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A putative major gene for rust resistance linked with a RFLP marker in sugarcane cultivar 'R570'

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Abstract Inheritance of resistance to rust was investigated in the self progeny of the sugarcane cultivar 'R570' also used to build a RFLP genetic map. Resistance was evaluated through both field and controlled greenhouse trials. A clear-cut 3 (resistant) : 1 (susceptible) segregation indicative of a probable dominant resistant gene was observed. This is the first documented report of a monogenic inheritance for disease resistance in sugarcane. This gene was found linked at 10 cM with an RFLP marker revealed by probe *CDSR29*. Other minor factors involved in the resistance were also detected.

Key words Major gene · Polyploidy · *Puccinia melanocephala* · Sugarcane · RFLP · Rust

Introduction

The genome organisation of current sugarcane cultivars is very complex. They are highly polyploid and aneuploid clones derived from interspecific hybridisation between *S. officinarum* L. (2n = 80), the first domesticated species, and a wild relative, namely *S. spontaneum* L. (2n = 40–128). Their chromosome number is in the range of 100 to 130, with a probable 10% contribution from the wild species. The basic chromosome numbers of *S. officinarum* and *S. spontaneum* are most likely different, $x = 10$ and $x = 8$, respectively (Sreenivasan et al. 1987; D'Hont et al. 1995; D'Hont et al. 1996). This genetic complexity makes breeding for all traits including resistance to diseases a difficult task, as qualitative Mendelian trait segregation seems to be the exception. A few morphological characters have been found to be controlled by one or two genes. For ring color, a segregation reported in *S. officinarum* fitted a monogenic determinism in the F_2 but not in the backcross progenies (Raghavan and Govindaswamy 1956). For ligular process, segregation fitted a digenic inheritance in a *S. officinarum* L. × *S. spontaneum* L. cross (Batcha and Palanichamy 1978). For diseases, however, no major resistance gene has yet been documented. The lagging behind of Mendelian genetics in sugarcane is partly due to its genome complexity, especially the high ploidy level of clones, which hides possible existing mutations, and also to practical limitations such as a difficulty in efficiently controlling pollination during crosses.

Common rust of sugarcane is caused by the fungus *Puccinia melanocephala* Syd P. Syd. The disease occurs world-wide and can cause high sugar tonnage losses in susceptible varieties (Purdy et al. 1983; Taylor et al. 1986; Comstock et al. 1992). Rust resistance is generally considered to be quantitatively inherited trait with a high heritability and a strong additive genetic variance component (Tai et al. 1981; Hogarth et al. 1983, 1993). The existence of rust pathogenic races has been reported in India, where the disease is probably endemic (Srinivasan and Muthaiyan 1965), but also in Florida where the disease was first detected in 1979 (Dean and Purdy 1984). In Australia, where rust was first reported in 1978, no pathogenic races have been detected (Taylor 1992).

Molecular markers provide a powerful tool to unravel the complex genome of sugarcane and enhance the determination of Mendelian bases for trait inheritance. In recent years, the use of molecular markers in this crop has increased rapidly. Genetic mapping was first performed on the wild species *S. spontaneum* (A1-Janabi et al. 1993; Da Silva et al. 1995). It was also undertaken on cultivated materials (D'Hont et al. 1994). Recently, a large-scale restriction fragment length polymorphism (RFLP) map was constructed based on a self progeny of cv 'R570' (Grivet et al. 1996).

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In the work reported here, we studied rust resistance inheritance in the self progeny of cv 'R570', through both field trials with natural infection and controlled greenhouse trials with artificial inoculation. We identified a discrete 3:1 segregation, which can be attributed to a major dominant resistance gene. This gene was shown to be linked, at 10 cM, with a simple molecular marker revealed by an RFLP probe.

