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INHERITANCE OF FIJI DISEASE RESISTANCE

by

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INHERITANCE OF FIJI DISEASE RESISTANCE

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INTRODUCTION

Plant breeding strategies for breeding for various characteristics vary according to the mode of inheritance involved for that character. If performance of seedlings can be predicted from the attributes of the parents, most of the genetic effects must be additive, and there will be a high heritability coefficient for that character. If, on the other hand, performance of the parents can not be used to predict performance of the progeny, then most of the genetic effects are likely to be non-additive, and there will be a low heritability. Low heritabilities also arise if there is much variability due to environment. Inheritance studies are designed to estimate genetic and envionmental effects, and to determine whether genetic effects are additive or non-additive.

Various projects have been conducted in recent years to study the inheritance of resistance to Fiji disease. In this report, the results of these studies are presented, and the implications for the plant breeding programme discussed.

METHODS AND MATERIALS

Two projects were designed to study the inheritance of Fiji disease and information collected for a third project was suitable for genetic analysis. Details of the projects are presented below:-

1. Project 699-7718

As part of this project, all ratoon 30-sett plots were inspected for Fiji disease and data on per cent infection were obtained for each plot. Cross means for per cent infection were calculated and correlated with mid-parent mean ratings based on the results of Fiji disease trials. This correlation is an estimate of the heritability on a family mean basis.

2. Project 699-7729

This project is being conducted on Bundaberg Station and is designed to study the mode of inheritance of resistance to Fiji disease. Two experiments are involved, one of which is discussed in this report.

In 1979, one hundred biparental crosses were planted in a 100 x 2 randomized complete-block experiment, with each plot having 35 seedlings. The crosses included 31 proven crosses planted at Bundaberg in 1979 plus 69 experimental crosses. In July, 1980, 12 seedlings from each plot were inspected for Fiji infection,

so that the per cent infection for each cross could be calculated. Estimates of total genetic, environmental, phenotypic variances and degree of genetic determination were made. Degree of genetic determination was estimated from the formula

$$g^2 = \sigma^2 G / \sigma^2 P$$

where g^2 = degree of genetic determination
$\sigma^2 G$ = genetic variance
$\sigma^2 P$ = phenotypic variance

Family means were correlated with mid-parent ratings to obtain an estimate of heritability on a family basis.

3. Project 699-7723

This project was designed to study the mode of inheritance of resistance to Fiji disease. It was conducted at the Pathology Farm.

The experiment consisted of seven factorials of crosses, i.e. all possible crosses between a set of female parents and a set of male parents. The factorials consisted of the following parents:-

A.	65C286 Co740 49R3863 Triton	x	Co622, F151, H36-7913, H44-2818, H49-3666, H52-246, Q68, Vesta, 59S55
B.	66C126 Phil56-98 Q77 60S223	x	Co6501, CP43-47, Q65, 63S782
C.	Co954 Q73 Q88 Q117	x	62C366, CP57-526, H36-7913,H52-246
D.	F144 62N1659 63S159	x	Co954, Co6605, L62-68, L62-86
E.	64A490 B54-163 65C286 CP55-14	x	Co331, Co475, 58N1000
F.	Cadmus CP52-68 60N1853 61N1184	x	63N1700, 39SN3821, Vesta

G. 65C573
NCo310 x CP44-155, CP52-68, L62-96
Q86

The fuzz was planted on Bundaberg Station in 1976 and 12 varieties from each cross were propagated at the Pathology Farm in 1977. Several crosses failed to produce any seedlings and some produced fewer than 12. However, germination was generally satisfactory.

Each cross was represented by 10 of the 12 varieties. For crosses with fewer than 10 varieties, the available varieties were repeated so that 10 stools were planted in each plot. The factorials of crosses together with their parent varieties and standards were planted in standard three-replicate Fiji field trials. Two or three factorials were included in each field trial.

Disease inspections were conducted in the first ratoon crop. The following data were collected for each plot:-

(a) Number of diseased stools.

(b) Number of diseased and healthy stalks for each plant in the plot.

(c) Visual rating (0-9 scale) for each plant in the plot.
The visual ratings were given using the following system:-

0	no symptoms
1	few galls, no stunting
2	many galls, no stunting
3	many galls, some stunting, bitten off tops on some plants
4	more severe than 3
5	stunting, 75-90 cm to TVD, fair amount of cane visible
6	more severe than 5
7	very severely stunted - some stalk showing
8	very severely stunted, blue beard to tuft, 25-35 cm to top of leaves
9	death

Analyses of variance for parent varieties and crosses were conducted for each factorial and the results were combined into one analysis. For parent varieties, the combined analysis had the following degrees of freedom and expected mean squares.

