Report

of the

Tomato Genetics Cooperative

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Department of Plant Breeding
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Foreword

The Tomato Genetics Cooperative is a group of researchers who share an interest in tomato genetics, and who have organized informally for the purpose of exchanging information, germplasm, and genetic stocks. The Report of the TGC is published annually and contains reports of work in progress by members, announcements, and updates on linkage maps and materials available. The research reports include work on diverse topics such as new traits or mutants isolated, new cultivars or germplasm developed, interspecific transfer of traits, studies of gene function or control and tissue culture. The data presented here are not to be used without the permission of the respective authors.

As of November 30, 1998, membership stood at over 200, from 34 countries. Requests for membership (US$15 plus $5 shipping if international) should be sent to Theresa Fulton, 252 Emerson Hall, Cornell University, Ithaca, NY 14853-1901.

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From the editors

Tomato Genetics Cooperative has changed hands this year, for the first time in over 10 years. We hope to uphold the tradition of cooperation and goodwill that has kept TGC going for 48 years. To keep the Report informative and current, we would also like to introduce two **new** items into future Reports. With the expansion of comparative genomics, it seems prudent to keep abreast of work in related species in addition to tomato. Therefore, we encourage the submission of articles from other Solanaceous species that may be relevant: comparison maps, orthologous genes, etc.

In addition, beginning next year we will be inviting a member of the tomato industry or a biotechnology company to give a commentary related to a contemporary plant breeding or genetics topic. Suggestions for commentary topics or authors, as well as any suggestions for additions or improvements to the Report are always welcome.

In hopes of getting the 1999 Report out earlier next year, the deadline for submissions is May 1, 1999. Submissions received after this date will be accepted but not guaranteed publication in the current issue. As always, articles should be as concise as possible, 2 pages maximum. Submissions (preferably in Microsoft Word) should be sent to the managing editor as Macintosh or compatible diskettes (with an included hard copy), emailed as attachments, or uploaded to the FTP site:

ftp://tgc.plbr.cornell.edu/

Most images can be included, preferably TIFF or EPS, but also Pict, Photoshop, B/W photos, Excel tables, and other graphics. For more information and links to some past issues, see the web site (still under construction as of this writing):

http://tgc.plbr.cornell.edu

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OPEN QUESTION TO MEMBERS FROM ROGER SEEBER

In a time of ever increasing gene loss, sorting through the blizzard of varietal names, local unknown tomatoes, new ones being created, and the large number of substantial collections seems like a timely project. Has anyone begun this or have any suggestions for the initiation of a genetic variety ID? Send responses to Roger Seeber, Biology Division, West Liberty State College, West Liberty, WV 26074, phone: (304) 336-7230, email: seeberrg@WLSVAX.WVNET.EDU

Developing Tomato and Tobacco Crops with Broad-Spectrum Resistance against Plant Pathogens by Genetic Transformation

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Tomato line Mocimor and tobacco line Samsun NN were transformed with *avrD* (Kobayashi *et al*. 1990) and a magainin 2 derivative lytic peptide gene (MSI-99) to examine possible use of these genes for enhancing plant resistance against phytopathogenic organisms. Both genes were put under an enhanced cauliflower mosaic virus promoter, and *Agrobacterium tumefaciens* strain LBA4404 was used as the transformation vector. Twenty-nine tomato transformants carrying *avrD* and 30 transformants carrying MSI-99 were obtained. In tobacco, 27 transformants carrying *avrD* and 57 transformants carrying MSI-99 were obtained. Transformants were initially confirmed by PCR analysis using construct-specific primers. Southern blot analysis using *nptII* and *avrD* genes as probes agreed with the PCR results.

In preliminary experiments, some transgenic tomato and tobacco plants were inoculated with different concentrations of *Pseudomonas syringae* pv. *tomato* and *P. syringae* pv. *tabaci*, respectively. Results from these tests suggested that expression of *avrD* and MSI-99 genes in tomato and tobacco may delay symptom development and disease severity when transformants were inoculated with bacterial concentrations of 10^4 and 10^5 CFU/ml. Development of homozygous transformed lines and further tests with bacterial and fungal pathogens are underway.

Acknowledgement. We thank Dr. Alan Blowers of Sanford Scientific for providing the constructs used.

Literature cited

Kobayashi DY, Tamaki SJ, and Keen NT. (1990) Molecular characterization of avirulence gene D from *Pseudomonas syringae* pv. *tomato*. Molec. Plant-Microbe Interact. 3: 94-102.

