Theor Appl Genet (2000) 100:557–563

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The genetics of rust resistance in sugar cane seedling populations

Received: 2 June 1999 / Accepted: 22 June 1999

Abstract The inheritance of rust resistance was studied in sugar cane seedling populations using a factorial mating design over 1 summer and 2 winter seasons. Frequency distributions for rust infection pooled over 2 winter seasons for resistant x resistant parents were highly skewed with the majority of progenies grouped towards the resistant classes, whereas crosses between susceptible x highly susceptible parents tended to be skewed with the majority of progenies grouped towards the susceptible ones. Both categories of crosses produced transgressive segregants at either extremes. Distribution of infection within progeny of the selfed resistant parent 'R 570' and distribution in the majority of crosses tended to support the hypothesis of a major gene with a dominant effect for resistance. However, the action of other minor genes acting in a quantitative way is also suggested. The female (F) and male (M) variance components were very important, and F×M interaction indicated the existence of non-additive genetic effects. F×S, M×S and F×M×S interaction mean squares were generally low or insignificant. Broad-sense heritability for the individual season ratings and for the combined ratings was high (0.75–0.90), whereas narrow-sense heritability was generally moderate (0.40–0.52) with the additive genetic effects accounting for 44–68% of the total genetic variation. The implications of these findings in the breeding for rust resistance in the local programme are discussed.

Key words Heritability · Major gene · *Puccinia melanoceplala* · Rust · Sugar cane

Communicated by H.F. Linskens

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Introduction

Sugar cane common rust *(Puccinia melanocephala* H. & P. Syd.) is an economically important disease in many sugar cane-growing countries. The disease has caused economic losses in susceptible varieties in various countries (Ryan and Egan 1989) and has called for changes in breeding and selection programmes to produce rust-resistant varieties (Tai et al. 1981, Hogarth et al. 1993). Breeding in sugar cane uses the sexual route to create new variations which are fixed in clonally propagated cuttings for successive selection stages.

In Mauritius the conditions favourable to the development of the rust pathogen are found in the humid and very humid regions found mostly in the centre and eastern sector of the island (rainfall >1800 mm, altitude >150 m), which represents about 40% of the area under cane. Sugar cane varieties bred for this zone need to be resistant to rust as the use of fungicides is neither cost-effective nor environmentally acceptable. Rust is most severe in young canes a few months after planting and in young regrowth (ratoons) following each year's harvest. A severe winter peak (June to September) and a less severe summer peak (December–January) of disease infection are encountered on juvenile canes in rust-prone areas in Mauritius. Rust epidemics in the mid-1980s which drastically reduced the cultivation of two commercial varieties ('M 2370/62', 'M 1030/71') in the humid and very humid zones (MSIRI 1985, 1987, 1989) have called for the systematic screening of promising varieties against the disease in final phase trials (Ramdoyal et al. 1996).

Cultivated sugar cane varieties are complex interspecific aneuploids with chromosome numbers ranging from 2n=100–130 (Sreenivassan et al. 1987). Chu et al. (1982) had assumed that rust-susceptible genes of modern commercial varieties are transmitted mainly by some *S. officinarum* clones which account for around 90% of the genome of commercial varieties (Bremer 1961, D'Hont et al. 1996) and suggested that the character was not likely to be determined by a single gene.

Results on the genetics of rust resistance are not conclusive. Tai et al. (1981) observed marked transgressive segregation towards susceptibility in bi-parental crosses and selfed families and suggested that resistance to rust was partially dominant. Intermediate heritabilities for rust resistance were reported by Tai et al. (1981) and Gonzalez et al. (1987), whereas high narrow-sense and broad-sense heritabilities were reported by Comstock et al. (1992) and Hogarth et al. (1993). Daugrois et al. (1996) attributed resistance to rust in the progeny of the selfed cultivar ‘R 570’ to a major gene with dominant effect.

A series of experiments was initiated under the Mauritian conditions to study the inheritance of rust to guide breeding strategies for the humid and very humid zones. A preliminary study suggested the involvement of a few genes with major effects (Ramdoyal et al. 1996). A second series of trials was set up in 1993 with repeated measurements over 3 seasons from 1994 to 1996 and aimed to unravel further the genetics of rust resistance.