Materials and methods

Plant material

Resistance was evaluated on the self progeny (P1) of the rust-resistant cultivar 'R570' that was used to build an RFLP genetic map (Grivet et al. 1996). 'R570', developed by the Centre d'Essai de Recherche et de Formation (CERF), is commercially very successful in Réunion and Mauritius. Both of the parents of 'R570', 'R445' and 'H328560', are resistant to the rust disease. Marker segregations enabled us to check that progeny individuals were indeed derived from the selfing of 'R570', and this then allowed us to discard a few outstanding individuals. Resistance to rust was evaluated in two different field trials and under controlled conditions in the greenhouse. Due to seed (cuttings) availability, the various trials involved slightly different materials. A second sample of 83 individuals of the self progeny of 'R570', (P2), was used to test the results obtained on the P1 population and improve the accuracy of the conclusions.

Rust susceptibility in the field

Population P1 was evaluated in two field trials planted at the CIRAD breeding station of La Mare (Réunion Island) where the rust pressure is present naturally (Peros and Lombard 1986). The first trial (trial 1) evaluated the susceptibility of 64 clones. It followed a completely randomised layout where each clone was represented by a single plot. Each plot consisted of one 3-m-long row of 6 plants. Rust susceptibility was evaluated on plant cane (as opposed to ratoon crop) in 1992.

The second trial (trial 2) evaluated the susceptibility of 58 clones, with all but 1 already included in trial 1. The clones were planted in a partially (half) balanced lattice layout with three replications. Field plots consisted of two rows 2 m in length with 1.5 m between the rows. Planting density was four three-eye cuttings per meter. Rust susceptibility was evaluated on plant cane in 1993 and first ratoon in 1994.

The P2 population was evaluated for rust susceptibility in 1995, in a randomised complete block layout with three replications. The field plots consisted of 2.5-m long row, with a planting density of four three-eye cuttings per meter.

For all of the trials, the susceptibility level of each plot was graded on a 1 to 9 scale (Tai et al. 1981), 1 being the most resistant (no pustule) and 9 the most susceptible (high pustule density and tissue death).

Rust susceptibility in controlled conditions

Sixty-three clones of P1 were evaluated under controlled conditions through greenhouse trials. The size of the greenhouse limited us to the evaluation of a maximum of 30 clones per trial. We estimated first the repeatability of the test by evaluating the same 20 clones in two different trials. We then evaluated another 43 clones in two other trials. In each trial, 4 clones of known susceptibility (from the most to the least susceptible: 'H49–5', 'B34104', 'R469', 'R570') were used as a control.

The protocol of rust susceptibility evaluation has already been described by Peros (1989). For each clone, 15 single-bud cuttings were planted in 0.8 l soil/pozzolana (v/v) pots. After 12 weeks, the last developed leaf of each of the five largest plants was inoculated by spraying uredospores under a settling tower. The inoculum was taken from field-infected plants and propagated in a greenhouse room on susceptible cultivar 'B4362'. Inoculum was calibrated to give approximately 60 germinated spores per square centimeter on agar medium. Inoculated plants were kept for 16 h at 100% relative humidity and then transferred to a greenhouse room. Disease expression was scored 15 days later by measuring uredinia density on 5 cm^2 per inoculated leaf. For each clone, the susceptibility measure was the mean of density obtained on the five leaves.

Mapping data

The construction of the genetic map on P1 has been reported elsewhere (Grivet et al. 1996). Mapping was performed by using 1 isozyme and 128 RFLP probes. This made 505 simplex markers available for the segregation analysis. Among those, 408 were linked into 96 cosegregation groups. These could be assembled into ten linkage groups on the basis of probes in common. Ninety-six markers remained unlinked. Eight probes had no marker involved in any cosegregation group. By discarding markers with more than 10% missing data, we were able to use 439 markers to search for associations with rust susceptibility. Probe *CDSR29*, which is associated with the main rust effect in P1, was used to reveal a RFLP on P2 according to the same RFLP protocol as Grivet et al. (1996).

