vs TME6		0.13	1.15	1.36
TME14 vs TME7		0.01	0.37	0.29
TME14 vs TME8		0.52	0.00	1.31
TME14 vs TME9		0.06	0.36	0.00

* P-value <0.05; ** P-value <0.01

a significant difference from the resistant landrace TME5 based on their crosses with TMS30555, and from resistant landraces TME9, TME12, and TME14, based on their crosses with TMS30572. The test further revealed significant differences between resistant landraces TME12 and TME11 based on their crosses with TMS30572. TME5 was also significantly different from TME8, TME12, and TME14, based on their crosses with TMS30555.

The moderately susceptible accession TMS30555 exhibited significant differences from resistant clones TMS30001, 58308, and TMS30572 based on their crosses with TMS30555 (Table 7). Susceptible landraces TME2, TME31 and TME117 were also different from the resistant clones TMS4(2)1425 and TMS90257 based on their crosses with TMS30572. The results further showed that TME2 was significantly different from TMS60142 based on their crosses with TMS30001 and TMS30555 and different from TMS90257, TME1, TME4, TME6, TME7, TME8, TME9, and TME11 from their crosses with TMS 30555. A significant difference between TME2 and TME11 was also detected when they were crossed with TMS30572. The susceptible landrace TME117 was significantly different from TME5, TME6, TME8, and TME11 based on their crosses with TMS30572.

Among the susceptible cassava accessions, the results further showed that TME2 was significantly different from TME10 and TME41 based on their crosses with TMS30555 and that TME117 and TME10 were significantly different based on their crosses with TMS30572 (Table 8).

DISCUSSION

Resistance to CMD is assessed by a number of resistance components including laboratory based

TABLE 7. Chi-square values for differences in the mean distribution of disease severity scores of F_1 progenies in crosses involving resistant and susceptible male parents when crossed with each of the female parents 30001 resistant to CMD, 30555 susceptible to CMD, and 30572 resistant to CMD

Male resistant source	Female parent			
	58308	30001	30555	30572
TMS 30001 vs TMS 30555	0.99	0.08	6.37*	1.10
TMS 30555 vs 58308	0.59	0.46	5.22*	0.02
TMS 30555 vs TMS 30572	1.54	2.51	10.99**	1.68
TMS 4(2)1425 vs TME2		1.10	1.62	6.63*
TMS 4(2)1425 vs TME10		0.00	0.00	1.30
TMS 4(2)1425 vs TME31		0.01	0.04	5.98*
TMS 4(2)1425 vs TME41		0.04	0.15	2.01
TMS 4(2)1425 vs TME117		0.14	0.16	8.83**
TMS 60142 vs TME2		7.63**	8.84**	1.48
TMS 60142 vs TME10		2.33	1.51	0.01
TMS 60142 vs TME31		1.34	1.93	0.99
TMS 60142 vs TME41		1.28	0.51	0.03
TMS 60142 vs TME117		0.83	2.14	1.80
TMS 90257 vs TME2		3.20	8.93**	9.05**
TMS 90257 vs TME10		0.74	2.18	2.70
TMS 90257 vs TME31		0.55	2.77	8.23**
TMS 90257 vs TME41		0.32	1.08	3.44
TMS 90257 vs TME117		0.18	3.07	11.46**
TME1 vs TME2		0.98	7.92**	0.58
TME1 vs TME10		0.01	0.71	0.02
TME1 vs TME31		0.02	1.16	0.34
TME1 vs TME41		0.01	0.08	0.00
TME1 vs TME117		0.07	1.50	0.63
TME2 vs TME4		1.96	7.86**	1.19
TME2 vs TME5		2.81	1.01	2.77
TME2 vs TME6		3.03	6.34*	3.32
TME2 vs TME7		1.92	8.74**	1.46
TME2 vs TME8		4.33*	13.22**	3.44
TME2 vs TME9		4.00	5.88*	1.20
TME2 vs TME31		0.77	2.42	0.13
TME4 vs TME41		0.00	0.07	0.07
TME10 vs TME5		0.13	1.15	0.00
TME10 vs TME6		0.43	0.04	0.08
TME10 vs TME7		0.00	0.56	0.05
TME10 vs TME8		1.14	2.09	0.02
TME10 vs TME9		0.36	0.31	1.07
TME10 vs TME11		0.21	0.03	0.12
TME10 vs TME12		0.17	2.87	2.22
TME10 vs TME14		0.01	1.55	0.89
TME11 vs TME2		2.26	4.15*	4.75*
TME11 vs TME31		0.16	0.04	4.23*
TME11 vs TME41		0.03	0.59	0.62
TME11 vs TME117		0.00	0.24	7.50**
TME12 vs TME41		0.02	1.23	1.17
TME14 vs TME31		0.02	1.97	0.44
TME14 vs TME41		0.01	0.48	0.27
TME31 vs TME4		0.05	1.05	0.75
TME31 vs TME5		0.09	0.35	2.24
TME31 vs TME6		0.30	0.26	2.76
TME31 vs TME7		0.01	0.91	0.99
TME31 vs TME8		0.86	2.55	2.97
TME31 vs TME9		0.20	0.62	0.73
TME41 vs TME5		0.00	2.55	0.17
TME41 vs TME6		0.11	0.19	0.40

