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Genetic mapping of grapevine (*Vitis vinifera* L.) applied to the detection of QTLs for seedlessness and berry weight

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Abstract Parental and consensus genetic maps of *Vitis vinifera* L. (2n = 38) were constructed using a F_1 progeny of 139 individuals from a cross between two partially seedless genotypes. The consensus map contained 301 markers [250 amplification fragment length polymorphisms (AFLPs), 44 simple sequence repeats (SSRs), three isozymes, two random amplified polymorphic DNAs (RAPDs), one sequence-characterized amplified region (SCAR), and one phenotypic marker, berry color] mapped onto 20 linkage groups, and covered 1,002 cM. The maternal map consisted of 157 markers covering 767 cM (22 groups). The paternal map consisted of 144 markers covering 816 cM (23 groups). Differences in recombination rates between these maps and another unpublished map are discussed. The major gene for berry color was mapped on both the paternal and consensus maps. Quantitative trait loci (QTLs) for several quantitative subtraits of seedlessness in 3 successive years were searched for, based on parental maps: berry weight, seed number, seed total fresh and dry weights, seed percent dry matter, and seed mean fresh and dry weights. QTLs with large effects (R^2 up to 51%) were detected for all traits and years at the same location on one linkage group, with some evidence for the existence of a second linked major QTL for some of them. For these major QTLs, differences in relative parental effects were observed between traits. Three QTLs with small effects (R^2 from 6% to 11%) were also found on three other linkage groups, for berry weight and seed number in a single year, and for seed dry matter in 2 different years.

Keywords *Vitis vinifera* L. · Genetic maps · QTLs · Seedlessness · Berry weight

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Introduction

Seedless grapevine has traditionnally been used mainly for raisin production. There has also been interest in seedless grapes for fresh fruit consumption, and consequently for many decades now in the development of new seedless cultivars (Loomis and Weinberger 1979). Two types of seedlessness, parthenocarpy and stenospermocarpy, can be found among grape genetic resources. However, only stenospermocarpy can lead to berry sizes compatible with commercial requirements for fresh fruit production. In stenospermocarpic berries, double fertilization occurs but seeds fail to fully develop because of early degeneration of endosperm and abnormal development of integuments (Ledbetter and Ramming 1989).

Stenospermocarpy, as measured by the degree of seed development, exhibits quantitative and qualitative variation (Striem et al. 1992). The classical source of seedlessness in grape breeding programs is Sultanina, also called Thompson Seedless. Initially, seedless cultivars related to Thompson Seedless were used as male parents in crosses with female seeded genotypes. The proportion of completely seedless genotypes obtained in these progenies, as evaluated by perceptibility, was low (Weinberger and Harmon 1964; Loomis and Weinberger 1979; Spiegel-Roy et al. 1990; Roytchev 1998). Later on, the use of seedless genotypes as female parents was made possible by in vitro embryo rescue, leading to larger proportions of completely seedless genotypes in progenies of seedless × seedless crosses (Spiegel-Roy et al. 1989; Ramming et al. 1990) or in progenies of seeded × seedless crosses with seedless males issued from seedless × seedless crosses (Ledbetter and Burgos 1994). More recently,

breeding research has also focused on the development of molecular markers linked to the genes controlling seedlessness (Striem et al. 1994, 1996). Such markers could be valuable tools for further increasing the proportion of seedless genotypes in progenies through a documented choice of parents, but also for early selection of seedless offspring to be transplanted to the field for phenotypic evaluation. They would thus allow the speeding up of breeding programs, along with in vitro techniques.

Recently, a model of genetic determinism for the degree of seed development was proposed by Bouquet and Danglot (1996), based on both a review of several segregation studies and the analysis of a progeny segregating for seedlessness (MTP3140) obtained by crossing two partially seedless genotypes. They assumed that this complex trait was under the control of three independent recessive genes regulated by a major dominant inhibitor gene, *SdI*. By means of bulk segregant analysis applied to the MTP3140 progeny, Lahogue et al. (1998) developed a SCAR marker, SCC8, linked to *SdI*, codominant in this progeny. This marker explained a large part (65–89%) of the total phenotypic variation of seedlessness component traits. All individuals homozygous for the $SCC8^+$ allele were completely seedless or had only small-sized seed traces (Class 1, as defined in Bouquet and Danglot 1996), and all individuals with normally developed seeds (Class 4) were homozygous for the $SCC8^-$ allele, except one recombinant individual. A few $SCC8^+/SCC8^-$ individuals also belonged to Class 1, but most of them were only partially seedless (Classes 2 and 3), with a large variation in the degree of seed development, ranging from unperceptible seed traces to large-sized hard traces (40–50% dry matter). These results partly confirmed the hypothesis of Bouquet and Danglot (1996), but part of the total phenotypic variance still remained unexplained. Finally, a recent study of other progenies (Adam-Blondon et al. 2001) showed that seeded individuals can be heterozygous at SCC8, which is also compatible with this hypothesis. However, it appears that the understanding of the genetic determinism of stenospermocarpic seedlessness is not sufficient yet to allow efficient marker-assisted selection (MAS) for seedlessness.

The objectives of the present paper were thus: (1) to develop genetic maps of a grapevine cross segregating for seedlessness and berry weight, with several markers including mainly AFLPs and SSRs, and also a morphological marker, berry color; (2) to use these maps to further investigate the genetic control of seedlessness component traits and berry weight (number, location, and effect of QTLs involved). Two genetic mapping studies of grapevine have been published so far (Lodhi et al. 1995; Dalbo et al. 2000) for complex interspecific hybrids. Therefore, we report here the first consensus map of *Vitis vinifera* L., as well as the first QTL detection results for grapevine.

Materials and methods

Mapping population

The mapping population, MTP3140, is a F_1 progeny obtained by crossing two partially seedless genotypes, MTP2223-27 (Dattier de Beyrouth × 75 Pirovano) with MTP2121-30 (Alphonse Lavallée × Sultanine), and using in vitro rescue of the embryos (Bouquet and Davis 1989). The 139 full-sib genotypes, the two parents, and the four grand-parents were overgrafted in 1993 in one replicate at the INRA Experimental Station Chapitre near Montpellier, France. This population was chosen because of its large and well-balanced segregation for seedlessness component traits.

Phenotypic evaluation of seedlessness, berry weight and berry color

For each genotype, 100–200 berries were randomly sampled, and the 100 largest ones were weighted (berry weight, BW). All the seeds (or seed traces) of these 100 berries were then extracted, counted (seed number, SN), weighted (seed total fresh weight, STFW), dried at 80 °C and weighted again (seed total dry weight, STDW). All these traits were measured in 1994 (Bouquet and Danglot 1996), 1995 and 1996. Three additionnal traits derived from these measures were also analyzed: seed percent dry matter (SDM = STDW/STFW), seed mean fresh weight (SMFW = STFW/SN) and seed mean dry weight (SMDW = STDW/SN). Although Striem et al. (1992) showed potential interest for including the degree of development of the endosperm and seed coat hardness as subtraits for the study of stenospermocarpic seedlessness in grapes, the test of this method that we carried out did not yield us reliable evaluation and classification of seeds and seed traces for these subtraits. Therefore, we have chosen to present only results concerning easily quantifiable seedlessness subtraits such as weights and numbers.

Year effect was tested for each trait using a linear model with two main effects (genotype and year) and no interaction. Phenotypic correlations between traits within each year were tested using Pearson correlation coefficients. These statistical analyses were performed with SAS (SAS Institute Inc., Cary, N.C., USA).

In grapes, berry coloration is determined by at least one major dominant gene (Durquety and Destandau 1967), except for pink coloration, which seems to be under the control of three dominant genes (Wagner 1967). The female parent had yellow-green berries, the male purple-red ones, and presence of the coloration was segregating in the progeny. In this study, berry color was thus scored as a qualitative trait, with black, blue, purple or red scored as the presence of coloration, yellow or green as absence, and pink as missing data. Quantitative variation of color intensity was not studied in this population because the power of QTL detection in each subgroup (colored/uncolored) would have been too low.

Genotyping

Total DNA was extracted from young expanding leaves of each progeny, purified, and quantified after the protocol of This et al. (1997).

