Simulation of the Effects of Host Resistance, Reversion, and Cutting Selection on Incidence of African Cassava Mosaic Virus and Yield Losses in Cassava

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ABSTRACT

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A simulation model, developed earlier to describe epidemics of African cassava mosaic, was used to investigate the effects of resistance and sanitation on epidemic severity and cassava productivity in successive annual cropping cycles. Parameters characterizing host resistance, secondary spread within plantings, latent period, and yield losses were incorporated into the model. Resistance and sanitation were modeled in two ways: reversion (the percentage of healthy cuttings derived from infected plants) and preferential cutting selection (the ratio of the number of cuttings from a healthy plant to the number from an infected one). When reversion or cutting selection occurred for several successive crop cycles in highly resistant cultivars, disease incidence increased during the first few annual crop cycles but ultimately reached an equilibrium considerably below 100%. At this equilibrium stage, new infections caused by transmission of the virus by insect vectors balanced "escapes" through reversion or cutting selection, and yield losses were limited. Respective and combined effects of host resistance, reversion, and cutting selection on disease incidence and yield losses are assessed.

African cassava mosaic virus (ACMV), transmitted by the whitefly *Bemisia tabaci* (Gennadius) and perpetuated through cuttings, causes severe losses annually in all cassava (*Manihot esculenta* Crantz)-growing areas of Africa (29). Breeding programs have been conducted for several decades to incorporate resistance against the disease (19). Symptom severity was the initial criterion of selection, but resistant cultivars also have other characteristics. In particular, ACMV does not become fully systemic in highly resistant cultivars (3,18,19,27), and the virus titer remains low (13). Consequently, spread within and among plantings is relatively slow (23,24,29), and some cuttings propagated from infected plants may revert to healthy plants (3,18,19,27). This phenomenon has been observed in several African countries and is termed "reversion" (15,21) or "self-elimination" (25). Differences in growth between healthy and diseased cassava plants may lead to discrimination in favor of healthy, vigorous source plants when cuttings are collected by farmers. This selection would result in an underrepresentation of cuttings from infected stems (10). The individual and combined contributions of host resistance, reversion, and cutting selection for ACMV control are assessed here with the use of a simulation model developed from a 6-yr multidisciplinary project on the epidemiology of ACMV in Ivory Coast (7,12).

MATERIALS AND METHODS

Disease progress model. Assessment of the primary spread of ACMV was based on a monomolecular differential equation with a time-dependent rate, $r_P(t)$:

$$dy_1/dt = r_P(t)(1 - y_1), \quad (1)$$

where y_1 is the disease incidence caused by primary infection and t the time in months. The function $r_P(t)$ describes the rate of disease progress (when the month of planting, P, is fixed) and incorporates an overall negative exponentially changing susceptibility with host age, $a(t)$, and a sinusoidal temperature-driven seasonal fluctuation, $s_P(t)$ (6). Rate of disease progress is measured by the equation

$$r_P(t) = k\, a(t)\, s_P(t)$$

in which k is a parameter that describes the host response to infection. Plant age-related susceptibility is assessed by the equation

$$a(t) = \alpha (t - \beta) \exp[-\epsilon (t - \beta)] + \delta$$

when $t > 1$; when $t \leq 1$, $a(t) = 0$. α, β, δ, and ϵ are parameters estimated by nonlinear regression. Seasonality is determined by

$$s_P(t) = \sigma + \mu \sin[\omega (t + P) + \phi],$$

where $\omega = 2\pi/12$, σ is the average rate, μ is the amplitude around the average, and ϕ is the phase; these parameters are estimated by nonlinear regression.

This model structure was set with ACMV disease progress curves obtained from a 6-yr experiment conducted at Adiopodoumé in Ivory Coast and validated with data from Tanzania (11,26). Values of the parameters of the function r_P calculated with cultivar CB (11) were α, 3.01; β, 1.07; ϵ, 1.37; δ, 0.23; σ, 0.46; μ, 0.35; and ϕ, −0.66. These values were used to perform the simulations.

Secondary spread of ACMV within plantings was described by a logistic model (2) with the same time-dependent rate, $r_P(t)$, applied in equation 1. It was also assumed that only symptomatic cassava contributed to secondary spread, because symptom expression and virus content were positively correlated (8). The equation for secondary spread, which describes the internal spread within plantings, is

$$dy_2/dt = r_P(t)\, \lambda\, y_{2,l}(1 - y_2), \quad (2)$$

where y_2 is the disease incidence caused by secondary spread, l is the latent period (defined as the time between infection and symptom expression), $y_{2,l}$ is the disease incidence at $t - l$, and λ is the coefficient of secondary spread.

Total disease spread, y, combining primary and secondary spread (y_1 and y_2 in equations 1 and 2, respectively), was modeled by the differential equations of Brasset and Gillingan (2):

$$dy/dt = dy_1/dt + dy_2/dt = r_P(t)(1 - y_1) + r_P(t)\,\lambda\, y_{2,l}(1 - y_2)$$
$$dy/dt = r_P(t)\,[(1 - y_1) + \lambda\, y_{2,l}(1 - y_2)],$$

which we condensed to

$$dy/dt = r_P(t)\,[(1 + \lambda\, y_l)(1 - y)] \quad (3)$$

assuming the correction factor in both equations 1 and 2 to be $(1 - y)$, with y_l the disease incidence at $t - l$. This differential equation was solved with the Runge-Kutta fourth-order numerical integration routine with a time step of 1 wk (4) by using the Stella II software package (22).

