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RFLP mapping of the *sugary enhancer1* gene in maize

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Abstract RFLP marker data from an $F_{2:3}$ population derived from a cross between a *sugary1* (*su1*) and a *sugary enhancer1* (*su1*, *sel*) inbred were used to construct a genetic linkage map of maize. This map includes 93 segregating marker loci distributed throughout the maize genome, providing a saturated linkage map that is suitable for linkage analysis with quantitative trait loci (QTL). This population, which has been immortalized in the form of sibbed $F_{2:3}$ families, was derived from each of the 214 F_2 plants and along with probe data are available to the scientific community. QTL analysis for kernel sucrose (the primary form of sugar) concentration at 20 days after pollination (DAP) uncovered the segregation of seven major QTL influencing sucrose concentration; a locus linked to umc36a described the greatest proportion of the variation (24.7%). Since maltose concentration has previously been reported to be associated with the *sel* phenotype, an analysis of probe associations with maltose concentration at 40 DAP was also conducted. The highly significant association of umc36a with maltose and sucrose concentrations provided evidence that this probe is linked to *sel*. Phenotypic evaluation for the *sel* genotype in each $F_{2:3}$ family enabled us to map the gene 12.1 cM distal to umc36a. In contrast to previous work where *sel* was reported to be located on chromosome four, our data strongly suggest that the *sugary enhancer1* locus maps on the the distal portion of the long arm of chromosome 2 in the maize genome.

Key words Sweet corn · *sel* · Sucrose · Quality

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Introduction

The traditional sweet corn hybrids grown in the U.S. are homozygous for the recessive endosperm mutant allele, *sugary1* (*su1*). The *su1* mutation has reduced levels of a starch debranching enzyme, thereby creating a highly branched and water-soluble form of starch, known as phytoglycogen, that imparts a creamy texture to the kernel (Dickinson et al. 1983; Pan and Nelson 1984). The rapid loss of sugars and moisture concentration of maturing *su1* kernels (Carey et al. 1982) restricts the duration of the post-harvest eating quality and allows only a narrow harvest window for profitable production by the sweet corn processing industry (Marshall 1987). This limitation has stimulated research into the commercial use of other maize endosperm carbohydrate mutations. There are at least 13 distinct single gene endosperm mutations in *Zea mays* L. that produce qualitative and quantitative differences in maize kernel carbohydrate metabolism (Hoistington et al. 1988). Nearly all of these mutations are recessive and result in amplified sucrose concentrations in immature kernels at the expense of reduced starch. Because of their superior post-harvest quality, three mutations, *shrunken2* (*sh2*), *brittle2* (*bt2*), and *sugary enhancer1* (*sel*) are now used in commercial production (Boyer and Shannon 1983). The mutants *sh2* and *bt2* are lesions in the two subunits of the gene for ADPG-pyrophosphorylase, an important enzyme in the conversion of sucrose to substrates for starch synthesis (Bhave et al. 1990; Shaw and Hannah 1992). The biochemical lesion of the *sel* gene is, as yet, unknown.

The phenotype of the *sel* gene was first reported 20 years ago (Gonzales et al. 1974, 1976). It was described as a single recessive modifier of *su1* (Ferguson et al. 1978, 1979). In the homozygous state in immature *su1* kernels, *sel* increases total sugars levels by 50–100% at the expense of starch content without a significant reduc-

tion in phytoglycogen concentration (Gonzales et al. 1976; La Bonte and Juvik 1990). Hence, *su1se1* kernels at harvest maturity have the desirable texture characteristics of standard *su1* sweet corn with the enhanced sugar concentration of super sweet cultivars (La Bonte and Juvik 1990). Significantly elevated maltose concentrations in mature-dry *su1se1* kernels have also been observed (Ferguson et al. 1979). Phenotypic selection criteria used for the identification of homozygous *su1se1* kernels in segregating populations include lighter yellow kernel color, slower rates of kernel moisture loss and a more finely wrinkled pericarp in mature dry kernels (Ferguson et al. 1979; La Bonte and Juvik 1990). However, phenotypic selection based on these traits is problematic, due to the impact of background and kernel maturity. The most reliable procedure by which to identify the *se1* phenotype is by sugar analysis of kernels harvested at 20 days after pollination (DAP). This procedure is destructive, time-consuming, and will only identify kernels homozygous for the *se1* gene.

