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CORRELATED RESISTANCE OF CASSAVA TO MOSAIC AND BACTERIAL BLIGHT DISEASES

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SUMMARY

The two most serious diseases of cassava (*Manihot esculenta* CRANTZ) are cassava mosaic disease (CMD) and cassava bacterial blight (CBB) (*Xanthomonas manihotis* STARR). Clone 58308, derived from the third backcross of the interspecific cross of cassava (*M. esculenta*) × ceara rubber (*M. glaziovii*), showed a high level of resistance to both diseases. Crosses of 58308 with several other clones which varied from susceptible to moderately susceptible to both diseases gave progenies with a significant genotypic correlation between resistance to both diseases ($r = 0.90$), apparently due to linkage. The heritabilities of resistance to the diseases were estimated at 50–70% for CMD and 25–65% for CBB. Resistance to both diseases is assumed to be polygenic. The correlated response to selection for CMD and for CBB was estimated.

INTRODUCTION

Cassava mosaic disease (CMD), first reported in East Africa by Warburg in 1894 (c.f. STOREY & NICHOLS, 1938) is endemic in all cassava growing areas of Africa and India, causing yield reductions of 20–90%. The sole vector is a whitefly, *Bemisia tabaci* (STOREY & NICHOLS, 1938; CHANT, 1958). The causal agents have not been identified, but virus etiology is suspected (STOREY & NICHOLS, 1938). Vegetative propagation ensures the perpetuation of the disease, and resistant cultivars are the only means of effective control. Breeding for resisrance was initiated by Storey and Nichols in East Africa in 1937 (NICHOLS, 1947). Resistance was not found in cultivars from the other cassava growing countries but the progenies of the third backcross of cassava (*Manihot esculenta*) × ceara rubber (*Manihot glaziovii*) showed promise (NICHOLS, 1947). Effective resistance was not found in East Africa, nor were the clones from interspecific crosses as productive as true cassava selections (JENNINGS, 1972). Research was discontinued in 1958.

Breeding for resistance to CMD was initiated in Nigeria by Beck in 1955 (EKANDEM, 1970), who introduced from East Africa seeds of an interspecific hybrid and identified the clone 58308 as resistant in 1958. Although the breeding work was discontinued in 1961, resistant clone 58308, fortunately, was maintained and showed continuous resistance for 20 years under heavy infection pressure; but the yield was very poor, both in quantity and quality (HAHN et al., 1973).

Resistance to CMD was reported by NICHOLS (1947) to be genetically controlled and

DOUGHTY (1958) suggested that it was multigenic. HAHN & HOWLAND (1972) postulated polygenic inheritance. From a seven parent diallel cross it was concluded that CMD resistance was recessive with a heritability of about 60% (HAHN et al., 1973).

Cassava bacterial blight (CBB) was first recorded in Brazil in 1912, and occurs in several other South American countries (LOZANO, 1975). In Africa it has only recently been recognized (IITA, 1972; WILLIAMS et al., 1973). The disease is now reported from many countries in Africa (TERRY, 1978), and has also been reported from Asia (LEU & CHEN, 1972; CHAN et al., 1975; BOONSUE & SINTHUPRAMA, 1975; DHARNAPUTRA, 1975).

The disease is devastating in Africa, especially in Nigeria and Zaire where cassava production is highest. It has caused complete loss of tuberous root yield, leaves and planting material during severe epiphytotics (TERRY, 1975). The potential for spread is very high as the disease is disseminated by vegetative cuttings, insects, humans, animals, tools, rain splash, infected leaves and wind-borne pellets carrying bacteria. Genetic resistance is the only effective means of control.

The clone 58308, which is resistant to CMD, is also resistant to CBB. It has produced many highly resistant progenies and has a high parental value for CBB resistance. Resistance appears to be polygenic and largely additive in nature (HAHN et al., 1974).

Our study investigated the genetic relationship between resistance to CMD and CBB, the heritabilities of resistance to both diseases and the correlated response to selection. The work was conducted at the International Institute of Tropical Agriculture (IITA) from 1972 to 1977.