The progeny was genotyped for 47 SSRs: 11 VVMD (5, 6: Bowers et al. 1996; 14, 24, 25, 27, 28, 31, 32, 34, 36: Bowers et al. 1999), three VVS (2: Thomas and Scott 1993; 16, 112: M.R. Thomas, CSIRO, unpublished), VH43 (M.R. Thomas, CSIRO, unpublished), and 32 markers developed by the international consortium of laboratories VMC (Vitis Microsatellite Consortium, managed by Agrogene SA, Moissy Cramayel, France), among which only a few have already been published (VrZAG25 and VrZAG79: Sefc et al. 1999; VMC4F3.1 and VMC4H5: Di Gaspero et al. 2000). Laboratories interested in unpublished markers developed by VMC should contact Agrogene SA. These SSR markers were selected to be well-spread over the 19 linkage groups according to the last available version of the unpublished reference SSR map of

Riaz and Meredith (139 SSRs mapped to 19 linkage groups and spanning 1,882 cM Kosambi, for a Riesling × Cabernet Sauvignon cross; unpublished data). An earlier version of this map was presented at the Plant and Animal Genome VIII Conference (January 9–12, 2000, San Diego, Calif.). SSR markers were also selected to be as polymorphic as possible, the preferred segregation type being 1:1:1:1 with four different alleles. SSR amplifications were performed on the parents and the progeny after the protocol of Loureiro et al. (1998), with 26 cycles of 1 min at 94 °C, 1 min at 56 °C and 1 min at 72 °C.

AFLP markers were produced after Vos et al. (1995), with adenosine and cytosine as the pre-selective nucleotides for *Eco*RI and *Mse*I primers, respectively, and then three selective nucleotides. Polymorphism was tested for 64 primer combinations, either on the two parents or on a random sample of 8–10 offspring individuals. Eighteen primer combinations were selected for genotyping the parents and the whole progeny. Since the expected map length was approximately 1,500–2,000 cM (Lodhi et al. 1995; Riaz and Meredith, unpublished results), we stopped adding new AFLP markers when the number of markers expected to saturate the map was reached (268 and 358, for 1,500 and 2,000 cM, respectively; after Beckmann and Soller 1983).

For both SSRs and AFLPs, amplification products were separated after the protocol of Loureiro et al. (1998). All gels were independently scored by two persons, and inconsistent or ambiguous scores were considered as missing data.

The parents and the progeny were also genotyped for four isozymes (GPI, PGM, AAT and PER) according to Boursiquot and Parra (1996) and Ben Abdallah et al. (1998).

Construction of genetic maps

The genotypic data for one codominant SCAR marker (SCC8) segregating 1:2:1, and two RAPD markers (P18-530 and C08-1020) segregating 3:1, developed by Lahogue et al. (1998) on the same mapping population, were included in the map.

The global segregation of each marker was tested for goodness-of-fit to the appropriate expected segregation ratio using a chi-square test. Segregation goodness-of-fit was also tested in both parents separately for loci segregating 1:1:1:1 or 1:2:1. AFLP loci for which the observed segregation strongly deviated from the expected one ($P < 0.0005$) were discarded, since they probably resulted from overlapping loci. As a preliminary step to mapping, the grouping of markers at LOD (logarithm of the odds ratio) 4.0 and the computation of 2-point recombination frequencies were performed using JOINMAP 2.0 (Stam and van Ooijen 1995), in order to check consistencies in the phases chosen by the software. Loci showing inconsistencies, i.e. considered as coming sometimes from one grand-parent and sometimes from the other grandparent, were removed from the data set.

One map was constructed for each parent according to the pseudo-testcross strategy first proposed by Grattapaglia and Sederoff (1994), using MAPMAKER/EXP 3.0 (Lander et al. 1987). For each parental map, we used markers segregating 1:1 in this parent, and markers segregating 1:1:1:1 or 1:2:1 (ab × ab, ab × a0 and a0 × ab cases), re-coded to keep the segregation information for this parent only. The genotypic classes for which parental origin could not be deduced were considered as missing data. Markers segregating 3:1 could not be used for parental maps. Because of unknown linkage phases, all genotypic data were re-coded before linkage analysis, by exchanging alleles (i.e. genotypes 'A' re-coded 'H' and vice versa). This re-coded data set was analyzed together with the original one. Linkage analysis of the whole data set thus yielded twice the expected number of linkage groups, with homologous groups containing the same markers in the same order. Linkage groups were determined using threshold values of 4.0 for LOD and 0.3 for recombination rate. The ERROR DETECTION option was used systematically with a 1% error probability.

A consensus map was also constructed using JOINMAP. Markers segregating 1:2:1 for the ab × a0 and a0 × ab cases could not be handled directly by this software, so we included them in a duplicated form, using the same re-coding as in parental maps. They were thus considered as two separate loci, one segregating only in the one-banded parent (suffix a0), and the other one segregating only in the two-banded parent (suffix ab). Linkage groups were determined using a LOD threshold value of 4.0. For all maps, the Kosambi mapping function (Kosambi 1944) was used to convert recombination fractions into map distances.

Estimation of genome size

The expected genome size for each parent was estimated using both the method of moments proposed by Hulbert et al. (1988), and the modified version of this method proposed by Chakravarti et al. (1991). To handle the problem of missing data with the method of Hulbert et al. (1988), we had to use a little approximation: instead of the exact number of informative plants in pairwise combinations of loci, we used the upper class bounds of the frequency distribution of these numbers, as given by JOINMAP JMSLA (i.e. 19 to 139 by 10). For both methods, the estimation of genome size was first performed using all mapped loci, and then excluding all AFLPs because these markers exhibited a clustered distribution on our maps. Confidence intervals for genome-length estimates were computed according to Gerber and Rodolphe (1994) for a bilateral type-I error rate $\alpha = 5\%$. Expected genome coverage (% of the estimated length of the genome containing no gap > 20 cM under the assumption of random distribution of markers), was estimated by the method of Beckmann and Soller (1983), modified to take into account the effects of chromosome ends (Lange and Boehnke 1982). We estimated the expected coverage obtained with all mapped markers (157 and 144, for maternal and paternal parents, respectively), even though using the estimated genome length obtained without AFLPs.

QTL detection

Framework parental maps for QTL detection were obtained with MAPMAKER by dropping markers within clusters until marker order was supported by LOD 0.5 or more. Markers dropped first were those with the largest probability of erronous genotypes (as given by MAPMAKER) and/or the largest number of missing data. Since genotype × year interactions could not be tested, the data from each year were analyzed separately. QTL detection was performed using two different methods. First, the non-parametric Kruskal-Wallis (KW) rank-sum test was applied to the global segregation of each locus using the NPAR1WAY procedure of SAS. A first-type error rate of 0.005 was used for individual tests. Then, composite interval mapping (CIM; Jansen and Stam 1994; Zeng 1994) was performed on each framework parental map using model 6 of QTL Cartographer 1.13 g (Basten et al. 1994, 1997). The maximum number of cofactors was set to five, and the window size to 10 cM. Cofactors were selected by forward and backward stepwise regression. Permutation tests with 989–1,000 permutations were performed in order to determine the experimentwise significance levels (Churchill and Doerge 1994; Doerge and Churchill 1996). For each trait, the LOD corresponding to an experimentwise type-I error rate of 20% was chosen as the threshold to declare QTLs significant. The maximum LOD value was used for the estimate of QTL position, and a one-LOD support interval for the confidence interval.

Results and discussion

Mapping data set

The total number of loci in the final mapping data set was 352 (Table 1). Two out of the 47 SSR markers, VVMD14 and VMC2F10, showed more than two bands per individual. It was assumed that these markers re-

Table 1 Number of markers of each type in the final mapping data set, and their segregation type

Marker type	Maternal 1:1	Paternal 1:1	3:1	1:2:1 with no null allele	1:1:1:1 3 alleles	1:1:1:1 4 alleles	Total
AFLP	116	99	78				293
SSR	7	12	1	7	17	8	52
Isozyme	2	1					3
RAPD			2				2
SCAR				1			1
Color		1					1
Total	125	113	81	8	17	8	352

vealed two loci each, suffixed a and b. The 64 AFLP primer-pairs tested yielded 2–34 polymorphic markers. Eighteen combinations (list available from the authors upon request) were used to genotype the whole progeny, yielding a total of 349 markers. Fifty one markers were removed from the data set because of inconsistencies in the phase chosen by JOINMAP, revealed during the preliminary screening step. The peroxidase locus was also removed for the same reason. Five AFLP loci showing very strong segregation distortions ($P < 0.0005$) were also discarded, leaving a total of 293 AFLP loci in the final mapping data set. Berry color segregated 1:1 in the progeny. Re-coding of the three SSR markers segregating 1:2:1 with a null allele (ab × a0 and a0 × ab cases) yielded six loci segregating 1:1, three in each parent.

Genetic maps

The complete maternal map consisted of 157 markers (116 AFLPs, 38 SSRs, two isozymes and one SCAR) mapped on 22 linkage groups covering 767.4 cM, with an average interval length of 5.7 cM. The complete paternal map consisted of 144 markers (98 AFLPs, 43 SSRs, one isozyme, one SCAR, and color) mapped on 23 linkage groups covering 816.0 cM with an average interval length of 6.7 cM. There were one and two unlinked loci at LOD 4.0 for the maternal and paternal maps, respectively.