Yield loss model. Earlier studies established the relationship between the date of symptom appearance and yield loss (6).

$$U = U_h - A\exp(-Bt), \quad (4)$$

where U is the yield of a diseased plant, U_h is the yield of a healthy plant, t is the plant age when symptoms appeared, and A and B are constants that characterize the yield loss in each cultivar. U_h was set to 1 (100%), so that U represented the yield of a diseased plant expressed as a fraction of a healthy plant. Another variable, Uc, is defined as the yield of a plant infected as a cutting. In field studies, little yield loss occurred if plants became infected after 4 mo of growth (6). Thus, to model the time of infection on subsequent yield, values of A and B were adjusted so that $U = Uc$ when $t = 0$ and $U = 0.9$ when $t =$ 4 mo. The total plot yield, W, was obtained by combining the n individual plant yields as affected by the age when the infection occurred:

$$W = \sum_{i=1}^{i=n} U(i, t). \quad (5)$$

Modeling host resistance, reversion, and cutting selection. Estimates of host resistance, latent period, and secondary spread parameters were needed to perform the simulations. Results from two experimental trials, arbitrarily named experiments 1 and 2, were used to obtain realistic values of these parameters. In experiment 1, disease progress was monitored in a collection of 29 cassava cultivars at Adiopodoumé showing a wide range of host resistance and including resistant cultivars derived from interspecific hybrids with the ceara rubber tree, *Manihot glaziovii* (19). The cassava were planted in December 1984 with 1- × 1-m spacing in a randomized block experimental design with four blocks, one plot of each cultivar per block, and 20 plants of each cultivar per plot. Disease incidence was assessed weekly, and symptomatic plants were removed to limit secondary spread. The parameter k in equation 1 was selected so that the calculated disease incidence value equaled the observed value 2 mo after planting. Disease progress curves of each cultivar were calculated from equation 1 with the corresponding k parameter and were compared with the observed epidemics.

Experiment 2 was a square of 0.49 ha planted in July 1983 comprising seven blocks of seven plots. Each plot contained 100 plants of cultivar CB arranged with 1- × 1-m spacing. In the plots of the western half of the trial, plants showing symptoms were allowed to remain in the field. In the plots of the eastern part, plants with mosaic were rogued as soon as they were noted. Disease progress was monitored in rogued and unrogued plots of three blocks, which differed in position and average disease incidence: block A was on the wind-exposed border, block B was in the middle of the experimental trial, and block C was between blocks A and B. The latent period, the season parameters of the function r_P, and the secondary spread coefficient λ were estimated from the results of experiment 2 (5). The highest correlation between changes in the numbers of adult whiteflies and subsequent changes in symptoms was found with a time lag of 6 wk, which was considered to be an estimate of the average length of the latent period. Season parameters varied between experiments (11) and consequently were estimated by nonlinear regression with SYSTAT statistical software (33) for experiment 2 from equation 1 to fit data from rogued plots of blocks A, B, and C. Then, the values of λ were estimated by iterative approximations for calculated values of y from equation 3 to fit the observed data in unrogued plots of blocks A, B, and C (28).

The following variables were defined: $y_{e,\,i-1}$ was the disease incidence at the end of year $i-1$, and $y_{b,\,i}$ was the disease incidence at the beginning of the following year i. Reversion, R, was defined as the percentage of healthy cuttings derived from infected plants. Then

$$y_{b,\,i} = (1 - R)\, y_{e,\,i-1}. \quad (6)$$

Cutting selection, S, was defined as the ratio between the probability that a cutting would be selected from a healthy plant compared with a diseased one. Then

$$y_{b,\,i} = y_{e,\,i-1}/[S + (1 - S)\, y_{e,\,i-1}]. \quad (7)$$

When reversion, R, and selection, S, were considered together, then

$$y_{b,\,i} = (1 - R)\, y_{e,\,i-1}/[S + (1 - S)\, y_{e,\,i-1}]. \quad (8)$$

Simulations. Simulations were run for March planting dates, the most common time of planting of cassava in Adiopodoumé. This time also corresponds to the period of highest inoculum levels (7). The growth cycle of cassava from planting to harvesting was considered to be 12 mo. Simulations of disease incidence and yield losses were run for 10 successive years. Values of reversion found experimentally in a collection of resistant cultivars were 5–95% (15). The average reversion value of these cultivars was approximately 50% (15). Selection ratios commonly had a value of 2 but were as high as 10 in very sensitive cultivars that exhibited conspicuous symptoms (J. M. Thresh, *personal communication*). These ranges of values for reversion and cutting selection were adopted in the simulations. Simulations of yield losses were run in a hypothetical cultivar with $Uc = 0.50$ (50% losses), a value that was consistent with the experimental data reported (32).