Chromosomal localization and marker-tagging of the *se1* gene in the maize genome will provide plant breeders and researchers with a tool to select for *se1* and will help in the understanding of the physiological role of this commercially desirable gene. In a previous report, maize B-A translocations (Beckett 1978) in combination with single-kernel sugar analysis were used to localize the *se1* gene on the long arm of chromosome four (La Bonte and Juvik 1991). The study presented here uses restriction fragment length polymorphism (RFLP) analysis of a population segregating for the *se1* to more precisely map the location of this gene in the maize genome.

Materials and methods

Plant material

IL731A*su1se1* is a maize inbred homozygous for both *su1* and *se1* (Carey et al. 1982). This inbred was hybridized with the *su1* inbred W678*su1Se1*, which is homozygous for wild-type allele (*Se1*) at the *sugary enhancer* locus. Self-pollination of the F_1 yielded F_2 plants that were individually selfed to generate F_3 seeds. One hundred seeds per family of 214 $F_{2:3}$ families and both parents were sown in flats containing a 1:1:1:2 soil mixture of soil:peat:perlite:vermiculite in May of 1992. After emerging, seedlings were hardened off for 10 days and transplanted into field plots on the University of Illinois at Urbana-Champaign. Equal amounts of leaf material were collected from 25 $F_{2:3}$ plants of each family, and these were bulked as one sample. Leaves were freeze-dried, ground to fine powder, and stored at −20 °C. After anthesis, 30–40 plants in each family were sib-pollinated.

Twenty days after pollination (DAP), eight ears from each family were harvested, frozen in liquid nitrogen, sealed in freezer bags, and stored at −80 °C. The kernels were later removed from each of the eight frozen cobs prior to analysis. A bulk 20-g sample (approximately 100 kernels) from the eight ears was freeze-dried and used for sugar analysis. The remaining ears were harvested at maturity (approximately 40 DAP), dried in forced air ovens at 35 °C to approximate 12% moisture concentration, and bulked for subsequent analysis. A subsample of 100 mature dry kernels from each $F_{2:3}$ family was weighed, freeze-dried and ground into a powder with a coffee grinder.

Sugar analysis

Previous studies have shown that sucrose accounts for 75–85% of the total sugar in immature sweet corn kernels (Juvik and La Bonte 1988). Maltose is not detected in 20 DAP kernels but can accumulate to up to 2–5% of kernel dry weight in mature dry seeds of lines homozygous for *se1* (Carey et al. 1982; Dickinson et al. 1991).

Sucrose and maltose concentrations in the 20 DAP and 40 DAP kernels, respectively, were determined by high pressure liquid chromatography (HPLC) using procedures described by Azanza et al. (1994). Two separate powdered kernel tissue samples from each of the $F_{2:3}$ families were analyzed for concentrations of sucrose at 20 DAP and maltose at 40 DAP.

