Theor Appl Genet (1997) 95:20–32

N. Nitta · M. L. Farman · S. A. Leong

Genome organization of *Magnaporthe grisea*: integration of genetic maps, clustering of transposable elements and identification of genome duplications and rearrangements

Received: 17 February 1997 / Accepted: 21 February 1997

Abstract A high-density genetic map of the rice blast fungus *Magnaporthe grisea* (Guy11 × 2539) was constructed by adding 87 cosmid-derived RFLP markers to previously generated maps. The new map consists of 203 markers representing 132 independently segregating loci and spans approximately 900 cM with an average resolution of 4.5 cM. Mapping of 33 cosmid probes from the genetic map generated by Sweigard *et al.* has allowed the integration of two *M. grisea* maps. The integrated map showed that the linear order of markers along all seven chromosomes in both maps is in good agreement. Thirty of eighty seven markers were derived from cosmid clones that contained the retrotransposon MAGGY (*M. grisea* gypsy element). Mapping of single-copy DNA sequences associated with the MAGGY cosmids indicated that MAGGY elements are scattered throughout the fungal genome. In eight cases, the probes associated with MAGGY elements showed abnormal segregation patterns. This suggests that MAGGY may be involved in genomic rearrangements. Two RFLP probes linked to MAGGY elements, and another flanking other repetitive DNA elements, identified sequences that were duplicated in the Guy11 genome. Most of the MAGGY cosmids also contained other classes of repetitive DNA suggesting that repetitive DNA sequences tend to cluster in the *M. grisea* genome.

Key words Rice blast · Linkage map · *Pyricularia grisea* · RFLP · MAGGY · Molecular map

Communicated by F. Mechelke

N. Nitta[1] · M. L. Farman[2] · S. A. Leong (✉)
Department of Plant Pathology, University of Wisconsin, 1630 Linden Drive, Madison, WI 53706, USA

S. A. Leong
USDA-ARS Plant Disease Resistance Research Unit, University of Wisconsin, 1630 Linden Drive, Madison, WI 53706, USA

Present addresses:
[1] Japan Tobacco Inc., Plant Breeding and Genetics Research Laboratory, 700 Higashibara, Toyoda, Iwata, Shizuoka 438, Japan
[2] Department of Plant Pathology, S-305 Agricultural Science Building-North, University of Kentucky, Lexington, KY 40546, USA

Introduction

The filamentous ascomycetous fungus, *Magnaporthe grisea* (Hebert) Barr [(*Pyricularia grisea*, sacc) *Pyricularia oryzae*, cavara] is a causative agent of rice blast disease, one of the most devastating diseases of rice (*Oryza sativa*). Because of its economic importance, considerable efforts have been made to understand the genetics and molecular biology of this fungus. Three different genetic maps for this organism have been reported (Romao and Hamer 1992; Skinner et al. 1993; Sweigard et al. 1993). One map, containing 98 RFLP markers, two isoenzymes and the mating-type locus (Skinner et al. 1990; Budde et al. 1993; Skinner et al. 1993), was later modified to include *avrCO39* (*AVR1-CO39*), a locus controlling cultivar specificity to rice cultivar CO39 (Smith and Leong 1994), and 14 telomere loci (Farman and Leong 1995). A second map was constructed utilizing the repetitive DNA sequence MGR586 as a genetic marker (Romao and Hamer 1992), while the third map was developed using cloned genes, cosmid clones, various repeated DNAs and a telomere-specific repeat, as RFLP probes (Sweigard et al. 1993). In addition *AVR2-YAMO* and *AVR1-TSUY*, two genes controlling cultivar specificity toward rice, and *PWL2*, a gene conferring host specificity to weeping lovegrass, were mapped.

In this paper we describe the addition of 87 new markers to the map of Farman and Leong (1995) and the integration of this map with that of Sweigard et al. (1993). The alignment of the two molecular maps serves to significantly increase the number of phenotypic and

molecular markers that are available to fungal researchers. In this regard it is noteworthy that Zhu et al. (1996) were rapidly able to map a gene involved in appressorium development and identify four markers that co-segregated fully with the *app*$^-$ phenotype and several other closely linked markers. This demonstrates the potential usefulness of this map for mapping and cloning additional characters related to pathogenicity.

Materials and methods

Fungal isolates

M. grisea isolates Guy11 (*MAT1-2*) and 2539 (*MAT1-1*) and their 61 random ascospore progeny were used to determine the segregation of RFLPs as described by Skinner et al. (1993).

Genomic DNA isolation

Mycelia from oatmeal plate cultures of *M. grisea* were used to inoculate 50 ml of liquid complete medium (CM) in 250-ml Erlenmeyer flasks. The flasks were incubated at 25–30°C with shaking at 200 rpm using an orbital shaker. After 2–3 days, the cultures were homogenized using a sterile Waring microblender and the sheared mycelium was shaken for 1 additional day in 50 ml of fresh medium. Mycelia were harvested when their density reached its maximum but before dark pigments were produced.

DNA extraction was by the CTAB method (Manicom et al. 1987) with modification. Lyophilized mycelium (0.2 g) was ground with a mortar and pestle in the presence of sand. The mycelial powder was mixed with 3 ml of hot (65°C) lysis buffer (0.5 M NaCl; 10 mM Tris-HCl, pH 7.5; 10 mM EDTA; 1% SDS) and 0.3 ml of 10% CTAB in 0.7 M NaCl to form a slurry, and then 3.0 ml of phenol/chloroform/isoamylalcohol (25 : 24 : 1, vol) was added. The mixture was incubated at 65°C for 15–30 min and centrifuged at 11500 g at 4°C for 15 min. The supernatant was treated with 10 μg/ml of RNase A followed by extraction with an equal volume of chloroform. To the supernatant, an 0.5 vol of 7.5 M ammonium acetate was added and the resultant solution was left on ice for 1 h or kept at 4°C overnight to precipitate protein, which was removed by centrifugation as above. DNA was precipitated by the addition of 0.54 vol of isopropanol, and the resulting pellet was washed with 70% ethanol, then dried and dissolved in 2.0 ml of TE. Polysaccharides contaminating the DNA sample were removed by ethanol precipitation according to the method of Michaels et al. (1994). The DNA thus obtained was quantified using a TKO 100 Fluorometer (Hoefer Scientific Instruments, San Francisco, Calif.). The DNA concentration was adjusted to 250 ng/μl in TE and kept at −80°C until use.

Southern-hybridization analysis

Restriction endonuclease-digested DNA was electrophoresed in 0.7% SeaKem LE agarose gels (1–1.5 μg/lane) in 0.5 TE (Maniatis et al., 1982). The electrophoresed DNA was transferred to a nylon membrane (Schleicher and Schuell, Keene, N.H.) by the method of Southern (1975). Plasmid or cosmid DNA was recovered from *Escherichia coli* using the alkali mini-prep method (Maniatis et al. 1982). Whole recombinant plasmids and cosmids were radiolabeled by nick translation (Maniatis et al. 1982). Cosmids containing repetitive DNA were digested by restriction endonuclease and single-copy fragments were isolated using 0.7% SeaPlaque gel or Gene Clean and radiolabeled by the random primer labeling technique. Hybridization conditions and removal of probe DNA were as described by Skinner et al. (1993).

RFLP markers

A cosmid library of genomic DNA from strain 2539 cloned in pMLF1 (Leong et al. 1994) was assayed by colony hybridization with MAGGY internal regions as probes (Farman et al. 1997b), and 57 MAGGY-hybridizing clones were identified in approximately 3500 cosmids screened. The cosmid DNAs were purified and used as a template to sequence 5′ and/or 3′ flanking regions of MAGGY (Farman et al. 1997). Based on the sequence of those flanking regions and restriction-enzyme-fragment analysis, the MAGGY cosmids were classified into 29 subgroups. Additional cosmid clones not hybridizing to MAGGY were randomly chosen and mapped as well.

Thirty three cosmid clones representing RFLP markers in the map of Sweigard et al. (1993) were provided by Dr. B. Valent (Dupont Co.). In order to identify repeated and single-copy DNA-containing fragments in the cosmids, cosmid DNAs were digested with restriction enzymes, electrophoresed in agarose and analyzed sequentially by Southern hybridization with total genomic DNA of strain 2539 and isolate Guy11 as probes. Repetitive DNA-containing fragments, which hybridized at high efficiency according to copy number, were detected by autoradiography after only a short exposure time (18 h). Single-copy bands were fractionated using low-melting agarose and employed as probes. When repetitive DNA was absent, the entire cosmid was labeled by nick translation and served as a probe. In order to identify enzymes that yielded informative DNA polymorphorisms, probes were used to survey blots of genomic DNA of strains Guy 11 and 2539 digested with various restriction enzymes.

Analysis of linkage

Ordered data showing the inheritance patterns of the 87 new markers within the progeny are presented in Appendix 1. Segregation data were analyzed using MAPMAKER Macintosh V2.0 (E. I. duPont de Nemours and Co.). Parameters for map construction were a minimum LOD (log of the odds) of 4.0 and a maximum recombination fraction of 0.25. The Kosambi mapping function was employed to compute recombination distances in centimorgans (cM). Linkage of markers separated by a recombination fraction greater than 0.2 was validated by chi square analysis.

Results and discussion

Genetic map

As shown in Fig. 1 (see also Table 1), mapping of the additional 87 RFLP markers (indicated in bold in Appendix 1) resulted in the generation of seven contiguous linkage groups representing the seven known chromosomes of *M. grisea*. Only telomere 6 remained unlinked. The distance between the terminal RFLP marker CH5-176H and telomere 6 was approximated as $\geqslant$40 cM by Farman and Leong (1995). Therefore the current genetic map spans approximately 860 cM. Thus, the estimated size of the mapped genome of *M. grisea* is about 900 cM. This value is in good agreement with the map sizes of 802 cM and 840 cM reported by Romao and Hamer (1992) and Sweigard et al. (1993), respectively.