RESULTS

Parameter estimation. For the 29 cultivars tested, the host response parameter, k (equation 1), ranged between 0.1 and 4. For instance, cultivars with final disease incidences of 20, 40, 80, and 100% had k values of 0.16, 0.23, 1.20, and 3.87, respectively. Disease progress curves calculated from equation 1 were similar to observed data for cultivars with values of $k = 0.16$ and 1.2 (Fig. 1). Estimated parameters from experiment 2 of the function r_P and of secondary spread, λ, resulted in calculated disease progress curves that were close to field observations (Table 1). The contribution of secondary spread to total spread differed among blocks, as indicated by λ estimates of 1–10. Consequently, values of $\lambda = 1$ and 10 were subsequently used in simulations to represent situations where secondary spread was either low or high, respectively.

Effect of reversion. Starting with uninfected material and without reversion or cutting selection, disease incidence increased and ultimately approached 100% after successive crop cycles, whatever the level of host resistance (Fig. 2). For instance, with a highly resistant cultivar ($k = 0.1$), 100% disease incidence occurred within 13 yr when $\lambda = 1$ and within 5 yr when $\lambda = 10$. By contrast, with a 50% reversion in a highly resistant cultivar ($k = 0.1$),

disease incidence increased during the first few crop cycles but ultimately reached an equilibrium below 100% (Fig. 2). The disease incidence level at equilibrium was 36% when $\lambda = 1$ and 76% when $\lambda = 10$. In both instances, the equilibrium was reached within 7 yr.

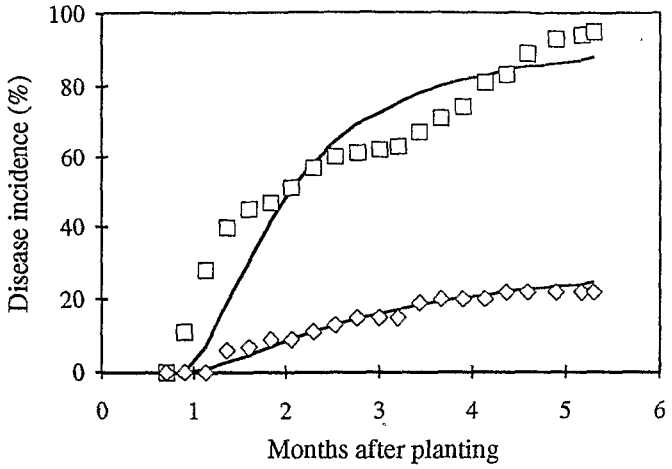

Fig. 1. Observed disease progress curves for cultivars CB (□) and Garimoshi (◇) at Adiopodoumé in 1983 and calculated disease progress curves (lines) assuming resistance parameter $k = 1.20$ for CB and 0.16 for Garimoshi.

TABLE 1. Estimated values of the season parameters α, μ, and ϕ[a] of the function r_P,[b] host response parameter (k), secondary spread coefficient (λ), and coefficient of determination (R^2) between calculated and observed disease progress curves for African cassava mosaic virus epidemics observed in three blocks of cassava cultivar CB at Adiopodoumé, Ivory Coast, in 1983

Block	α	μ	ϕ	k	λ	R^2
A	0.46	0.69	−0.68	2.0	3.5	0.96
B	0.44	0.64	−0.66	2.4	1.0	0.97
C	0.17	0.26	−0.87	1.8	10.0	0.92

[a] α = Average rate; μ = amplitude around the average; and ϕ = the phase.
[b] The rate of disease progress when the month of planting (P) is fixed.

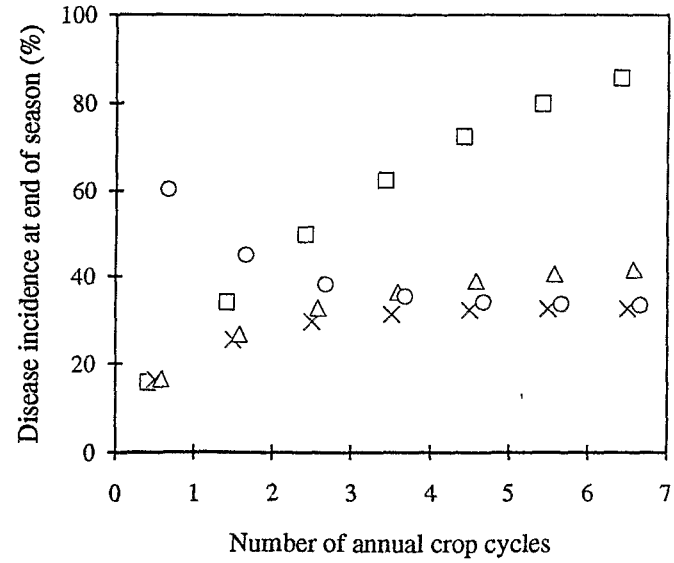

Fig. 2. Simulated disease incidence at the end of the season after successive crop cycles in a highly resistant cultivar ($k = 0.1$); secondary spread coefficient $\lambda = 1$. □ = Simulation done without reversion ($R = 0\%$) or cutting selection ($S = 1$) and with no infected cuttings in the initial planting; × = simulation at $R = 50\%$, without cutting selection, and with no infected cuttings; ○ = simulation at $R = 50\%$, without cutting selection, and with 100% infected cuttings; and △ = simulation done without reversion, with a selection ratio of 2, and with no infected cuttings.

