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Additive inheritance of resistance to pod rot caused by *Phytophthora palmivora* in cocoa

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Summary. Quantitative inheritance of resistance to *Phytophthora* pod rot (Ppr) was studied in cocoa hybrid progeny from 12 Trinitario × Amazonian crosses and their reciprocal crosses. The crossing scheme was similar to a factorial design. Disease was assessed by the number and percentage of infected pods on each tree. Highly significant differences due to general combining abilities (GCA) were obtained for all characters, except for the GCA of Trinitario on total pod production. Differences for specific combining ability (SCA) were not significant for all characters. There were no significant differences between reciprocal crosses. The Trinitario clone K82 provided the only source for the hybrid progenies of strong Ppr resistance to the hybrid progenies, while K20 provided moderate resistance. Other parental clones – KA2-101, KA5-201, KEE 2, KEE 5, and KEE 52 – produced progenies which were susceptible to Ppr. It is evident that resistance to Ppr in cocoa is inherited additively. Maternal and cytoplasmic effects were assumed to have no influence on inheritance of resistance. It is also concluded that resistance to Ppr of the kind shown by K82 is likely to be horizontal resistance. Breeding for high-yielding cultivars combined with Ppr resistance is the most effective way of controlling Ppr of cocoa on the crops of growers with small holdings in Papua New Guinea.

Key words: Cocoa – Disease resistance – *Phytophthora* pod rot – General combining ability – Reciprocal crosses

Introduction

In Papua New Guinea, vascular-streak dieback, caused by *Oncobasidium theobromae*, and pod rot (Ppr) and canker, caused by *Phytophthora palmivora* (Butl.) Butl., are the three major diseases in cocoa (Tan 1981, 1987). Recently, pink disease caused by *Corticium salmonicolor* has also become a problem in some areas. Ppr has been estimated to destroy on average 10% of the world cocoa crop annually (Gregory 1985). From the long-term records of the Lowland Agriculture Experimental Station (L.A.E.S.) at Keravat, yield loss from Ppr was high in the late 1950s (Thrower 1960), and declined to a low level from 1962 to 1968 (Hicks 1975). The incidence of Ppr gradually increased until in 1980 it caused about a 40% loss of crop (McGregor 1981). Yield loss is even higher for some very susceptible clones, being estimated at from 50% to 100% (McGregor 1981). In order to reduce the great economic losses due to Ppr, attempts have been made to control it by the application of fungicides, and adopting breeding and cultural measures. However, under the current low market price of cocoa beans, a major drawback to control of Ppr by fungicides is the increased cost of their production. Most importantly, chemical sprays for control of Ppr are rarely used by growers with small holdings, who are now producing more than 60% of the crop in Papua New Guinea. Therefore, breeding for disease resistance is the most economical and acceptable method of disease control.

Several studies have reported on the inheritance of resistance to Ppr under field conditions in different countries. Results suggested that Ppr resistance in cocoa was inherited quantitatively and might be controlled by dominant or partial-dominant genes (Rocha 1974; Soria 1974, 1978; Soria and Esquivel 1970). Recent evidence has shown that substantial genetic variations were attributable to additive gene effects in controlling Ppr resistance when assessed by artificial inoculation techniques (Sitapai and Kennedy 1988). Selection for high-yielding trees with low susceptibility to Ppr in the resistant crosses was possible (Amponsah 1988).

The present study was conducted to determine the relative importance of additive and nonadditive genes for field resistance to Ppr in cocoa. Disease severity was expressed as the number and percent of infected pods per tree among the progenies. The relationship between resistance and pod production and yield, and its implication in the breeding program for developing high-yielding and Ppr-resistant cultivars, are discussed.

Materials and methods

Four Trinitario female parents (K20, K82, KA2-101, and KA5-201) were crossed with three Amazonian male parents (KEE 2, KEE 5, and KEE 52) in all combinations. Reciprocal crosses were also made using the Amazonian clones as female parents and the Trinitario clones as male parents. Information on the resistance to Ppr of the seven parents was obtained either from the observation blocks or from clonal trials at L.A.E.S. (Tan 1987). K82 was consistently rated as resistant and K20 as less susceptible, while all other clones suffered from heavy pod loss due to Ppr. The 24 hybrid progenies were planted in the field in August 1983. The experiment was arranged in a random complete-block design with four replicates. Each plot consisted of 16 trees, spaced in a 4-m triangular pattern, giving a density of approximately 721 trees ha^{-1}. The entire experimental block was surrounded by at least four to five guard rows. Fertilizer was applied uniformly to each tree (N : P : K, 15 : 15 : 15) at the rate of 3×280 g $tree^{-1}$ $year^{-1}$. Overhead shade (*Gliricidia* species) was maintained at a level of about 80% interception. This, combined with no canopy pruning on cocoa trees, provided favorable conditions for infection by *P. palmivora*.