Materials and methods

Plant material

The crosses were made in the glasshouse under artificial conditions (Ramdoyal et al. 1995) between May and June 1992 between six female varieties, (two resistant, ‘M 2077/78’, ‘M 937/77’; two slightly susceptible: ‘M 695/69’, ‘S 17’; two susceptible, ‘Q 96’, ‘M 555/60’) and two male varieties, ‘R 570’ (resistant) and ‘M 1030/71’ (highly susceptible). The two male parents were also selfed, and a reciprocal cross was carried out between the two male parents, in which case flowering stalks of the two varieties were obtained from environments which favoured either female or male characteristics, respectively. Warm conditions during flower development promote anther dehiscence and increase the fertility of pollen in a number of varieties, whereas cool environments inhibit anther dehiscence. A factorial mating system was used with eight female varieties (including selfing and reciprocal crosses) and two male varieties, which resulted in 16 families.

However, only 12 families which included 4×2 factorial crosses produced enough seedlings to start the experiment. Seed setting in sugar cane meets with varying success mainly as a consequence of pollen and ovule sterility that frequently results in low set set or its absence entirely in a large number of crosses, particularly under sub-tropical conditions with low night temperatures (Nuss 1978; Berding 1981). The other 4 crosses were included in the trial at a later date. Families were raised from seeds in sowing trays, and each seedling was transplanted to a 10-cm plastic pot at the four- to five-leaf stage. After 3 months of hardening the seedlings were transferred to the experimental site in a very humid environment at Bonne Veine (altitude, 470 m, annual rainfall, 2800 mm, yearly mean minimum and maximum temperatures of 14°C and 26.7°C, respectively). The first series of 12 crosses was transplanted in April 1993, whereas the other 4 families were transplanted to the experimental site in December 1993 after a second series of crossing and sowing.

The design used was a randomised complete block with two replicates, and the randomisation of families was done within each of the two series of crosses. Each family was split in two groups of 52 progenies allocated to each replicate. The families were planted in two rows of 26 progenies per row for each replicate, with 1 m within each row and 1.5 m between rows.

Parents established as single stools in pots from one-eyed cuttings were transplanted at regular intervals in each family row. Each female and each male parent was replicated four times within each family row per replicate. Eight control varieties were established as single stools in pots from one-eyed cuttings, ‘M 3035/66’, ‘R 570’ (resistant), ‘M 2173/63’, ‘M 657/66’ (slightly susceptible), ‘M 147/44’, ‘M 442/51’ (susceptible), ‘M 1030/71’, ‘B 4362’ (highly susceptible), and included in the trials. The control varieties also included the two male parents ‘R 570’ and ‘M 1030/71’, which are among the control varieties normally included in rust trials in Mauritius. The controls were planted at regular intervals within the family rows and were replicated across the families; there were as many replicates of each control as there were families.

Each family row, which included progeny, parents and controls, was bordered on each side by an inoculum row of the highly susceptible variety ‘M 1030/71’. The total experimental site occupied an area of around 0.78 ha.

Disease evaluation

The first series of seedlings that had been transplanted in April 1993 and that had reached a stage of growth conducive to rust infection were evaluated on the January 13, 1994 during the summer peak epiphytotic. Subsequently, the whole trial was shaved in April 1994 and at the beginning of March 1995 such that the developmental stage of the regrowths would coincide with the winter peak infections of 1994 and 1995. Winter evaluations were conducted on September 6, 1994 and June 26, 1995. All progenies, parents and controls were given a visual rating for rust on a scale of 1–6 based on infection on all leaves. Ratings were graded as follows: 1, no symptoms – resistant (R); 2, light infection, few lesions on lower leaves – slightly susceptible (SS); 3, slight infection, moderate number of lesions on lower leaves – slightly susceptible (SS); 4, moderate infection, numerous lesions on old leaves and few lesions on young leaves – susceptible (S); 5, heavy infection, abundant lesions on old leaves and numerous lesions on young leaves with or without some necrosis – susceptible (S); 6, very heavy infection, abundant lesions on old and young leaves with necrosis and death of leaves – highly susceptible (HS).

The first summer rating was conducted by four teams, but the 2 subsequent winter ratings were conducted by one team only to remove any bias which could arise from subjectivity of the enumerator.