Race composition of the natural epiphyte pathogen population of *Pseudomonas syringae* pv. *tomato* in Bulgaria

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Bacterial speck on tomato caused by *Pseudomonas syringae* pv. *tomato* (*Pst*) is an important disease in many tomato growing areas worldwide (Gitaitis et al., 1992). *Pst* can be present as an epiphyte on tomato (McCarter et al., 1983; Marino and McCarter, 1991) and a wide range of annual and perennial late spring weeds (Bogatzevska, 1988; Boneva et al., 1990; Marino and McCarter, 1991) and survives on seeds of tomato (McCarter et al., 1983) and weeds (Vitanov and Bogatzevska, 1988; Bogatzevska and Boneva, 1990). Strains of *Pst* are divided into two races- R0 and R1, based on the different reactions on tomato genotype Ontario 7710 (Lawton and Mac Neill, 1986). Race 1 was discovered in Canada (Lawton and Mac Neill, 1986), Bulgaria (Bogatzevska et al., 1989), and Italy (Buonaurio et al., 1996).

The collection of Bulgarian *Pst* strains was tested by the method of Bogatzevska et al. (1989). The following differential species were used: cv. Ontario 7710 (resistant *Pto*) and susceptible cv. Chico (Lawton and Mac Neill, 1986).

The isolates were gathered during the period 1990-1997 from: 1) different tomato species (leaves, stems, fruit, flowers) with symptom and symptomless development of bacterial speck; 2) mass seed production and experimental fields from whole Bulgaria; 3) weeds from the usual crop and regional association, on which *Pst* was in resident phase. The identification was done by the basic differential tests for *Pseudomonas syringae* pv. *tomato* (Bradbury, 1986; Schaad, 1988; Young and Triggs, 1994).

Controls were: **Pst- 1778 R1, Pst- 1776 R0** (Gottinger, Germany), **Pst- 140 R0, Pst- 148 R1** – collection of the Plant Protection Institute.
A total of 184 *Pst* strains were tested, from which 106 belonged to race 0 and 78 to race 1. The natural population of *Pst* contained both races, but race 0 predominated. Only race 0 was distributed on the mass tomato crops and the weeds from the usual crop and regional association. The cultivated tomato species were sensitive to race 0. The Bulgarian strains differentiated as race 1 formed large number of spots on the leaves and stems of Ontario 7710. The control *Pst* 1778 R1 caused HR without the appearance of typical necrotic spots on the leaves and stems. Race 1 predominates in the experimental fields of the Institute of Genetics and the Plant Protection Institute, where a large variety of wild species and lines are cultivated.

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Transformation of tomato with an endochitinase gene from the biocontrol fungus *Trichoderma harzianum*

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The goal of this study was to determine whether tomato plants transformed with a *T. harzianum* endochitinase gene have increased resistance to fungal pathogens. The gene *ThEn-42*, encoding the *T. harzianum* endochitinase, has been cloned at Cornell (Lorito et al., 1993).

Fifteen transgenic tomato plants (cv. Moneymaker) were obtained after *Agrobacterium tumefaciens* –mediated transformation with p35ThEn42 (35CaMV promoter, genomic DNA with 3 introns, fungal signal peptide) and 15 with pStudI (double 35S CaMV promoter, AMV enhancer, endochitinase cDNA, fungal signal peptide). Thirty two transformants were obtained with pSAN206BI (UBQ3 (polyubiquitin) promoter, cDNA, fungal signal peptide removed) and 38 with pSAN208BI (UBQ3 promoter, cDNA, pea vicilin gene signal peptide). Plants were initially confirmed as transformants using PCR and NPTII protein-specific ELISA. Endochitinase activity was measured by a fluorimetric assay using 4-methylumbelliferyl-_-D-N, N', N''-triacetylchitotriose as a substrate. pStudI primary transformants had 2-150 times higher endochitinase activity than control plants. Endochitinase activity in plants transformed with the other three constructs was comparable to that of control plants, so these transformants were not studied further. Transformation of pStudI plants was further confirmed by Southern blotting. Expression of the endochitinase was confirmed by Western blotting using endochitinase-specific antibodies. Only pStudI plants showing high endochitinase activity had the expected ~40kDA protein band.

Seeds were collected from four different pStudI tomato plants expressing high endochitinase activity and one plant with low endochitinase activity. T1 progeny were used to determine if endochitinase provides resistance to *Fusarium oxysporum* fsp. *lycopersici.* Seventeen days old seedlings (15-60 from each line, seed supply and germination permitting) were inoculated by dipping roots in a suspension of fungal mycelium. Plantlets were screened after 16 days, and disease severity was recorded (0 = healthy plant, 10 = dead plant). The experiment was repeated twice. Both transgenic and control plants displayed severe disease symptoms, and no statistically significant differences in performance of plants were found.