Pod production was recorded for each individual tree at bi-weekly intervals. The number of healthy and infected pods was recorded from 1985 to May 1989. The percentage of Ppr infection was calculated from the whole recording period on a yearly basis, whereas high level of percent Ppr only accounts for the months with high Ppr incidences during the wet season, from the period January 1988 to May 1989. Potential yield of dry beans per hectare from each progeny was estimated from total pod production (the sum of healthy and infected pods) divided by pod value and multiplied by 721 (trees ha^{-1}) and a correction factor of 0.83.

Analysis of variance was performed in the same way as for the factorial design of Comstock and Robinson (1952), which enabled the total variation to be partitional into three main sources, i.e., general combining ability (GCA) of Trinitario, Amazonian, and differences between reciprocal crosses. Variation for interactions of Trinitario × Amazonian, reciprocal × Trinitario, reciprocal × Amazonian, and Trinitario × Amazonian × reciprocal was also obtained. Differences in GCA were regarded as being due to additive genes, and differences in specific combining ability (SCA) (Trinitario × Amazonian) as being due to nonadditive genes. The relative importance of GCA and SCA contributing to the total variation of the hybrid progenies for characters associated with resistance and yield was expressed as r^2 values, according to the method described by Simmonds (1979). Estimates of GCA effects for Trinitario and Amazonian parents for Ppr resistance and their respective standard errors were calculated according to Park and Gerhold (1986). The negative values of GCA effects indicated good combiners for number and percent of infected pods, because lower values indicated more resistance to Ppr. Conversely, good combiners for total pod production and potential yield should have positive GCA effects.

Results

The initial level of inoculum of *P. palmivora* in the experimental block was low, as shown by the average of only 3.9% and 8.4% infected pods in 1986 and 1987, respectively. From January 1988 to May 1989, inoculum had built up to a high level, resulting in an average of 21.3% infected pods for the 24 progenies being tested. Therefore, data from this period were used for the interpretation of resistance to Ppr. Significant correlation coefficients were found among all four replicates for both number and percent of infected pods ($r = 0.42–0.68$, $df = 22$). The results show that during that period, inoculum was uniformly distributed over the experimental area and that the resistant and susceptible progenies were well differentiated in all replicates. The inoculum level was evidently high throughout the area, since certain plots containing susceptible progenies suffered more than 53% pod loss due to Ppr.

Rainfall was found to be moderately correlated with Ppr incidence ($r = 0.45$, $df = 22$) over a period of 2 years. A coefficient of determination of about 20.3% suggested that there were other environmental factors also influencing Ppr infection. However, high and low incidences of Ppr can still be clearly differentiated from the monthly records in this experiment.

Mean values of number of healthy pods, total pod production, and number and percent of infected pods recorded in 1988 to May 1989 for the 12 Trinitario × Amazonian crosses and their reciprocal crosses are shown in Table 1. Hybrid progenies displayed a wide range of susceptibility to Ppr, from less than 10% Ppr infection for crosses with K82 as one of the parents to greater than 30% for the KA5-201 crosses. Analysis of variance (Table 2) revealed that highly significant differences were obtained for GCA of both Trinitario and Amazonian for all characters studied, with the exception of GCA of Trinitario on total pod production. The results are further illustrated in Fig. 1, where observed data (Y) are plotted against the predicted values (X) from parental GCA for the four Ppr-resistant characters (Simmonds 1979). High r^2 values were obtained, indicating that GCA accounted for a very large portion of variation between hybrid crosses for these characters, which ranged from 81% for number of healthy pods to 91% for percentage of Ppr measured at high-level infection. Very high r^2 values were also found for total pod production (82%) and potential yield (80%). Nonsignificant differences were found for reciprocal crosses and SCA was found for all characters. Interactions between reciprocal and Trinitario were nonsignificant for all characters, while significant difference ($P = 0.05$) for reciprocal × Amazonian was obtained for percent of infected pods measured during the months when Ppr incidence was high. These results provided evidence that whether or not

Table 1. Mean values of Trinitario × Amazonian (T × A) crosses and their reciprocal crosses (A × T) for number of healthy pods and infected pods, Ppr (%), pod production, and potential yield