Such equilibria were stable and did not depend on the initial level of infection in the planting material. For example, when starting from a 100% infected plot, disease incidence in the highly resistant cultivar ($k = 0.1$) decreased progressively in successive cycles (Fig. 2) to reach the equilibria found previously (36 and 76% with $\lambda = 1$ and 10, respectively). Only the number of crop cycles to reach the equilibrium depended on the initial level of infection in the planting material: the equilibrium value of 36% (when $\lambda = 1$) was reached after 4 yr when starting with uninfected material, but it required 6 yr to reach this value when the initial level of infection was 100%.

The equilibrium level of disease incidence reached after several successive crop cycles was influenced by the reversion rate and the level of host resistance of the cultivar. However, their respective impacts on the equilibrium value were different. For instance, when $R = 50\%$, an equilibrium below 100% was reached only in cultivars with high levels of host resistance ($k \leq 0.1$) (Fig. 3). By contrast, in cultivars with high levels of host resistance ($k \leq 0.1$), an equilibrium below 100% was apparent whenever $R \geq 20\%$ (Fig. 4). Then, the equilibrium value gradually dropped with higher reversion rates.

Effect of selection. If cuttings derived from healthy plants were selected preferentially, disease incidence gradually increased during the first crop cycles but commonly reached an equilibrium below 100% (Fig. 2). The disease level reached after several years

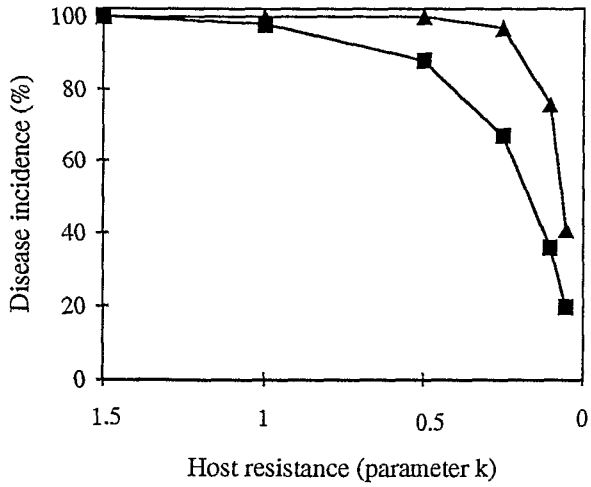

Fig. 3. Simulated relationships between final disease incidence after 10 successive crop cycles and the level of host resistance (k) in a cultivar with a 50% reversion rate (R) when the coefficient of secondary spread (λ) was either 1 (■) or 10 (▲).

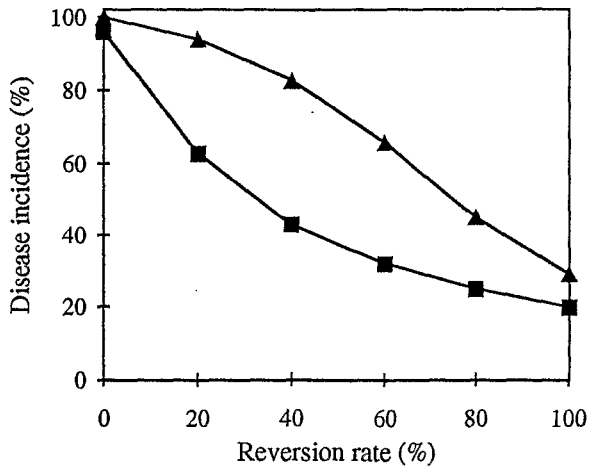

Fig. 4. Simulated relationships between final disease incidence after 10 successive crop cycles and the reversion rate (R) in a highly resistant cultivar ($k = 0.1$) when the coefficient of secondary spread (λ) was either 1 (■) or 10 (▲).

depended on the level of host resistance and on the selection ratio of the cultivar (Fig. 5). An equilibrium below 100% occurred whatever the hypotheses on secondary spread when a selection ratio of 4 was combined with a high level of host resistance ($k \leq 0.1$.) (Fig. 6). The equilibrium value dropped sharply when higher selection ratios were applied, and minimal disease incidence was approached when $S \geq 6$ (Fig. 6).

Effects of reversion and cutting selection. The effects of reversion and cutting selection reinforced each other. An equilibrium below 100% was reached, whatever the hypotheses on the secondary spread, in cultivars with $R \leq 20\%$ as long as $S \geq 2$ (Fig. 7). Minimal disease incidence (almost as low as that reached when starting from a virus-free planting) was reached in varieties with $R \geq 60\%$, as long as $S \geq 4$.