Markers in the region between CH5-176H and telomere 6 are also absent in the map of Sweigard et al. (1993). The distance between marker CH5-176H and telomere 6 was determined to be approximately 580 kb

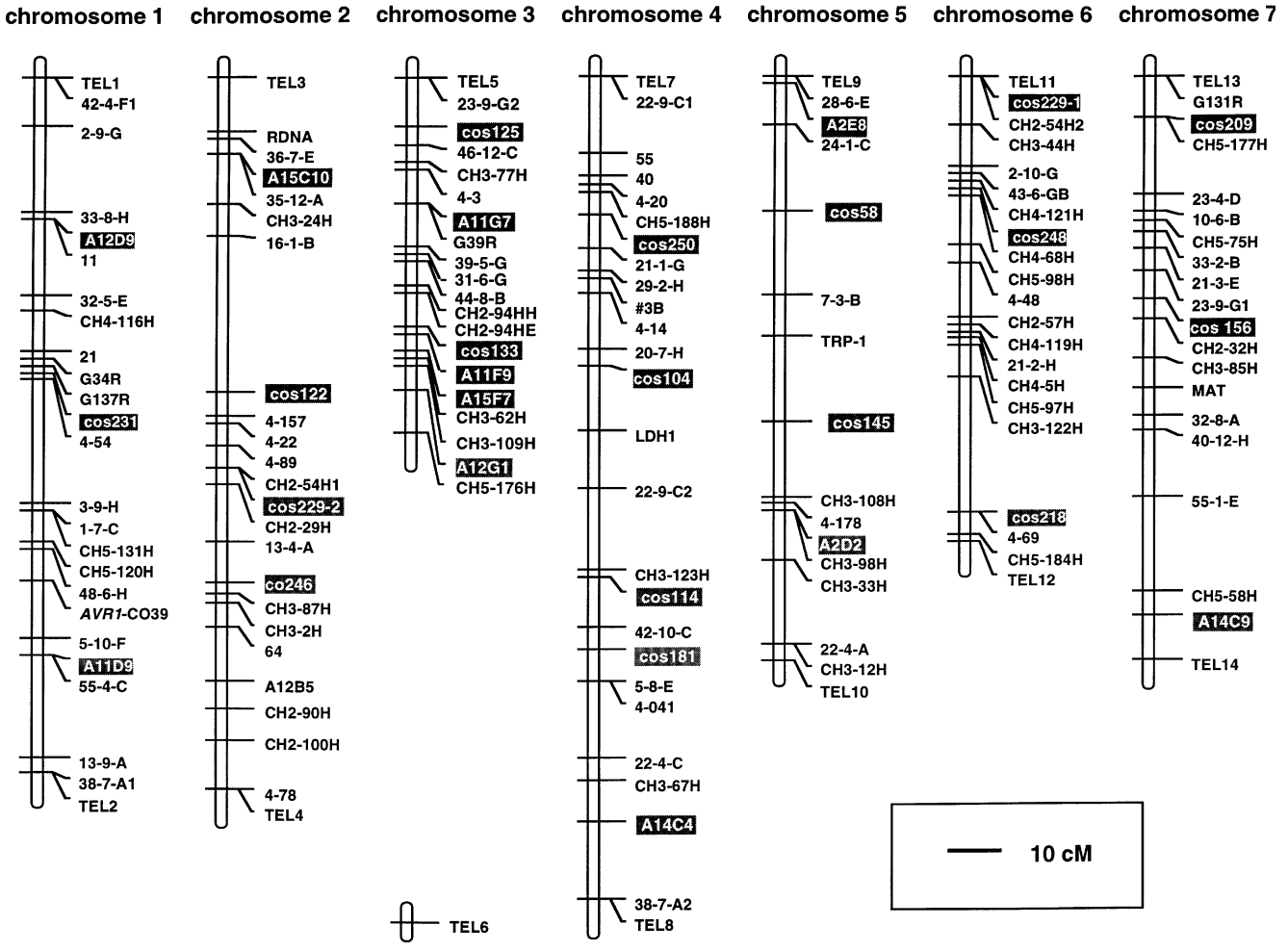


Fig. 1 Genetic map of a cross between *M. grisea* isolates Guy 11 and 2539. Only one marker representing each independently segregating locus is shown. Additional co-segregating markers are listed in Table 1. Markers derived from the map of Sweigard et al. (1996) are in *shaded boxes*

in the Guy11 genome and 530 kb in that of 2539 (Farman and Leong 1995). This region of chromosome 3 is clearly subject to an unusually high level of recombination. As a comparison, marker 11 and telomere 1 are separated by 1.8 Mb and are genetically linked (Farman and Leong 1995). This finding, along with the lack of RFLP markers in the region, may indicate that this region is highly homologous in the parental genomes.

While constructing the original map, distorted segregation (approximately 2:1) was observed for one marker, CH5-58H (Skinner et al. 1993). Mapping of telomeres identified a similar bias at telomeres 9 and 14 (Farman and Leong 1995). Two-dimensional analysis of telomeric restriction fragments suggested that the bias was caused by skewed inheritance rather than by the segregation of two loosely linked loci (Farman and Leong 1995). In the present study, this was confirmed by the identification of linked markers sharing the same bias and by the ability to place these markers on the map without having to infer double crossovers between them and existing linkage groups. In the case of markers near telomere 9, the distortion was progressively resolved towards the internal markers with marker cos58 showing no bias at a level of significance $P = 0.05$ (Appendix 1). Similarly, the three markers at the tip of chromosome 7 showed statistically significant distortion, which also affected linked markers extending 80 cM in from the telomere. Neither region showed segregation bias in the cross used to construct the Sweigard map (J. Sweigard, personal communication).

Placement of RFLPs identified by cosmid markers provided by Dr. B. Valent on the genetic map constructed in our laboratory enabled complete integration of the two maps (Skinner et al. 1993; Sweigard et al. 1993). The integrated map, shown in Fig. 2, potentially contains over 200 independently segregating loci, with an average resolution of 4.5 cM. The identification of markers that map close to a target gene will now enable cross-referencing between maps, possibly leading to the discovery of other closely linked markers. In this manner, a region containing a gene involved in appres-

Table 1 Additional markers mapping to locations represented on the *M. grisea* genetic map (Fig. 1)

CH#[a]	Marker indicated on map	Co-segregating markers			
1	CH4-116H	40-12-H			
	21	3-5-E			
	G34R	4-145			
	G137R	LDH3			
	cos23	50			
	CH5-131H	1-7-C			
	CH5-120H	1.2H			
2	4-22	4-10	52		
	CH2-54H1	CH4-112H	CH4-137H		
	13-4-A	43-6-GA			
	CH3-2H	CH3-87H			
3	G39R	CH3-125H	7-12H	37-2-H	
	cos133	CH2-59H	CH5-153H	CH3-73H	A1A2
	A11F9	A14B9	A14D5	A16C12	
	CH3-62H	CH5-196H	CH5-67H	37-10-G	
	A12G1	18	CH3-91H		
4	TEL7	4-181	4-194	42-4-F3	
	cos250	4-183	4-183	G121R	
5	TEL9	22-2-D			
	CH3-98H	A2D2			
6	TEL11	47-12-G			
	43-6-GB	CH4-173H			
	CH4-121H	CH5-191H			
	cos248	CH3-132H	CH4-131H	CH4-133H	CH5-185H
	CH4-68H	CH5-181H			
	CH2-57H	CH3-113H	CH4-60H	CH4-161H	CH5-167H
	CH4-5H	CH5-61H	37-11-F		
7	21-3-E	39	4-146		
	CH3-85H	CH4-537H			

[a] Chromosome number

sorium development was using a map-based approach within six months (R. Dean, personal communication).

A large reciprocal translocation had previously been identified between the parental strains 4224-7-8 and 6043 used in the Sweigard cross (1993). In the present study, the chromosomal associations of markers mapping on each translocated arm indicated that it was the 4224-7-8 parent that had suffered the translocation event (Fig. 3). This was not surprising as 6043 is an F_1 progeny of Guy11 and 2539, whose genomes show near perfect synteny.

A group of markers that co-segregated in the map constructed by Sweigard et al. (1993) was divided by a single crossover which occurred in one of the Guy11 $\times$ 2539 progeny. The mapping resolution was, therefore, slightly improved in this apparently recombination deficient region of chromosome #2.

Skinner et al. (1993) reported that marker CH2-54H was located on chromosome 2 in 2539 (designated as marker CH2-54H2) but was tightly linked to a telomere of Guy11 chromosome 5 (designated as marker CH2-54H1). In this study, cos229 showed two polymorphic patterns; one was a strong hybridization signal that co-segregated with CH2-54H2 and the other yielded a faint signal mapping at CH2-54H1 (data not shown). This result indicates that cos229 most likely overlaps with a breakpoint of the translocated sequences contaning the CH2-54H probe.

One marker, 42-4-F, only hybridized strongly with the 2539 genome. As this is a laboratory strain derived from crosses between rice and grass pathogens, this marker appears to contain grass pathogen-specific DNA sequences.