TABLE 7. Contd.

Male resistant source	Female parent			
	58308	30001	30555	30572
TME41 vs TME7		0.07	0.01	0.01
TME41 vs TME8		0.53	0.62	0.31
TME41 vs TME9		0.05	0.00	0.35
TME117 vs TME4		0.06	1.29	1.95
TME117 vs TME5		0.04	0.03	4.15*
TME117 vs TME6		0.03	0.52	4.72*
TME117 vs TME7		0.23	1.16	1.92
TME117 vs TME8		0.33	2.73	5.46*
TME117 vs TME9		0.00	0.88	2.62
TME117 vs TME12		0.00	3.17	0.34
TME117 vs TME14		0.06	2.18	1.43

* P-value <0.05; ** P-value <0.01

methods such as virus resistance (virus content estimated by ELISA), as well as field based assessments, incidence (percentage of infected plants) and symptom intensity or severity (Fargette *et al.*, 1996). Fargette *et al.* (1996) demonstrated a significant correlation between symptom severity and ACMV titre among resistant genotypes. Although, vector transmission of the virus is dependent on whitefly activity which would vary in different environments, an assessment of an accession resistance potential is generally based on its resistance in different environments. Recent studies which used artificial inoculation methods to study the reaction of cassava genotypes to the viruses causing the disease, gave similar CMD resistance status to genotypes classified as resistant or susceptible based on field observation across environment (Ogbe *et al.*, 2002; Ariyo *et al.*, 2003), which further proves that multilocation testing is a reliable means of assessing CMD resistance potential in a population. Thus, to obtain more precise genetic information on the genotypes tested, our experiments were conducted in different environments (i.e., locations and years), to take into account the GXE effect on the expression of the trait. Furthermore, our analysis were based on severity at 12 WAP which has previously been documented as the period when CMD incidence is high (Leuschner, 1978; Ogbe *et al.*, 1996), plants are generally more susceptible to secondary infection (Fargette *et al.*, 1994) and affects yield of storage roots at harvest.

TABLE 8. Chi-square values for differences in the mean distribution of disease severity scores of F_1 progenies in crosses involving susceptible male parents when crossed with each of the female parents 30001 resistant to CMD, 30555 susceptible to CMD and 30572 resistant to CMD

Male parent	Female		
	30001	30555	30572
TME2 vs TME31	0.77	2.42	0.13
TME2 vs TME41	2.47	8.09**	1.64
TME10 vs TME2	1.71	5.38*	2.84
TME10 vs TME31	0.00	0.13	2.26
TME10 vs TME41	0.11	0.40	0.15
TME31 vs TME41	0.08	0.74	1.22
TME10 vs TME117	0.28	0.36	4.19*
TME117 vs TME2	3.01	0.88	0.00
TME117 vs TME31	0.19	0.08	0.19
TME117 vs TME41	0.04	1.01	2.77

* P-value <0.05; ** P-value <0.01

In many cases, resistance to plant viruses is under a simple genetic control involving a single dominant or recessive gene (Fraser, 1990). However, there are reports in the literature indicating that resistance to some plant viruses is under a complex genetic control (Hahn and Howland, 1972; McMullen *et al.*, 1994; Caranta and Plaloix, 1996; Melchinger *et al.*, 1998).