Source	**d.f.**	**Expected mean squares**
Varieties	49	$\sigma^2 s + 10\sigma^2 E + 30\sigma^2 G$
Error	98	$\sigma^2 s + 10\sigma^2 E$
Within plot error	1 319	$\sigma^2 s$

where $\sigma^2 s$ = plant to plant environmental variance
$\sigma^2 E$ = plot to plot environmental variance
$\sigma^2 G$ = genetic variance

For crosses, the combined analysis had the following degrees of freedom and expected mean squares:-

Source	d.f.	Expected mean squares
Females	19	$\sigma^2W + 10\sigma^2E + 30\sigma^2MF + 128.7\sigma^2F$
Males	23	$\sigma^2W + 10\sigma^2E + 30\sigma^2MF + 111.3\sigma^2M$
Females x males	57	$\sigma^2W + 10\sigma^2E + 30\sigma^2MF$
Error	191	$\sigma^2W + 10\sigma^2E$
Within plot error	2 627	σ^2W

where σ^2W = plant to plant variation

= $\sigma^2s + 1/2\sigma^2A + 3/4\sigma^2D$

σ^2s, σ^2E = as for parent analysis

σ^2MF = variance component due to male x female interaction

= $1/4\sigma^2D$

σ^2M = variance component due to differences between males

= $1/4\sigma^2A$

σ^2F = variance component due to differences between females

= $1/4\sigma^2A$

and σ^2D = non-additive genetic variance

σ^2A = additive genetic variance

genetic variance, $\sigma^2G = \sigma^2A + \sigma^2D$

phenotypic variance, $\sigma^2P = \sigma^2W + \sigma^2E + \sigma^2MF + \sigma^2M + \sigma^2F = \sigma^2s + \sigma^2E + \sigma^2A + \sigma^2D$

Regressions of offspring means on mid-parent means were performed for each factorial, and these were also combined into one analysis. In this case, the regression coefficient is an estimate of heritability on a family basis, and the mean cross product is an estimate of $1/2\sigma^2A$.

RESULTS

Project 699-7718

The results of this project will be discussed fully in a separate project report. However, information obtained on heritability is worthy of comment in this report.

Inspections for per cent Fiji disease were made in the first and second ratoon crops of the 1977 30-sett plots and in the first ratoon crop of the 1978 30-sett plots. Mean per cent diseased stools and parent ratings for crosses with eight or

more seedlings are presented in Tables 1 and 2 for the 1977 and 1978 30-sett plots respectively.

The best estimate of heritability of Fiji disease resistance on a family mean basis is provided by the correlation between mean per cent diseased stools and mid-parent rating. The relevant correlations are presented below:-

Trial	Characters	Correlation
1977 30-sett plots	Per cent diseased stools (1R) and mid-parent rating	0.75
1977 30-sett plots	Per cent diseased stools (2R) and mid-parent rating	0.74
1978 30-sett plots	Per cent diseased stools (1R) and mid-parent rating	0.69

Project 699-7729

Cross means and parent ratings are presented in Table 3.

The analysis of variance for this project showed that the per cent of seedlings diseased varied from 83.3 (71N814 x H49-3666) to zero (W60-61 x Co6602) with a general mean of 28.8 per cent. Infection was sufficiently heavy for the differences between crosses to be highly significant. The two replicates of the experiment were in different parts of the field, and it is worth noting that there was a highly significant difference between replicate means.

The coefficient of variation was improved from 48.2 per cent to 35.7 per cent by using an arcsin transformation. Genetic analysis based on transformed and untransformed data gave the following results:-

	Untransformed	Transformed
Genetic variance, σ^2G	437.94	227.96
Environmental variance, σ^2E	192.33	116.40
Phenotypic variance, σ^2P	630.26	344.36
Degree of genetic determination, $g^2 = \sigma^2G/\sigma^2P$	0.69	0.66

Thus the important statistic, g^2, is not greatly affected by transformation of data.

The correlation between family mean and mid-parent ratings was 0.63, which is slightly lower than comparable estimates obtained in project 699-7718. Using the arcsin transformation, the correlation was 0.65.

Table 1

Per cent diseased stools and parent ratings for crosses in the 1977 30-sett plots