Distribution of the retrotransposon MAGGY

The copy number of MAGGY in 2539 was determined to be approximately 42 by counting bands in a Southern blot (data not shown). This figure is lower than that ($\geqslant$ 50) found in most rice-infecting strains (Shull and Hamer 1994; Farman et al. 1996; Tosa et al. 1995) because 2539 is a laboratory strain developed by crossing isolates from rice with a grass pathogen which lacks MAGGY (Shull and Hamer 1994). Fifty eight cosmids representing 29 distinct MAGGY loci (some of which contained more than one MAGGY element were identified in a genomic library of 2539 DNA. Twenty eight

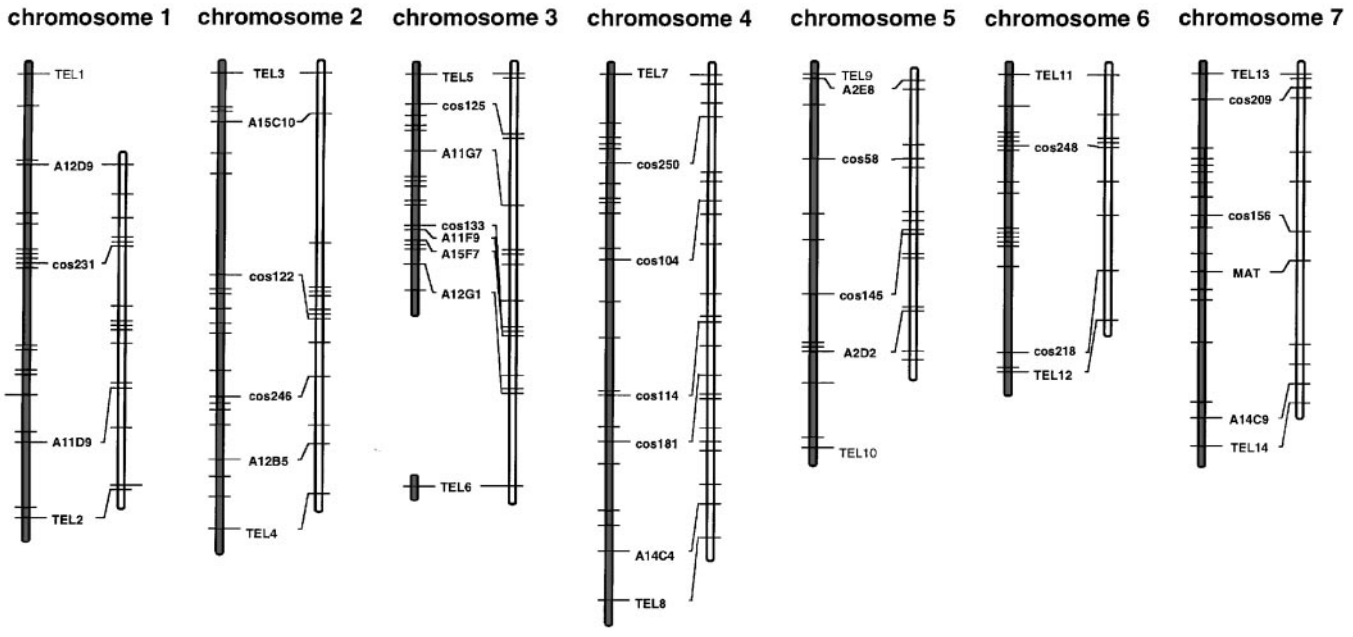


Fig. 2 Integration of two genetic maps of *M. grisea*. The synteny relationships between chromosomes in the genetic map shown in Fig. 1 (*shaded backbone*) and that published by Sweigard et al. (1993) (*unshaded backbone*) are indicated by comparative map locations of markers highlighted in *bold type*

of the elements, representing approximately 75% of the MAGGY insertions in the 2539 genome, were mapped to unique locations (Fig. 4) indicating that MAGGY is dispersed throughout the genome of 2539. It was not possible to map the final cosmid, as linked single-copy DNA did not hybridize well to *M. grisea* genomic DNA (data not shown). The genomic distribution of MAGGY elements resembles that of the inverted repeat transposon MGR586 (Romao and Hamer 1992; Farman et al. 1996) which also maps to dispersed locations.

Genomic duplications and rearrangements in regions associated with MAGGY probes

In the present study, we used co-dominant, single-copy probes flanking the repeated MAGGY element to identify RFLP markers. This proved to be very informative with respect to identifying rearrangements and duplications. When using dominant markers, such as repeated DNAs, telomeric RFLPs, RAPDs, AFLPs, etc., it is not possible to identify rearrangements associated with the recessive allele. Similarly, it is not easy to determine from which marker a new allele is derived. Another advantage of mapping repeats by associated single-copy probes is that they provide a single-copy DNA-based frame of reference for element dispersion.

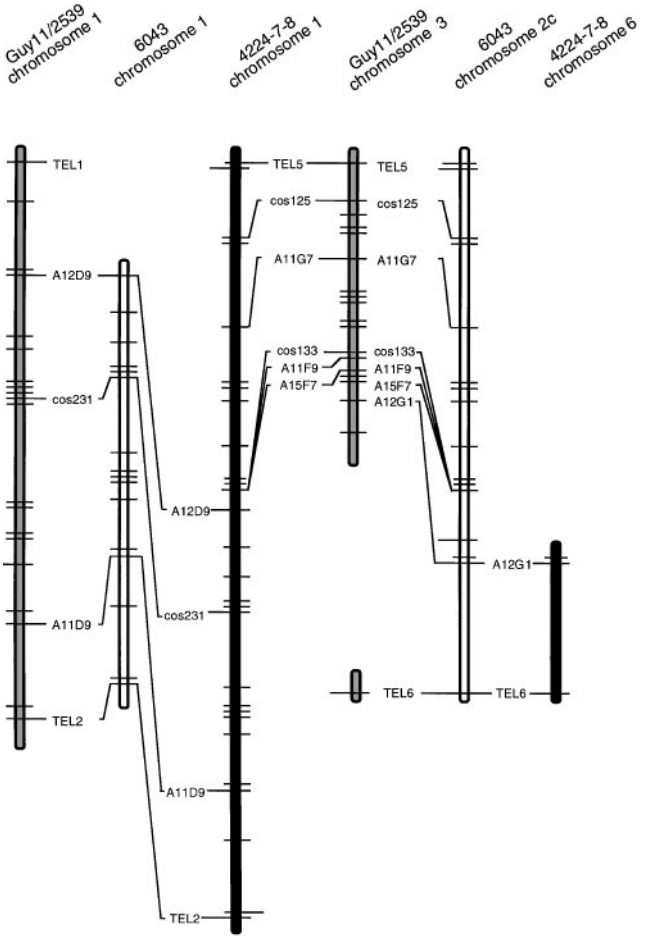


Fig. 3 Detail of a translocation occurring in strain 4224-7-8. Chromosome arms of each parent are represented as follows: Guy11 (=2539) – *shaded backbone*; 6043 – *open backbone*; 4224-7-8 – *black backbone*

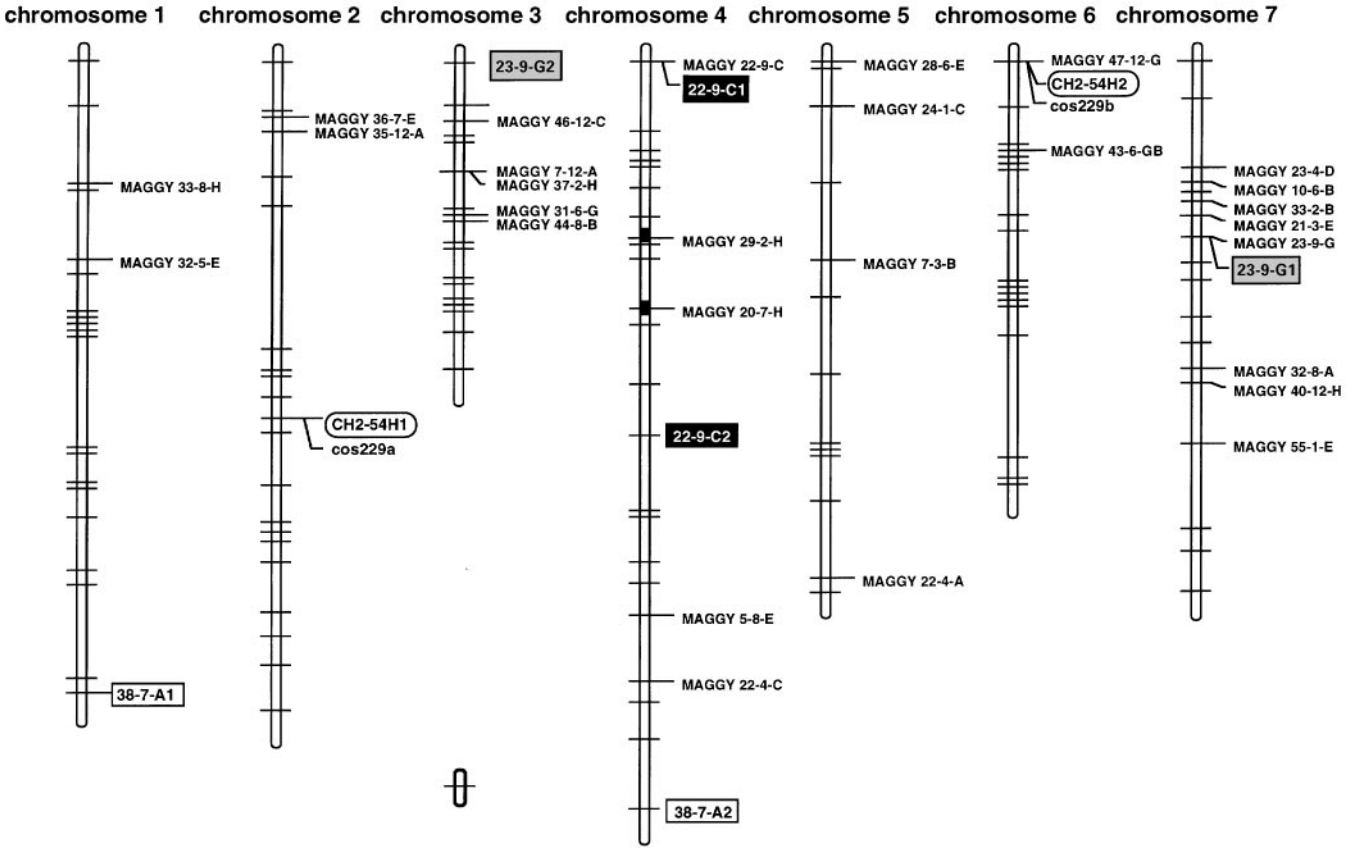


Fig. 4 Map positions of the MAGGY retrotransposon in the genome of 2539, and MAGGY-associated rearrangements and duplications. Locations of MAGGY retrotransposon insertions are denoted by the 'MAGGY' descriptor followed by the corresponding cosmid identification number. Duplicated markers are displayed in *boxes* and a translocated marker is highlighted with a *rounded box*. Two sites of rearrangement identified in progeny isolates are indicated by *black boxes* on the affected chromosome segments