RFLP analysis

Total maize genomic DNA was isolated from 600 mg of finely ground freeze-dried leaf tissue for each $F_{2:3}$ family using the CTAB procedure (Saghai-Maarof et al. 1984). DNA was stored at −20 °C for subsequent analysis. A set of 200 probes provided by the Brookhaven National Laboratory (bnl), Pioneer Hi-Bred International (p and npi), and University of Missouri, Columbia (umc) were screened for polymorphism among the parental lines using *Eco*RI: 62% of them (125) showed polymorphism. For the genotyping of the segregating population, we chose 88 probes plus three cDNAs clones from known structural genes [*sh1* (Sheldon et al. 1983), *sh2* (Bhave et al. 1990), and *dhn1* (Close et al. 1989)] known to be polymorphic between the parents. The RFLP probes were chosen to score the population on the basis of previous maps (Beavis and Grant 1991; Burr and Burr 1991; Burr et al. 1988; Coe et al. 1988; Gardiner et al. 1993; Helentjaris et al. 1986), allelic band separation, and ease of evaluation. Three probes (umc36, umc113, and npi276) gave us two informative bands (designated a and b based on size), and the rest were scored for one band. This number of segregating probes were considered to be sufficient for mapping the population without additional endonuclease digestions.

Ten micrograms of genomic DNA was digested with 25 units of the restriction endonuclease, *Eco*RI, for 4–6 h at 37 °C and fractionated for 16 h in a 0.8% agarose gel at 30 volts. Southern blotting to Hybound N+ membranes was conducted according to the manufacturer's instructions. Gels were treated with 0.4 *M* NaOH for 10 min, and DNA was transferred to the membranes with 0.4 *M* NaOH as the transfer buffer for a minimm of 6 h. After blotting, the membranes were briefly washed in 2 × SSC. Portions of agarose plugs containing the inserts (50–100 ng of DNA) were used for oligolabelling (Feinberg and Vogelstein 1983). Hybridization was performed in hybridization bottles containing 6 × SSC, 0.5% SDS, 0.025 *M* $NaPO_4$, 5 × Denharts, and 5 mg/ml denatured salmon sperm DNA at 65 °C. Membranes were prehybridized for 1 h, then the labeled probe was added to the bottle without replacement of the buffer. The hybridization of a minimum of 12 h was followed by a 15-min wash with 2 × SSC; 0.2% SDS at 65 °C, a 15-min wash with 1.5 × SSC; 0.2% SDS at 65 °C, and a 15-min wash with 1 × SSC; 0.2% SDS at 65 °C. Wet membranes were wrapped and exposed to X-Ray film for 1–5 days. After exposure, membranes were striped of radioactivity by placing them in boiled water with 0.6% SDS and subsequently shaken in this solution at room temperature until it cooled down or for a minimum of 30 min.

Two morphological markers were also scored in the population. Coleoptyle anthocyanin pigmentation (CAP) was scored on young seedlings of the $F_{2:3}$ families. IL731A*su1se1* has a purple coleoptyle while W678*suSe1* has a green one. The population was evaluated for the *se1* gene based on the kernel color, endosperm size, and pericarp texture of oven-dried $F_{2:3}$ seeds harvested at 40 DAP from each family (La Bonte and Juvik 1990).

Linkage analysis and QTL mapping

Multipoint maximum likelihood linkage analysis was performed (minimum LOD of 3.0 with a recombination fraction of 0.4) using the Macintosh computer version of MAPMAKER (Lander et al. 1987)

with the Kosambi mapping function (Kosambi 1944). Individuals marker loci were tested for linkage to quantitative trait loci (QTL) by analysis of variance of mean performance of marker genotypic classes (Edwards et al. 1987). Single-factor analysis of variance was performed on all loci for sucrose in 20 DAP kernels and for maltose of dry seeds using the one-way ANOVA from the PROC GLM routine of SAS (SAS Institute 1988). The association of marker loci with QTL was considered to be significant when the *F*-test exceeded a value necessary for a probability value less than 0.01. This probability level was chosen to avoid Type-1 error, since we assume a highly significant effect of the *sel* gene on kernel sucrose concentration. The results of the analysis of variance were reported using probe association scores [PAS = $\log_{10}$ (1/*P* value)] and R^2 values (percentage of the total variation accounted for by each marker). PAS = 1.3, 2, and 3 are equivalent to *P* values of 0.05, 0.01 and 0.001, respectively. Significant differences in marker class means were interpreted as indicating the linkage of marker loci to QTL controlling sugar concentrations.

