Theoretical and Applied Genetics 42, 262—266 (1972)

Quantitative Genetics of Sugarcane

IV. Genetics of Fiji Disease Resistance

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Summary. 1. Fiji disease is an important virus disease of sugarcane in many countri es. In Fiji, its vector is the leafhopper *Perkinsiella vitiensis*.

2. The usual measure of resistance under insectary test conditions is the number of days required after exposure to viruliferous hoppers for half the test plants of a clone to show the diagnostic symptoms (SD_{50}). Previous studies have shown this measure is related to resistance under field conditions and is convenient to record.

3. A quantitative genetic analysis has been made of resistance to Fiji disease in a hybrid sugarcane population.

4. The SD_{50} measure of resistance showed a heritability and repeatability of about 0.4. These results indicated that Fiji disease resistance can be improved by breeding and selection.

5. No unfavourable correlations were detected between resistance and other commercial attributes.

6. In a mixed stand, sugarcane clones differ for the number of leafhoppers found on them. However no necessary relationship was detected between this character and resistance to Fiji disease.

Introduction

Fiji disease is a virus disease of sugarcane (*Saccharum officinarum* L.) and some closely related species, *S. robustum, S. edule* and *Erianthus maximus*. It causes stunted growth and its diagnostic symptoms are small galls on the undersurface of leaves (Hughes and Robinson, 1961). It occurs in the South Pacific (Australia, Fiji, New Guinea, Samoa etc.), the Philippines and Madagascar. The disease poses a threat to many sugar producing countries, indeed Martin (1947) considered it the most serious of all sugarcane diseases. Fiji disease is transmitted by the leafhopper *Perkinsiella vitiensis* Kirk. in Fiji (Husain, Brown, Hutchinson and Wismer, 1967) and by other *Perkinsiella* species in other countries (Hughes and Robinson, 1961). Transmission is thought to occur whilst the leafhopper feeds from the phloem tissue in leaves and leaf sheaths (Baber and Robinson, 1951).

Modern commercial hybrids can acquire genes for resistance to Fiji disease from either *S. officinarum* or *S. spontaneum* sources. Breeding and selection programmes for resistance to the disease are based on treating resistance as a polygenic character. Simmonds (1969) has argued that polygenic resistance to plant diseases is liable to be more stable and effective than oligogenic, i. e. where the resistance arises from a few genes with large effect. The success of such programmes will depend on adequate heritability and repeatability for resistance as well as the absence of restrictive correlations with other commercially important characters. For example, Hughes and Hooker (1971) detected a largely additive action of genes conditioning resistance to northern leaf blight in maize. This paper provides evidence that these requirements are met in the case of Fiji disease of sugarcane.

Material and Methods

The experimental material consisted of 141 hybrid sugarcane clones, each derived from crosses made as part of the normal breeding programme in Fiji. The clones were chosen at random from about 7000 clones in Stage 2 of the selection programme. Three full-sib progeny represented each of 47 families, with four replications per clone. Brown, Daniels and Latter (1968, 1969) gave details of the origin of the clones, the experimental design, the breeding behaviour of the population, the analysis of variation and correlation for 42 vegetative characters (x_1 to x_{42}), and the assumptions on which these analyses were based. The present study used these same data for the correlation analysis with Fiji disease resistance, as estimated by five disease variables (x_{43} to x_{47}) defined below.

For individual characters, the genetic parameters of most use were h_f^2, the heritability based on the between family component of variance, and r_c, the clonal repeatability measured as the resemblance between asexual propagules of the same genotype. Large standard errors prohibited further analysis of the genetic variance into additive and dominance components. For the correlation analysis, genetic correlations were estimated from the between families covariance, clonal correlations from between clones covariance, and the environmental correlations from the within clones covariance. The genetic correlations (analogous to heritability) will detect important associations likely to persist from one sexual generation to the next, whilst the clonal correlations (analogous to repeatability) predict joint responses to clonal selection. The environmental correlations measure the effect of environmental factors on the joint variation of two characters over propagules.

The Fiji disease resistance of each clone was measured by a standard insectary method (Daniels, Husain, Hut-

chinson and Wismer, 1969). Small plants, 16—20 days after germination from single-bud stalk pieces, were exposed for 10 days to viruliferous leafhoppers in an insectary. The population density of the leafhoppers in the insectary was sufficiently high to ensure that all plants were attacked despite any possible clonal preferences by the leafhoppers. Each plant was then removed from the insectary and the latent period (the number of days until the appearance of disease symptoms) recorded. Each replication of each clone consisted of six plants i. e. over the four replications, there were 24 plants per clone. The experimental design, both for siting clones in the insectary and for latent period observation, was the same as that used in the original field experiment (Brown et al., 1968). Observations ceased after 184 days, when further appearances of diseased plants were rare.