Cross	Number of seed-lings	Per cent diseased stools - 1R		Per cent diseased stools - 2R		Parent rating	
		Mean ± s.d.	Range	Mean ± s.d.	Range	Female	Male
CP45-184 x CP53-19	21	19.7 ± 28.9	0-68	41.4 ± 41.3	0-100	6	4
CP51-21 x 67N1509	8	12.1 ± 13.5	0-38	15.8 ± 18.0	0-50	2	7
CP53-19 x F150	12	30.0 ± 37.3	0-56	62.4 ± 56.5	4-100	4	6
CP53-19 x 62N1659	36	9.3 ± 17.9	0-55	19.5 ± 30.2	0-90	4	3
H49-104 x Q99	12	14.2 ± 17.4	0-50	23.5 ± 37.7	0-100	7	1
54N7096 x CP53-19	15	24.6 ± 32.1	0-58	45.5 ± 48.0	0-100	5	4
55N689 x CP53-19	30	22.4 ± 27.3	0-58	45.6 ± 34.1	0-93	4	4
58N829 x CP49-50	14	24.6 ± 40.7	0-89	44.6 ± 50.4	0-100	7	4
58N1868 x CP53-19	31	22.4 ± 30.2	0-72	43.6 ± 33.3	0-100	5	4
60N1853 x H44-2818	41	38.7 ± 28.3	0-94	65.9 ± 28.3	0-100	3	9
60N1853 x H49-3666	41	44.3 ± 28.4	0-96	76.9 ± 28.8	0-100	3	8
60N2111 x CP53-19	27	24.4 ± 36.0	0-100	48.4 ± 35.5	0-100	4	4
60 N2111 x H49-104	42	36.2 ± 28.1	0-90	59.3 ± 28.5	0-100	4	7
62N1659 x CP49-50	10	8.7 ± 17.8	0-50	20.2 ± 46.1	0-96	3	4
62N1659 x H49-3666	19	36.7 ± 41.7	0-100	64.8 ± 42.3	0-100	3	8
67N2254 x 65N809	39	31.4 ± 29.5	0-86	50.6 ± 29.7	0-100	2	8
67N3140 x 67N1509	8	38.6 ± 40.7	8-71	55.8 ± 68.1	4-100	1	7
67N3184 x CP44-101	9	7.8 ± 12.1	0-36	20.0 ± 30.2	0-92	5	2
NCo310 x CP44-101	11	11.7 ± 15.5	0-42	34.8 ± 47.2	0-88	8	2
NCo310 x CP53-19	8	27.3 ± 36.9	7-52	60.0 ± 72.2	32-100	8	4
NCo310 x 54N7096	30	33.0 ± 33.7	0-96	55.6 ± 34.2	0-100	8	5
NCo310 x Q58	18	38.2 ± 43.2	8-78	72.8 ± 43.4	20-100	8	8
Q63 x Q28	9	8.4 ± 8.3	0-21	17.9 ± 12.2	0-38	5	2
Q77 x CO475	10	66.0 ± 62.4	8-100	82.2 ± 58.4	22-100	8	9
Q79 x CP53-19	70	21.2 ± 21.9	0-81	40.4 ± 21.7	0-100	3	4
Q79 x H49-3666	24	47.8 ± 37.6	0-95	72.6 ± 37.3	8-100	3	8
Q79 x 39SN3821	13	31.3 ± 45.9	0-88	55.1 ± 53.0	4-100	3	6
Q90 x H50-3511	18	49.1 ± 44.1	0-92	82.0 ± 43.3	18-100	5	9
Q93 x CP49-50	20	25.2 ± 39.5	0-100	41.6 ± 42.5	0-100	7	4
Q93 - 54N7096	11	43.9 ± 55.7	0-82	68.0 ± 59.6	4-100	7	5
Q100 x 61N1184	32	17.8 ± 28.5	0-87	29.9 ± 32.5	0-100	4	8
Q117 x H52-246	9	51.7 ± 57.7	19-89	89.7 ± 64.4	54-100	4	8
60S223 x CO6501	12	6.7 ± 14.5	0-50	15.6 ± 37.1	0-96	2	1
Trojan x H50-3511	9	43.9 ± 57.4	0-100	68.2 ± 65.7	4-100	1	9

Table 2

Per cent diseased stools and parent ratings for crosses in the 1978 30-sett plots

Cross	Number of seedlings	Per cent diseased stools		Parent rating	
		Mean ± s.d.	Range	Female	Male
65C573 x Col001	12	73.9 ± 54.6	0-100	7	6
65C573 x CP44-155	9	57.0 ± 63.9	12-100	7	4
65C573 x CP52-68	11	57.9 ± 56.2	15-100	7	6
65C573 x L62-96	12	57.3 ± 53.6	0-100	7	8
Col007 x 61N567	8	26.5 ± 56.8	0-100	3	3
60N795 x 59S55	13	32.3 ± 51.7	0-95	6	7
62N1659 x 66C807	15	24.7 ± 46.8	0-100	3	2
62N1659 x Co954	11	26.9 ± 37.9	0-85	3	1
62N1659 x L62-86	18	2.5 ± 6.2	0-25	3	1
62N1659 x L62-96	24	18.8 ± 34.7	0-100	3	8
62N1659 x Phil54-60	11	39.9 ± 57.6	0-100	3	9
67N3184 x CP44-101	32	18.9 ± 32.3	0-100	5	2
67N2254 x 65N809	13	41.5 ± 50.5	0-100	2	8
NCo310 x CP33-372	11	36.5 ± 48.8	4-96	8	6
NCo310 x CP44-101	24	29.9 ± 38.0	0-96	8	2
NCo310 x 54N7096	28	60.0 ± 34.8	0-100	8	5
NCo310 x Q58	15	55.2 ± 46.9	0-96	8	8
Q79 x CP53-19	16	37.8 ± 47.9	0-88	3	4
Q91 x 59S55	8	33.6 ± 51.1	0-94	4	7
Q93 x 54N7096	34	62.2 ± 31.2	0-100	7	5
Q100 x 61N1184	16	34.8 ± 47.6	0-100	3	8
Q101 X CO6501	8	14.3 ± 16.5	0-47	4	1
Q117 x H52-246	10	97.0 ± 58.1	86-100	4	8
49R3863 x 59S55	10	50.4 ± 61.1	0-100	2	7
60S223 x CO6501	21	13.7 ± 27.7	0-96	2	1
60S7493 x 66C807	22	16.5 ± 33.4	0-100	4	2
60S7493 x L62-96	9	49.0 ± 64.2	0-94	4	8

Table 3

Per cent diseased seedlings and parent ratings for 1979 original seedling experiment