For the consensus map, all 352 loci were linked at LOD 4.0, but 51 could not be mapped with the mapping parameters we used in JOINMAP. The consensus map consisted of 301 markers mapped on 20 linkage groups, covering 1,002 cM with an average interval length of 3.6 cM (Fig. 1). Groups were numbered according to the reference SSR map of Riaz and Meredith (unpublished results), based on common markers. Some groups unlinked in our map were linked in the reference map (groups 1 and 3). Inversely, some groups unlinked in the reference map were linked in our map (groups 9–16 and 13–15). For groups x and y, there were no markers in common with the reference map.

Too few (only 17) pairs of linked SSR loci were available on both parental maps to allow a reliable comparison of recombination rates between parents. However, they were probably not very different, since marker order and distances were generally well-conserved between parental and consensus maps, with only one inversion on group 5 (Fig. 1).

Table 2 Distorted loci and significance level for the chi-square test of goodness-of-fit to the expected segregation ratio: $*P < 0.05$, $**P < 0.01$, $***P < 0.005$, $****P < 0.001$

Locus	Linkage group	Significance level	Expected segregation ratio
AAG/CTT-A	1	*	1:1
AAC/CAT-D	1	*	1:1
ACC/CTT-M	2	**	1:1
ACC/CTT-Y (unmapped)	2	**	1:1
AAG/CAA-G	4	**	3:1
ACA/CTT-C	4	**	1:1
ACA/CTG-K	6	****	1:1
AAG/CTT-I	6	**	1:1
ACA/CTG-L	6	*	1:1
PGM	7	*	1:1
ACA/CAA-V	8	*	3:1
ACA/CAT-D	8	*	1:1
AGC/CAA-R	8	**	1:1
AAG/CAA-K	8	*	1:1
AAG/CAA-L	8	*	1:1
AAG/CTA-W	9–16	***	1:1
ACA/CAT-P	10	*	1:1
ACA/CTC-M	19	*	1:1
ACA/CAT-W	x	*	1:1

Nineteen loci showed significant distortion in their global segregation (Table 2). Among loci for which segregation distortion could also be tested in both parents separately, two loci segregating 1:1:1:1 showed distorted segregation in one parent only (VMC3C11.1 for the female parent and VMC2E7 for the male parent), and one locus segregating 1:2:1 (VVMD34) showed distorted segregation in each parent. Only two regions of the genome exhibited small groups of loci with distorted segregation. Given that the offspring were issued from in vitro embryo rescue and that the percentage of ovules which yielded viable plantlets was low (approximately 13%; Bouquet and Davis 1989), a much higher level of segregation distortion could have been expected. This indicates that only very little viability selection occurred during the in vitro step.

AFLP markers provided a fast way to construct maps for QTL detection on the grapevine F_1 population MTP3140. However, they tended to show a clustered distribution, which has often been reported for maps of other plant species using *Eco*RI–*Mse*I AFLPs (Castiglioni et al.

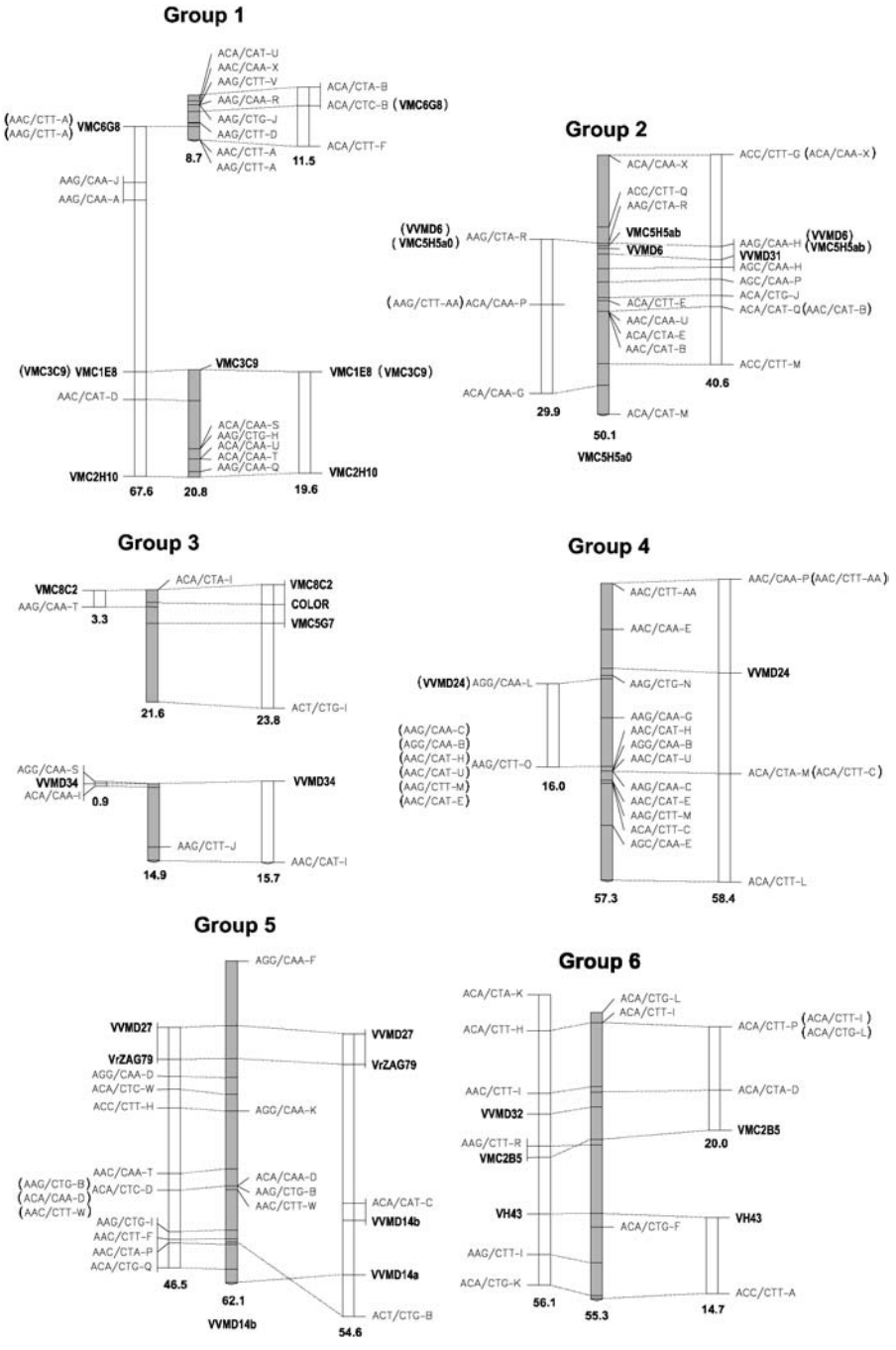


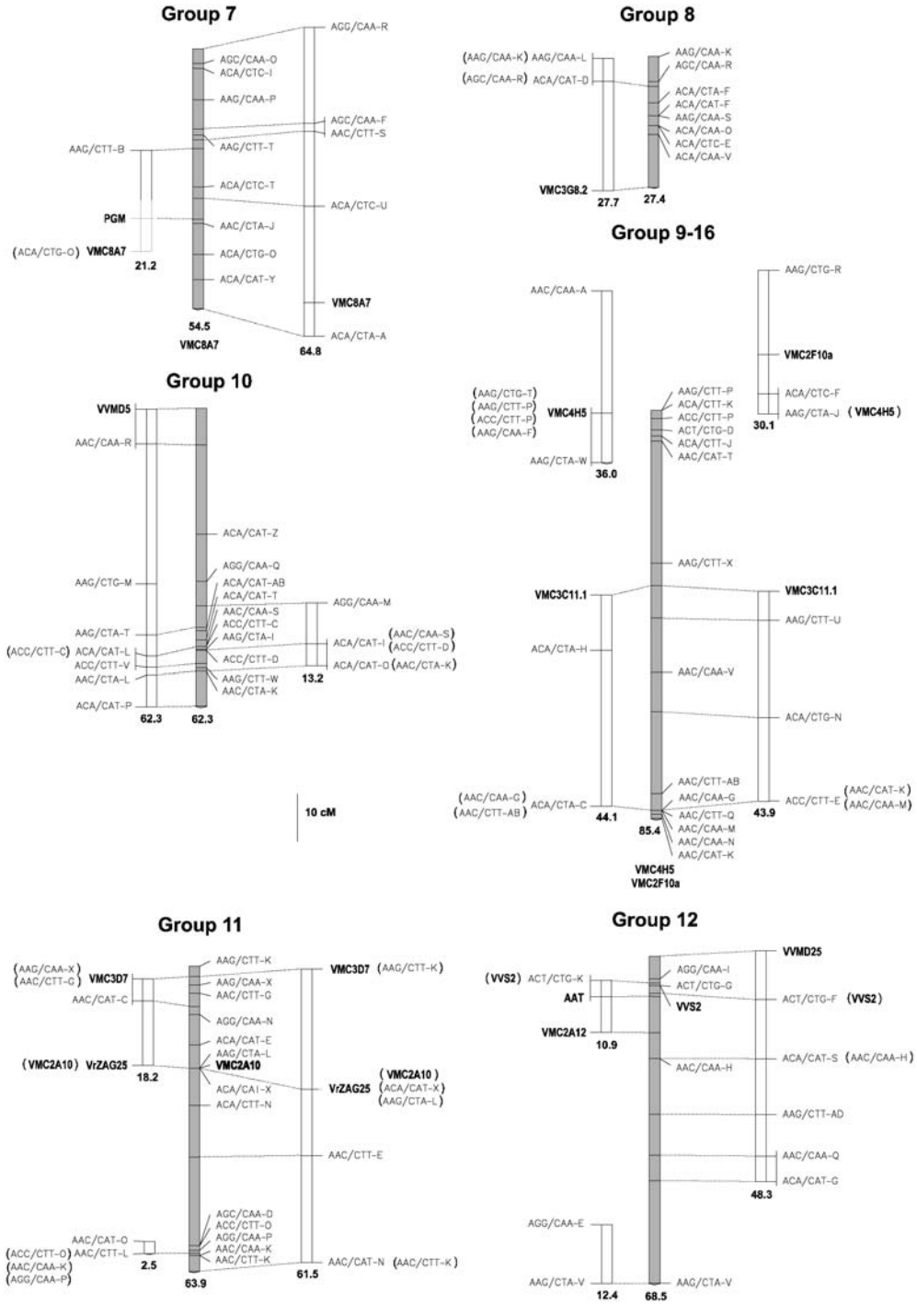
Fig. 1 Genetic linkage maps of the cross MTP3140. *Grey bars* are the linkage groups of the consensus map. *White bars* with names of markers on the left and on the right are the linkage groups of the maternal and paternal maps, respectively. *Dashed lines* connecting bars indicate markers common between groups. AFLP markers are named by the *Eco*RI-*Mse*I primer combination used for the amplification, followed by a *letter* indicating the fragment position on the gel (with alphabetical order ranking beginning with large fragments). The length of each linkage group (in Kosambi cM) is indicated below the group. On parental maps, the positions of markers for which the order was supported by a LOD value between 0.5 and 2.0 are connected by a *vertical bar*. Markers that could not be placed on parental maps with an order LOD support of at least 0.5 are given *in parentheses* beside the closest mapped marker. The *names* of unmapped but linked SSR loci are given below the consensus linkage groups