Five disease variables were calculated:

x_{43} percentage of plants (out of 6) which expressed disease symptoms by 100 days.

x_{44} percentage of plants which expressed disease symptoms by 184 days.

x_{45} SD_{50} estimated at 100 days, i. e. the number of days required for the first 3 of the 6 plants to express disease symptoms. Healthy plants at 100 days were recorded as 100.

x_{46} SD_{50} estimated at 184 days.

x_{47} mean number of days before symptoms were detected. Only plants which showed symptoms (i. e. by 184 days) were included.

Thus variables x_{43} and x_{44} were measures of the *degree* or percentage of infection, variable x_{47} was a measure of the *rapidity* to symptom expression, and variables x_{45} and x_{46} were particular functions of these two dimensions of the severity of disease expression.

Two of the 141 clones were not available for resistance testing. The importance of this missing data was examined by (1) eliminating the respective families to give a balanced analysis, (2) estimating the performance of the missing clones from those of the other two clones in the respective families. The differences between the two sets of analyses were trivial; the missing data is unlikely to upset conclusions drawn from this experiment.

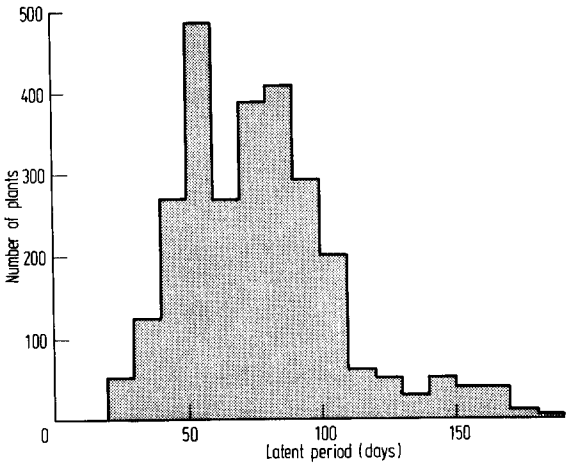

Fig. 1. Distribution of the latent periods of 2770 individual plants which expressed disease symptoms by 184 days

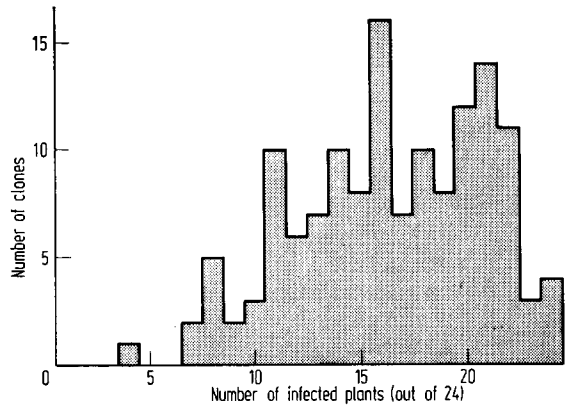

Fig. 2. Distribution of the degree of infection expressed by the 139 clones by 100 days (x_{43}). The total number of plants per clone was 24

Results

Expression of resistance to Fiji disease. Fig. 1 is a histogram of the distribution of latent periods for individual, diseased plants. Of the 3336 plants, 68% showed symptoms by 100 days, and 83% by 184 days. Disease symptoms appeared in at least three of the four replications for all 139 clones i. e. there were no immune clones. Indeed, only four clones had one symptomless replication (of six plants) after 184 days. Figure 2 is a histogram of the number of clones showing various numbers of diseased plants (out of 24) by 100 days. Resistance is a relative, continuous character in terms of both speed of expression

Table 1. *Estimates of population parameters for measures of Fiji disease resistance*

No	x_{43}	x_{44}	x_{45}	x_{46}	x_{47}
Character	% Infection	% Infection	SD_{50}	SD_{50}	Mean latent period
When assessed	100 days	184 days	100 days	184 days	184 days
Mean	68%	83%	76.1 days	83.4 days	95.8 days
Coefficients of variation (%)					
— genotypic	23	11	16	22	19
— environmental	31	22	18	33	24
— phenotypic	38	25	24	40	30
Heritability (h^2_f)	.34 ± .11	.11 ± .08	.41 ± .12	.33 ± .10	.35 ± .11
Clonal repeatability (r_c)	.35 ± .05	.21 ± .05	.45 ± .05	.31 ± .05	.39 ± .05

All h^2_f and r_c estimates are statistically significant at $P < .01$ except for h^2_f, x_{44}.