Cross	% seedling infection	♀ rat.	♂ rat.
71N814 x H49-3666	83.33	*	8
Q117 x 62N865	79.17	5.5	8.5
Q79 x CP53-19	66.67	2	4
63N1700 x 63S229	66.67	6	3
NCo310 x 54N7096	62.50	8	5
70N1536 x CP50-11	58.33	8	3
58N829 x 59S55	54.17	7	7
61N1184 x 58N829	54.17	8	7
Q101 x 58N1868	54.17	4	5
60N1853 x H44-2818	54.17	4	9
61N1726 x Co440	54.17	9	4
60S7493 x 64B33	50.00	4	1
61N1017 x 65A17	50.00	7	6
Q68 x 63N1700	50.00	3	6
64C386 x CP33-19	45.83	5	4
CP45-184 x CP53-19	45.83	6	4
67N2643 x CP50-11	45.83	*	3
NCo310 x Q58	45.83	8	8
59N154 x Vesta	45.83	2	4
Q113 x 58N1868	45.83	5	5
61N1017 x Q68	45.83	7	3
Q117 x H52-663	45.83	5.5	4
Q93 x CP44-101	41.67	7	2
Q101 x 70N959	41.67	4	8
Co1007 x 54N7096	37.50	3	5
L56-7 x CP49-50	37.50	2	3
Q86 x Co331	37.50	7	4
59C879 x 70N1691	37.50	7	*
58N1868 x Co440	37.50	5	4
62N1659 x B42231	37.50	4	8
Q86 x N6	37.50	7	3
Q93 x Co662	37.50	7	3
63S159 x 71B6	37.50	7	*
Q95 x CP43-47	33.33	3	4

Table 3 (Continued)

Cross	% seedling infection	♀ rat.	♂ rat.
65C152 x 61N1232	33.33	4	8
CP55-14 x 67N1509	33.33	5	7
L56-7 x 64B33	33.33	2	1
75N1685 x CP50-11	33.33	*	3
NCo310 x CP44-101	33.33	8	2
CP50-11 x 61N1232	33.33	3	8
61N1184 x Vesta	29.17	8	4
67N2643 x 63S782	29.17	*	3
Q63 x R397	29.17	5	3
Co331 x 63N1700	29.17	4	6
Q87 x CP57-614	29.17	2	2
Q100 x H57-1472	29.17	3	7
CP45-184 x 33MQ371	29.17	6	6
62N1659 x CP49-50	29.17	4	3
L56-7 x N6	29.17	2	3
TS64-375 x Q68	29.17	*	3
60N1853 x 67N1352	25.00	4	*
64C386 x Co6602	25.00	5	0
F144 x CP44-155	25.00	4	4
70N1564 x Co331	25.00	8	4
62C383 x CP44-101	25.00	8	2
CP55-14 x CP6605	25.00	5	3
54N7096 x CP50-11	25.00	5	3
60N795 x 60S7473	25.00	6	4
60S7493 x 70N1691	25.00	4	*
63N1700 x Co622	20.83	6	3
L56-7 x CP53-19	20.83	2	4
64BN4423 x CP50-11	20.83	2	3
L56-7 x CP57-614	20.83	2	2
62N1659 x Vesta	20.83	4	4
61N49 x CP43-47	16.67	4	4
66C807 x Col148	16.67	2	1
Co740 x Co6602	16.67	3	0
NCo310 x Co6602	16.67	8	0
65C573 x CP44-155	16.67	4	4

Table 3 (Continued)

Cross	% seedling infection	♀ rat.	♂ rat.
Q79 x CP43-47	16.67	2	4
59C879 x Col148	16.67	7	1
Col007 x Co6602	16.67	3	0
CP61-37 x Col148	16.67	6	1
60N1853 x Q68	16.67	4	3
61N1184 x Co6501	16.67	8	1
68N1680 x Col001	16.67	2	6
60N2111 x MQ71-24	12.50	4	0
60N1853 x 60S7473	12.50	4	4
65C152 x Co440	12.50	4	4
66C789 x CP44-101	12.50	1	2
Co740 x 63B28	12.50	3	0
CP49-50 x CP44-101	12.50	3	2
70N1671 x CP50-11	12.50	*	3
63S159 x CP63-588	12.50	7	1
62N1659 x CP50-11	8.33	4	3
Q96 x CP44-101	8.33	7	2
66C807 x CP63-588	8.33	2	1
58N2061 x CP50-11	8.33	1	3
67N2254 x 63S229	8.33	2	3
Q87 x Col148	8.33	2	1
Q87 x 70N1691	8.33	2	*
63S782 x CP50-11	8.33	3	3
66C126 x Co6501	4.17	1	1
CP49-50 x Co331	4.17	3	4
CP51-21 x CP63-588	4.17	2	1
60N1853 x MQ71-24	4.17	4	0
68N4528 x 63S782	4.17	*	3
Q96 x CP63-588	4.17	7	1
60S7493 x CP57-614	4.17	4	2
W60-61 x Co6602	0.00	2	0

* No parent rating available

Project 699-7723

Cross and parent means for per cent diseased stalks/stool and visual ratings are presented in the Appendix.

Unfortunately, the level of infection in this project was very high, and many parent varieties and crosses passed their threshhold of resistance. As a result, many moderately resistant varieties became heavily infected, and discrimination between varieties became difficult. Because of this, it was decided to supplement the traditional method for assessing resistance (based on per cent of stools diseased) with a visual rating of each stool. The visual rating took into account the effect of the disease on the plant (see materials and methods). In addition, number of infected stalks per stool was obtained.

The mean per cent infected stools for each parent variety and cross is presented in Table 4 together with coefficients of variation.

Table 4

Per cent diseased stools for parent and progeny analyses for each factorial in Project 699-7723

Factorial	Parent varieties		Crosses	
	Mean	c.v.	Mean	c.v.
A	95.9	7.0	81.8	14.1
B	70.1	19.6	65.1	21.9
C	82.9	16.9	87.1	13.1
D	85.5	12.8	69.2	20.1
E	95.9	8.7	90.0	13.2
F	95.1	5.2	89.6	12.5
G	91.0	18.2	93.4	8.6

One interesting feature of Table 4 was that the means for crosses were generally less than those for parent varieties.