During the course of mapping the MAGGY elements, aberrant segregation patterns were observed with some probes in some progeny (Fig. 5 and Table 2). Skinner et al. (1993) also reported finding a number of aberrant hybridization patterns in progenies using randomly isolated single-copy RFLP probes; however, the frequency and significance of these events was much more limited. Twenty eight percent (8/28) of the MAGGY-associated markers showed evidence of rearrangements, while only 2% (5/189) of the other (non-repeat associated) markers produced aberrant progeny genotypes (Table 2).

In this study, the MAGGY-associated markers 29-2-H and 20-7-H revealed non-parental alleles in several progeny and the new allele was common to all aberrant progeny (Fig. 5). The two loci were only loosely linked and the affected progeny were different for each probe, indicating that these events were independent. Molecular characterization of the 29-2-H locus of 2539 revealed three tandemly arranged MAGGY elements each separated by lone LTRs, all oriented in the same 5′-3′ direction relative to the orientation of the internal ORFs (Fig. 6).

In the case of 29-2-H, analysis of the genotype of surrounding markers indicated that the Guy11 locus had rearranged to produce the new allele. This, together with the observation that the rearrangement did not occur in a second cross, implies that it occurred within a sector of the Guy11 colony prior to mating. We suspect that Guy 11 may also contain MAGGY elements at the 29-2-H location which may have recombined to produced the new allele. This is not an unreasonable assumption as Guy 11 was found to contain at least two MAGGY elements at or near the same integration site as the MAGGY associated with 32-5-E in 2539; and this MAGGY was also shared by three other rice pathogenic isolates (Farman et al. 1996).

In contrast to the rearrangement identified by marker 29-2-H, that revealed by marker 20-7-H appeared to have occurred in the 2539 colony. Interestingly, 29-2-H and 20-7-H are linked (16.2 cM distance, Fig. 1) yet the markers between them segregate normally, ruling out the occurrence of an intermarker genomic deletion in the aberrant progeny. Moreover, other probes derived from cosmids 29-2-H and ♯3B did not reveal non-parental RFLPs (Appendix 1).

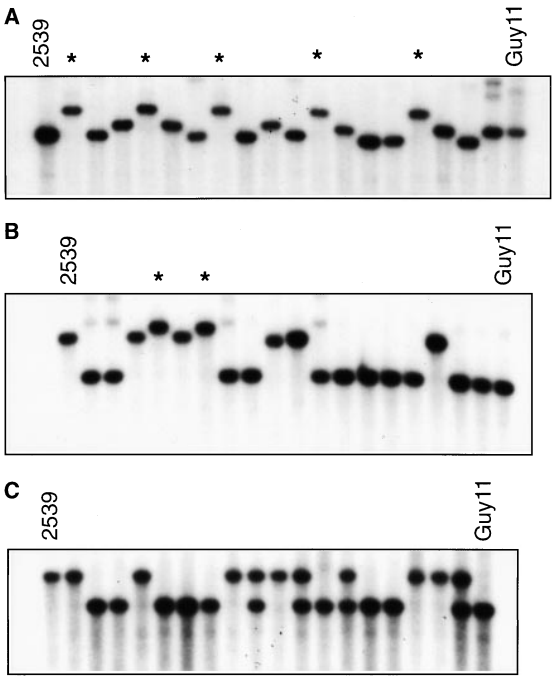


Fig. 5A–C Segregation patterns of markers showing unusual inheritance of RFLPs. Autoradiograms of representative Southern blots are shown. The positions of the parental DNA samples are indicated and progeny DNA samples were loaded in between. **A** Pattern observed for probe 29-2-H showing a rearrangement which produced a new allele. **B** Pattern observed for probe 20-7-H showing a rearrangement which also produced a new allele. **C** Pattern observed for duplicated probe 22-9-C

Table 2 Aberrantly segregating RFLP markers[a]

Chromosome	Marker[b]	No. of progeny exhibiting new RFLP[c]
#1	38-7-A (repetitive)	2/61
#2	35-12-A(MAGGY)	2/61
	A12B5 (repetitive)	1/61
	CH3-87H	1/61
	CH2-90H	1/61
#3	CH3-91H	1/61
	31-6-G(MAGGY)	2/61
	7-12-A(MAGGY)	1/61
	cos125	1/61
#4	21-1-G	1/61
	29-2-H(MAGGY)	7/61
	20-7-H(MAGGY)	8/61
	40	1/61
#5	cos58	1/61
#6	47-12-G(MAGGY)	2/61
	43-6-GB(MAGGY)	1/61
#7	23-9-G(MAGGY)	1/61
	cos156	1/61

[a] Markers producing new alleles that were different from either parental allele are represented
[b] Where known, additional features of cosmid clones are provided
[c] Number of aberant progeny/total progeny

Three markers, 22-9-C, 23-9-G and 38-7-A, which were all associated with repetitive DNAs, are duplicated in the Guy 11 genome. These loci are indicated in Fig. 4 by marker numbers with suffixes 1 and 2. Interestingly, For each of the duplicated and translocated sequences found in the Guy11 genome, at least one copy was located at a telomere and, in each case, only one copy of these sequences was present in the 2539 genome. In this context it is worthwhile to note that Guy 11 harbors the largest number of MAGGY elements that we have observed among collections of rice-infecting strains of *M. grisea* (Farman et al. 1996). Further experiments will be required to determine whether MAGGY is contributing to these genomic alterations.

Clustering of repetitive DNAs

MAGGY elements present in 29-2-H, 32-5-E and 23-9-G were mapped by using probes derived from single-copy regions immediately flanking the 3′ LTR. Other MAGGYs, however, were impossible to map using this strategy because the flanking region was also found to be repetitive. This finding was unusual because approximately 80% of the cosmid clones of 2539 have few or no repetitive DNA-containing fragments (data not shown). When total genomic DNAs of Guy11 and 2539 were used as hybridization probes to detect repetitive DNAs on Southern blots of digested MAGGY cosmid DNAs, most clones were found to possess additional fragments containing other repetitive DNA species. These Southern blots were rehybridized with probes derived from characterized repetitive elements from *M. grisea* including the inverted repeat transposons Pot2 (Kachroo et al. 1994) and MGR586 (Hamer et al. 1989, Farman et al. 1996) and the SINE elements, MgSINE and Ch-SINE (Kachroo et al. 1996). Solo-LTRs of MAGGY were also used. Skinner et al. (1993) reported on the occurrence of nine classes of repeated DNA in the *M. grisea* genome, one of which was derived from MAGGY (Farman et al. 1996). Some representatives of these nine repeat classes were also used as probes to the MAGGY-containing cosmids.

As shown in Table 3, MAGGY cosmids often hybridized with other types of repetitive DNA. In particular MAGGY was frequently associated with MGR586, Pot2 and the Mg-SINEs. Based on the estimated copy numbers of these elements, it appeared that they were associated with MAGGY more frequently than would be expected by chance. This was tested statistically for MGR586 (Hamer et al. 1989) and Pot2, by comparing the observed frequency of association with the expected frequency based on their copy numbers, the genome size, and the average cosmid insert size.

It was necessary to make an adjustment to the estimated genome size of 38 Mb (Hamer et al. 1989) as strain 2539 (Leung et al. 1988), from which the library

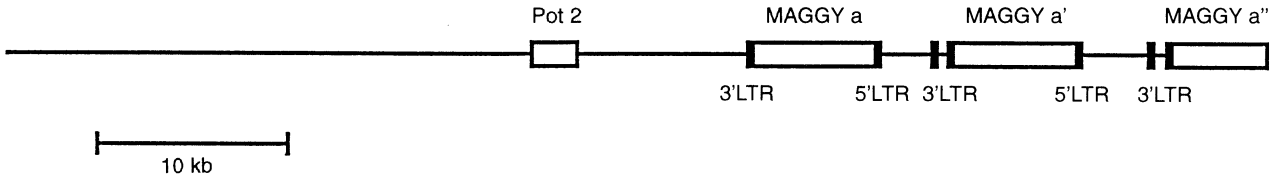


Fig. 6 Restriction map of a genomic region affected by a rearrangement event. Locations of a Pot2 element, MAGGY elements and solo-LTRs are indicated

was constructed, was derived by crossing rice-pathogenic strains which possess many copies of MAGGY and MGR586 with other strains which lack these elements entirely. Consequently, only a fraction of the genome of 2539 contains these elements and it is important to consider only this portion when calculating the expected number of cosmids possessing MAGGY and MGR586. The three final crosses in the pedigree of strain 2539 involved two backcrosses of a strain lacking these elements to a rice pathogenic isolate which possesses them (Leung et al. 1988; Farman and Leong 1996). Therefore, approximately 75% of the genome of 2539 should be derived from the rice pathogen, which equates to approximately 28.5 Mb. This fraction would be represented by 805 non-overlapping cosmids (average insert size = 35.4 kb). The expected number of cosmids containing both MAGGY and MGR586 is then calculated as the product of the proportion of cosmids containing each element multiplied by the number of cosmids. The observed number of six cosmids is in six-fold excess of the expected number (1.0) and the probability of this degree of association occurring by chance is 0.002 as determined by a Fisher's exact test. It should be noted that the corrected genome size is an estimate. The test was also performed by underestimating the proportion of the 2539 genome contributed by the rice pathogen (50%). Using a corrected genome size of 19 Mb, the probability of random association was determined to be 0.015. Similar tests for MAGGY and Pot2 returned probabilities of 5.6×10^{-8} and 6.7×10^{-6} for estimated contributions from the rice pathogen genome of 75% and 50%, respectively. This