MATERIALS AND METHODS

In 1971, several crosses were made between clone 58308 (resistant to both CMD and CBB), Isunikakiyan (moderately resistant to CMD and CBB), 58198 (moderately resistant to CMD and CBB) and cultivar 60444 (susceptible to both diseases). In 1972 about 10000 F_1 hybrid seeds of these crosses were planted in the nursery and later seedlings were transplanted to the field at a spacing of 1 m square. Two plants each of 719 clones out of the 10000 were replanted in the same field in 1973. They were inoculated 3 months after planting with CBB bacteria during the rainy season using a stem puncture method. Plants were exposed to CMD by a high field population of CMD carrying whiteflies.

CMD scoring was based on five classes:

class 1 – apparent field resistance, no symptoms observed;
class 2 – a mild chlorotic pattern over entire leaflets, or mild distortion only at the base of the leaflets with the remainder of the leaflets appearing green and healthy;
class 3 – strong mosaic pattern throughout the leaf, narrowing and distortion of the lower one-third of leaflets;
class 4 – severe mosaic, distortion of two-thirds of the leaflets and general reduction of leaf size;
class 5 – severe mosaic, distortion of all leaflets, twisted and misshapen leaves.

CBB scoring was based on 5 classes:

class 1 – no symptoms;
class 2 – only angular leaf spotting;

class 3 – extensive leaf blight and leaf wilt, defoliation, gum exudation on stems and petioles;
class 4 – extensive leaf blight, wilt, defoliation and stem die-back;
class 5 – complete defoliation and stem die-back, stunting and die-back of lateral shoots.

The first reading was normally made at three to four months and the second at six months after planting.

Fifty-two selected families (seedlings) from the 1973 seedling nursery were planted in a randomized complete block design with two replications in two locations in 1974. Ten seedlings per plot were planted at a spacing of 1 m on single ridges 1 m apart. However, only the data from one location was used for analysis because of rodent damage at the other location.

About 1000 clones selected from the 10000 seedlings of 1972 were planted on 7 m ridges without replication in 1973. However, standard cultivator 60444 was planted every 10 rows. Open pollinated seeds were collected in 1973 from each clone of the 483 selected out of the 1000 and per half-sib progeny 20 seedlings were planted in the field in 1974. The progeny mean scores of both CMD and CBB were regressed on the respective parent scores to estimate heritability by multiplying by 2.

In 1975, crosses of 60444 × 58308 and Isunikakiyan × 58308 were made. The 62 and 76 hybrid clones resulting from these crosses, respectively, were planted in 1976 with rooted green stem cuttings in a randomized complete block design with three replications, one plant being planted in each plot. The mean CMD and CBB scores of the three plants were used to compute correlation coefficients. Other planting and inoculation methods were the same as in earlier experiments.

RESULTS

Correlation. Correlation coefficients between resistance to CMD and CBB were computed using the data of individual plants from the four crosses made in 1971 and planted in 1972 and 1973. There was a significant correlation between CMD and CBB resistance for the 58308 × Isunikakiyan cross in 1972, but other crosses showed no significant correlations. In 1973 the crosses of 58308 × Isunikakiyan and 58308 × 60444 showed no significant correlations, but 58308 × 58198 and Isunikakiyan × 60444 showed significant correlations between resistance to CMD andCBB. Correlation coefficients between CMD and CBB resistance obtained using the replicated data for two crosses made in 1975 and tested in 1976, are presented in Table 1. Highly significant correlation coefficients were found for both crosses.

Table 1. Correlation coefficients (r) between CMD and CBB resistance computed with replicated clonal data for two crosses.

Cross	Clones	r
60444 × 58308	62	0.85**
Isunikakiyan × 58308	76	0.51**

**Significant at 1 % level.

Table 2. Repeatability (seedlings vs. clone) of CMD and CBB incidences.