Results and discussion

Construction of the sweet corn linkage map

All RFLP markers segregated according to the expected Mendelian ratios (data not shown). On the basis of the criteria described above, the MAPMAKER program (Lander et al. 1987) yielded 11 linkage groups. Reduction of the LOD score to 2.15 joined npi286 and npi447 to form 1 linkage group in an orientation similar to previously published maps of chromosome one (Beavis and Grant 1991; Burr and Burr 1991; Burr et al. 1988; Coe et al. 1993; Gardener et al. 1993; Helentjaris et al. 1986). The RFLP linkage map is based on the analysis of 214 $F_{2:3}$ families and contains 92 loci that cover 1734.5 cM, with an averaged distance between markers of 18.8 cM (Fig. 1). The population size and map density are comparable to those of other published maize maps and are suitable for QTL analysis (Darvasi et al. 1993; Tanksley 1993). The location and the order of markers on the map were the same as those previously reported for dent lines (Beavis and Grant 1991; Burr and Burr 1991; Burr et al. 1988; Coe et al. 1988; Gardiner et al. 1993; Helentjaris et al. 1986). While sweet and dent maize have somewhat distinct racial origins (Anderson and Brown 1952; Tapley et al. 1934), their respective maps display no major chromosomal rearrangements. This is in agreement with the full fertility observed from crosses between sweet and field corn lines.

Sugar analysis

The inbred W6786*su1Sel* displayed low kernel sucrose (112 mg/g dry weight) at 20 DAP and no detectable amounts of maltose at 40 DAP. In contrast, IL731A*su1sel* contained over 3 times more sucrose (341 mg/g) in 20 DAP kernels and maltose levels of 23 mg/g in 40 DAP kernels (Table 1). Sucrose concentration was found to be normally distributed among the $F_{2:3}$ families (Fig. 2A). Almost half of the families had no detectable amounts of maltose. In order to perform single-factor analysis of variance we normalized the maltose data using the natural log transformation (Fig. 2B, C). $F_{2:3}$ families displayed transgressive segregation for both sucrose and maltose, indicating a possible complementary action of alleles from both of the parents. Generation means for both characteristics fell between both parents. The wide range in the means of these traits between the parents improves the power and resolution of single-factor analysis of variance to detect QTL-marker associations (Darvasi et al. 1993; Tanksley 1993).

QTL analysis

Seven unlinked chromosomal regions which were significant at a probability level of $P = 0.01$ or less were