Data analysis

The rust susceptibility scores obtained from the greenhouse trial were transformed by square root in order to stabilise the variance. Quantitative data analyses were performed using the SAS computer program (SAS Institute 1988). Normality of the different susceptibility measures was evaluated through the test of Shapiro and Wilk (1965) using the procedure UNIVARIATE. In field trial 2, in which each clone was repeated three times in a partially balanced lattice layout, the least square means were recovered with the GLM procedure. The VARCOMP procedure was used to estimate the genetic variance component σ_g^2 from which the broad-sense heritability on a plot basis, h_1^2 and on an entry mean basis, h_2^2, were deduced:

$$h_1^2 = \frac{\sigma_g^2}{\sigma_g^2 + \sigma_e^2},$$

$$h_2^2 = \frac{\sigma_g^2}{\sigma_g^2 + \frac{\sigma_e^2}{n}},$$

where n is the mean number of replications for each clone, and σ_e^2 the error variance component. The same method was used to estimate the heritabilities of the greenhouse test on the 20 clones evaluated in two different trials, which were considered as two blocks.

Significant associations between each of the 439 markers and the different rust susceptibility measures were detected using the non-parametric test of Kruskal-Wallis available on the computer program MAPQTL 2.2 under development at CPRO-DLO, the Netherlands (J. W. Van Ooijen, personal communication). This test is particularly adapted to treat ordinal data that do not follow a normal distribution (Van Ooijen et al. 1992).

Detection and estimation of linkage between Mendelian factors were performed according to Mather (1957).

Results

Rust susceptibility in the field

Rust susceptibility in population P1 was evaluated on three crops under natural inoculation, two on plant cane (trial 1, 1992· trial 2, 1993) and one on first ratoon (trial 2, 1994). This gave three different evaluations of rust sus-

ceptibility in different ecological (year and trial) and physiological (plant cane and ratoon) environments.

The distributions displayed by the three rust susceptibility measures were all distinct from a normal distribution with the test of Shapiro and Wilk at the 5% level, and tended to be "L shaped" with a majority of resistant clones and a minority of dispersed sensitive clones. All three measurements were highly correlated (Table 1). The heritability of the rust susceptibility was evaluated in the second trial and was very high on an entry mean basis in both plant cane (0.94) and first ratoon (0.97), thereby showing a high genetic variance component and good control of the environmental variation in the trial.

Rust susceptibility under controlled conditions

A set of 20 clones of population P1 were evaluated twice in two different experiments, thereby enabling us to evaluate the heritability of the greenhouse test, which was 0.97 on a plot basis. On the basis of this high value a high level of confidence could be attributed to the estimation of rust susceptibility in a single greenhouse test. We thus evaluated another 43 clones, each clone being repeated once. Variability in the self progeny appeared to be high. While the majority of the clones were as resistant as 'R570', several clones were more susceptible than 'R469', and 1 was as susceptible as 'H49.5'. As for the field trials, the distribution of the greenhouse measurement was not normal and tended to be "L shaped". The greenhouse test was highly correlated with the three field susceptibility measures (Table 1).

Identification of a discrete 3:1 distribution in the progeny

Although rust susceptibility may appear as a quantitatively distributed trait when the different measures are considered independently, a clear-cut segregation was revealed when the two measures were considered simultaneously (Fig. 1). This allowed individual progeny to be classified into two populations, one homogeneous with highly resistant clones and the other heterogeneous, with clones of various susceptibility levels. The clones of this second population will subsequently be designated as "susceptible" for simplification. A discrepancy appeared for 1 clone between field and greenhouse measures. This clone was resistant in the field and very susceptible in the greenhouse. All other clones presented a proportionate response in the greenhouse and the field trials (Fig. 1).

For 63 of the 65 P1 individuals we had a value for at least two of the three measures of susceptibility in the field. It was thus possible to score each of them as "resistant" or "susceptible" on the basis of two-dimensional plots: 45 were quoted "resistant" and 18 "susceptible". The ratio of resistant versus susceptible clones was not different from a 3:1 ratio ($\chi^2 = 0.48$), which is the expected segregation ratio for a simplex dominant resistance gene.