Progeny	No. of healthy pods no./tree/yr		No. of infected pods no./tree/yr		Ppr %		HL Ppr%[a]		Pod production no./tree/yr		Potential yield kg/ha/yr	
	T×A	A×T	T×A	A×T	T×A	A×T	T×A	A×T	T×A	A×T	T×A	A×T
K20 × KEE2	50.6	65.3	18.8	12.0	27.0	15.5	40.4	28.5	69.5	77.4	2,132	2,040
K20 × KEE5	59.7	59.2	14.5	14.7	19.5	19.8	34.8	33.8	74.2	74.0	2,627	2,380
K20 × KEE52	41.1	37.2	9.7	10.7	19.0	22.3	33.1	38.7	50.9	47.9	1,570	1,825
K82 × KEE2	61.8	62.0	6.1	6.7	8.9	9.7	21.0	20.9	67.9	68.7	1,736	1,575
K82 × KEE5	69.8	66.7	7.3	7.5	9.4	10.1	23.9	23.1	77.1	74.2	2,284	2,074
K82 × KEE52	37.2	45.1	8.0	7.7	17.6	14.5	24.0	28.8	45.3	52.8	1,426	1,698
KA2-101 × KEE2	56.2	55.2	18.0	11.8	24.2	17.5	35.3	29.6	74.2	67.1	1,873	1,587
KA2-101 × KEE5	57.7	45.0	15.5	14.0	21.1	23.7	33.5	39.2	73.3	59.0	2,345	1,801
KA2-101 × KEE52	40.6	39.9	15.7	14.7	27.8	26.8	43.3	41.7	56.3	54.7	2,066	1,971
KA5-201 × KEE2	59.9	62.2	20.4	16.5	25.3	20.8	39.7	32.9	80.4	79.0	2,186	2,119
KA5-201 × KEE5	45.8	46.2	19.2	22.3	29.5	32.5	40.9	43.5	65.0	68.6	2,477	2,319
KA5-201 × KEE52	41.2	38.3	15.7	17.2	27.4	30.9	41.0	43.6	57.1	55.6	1,919	1,860
Experimental mean	51.8		13.5		20.8		33.9		65.4		1,996	
LSD (0.05)	14.4		5.8		9.4		8.2		14.0		649.8	

[a] Data only from months with high levels of Ppr infection

Table 2. Combining analysis of variances for pod production, number of healthy pods, number of diseased pods, percent Ppr, and potential yield

Sources of variation	*df*	No. of healthy pods	No. of infected pods	Ppr (%)	HL[a] Ppr (%)	Total pod production	Potential yield
GCA (Trinitario)	3	780.2*	1,215.67**	1,130.11**	1,003.67**	138.5	994,312**
GCA (Amazonian)	2	7,626.8**	70.41	174.73*	391.62**	9,024.0**	3,322,365**
SCA (Trinitario × Amazonian)	6	540.3*	54.85	68.08	50.56	500.3*	469,069*
Reciprocal (R)	1	0.4	63.37	32.82	45.26	53.3	186,180
R × T	3	168.6	31.90	10.75	5.84	273.1	593,169*
R × A	2	295.4	119.57	131.07	137.44*	81.9	544,146
R × T × A	6	178.9	20.56	40.03	52.84	134.7	94,551
Error	69	235.4	38.23	45.45	34.52	220.9	208,000
r^2		0.81	0.90	0.86	0.92	0.82	0.80

*,** Significant at the 5% and 1% levels of probability, respectively
[a] Data from months with high level of Ppr infection

Table 3. Estimate of GCA effects and standard errors from the seven parental clones in Ppr resistant characters, production, and potential yield

Parental clones	No. of healthy pods	No. of infected pods	Ppr	HL Ppr	Pod production	Potential yield
Trinitario						
K20	0.3 (3.0)[a]	−0.1 (2.8)	−0.3 (2.6)	0.9 (2.0)	0.2 (3.0)	99 (172)
K82	5.2 (2.6)	−6.3 (1.8)	−9.2 (1.9)	−10.3 (1.9)	−1.1 (2.5)	−197 (127)
KA2-101	−2.7 (2.3)	1.4 (2.0)	2.7 (3.2)	3.1 (1.7)	1.3 (2.6)	−55 (138)
KA5-201	−2.9 (3.1)	5.0 (2.6)	6.9 (2.6)	6.3 (2.0)	2.2 (3.4)	155 (156)
Amazonian						
KEE2	7.3 (4.6)	0.2 (3.9)	−2.2 (4.0)	−2.9 (3.2)	7.6 (4.4)	−90 (212)
KEE5	4.4 (3.4)	0.8 (2.9)	−0.1 (2.8)	0.1 (2.4)	5.2 (3.7)	292 (171)
KEE52	−11.7 (4.1)	−1.1 (2.6)	2.4 (3.4)	2.8 (2.8)	−12.8 (4.5)	−200 (100)