Table 3 Repetitive elements hybridizing with MAGGY-cosmids and overlapping cosmids

MAGGY-cosmid	Overlapping cosmids	Ch.#	Solo-LTR	POT-2	SINE A	SINE B	MGR 586	SK3	SK12	SK31	SK36
33-8-H		1									
32-5-E	20-10-E, 40-12-H					+			+		
36-7-E	5-7-C, 32-1-B	2		+		+		+	+	+	+
35-12-A					+		+				
46-12-C	3			+		+	+	+	+	+	
7-12-A						+					
37-2-H											
31-6-G					+				+		
44-8-B	40-5-H			+			+	+			+
22-9-C	23-7-H	4			+				+		
29-2-H	24-6-H, #3B		++[a]	+				+			
20-7-H											
5-8-E				+		+		+	+		
22-4-C			+					+			
28-6-E	41-6-D	5		+				+			
24-1-C											
7-3-B	42-1-B			+	+		+	+	+	+	
22-4-A	4-8-G, 4-12-E			+		+		+	+	+	
47-12-G	35-2-A, 43-6-H	6				+	+		+	+	
43-6-GB	20-3-B		+	+				+			
23-4-D		7		++		+		+	+		
10-6-B	13-8-B, 25-11-A		+							+	
33-2-B				+				+			
21-3-E											
23-9-G	22-6-C			+				+		+	
32-8-A											
40-12-H	40-10-D									+	
55-1-E	13-4-H			+		+	+	+	+		
39-10-E	41-7-D, 47-12-C	?						+			

[a] There are two copies of solo LTRs in this cosmid contig

confirms the conclusion that these three families of elements tend to cluster in the genome.

The pattern of hybridization observed with some of the uncharacterized repeats identified by Skinner et al. (1993) indicated that SK3 and SK12 most likely contain parts of Pot2 and Mg-SINE, respectively (Table 3). Other repeats remain uncharacterized. In the case of MAGGY cosmid 41-6-D, the *Eleusine*-pathogen-specific retroelement grasshopper (*grh*) (Dobinson et al. 1993) was also found. This was unexpected as these two elements are exclusive to rice and grass pathogen genomes, respectively. This indicates that a recombination between rice and grass pathogen genomes most likely occurred in this region during the crosses used to develop 2539. Alternatively, one element may have transposed into the vicinity of the other.

The apparently clustered distribution of repeated DNAs in the *M. grisea* genome is intriguing. In the present study, the MAGGY elements appear to be frequently associated with one another and with other transposable elements; three of the 29 MAGGY loci analyzed herein possessed two or more copies of the MAGGY element. In previous studies we have identified a MAGGY element inserted into another (Farman et al. 1996), a Pot2 element inserted into a LINE element (Kachroo et al. 1994), and a SINE in Pot2 (Kachroo et al. 1996). It appears that certain chromosomal regions may be sinks for transposable elements. This parallels the finding that intergenic regions of maize are riddled with retrotransposons inserted into one another (SanMiguel et al. 1996). As MAGGY, Pot2 and the Mg-SINE elements are all found embedded in AT-rich DNA regions (Farman et al. 1996; Kachroo et al. 1994, 1996), it seems plausible that element clustering results from a tendency to integrate preferentially into these regions due to better accessibility for recombination.

In conclusion, the addition of more markers to the existing genetic map of *M. grisea* resulted in a clearer picture of genome organization and evolution in *M. grisea*. In particular, mapping of markers associated with the repetitive MAGGY element enabled the documentation of several interesting genomic rearrangements and duplications that would otherwise have gone unnoticed.

Acknowledgments The authors thank Scott Tingey (E. I. duPont de Nemours and Co.) for providing Mapmaker Macintosh V2.0 and Barbara Valent (E. I. duPont de Nemours and Co.) for graciously supplying cosmid RFLP probes and mapping data from their map. Many thanks are due to Murray Clayton for assistance with statistical analyses. This work was supported by the United States Department of Agriculture, and grants from Japan Tobacco Inc. and the Rockefeller Foundation to SAL.

Appendix 1

Chromosomal molecular marker constitutions of 61 progeny from a single cross of *M. grisea* isolates Guy 11 and 2539. All markers from the present study and those of Skinner et al. (1993), Smith and Leong (1994), and Farman and Leong (1995) and ordered as they appear on the map (co-segregating markers may not be in order). New markers from this study are highlighted in bold. Markers followed by (M) contain MAGGY. Each column of data represents a single progeny. A, allele inherited form Guy 11; C, allele inherited from 2539; m, polymorphic band was missing; d, polymorphic band different from either parent; b, allele shows inheritance from both parents; ( ), blank space indicates data not acquired. Only data representing the parental phenotypes were used for construction of the genetic map (Fig. 1)

```
Chromosome 1:
Progeny no.        5    10   15   20   25   30   35   40   45   50   55   60
                   .    .    .    .    .    .    .    .    .    .    .    .
TEL1           AACAAACAAACCCACAC AC CACACCACACAACCAAA CAAA CCAAACACCAACCCACC
42-4-F1        AACAAACAAACCCACACCACCCACACCACACAACCAAACCAAACCCAAACACCAACCCACC
2-9-G          CACAAACAAACACACACCACCCACAACACAAAACCAAACCAAACCCAAACACCCACCCAAC
33-8-H(M)      CCAAAACAAACAAACACCAACCACAACACAAAACCCAACCACACACAAAAACCCCCCCCAC
A12D9          CCAAAACAAACAAACACCAACCACAA ACCAAACCCAACCACACACAAAAACCCCCCCCAC
11             CCAAAACAAACAAACACCAACCACAACACCAAACCCAACCACACACAAAAACCCCCCCCAC
32-5-E(M)      CCAAAACAAAAAAACACCAACCACAACACCAAACCCAACAACAAACAAAAACACAAAAAAC
40-12-H(M)     ACAAAACAAAAAAACACCAACCACAACACCAAACCCAACAACAAACAAAACCACAAAAAAC
CH4-116H       ACAAAACAAAAAAACACCAACCACAACACCAAACCCAACAACAAACAAAACCACAAAAAAC
3-5-E          ACACCACCAAAAAACACCAACCAAAACACCAAACCCAACAACAACCAAAACCACAAAAAAC
21             ACACCACCAAAAAACACCAACCAAAACACCAAACCCAACAACAACCAAAACCACAAAAAAC
G34R            ACACCACCAAAAAACACCAACCAAAACACCAAACCCAACAACACCCAAAACCACAAAAAAC
4-145          ACACCACCAAAAAACACCAACCAAAACACCAAACCCAACAACACCCAAAACCACAAAAAAC
G137R          ACACCACCAAAAAACACAAACCAAAACACCAAACCCAACAACACCCAAAACCACAAAAAAC
LDH3           ACAC ACCAA AAACACAAACCAAAACACCAAACCCAACAACACCCAAAACCAC A AA C
4-163          ACACCACCAAAAAACACAAACCAAAACACCAAACCCAACAACACCCAAAACCACAAAAAAC
cos231         ACACCACCAAAAAACACAAACCAAAACACCAAACCCAACAACAACCAAAACCACAAAAAAC
```

50	ACACCACCAAAAAACACAAACCAAAACACCAAACCCAACAACAACCAAAACCACAAAAAAC
3-12-F	ACACCACCAAAAAACACAAACCAAAACACCAAACCCAACAACAACCAAAACCACAAAAAA
4-54	ACACCACCAAAAAACACAAACCAAAACACCAAACCCAACAACAACCAAAACCAAAAAAAAC
3-9-H	CCACCCACCAAACACACAAACCAACCCACCAAACCAAACAACAACCAACCCCAACAAACCA
1-7-C	CCACCCACCAAACACACAAACAAACCCACCAAACCAAACAACAACCAACCCCAACAAACCA
CH5-131H	CCACCCACCAAACACACAAACAAACCCACCAAACCAAACAACAACCAACCCCAACAAACCA
1.2H	CCACCCACCAACCACACAAACACACCCACAAAACCAAACAACAACCAACACCAACAAACCA
CH5-120H	CCACCCACCAACCACACAAACACACCCACAAAACCAAACAACAACCAACACCAACAAACCA
48-6-H	CCACCCACCAACCACACAAACACACCCACAAAACCAAACAACAACCAACACCAACAACCCA
AVR1-C039	CC A CCA CCA ACC CACCC C AAACCAAACAACC C C CACCAA A CCCA
5-10-F	ACACCAACCAACAACACAACCACACCAACAAAAACAACCAACCACCCACCCCAACCACCC
A11D9	ACACCAACCAACAACACAACCAAACCAACACAAACAACCAACCACCCACCCCAACCACCCA
55-4-C	ACACCAACCAA AACACAACCAAACCAACACAAACAACCAACCACCCACCCCAACCACCCA
13-9-A	ACACCAACCAACAACACACACCAACAAACACAAACAACACCCmCCCCCACCAAACCACCCC
38-7-A	ACAmCAACCAACAACACACACCAAdAAACmCAAAAAAmACCCdCCCCCACCAAACCACCCC
TEL2	ACACCAACCAACAA ACACACCAACAAA ACAAAAAAAACCCC CCCACCAAACCACCC