Analysis

Basic statistics were calculated for the crosses at each date of evaluation and on pooled data for the last 2 winter evaluations. Weighted means for the families were calculated from the distributions of the progenies in each reaction classes according to Sokal and Rohlf (1981). The model of a major gene with dominance effect proposed by Daugrois et al. (1996) from the progeny derived from selfing the resistant parent ‘R 570’ was tested using the chi-square test for goodness of fit. The individual season rating was analysed according to the basic analyses of variance for progeny and parents according to the factorial mating design proposed by Hogarth et al. (1981). The data combined over the evaluation seasons were analysed using a repeated-measure design. This analysis was conducted for the first series of factorial crosses (4 females×2 males) pooled over the three evaluation seasons whereas the whole set of factorial crosses (8 females×2 males) was pooled over the last 2 winter seasons. The additive (σ^2_A) and non-additive (σ^2_D) genetic variances were derived from variance components attributed to the female, male and the interaction between female and male as follows:

$$\sigma^2_A=2(\sigma^2_f+\sigma^2_m)$$

$$\sigma^2_D=4\sigma^2_{fm}$$

where σ^2_f, σ^2_m and σ^2_{fm} are the female, male and female×male components of variance, respectively. The total genetic variance (σ^2_G) is the sum of σ^2_A and σ^2_D ignoring the component of epistatic variance. The phenotypic variance (σ^2_P) is the sum of genetic and environmental (σ^2_E) component ($\sigma^2_P=\sigma^2_G+\sigma^2_E$), where σ^2_E has been derived from the residual mean square from the basic analysis of the parent and control clones combined together into a single analysis. Estimates of narrow-sense (h^2_n) and broad-sense (h^2_b) heritability were computed from appropriate variances as follows: $h^2_n=\sigma^2_A/\sigma^2_P$ and $h^2_b=\sigma^2_G/\sigma^2_P$.

Results

The mean seasonal rating for control and parent varieties indicated a more severe level of infection in winter seasons than in the summer season (Table 1) with similar ratings across the 2 winter seasons. Data from an individual season rating or combined rating for both controls and parents showed that the clones could consistently be classified across time (seasons) as being resistant or susceptible.

Slight changes in scores were evident within the resistant (R) and slightly susceptible (SS) groups only. The control variety ‘M 2173/63’, normally classified as slightly susceptible, behaved as a resistant clone in all seasons. The parent variety ‘M 695/69’ (SS) had a mean infection rate over the seasons which was lower than of the resistant parent (and control) ‘R 570’.

Weighted means for rust rating of families for the individual season ratings and for the pooled winter ratings are shown in Table 2. In general, resistant x resistant (R×R) crosses had the lowest mean infection rate, whilst highest mean infection rate was recorded for the cross between the susceptible parent, ‘M 555/60’, and the highly susceptible one, ‘M 1030/71’, (S×HS). The mean rating recorded for the selfed family of the highly susceptible parent ‘M 1030/71’ was unexpectedly low. This rating should be viewed with caution because of the high mortality and poor growth observed in this family. In general, mortality rate of the progenies varied largely between the families.

Two female varieties, ‘S 17’ (SS) and ‘M 555/60’ (S), tended to give progenies with higher mean infection rates when crossed with either the resistant male ‘R 570’ or the highly susceptible one, ‘M 1030/71’. In fact, higher mean infection rates were recorded for these two female parents than for the other parents classified in the same susceptibility classes (Table 1).

Frequency distributions for rust infection of families pooled over the 2 winter seasons are shown in Fig. 1 for crosses with the resistant male parent ‘R 570’ (Fig. 1A) and for crosses with the highly susceptible one, ‘M 1030/71’ (Fig. 1B). Segregation of the selfed male parent ‘M 1030/71’ is not shown on account of only a very few progenies which were common to both evaluation dates. Distributions for the R×R crosses were highly skewed with the majority of the progenies grouped towards resistant classes, whereas the distributions for S×HS crosses and 1 SS×HS cross were skewed in the other direction, with the majority of the progenies grouped towards the susceptible classes. All of the progenies of 1 S×HS cross (‘M 555/60’×‘M 1030/71’) were distributed in the susceptible and highly susceptible classes (4–5).