Cross	n	CMD	CBB
58308 × Isunikakiyan	252	0.41**	0.99**
60444 × 58308	193	0.31**	1.00**
60444 × Isunikakiyan	83	0.11	0.98**
58308 × 60444	128	0.35**	1.00**
58308 × 58198	63	0.25*	0.92**

*, ** Significant at 5% and 1% levels, respectively. n = number of observations.

Repeatability. In order to investigate whether the observations of CMD and CBB at the seedling stage are related with those at the subsequent clonal stage, correlations of both CMD and CBB data of 1972 seedlings and 1973 clones from the seedlings were obtained and are given in Table 2.

Repeatabilities were determined for CMD and CBB measurements and were significant, but the former were intermediate to low for CMD, but were very strong for CBB, suggesting that there was in general weak agreement for CMD but good agreement for CBB between the observations made at the seedling stage and those at the clonal or cutting stage.

Heritability. Narrow sense heritabilities of resistance to CMD and CBB were estimated by multiplying by 2 regression coefficients of the half-sib progeny mean scores of both CMD and CBB (1974 data) on respective parent scores (1973 data). Both CMD and CBB showed significant heritabilities, CMD being higher with 35% and 30% than CBB with 28% and 16% for the first and second readings.

Broad sense heritabilities of CMD and CBB resistance also estimated from variance components from half-sib family trials of 1974, were 44% for CMD and 48% for CBB. Broad sense heritabilities of CMD and CBB were also estimated as 68% and 69%, respectively, using the unreplicated data of only 952 clones out of 1000 planted in 1973 with the standard cultivar (156 observations) planted every 10 rows. Error variance was estimated using the data of the standard cultivar.

Phenotypic and genotypic correlations. Phenotypic and genotypic correlations between resistance to CMD and CBB were estimated as 0.4228** and 0.8992**, respectively from the data for half-sib families of 1974. Phenotypic and genotypic correlations were also estimated using 952 clonal data of CMD and CBB as 0.0051 and 0.6891, respectively, the former being not significant but the latter highly significant.

Correlated response to selection. From the half-sib family trial of 1974, genotypic correlation (r_{gxy}) between CMD and CBB was found to be 0.8992, heritabilities (h^2) of CMD and CBB resistance 56% and 24%, respectively and genetic variance components of CMD and CBB $\sigma_{gx}^2 = 0.3798$ and $\sigma_{gy}^2 = 0.0766$, respectively. With these genetic statistics, correlated response to selection for CMD and vice versa were estimated according to FALCONER (1960) as follows:

$$\Delta G_y = r_{g_{xy}} \frac{\sigma_{g_y}}{\sigma_{g_x}} \Delta G_x = 0.4038\ \Delta G_x$$

$$\Delta G_x = r_{g_{xy}} \frac{\sigma_{g_x}}{\sigma_{g_y}} \Delta G_y = 2.0023\ \Delta G_y$$

where ΔG_x is genetic gain of CMD and ΔG_y of CBB, and σ_{g_x} and σ_{g_y} are square roots of genetic variance components of CMD and CBB.

DISCUSSION

Cassava is a vegetatively propagated and normally cross pollinated crop that is genetically heterozygous. Cassava (*M. esculenta*) has 2n = 36 chromosomes and is probably an allotetraploid (JENNINGS, 1970). Ceara rubber (*M. glaziovii*) also has 2n = 36 chromosomes. The two species are closely related. The pachytene karyotype of ceara rubber has several features in common with cassava, however, there are a number of differences. Pachytene analysis of the F_1 hybrid of the species confirmed several of the karyological differences established by comparative karyology of the parents. The pachytene analyses of the backcross plants provided evidence for random transmission of at least some of the parental chromosomal types through the male gametes of the F_1 hybrid (personal communication, MAGOON, 1971).

LUSH (1948) and LERNER (1958) suggested that if genotypic correlation is due to pleiotropic genes, selection for two traits in the same direction will cause a negative change. However, our study indicates a different type of gene effect.