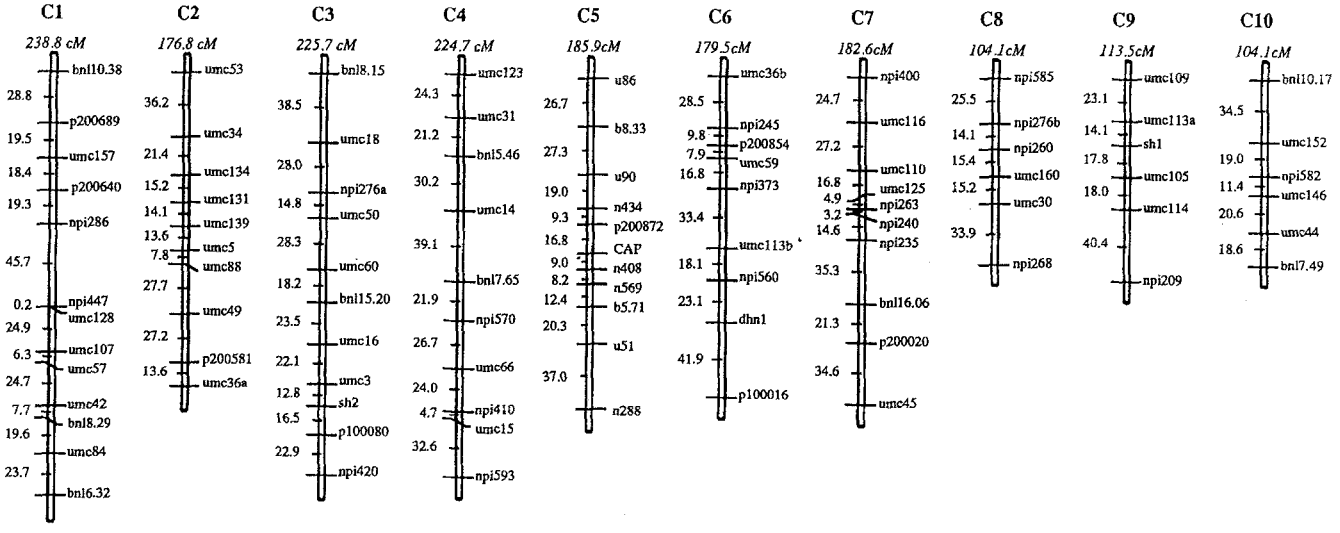

Fig. 1 RFLP linkage map of sweet corn constructed on the basis of data from the cross W6786 × IL731A. Distances between markers were calculated using the Kosamb's mapping function. The total length of the map is 1734.5 cM with an average distance betweeen markers of 18.8 cM

Table 1 Parental and $F_{2:3}$ means, standard deviations (SD), coefficients of variation (c.v.), and range of variation in the $F_{2:3}$ generation for each of the characteristics under study

Trait	Units	IL731A *su1se1*	W67856 *su1Se1*	$F_{2:3}$ Families			
				Mean	SD	C.V. (%)	Range
Sucrose at 20 DAP	mg/g	341	112	219	54.8	25.0	96–393
Maltose at 40 DAP	mg/g	23.5	0	2.75	4.99	1.81	0–28.5

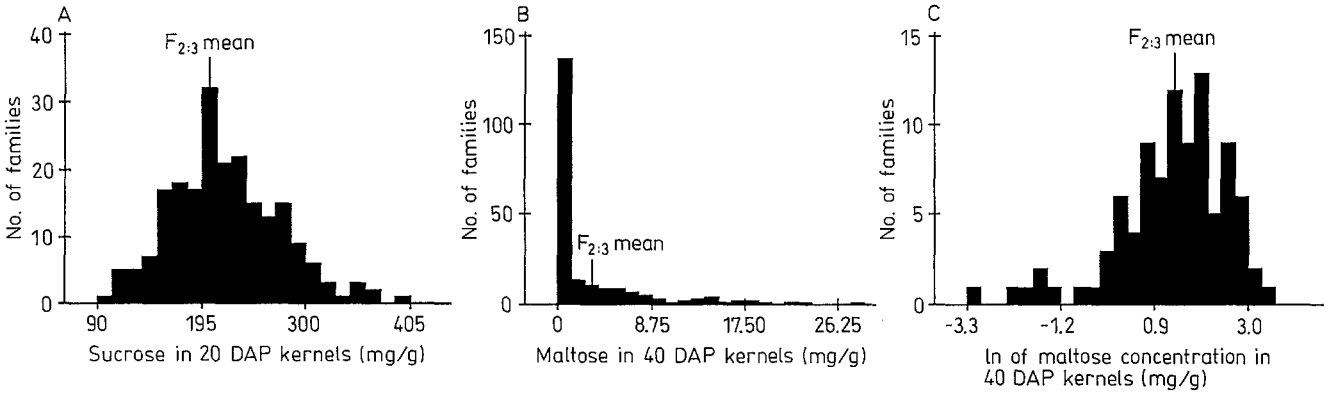

Fig. 2A–C Histograms of the distribution of the $F_{2:3}$ families for sucrose concentration at 20 DAP (**A**) and maltose concentration at 40 DAP (**B, C**)

detected for sucrose concentration in 20 DAP kernels (three on chromosome 1 and one on each of the chromosomes 2, 3, 4 and 5), and two regions were detected for maltose in 40 DAP kernels (on chromosomes 2 and 6). The results of the analysis of variance are reported using probe association scores [PAS = $\log_{10}(1/P$ value)] and R^2 values (percentage of the total trait variation accounted for by each marker) (Table 2).