Analyses of variance were performed on parent varieties and crosses for each factorial. Combined analyses were also performed.

Characters analysed were:-

(a) Per cent stalks/stool infected
(b) Arscin (a)
(c) Per cent stalks/plot infected
(d) Arcsin (c)
(e) Per cent stools infected
(f) Arcsin (e)
(g) Visual rating

Only (a), (b), and (g) could be analyzed on a plant basis, all other analyses being performed on a plot basis. Estimates of variance components and genetic and environmental parameters are presented in Tables 5 and 6 for parent varieties and crosses respectively. These estimates are all based on the combined analysis.

Table 5

Estimates of variance components and genetic and environmental parameters for parent varieties

Statistic	Character*						
	(a)	(b)	(c)	(d)	(e)	(f)	(g)
σ^2_s	565 ± 22	446 ± 17					3.12 ± .12
σ^2_E	70 ± 18	55 ± 14	111 ± 16	93 ± 13	121 ± 17	130 ± 18	0.55 ± .12
σ^2_G	377 ± 83	306 ± 67	374 ± 82	265 ± 59	244 ± 57	190 ± 47	4.69 ± .99
σ^2_P	1012 ± 86	807 ± 70	485 ± 82	357 ± 59	365 ± 57	320 ± 48	8.36 ± 1.00
g^2	.37 ± .05	.38 ± .05					.56 ± .05
g^2 (family)	.75 ± .04	.76 ± .04	.77 ± .04	.74 ± .05	.67 ± .06	.59 ± .06	.84 ± .03

* for key to characters (a)-(g), see text

Parent-offspring regressions were calculated for characters (a) to (g) for each factorial and were combined into one analysis. In addition, offspring means were correlated with mid-parent ratings, using ratings that had been established in previous trials. These results are presented in Table 7 in addition to estimates of σ^2A obtained from the regression analysis.

In Table 8, the two estimates of σ^2A and σ^2G from different analyses are compared.

The within plot mean square, σ^2W, in the progeny analyses is a function of plant to plant environmental variance and fractions of σ^2A and σ^2D (see materials and methods). Plant to plant environmental variance is estimated in the analysis of parent varieties while σ^2A and σ^2D are estimated from the progeny analyses. Therefore, by subtracting these estimates from σ^2W, a second estimate of σ^2s, the plant to plant environmental variance, may be obtained. These estimates are presented in Table 9.

Table 6

Estimates of variance components and genetic and environmental parameters for crosses

Statistic	Character*						
	(a)	(b)	(c)	(d)	(e)	(f)	(g)
σ^2_W	1116 ± 31	867 ± 24					7.40 ± .20
σ^2_E	40 ± 16	33 ± 12	144 ± 15	89 ± 9	160 ± 16	131 ± 13	.25 ± .10
σ^2_{MF}	133 ± 34	107 ± 27	167 ± 40	84 ± 21	141 ± 36	103 ± 27	.98 ± .24
σ^2_F	87 ± 41	68 ± 32	101 ± 48	61 ± 27	52 ± 31	36 ± 22	.87 ± .37
σ^2_M	91 ± 41	77 ± 34	76 ± 39	72 ± 30	39 ± 28	36 ± 23	1.11 ± .42
σ^2_A	356 ± 118	288 ± 95	354 ± 127	265 ± 82	181 ± 87	143 ± 66	3.95 ± 1.12
σ^2_D	531 ± 137	428 ± 110	669 ± 160	336 ± 85	563 ± 145	411 ± 109	3.90 ± .97
σ^2_G	888 ± 153	717 ± 123	1023 ± 170	601 ± 102	745 ± 135	555 ± 103	7.86 ± 1.32
σ^2_P	1467 ± 66	1151 ± 53	1167 ± 169	690 ± 101	905 ± 133	685 ± 101	10.60 ± .60
h^2	.24 ± .07	.25 ± .07					.37 ± .09
g^2	.61 ± .08	.62 ± .09					.74 ± .09
h^2 (family)	.34 ± .10	.34 ± .10	.30 ± .10	.38 ± .10	.20 ± .09	.21 ± .10	.45 ± .09
g^2 (family)	.85 ± .03	.86 ± .03	.88 ± .02	.87 ± .02	.82 ± .03	.81 ± .03	.89 ± .02

Table 7

Regression of offspring means on mid-parent means for characters (a) to (g) and correlation of offspring means with mid-parent ratings

Character	Regression analysis		Correlation with mid-parent rating
	Regression coefficient	σ^2_A	
(a)	.505 ± .125	190.2	.584
(b)	.456 ± .125	140.9	.580
(c)	.507 ± .133	188.8	.609
(d)	.422 ± .125	113.5	.616
(e)	.453 ± .138	121.4	.443
(f)	.438 ± .133	95.1	.481
(g)	.425 ± .109	1.86	.660

Table 8

Comparison of estimates of genetic variances from different analyses

Character	σ^2_A				σ^2_G		
	Parent-offspring covariance, A_1	Progeny, A_2	A_1/A_2	σ^2_D	Parents, G_1	Progeny, G_2	G_1/G_2
(a)	190	356	.53	531	377	888	.42
(b)	141	288	.49	428	306	717	.43
(c)	189	354	.53	669	374	1023	.37
(d)	114	265	.43	336	265	601	.44
(e)	121	181	.67	563	244	745	.33
(f)	95	143	.66	411	190	555	.34
(g)	1.86	3.95	.47	3.90	4.69	7.86	.60