Yield losses. Reversion and selection resulted in yield losses that were less than those caused by 100% infection of cuttings, even if the final disease incidence ultimately reached 100%. For instance, in a cultivar with high host resistance ($k = 0.1$), a reversion rate of 20%, and a selection ratio of 2, disease incidence after 10 yr was 100%, but yield losses were 11 and 34% if $\lambda = 1$ and 10, respectively (Fig. 8), which were much less than the 50% loss that occurred without reversion or cutting infection. Yield losses also depended on the disease incidence reached at equilibrium; the lower the disease incidence at equilibrium, the lower the estimated yield losses (Fig. 8). Regardless of the values of the secondary spread coefficients, yield losses were below 10% whenever $R \geq 40\%$ and $S \geq 2$ (Fig. 8).

DISCUSSION

The simulation studies described here provided insights into the likely impact of some characteristics of resistance and sanitation on the development of ACMV epidemics. Without reversion and cutting selection, disease incidence increased in successive plantings of the same clonal stock and ultimately reached 100%, whatever the degree of host resistance. By contrast, when reversion or cutting selection occurred, disease incidence reached an equilibrium value below 100%. At equilibrium, the percentage of plants that were not infected as a result of reversion or cutting selection balanced the new virus transmissions by whiteflies. This result has three practical consequences: 1) it emphasizes the potential of resistant cultivars with reversion to control ACMV, since these cultivars not only suffered lower yield losses when infected but were less likely to become heavily infected, even after many years

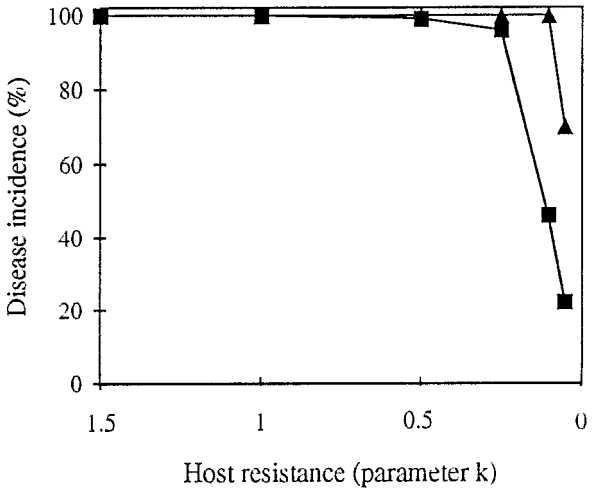

Fig. 5. Simulated relationships between final disease incidence after 10 successive crop cycles and the level of host resistance (k) in a cultivar with a selection ratio (S) of 2 when the coefficient of secondary spread (λ) was either 1 (■) or 10 (▲).

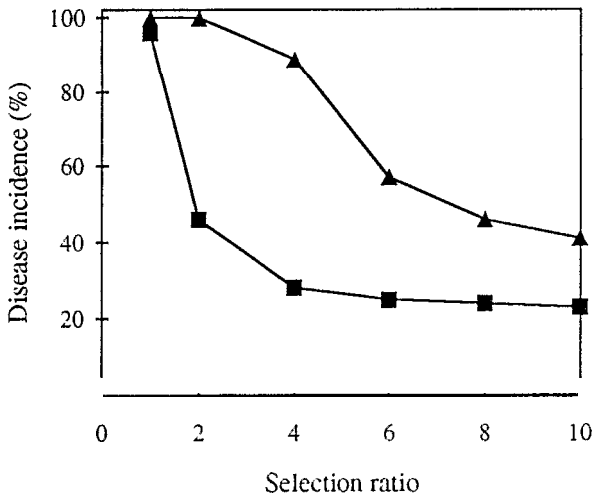

Fig. 6. Simulated relationships between final disease incidence after 10 successive crop cycles and the selection ratio (S) in a highly resistant cultivar ($k = 0.1$) when the coefficient of secondary spread (λ) was either 1 (■) or 10 (▲).

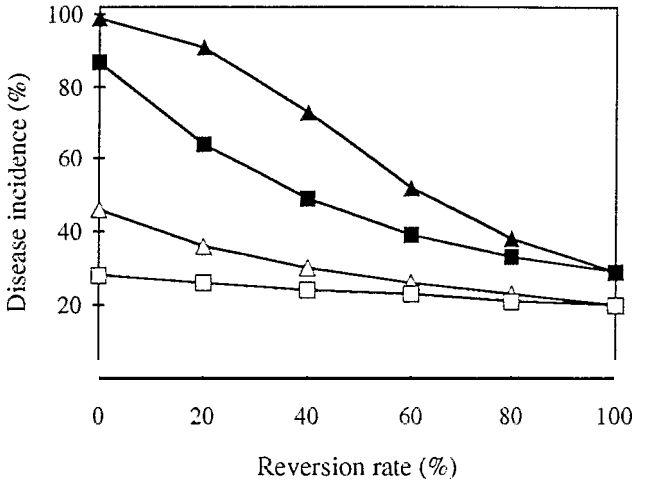

Fig. 7. Simulated relationships between final disease incidence after 10 successive crop cycles and the reversion rate (R) in a highly resistant cultivar ($k = 0.1$) with different selection ratios (S) and coefficients of secondary spread (λ): △, $S = 2$ and $\lambda = 1$; □, $S = 4$ and $\lambda = 1$; ▲, $S = 2$ and $\lambda = 10$; and ■, $S = 4$ and $\lambda = 10$.