Clone 58308, a source of resistance to CMD and CBB was derived from the third backcross of *M. esculenta* × *M. glaziovii*, the latter being a source of high level resistance to both diseases. When selection was applied to the progenies from the cassava crosses with 58308, correlated responses of CMD resistance with that of CBB were observed and it was possible to improve cassava for resistance to both diseases after a few generations of selection (IITA, 1972–77). Therefore, it could be postulated that correlation between resistance to CMD and CBB may not be due to pleiotropic genes, but may be due to linkage of genes for the two attributes. A report by MAGOON (personal communication, 1971) on random transmission of some of the parental chromosomal types of the F_1 hybrids of the species into the backcross progenies to cassava, further supports the possibility of linkage between genes for CMD and CBB resistance rather than pleiotropism.

Resistance to CMD and CBB is assumed to be polygenic (HAHN & HOWLAND, 1972; HAHN et al., 1974). Therefore, resistance to the diseases must be attributed to the combined action of a number of loci which are linked on a chromosome or a set of chromosomes of a genome or genomes. Since cassava is genetically heterozygous and probably an allotetraploid, a study on the genetic mechanism of resistance to CMD and CBB is rather complicated and difficult.

When interspecific crosses with ceara rubber as a source of resistance to CMD and CBB are made, an appropriate breeding method to break the linkage of the resistance genes and those governing poor agronomic characters and to accumulate the resistance genes should be adopted in order to effectively incorporate these genes controlling resistance to CMD and CBB, while retaining other desirable genes of cassava. Population improvement with a half-sib family selection scheme having relatively large

families was successfully used to improve cassava for resistance to these two major diseases and for high yield, quality and resistance to lodging (IITA, 1972–77).

Genetic correlation between resistance to CMD and CBB indicates that selection for CMD resistance in a breeding program also may result in genetic progress in CBB resistance and vice versa. This has not been proven experimentally but our breeding experience indicates that it is very likely as shown by the rapid progress in developing resistance to both CMD and CBB after a few generations of selection. Theoretically, genetic gain in CMD resistance in response to selection for CBB resistance should be greater than that in CBB resistance when selection is made for CMD resistance.

CBB incidence is dependent upon environmental conditions, especially with amount and distribution of rainfall and with temperature, but CMD incidence is less variable under IITA conditions because of high vector population. Therefore, favorable environmental conditions for CBB symptom expression need to be provided for effective selection of cassava for resistance to CBB. Frequent overhead irrigation and inoculum must be applied. This environmental influence is reflected in the lower heritability estimates of CBB resistance compared with that of CMD. Use of replicated data may improve its heritability estimate, however, higher repeatability of CBB scores than those of CMD indicated that once individuals or clones are selected for resistance under favorable environmental conditions, there will be better agreement between scoring at seedling and clonal stages than in CMD.

More than 2000 cassava germplasm accessions from Latin America were very susceptible to CMD and CBB (IITA, 1973-77), even though Central Latin America is the origin of cassava and related species. Germplasm from Brazil has shown resistance to CBB, even though accessions were not as highly resistant as the progenies from the interspecific cross with *M. glaziovii*. LOZANO (1975) reported that the pathogen of CBB in Latin America is the same as that in Africa. Nine isolates of the CBB pathogen differed in their behavior on three cultivars, but resistant clones were resistant to all the isolates (IITA, 1977). This suggests that resistance to CBB is race-non-specific in nature (PARLEVLIET & ZADOKS, 1977). Resistance to CMD has been effective in many countries in West Africa, Central Africa, East Africa and India (IITA, 1973–77). Whether or not resistance to CMD over localities will prove to be race-non-specific depends on information on pathogenic variation.

REFERENCES

BOONSUE, B. & S. SINTHUPRAMA, 1975. Country presentation: The international exchange and testing of cassava germplasm. Proc. Interdisciplinary Workshop. CIAT, Palmira, Colombia. IDRC-049e: 26–28.

CHAN, S. K., S. L. TAN, & S. L. GEH, 1975. Country presentation: Malaysia. The international exchange and testing of cassava germplasm. Proc. Interdisciplinary Workshop. CIAT, Palmira, Colombia. IDRC-049e: 19–20.