Table 1 Heritability and correlations between the different measures of rust in the field and in controlled conditions on the selfed progeny of R570

Trial	N[a]	h_1^2	h_2^2	Pearson correlation coefficients[b]			
				Trial 1 pc	Trial 2 pc	Trial 2 r	gh trial
Trial 1 pc	64	–	–	–	0.89	0.90	0.79
Trial 2 pc	58	0.86	0.94	–	–	0.95	0.75
Trial 2 r	58	0.93	0.97	–	–	–	0.75
gh trial	63	0.97	0.99	–	–	–	–

[a] N, number of progenies evaluated; h_1^2, heritability on a plot basis; h_2^2, heritability on an entry mean basis; pc, plant cane; r, ratoon; gh, greenhouse

[b] All correlations are significant at $P = 10^{-4}$

Fig. 1A, B Representation of rust susceptibility measures on two-dimensional plots in population P1. **A** Rust susceptibility observed in field trial 2 on plant cane (*pc*) versus ratoon (r). **B** Rust susceptibility observed on ratoon of field trial 2 versus greenhouse test. Measures in the field were graded visually on a 1 to 9 scale; measures in the greenhouse are the square root of the mean uredinia density observed on the leaves. One individual showing an inconsistant response between the two measures in plot **B** is represented by a ◇

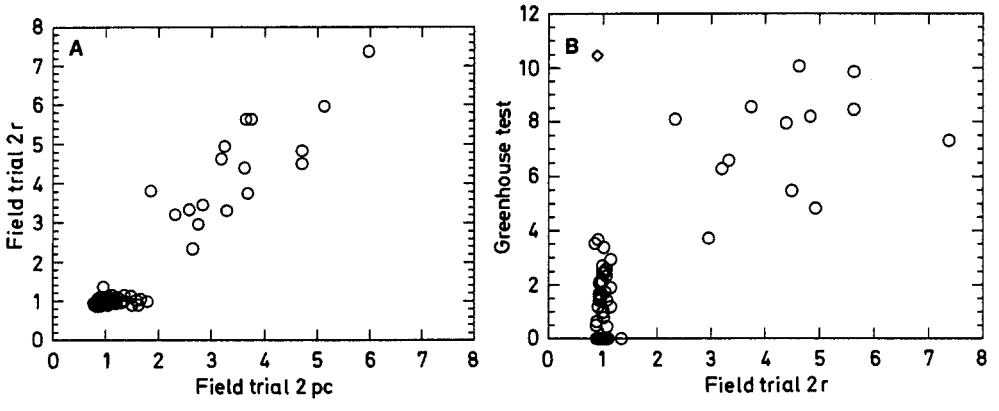


Table 2 QTL detected with Kruskal-Wallis test at the 5% level for the different measurements of rust susceptibility (*pc* plant cane, *r* ratoon, *gh* greenhouse)

Probe[a]	Cosegregation group[a]	D	Kruskal-Wallis test statistic and level of significance			
			Trial 1 pc[c]	Trial 2 pc[c]	Trial 2 r[c]	gh trial[c]
SG12	IV-1	8	7.9*	–	–	–
UMC132	IV-1		7.9*	–	–	–
Adh1	IV-5		10.5*	–	–	–
Adh1	VIII-1	5	–	8.3*	–	–
UMC15	VIII-1	7	–	9.0*	–	–
BNL3.04	VIII-1		–	8.6*	–	–
UMC6	VIII-2	0	9.6*	–	–	10.8**
UMC44	VIII-2	0	9.6*	–	–	10.8**
CDSR125	VIII-2	6	9.6*	–	–	10.8**
SSCIR86	VIII-2		–	–	–	10.8**
UMC137	X-1		8.1*	–	–	–
CDSR29	–		28.4****	27.8****	18.9***	15.5***

* $P \leq 0.0005$, ** $P \leq 0.0001$, *** $P \leq 10^{-4}$, **** $P \leq 10^{-5}$
[a] Grivet et al. (1996)
[b] Distance (in centi Morgans) between linked markers
[c] pc: plant cane; r: ratoon, gh: greenhouse