Table 9

Estimates of variance components involved in the within plot mean square for the progeny analyses

Analysis	σ^2_W (P.A.)	σ^2_s (V.A.)	$\frac{1}{2}(\sigma^2_A + \frac{3}{4}\sigma^2_D)$ (P.A.)	$\sigma^2_W - \sigma^2_P$ $(=\frac{1}{2}\sigma^2_A + \frac{3}{4}\sigma^2_D)$	$\sigma^2_W - (\frac{1}{2}\sigma^2_A + \frac{3}{4}\sigma^2_D)$ $(=\sigma^2_{s'}$ P.A.)
(a)	1 116	565	576	551	540
(b)	867	446	465	421	402
(g)	7.40	3.12	4.90	4.28	2.50

P.A. = progeny analysis

V.A. = parent varieties analysis

DISCUSSION

In each of the three projects, estimates of the correlation between mean per cent diseased stools for a family and mid-parent mean rating were obtained. Estimates ranged from 0.75 in project 699-7718 to 0.63 in project 699-7729 to 0.44 in project 699-7723. Due to the very severe infection in project 699-7723, discrimination between crosses and parent varieties based on per cent stools infected was very poor, and this has a detrimental effect on the estimation of correlations. For project 699-7723, the visual estimate of rating provided better discrimination between crosses and parent varieties, and the correlation between this character and mid-parent mean rating was 0.66. This correlation is comparable with the correlations between mean per cent diseased stools for a family and mid-parent mean from the other projects.

These correlations are estimates of heritability on a family mean basis. The values obtained are quite high, and indicate that considerable progress in breeding for resistance can be made by selecting parents with resistance.

In the inheritance project (699-7729) on Bundaberg Station, the heritability estimate of 0.63 was not much less than the estimate of degree of genetic determination of 0.69. These estimates were derived from different analyses, but suggested that most of the genetic variance was additive. If this is the case, there is little to be gained from progeny testing, i.e. testing each family for its resistance to Fiji disease.

However, the results of project 699-7723 on the Pathology Farm show that non-additive genetic variance, $\sigma^2 D$, is very important compared to $\sigma^2 A$, the additive genetic variance (Table 6). In view of the high infection pressure, visual rating (analysis (g) is regarded as the most reliable method of assessment but, even for this character, $\sigma^2 D$ is equal to about half the genetic variance. If this result is correct, some progress could be made by selecting resistant parents but it would also be desirable to conduct some progeny testing. For characters other than visual rating, the proportion of non-additive genetic variance was much higher.

An indication of the possible importance of progeny testing can be obtained by comparing h^2 and g^2 on a family basis in Table 6. For estimating gains from selection, h^2 would be used if selection of resistant crosses depended on selection of parents whereas g^2 would be used if progeny testing were conducted. For visual ratings, progeny testing would be about twice as successful as selecting parents.

However, there is some doubt about the reliability of the estimates of genetic variances, due to the severe infection pressure. There are at least two ways in which this could have caused an interaction between males and females and, therefore, produced a high estimate for non-additive variance.

Firstly, the case of two crosses of almost equal susceptibility may be considered. Under high infection pressure, the progeny from one cross may pass its threshhold of resistance and become heavily infected, while the other cross may have a much lower infection level because it has not passed its threshhold of resistance. Secondly, the case of one highly susceptible cross and one moderately susceptible cross may be considered. The highly susceptible cross will always have a high level of infection, but the moderately susceptible cross only has a high level of infection when infection pressure is high. Under these circumstances, there will only be a small difference in family means whereas there would normally be a large difference. When factors such as the two discussed are operating, interaction will result.

However, despite these arguments, it is very easy to find examples of interaction in the tables of means. For example, in factorial A, consider the visual ratings for the crosses between 65C286 and Co740 as females and Co622 and F151 as males:-

	Co622	F151
65C286	5.9	2.9
Co740	2.3	5.4

In this two-way table, there are virtually no differences between males or females, but there is a large interaction.

Evidence discussed later indicates that epistasis was probably unimportant, which means that most of the non-additive effects are due to dominance. This could well be the case if the example of 49R3863 and Q68 in factorial A is considered. Both of these varieties are moderately resistant with 49R3863 being rated as a 2 and Q68 as a 3. However, under the conditions of this trial, both varieties had 100 per cent infection.

Mean visual ratings were 9 for 49R3863 and 8.65 for Q68. Obviously both varieties had passed their threshhold of resistance. However, the interesting point is that progeny of 49R3863 and Q68 showed good resistance. The mean visual rating of progeny of 49R3863 was 3.9 while that for Q68 was 4.5. The mean visual rating for the cross 49R3863 x Q68 was only 1.2. This situation could arise if resistance is controlled by a number of dominant genes.

Another very interesting cross is 62N1659 x L62-68 in factorial D. Mean visual ratings for the parents were 7.0 for L62-68 and 5.1 for 62N1659 whereas

the cross had a mean visual rating of 0.03 and a mean per cent infected stalks/stool of 0.8. Unfortunately, this cross only had three seedlings (which were repeated to give 10 stools per plot), but it would seem to have exceptionally high resistance despite the relatively susceptible reactions of its parents. If the parents were largely homozygous for the alleles controlling resistance, it is possible that the cross could be much more resistant if dominance is important. Because of the exceptional resistance of this cross, every effort should be made to repeat the cross and retest it for resistance.