In vitro studies were done to determine if pStudI tomatoes have increased resistance to the early blight fungus *Alternaria solani* and to *Rhizoctonia solani*. Three leaflets (one from each of three different leaves on the same plant) were collected and endochitinase activity determined in *A. solani* studies. Endochitinase activity in the transgenic plants tested was up to 80 times higher than in controls. Seven days after inoculation with *A. solani* disease severity was recorded (0 = no lesions, 5 = lesions >5 mm in diameter). Although transgenic lines showed slightly reduced chlorosis around lesions in comparison to control lines, there were no significant differences in lesion sizes between transgenic plants and controls.

For studies with *R. solani,* 12 days old transgenic plantlets were planted in deep Petri dishes containing water agar blended with fungal mycelium. Disease severity was recorded every other day starting from the seventh day post-inoculation. Results were analyzed using a statistical model to determine when disease severity reached the rating of “5” (plant has lost lower leaves, some lesions on the stem, wilting). As in previous experiments, transgenic plants did not show increased resistance to fungus in comparison with controls.

In our studies, elevated levels of endochitinase alone did not increase resistance to several fungal pathogens of tomato plants. The enzyme concentration in our plants may have been too low to provide effective protection. *F. oxysporum* invades plant via vascular tissue where endochitinase probably is not expressed. Also, chitin in fungal cell walls normally is covered by other polysaccharides, which might decrease the effect of endochitinase on the pathogen. Transgenic plants that combine endochitinase with other lytic enzymes (or other transgenic proteins) may provide more positive results.

Acknowledgements

We would like to thank Dr. Gary Harman and Dr. Christopher Hayes (Cornell University) for providing constructs p35ThEn42 and pStudI and endochitinase specific antibodies and Dr. Alan Blowers (Sanford Scientific) for providing pSAN206BI and pSAN208BI.

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Lorito *et al*. (1993) Phytopath. 83:302

Bco, a corolla pigment intensifier on chromosome 7.

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In early backcross derivatives of *L. esculentum* 'VF36' x *Solanum lycopersicoides* 'LA2951', variation for flower color intensity was observed. The corolla color of plants in some families was consistently brighter, of a deeper yellow (nearly yellow-orange) hue, than the pale-yellow typical of 'VF36'. The color classes are relatively discrete and plants can be reliably classified by comparing several flowers at different stages on an inflorescence. Differences are more pronounced under field conditions, and among senescent flowers, those of the variant retaining color longer than normal.

Segregation was first detected in a BC_2F_2 family (94L1271), which yielded 16 normal : 6 mutant, consistent with a monogenic recessive (X^2=0). However, analysis of several isozyme loci segregating in this population told a different story. The corolla trait showed complete association with the chromosome 7 marker *Got-3*, which segregated 16 +/+ : 6 +/S : 0 S/S (wherein S=*S. lycopersicoides* allele), a significant distortion from the expected 1:2:1 ratio (X^s=27.8***). All *Got-3*$^{+/S}$ genotypes showed the corolla intensifier trait, while all the *Got-3*$^{+/+}$ genotypes had normal flowers. Association with another chromosome 7 marker, *Got-2*, was less intense: the one *Got-2*$^{+/S}$ – *Got-3*$^{+/+}$ recombinant had normal flowers. A larger population (95L2461) produced similar results: segregation for *Got-3* was distorted (40 +/+ : 42 +/S : 6 S/S; X^2=27.1***), and all *Got-3*$^{S/-}$ plants showed the corolla intensifier trait, with a somewhat more extreme phenotype in S/S homozygotes than heterozygotes. Based on these results, we conclude this trait is probably monogenic and at least partially dominant, for which we propose the gene symbol *Bco* (Brilliant corolla).

To investigate the linkage relations of *Bco*, homozygotes were crossed to the multiple marker stock LA1164, containing the genes *var* (variabilis) and *not* (notabilis). Segregation and recombination were monitored in the testcross to the recessive marker stock. *Bco* could not be scored in all *var* plants due to partial epistasis: *var*, which causes a general chlorosis in the shoots, also reduces corolla color intensity, thereby obscuring the effect of *Bco*. The other marker genes, all unambiguous, showed distorted segregations, particularly evident when the F_1 was used as female parent (Table 1). No distortion was seen in either direction in the control crosses. Since the original donor line contained a *S. lycopersicoides* segment around *Got-3* and *Bco* of 30cM or more (Figure 1), the non-Mendelian segregation ratios were presumably due to linked genes under gametophytic and/or zygotic selection.

No recombination between *Bco* and *Got-3* was detected in either population, suggesting they are tightly linked (Table 2). *Got-3* and *Bco* showed a closer association with *var* (0.9cM) than with *not* (23cM). The two *var* – *Got-3* recombinants detected were nonrecombinant for *var* – *not*, suggesting *Got-3* and *Bco* are distal to *var* as indicated (Figure 1). Recombination was reduced approx. 5 fold in the *Got-3* – *var* interval, and approx. 2 fold in the *var* - *not* interval, compared to published maps (Figure 1).