R×R crosses including the selfing of the resistant parent (‘R 570’) produced 70–80% of progenies in the resistant class. This proportion was reduced to around 40% in S×R crosses and to 0–12% in S×HS crosses. The pattern of segregation was nearly similar for the reciprocal crosses between the two male varieties. However, a slight maternal effect was evident depending upon the parent that was used as the seed-bearing one. Hence, a higher proportion of progenies was resistant (53%) when ‘R 570’ was used as the female parent as compared to

Table 1 Weighted means of rust ratings for control and parent varieties evaluated in summer and winter seasons

Control varieties	Category[a]	Summer rating January 13 (1994)	Winter rating		Pooled winter ratings
			September 6 (1994)	June 26 (1995)	
M 3035/66	R	1.00	1.00	1.00	1.00
R 570	R	1.10	1.17	1.33	1.35
M 2173/63	SS	1.00	1.00	1.00	1.00
M 657/66	SS	1.11	1.52	1.16	1.52
M 147/44	S	2.16	2.86	2.08	2.71
M 442/51	S	2.76	4.00	4.44	4.50
B 4362	HS	3.82	3.90	4.40	4.25
M 1030/71	HS	4.00	4.72	4.68	5.11
Mean of controls		*2.12*	*2.52*	*2.51*	*2.68*
Parents					
R 570	R	1.05	1.19	1.36	1.44
M 937/77	R	1.06	1.00	1.00	1.00
M 2077/78	R	1.19	1.04	1.03	1.07
M 695/69	SS	1.07	1.16	1.00	1.17
S 17	SS	1.37	2.62	2.18	2.71
Q 96	S	1.95	3.71	3.63	3.86
M 555/60	S	2.73	4.21	3.79	4.21
M 1030/71	HS	3.92	4.93	4.95	5.10
Mean of parents		*1.79*	*2.48*	*2.37*	*2.57*

[a] R, Resistant; SS, slightly susceptible; S, susceptible; HS, highly susceptible

'M 1030/71'. Conversely, 24% of progenies were classified as highly susceptible when 'M 1030/71' was used as the female parent as compared to only 3% for 'R 570'.

Transgressive segregants towards the high susceptibility classes were evident in R×R crosses, whereas segregants towards the resistant classes were also evident in S×HS crosses. The mode of segregation in the self progeny of the resistant parent 'R 570' suggested a high degree of heterogeneity in that parent (Fig. 1). When the progenies resulting from the selfing of 'R 570' were allocated into two groups of resistant: susceptible based on a complete absence of symptoms (resistant) and presence of symptoms (susceptible), a 3:1 ratio was observed which supports the presence of a major resistant gene with dominant effect segregating in a heterozygous way. Based on this model, the observed and expected number of progenies within each cross were tested by chi-square analysis (Table 3). The results tended to support the major gene hypothesis for resistance for the majority of crosses except for cross 'S 17'×'R 570' which showed a large deviation from expected ratio.

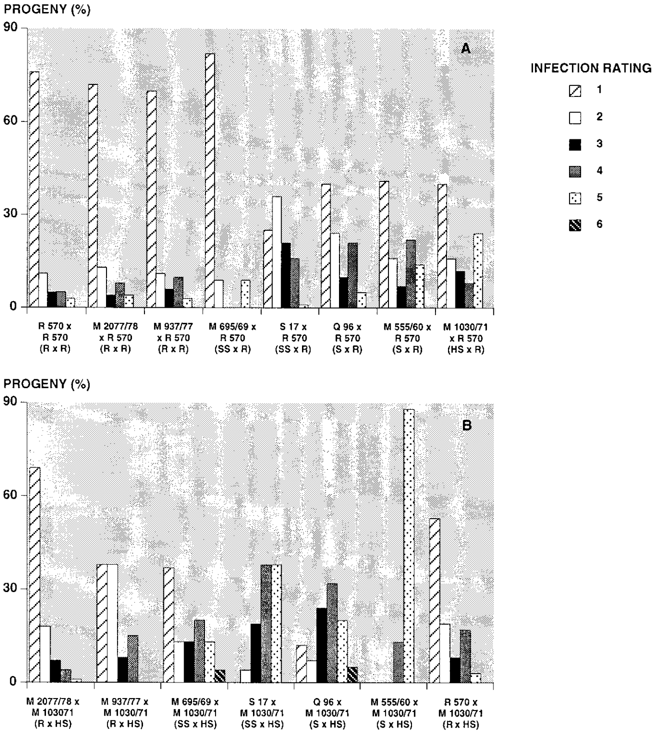

Fig. 1A, B Frequency distributions of rust rating for crosses with the resistant male parent (**A**) and the highly susceptible male parent (**B**) pooled over 2 winter seasons