The most significant probe association for sucrose concentration was with umc36a (PAS = 12.3) at a terminal region on the long arm of chromosome 2. This probe explains 24.7% of the variation in fresh kernel sucrose concentration. The magnitude of the association of this probe with kernel sucrose suggests it is linked to the *se1* locus. Linkage of umc36a with a QTL influencing kernel maltose concentration at 40 DAP (PAS = 2.4) provides additional support for this finding since previous reports have shown an association between elevated maltose concentration and the presence of the *se1* gene (Ferguson et al. 1979; La Bonte and Juvik 1990). The seven other regions found to contain QTL influencing sucrose and maltose concentration described considerable lower amounts of the total phenotypic variation (R^2 from 4.7 to 13.9) (Table 2).

Table 2 Summary of the significant associations between RFLP markers and sucrose and maltose concentrations detected using one-way ANOVA

Chr[a]	Probe	Genotypic means[b]			F[c]	R^{2d}	PAS[e]
		1	2	3			
Sucrose concentration at 20 DAP							
1L	npi447	207	215	242	6.3	5.8	2.7
1L	umc42	206	217	240	5.2	4.9	2.4
1L	bn16.32	224	220	256	4.8	5.9	2.0
2L	umc36a	191	214	270	32.7	24.7	12.3
3C	umc50	198	216	250	10.9	10.4	4.5
4L	npi593	202	231	213	5.2	4.8	2.2
5S	npi434	203	220	239	5.2	4.7	2.2
Maltose concentration at 40 DAP[f]							
2L	umc36a	1.9	2.3	5.5	6.0	4.8	2.4
6S	umc59	0.5	2.5	5.6	6.6	13.9	2.7

[a] Chromosome region
[b] 1, homozygous W6786; 2, heterozygous W6786/IL731A; 3, homozygous IL731A
[c] F value from the ANOVA
[d] Percentage of the total variation explained by each RFLP marker
[e] Probe Association Score
[f] ANOVA was conducted using a logarithmic transformation of the data

Mapping of the *se1* gene

To confirm the location of the *se1* gene on chromosome 2, oven-dried kernels of the $F_{2:3}$ families harvested at 40 DAP were scored for the *se1* phenotype based on lighter kernel color and a more finely wrinkled pericarp texture (La Bonte and Juvik 1990). The monogenic segregation of this marker did not deviate from the expected Mendelian ratio (45 *se1/se1*, 111 +/*se1*, and 49 +/+, with a $\chi^2 = 1.56$, 2 *df*, $0.5 > P > 0.25$). The linkage relationships between this marker and all the other 92 markers were analyzed by MAPMAKER using a LOD of 3.0 and a recombination fraction of 0.4. *Se1* was mapped to chromosome 2, 12.1 cM distal to umc36a (Fig. 3). The *se1* marker had a PAS of 21.8 with 20 DAP kernel sucrose concentration and explained 42.1% of the total vari-