Bco joins a large number of apparently monogenic morphological traits that have been observed in derivatives of *S. lycopersicoides* (Chetelat unpublished; Rick et al. 1988). The majority are novel traits, although a few, such as *Wa* (White anthers), and *Di*^{s} (Dialytic anthers), are observable in the wild parent itself. *Bco* belongs to the latter category, since *S. lycopersicoides* also displays brightly colored corollas. It seems likely that a more intense corolla pigmentation would enhance attraction of pollinating insects. If so, *Bco* would be among numerous floral characteristics (e.g. dialytic anthers, terminal anther pores, exserted stigmas, compound and 'showy' inflorescences, self-incompatibility, etc.) that promote outcrossing in this allogamous species.

Literature cited:

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Tanksley, S. D., and C. M. Rick (1980) Isozymic gene linkage map of the tomato: applications in genetics and breeding. Theor. Appl. Genet. 57:161-170

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Table 1. Single-locus segregations in the repulsion testcross *Bco-Got*^{S}-+-+ / +-+-*var-not* x +-+*var-not* /+-+-*var-not*. In the direct testcross, the F_1 is used as female parent, in the reciprocal as male parent. Data from the two control crosses to 'VF36', direct and reciprocal, did not differ significantly, so are pooled.

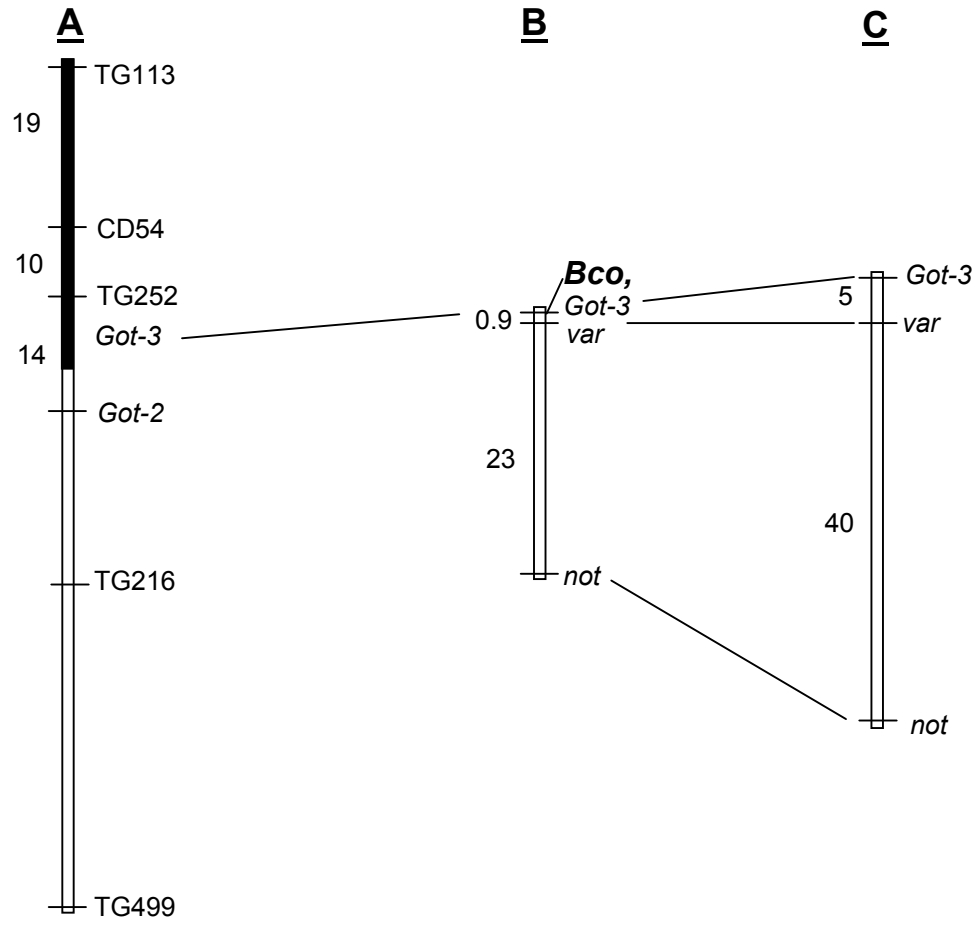
	Direct (F_1=female)			Reciprocal (F_1=male)			Control (pooled)		
Gene	+	mutant	X^2	+	mutant	X^2	+	mutant	X^2
Got-3	118	5	104***	72	43	7.3**	-	-	-
var	6	117	100***	42	73	8.4**	70	62	0.37
not	30	93	32.3***	51	64	1.5	65	67	0.008

Table 2. Joint segregations and recombination fractions for the repulsion testcross *Bco-Got*^{S}-+-+ / +-+-*var-not* x +-+*var-not* /+-+-*var-not*..