1999; Remington et al. 1999; Young et al. 1999; Ky et al. 2000; Lespinasse et al. 2000), although at least one such study detected no significant clustering (Arcade et al. 2000). It is usually suggested that these clusters correspond to highly methylated centromeric regions.

Genome length and coverage

Estimates of genome length were always slightly larger for the male, than for the female, parent (see Table 3), and with the modified Hulbert (MH) than with the Hulbert (H) method. They substantially decreased when computed without AFLPs, and so did their precision. Using the Kosambi mapping function, estimates ranged from 1,028 to 1,639 cM for the female genome, and from 1,330 to 1,908 for the male genome. Estimates of expected genome coverage under the assumption of a random distribution of markers varied from 84% to 92% for Kosambi estimations. It was slightly lower in the male than in the female parent, due to a smaller number of mapped markers and to a larger expected genome length. Total effective map lengths, excluding segments located more than 20 cM from the nearest markers, were

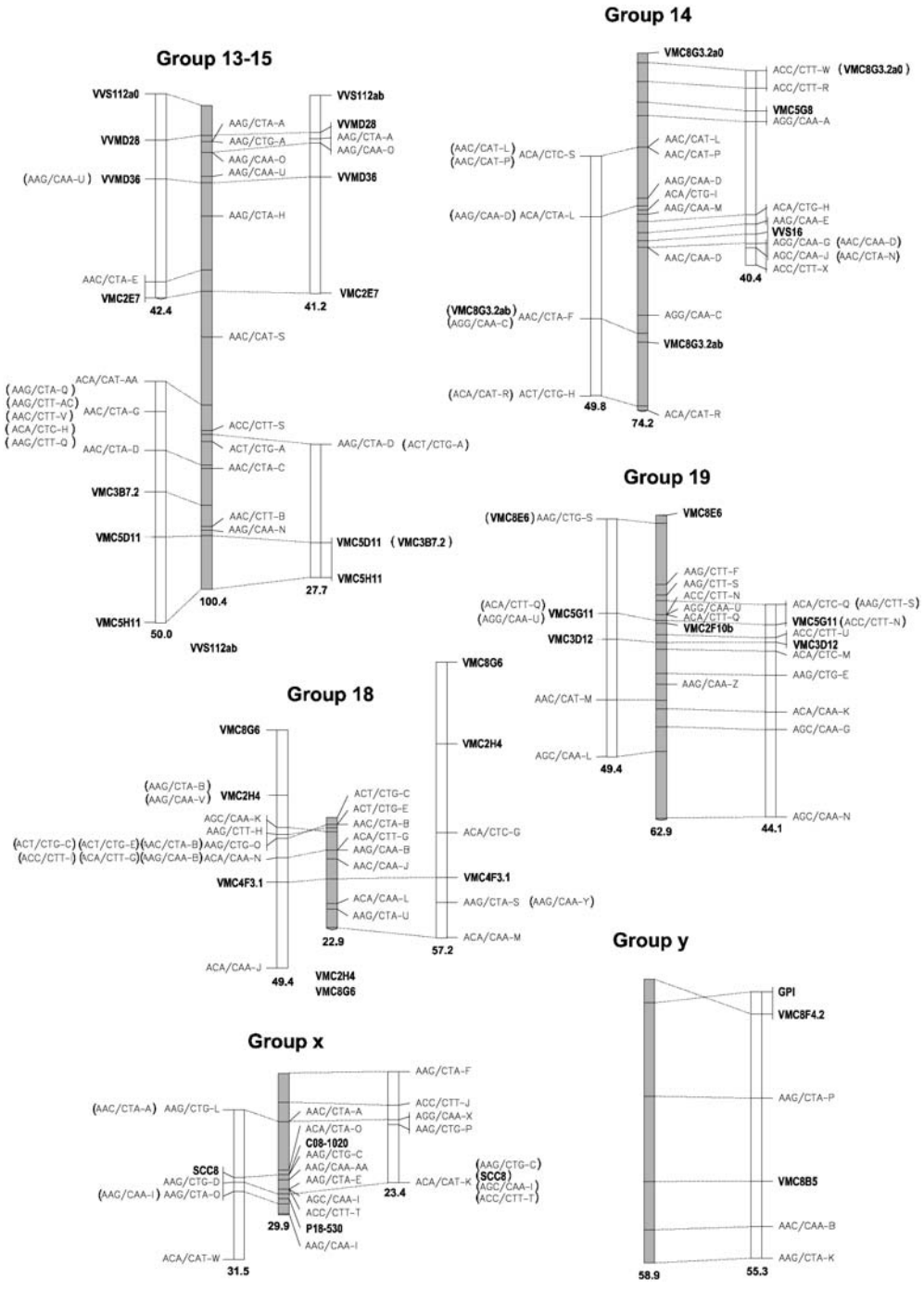
Fig. 1 (continued)



729.7 cM and 801.6 cM for the maternal and paternal parent, respectively.

In the consensus map, there was no unlinked locus at LOD 4.0, and only two gaps slightly larger than 20 cM, which suggests that this map was not very far from saturation. We a priori stopped adding new AFLP markers when the number of markers expected to saturate a 2,000-cM map was reached. However, effective genome coverage for Kosambi estimations was lower (59–71% and 60% for the maternal and paternal parent, respectively) than expected, due to clustering of the AFLPs. Moreover, effective genome coverage was lower for parental maps than for the consensus map, with smaller total map lengths and more gaps larger than 20 cM (six in each parental map). This was probably because only 46 and 42% of the loci of the consensus map could be mapped in the maternal and paternal parents, respectively. Nevertheless, with a genome coverage of at least 60%, parental maps proved a useful tool to rapidly perform a first QTL analysis.