[a] Standard error

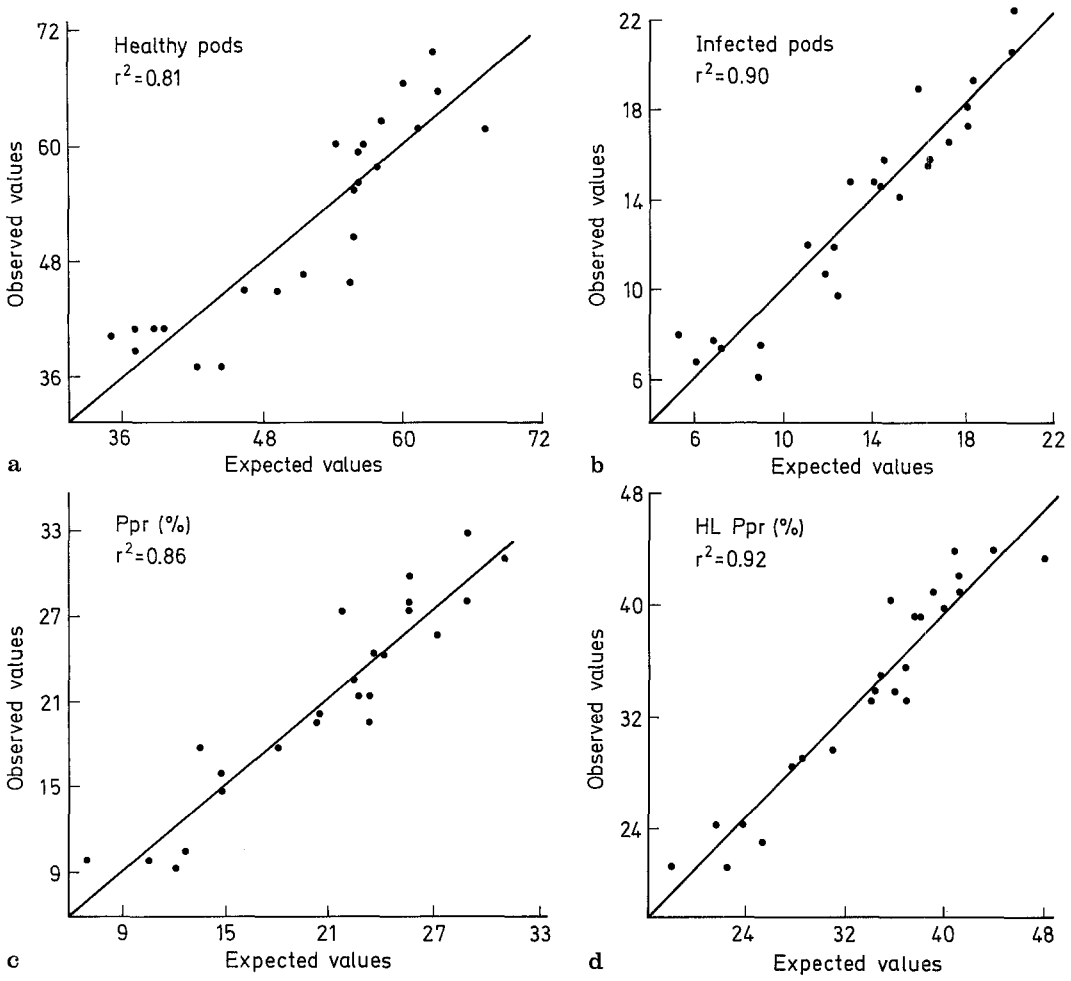

Fig. 1 a–d. Combining abilities for four *Phytophthora* pod rot related characters (**a–d**), observed values of progenies plotted against expected values from parental GCA

Amazonian parents were used as male or female parents had no effect on their progenies for disease resistance, except on percentage of Ppr at high level.

Estimates of GCA effects of the Trinitario and Amazonian parents for pod production and Ppr resistance are given in Table 3. Trinitario clone K82 is the parent that produced progenies with Ppr resistance, while Trinitario clones KA5-201 gave progenies susceptible to Ppr. Estimates of GCA effects in Amazonian parents were associated with large standard error for all characters measured.