RFLPs associated with rust resistance loci

In a first round of analysis, the four measures of rust susceptibility were considered as quantitative traits, and quantitative trait loci (QTLs) were searched for using the test of Kruskal-Wallis. With respect to the high number of tests performed, a threshold of $P = 0.005$ was retained to determine significant associations. For all four measures, a highly significant association ($P < 0.0001$) was found with marker *CDSR29-H5* (fifth largest fragment in the *Hin*dIII profile), which was revealed by probe *CDSR29* (Table 2). This marker as well as the other four simplex markers revealed by probe *CDSR29* were not involved in any cosegregation group, thus the QTL could not be positioned on the composite map of 'R570' (Grivet et al. 1996). Five other putative QTLs of smaller effect were found associated with one or two specific measures of rust susceptibility (Table 2). Considering the high number of marker-trait associations investigated, they may include artefacts. However, it is noteworthy that one of those minor QTLs repeatedly arose in two of the four trials. This QTL was located along cosegregation group VIII-2.

Table 3 Contingency table showing linkage between the putative resistance major gene and marker *CDSR29-H5* of probe *CDSR29* in the selfed progeny of 'R570' on population P1, population P2 and on the pooled population

Rust resistance major gene	Marker *CDSR29-H5*			
	Present	Absent	Missing data	Total
Present	96[a] (40[b], 56[c])	4 (3, 1)	2 (2, 0)	102 (45, 57)
Absent	9 (2, 7)	31 (13, 18)	4 (3, 1)	44 (18, 26)
Total	105 (42, 63)	35 (16, 19)	6 (5, 1)	146 (63, 83)

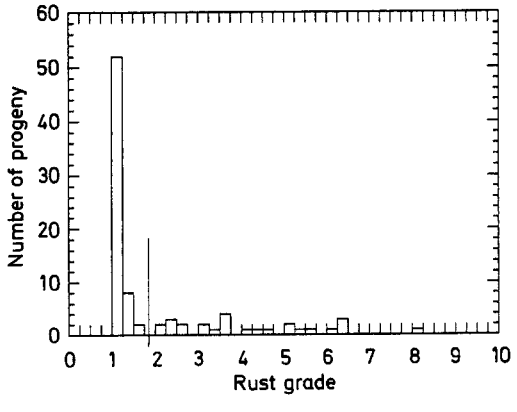
[a] Pooled data on populations P1 and P2
[b] Contribution of population P1
[c] Contribution of population P2

We then investigated linkage of the putative major gene, graded in a presence versus absence fashion, with the 439 markers of the map and found a strong linkage with marker *CDSR29-H5*. The χ^2_L test, which measures linkage independently of segregation distortions with 1 *df* (Mather 1957), was $\chi^2_L = 38.6$ ($P < 0.00001$). The distance between the two Mendelian factors was 8.8 cM with the Haldane mapping function.

Confirmation of the linkage data on another progeny sample

The P2 progeny, consisting of 83 clones, was observed in the field in 1995, and the clones were scored on the 1–9 scale for rust susceptibility. Keeping in mind the possible existence of a major resistance gene, we considered the 63 clones with a score lower than 2 to be highly resistant, while the 26 clones with a grade higher than or equal to 2 were considered to be susceptible (Fig. 2). The ratio of resistant versus susceptible clones was not differ-

Fig. 2 Distribution of rust susceptibility scores in population P2. The *vertical bar* represents the threshold retained to distinguish between individuals bearing the resistance gene and those that do not have it



ent from 3:1 ($\chi^2 = 1.77$). The scoring of marker *CDSR29-H5* on the same individuals permitted us to confirm the linkage with the putative rust major gene ($\chi_2 = 51.0$, distance of 11.1 cM). The χ^2 test measuring discrepancy with 1 *df* between the recombination rate obtained with the two bodies of data (Mather 1957), P1 and P2, was not significant ($\chi = 0.054$). Rust and marker data of P1 and P2 were then pooled, giving a $\chi_L^2 = 89.6$ and a genetic distance of 9.5cM between the putative major gene and marker *CDSR29-H5* (Table 3).