Gene	Direct (F_1=female)			Reciprocal (F_1=male)			Control (pooled)		
	+	*var*	cM±SE	+	*var*	cM±SE	+	*var*	cM±SE
Got-3^{S}	5	0	0.8±0.8	42	1	0.9±0.9	-	-	-
+	1	117		0	72		-	-	
not	2	92	24.6±3.8	7	57	21.2±3.7	33	34	n.s.
+	3	26		35	16		37	28	
	+	*Got-3*		+	*Got-3*				
not	91	2	23.2±3.8	59	9	22.0±3.8			
+	24	3		15	33				
Bco	0	5	0.0	0	42	0.0			
+	57	0		31	0				

[Editor's note: this figure, as originally published in TGC, contained an error in the drawing of the chromosome on the left – the correct version is represented here]

Figure 1. A) RFLP map of chromosome 7 (from Tanksley et al., 1992; approx. location of *Got-3* from Meglic and Chetelat, unpublished); solid section indicates the introgressed *S. lycopersicoides* segment containing *Bco* and *Got-3.* B) Map location of the *Bco* and *Got-3* genes from the present study (data pooled from direct and reciprocal crosses). C) Distances from classical maps of Tanksley et al. (1992) and Tanksley and Rick (1980).



Mapping of the *Ph-3* gene for late blight from *L. pimpinellifolium* L 3708

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The mapping of a partially dominant gene for late blight resistance from AVRDC *L. pimpinellifolium* accession L3708 (Black et al. 1996) has been done using AFLP markers and bulk segregant analysis approach. An F2 population generated from the cross of AVRDC inbred line CLN657-BC1F2-274-0-15-4 (susceptible) x L3708 (resistant) was used for the analysis. Prior to late blight inoculation, young leaves from each F2 plant were harvested for DNA isolation. After disease reactions had been scored, DNA from 9 highly resistant plants and 8 highly susceptible plants were used to generate an R-pool and S-pool, respectively. One hundred and twenty combinations of 3-base selective nucleotide AFLP primer pairs were used to assay R-pool and S-pool together with resistant and susceptible parents. DNA bands from six pairs were found to correspond to the resistant parent and R-pool or the susceptible parent and S-pool. These primer pairs were then used to genotype the whole population of 72 F2 plants. The correlation between these loci and disease resistance phenotype was analyzed using ANOVA and found to be highly significant ($P<0.0001$) These DNA bands were then cloned and used to hybridize with DNA from the introgressed lines population (IL) (Eshed et al., 1992) to find the location of these fragments. Hybridization results revealed that the line IL9-3, which represents the introgressed piece of the *L. pennellii* long arm of chromosome 9, was different from the others. To confirm the position of the DNA markers, we selected RFLP markers from the long arm of chromosome 9 (Tanksley et al. 1992) and used them for hybridization with a second F2 population from the same cross segregating for the *Phytophthora infestans* resistance gene and found that AFLP derived markers were associated with RFLP markers on chromosome 9 and mapped as shown in Fig 1.

Two genes conditioning late blight resistance in tomato have been reported: *Ph-1* on chromosome 7 (Clayberg et al 1965) and *Ph-2* on chromosome 10 (Turkeensteen (1973), Moreau et al 1998). We conclude that the gene identified in this experiment is a new locus for which we propose the symbol *Ph-3*.