In this study, the segregation of the F_1 progenies of the various crosses into the five disease severity classes demonstrates polygenic inheritance of resistance to CMD in the cassava landraces and the improved clones derived from 58308. This is

in agreement with earlier studies on CMD resistance on the 58308 source of resistance (Hahn and Howland, 1972; Jennings, 1976) and later among some of the landraces and the improved cassava accession (Lokko *et al.*, 1998). Akano *et al.* (2002), however, reported the presence of a major dominant gene for resistance in a resistant African landrace TME3 based on single marker regression analysis of a segregating F_1 mapping population. The presence of resistant phenotypes in the crosses involving susceptible by susceptible parents implies that the susceptible accessions, five of which were landraces, possess recessive resistance genes. This is also in agreement with studies on the 58308 source of resistance (Hahn and Howland, 1972). The presence of transgressive segregation is further evidence of polygenic control and allelic difference for resistance to CMD, where the alleles from diverse accessions act cumulatively to enhance the degree of resistance in their progenies.

The minimum number of effective factors affecting a quantitative trait is expected to range from 1 to 2 multiplied by the haploid number of the species (Lynch and Walsh, 1998). In this study, the number of effective factors estimated ranged from 2 to 5. Linkage, gene interaction such as epitasis, and the presence of transgressive segregants could cause a reduction in the estimated number of effective factors (Fenster and Ritland, 1994). However, there are no previous reports on the number of effective factors responsible for resistance to CMD to compare with these results. The results showed that both the resistant improved accessions and resistant landraces are capable of contributing resistant genes to their progenies. It also shows that resistant genes from different sources of resistance complement one another to enhance resistance. The ability of susceptible accessions to contribute effective factors towards progeny resistance has significant importance in cassava breeding. This is from the stand point that enhanced resistance to CMD would be incorporated into new genotypes being developed when a susceptible accession with the desired agronomic traits is a parent.

Wang *et al.* (1998) showed that if the genes for resistance in the parents involved in resistant by resistant crosses are allelic, there would be no plants with mosaic symptoms. In this study, the occurrence of susceptible progenies in the resistant by resistant crosses suggested allelic differences for CMD resistance genes among the resistant accessions. Although the analysis of variance did not reveal significant difference in the response of the resistant parents used in the NCD-II which all the majority of resistant parents with the exception of 58308, the GCA effects of the males was significant, and the Cochran-Mantel-Haenszel statistic revealed the individual resistant parents which contributed to the significant differences among crosses detected in the analysis of variance. Furthermore significance of the FxM SCA effect implied that some crosses were either more resistance or more susceptible than average, and again the pairwise test revealed the parental genotypes whose progenies contributed to this variation. The pair-wise test of the individual crosses revealed allelic differences for resistance to CMD between resistant genotypes TMS4(2)1425 and TME9, TME12 and TME14, between TMS60142 and TME5, between TMS90257 and TME4, TME5, TME12, and TME14, between TME5, and TME8, TME12, and TME14 and between TME11 and TME12, when these genotypes were used as parents. The lack of significant differences for other sources of resistance indicates that they are similar in the expression of resistance in their crosses. However, differences in the mean disease severity score and number of effective factors contributed by the various resistant accessions showed that they differed by minor genes which were influenced by the specific cross.

While both the analysis of variance and the Cochran-Mantel-Haenszel statistic did not detect significant differences due to reciprocal effects, significant differences among some sources of resistance detected when either TMS30555 or TMS30572 was the female parent, and the lack of significant differences among all sources of resistance when TMS30001 was the female parent indicates that the expression of resistance is influenced by the nature of the female parent. The resistant landrace TME5 for instance, showed significant differences from improved accessions, TMS60142 and TMS90572 and landraces TME8, TME12, and TME14 only when TMS30555 was the female parent.

As was expected, in many cases alleles

contributed by the susceptible parents towards resistance were different from those contributed by the resistant parents but in others, the susceptible accessions differed from the resistant accessions only in minor genes which expressed differently in the specific crosses.

CONCLUSION

Resistance to CMD in the accessions studied is under a complex genetic control and both resistant and susceptible accessions contribute favourable genetic factors towards resistance in their progenies. There are allelic differences between the various sources of resistance to CMD and these complement one another to increase the number of progenies with enhanced resistance to CMD. These accessions are, therefore, a recommended source of germplasm to diversify resistance while developing new genotypes with desired agronomic traits and enhanced resistance to CMD.

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