Until the results of the second Bundaberg inheritance experiment are available, it would be prudent to assume that non-additive genetic variance is important. This should be considered when planning a breeding strategy.

Comparison of estimates of genetic variance

Additive genetic variance was estimated from the progeny analysis and from the covariance of parents and offspring while total genetic variance was estimated from the progeny analysis and the parent analysis (Table 8). As discussed above, the progeny analyses were affected by the heavy infection pressure. However, the parent analyses were even more heavily infected. This is because once a parent variety passed its threshhold of resistance, it normally became 100 per cent infected. With crosses, individual susceptible varieties were heavily infected but usually a few varieties were more resistant and were not infected, so that there tended to be more variation between crosses. An indication of the heavier infection for parent varieties can be obtained from Table 4 which shows that mean per cent stools infected for parents was generally higher than for progeny. For example, in factorial A, seven of the 13 parents were 100 per cent infected whereas only six of the 36 crosses were 100 per cent infected.

As a result, differences between varieties were minimized, and this reduced the estimates of genetic variance. It aslo reduced the range of means, which would have reduced the covariance of offspring means and mid-parent means from which an estimate of σ^2A is made.

This effect is shown up in Table 8 where the two estimates of σ^2A and σ^2G are compared. Theoretically, the estimate of σ^2A from the parent-offspring covariance should be greater than the estimate from the progeny analysis (Hogarth, 1977), and the estimate of σ^2G from the parent analysis should be greater than that from the progeny analysis. In fact, the reverse was true for both σ^2A and σ^2G. This was almost certainly due, in both cases, to the reduced variation for parent varieties but is also an indication of no epistatic variance.

The poor discrimination between mid-parent means explains why higher estimates of heritability on a family basis were obtained by correlating offspring means with mid-parent ratings compared with finding the regression of offspring means on mid-parent means for the same character (Table 7). The mid-parent ratings were based on previous trial results, and did not depend on the experiment at all.

Comparison of estimates of environmental variance

From Tables 5 and 6, estimates of plot to plot environmental variance, σ^2E, may be compared for the parent and progeny analyses. The two estimates compare well, except possibly for visual rating.

For the parent analysis, an estimate of plant to plant environmental variance, σ^2s, is also obtained. In the progeny analysis, σ^2s is confounded with components of genetic variance but, in Table 9, these have been subtracted from the within plot mean square to give an estimate of σ^2s for the progeny analysis. The two estimates of σ^2s agree very well. The estimate of σ^2s from the progeny analysis is confounded with components of epistatic variance (Hogarth, 1977), and would be expected to be greater than the estimate from the parent analysis if epistasis were important. For each of the three analyses, the estimate was lower, so there was no evidence to suggest epistatic effects.

Transformation of data

Results have been presented for analyses based on raw data and for analyses using an arcsin transformation. For the estimation of genetic statistics such as h^2 and g^2, the transformation made very little difference. In any publication of these results, it should be stated that the arcsin transformation was studied but made little difference. Analyses with untransformed data only should be presented.

Breeding strategy

There is ample evidence from the three projects to show that selection of parents with Fiji resistance is effective for selecting crosses with resistance. However, the fact that substantial non-additive genetic variance was demonstrated in the project at the Pathology Farm indicates that an attempt should be made to conduct progeny testing, at least until the results of the second Bundaberg inheritance project are available.

A further indication that progeny testing is desirable is provided by the results presented in Tables 2 and 3. It may be seen from the standard deviations and ranges that the level of resistance within a cross is highly variable. Highly susceptible crosses such as 65C573 x Col001, NCo310 x 54N7096 (Table 2), 60N1853 x H49-3666, 60N1853 x H44-2818, and 62N1659 x H49-3666 (Table 3) are still capable of producing resistant progeny. All of these crosses produce a high proportion of varieties with acceptable yield. Rejection of the crosses on the basis of Fiji disease susceptibility would prevent the selection of high yielding varieties with Fiji disease resistance. However, progeny testing at an early stage of selection would be necessary to avoid the selection of large numbers of attractive varieties that had to be discarded at a later stage due to Fiji disease susceptibility.

Most proven crosses have undergone progeny testing already in the 30-sett plots where susceptible varieties become infected and are easily identified. Therefore, we only need to concern ourselves principally with experimental crosses.

The best method would seem to be to plant experimental crosses in bunches between rows of infected material, and to select the bunches in the ratoon crop. In the longer term, if infection of bunches in the glasshouse is shown to be an

effective screening technique, this method could replace the former method. Infection in the glasshouse has the advantage that selection can take place in the plant crop. In addition, it would not be necessary to plant infection rows.

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APPENDIX

Means for crosses and parents in Project 699-7723

Factorial A

(a) Per cent infected stalks/stool

Female	Male									Female parent mean
	Co622	F151	H36-7913	H44-2818	H49-3666	H52-246	Q68	Vesta	59S55	
65C286	81.7	66.8	48.7	100.0	91.6	94.4	82.1	55.8	91.6	87.3
Co740	42.4	76.0	62.5*	100.0	93.1	93.3*	66.6	46.5*	74.8	91.7
49R3863	49.9	46.0	62.5	67.7	71.3	90.2	24.5	48.1	63.4	100.0
Triton	83.6	95.4	78.5	100.0	93.8	100.0	71.3*	37.9	95.4	86.4
Male parent mean	70.3	97.6	90.6	100.0	100.0	100.0	100.0	74.4	99.2	