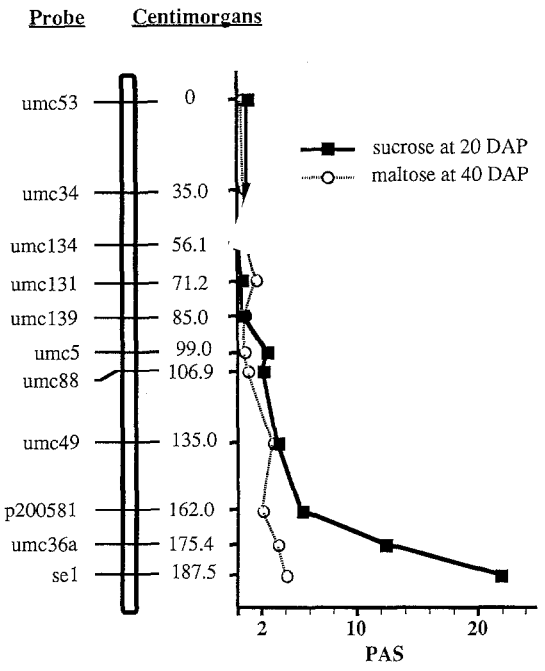

Fig. 3 RFLP map of chromosome 2 and PAS obtained from the ANOVA looking at the association between each marker on chromosome 2 and sucrose and maltose concentrations

ation for sucrose in the population. The PAS (3.3) for *sel* for kernel maltose at 40 DAP was also significantly associated. Our data as well as several previous reports suggest that the *sel* mutation is responsible for a lesion in endosperm starch metabolism that results in both enhanced sucrose at 20 DAP and maltose at 40 DAP. The actual metabolic process resulting in the associated amplification of these two sugars at different stages of kernel development awaits elucidation.

These results are in disagreement with the previously postulated position of the *sel* locus on the long arm of chromosome 4 (La Bonte and Juvik 1991). We feel that the use of maize B-A translocation stocks to map genes to specific arms of the chromosomes is not as rigorous a method as linkage studies with a saturated RFLP map of the genome. In this study we observed linkage of a QTL affecting sucrose to npi593 on the long arm of chromosome 4. It may be that this gene, which exerts a smaller effect of kernel sucrose, was the one observed in the earlier study by La Bonte and Juvik (1991).

Based on the dosage effect of the *sel* gene on kernel sugar and the assumption that this allele is recessive, we would expect the sucrose concentration mean for those families heterozygous for the *sel* gene to be greater than that of the families homozygous for *sul*, since one-quarter of the $F_{2:3}$ kernels will be homozygous for *sel*. The theroretically calculated mean sucrose concentration for the heterozygous class is 203 mg/g sucrose, which is comparable to the 210 mg/g value that was observed. This is also in agreement with the observed phenotypic segregation of 3:1 of *sel* (Ferguson et al. 1978; La Bonte and Juvik 1990). The incremental increase in the PAS of sucrose with linked probes at decreasing distances to the *sel* locus (Fig. 3) indicates that this gene has a major effect on kernel carbohydrate metabolism during kernel development. This progressive increase in association scores provides support for the location of the *sel* locus near the terminus of the long arm of chromosome 2. No other putative gene influencing kernel carbohydrate metabolism has been mapped to this region (Coe 1993).

Possible involvement of other known structural genes

Except for the QTL associated with umc50 on chromosome 3 and umc59 on chromosome 6, all other probe associations to the investigated traits were with loci that had minor effects on the sucrose and maltose concentrations and described less than 8% of the total variability. Genes linked to umc50 and umc59 apparently influence kernel carbohydrate metabolism. It is interesting to note that in comparing the location of umc50 with the Maize Genetics Cooperation Gene List and Working Map (Coe 1993), this QTL mapped close to the *hexokinase1* (*hex1*) locus. Alleles of the *hex1* locus with varying enzyme activity would influence the amount of sucrose partitioned in the synthesis of either lipids or carbohydrates. As for umc59, no previously mapped structural genes that influence maize kernel carbohydrate metabolism have been found on chromosome 6.

In summary, our data strongly suggest that the *sel* gene is on the distal end of chromosome 2, approximately 12.1 cM distal to umc36a. Polymorphism at this locus, in a population segregating for the *sel* gene, can be utilized to select for plants homozygous or heterozygous for this desirable gene at the seedling stage. This would allow for the rapid introgression of this gene into elite maize backgrounds. To improve a system for molecular-assisted selection and to elucidate the physiological role of the *sel*, more detailed mapping is currently in progress.

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