Intercharacter relationships among pod production, yield, and Ppr-resistant characters are given in Table 4. Percentages of Ppr from both measurements were positively correlated with the number of infected pods, but negatively correlated with the number of healthy pods. Pod production was highly correlated with the number of healthy pods and potential yield. No significant correlation was found between pod production and Ppr-resistant characters, either on number or percentage of infected pods. Surprisingly, there was positive correlation

Table 4. Intercharacters' correlation coefficients ($df=22$) for pod production, yield production, yield, and *phytophthora* pod rot resistant characters

Characters	Healthy pods	Infected pods	Ppr %	HL[a] Ppr %	Yield
Pod product	0.90**	0.23	−0.22	−0.20	0.58**
Healthy pods		−0.20	−0.62**	−0.58**	0.30
Infected pods			0.88**	0.84**	0.55**
Ppr (%)				0.96**	0.30
HL Ppr (%)					0.36
Yield					

*,** Significant at 1% level of probability
[a] Data only from months with high level of Ppr infection

($P=0.01$) between potential yield and number of infected pods. However, this result was somewhat different from the stepwise multiple regression analysis (Table 5) (Draper and Smith 1966). Of all the five characters used as independent variables in predicting yield, the number of healthy pods, number of infected pods, and pod value

Table 5. Stepwise multiple regression using *Phytophthora* pod rot resistant for the prediction of yield in cocoa

Independent variables	Standarized partial regression coefficients				
Healthy pod number	1.074**	1.042**	1.029**	1.209**	–
Infected pod number	0.402	0.445	0.470**	–	0.469
Ppr (%)	0.173	0.030	–	–	–
HL Ppr (%)	−0.094	–	–	–	
Pod value	−0.921**	−0.908**	−0.910**	−1.063**	−0.250
R^2	0.98 **	0.98 **	0.98 **	0.78 **	0.35 **

** Significant at 1% level of probability

were the major determinants of yield ($R^2=0.98$). The percentage of Ppr from both measurements was found to be unimportant. Adding these two variables into the equation did not significantly improve the model; R^2 value remained unchanged. Moreover, the number of healthy pods appeared to be more important than the number of infected pods in predicting yield. The contribution of healthy pod and pod value to the total variation ($R^2=0.78$, $P=0.01$) was greater than the total variation ($R^2=0.35$) which accounted for the number of infected pods and pod value in the regression equation.

Discussion

The present study demonstrates that Ppr resistance, expressed as the number of infected pod and percentage of Ppr infected pods measured during periods of both high and normal field levels of Ppr infection, is quantitatively inherited. It suggests that several genes are probably involved in Ppr resistance, although the number of genes and individual genes controlling resistance could not be accurately identified. This agrees with previous reports from other cocoa-growing areas in Africa and South America (Asare-Nyako and Amponsah 1973; Soria 1978; Partiot 1975). The results also further indicate that gene effects are predominantly additive for the characters associated with Ppr resistance, therefore, selection for resistant hybrid progenies must be effective in a breeding program.

Reciprocal differences were found to be nonsignificant for the various assessments of Ppr resistance. Therefore, maternal and cytoplasmic effects were assumed not to influence resistance. The Trinitario and Amazonian parents contributed their resistance equally to their offsprings, regardless of whether they were used as male or female parents. However, Blaha and Lotode (1977) observed some reciprocal differences among hybrid crosses for Ppr resistance.

From previous clonal trials, Trinitario K82 was known to have resistance to Ppr (Tan 1987). Present results also showed that K82, either as female or male parents, provided the best source of Ppr resistance to the