CHANT, S. R., 1958. Studies on the transmission of cassava mosaic virus by *Bemisia spp.* (Aleyrodidae). Ann. App. Biol. 46(2): 210–215.

DHARNAPUTRA, T. S., 1975. Country presentation: Indonesia. The international exchange and testing of cassava germplasm. Proc. Interdisciplinary Workshop. CIAT, Palmira, Colombia. IDRC-049e: 13–14.

DOUGHTY, L. R., 1958. Cassava breeding for resistance to mosaic and brown streak viruses. 1958 EAFRO Annual Report: 48–55.

EKANDEM, M. J., 1970. Cassava research in Nigeria before 1967. Federal Report of Agric. Research, Ibadan, Nigeria. Memo No. 103.

FALCONER, D. S., 1960. Introduction to quantitative genetics. Ronald Press Co., New York. pp. 365.
HAHN, S. K. & A. K. HOWLAND, 1972. Breeding for resistance to cassava mosaic disease. Proc. Cassava Mosaic Workshop. Ibadan, Nigeria. pp. 37–39.
HAHN, S. K., A. K. HOWLAND & E. R. TERRY, 1973. Cassava breeding at IITA. Proc. Int. Trop. Root Crop. Symp., Ibadan, Nigeria. pp. 4–10.
HAHN, S. K., A. K. HOWLAND & C. A. OKOLI, 1974. Breeding for resistance to cassava bacterial blight at IITA. Proc. Workshop on Cassava Bacterial Blight in Nigeria. Umudike, Nigeria. pp. 11–14.
IITA, 1972. 1972 Annual Report of Root, Tuber and Vegetable Improvement Programs. IITA, Ibadan, Nigeria.
IITA, 1973. 1973 Annual report of root and tuber improvement program. IITA, Ibadan, Nigeria.
IITA, 1974–1977. Annual Reports. IITA, Ibadan, Nigeria.
JENNINGS, D. L., 1970. Cassava in Africa. Field Crop Abst. 23: 271–278.
JENNINGS, D. L., 1972. Breeding for resistance to cassava viruses in East Africa. Proc. Cassava Mosaic Workshop. Ibadan, Nigeria. pp. 40–47.
LERNER, I. M., 1958. Population genetics and animal improvement. Cambridge University Press, pp. 298.
LEU, L. S. & C. T. CHEN, 1972. Bacterial wilt of cassava *Manihot utilissima* POHL, caused by *Xanthomonas manihotis* (ARTHUR-BERTHET) STARR. Plant Prot. Bull. (Taiwan), 14(1): 17–26.
LOZANO, J. C., 1975. Bacterial blight of cassava. PANS 21(1): 38–43.
LUSH, J. L., 1948. The genetics of populations. Iowa State University, Ames, Iowa. pp. 381.
NICHOLS, R. F. W., 1947 Breeding cassava for virus resistance. East Afr. Agric. Journal 12: 184–194.
PARLEVLIET, J. E. & J. C. ZADOKS, 1977. The integrated concept of disease resistance: a new view including horizontal and vertical resistance in plants. Euphytica 26: 5–21.
STOREY, H. H. & R. F. W. NICHOLS, 1938. Studies on the mosaic diseases of cassava. Ann. appl. Biol., 25: 790–806.
TERRY, E. R., 1975. Cassava bacterial blight. International exchange and testing of cassava germplasm in Africa. Proc. Interdisciplinary Workshop. IITA, Ibadan, Nigeria. IDRC-063e: 23–25.
TERRY, E. R., 1978. Cassava bacterial diseases. Proc. Cassava Protection Workshop. CIAT, Cali, Colombia. Pp. 75–84.
WILLIAMS, R. J., S. D. AGBOOLA & R. W. SCHNEIDER, 1973. Bacterial wilt of cassava in Nigeria. Plant Disease Reporter 57(10): 821–827.