Table 2. *Correlations among measures of Fiji disease resistance. Above diagonal: upper-genetic, lower-clonal correlations. Below diagonal: environmental correlation*

No.	Character	x_{43}	x_{44}	x_{45}	x_{46}	x_{47}
x_{43}	% Infection (by 100 days)	—	1.13 ± .15 0.92 ± .04	−0.92 ± .05 −0.96 ± .03	−0.94 ± .04 −1.01 ± .02	−0.97 ± .02 −1.00 ± .01
x_{44}	% Infection (by 184 days)	0.71 ± .03	—	−1.10 ± .21 −0.78 ± .08	−1.06 ± .14 −0.91 ± .05	−1.06 ± .11 −0.92 ± .03
x_{45}	SD_{50}' (100 days)	−0.63 ± .03	−0.44 ± .04	—	1.01 ± .03 0.96 ± .02	1.01 ± .02 0.97 ± .02
x_{46}	SD_{50} (184 days)	−0.71 ± .03	−0.68 ± .03	0.76 ± .02	—	0.99 ± .02 1.01 ± .02
x_{47}	Mean latent period (over 184 days)	−0.89 ± .01	−0.86 ± .02	0.72 ± .02	0.81 ± .02	—

All correlations are significantly different from zero at the 1% level.

(Fig. 1) and percentage diseased plants after some interval (Fig. 2).

Genetics of Fiji disease resistance. Table 1 shows the means, coefficients of variation, heritabilities (h_f^2) and clonal repeatabilities (r_c) for the five measures of resistance. The measure which had the highest heritability and clonal repeatability (~.4) and the lowest environmental coefficient of variation was SD_{50} at 100 days. The magnitude of these estimates suggests that substantial improvement may be achieved by breeding and selection for disease resistance, and that the disease testing method is adequate. Improvements have been made to the testing method since this experiment (Husain and Hutchinson, in press), and it is probable the accuracy of these estimates can be further improved upon.

Table 2 shows genetic, clonal and environmental correlations between the five disease variables. The genetic and clonal correlations were close to unity, showing these five measures of disease resistance to be genetically equivalent. SD_{50} is related to field Fiji disease resistance (Daniels et al., 1969); these results show both components of SD_{50} *viz.* speed of symptom expression and percentage diseased plants, are also related to disease resistance.

Correlation between Fiji disease resistance and other characters. No genetic or clonal correlation between the disease variable SD_{50} and any of the physical or biochemical characters studied was significant. Table 3 shows the SD_{50} correlations with the more important characters. From a breeding and selection point of view, these results are encouraging; no unfavourable correlations were detected between disease resistance and commercially important characters.

Correlation between Fiji disease resistance and leafhopper clonal preferences. We (Brown et al., 1968) previously found that the number of leafhoppers counted in field plots of different clones was a clonal character with a coefficient of variation of 87% and with h_f^2 and r_c estimates of about 0.35 on a log scale. However, the correlations between disease resistance (SD_{50}, x_{45}) and leafhopper count per plot (x_5) were:

genetic	0.35 ± .36
clonal	0.01 ± .15
environmental	0.02 ± .05

There is therefore no evidence from this experiment that preferred clones in the field are also more susceptible to Fiji disease.

Basis of leafhopper clonal preferences. The field experiment in which the leafhopper counts were made can be regarded as a replicated ecological assay of factors affecting leafhopper distribution. Each clone represents a particular "micro-environment", specified by its character set (x_6—x_{42}). These characters affect the *extent* and/or the *quality* of environmental space available to the leafhoppers. For example, the number of stalks per plot (x_{17}) and other yield characters may be regarded as effecting the amount of space, whereas leaf moisture (x_8) and leaf width (x_{13}) may be regarded as affecting the nature of the space.

Table 4 gives the correlations between leafhopper density (x_5) and other variables which were statistically significant. Significant genetic or clonal correlations occurred only for leaf characters affecting quality. This was in contrast to the prevalence of significant, low environmental correlations (~0.20) for yield factors related to the extent of the environment.