(b) Visual ratings

Female	Male									Female parent mean
	Co622	F151	H36-7913	H44-2818	H49-3666	H52-246	Q68	Vesta	59S55	
65C286	5.9	2.9	3.9	7.5	7.4	8.0	6.1	3.1	7.0	6.0
Co740	2.3	5.4	3.7*	8.1	7.3	7.2*	4.9	2.7*	3.1	8.1
49R3863	2.4	2.6	4.0	5.4	5.6	6.5	1.2	4.0	3.4	9.0
Triton	6.3	6.2	4.4	8.3	7.9	8.4	5.6*	2.2	7.7	5.5
Male parent mean	4.2	7.7	6.3	8.2	8.4	9.0	8.7	5.2	7.9	

* Missing plot estimate

Note: Male and female parent mean values are from parent analysis; cross means are from progeny analysis

Factorial B

(a) Per cent infected stalks/stool

Female	Male				Female parent mean
	Co6501	CP43-47	Q65	63S782	
66C126	23.5	55.3	24.5	30.0	5.6
Phil56-98	77.9	50.1	80.6	57.2	100.0
Q77	63.4	62.5	81.6	65.0	93.6
60S223	28.2	61.7	32.1	36.4	71.1
Male parent mean	7.8	74.5	98.4	33.7	

(b) Visual rating

Female	Male				Female parent mean
	Co6501	CP43-47	Q65	63S782	
66C126	1.6	3.3	1.7	2.6	0.4
Phil56-98	6.1	4.2	6.6	4.2	8.6
Q77	5.1	4.3	5.1	4.6	7.9
60S223	1.8	3.9	2.3	2.4	4.3
Male parent mean	0.2	4.5	5.8	2.1	

Factorial C

(a) Per cent infected stalks/stool

Female parent	Male				Female parent mean
	62C366	CP57-526	H36-7913	H52-246	
Co954	49.5	77.9	72.5	99.8	75.7
Q117	84.7	85.6	84.4	95.4	99.3
Q73	58.2	59.8	62.0	87.8	21.3
Q88	72.3	88.8	59.3	89.8	78.8
Male parent mean	70.4	97.2	56.0	100.0	

(b) Visual rating

Female parent	Male				Female parent mean
	62C366	CP57-526	H36-7913	H52-246	
Co954	2.8	3.5	4.9	6.4	2.6
Q117	6.6	6.7	6.3	8.0	8.0
Q73	3.9	4.2	4.5	5.9	1.3
Q88	4.6	5.6	4.0	7.2	5.0
Male parent mean	4.8	7.9	3.6	9.0	

Factorial D

(a) Per cent infected stalks/stool

Female parent	Male				Female parent mean
	Co954	Co6605	L62-68	L62-86	
F144	58.2	64.5*	66.6	36.5	93.1
62N1659	63.2	47.6	0.8	59.4	85.0
63S159	66.7	80.5	62.4	67.6*	91.9
Male parent mean	75.5	79.1	99.3	59.7	

(b) Visual rating

Female parent	Male				Female parent mean
	Co954	Co6605	L62-68	L62-86	
F144	3.3	4.4*	3.8	2.0	5.0
62N1659	2.5	3.5	0.0	3.2	5.1
63S159	4.1	5.9	5.3	4.8*	7.6
Male parent mean	2.6	6.4	7.0	2.9	

* Missing plot estimate

Factorial E

(a) Per cent infected stalks/stool

Female parent	Male			Female parent mean
	Co331	Co475	58N1000	
64A490	73.1	95.8	75.7	84.0
B54163	90.9	69.4	94.5	95.6
65C286	77.8	93.5	76.5	89.1
CP55-14	92.8	86.1	98.0	89.6
Male parent mean	78.5	100.0	93.8	

(b) Visual rating

Female parent	Male			Female parent mean
	Co331	Co475	58N1000	
64A490	5.1	8.3	6.9	6.6
B54163	6.6	6.1	8.1	8.1
65C286	5.5	7.4	5.9	6.1
CP55-14	7.0	7.0	8.3	7.4
Male parent mean	2.8	9.0	8.2	

Factorial F

(a) Per cent infected stalks/stool

Female parent	Male 63N1700	39SN3821	Vesta	Female parent mean
Cadmus	81.9	86.5	69.6	89.2
CP52-68	71.7*	79.1	69.3	100.0
60N1853	93.0	96.5	79.6	95.5
61N1184	86.0	100.0	87.1	99.2
Male parent mean	99.6	98.4	71.6	

(b) Visual rating

Female parent	Male 63N1700	39SN3821	Vesta	Female parent mean
Cadmus	6.5	7.2	4.9	8.0
CP52-68	4.5*	6.0	5.0	8.1
60N1853	5.8	7.9	5.7	6.2
61N1184	5.7	8.6	7.4	8.2
Male parent mean	6.6	7.3	6.3	

* Missing plot estimate

Factorial G

(a) Per cent infected stalks/stool

Female parent	Male			Female parent mean
	CP44-155	CP52-68	L62-96	
65C573	84.1	96.3	92.9	56.1
NCo310	90.4	76.5	100.0	100.0
Q86	61.1	91.7	88.6	100.0
Male parent mean	93.3	91.7	75.9	

(b) Visual rating

Female parent	Male			Female parent mean
	CP44-155	CP52-68	L62-96	
65C573	5.8	7.0	6.9	1.9
NCo310	6.0	4.6	8.1	7.6
Q86	4.0	6.7	6.8	7.9
Male parent mean	3.3	7.9	6.6	