Mean squares for the clonal (parents and controls) analysis pooled over 3 seasons for the 4×2 factorial crosses and 2 seasons for the 8×2 factorial crosses are shown in Table 4; those for the progeny analysis with expected mean squares are presented in Table 5. All clones differed significantly in their reactions to rust (Table 4). Differences between seasons were significant for the pooled summer and winter ratings, whereas the 2 winter seasons did not differ in rust severity and confirmed the observations reported earlier in Table 1. Clone×season interaction was significant only when the summer rating was included in the analysis. Based on the progeny analysis (Table 5) both female and male variances were highly significant. The highly significant female×male interaction indicated the existence of non-additive genetic effects. The season effect indicated that rust severity will change between seasons. The female×season or male×season interaction mean squares were generally very low. It could not be ascertained whether additive genetic effects would interact with season but this is less likely to be so. However, if the summer ratings (lower infection rates) were ignored, the non-significant F×M×S interaction indicated that non-additive genetic effects do not change over seasons.

Table 2 Weighted means of rust ratings for families evaluated in summer and winter seasons

Families	Category	Summer rating January 13 (1994)	Winter rating September 6 (1994)	Winter rating June 26 (1995)	Pooled winter ratings
R 570×R 570	R×R	1.35	1.36	1.45	1.39
M 2077/78×R 570	R×R	1.14	1.48	1.58	1.51
M 937/77×R 570	R×R	1.37	1.56	1.54	1.55
M 695/69×R 570	SS×R	–	1.33	1.47	1.36
S 17×R 570	SS×R	1.84	2.31	2.26	2.14
Q 96×R 570	S×R	1.71	2.06	2.27	2.11
M 555/60×R 570	S×R	2.26	2.45	2.32	2.38
M 1030/71×R 570	HS×R	1.91	2.00	2.79	2.38
R 570×M 1030/71	R×HS	1.66	1.88	2.10	1.86
M 2077/78×M 1030/71	R×HS	1.64	1.35	1.64	1.43
M 937/77×M 1030/71	R×HS	–	1.62	2.11	1.81
M 695/69×M 1030/71	SS×HS	1.79	2.54	2.57	2.57
S 17×M 1030/71	SS×HS	3.35	3.62	4.29	3.90
Q 96×M 1030/71	S×HS	2.10	3.24	3.37	3.28
M 555/60×M 1030/71	S×HS	–	3.75	4.90	4.38
M 1030/71×M 1030/71	HS×HS	–	1.50	2.88	2.33
Mean season ratings		*1.84*	*2.13*	*2.47*	

Table 3 Observed and expected ratios of resistant (R, absence of symptoms) and susceptible (S, presence of symptoms) progenies for families based on a major gene hypothesis and the chi-square test for gootness of fit

Families	Type of cross	Number of progeny	Observed ratio R:S	Expected ratio R:S	χ^2 [a]
R 570×R 570	R×R	37	28:9	27.75:9.25	0.009
M 2077/78×R 570	R×R	53	38:15	39.75:13.25	0.157
M 937/77×R 570	R×R	70	49:21	52.50:17.50	0.685
M 695/69[b]×R 570	SS×R	11	9:2	8.25:2.75	0.031
S 17[c]×R 570	SS×R	80	20:60	40:40	20.000
Q 96×R 570	S×R	42	17:25	21:21	1.540
M 555/60×R 570	S×R	90	37:53	45:45	2.855
M 1030/71×R 570	HS×R	25	10:15	12.5:12.5	1.040
R 570×M 1030/71	R×HS	36	19:17	18:18	0.111
M 2077/78×M 1030/71	R×HS	68	47:21	34:34	9.956
M 937/77×M 1030/71	R×HS	13	5:8	6:6	0.750
M 695/69×M 1030/71	SS×HS	46	17:29	23:23	3.152
S 17×M 1030/71	SS×HS	26	0:26	0:26	0
Q 96×M 1030/71	S×HS	41	5:36	0:41	0.738
M 555/60×M 1030/71	S×HS	8	0:8	0:8	0