Fig. 1 (continued)



Comparison of genome sizes

Estimates of genome length ranged between 1,028 and 1,908 cM Kosambi depending on the parent, the estimation method and the set of loci used for the estimation (Table 3). Chakravarti et al. (1991) showed that the Hulbert method (H) yields overestimates of genome length, even when the number of markers and/or individuals are increased. The modified Hulbert method (MH) is expected to give less-biased and thus lower estimates, with a bias further decreasing when many markers or individuals are used. For both H and MH methods, missing data are expected to largely inflate estimates, with a larger increase for H than for MH. Contrary to these expectations, we found higher estimates with MH than with H in this study. However, their confidence intervals were largely overlapping. Moreover, the violation of the assumption of equal chromosome length was shown to have little effect on the H method but its effect was not studied for the MH method. Since the theoretical effect

Table 3 Estimates of genome length and genome coverage for the parents of the mapping population. Genome length was estimated by both the Hulbert method (Hulbert et al. 1988) and a modified Hulbert method (method No. 3 of Chakravarti et al. 1991), either including all markers mapped or excluding AFLPs. Ninety five percent confidence intervals are given in brackets. Estimations of genome coverage were performed as described in Materials and methods. Genetic distances were obtained with both Haldane (Hal) and Kosambi (Kos) mapping functions

Parent		Hulbert method				Modified Hulbert method			
		Genome length with all markers	Genome length without AFLPs	Expected genome coverage (%)	Observed genome coverage (%)	Genome length with all markers	Genome length without AFLPs	Expected genome coverage (%)	Observed genome coverage (%)
Maternal	Hal	2,073 (1,908–2,268)	1,321 (954–2,145)	86	–	2,166 (1,994–2,370)	1,639 (1,184–2,662)	80	–
	Kos	1,576 (1,451–1,725)	1,028 (742–1,670)	92	71	1,639 (1,509–1,793)	1,240 (895–2,014)	88	59
Paternal	Hal	2,398 (2,178–2,669)	1,719 (1,266–2,677)	75	–	2,522 (2,290–2,807)	1,724 (1,270–2,685)	75	–
	Kos	1,826 (1,658–2,032)	1,330 (980–2,072)	84	60	1,908 (1,733–2,123)	1,341 (988–2,089)	84	60

of non-uniform distribution of markers is unknown, we performed genome length estimation both with and without AFLPs. According to Chakravarti et al. (1991), who recommended to use at least 20 loci, the number of loci remaining in the subset without AFLPs was large enough to yield a reliable estimate. We obtained smaller estimates without AFLPs, but with much-larger confidence intervals. To sum up, all the estimates of genome length given here should be considered as overestimates, and those obtained without AFLPs (1,028–1,341 cM Kosambi) are probably less biased than those obtained with all markers.

Large differences in total map length were observed between our map (1,002 cM Kosambi) and the Riaz and Meredith one (1,882 cM Kosambi; unpublished results). Moreover, most of the 24 distances between linked SSR pairs that were available for both maps were much smaller in ours than in the Riaz and Meredith one (Table 4). A few differences might be attributed to differences in locus order, but this is not the case for most of them. Statistical artifacts could not explain such differences in recombination rates, since the same version of the same mapping software was used, as well as the same mapping function (Kosambi). Differences in the number of loci with distorted segregations could not either, and for two reasons: (1) there were slightly more distorted loci (among loci segregating 1:1 or 1:1:1:1) in the Riaz and Meredith map (7% in the female parent and 15% in the male parent) than in ours (9% in the female parent and 4% in the male parent), which is expected to reduce rather than enlarge distances in their map compared to ours; (2) segregation distortions do not affect the modified LOD scores used by JOINMAP, which are derived from a chi-square test. To our knowledge, no result concerning the effect of missing data on genetic map distances is available in the literature.

Differences in recombination rates between individuals, either global or restricted to some particular genomic regions, have been reported for several plant and animal species (Karp and Jones 1983; Tulsieram et al. 1992; Williams et al. 1995; Causse et al. 1996 and references therein; Simianer et al. 1997, Ursul and Zhuchenko 1998). Different biological hypotheses could explain such discrepancies. First, differences in the inbreeding level of the parents might be involved, because it could lead to better DNA homology, and thus to more recombinations, within more-inbred parents than within less-inbred ones. A negative correlation between recombination level and genetic distance between parents has often

Table 4 Genetic distances between linked SSR pairs available for both our consensus map and the Riaz and Meredith one

SSR pair	Genetic distance (cM Kosambi)		Linkage group
	Our map	Riaz and Meredith map	
VMC2H10-VMC3C9	20.8	19.5	1
VMC2H10-VMC1E8	20.8	54.5	1
VMC3C9-VMC1E8	0.0	35.4	1
VVMD6-VMC5H5ab	0.5	71.9	2
VVMD6-VVMD31	1.1	120.2	2
VMC5H5ab-VVMD31	1.6	48.4	2
COLOR-VMC5G7	4.0	18.1	3
VVMD27-VrZAG79	6.4	44.8	5
VVMD14A-VrZAG79	43.2	98.3	5
VVMD14A-VVMD27	49.6	53.7	5
VMC2B5-VVMD32	6.2	73.1	6
VMC3D7-VrZAG25	19.2	65.4	11
VMC3D7-VMC2A10	19.2	33.5	11
VrZAG25-VMC2A10	0.0	98.9	11
VMC2A12-VVS2	9.9	32.5	12
VVS2-VVMD25	6.1	37.8	12
VVMD25-VMC2A12	16.0	70.3	12
VVMD28-VVMD36	9.9	11.9	13–15
VVMD28-VMC2E7	32.4	49	13–15
VVMD36-VMC2E7	22.5	37.1	13–15
VMC5H11-VMC5D11	11.1	16.4	13–15
VMC5H11-VMC3B7.2	17.4	53.3	13–15
VMC5D11-VMC3B7.2	6.3	69.7	13–15
VMC5G11-VMC3D12	4.5	6.4	19

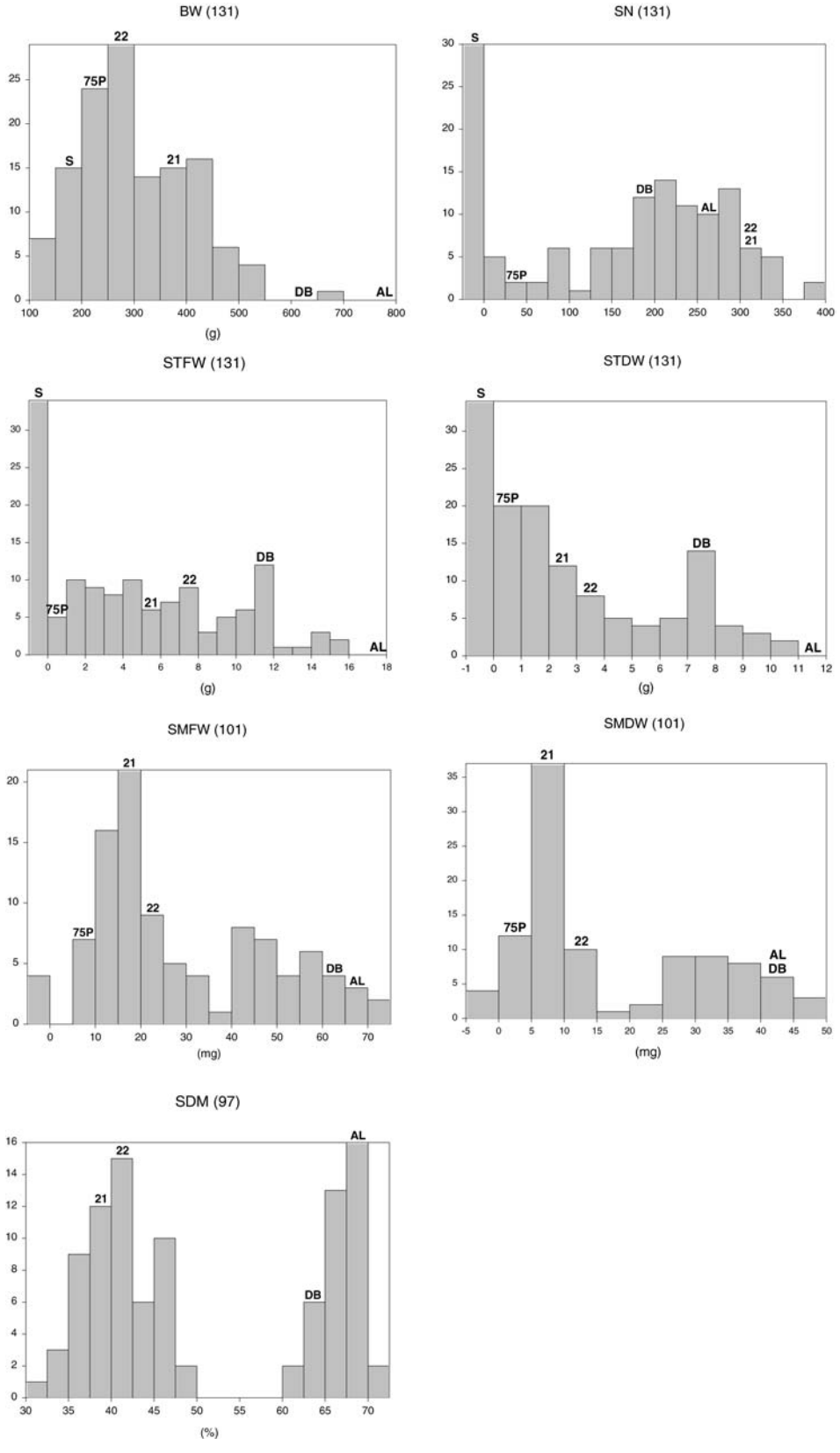


Fig. 2 Distributions of phenotypic data for all traits in 1995. Sample sizes are given *in brackets* for each trait. The *names* of parents (22 = 2223–27, 21= 2121–30) and grand-parents (DB = Dattier de Beyrouth, 75P = 75 Pirovano, AL = Alphonse Lavallée, S = Sultanine) are indicated above the classes containing their value