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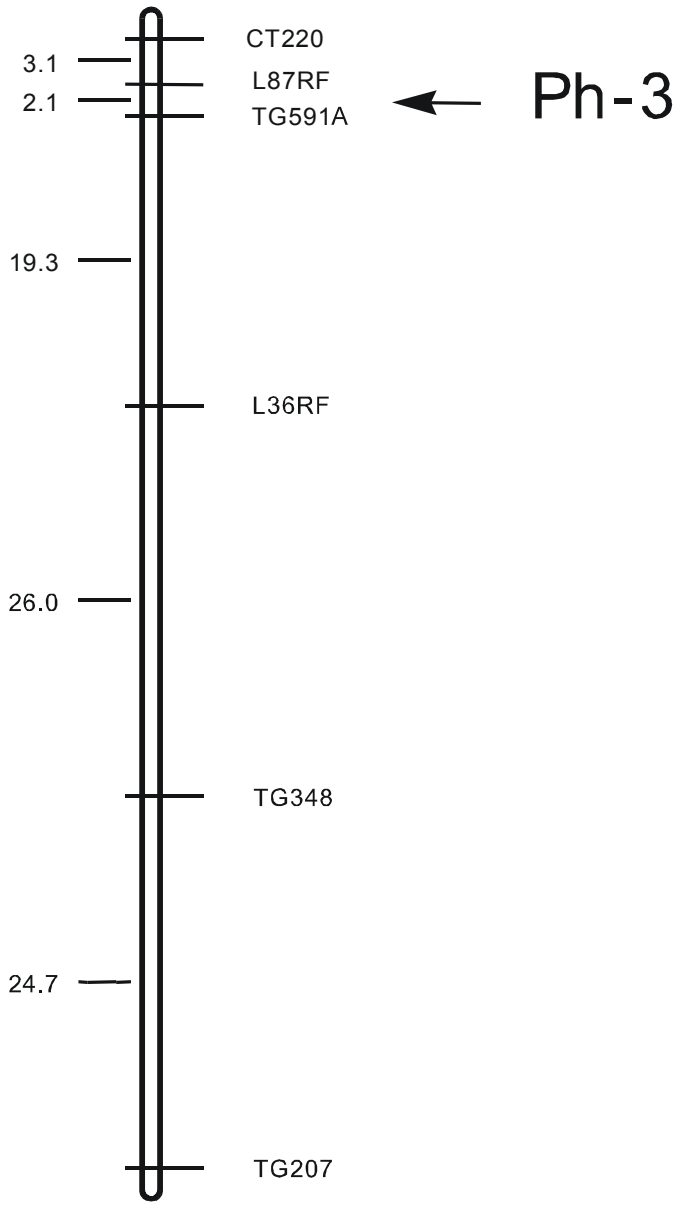
Figure 1 Mapping of Ph-3 on tomato chromosome 9



Mapping of earliness (*er12.1*) gene in tomato

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Earliness is an economically important trait for both processing and fresh market tomatoes in that it is crucial for regions with short growing seasons and is desirable for taking advantage of early season high prices and for staggering tomato production.

The most common form of earliness in tomatoes is early maturity due to an earlier switch from vegetative to reproductive growth. This type of earliness is generally measured by determining the number of days from sowing to the appearance of the first ripe fruit (Kemple and Gardner 1992). Unfortunately, this type of earliness is associated with a decrease in fruit size (Banerjee and Kalloo 1989). A new form of earliness was identified in the 1980s in the variety 'Early Cherry' (M. Mutschler, pers. comm.). This type of early maturity is the result of a decreased length of time between anthesis and the first ripe fruit. An F2 population of 104 plants was generated from a cross between *L. esculentum* cv E6203 x *L. pimpinellifolium* (cv. 'Early Cherry'). This population was used for molecular marker analysis to identify QTLs for earliness and to map the genes controlling the trait. A total of 530 RAPD markers were first screened on parental DNA to find the markers with polymorphism. The polymorphic markers were then used in the F2 population. Forty-eight RAPD markers segregated in the F2 population and fell into 6 linkage groups. Two of the markers (OP106 and OP529) were found to be strongly ($p<0.0001$) linked to earliness. These markers explained 19% and 17% of the total phenotypic variation for earliness, respectively. These markers (OP106 and OP529) eventually mapped to chromosome 12 between TG283 and CT394 and chromosome 5 between TG503 and TG96 close to *rin* gene, respectively. The QTL on chromosome 5 (er5.1) is also associated with fruit weight, but the QTL on chromosome 12 (er12.1) seems to be free of any associated effect and may be useful in the development of early maturity lines.

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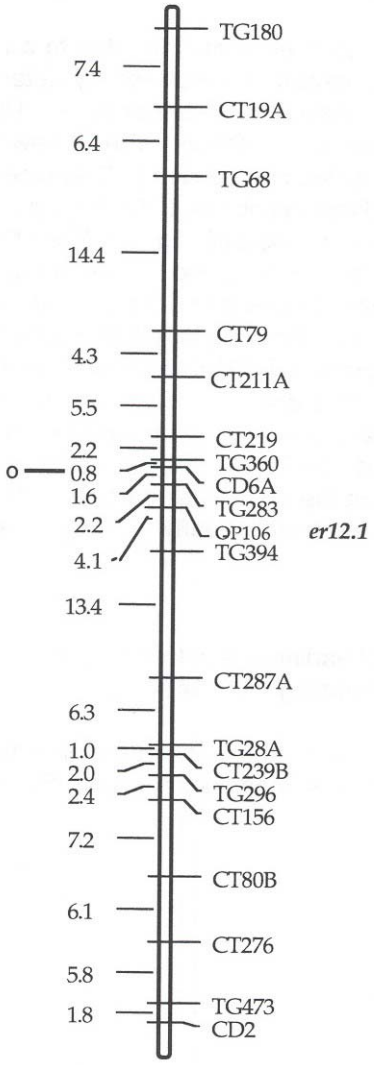

Figure 1. Map of tomato chromosome 12 showing position of the *er12.1* gene, The linked RAPD markers. o=centromeric region

Mapping of fruit shape QTL controlling the bell-pepper phenotype in tomato.