[a] Chi-square (χ^2)=3.84 at the 5% significance level
[b,c] Behave as resistant and susceptible parents, respectively

Table 4 Analysis of variance for control and parent clones for rust rating pooled over 3 seasons for 4×2 factorial set of crosses and pooled over 2 winter seasons for the complete set of 8×2 factorial crosses

Source of variation	Summer and winter ratings (4×2 set of crosses)		Winter ratings (8×2 set of crosses)	
	df	MS	*df*	MS
Replicates	1	0.01 ns	1	0.01 ns
Clones	13	180.92**	13	16.15**
Error (e_a)	13	1.38	13	0.67
Seasons	2	28.59**	1	0.09 ns
Clones×seasons	26	3.16**	13	0.90 ns
Error (e_b)	28	0.72	14	0.64
Within[a]	1066	0.21	851	0.21

** $P<0.01$; ns, not significant
[a] Estimates the environmental component of variance, σ^2_E

Table 5 Analysis of variance for progenies for rust rating pooled over 3 seasons for 4×2 factorial set of crosses and pooled over 2 winter seasons for the complete set of 8×2 factorial crosses

Source of variation	Summer and winter ratings[a]		Winter ratings[b]		EMS[c]
	df	MS	*df*	MS	
Replicate	1	7.83 ns	1	4.08 ns	
Female (F)	3	105.13**	7	57.03**	$\sigma^2_w+n\sigma^2_{e2}+ns\sigma^2_{e1}+nr\sigma^2_{mfs}+nrs\sigma^2_{mf}+nrsm\sigma^2_f$
Male (M)	1	138.00**	1	150.10**	$\sigma^2_w+n\sigma^2_{e2}+ns\sigma^2_{e1}+nr\sigma^2_{mfs}+nrs\sigma^2_{mf}+nrsf\sigma^2_m$
Female×male	3	39.30**	7	18.40**	$\sigma^2_w+n\sigma^2_{e2}+ns\sigma^2_{e1}+nr\sigma^2_{mfs}+nrs\sigma^2_{mf}$
Error (e_1)	7	3.26	14	1.72	$\sigma^2_w+n\sigma^2_{e2}+ns\sigma^2_{e1}$
Season (S)	2	23.14**	1	6.10*	
F×S	6	4.50*	7	1.24*	$\sigma^2_w+n\sigma^2_{e2}+nr\sigma^2_{mfs}+nrf\sigma^2_{ms}$
M×S	2	0.47 ns	1	4.61**	$\sigma^2_w+n\sigma^2_{e2}+nr\sigma^2_{mfs}+nrm\sigma^2_{fs}$
F×M×S	6	5.83*	7	1.18 ns	$\sigma^2_w+n\sigma^2_{e2}+nr\sigma^2_{mfs}$
Error (e_2)	16	1.63	14	0.37	$\sigma^2_w+n\sigma^2_{e2}$
Within-plot	1030	1.13	1237	1.63	σ^2_w

* $P<0.05$; ** $P<0.01$; ns, not significant
[a] n=22.5, r=2, s=3, f=4, m=2
[b] n=20.7, r=2, s=2, f=8, m=2
[c] Based on Comstock et al. (1992)

Genetic parameters derived from the progeny analysis from the individual season rating and from the combined seasonal ratings indicated that additive genetic effects accounted for 44–68% of the total genetic variation. Narrow-sense heritability (h^2_n) varied from 0.40 to 0.52 when derived from the progeny analysis and ranged from 0.39 to 0.64 when the estimates were derived from offspring-mid-parent regression analysis.

Broad-sense heritability (h^2_b) was observed to be very high (Table 6) for the individual season and for the combined ratings (0.75–0.90). These values indicate a high genetic component for rust reaction.