been mentioned in crosses of inbred species (Paterson et al. 1990; Causse et al. 1994), although this is not systematic (Causse et al. 1996). In heterozygous species such as *Vitis vinifera*, the genetic distance between grand-parents (as measured by the heterozygosity of parents) should be considered instead of the genetic distance between parents. However, out of the 73 loci that were tested on the four parents of our cross and the Riaz and Meredith cross, 84% were heterozygous for the female and 90% for the male parent of our cross, and 78% were heterozygous for the female and 75% for the male parent of the Riaz and Meredith cross. These levels of heterozygosity seem too close to be at the origin of the large differences in map distances observed between the two maps. Second, evidence of the genetic control of recombination rate, chiasma frequency, or chiasma distribution has been found in some plant species (Pagliarini 1980; Karp and Jones 1983; Abirached-Darmency et al. 1992; Tulsieram et al. 1992). Third, some environmental factors might influence chiasma frequency (Powell and Nilan 1963; Corazza-Nunes et al. 1993). For the last two hypotheses, no data are currently available for grapevine. It should be noted that the total map length of the *Vitis* maps published so far (1,196 and 1,477 cM in Lodhi et al. 1995; 1,199 cM and 1,470 cM in Dalbo et al. 2000) were intermediate between the Riaz and Meredith one and ours. Given their extent, differences in recombination rates observed not only within the *Vitis* genus but even within the *Vitis vinifera* species may have some negative consequences on the potential efficiency of MAS. Therefore, it would be useful to know more about the major determinants of these differences.

Distribution of phenotypic data

Distributions of 1995 phenotypic data are shown in Fig. 2. Distributions for 1994 and 1996 (data not shown) were very similar. Parental values were always very close to each other. They were intermediate between the values of seeded (Alphonse Lavallée and Dattier de Beyrouth) and seedless (75 Pirovano and Sultanine) grand-parents for all traits except seed number. Transgressive segregations were observed for all traits. No distribution was normal. In the progeny, there was a large class of completely seedless genotypes, i.e. with no seeds at all. These were most-probably not parthenocarpic but true stenospermocarpic, as strongly suggested by the relatively uniform and medium size of their berries, the absence of parthenocarpic genotypes among the ascendants of the progeny, and the difficulty to detect and measure the smallest seed traces. Therefore we included them in the analysis. This yielded a zero class for SN, STFW and STDW. Since it was not possible to determine SDM, SMFW and SMDW for this class, these genotypes were not included on the charts. These three traits showed bimodal distributions.

Methodology of QTL detection

For CIM, the LOD threshold for an experimentwise type-I error rate of 20%, as determined through 989–1,000 permutations, varied from 2.56 to 3.12 depending on trait and year. The choice of a high experimentwise type-I error rate for our QTL detection can be discussed. Such a high rate is commonly chosen for QTL detection experiments (e.g. Melchinger et al. 1998), and is even advised for exploratory QTL detections (Beavis 1998). Moreover, *a priori* we knew that at least one major QTL was segregating in the progeny studied, which was expected to lower the power detection of minor QTLs. So we chose to allow for a relatively high probability of false-positive QTLs.

QTL detection was performed both with a non-parametric Kruskal-Wallis test (KW; data not shown) and CIM (results summarized in Table 5). Although CIM is expected to be more-powerful, we also used KW, first because we could perform CIM detection only on parental maps, which were less-saturated than the consensus map, and second because in some cases the residuals obtained with CIM were not normally distributed, which might induce nonsense results. In addition to the major QTLs, three minor QTLs were detected with both methods (on groups 1, 12 and 13–15). Three putative minor QTLs were detected with CIM only (on groups 6, 11 and 13–15), probably due to the larger power of CIM. Conversely, several putative minor QTLs were detected with KW only (on groups 4, 5, 7, 9–16 and 13–15). Some of them (on group 4), could not have been detected with CIM because they were located in a part of the genome not covered by the maternal map. Other ones (on groups 5, 7 and 9–16) were more difficult to explain because the loci significant with KW segregated in one parent only and, in this case, CIM should be more powerful than a non-parametric method. Lastly, the locus significant with KW on group 13–15 segregated in both parents and, in such a case, detection from the consensus map might be more powerful than detection from each parental map. Therefore, the use of both methods was useful in our case to obtain reliable results. That is why we distinguished between ‘QTLs’ (detected with both methods and shown on Fig. 3) and ‘putative QTLs’ (detected by one method only; data not shown, since they still have to be confirmed).

QTLs for seedlessness and berry weight

For berry weight (BW), we found one QTL with large effect for all 3 years (R^2 = 25–38) on group x, and one QTL with small effect for 1994 on group 12 (R^2 = 6). On group x, two LOD peaks were found on the maternal map and one on the paternal map, but confidence intervals were overlapping. For this QTL, the effect was larger in the maternal (R^2 = 30–38) than in the paternal parent (R^2 = 25–29).

For seed number (SN), we found one QTL on group x with a large effect in the maternal parent for all 3 years

Table 5 Location, significance and effect of individual QTLs detected using QTLCartographer. Group = linkage group, Parent = parent in which the QTL was detected (M for maternal and P for paternal), Peak = QTL position as estimated by the location of the local LOD maximum in cM from the 'top' of the linkage group, Nearest marker = marker nearest to the QTL position, Interval = confidence interval of QTL position in cM, LOD = LOD value at QTL position, LOD threshold = experimentwise LOD threshold for type-I with t for the 'top' of the linkage group, and b for the 'bottom' error rates of 20% and 5% based on 1,000 permutations, *S* = test statistic for the normality of residuals under the H1 hypothesis that there is a QTL at this position (* for $P < 0.05$ and ** for $P < 0.01$), R^2 = proportion of the total phenotypic variance explained by the QTL, *a* = estimate of the additive effect (effect of substituting one parental allele for the other) on the trait