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'Yellow Stuffer' is a tomato cultivar that resembles a bell pepper in appearance, having four prominent locular lobes and a high proportion of internal air space. In an effort to map the genes responsible for the distinctive shape of 'Yellow Stuffer', an F_2 population of 48 individuals from the cross *L. esculentum* cv 'Yellow Stuffer' x *L. pimpinellifolium* LA1589 was analyzed. Bumpiness, the degree to which the fruit displayed the bell-pepper phenotype, was evaluated visually on a scale of 1-5, where 1= completely spherical like the *L. pimpinellifolium* parent and 5 = highly lobed like the 'Yellow Stuffer' parent. Genotypic data for 86 segregating molecular markers spanning the entire tomato genome were also generated for the mapping population.

Analysis of the genotypic and phenotypic data with QGene software (Nelson, 1997) revealed significant ($P < 0.01$) associations between several markers on chromosomes 2, 5 and 8 and the bumpiness trait (Figure 1). The degree of dominance statistic (d/a) indicates that for QTLs on chromosome 2 and 5 bell-pepper shape is partially recessive, whereas on chromosome 8, the gene effect is more nearly additive (Table 1). It is especially interesting to note that these bumpiness QTLs fall into regions of the genome in which other loci controlling fruit development have been mapped (Figure 1). The interval between TG469 and TG151 on chromosome 2, which appears to account for approximately 30% of the bumpiness phenotype, also contains *ovate* (Ku , personal comm.) and *fw2.2* (Alpert et al., 1995), the major fruit weight QTL in tomato. Moreover, the bumpiness loci on chromosomes 5 and 8 fall in the vicinity of other QTLs involved in fruit size and shape, namely*fw5.1* and *fs8.1* (Tanksley et al., 1996). Thus, it is tempting to speculate that perhaps many of the genes controlling fruit development are clustered in just a few regions of the genome which may point to a common evolutionary origin. Alternatively, it is possible that the stuffer trait is allelic with *ovate* or another fruit shape gene. Further investigation of the trait should elucidate these issues.

Table 1. QTLs detected for bell-pepper fruit shape (bmp).

locus	chromosome	marker	R^2 %	P value	d/a
bmp2.1	2	TG469	33	0.0001	-0.46
	2	TG151	28.9	0.0007	-0.46
bmp5.1	5	TG619	22.7	0.004	-0.81
bmp8.1	8	TG176	20.5	0.006	-0.1

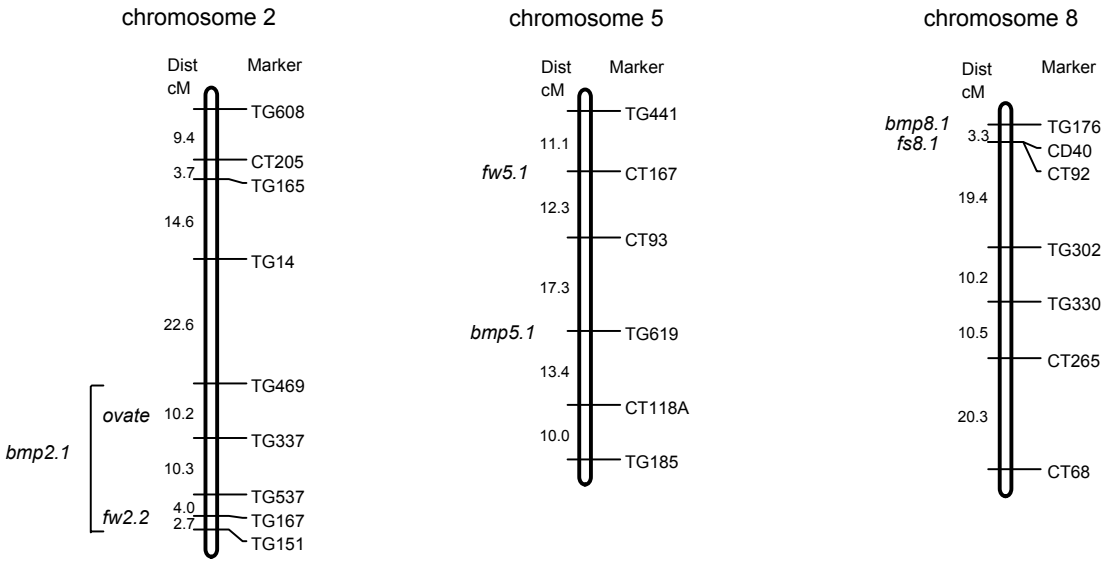

Figure 1. Molecular linkage maps of chromosomes 2, 5, and 8 with the positions of significant fruit development QTLs indicated to the left.