Chromosome 2:

Progeny no.	5	10	15	20	25	30	35	40	45	50	55	60
	.	.	.	.	.	.	.	.	.	.	.	.
TEL3	AAAAAC CCCAACCCAACACCAAAAACAACCCACAACCCC AA CAAAC CCCAACAAC C											
RDNA	AAAAACACCCAACCCAACACCACAAACACACCACAACCCCAAACCACACACCCCCCAACAC											
36-7-E(M)	AAAAACACCCAACCCAACACCACAAACACACCACAACCCCAAACCACACACCCCCACAACAC											
A15C10	AAAAACACCCAACCAAACACCACACACACACCACAACCCCAAACCACACACCCCCACAACAC											
35-12-A(M)	AdAAACACCCAACCAAACACCAdACACACACCACAbCCCCAAACCACACACCCCACAACAC											
CH3-24H	AAmAACACCCACCCAAAmCCCmCAmbCACACCACACCbCCAAACCACACACCCCACAACAC											
16-1-B	AACAACACCCACCCAAAACCAACAACCACACCACACCACCAAACCACACACCCCCCAACAC											
cos122	AACAAACACAACCCAAAACCACACACCACACCAACCAAACCAAACCACCCACACCCCCAAAA											
4-157	AACAAACACAACCCAAAAACACACACCACACCAACCAAACCAAACACCAACACCCCCAAAA											
4-10	AACAAACACAACCCAAAAACACACACCACACCAACCAAACCAACCACCAACACCCCCAAAA											
4-22	AACAAACACAACCCAAAAACACACACCACACCAACCAAACCAACCACCAACACCCCCAAAA											
52	AACAAACACAACCCAAAAACACACACCACACCAACCAAACCAACCACCAACACCCCCAAAA											
4-89	AACAAACACAACCCAAAAACCCACACCACACCAACAAAACCAACCACCACCACCCCCAAAA											
CH2-54H1	AACAAACACAACCCAAAAACACACACCACACCAACAAAACCAACCCACACCACCCCCAAAA											
cos229-2	AACAAACACAACCCAAAmACACACACCACACCAACAAAACCAACCCACACCACCCCCAAAA											
CH4-112H	AACAAACACAACCCAAAAACACACACCACACCAACAAAACCAACCCACACCACCCCCAAAA											
CH4-137H	AACAAACACAACCCAAAAACACACACCACACCAACAAAACCAACCCACACCACCCCCAAAA											
CH2-29H	AACAAACACAACCCAAAAAACACACACCACACCAACAAAbCCAAACCACACCACCCCCAAAA											
13-4-A	AACAAACACAACCCAAACAAACACAACACCCCAACAAAACCAACCCACACCACCCCAAAAA											
43-6-GA	AACAAACACAACCCAAACAAACACAACACCCCAACAAAACCAACCCACACCACCCCAAAAA											
cos246	AAAAAACACAAACCAACCAAACACAACACCCCAACAAAACAAACCCACACAACCCCAAAAA											
CH3-87H	AAAAAACACAAACCAACCAAACACAACACCACAACAAAACAAACCCACACAACCCCAdAAA											
CH3-2H	AAAAAACACAAACCAACCAAACACAACACCACAACAAAACAAACCCACACAACCCCACCAA											
64	AAAAAACACAAACAAACCAAACACAACACCACAACAAAACAAACCCACACAACCCAACCCA											
A12B5	AAAAAACACAAACAAACCAAACACAACACCACAAAACAAAAAAACCACACAAACCCACCCd											
CH2-90H	AAAAACCACAAACAAACCAAACACAACACCACAAAACAAAAAACCCAdACAACCCCACCCC											
CH2-100H	AAAAACCCAAACAAACCAAACACAACAACACAAAACCAAAAAACCACACAACCCCACCCC											
4-78	AAAAACCCAAAACAAACCAAACACAACAACACAAAACCACACCCCCACACACCCCCACCCC											
TEL4	AAAAACCCAAAACAAACCAAACACAACAACACAAAACCAC CCCCCA ACACCCCCACCCC											

Chromosome 3:

Progeny no.	5	10	15	20	25	30	35	40	45	50	55	60
	.	.	.	.	.	.	.	.	.	.	.	.
TEL5	CCACCC ACACACAACACCAAAAACCCCCA AACAACCCACCC CAACAAAACAAACCACA											
23-9-G2	CACCCAACACACAACACCAAAAACC CCACAACAACCCACCCACAACAAAACAAACCACA											
cos125	CCACCCAACACACAACACCAAAAACCCCCACAACAACCACCCCCdAAAAAAACAACCCACA											
46-12-C(M)	CCACCCAACACACAACAACCAAACCCCCACAACAACCACCCCCAAAAAAAACAACCCACA											
CH3-77H	CCACCCAACACACAACAACCAAAACCCCCCCAACAACCACCCCCAAAAAAAACAACACACA											
4-3	CCACCCACCACACAACAACCAAAACCCCCCCAACAACCACCCCCAAAAAAAACAACACACA											
A11G7	CCACCCACCCCACACCAACCAACACCCCCCCAACAACCACCCCCAACAAAAACAACACACA											
7-12-A(M)	CCACCCACCCCACACCAACCAACAdCCCCCCAACAACCACCCCCAACAAAAACAACACACA											
37-2-H(M)	CCACCCACCCCACACCAACCAACACCCCCCCAACAACCACCCCCAACAAAAACAACACACA											

```
CH3-125H      CCACCCACCCCACACCAACCAACACCCCCCCCAACAACCACCCCCAACAAAAACAACACACA
G39R           CCACCCACCCCACACCAACCAACACCCCCCCCAACAACCACCCCCAACAAAAACAACACACA
39-5-G         ACACCCACCCACCACCAACCAACACCCCCCCCAACAACCCCCCCCAACAAAAACAACAAACA
31-6-G(M)      ACACCCACCCACCACAAACdAAdACCCCCCCAACAACCCCCCCCCAACAAAAACAACAAACA
44-8-B(M)      ACACCCACCCACCACAAACCAACACCCCCCCCAACAACCCCCCCCAACAAAAACAACAACCA
CH2-94HH       ACACCCACCCACCAAAAACCCACmCCCCCACAACAACCCCCCCCCAACAAAAACAACAACCA
CH2-94HE       ACACCCACCCACCAAAAACCCACmCCCCCACAACAACCCCCCCCAAACmAAAACAACAACCA
cos133         ACACCCACCCACCAAAAACCCAAACCCCCCACAACAACCCACCCAAACAAAAACAACCCCCA
A1A2           ACACCCACCCACCAAAAACCCAAACCCCCCACAACAACCCACCCAAACAAAAACAACCCCCA
CH5-153H       ACACCCACCCACCAAAAACCCAAACCCCCCACAACAACCCACCCAAACAAAAACAACCCCCA
CH3-73H        ACACCCACCCACCAAAAACC AAACCCCCA AACAACC ACCCAAACAAAAACAACCCCCA
CH2-59H        ACACCCACCCACCAAAAACCCAAACCCCCCACAACAACCCACCCAAACAAAAACAACCCCCA
A11F9          ACACCCACCCACCAAAAACCCAAACCCCCAACAACAACCCACCCAAACAAAAACAACCCCCA
A14B9          ACACCCACCCACCAAAAACCCAAACCCCCAACAACAACCCACCCAAACAAAAACAACCCCCA
A14D5          ACACCCACCCACCAAAAACCCAAACCCCCAACAACAACCCACCCAAACAAAAACAACCCCCA
A16C12         ACACCCACCCACCAAAAACCCAAACCCCCAACAACAACCCACCCAAACAAAAACAACCCCCA
A15F7          ACACCCACCAACCAAAAACCCAAACCCCCAACAACAA CCACCCAAACAAAAACAACCCCAA
37-10-G        ACACCCACCAACAAAAAACCCAAACCCCCAACAACAACCCACCCAAACAAAAACAACCCCAA
CH5-67H        ACACCCACCAACAAAAAACC AAACCCCCAACAACAACCCACCCAAACAAAAACAACCCCAA
CH5-196H       ACACCCACCAACAAAAAACCCAAACCCCCAACAACAACCCACCCAAACAAAAACAACCCCAA
CH3-62H        ACACCCACCAACAAAAAACCCAAACCCCCAACAACAACCCACCCAAACAAAAACAACCCCAA
CH3-109H       AC CCCACCAACACAAAACCCAAACCCCCAACAACAACCCACC A AC AAAACA CCCCAA
A12G1          ACACCCACCAACACCACACCCAAACCCCCAACAACAACCCAACCAAACAAAAACAACCCCAA
18             ACACCCACCAACACCACACCCAAACCCCCAACAACAACCCAACCAAACAAAAACAACCCCmA
CH3-91H        ACAdCCACCAACACCACACCCAAACCCCCAACAACAACCCAACCAAACAAAAACAACCCCmA
CH5-176H       ACACCCACCACAACCACACCCAAACCCCCACCAACAACCCAAACAAACAAAAACAACCCAAA
TEL6           ACCAACCACACAAACCCAACCCACCCACACACAAAACAAAAACAACACCAAAAAACAAACC
```