Trait	Year	QTL location					LOD	LOD threshold		*S*	QTL effect	
		Group	Parent	Peak	Nearest marker	Interval		$\alpha = 0.20$	$\alpha = 0.05$		R^2	*a*
BW	1994	x	M	14	SCC8	8–16	13.61	2.62	3.66	2.34	36	–147
		x	M	21	AAG/CTA-O	0–29	10.11	2.62	3.66	4.03	34	–142
		x	P	22	ACA/CAT-K	14–b	9.49	2.64	3.44	1.98	29	–128
		12	M	2	AAT	t–b	2.65	2.62	3.66	0.71	6	57
	1995	x	M	14	SCC8	8–16	14.32	2.56	3.35	0.28	38	–135
		x	M	21	AAG/CTA-O	2–27	11.76	2.56	3.35	1.95	36	–132
		x	P	18	ACA/CAT-K	10–b	9.13	2.76	3.66	13.32**	27	–114
	1996	x	M	14	SCC8	8–16	10.33	2.65	3.59	2.23	30	–173
		x	M	23	AAG/CTA-O	2–29	8.52	2.65	3.59	3.16	31	–174
		x	P	16	AAG/CTG-P	7–b	6.38	2.77	3.50	3.02	25	–156
SN	1994	x	M	12	SCC8	6–16	17.18	2.70	3.44	9.15*	51	–178
		x	M	21	AAG/CTA-O	2–25	13.31	2.70	3.44	5.45	48	–174
		x	P	22	ACA/CAT-K	16–b	4.88	2.82	3.52	1.86	15	–95
	1995	x	M	12	SCC8	8–16	14.86	2.65	3.54	2.63	41	–157
		x	M	19	AAG/CTA-O	2–25	11.57	2.65	3.54	0.79	33	–139
		1	M	37	VMC1E8	23–53	3.70	2.74	3.68	2.49	11	–80
	1996	x	M	12	SCC8	6–16	12.92	2.75	3.68	10.04**	41	–146
		x	M	19	AAG/CTA-O	2–25	9.75	2.75	3.68	4.76	32	–127
		x	P	22	ACA/CAT-K	12–b	3.17	2.80	3.69	1.52	11	–74
STFW	1994	x	M	14	SCC8	10–16	14.89	2.61	3.57	43.33**	41	–6.21
		x	M	21	AAG/CTA-O	17–29	11.62	2.61	3.57	57.89**	40	–6.13
		x	P	22	ACA/CAT-K	16–b	9.28	2.68	3.55	24.95**	27	–5.15
	1995	x	M	14	SCC8	8–16	18.83	2.65	3.54	2.02	49	–6.47
		x	M	19	AAG/CTA-O	2–27	13.84	2.65	3.54	2.54	43	–6.02
		x	P	20	ACA/CAT-K	14–b	8.91	2.77	3.71	1.96	26	–4.84
	1996	x	M	12	SCC8	4–16	14.17	2.76	3.67	2.27	46	–7.45
		x	M	21	AAG/CTA-O	t–27	9.74	2.76	3.67	3.23	39	–6.84
		x	P	18	ACA/CAT-K	10–b	9.29	2.77	3.70	1.13	35	–6.46
STDW	1994	x	M	14	SCC8	8–16	12.43	2.60	3.48	54.88**	34	–3.70
		x	M	21	AAG/CTA-O	2–29	10.02	2.60	3.48	63.58**	32	–3.62
		x	P	22	ACA/CAT-K	14–b	10.06	2.70	3.61	64.64**	30	–3.56
	1995	x	M	14	SCC8	8–16	16.80	2.64	3.65	3.59	44	–4.17
		x	M	19	AAG/CTA-O	0–27	12.60	2.64	3.65	6.66*	39	–3.91
		x	P	20	ACA/CAT-K	14–b	11.06	2.72	3.65	1.60	31	–3.54
	1996	x	M	14	SCC8	8–16	12.69	2.67	3.77	5.23	40	–4.14
		x	M	19	AAG/CTA-O	0–29	8.81	2.67	3.77	9.20*	32	–3.69
		x	P	22	ACA/CAT-K	14–b	11.05	2.82	3.79	2.91	37	–3.99
SDM	1994	x	M	29	ACA/CAT-W	16–b	6.14	2.86	3.72	4.02	27	–14.5
		x	P	18	ACA/CAT-K	12–b	10.13	2.91	3.87	3.75	40	–16.3
		13–15	P	0	AAG/CTA-D	t–10	3.15	2.91	3.87	1.94	10	–8.3
	1995	x	M	14	SCC8	6–16	7.31	2.94	3.88	3.08	29	–16.4
		x	M	27	ACA/CAT-W	17–b	6.20	2.94	3.88	3.47	33	–16.4
		x	P	18	ACA/CAT-K	10–b	8.30	2.97	3.95	5.23	37	–16.9
	1996	x	M	14	SCC8	6–17	5.18	2.98	3.94	3.25	26	–14.3
		x	M	27	ACA/CAT-W	2–b	4.08	2.98	3.94	4.77	27	–13.9
		x	P	10	AGG/CAA-X	7–b	7.58	3.12	4.25	0.22	32	–14.7
		x	P	16	AAG/CTG-P	4–b	7.28	3.12	4.25	1.03	35	–15.8
SMFW	1994	x	M	14	SCC8	6–16	6.94	2.94	3.95	1.00	25	–16.8
		x	M	27	ACA/CAT-W	2–b	5.90	2.94	3.95	1.20	26	–16.7
		x	P	18	ACA/CAT-K	10–b	5.36	2.87	3.87	0.12	23	–14.2
	1995	x	M	14	SCC8	6–16	7.39	2.82	3.83	2.07	26	–22.5
		x	M	31	ACA/CAT-W	23–b	7.46	2.82	3.83	2.61	31	–24.0
		x	P	18	ACA/CAT-K	12–b	8.56	3.00	3.85	0.67	38	–24.7
	1996	x	M	31	ACA/CAT-W	16–b	3.05	2.93	3.91	6.66*	19	–20.7
		x	P	12	AAG/CTG-P	7–20	7.00	2.99	4.10	0.21	26	–23.3

Table 5 (continued)

Trait	Year	QTL location					LOD	LOD threshold		*S*	QTL effect	
		Group	Parent	Peak	Nearest marker	Interval		$\alpha = 0.20$	$\alpha = 0.05$		R^2	*a*
SMDW	1994	x	M	29	ACA/CAT-W	16–b	5.82	2.80	3.79	2.07	25	–12.1
		x	P	18	ACA/CAT-K	10–b	7.19	2.99	3.91	0.28	29	–12.0
	1995	x	M	14	SCC8	6–16	7.97	2.85	3.79	2.90	28	–17.6
		x	M	31	ACA/CAT-W	21–b	7.12	2.85	3.79	2.79	31	–17.8
		x	P	18	ACA/CAT-K	12–b	9.29	2.90	3.83	0.53	41	–19.0
	1996	x	M	14	SCC8	0–17	3.90	3.03	3.99	4.29	17	–13.6
		x	P	10	AGG/CAA-X	7–20	8.62	3.02	4.05	0.58	48	–18.0

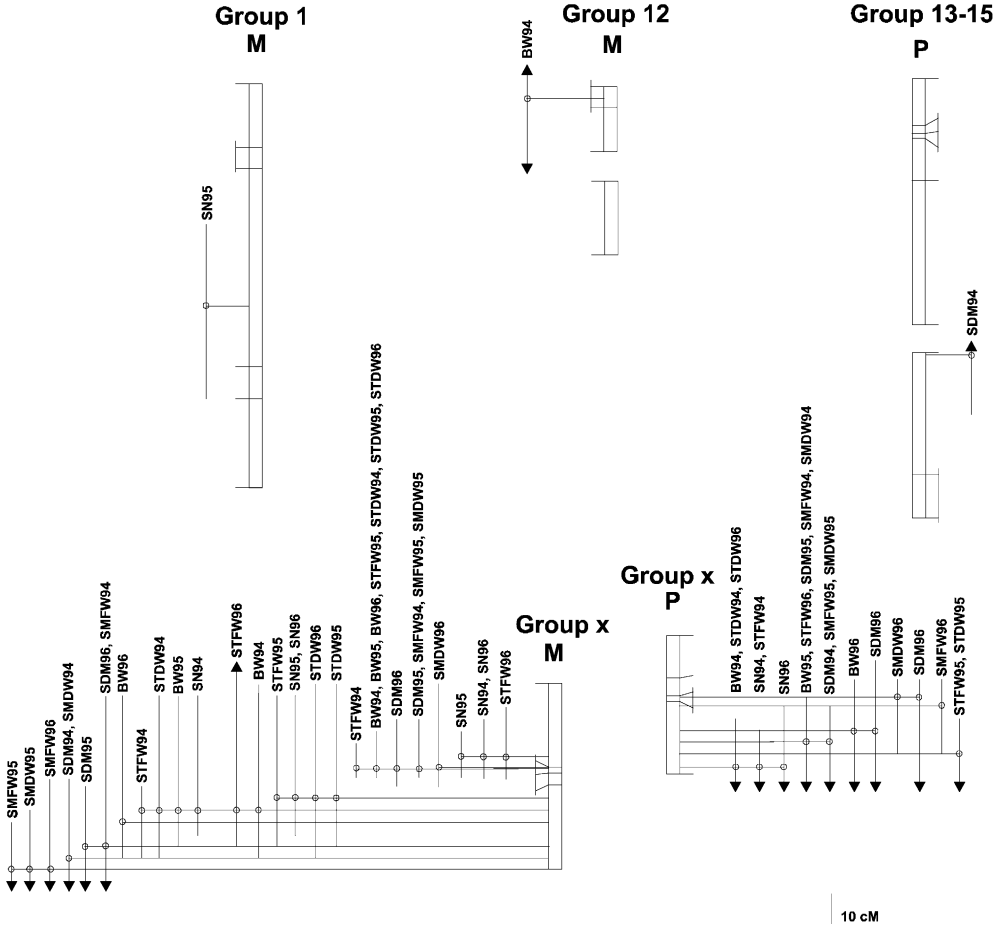


Fig. 3 QTLs for berry weight (*BW*) and seedlessness traits (*SN*: seed number; *STFW*: seed total fresh weight, *STDW*: seed total dry weight; *SDM*: seed dry matter; *SMFW*: seed mean fresh weight; *SMDW*: seed mean dry weight), detected by Composite Interval Mapping on parental maps. *M*: maternal linkage group, *P*: paternal linkage group. One-LOD support confidence intervals are represented by *vertical bars*, with an *arrow* at the end whenever extending beyond the end of the linkage group. QTL positions are represented by *horizontal bars*

(R^2 = 32–51), and a smaller effect in the paternal parent, for 1994 and 1996 only (R^2 = 11–15). Here again, there were several LOD peaks, but confidence intervals were overlapping. Another QTL, with small effect (R^2 = 11), was detected on group 1 in 1995 only.

For seed total fresh and dry weights (STFW and STDW, respectively) in the 3 years, one QTL with large effect (R^2 = 26–49) was found on group x, with several LOD peaks but overlapping confidence intervals. However, there was an exception for STFW94, where two distinct QTLs (non-overlapping confidence intervals) were found in the maternal parent. The effect of these major QTLs tended to be larger in the maternal parent (R^2 = 32–49) than in the paternal one (R^2 = 26–37).

For seed percent dry matter (SDM), a major QTL was again detected on group x for all 3 years, as well as a second one (non-overlapping confidence intervals) for 1995. But for this trait, the maternal effect (R^2 = 26–33) tended to be smaller than the paternal effect (R^2 = 32–40). A QTL with a small effect (R^2 = 10) was detected on group 13–15, in 1994 with CIM and in 1996 with KW.