Discussion

The present study revealed the presence of a probable simplex major gene conferring resistance to rust disease in sugarcane cv 'R570' and located this gene at 10 cM from an RFLP marker. The existence of a major factor in this variety had already been suggested in a preliminary report of experiments conducted in Mauritius (Saumtally et al. 1994). Our results established its existence with a high level of confidence on the basis of the following:

- characterisation of progeny individuals with RFLP markers identified valid self-progeny clones of 'R570'. This is important, for the lack of an efficient control of pollination is a common source of disturbance in genetic studies on sugarcane;
- the progenies were evaluated in several experiments, and in all of these high heritabilities and high mutual correlations were apparent. Scoring in the field and in the greenhouse varied for only 1 progeny clone, which expressed rust susceptibility only in the greenhouse trial. This discrepancy will require further investigation. It may be due to genotype × environment interaction, for rust susceptibility is largely influenced by environmental factors such as soil (Anderson et al. 1990), climate (Peros et al. 1993) and plant age (Albuquerque 1958). A mislabelling can, however, not be excluded;
- the segregation ratio fitted very well with the expected 3:1 ratio for a dominant simplex gene;
- linkage with a DNA marker was established with a high level of confidence ($\chi_L^2 = 89.6$). This permitted us to associate the resistance versus susceptibility of progeny clones with the presence versus absence of a specific chromosomal segment. Even when rust susceptibility was treated as a quantitative trait, the association with the marker was very highly significant for all measures, indicating the presence of a QTL with a very large effect.

Beyond the segregation of the major gene, a large rust susceptibility level variation still existed in the susceptible progeny class. The high heritability on a plot basis suggests that the origin of this variation is at least partially genetically determined. It could be explained by the segregation of QTLs with small effects, such as the one borne by cosegregation group VIII-2. This marginal polygenic variation may explain why a few progeny clones were difficult to classify as resistant or susceptible. A significant genotype × year interaction was noticed in trial 2 (data not shown), indicating that clone ranking for susceptibility can be affected to some extent by the environment (physiological or ecological since year effect is superimposed with ratoon effect in our trial). This could explain why the clear-cut segregation for rust susceptibility appeared more clearly when two measurements were taken simultaneously. When clones do not bear the major gene but have favorable alleles at QTL with small effects, they may appear as resistant in one given trial, whereas the susceptibility might be revealed when several observations are available.

Robinson (1976) considered that vertical pathosystems do not exist in sugarcane, and he stressed that oligogenically inherited resistance has never been recorded in this crop. Since pathogenic races may exist in *P. melanocephala*, the identification of a major gene raises the question of whether it corresponds to a gene-for-gene relationship. The genetics of rust resistance has been very well documented in maize and to a lesser extent in sorghum, two diploid crops belonging to the Andropogoneae tribe, like sugarcane (Hooker 1985). Main specific rust pathogens of maize and sorghum belong to the *Puccinia* genus (respectively, *P. sorghi* and *P. purpurea*). In both species, a general polygenic resistance and a monogenic pathogen race-specific resistance have been characterised. The analogy with our results is noteworthy. Nevertheless, the Flor (1971) gene-for-gene concept has been developed on diploid crop, and a transposition to sugarcane may not be appropriate given the high ploidy level of the cultivars. Indeed, around ten copies of the resistance locus are present in a given clone, each one possibly bearing a race-specific allele. Extending the genetic map of 'R570' will reveal the position of the major gene on a linkage group and thus enable a comparison with the *Rp* maize resistance gene positions on the maize map. If the positions concur, it will be indicative of a probable homology of the gene detected here in sugarcane with the corresponding *Rp* gene of maize.

The development of a polymerase chain reaction (PCR)-based marker closely linked to the major resistance gene could be helpful in screening for rust resistance in large progeny samples although rust resistance is relatively easy to select for on the basis of the phenotype, thanks to its high heritability.

Beyond the specific case of the rust disease, our result is important since it is the first report of a monogenic determinism of disease resistance in the complex polyploid that sugarcane is. Should this exist for the main sugarcane pathogenes for which field trials are usually very laborius, molecular markers could be very useful in assisting breeding.

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