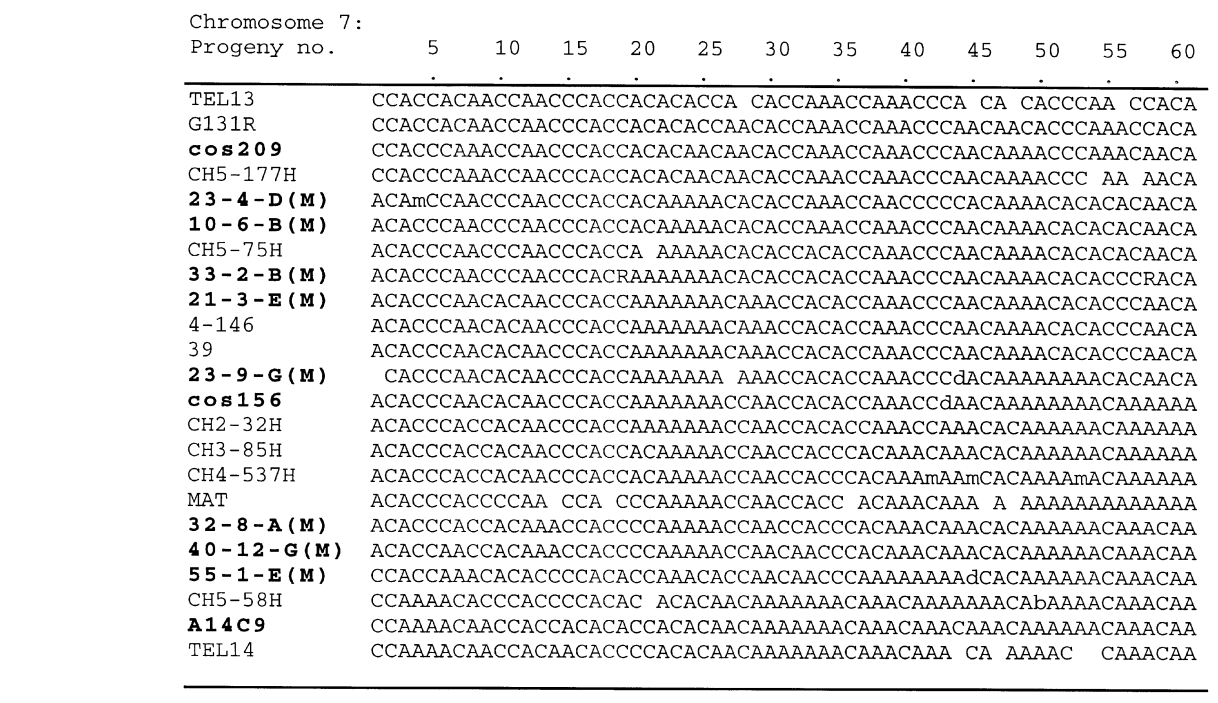
```
Chromosome 4:
Progeny no.        5    10   15   20   25   30   35   40   45   50   55   60
                   .    .    .    .    .    .    .    .    .    .    .    .
TEL7           CCCAAACACAACCC  CCAA CACAAAAA  ACCCACAACAAA CC ACAAC C  ACA A
22-9-C(M)      CCCAAACACAACCCCCCCAACCACAAAAACCACCCACAACAAACCCCCACAACC CCACACA
4-194          CCCAAACACAACCCCCCCAACCACAAAAACCACCCACAACAAACCCCCACAAC CCCACACA
4-181          CCCAAACACAACCCCCCCAACCACAAAACCACCCACAACAAACCCCCACAACmCCCACACA
55(R)          CCACCACACAACCCCCACAACCACAAACACCACCCACAACAAACCCCACCAACCCCCAACCA
40             CCAdCACACAACCCCCACAACCACAAACACCAACCACbACAAACCCCAbAAACCCCCAACCA
4-20           CCACCACACAACCCCCACAACCACAAACACAAACCACAACAAACCCAAAAACCCCCAACCA
CH5-188H       CCACCACACAACCCCCACAACCACAAACACAAACCACAACAAACCCAACAACCCCCAACCA
cos250         CCACCACACAACACCCACAACCACAAACACAAACCACAACAAACCCAAACACCCCCAACCA
4-183          CCACCACACAACACCCACAACCACAAACACAAACCACbACAAACCCAAACACCCCCAACCA
G121R          CCACCACACAACACCCACAACCACAAACACAAACCACAACAAACCCAAACACCCCCAACCA
4-186          CCACCACACAACACCmmCAACCACAAACACAAACCACAACAAACCCAAACACCCCCAACCA
21-1-G         ACACCACAAAACAACdACAACCCCAAACACAAACCACAACAAACCCAAACACCCCCAACCA
29-2-H(M)      dCACCACAdAACAACCACdACCCCCdACACAAdCCACdACAAACCCAAdCACCCCCAACAC
#3B(M)         ACACCACAAAACAACCACAACCCCAAACACAAACCACAACAAACCCAAACACCCCCAACAC
4-14           ACACCACAAAAAACCCACAAmCCCAAACACAAACCACAACAAACCCAAACACCCCCAACAC
20-7-H(M)      AdAddAdAAdAACCCCACAACdCdAACCAAAAACAACCACAAACCCACACACCCCdAACAC
cos104         ACACCACAACAACCCCACAACCCCAAACCAAAACAACCACAAACCCACACACCCCCAACAC
LDH1           ACACCACAAC ACCCCACAACCACAAACCAAAAAAACAACAAAAACACACACCC C AACC
22-9-C2        ACA   A A  ACCCCAC  CC C     AA AAA C  C   CACA A  CC AC A A
CH3-123H       ACACCCAAACCCCCCCCCCA AACCCAACAAAAAAACAACACACACACAAAACCAAAAAAC
cos114         ACACCCAAACCCCCCCCCCACACCCCAACAAAAAAACAACACACACACAAAACCAAAAAAC
42-10-C        ACCCCCAACCCCACCCCCCACACACCAACACAAAAACAACAAACACACAAAAC AAAAAAC
cos181         ACACCCAACCCCACCCCCCACACACCAACACAAAAACAACAAACACACAAAACCCAACAAC
4-041          ACACCCAACCCCACCCCCCACCCAACAACACAAAAACAACAAACACAAAAAAACCAACAAC
5-8-E(M)       ACACCCAACCCCACCCCCCACCCAACAACACAAAAACAACAAACACAAAAAAACCAACAA
22-4-C(M)      ACACCCACCCACACACCCCACCCAACAAAACAACCAAACCAAACACAAAAAAACCAACAAA
CH3-67H        ACACCCACCCACACACCCCACCCAACCAAACAACCCAACCAAACCCAAAAACACCAACAAA
A14C4          ACACCCACCCACACAACCAACCCAACCAAACAACCCAACCAAACCCAAAAACCCCAACACA
38-7-A2        ACAACCACCAACCCAACCAACCCACAAAAACAACCCAACCAACCCCAAAAACCCCAAAACC
TEL8           ACA CCAC AACCCAACCAAC CACAAAAACAACCCAACCAAC CCAAAAACCCCAAAACC
```

Chromosome 5:

Progeny no.	5 .	10 .	15 .	20 .	25 .	30 .	35 .	40 .	45 .	50 .	55 .	60 .
TEL9	AACAAACAAACCC AAAAACCCAACAAACCCCAAACAAAAAAC ACAACAAACAAAAAAA											
28-6-E(M)	AACAAACAAACCCCAAAAACCCAACAAACCCCAAACAAAAAACACACAACAAACAAAAAAA											
A2E8	AACAAACAAAC CCAAAAACCCAACAAACCCCAAACACAAAACACACAACAAACAAA AAA											
22-2-D	AACAAACAAACCCCAAAAACCCAACAAACCCCAAACACAAAACACACAACAAACAAAAAAA											
24-1-C(M)	AACAACAAACCCCCAAAAACCCAACAAACCCCCAACACAAAACACACAACAAAAAAAAAAA											
cos58	AACAACACCCCCACAAAAACCCAACAAACCCCCAACCCAACCCACCCAAAAAAAAAACCd											
7-3-B(M)	CACAACACCCCCCCAAAACCAAAACAAACCCCCCACCCCACCCAACAAAAAACAAAAACCA											
19-10-A	CCAAACACCCCC CCCAACCAAAACAAACCCCCCACCACACCCAACAAAAAACAAAAACCA											
cos145	CCACCCACACACCCCCAAACACAACAAACCACCCACCAAACACAACACAAAAAACAACAACCA											
CH3-108H	CCACCCACACCCCACCCAACCCACCAAACCACCAACCAAAAAACCACAAAAAACCAC											
4-178	CCACCCACACCCCACCCAACCCAACAAACCACCAACCAAAAAACCACAAAAAACCACAACCA											
CH3-98H	CCACCCACACCCCACCCAACCCAACAAACCACCAACCAAAAACAACAAAAAACCACAACCA											
A2D2	CCACCCACACCCCACCCAAC CAACAAACCACCAACCAAAAACAACAAAAAAC ACAACCA											
CH3-33H	CCACCCACACCCCCC AACCCACCAbACCACCACCCAAAAACAACAAAAAACCAbACAAA											
22-4-A(M)	ACACCCACACCCCCCCCAACCCACCCACCCACAACACAAAAAAACAAACAAACCACACAAC											
CH3-12H	ACACCCACACCCCCCCCAACCCACCCACCCACAACACAAAAAAACAAACAAACCACACAAC											
TEL10	ACACCCACACCCCCCCCAACCCACCCACCCACAACACCAAAAA CAAACAAACCACACACC											

Chromosome 6:

Progeny no.	5 .	10 .	15 .	20 .	25 .	30 .	35 .	40 .	45 .	50 .	55 .	60 .
TEL11	AACCCAAAACCACAACC CACACCAAACCACCC AACCCAACAACACACCCAACCAACACC											
cos229a	AACCCAAAACCACAACCmCACACCAAACCACCCAAACCCAACAACACACCCAACCAACACC											
47-12-G(M)	AACCCAAAACCACAACCmCACACCAAAdCACCCAAACCCAACAACACACCCAACCAAdACC											
CH2-54H2	AACCCAAAACCACAACCCCACACCAAACCACCCAAACCCAACAACACACCCAACCAACACC											
CH3-44H	AACCCAAACCCACACCACCACACCAAACCACCCAAACCAAACCACACACCCAACCCACACC											
2-10-G	AACCCCACCCCACACCACCACACCCAACCACCCAAACCACACCCCACACCCAACCCACACC											
43-6-GB(M)	AACCCCACCCCdCACCACCACACCCAACCACCCAAACCACACCCCACACCCAACCAACACC											
CH4-173H	AACCCCACCCCACACCACCACACCCAACCACCCAAACCACACCCCACACCCAACCAACACC											
CH4-121H	AACCCCACCACACACCACCACACCCAACCACCCAAACCACACCCCACACCCAACCAACACC											
CH5-191H	AACCCCACCACACACCACCACACCCAACCACCCAAACCACACCCCACACCCAACCAACACC											
cos248	AACCCCACCACCCACCACCACACCCAACCACCCAAACCACACCCCACACCCAACCAACACC											
CH3-132H	AACCCCACCACCCACCACCA ACCCAACCACCCAAACCACACCCCACACCCAACCAACAC											
CH4-133H	AACCCCACCACCCACCACCACACCCAACCACCCAAACCACACCCCACACCCAACCAACACC											
CH5-185H	AACCCCACCACCCACCACCACACCCAACCACCCAAACCACACCCCACACCCAACCAACACC											
CH4-131H	AACCCCACCACCCACCACCACACCCAACCACCCAAACCACACCCCACACCCAACCAACACC											
CH4-68H	AACCCCACCACCCACCACCACACCCAACCACCCAAACCACACCACACACCCAACCAACA C											
CH5-181H	AACCCCACCACCCACCACCACAmCCAACCACCCAAACCACACCACACACCCAAmCAACACC											
CH5-98H	AACAACCCCACACACCACCACACCCAACCACACAAACCACACCCCACACCCAACCAACACC											
4-48	AACAACCCAACACACCACCACACCCAACCACACAAACCACACCACACAC CAACCAACACC											
CH2-57H	AACAACCCAACACACCCCCACACCCAACCAAACACAAAAAACCACACACACAACCAACACC											
CH3-113H	ACAACCCAACACACCCCCACACCCAACCAAACACAAAAAACCACACACACAACCAACACC											
CH4-60H	AACAACCCAACACAbCCCCACACCCAACCAAACACAAAAAACCACACACACAACCAACACC											
CH4-161H	AACAACCCAACACACCCCCACACCCAACCAAACACAAAAAACCACACACACAACCAACACC											
CH5-167H	AACAACCCAACACACCCCCACACCCAACCAAACACAAAAAACCACACACACAACCAACACC											
CH4-119H	AACAACCCAACACACCCCCACACCCAACCAAACACAAAAAACCACACAAACAACCAACACC											
21-2-H	AACAACCCAACACACCCCCACACCCAACCAAAAACAAAAAACCACACAAACAACCAACACC											
37-11-F	AACAACCCAACACACCCCCACACCCAACCAAAAACAAAAAACAACACAAACAACCAACACC											
CH5-61H	AACAACCCAACACACCCCCACACCCAACCAAAAACAAAAAACAACACAAACAACCAACAC											
CH4-5H	AACAACCCAACACACCCCCACACCCAACCAAAAACAAAAAACAACACAAACAACCAACACC											
CH5-97H	AACAACCCAACACACCCCCACACCCAACCAAAAACAAAAACCAACACAAACAACCAACACC											
CH3-122H	AACACCCCAACACACCCCAACACCCAACCAAAAACAAAAACCCACACACACAACCAACACC											
cos218	ACCAACCCCACACACCCCCACACCCACCAAAAACCAAACACAAACCAAAACAACCCACACA											
4-69	ACCAACCCCACACACCCCCACACCCACCAAAAACCAAACACAAACCAAAACAACCCACACA											
CH5-184H	ACCAACACCACAAACCCCCACACCCACCAAAAACCAAACACAAACCAAAACCACCCACACA											
TEL12	ACCAACACCACAA CCCCCACACCCACCAAAAACCAAACAAAAACCAAAACCACCCACACA											



References

Budde AD, Smith JR, Farman ML, Skinner DZ, Leong SA (1993) Genetic map of the fungus *Magnaporthe grisea*. In: O'Brien SJ (ed) Genetic maps, 6th edn. Cold Spring Harbor Laboratory, Cold Spring Harbor, New York, pp 3.110–3.111

Dobinson KF, Harris RE, Hamer JE (1993) Grasshopper, a long terminal repeat (LTR) retroelement in the phytopathogenic fungus *Magnaporthe grisea*. Mol Plant-Microbe Interact 6:114–126

Farman ML, Leong SA (1995) Genetic and physical mapping of telomeres in the rice blast fungus, *Magnaporthe grisea*. Genetics 140:479–492

Farman ML, Leong SA (1996) Genetic analysis and mapping of avirulence genes in *Magnaporthe grisea*. In: Bos CJ (ed) Fungal genetics: principles and practice. Marcel Dekker Inc., New York, Basel, Hong Kong, pp. 295–315

Farman ML, Tosa Y, Nitta N, Leong SA (1996) MAGGY, a retrotransposon in the genome of the rice blast fungus, *Magnaporthe grise*a. Mol Gen Genet 251:665–674

Hamer JE, Farrall L, Orbach MJ, Valent B, Chumley FG (1989) Host species-specific conservation of a family of repeated DNA sequences in the genome of a fungal plant pathogen. Proc Natl Acad Sci USA 86:9981–9985

Kachroo P, Leong SA, Chattoo B (1994) Pot2, an inverted repeat transposon from the rice blast fungus *Magnaporthe grisea*. Mol Gen Genet 245:339–348

Kachroo P, Leong SA, Chattoo B (1996) Mg-SINE: a short interspersed nuclear element from the rice blast fungus *Magnaporthe grisea*. Proc Natl Acad Sci U.S.A. 92:11125–11129

Leong S, Farman M, Smith J, Budde A, Tosa T, Nitta N (1994) Molecular-genetic approach to the study of cultivar specificity in the rice blast fungus *Magnaporthe grisea*. In: Zeigler R, Leong S, Tang P (eds) Rice Blast Disease, CABI, London, pp. 88–110

Leung H, Borromeo ES, Bernado MA, Notteghem JL (1988) Genetic analysis of virulence in the rice blast fungus *Magnaporthe grisea*. Phytopathology 83:1427–1433

Maniatis T, Fritsch EF, Sambrook J (1982) Molecular cloning. Cold Spring Harbor Laboratory, Cold Spring Harbor, New York

Manicom BQ, Bar-Joseph M, Rosner A, Vigodsky-Haas H, Kotze JM (1987) Potential applications of random DNA probes and restriction fragment length polymorphisms in the taxonomy of the Fusaria. Phytopathology 77:669–672

Michaels SD, John MC, Amasino RM (1994) Removal of polysaccharides from plant DNA by ethanol precipitation. BioTechniques 17:274–276

Romao J, Hamer JE (1992) Genetic organization of a repeated DNA sequence family in the rice blast fungus. Proc Natl Acad Sci USA 89:5316–5320

SanMiguel P, Tikhonov A, Jin Y-K, Motchoulskaia N, Zhakarov D, Melake-Berhan A, Springer PS, Edwards KJ, Lee M, Avramova Z, Bennetzen JL (1996) Nested retrotransposons in the intergenic regions of the maize genome. Science 274:765–768

Shull V, Hamer JE (1994) Genomic structure and variability in *Pyricularia grisea*. In: Zeigler RS, Leong SA, Teng PS (eds) Rice Blast Disease, CABI International, Wallingford, UK, pp 65–86

Skinner DZ, Leung H, Leong SA (1990) Genetic map of the blast fungus *Magnaporthe grisea*. In: O'Brien SJ (ed) Genetic maps, 5th edn. Cold Spring Harbor Laboratory, Cold Spring Harbor, New York, pp 3.82–3.83

Skinner DZ, Budde AD, Farman ML, Leung H, Leong SA (1993) Genome organization of *Magnaporthe grisea*: genetic map, electrophoretic karyotype, and occurrence of repeated DNAs. Theor Appl Genet 87:545–557

Smith JR, Leong SA (1994) Mapping of a *Magnaporthe grisea* locus affecting rice (*Oryza sativa*) cultivar specificity. Theor Appl Genet 88:901–908

Southern EM (1975) Detection of specific sequences among DNA fragments separated by electrophoresis. J Mol Biol 98:503–517

Sweigard JA, Valent B, Orbach MJ, Walter AM, Rafalski A, Chumley FG (1993) Genetic map of the rice blast fungus *Magnaporthe grisea*. In: O'Brien SJ (ed) Genetic maps, 6th edn. Cold Spring Harbor Laboratory, Cold Spring Harbor, New York, pp 3.112–3.115

Tosa Y, Nakayashiki H, Hyodo H, Mayama S, Kato H, Leong SA (1995) Distribution of retrotransposon MAGGY in *Pyricularia* species. Ann Phytopathol Soc Japan 61:549–554

Zhu H, Whitehead DS, Lee Y-H, Dean RA (1996) Genetic analysis of developmental mutants and rapid chromosome mapping of *APP1*, a gene required for appressorium formation in *Magnaporthe grisea*. Mol Plant-Microbe Interact 9:767–774