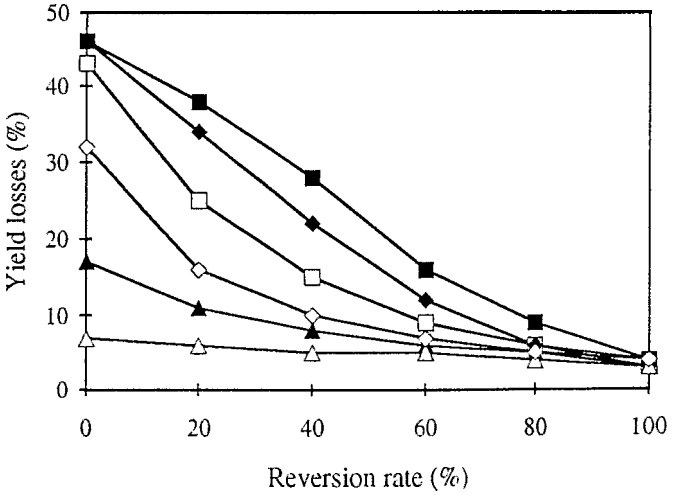

Fig. 8. Simulated relationships between yield losses and the rate of reversion (R) with different selection ratios (S) and coefficients of secondary spread (λ): □, without selection and $\lambda = 1$; ◇, $S = 2$ and $\lambda = 1$; △, $S = 4$ and $\lambda = 1$; ■, without selection and $\lambda = 10$; ◆, $S = 2$ and $\lambda = 10$; and ▲, $S = 4$ and $\lambda = 10$.

of successive crop growth; 2) it underlines the potential of "imperfect" sanitation techniques, i.e., simple preferential selection of healthy cuttings as opposed to more systematic selections of healthy cuttings and eradication of diseased plants as successfully implemented in areas with low inoculum pressure (1,14,16,17); and 3) it shows that the losses suffered when reversion occurred were much fewer than expected in totally infected planting material.

Simulation studies indicated that the disease incidence reached after several successive crop cycles was critically dependent on the level of host resistance, the reversion rate, and the cutting selection ratio. A disease incidence below 100% at equilibrium was reached only in cultivars combining high host resistance ($k = 0.1$, the order of magnitude of the most resistant improved cultivars available) with $R \geq 20\%$ or $S \geq 2$. In the "extreme" scenarios associating high host resistance ($k = 0.1$) with high reversion rate ($R \geq 60\%$) or high cutting selection ($S \geq 4$), disease incidence and yield loss values after several crop cycles were almost as low as those obtained when virus-free cuttings were planted each year.

The equilibrium reached with reversion alone differed from that induced by cutting selection. When reversion occurred, whatever the final disease incidence, a proportion of cuttings escaped infection after each crop cycle and the equilibrium reached was stable. By contrast, selection of virus-free cuttings was dependent on the disease incidence at the end of each crop cycle: no equilibrium was reached if the plot became fully or heavily infected, because of the lack of healthy plants available. Selection and reversion complemented each other. However, they are not likely to occur together in nature because reversion is a feature mainly of highly resistant plants that tend to tolerate infection and show few symptoms. Thus, technology more sophisticated than simple visual assessment would be needed to practice an improved selection procedure in highly resistant cultivars.

Because Adiopodoumé is an area with exceptionally high inoculum levels (9), it is likely that equilibria with disease incidence below 100% would occur in many other areas of Africa where inoculum levels are lower, even in less resistant cultivars with lower reversion rates or cutting selection ratios. This is consistent with repeated observations of significant percentages of symptomless cassava occurring within infected fields in several countries, despite many years of cultivation (10,20,30). This was sometimes attributed to the loss of symptoms under specific conditions (plant maturity, drought, poor growth, or attack by mealybugs or green mites) but could also reflect the proportion of healthy cassava plants remaining at equilibrium.

If the long-term effects of reversion and cutting selection on disease incidence and yield losses suggested by these simulation studies are verified experimentally and if equilibria below 100% are found to occur widely in long-term multilocation trials, the management of ACMV in Africa should be reassessed, putting more emphasis on the use and integration of resistant cultivars and on sanitation techniques (31,32). In particular, when the benefits of reversion are considered, the general use of ACMV-resistant cultivars with the available level of reversion and host resistance would be the key component of any disease-control strategy. In areas with low inoculum levels, it is likely that ACMV incidence and yield losses would be so limited with such cultivars that phytosanitation techniques would become unnecessary. Phytosanitation techniques may have a significant impact only in areas with high inoculum levels or with less resistant cultivars. Even then, the long-term effect of cutting selection on ACMV incidence and yield losses should be assessed before the scope of an extended sanitation program based on selection of healthy material and roguing is determined.