Thus the basis of clonal preferences by leafhoppers is related to clonal characters which affect favourability of the environment rather than to those affecting its extent. Nonetheless the environmental

Table 3. *Correlations between Fiji disease resistance (SD_{50}-100 days, x_{45}) and commercially important characters*

No.	Character	Genetic	Clonal	Environmental
x_{16}	Yield per plot (kg)	.02 ± .28	.02 ± .12	−.05 ± .05
x_{17}	Stalks per plot	.11 ± .34	.06 ± .11	−.05 ± .05
x_{21}	Starch (ppm)	−.11 ± .23	−.22 ± .12	.10 ± .05
x_{24}	Fibre %FW	.41 ± .30	−.04 ± .11	.00 ± .05
x_{27}	Sucrose %FW	−.08 ± .25	.05 ± .12	−.02 ± .05
x_{32}	Sucrose per plot (kg)	.00 ± .29	.03 ± .12	−.04 ± .05
x_{37}	Stalk weight (kg)	−.22 ± .29	−.10 ± .11	.00 ± .05

correlations show that variation for characters of this latter type does influence leafhopper density. Note that these correlations account for only a small portion of the variance in leafhopper counts between clones. This perhaps arises from the measurement of the characters at quite different times as well as the possible effects on preferences of other characters not specifically studied here.

Population densities of leafhoppers can reach a point where serious physical damage results (Harris, 1970). In such situations clonal preferences *per se* could be important if they were related to factors controlling population density in a pure stand. However from the viewpoint of the epidemiology of and the resistance testing for Fiji disease, the relevant point is whether preferred clones are also more susceptible to Fiji disease. Bell (1935), North and Baber (1935) and Sigwalt (1963) suggested such a relationship but Baber and Robinson (1951), and Husain *et al.* (1967) under insectary test conditions, found no evidence of it. In the absence of a positive relationship, resistance testing may be prejudiced particularly at low population densities; some disease-susceptible but unpalatable clones may escape infection. The practice of confining hoppers to individual plants by small voile cages or glass cylinders (Husain and Hutchinson, in press) would appear a desirable innovation.

Table 4. *Statistically significant correlations between leafhopper count (x_5) and vegetative characters*

No.	Character	Genetic	Clonal	Environmental
x_6	Stalks per plot			.29 ± .05**
x_7	TVD leaf weight (g)		.30 ± .13*	.20 ± .05**
x_8	TVD leaf moisture (%w/w)		.37 ± .11**	
x_{12}	Early yield grade			.27 ± .05**
x_{13}	TVD leaf width (cm)	.56 ± .22*	.32 ± .11*	
x_{16}	Yield per plot (kg)			.24 ± .05**
x_{17}	Stalks per plot			.28 ± .05**
x_{18}	Nodes per stalk			.10 ± .05*
x_{19}	Stalk length (cm)			.23 ± .05**
x_{21}	Starch (ppm)			−.14 ± .05*
x_{24}	Fibre %FW			−.13 ± .05*
x_{29}	Sucrose %FW			.10 ± .05*
x_{32}	Sucrose per plot (kg)			.22 ± .05**

n. b. (1) *, ** Statistically different from zero at $P < .05$, $P < .01$. (2) In addition, environmental correlations for x_{33}, x_{34}, x_{35}, x_{39}, x_{41} and x_{42} were statistically different from zero at the 1% level.

Discussion

Resistant variables. Which amongst the five measures of resistance is the best one to use in practice? The correlations in Table 2 argue that the five measures are equivalent genetically. Therefore the choice can be made on the basis of clonal repeatability and/or convenience. The estimates of r_c in Table 1 support the choice of x_{45}. Furthermore, when testing facilities are limited, the termination of each test at 100 days (as for x_{45}) allows more clones to be tested and hence susceptible clones to be discarded at an earlier stage in a selection programme.

Genetics of Fiji disease resistance. The data in the histograms and tables demonstrate the expression of resistance as a continuous variable and that its variation is in part genetic. Whether this genetic component is due to many polygenes of small effect and not to a few oligogenes whose segregation is masked by environmental or other polygenic variation, is still unproven.

Leafhopper clonal preferences. Clonal differences in population density of leafhoppers have been observed in the past. Mungomery and Bell (1933) and Robinson and Martin (1956) considered that these insects prefer vigorous healthy plants, and North and Baber (1935) found higher densities on hybrid POJ and Co clones compared with "wild" clones of *S. spontaneum*. The results of the analysis of covariance described above have suggested that important sources of the clonal distribution pattern are leaf physical characters which alter the favourability of a given amount of environmental space.

Acknowledgements

We are indebted to the Colonial Sugar Refining Company Limited for permission to publish this paper, to Mr. A. A. Husain who supervised the Fiji disease tests, and to Mr. J. Daniels for discussion.

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Note added in proof: Since the preparation of this paper, the following useful general review has appeared: Hooker, A. L., Saxena, K. M. S.: Genetics of disease resistance in plants. Ann. Rev. Genet. **5**, 407—424 (1971).

Received November 8, 1971

Communicated by R. W. Allard

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