Table 6 Estimates of genetic variance components and heritability for rust derived from progeny analysis and offspring-mid-parent regressions (OP) for the individual and combined ratings

Rating season	σ^2_A	σ^2_D	σ^2_G	$\sigma^2_{A/}\sigma^2_G$	σ^2_E	σ^2_P	h^2_b (σ^2_G/σ^2_p)	h^2_n (σ^2_A/σ^2_p)	h^2_n (OP)
Summer January 94	0.603	0.278	0.880	0.685	0.286	1.166	0.75	0.52	0.51
Winter September 94	0.783	0.624	1.407	0.557	0.207	1.614	0.87	0.49	0.39
Winter June 95	0.836	0.460	1.296	0.645	0.276	1.572	0.82	0.46	0.64
Combined all dates (4×2 sets)	0.854	1.068	1.922	0.444	0.208	2.130	0.90	0.40	0.58
Combined winter (8×2 sets)	1.116	0.820	1.936	0.576	0.212	2.148	0.90	0.52	0.53

σ^2_A, σ^2_D, σ^2_G, σ^2_E, σ^2_P are, respectively, the additive, non-additive, total genetic, environmental, and phenotypic components of variance

Discussion

This study showed that resistance to rust has a strong genetic component. Controls and parents could consistently be classified across time as being resistant or susceptible. Slight changes in scores were confined to within the resistant and slightly susceptible groups only. The pattern of segregation for progenies resulting from selfing of the resistant parent ‘R 570’ confirmed the heterozygous nature of that parent. When these progenies were allocated into two groups of resistant : susceptible based on the complete absence (resistant) and presence of symptoms (susceptible), a 3:1 ratio was obtained which supports the presence of a major resistant gene segregating in a heterozygous way. These results confirm those of Daugrois et al. (1996) who attributed resistance to rust in the self progeny ‘R 570’ to a major gene with dominant effect that could be linked at 10 cM with a restriction fragment length polymorphism (RFLP) marker revealed by the probe *CDSR29-H5*. Crosses between parents of various susceptibility levels with ‘R 570’ and ‘M 1030/71’ tend to support these findings except for 1 cross which showed a major deviation. However, the presence of one gene with major effect does not explain the overall susceptibility levels within the crosses, which tend to suggest the existence of other genes with smaller effects acting in a quantitative way. When rust susceptibility was treated as a quantitative trait, the association with the marker in the self progeny of ‘R 570’ indicated the presence of a quantitative trait locus (QTL) with a very large effect (Daugrois et al. 1996). These authors detected, though not systematically, the presence of few QTLs with minor effects associated with rust susceptibility.

The high broad-sense heritability (0.75–0.90) agrees with the estimates of Comstock et al. (1992) and advocates that selection for rust would be highly effective early in the selection programme in asexual populations in the presence of disease pressure, while the combined analysis suggests that the trait is highly repeatable. Susceptible families and/or susceptible clones within families can be efficiently eliminated from segregating populations evaluated in rust-prone areas. Narrow-sense heritability derived from the progeny analysis was in the range 0.40 to 0.52 and was close to the estimates of Hogarth et al. (1993), suggesting that the level of susceptibility in the progeny can be predicted to some extent from the individual susceptibilities of the parents. Additive genetic effects accounted for 44–68% of the total genetic variation. The highly significant female×male interaction shows that deviation from expectancy based on the individual susceptibility of the parents is real but would not be of importance in R×R parents, which may cumulate the positive effects of some minor genes as suggested earlier.

Under Mauritian conditions, R and SS varieties are released for cultivation in rust-prone areas. This study showed that R×R and SS×R crosses produced 82–92% of progenies that would be resistant or slightly susceptible (score 1–3) to rust whereas S×R or HS×R crosses produced 64–74% progenies in the R and SS categories. When the resistant parent was used as the seed-bearing one, 3 R×HS crosses produced 80–94% progeny in the R and SS classes. This tends to confirm both the dominant nature of resistance as well as the possible maternal influence of the seed-bearing parent as observed in the reciprocal cross between the resistant parent, ‘R 570’, and the slightly susceptible one, ‘M 1030/71’. Emphasis will be laid on R×R, R×SS and SS×SS crosses in the Mauritian breeding programme. The limited availability of these categories of parents may necessitate the use of crosses between susceptible males and resistant females. Susceptible to highly susceptible progenies can be rejected safely from these categories of crosses early in the selection programme in the presence of disease pressure.

Acknowledgments We thank Dr. J.C. Autrey, Director, and Dr. C. Soopramanien, Deputy Director, for reviewing the paper and are grateful to Mrs. C. Ramnawaz and Mrs. S. Seeburrun for their assistance in computation. The experiments comply with the current laws of the Republic of Mauritius.

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