LITERATURE CITED

1. Bock, K. R. 1983. Epidemiology of cassava mosaic disease in Kenya. Pages 337-347 in: Plant Virus Epidemiology. R. T. Plumb and J. M. Thresh, eds. Blackwell Scientific, Oxford.
2. Brasset, P. R., and Gilligan, C. A. 1988. A model for primary and secondary infection in botanical epidemics. J. Plant Dis. Prot. 95:352-360.
3. Cours-Darne, G. 1968. Improving cassava in Africa. Pages 330-339 in: The Abidjan Conference. Agricultural Research Priorities for Economic Development in Africa. U.S. National Academy of Sciences, Washington, DC.
4. Doucet, P., and Sloep, P. B. 1992. Mathematical Modelling in the Life Sciences. Elis Horwood, New York.
5. Fargette, D., Fauquet, C., Grenier, E., and Thresh, J. M. 1990. The spread of African cassava mosaic virus into and within cassava fields. J. Phytopathol. 130:289-302.
6. Fargette, D., Fauquet, C., and Thouvenel, J.-C. 1988. Yield losses induced by African cassava mosaic virus in relation to mode and date of infection. Trop. Pest Manage. 34:89-91.
7. Fargette, D., Jeger, M., Fauquet, C., and Fishpool, L. D. C. 1994. Analysis of temporal disease progress of African cassava mosaic virus. Phytopathology 84:91-98.
8. Fargette, D., Thouvenel, J.-C., and Fauquet, C. 1987. Virus content of leaves of cassava infected by African cassava mosaic virus. Ann. Appl. Biol. 110:65-73.
9. Fargette, D., and Thresh, J. M. 1994. Ecology of African cassava mosaic. Pages 269-282 in: Ecology of Plant Pathogens. J. P. Blakeman and B. Williamson, eds. C.A.B. International, Oxford.
10. Fargette, D., Thresh, J. M., and Otim-Nape, G. W. 1994. The epidemiology of African cassava mosaic geminivirus: Reversion and the concept of equilibrium. Trop. Sci. 34:123-133.
11. Fargette, D., and Vié, K. 1994. Modeling the temporal primary spread of African cassava mosaic virus into plantings. Phytopathology 84:378-382.
12. Fauquet, C., and Fargette, D. 1990. African cassava mosaic virus: Etiology, epidemiology, and control. Plant Dis. 74:404-411.
13. Fauquet, C., Fargette, D., Desjardin, J., Leylavergne, F., Colon, L., and Thouvenel, J.-C. 1986. Multicomponent resistance of cassava to African cassava mosaic virus. Pages 7-9 in: Sec. 7, Proc. Int. Workshop Epidemiol. Plant Virus Dis., 3rd.
14. Fauquet, C., Fargette, D., and Thouvenel, J.-C. 1988. Some aspects of the epidemiology of African cassava mosaic virus in Ivory Coast. Trop. Pest Manage. 34:92-96.
15. Fauquet, C., Fargette, D., and Thouvenel, J.-C. 1988. Selection of healthy cassava plants obtained by reversion in cassava fields. Pages 146-149 in: Proc. Int. Sem. Afr. Cassava Mosaic Virus Dis. Control. C. Fauquet and D. Fargette, eds. CTA, Wageningen, the Netherlands.
16. Guthrie, J. 1990. Controlling African Cassava Mosaic Disease. CTA, Wageningen, the Netherlands.
17. Jameson, J. D. 1964. Cassava mosaic disease in Uganda. E. Afr. Agric. For. J. 29:208-213.
18. Jennings, D. L. 1960. Observations on virus diseases of cassava in resistant and susceptible varieties. I. Mosaic disease. Emp. J. Exp. Agric. 28:23-34.
19. Jennings, D. L. 1994. Breeding for resistance to African cassava mosaic virus in East Africa. Trop. Sci. 34:110-122.
20. Otim-Nape, G. W. 1993. Epidemiology of the African cassava mosaic geminivirus disease (ACMD) in Uganda. Ph.D. thesis. University of Reading, Reading, England.
21. Pacumbaba, R. P. 1985. Virus-free shoots from cassava stem cuttings infected with cassava latent virus. Plant Dis. 69:231-232.
22. Richmond, B., Peterson, S., and Boyle, D. 1990. Stella II. User's Guide. High Performance Systems, Hanover, NH.
23. Rossel, H. W., Asiedu, R., and Dixon, A. G. O. 1992. Resistance of cassava to African cassava mosaic virus: What really pertains. Trop. Root Tuber Crops Bull. 6:2.
24. Rossel, H. W., Changa, C. M., and Atiri, G. I. Quantification of resistance to African cassava mosaic (ACMV) in IITA-improved mosaic-resistant cassava breeding material. In: Proc. Int. Soc. Trop. Root Crops. 1992. (In press.)
25. Rossel, H. W., Thottappilly G., Van Lent, J. M., and Huttinga, H. 1988. The etiology of cassava mosaic in Nigeria. Pages 43-56 in: Proc. Int. Sem. Afr. Cassava Mosaic Virus Dis. Control. C. Fauquet and D. Fargette, eds. CTA, Wageningen, the Netherlands.
26. Storey, H. H., and Nichols, R. F. W. 1938. Virus diseases of East African plants. VII. A field experiment in the transmission of cassava mosaic. E. Afr. Agric. J. 6:446-449.
27. Storey, H. H., and Nichols, R. F. W. 1938. Studies on the mosaic diseases of cassava. Ann. Appl. Biol. 25:790-806.
28. Teng, P. S. 1981. Validation of computer models of plant disease epidemics: A review of philosophy and methodology. J. Plant Dis. Prot. 88:49-63.
29. Thresh, J. M., Fargette, D., and Otim-Nape, W. G. 1994. Effects of African cassava mosaic geminivirus on the yield of cassava. Trop.