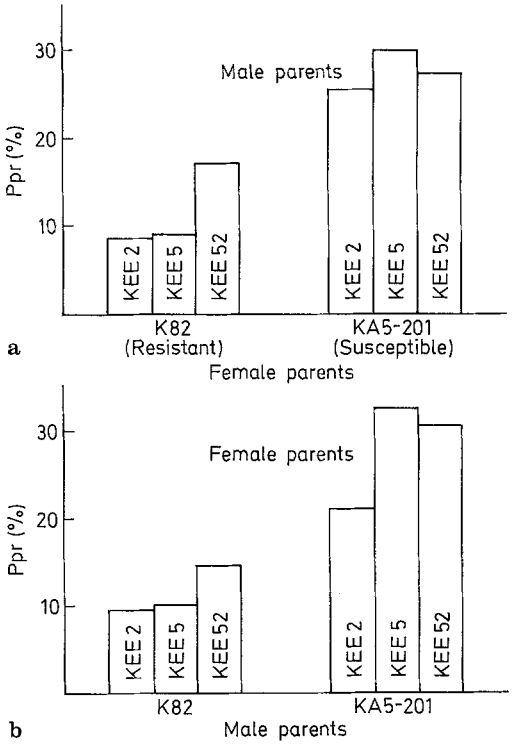

Fig. 2 a and b. Comparison among hybrid progenies with one of the parents being either resistant (K82) or susceptible (KA5-201) (**a** and **b**), and their reciprocal differences [(a) vs (b)], for Ppr (%)

progenies, while KA5-201 consistently produced Ppr-susceptible progenies (Fig. 2). K20 contributed moderate Ppr resistance in the crosses. Progenies involving KA2-101 also produced Ppr-susceptible progenies. The results agreed well with previous reports (Rocha 1974; Rocha and Vello 1971; Toxopeus 1974; Soria 1978; Soria and Esquivel 1970) that resistance to Ppr can be transmitted from the parents to the offspring. The higher the level of resistance of the clones used as parents in a breeding program, the higher the proportion of resistant individuals that can be obtained in the progenies.

The resistance to Ppr exhibited by K82 and its progeny is partial resistance. Infection still occurs in the resistant clone, but the number and proportion of infected

pods are lower than in clones regarded as being susceptible. The present results also revealed that none of the progenies was immune to Ppr infection. Polygenic, partial resistance is usually but not universally accepted as corresponding to "horizontal" resistance as defined by Vanderplank (1963). It is also usually regarded as durable resistance, because it presumably shows no interaction with races of the pathogen (Vanderplank 1963). Breeding for durable resistance is of great importance for this perennial crop. It must be emphasized that any resistance incorporated into high-yielding material must remain effective throughout the economic life of the plant material, which normally is at least 15 years. There is evidence from Papua New Guinea that Ppr resistance of clone K82 is indeed durable. K82 has been grown widely as commerical, vegetatively propagated plant material throughout Papua New Guinea. Its Ppr resistance still remains effective against the pathogen in different environments since it was first recognized in the early sixties (Hicks 1967, 1975).

Phytophthora pod rot occurs in Papua New Guinea and is caused by *Phytophthora palmivora* (Butl.) Butl., although at least four species of *Phytophthora* have been reported as pathogens of the disease in other countries (Zentmyer 1988). Different strains and mating types of *P. palmivora* have been reported in different cocoa-growing areas (Spence 1961; Zentmyer and Mitchell 1971; Ram and Figueiredo 1971). There is little information on different races of *P. palmivora* in Papua New Guinea. Arentz (1986) reported that all the isolates on cocoa pods and soil from cocoa plantations were identified as the A2 mating type, although the A1 mating type was also found in certain cocoa-growing areas and forest soils. Based on the present study, it is not possible to relate Ppr resistance to race nonspecificity, because presumably only one pathotype of *P. palmivora* was involved in the resistance.

The stepwise multiple regression analysis showed pod production was not related to any of the resistant characters. There was also no evidence that resistance was negatively associated with yield. This is in contrast to the Thorold effects (Thorold 1956), a phenomenon reported for cocoa in West Africa, but agreed with a recent report by Amponsah (1988). Therefore, it should be possible to combine high-yielding characters with Ppr-resistant characters, since both characters were controlled predominantly by additive genes. After all, breeding for high yielding remains the main objective in cocoa-breeding programs (Kennedy et al. 1987). K82 and another Ppr-resistant Trinitario clone (KA2-106) are being used extensively in seed gardens to produce new cocoa cultivars, which are composed of multiple lines with different degrees of Ppr resistance in combination with high yield, good pod and bean characters, and resistance to vascular-streak dieback (Tan and Tan 1988, 1989).

Environmental factors, especially temperature, rainfall, and humidity, are well-known critical factors in the development of Ppr disease in cocoa (Dakwa 1977). It is obvious that the macro- and micro-climate can affect disease development and epidemiology (Gregory and Maddison 1981). Similarly, good field management such as the use of sanitation, regular and complete harvesting, frequent pruning of cocoa trees, and low shade would reduce disease development and enhance the effect of resistance of genotypes with moderate or partial resistance to Ppr. Moreover, fungicide application can be integrated with use of resistance and cultural controls to provide maximum protection against crop loss due to Ppr in areas where the disease reaches epidemic levels.

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