For seed mean fresh and dry weights (SMFW and SMDW, respectively), one major QTL was detected on group x in 1994 and 1996, as well as a second one (non-overlapping confidence intervals) in 1995. Here again, the maternal effect (R^2 = 17–31) tended to be smaller than the paternal effect (R^2 = 23–48).

In summary, QTL detection in the MTP3140 progeny revealed one genome region, on group x, with a major effect on both berry weight and seedlessness in all years, as well as three regions on distinct groups, with minor effects on either berry weight or seedlessness in 1 or 2 years (Fig. 3). The major region on group x included the two RAPDs found to be tightly linked to the *SdI* gene by Lahogue et al. (1998), as well as the SCAR marker SCC8 developed by these authors. But, for a few traits (STFW94, SDM95, SMFW95 and SMDW95), we found some evidence for the presence of two distinct QTLs segregating in the maternal parent in this region, one close to *SdI*, and the other one 10–20 cM apart from *SdI*. Reducing the CIM window size to 5 cM yielded exactly the same results for significant QTLs. For most other traits, there were two LOD peaks, but confidence intervals were overlapping. This result should be checked on a larger population in order to obtain a better precision of QTL location. Alternatively, other estimation methods of confidence intervals for QTL position could be used, since for CIM their computation is still an unsolved problem (Visscher et al. 1996). Therefore, the LOD support method was used here only as an approximation, but Kao et al. (1999) showed that at least one other method (using asymptotic standard deviation) can yield much narrower intervals than the LOD support method. Moreover, individual effects of the QTLs detected on this linkage group ranged between 11 and 51%, and were thus always lower than the effects of SCC8 alone (65–89%), as reported by Lahogue et al. 1998. This is another argument in favor of the actual presence of two distinct QTLs for most traits on this linkage group. On the other hand, the possibility of erroneous locus order on the maternal map in this region cannot be totally ruled out, since the two LOD peaks found with CIM were also always detected with Simple Interval Mapping (data not shown). To check this, it is necessary to map more markers in this region, so that different subsets of loci can be used to construct maps and carry out QTL detection. This would also allow us to look for QTLs segregating in the paternal parent in this region.

Highly significant ($P < 0.5$) positive phenotypic correlations were observed within years between berry weight and all seedlessness traits, and also between most seedlessness traits (Table 6). These correlations were at least partly of genetic origin, since on group x, most of the major QTLs detected for berry weight and for the different seedlessness traits were co-located. Such genetic correlations might be due either to pleiotropy or to tight linkage. The main hypothesis in favor of pleiotropy is physiological. It involves the production of gibberellins by the seeds, which affect berry growth. Performing multiple interval mapping (MIM) analysis might help separating linked QTLs since it is expected to yield more-precise estimates of QTL location than CIM analysis (Kao et al. 1999).

For the QTLs located close to *SdI* on group x, there were differences in R^2 between parents, with variations among traits in the extent and direction of these differences but relative stability among years. Maternal QTLs had larger effects than paternal ones for BW, SN, STFW and STDW. Conversely, maternal QTLs had smaller effects than paternal ones for SDM, SMFW and SMDW. Thus, two main groups of traits could be distinguished based on this criterion: berry weight and seed traits dependent on seed number (SN, STFW, STDW) on the one hand, and seed traits independent on seed number (SDM, SMFW, SMDW) on the other hand. Assuming pleiotropy, three hypothesis may explain such differences. First, these major QTLs might interact with minor ones, or with the genetic background, in a different way for the different traits. MIM analysis would allow one to test for epistasis between QTLs (Kao et al. 1999), and in particular for interactions between major and minor QTLs which were part of the assumption of Bouquet and Danglot (1996). Second, the direct proportionality relationships between some traits due to their very definition (e.g. between SDM and STFW or STDW) might be at the origin of the variation in parental R^2 differences among traits. Of course, mathematically independent traits such as berry weight and seedlessness traits are not concerned by this hypothesis. Third, there might be a second QTL linked to the first major one found near *SdI*, segregating in the paternal parent but undetected in this study (e.g. in the part of group x that was not covered by the paternal map), and with differing effects for these traits. For example, if the paternal QTL detected in this study had a strong pleiotropic effect on both SN and SDM, the presence of another paternal QTL for SN only, linked in repulsion and undetected in this study, could have led to a lower resulting paternal effect for SN than for SDM.

The effects of the major QTLs on group x, as measured by R^2, varied substantially among years (difference up to 19). These variations could be attributed to year effects, which were highly significant ($P < 0.001$) for all

Table 6 Phenotypic correlations between traits (Pearson correlation coefficients), within each year. All correlations were significant ($P < 0.5$), except those indicated by NS

		SN	STFW	STDW	SDM	SMFW	SMDW
BW	1994	0.67	0.84	0.82	0.65	0.76	0.75
	1995	0.47	0.77	0.77	0.59	0.75	0.73
	1996	0.61	0.77	0.76	0.54	0.65	0.64
SN	1994		0.78	0.69	0.41	0.43	0.41
	1995		0.69	0.55	–0.08 NS	0.10 NS	0.03 NS
	1996		0.77	0.65	0.25	0.22	0.19 NS
STFW	1994			0.98	0.78	0.90	0.90
	1995			0.97	0.74	0.87	0.84
	1996			0.97	0.74	0.85	0.84
STDW	1994				0.84	0.91	0.94
	1995				0.86	0.91	0.92
	1996				0.86	0.86	0.90
SDM	1994					0.83	0.91
	1995					0.86	0.91
	1996					0.76	0.88
SMFW	1994						0.98
	1995						0.99
	1996						0.97

traits, except STDW ($P > 0.5$). They could also be due to interactions between genetic and environmental effects, but such interactions could not be tested in our unreplicated field design. On group x, QTL effects also varied widely among traits, which probably reflected differences in heritability, i.e. in the relative effect of the environment on total phenotypic variation, in addition to the potential causes discussed above.

In addition to the major QTLs on group x, minor QTLs were detected on three distinct groups, for BW (in 1 year), SN (in 1 year), and SDM (in 2 years) on groups 12, 1 and 13–15, respectively. No co-location between QTLs for berry weight and seedlessness was found, suggesting that the genetic correlations between these traits were only due to the major QTLs of group x. However, it is also possible that the minor QTLs for seedlessness had pleiotropic effects, but that the indirect effect on berries through seeds was too weak to allow detection of QTLs for berry weight.

Bouquet and Danglot (1996) assumed that three minor genes were involved in the determinism of seedlessness, whereas we detected only two genomic regions. This suggests that one minor QTL for seedlessness might not have been detected in this study, due to incomplete genome coverage.

Striem et al. (1994, 1996) found RAPD markers with significant effects on several seedlessness subtraits, mainly total and mean seed fresh weight per berry. Additive effects reported for these markers had the same order of magnitude as those found in our study for the major QTLs on group x. But testing the effects of unmapped markers did not allow us to estimate the number and location of QTLs involved in seedlessness. The construction of the genetic maps reported here allowed a more-systematic detection and characterization of these QTLs.

Minor QTLs were detected only in a single year, except the QTL for SDM on group 13–15. This might be due to year effects and/or to genotype × year interactions. Alternatively, this might be due to a limited detection power because of the combination of a moderate population size with at least one major QTL responsible for most of the total phenotypic variation. In this case, a larger population fixed for this (these) major QTL(s) would be required to better describe the minor genes involved in berry weight and seedlessness.

Conclusion

We developed genetic maps covering the major part of the genome for a *V. vinifera* cross between two partially seedless genotypes and used them to carry out a first QTL detection for seedlessness and berry weight. For more complete QTL detection, further saturation of these maps is required. It will be easily achieved thanks to the current development of grapevine reference microsatellite maps, avoiding the drawbacks of AFLP randomness and clustering. Although incomplete, the maps presented in this study have already allowed us to detect several QTLs for stenospermocarpic seedlessness and berry weight, confirming the existence of one major QTL at the same location for all traits studied, suggesting the presence of a second major QTL for some of these traits on the same linkage group, and confirming the existence of at least three independent minor QTLs (two for seedlessness and one for berry weight). In a marker-assisted selection perspective, our results already suggest two possibilities to try dissociating the unfavorable correlation between berry weight and seedlessness. First, a few minor QTLs specific for berry weight or seedlessness could be used. Second, parental differences in the effect of major QTLs could be exploited whatever the exact underlying determinism. Further information on the nature of genetic correlation between major QTLs for berry weight and seedlessness is needed in order to be able to fully control recombinations. However, the extent of dissociation of these traits will probably still be limited based on the present results alone. It is likely that variations of berry weight independent of seedlessness will also have to be used in order to simultaneaously obtain satisfying levels of seedlessness and berry weight.

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