Sci. 34:26-42.
30. Thresh, J. M., Fishpool, L. D. C., Otim-Nape, G. W., and Fargette, D. 1994. African cassava mosaic virus: An underestimated and unsolved problem. Trop. Sci. 34:3-14.
31. Thresh, J. M., and Otim-Nape, G. W. 1994. Strategies for controlling African cassava mosaic geminivirus. Adv. Dis. Vector Res. 10:215-236.
32. Thresh, J. M., Otin-Nape, G. W., and Jennings, D. L. 1994. Exploiting resistance to African cassava mosaic virus. Aspects Appl. Biol. 39:51-60.
33. Wilkinson, L. 1992. SYSTAT: The System for Statistics. Version 5.2. SYSTAT, Inc., Evanston, IL.

Genetics

Chromosomal Location of Genes for Stripe Rust Resistance in Spring Wheat Cultivars Compair, Fielder, Lee, and Lemhi and Interactions of Aneuploid Wheats with Races of *Puccinia striiformis*

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ABSTRACT

Chen, X. M., Jones, S. S., and Line, R. F. 1995. Chromosomal location of genes for stripe rust resistance in spring wheat cultivars Compair, Fielder, Lee, and Lemhi and interactions of aneuploid wheats with races of *Puccinia striiformis*. Phytopathology 85:375-381.

The spring wheat (*Triticum aestivum*) cultivar Lemhi has one gene, and cultivars Lee, Compair, and Fielder have two genes each for resistance to stripe rust, caused by *Puccinia striiformis*. To determine the chromosomal locations of the genes, the cultivars were crossed with susceptible disomic Chinese Spring and a set of 21 Chinese Spring aneuploids. Monosomic F_1 plants were cytologically determined, grown in a greenhouse, and self pollinated to produce F_2 seed. F_2 seedlings and their parents were inoculated with selected North American races of *P. striiformis*. The results confirmed that *Yr6* in Fielder is on chromosome 7B and *Yr8* in Compair is on chromosome 2D; and they show that *YrLem* in Lemhi is on chromosome 1B, *YrLe1* in Lee is on chromosome 4D, *YrLe2* in Lee is on chromosome 6D, *YrCom* in Compair is on chromosome 5B, and *YrFie* in Fielder is on chromosome 6D. None of the Lee genes that we detected with North American races is *Yr7*. We propose official gene designations *Yr19* for *YrCom*, *Yr20* for *YrFie*, *Yr21* for *YrLem*, *Yr22* for *YrLe1*, and *Yr23* for *YrLe2*.

Additional keywords: cytogenetics, gene interaction, host-pathogen interaction, monosomic analysis, yellow rust.

Stripe rust (yellow rust), caused by *Puccinia striiformis* Westend., is an important disease of wheat (*Triticum aestivum* L.) in many regions of the world (34). In North America, the disease is most destructive in the western United States and sometimes destructive in the south central United States. Growing resistant cultivars is the most economical method of controlling the disease (18).

The spring wheat cultivars Lemhi, Lee, and Fielder are used to differentiate North American races of *P. striiformis* (19); Compair is used to differentiate European races; and Lee is used to differentiate world races of *P. striiformis* (34). An understanding of the genetics of the differential cultivars improves their usefulness in identifying races and breeding wheats for resistance. Macer (22) designated *Yr7* as the resistance gene in Lee I; however, he pointed out that the designation was not proved by a complete set of diallel crosses. Later, McIntosh et al (25) reported that *Yr7* was located on chromosome 2B because of its linkage with *Sr9g*, which confers resistance to stem rust (*Puccinia graminis* f. sp. *tritici*). Compair was developed by Riley et al (27,28) by crossing the hexaploid wheat cultivar Chinese Spring with *Aegilops comosa*. They reported that Compair had a dominant gene for resistance to the tested European races and demonstrated that the gene was transferred to wheat chromosome 2D by genetically induced homoeologous recombination with *A. comosa* chromosome 2M. The gene was later designated by Macer (22) as *Yr8*.

In inheritance studies of resistance to stripe rust, Chen and Line (3-6,8) reported that Lemhi has one gene and Lee, Compair, and Fielder have two genes each for resistance to North American races of *P. striiformis*. Based on the results of diallel crosses and race reactions, they identified one of the Fielder genes as *Yr6*, which has been reported in Heines Kolben, Heines Peko, and other cultivars (13,22,29). Gene *Yr6* in Heines Kolben and Heines Koga II was subsequently reported to be located on chromosome 7B (11,17). Because Lee is used to differentiate both North American and European races and its resistance to European races has been attributed to *Yr7*, Chen and Line (5,6) suggested that one of the genes they detected in Lee should be *Yr7*. Similarly, Chen and Line (3,5,6,8,18) suggested that one of the genes in Compair should be *Yr8*. The Lemhi gene and the additional genes in Lee, Compair, and Fielder were shown to be different from other reported genes and were therefore provisionally designated