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Identification of QTL for late blight resistance from *L. pimpinellifolium* L3708

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Quantitative trait loci (QTL) for *Phytophthora infestans* resistance were mapped in an F2 population derived from a cross between a susceptible *L. esculentum* line (NC23-2(93), provided by R. Gardner, N. Carolina State Univ.) and the resistant *L. pimpinellifolium* accession L3708 (provided by L. Black, AVRDC). Although the resistance from L3708 has been described as a single additive gene (Black et al., 1996), when the F2 were grown in a virulent field in California, the plants showed a continuous distribution from resistance to susceptibility (Figure 1). This observation indicated that it is likely that resistance to California isolates of the pathogen is controlled by more than one gene.

A total of 115 RFLP markers covering the entire tomato genome were assayed on the F2 population and two QTL associated with resistance were identified. One QTL, located on chromosome 6, spans nearly 40 cM and includes 9 RFLP markers (5 with $p<0.001$, 4 with $p<0.01$) and one morphological marker, *sp*. Within this QTL, the RFLP markers are spaced at intervals of less than 10 cM. The three most significant ($P = 0$) markers, CT206, TG314 and TG578, account for 19.5, 18.4 and 19.0%, respectively, of the variation in the resistance phenotype (Figure 2, Table 1). Because *Sp*, the self-pruning locus, is included within the QTL region and because the *L. pimpinellifolium* alleles for both *Sp* and late blight resistance show the same gene action (dominance), it is possible that disease resistance is a pleiotropic effect of *Sp*. In other words, it may be that F_2 individuals that are indeterminate are growing out of the disease and, therefore, appear to be resistant. The second QTL linked to *P. infestans* resistance is located on chromosome 8 (Table 1). This region is much less significant ($p<0.05$) than the QTL on chromosome 6 and the most significant marker, CT111, accounts for only 6.5% of the variation in the disease phenotype.

Currently, F4 lines derived from selected F2 individuals are being grown in California. These plants will be scored for their disease resistance and *sp* phenotypes. RFLP markers linked to resistance will be scored in this population, thus, this experiment should confirm the linkage of specific markers to disease resistance. In addition, analysis of these plants, especially those individuals which have a recombination between TG279 (the marker most closely linked to *Sp*) and CT206 should clarify whether or not resistance is solely an effect of the *Sp* locus.

To date, three genes controlling late blight resistance in tomato have been identified. *Ph-1* is located on chromosome 7 (Peirce, 1971), *Ph-2* was mapped to chromosome 10 (Moreau et al., 1998), and *Ph-3* was mapped to chromosome 9 (Chunwongse et al., 1998). None of these genes were detected in our population infected with California isolates of *P. infestans*.

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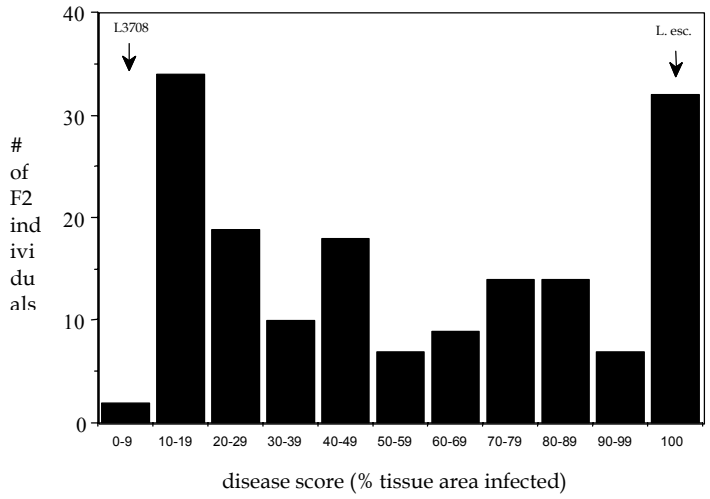

Figure 1. Disease scores for 166 individuals of the F2 population infected with *P. infestans*.

Table 1. RFLP markers with significant associations with late blight resistance, their chromosomal locations, R^2 and P and D/A (dominance/additivity) values.

marker	chromosome	R^2 value	P value	D/A
CT206	6	0.195	<0.0001	-0.9
TG314	6	0.184	<0.0001	-1.1
TG578	6	0.190	<0.0001	-1.8
TG477	6	0.144	0.0001	-0.9
TG279	6	0.135	0.0003	-1.2
TG552	6	0.114	0.0023	-1.2
TG99	6	0.123	0.0024	-1.2
TG336	6	0.079	0.0055	-1.4
TG253	6	0.095	0.0072	-1.0
CT111	8	0.065	0.0128	-1.1
CT68	8	0.080	0.0171	0.5
CT265	8	0.065	0.0226	-0.4
TG330	8	0.055	0.028	-0.4
TG434	8	0.066	0.029	0.3

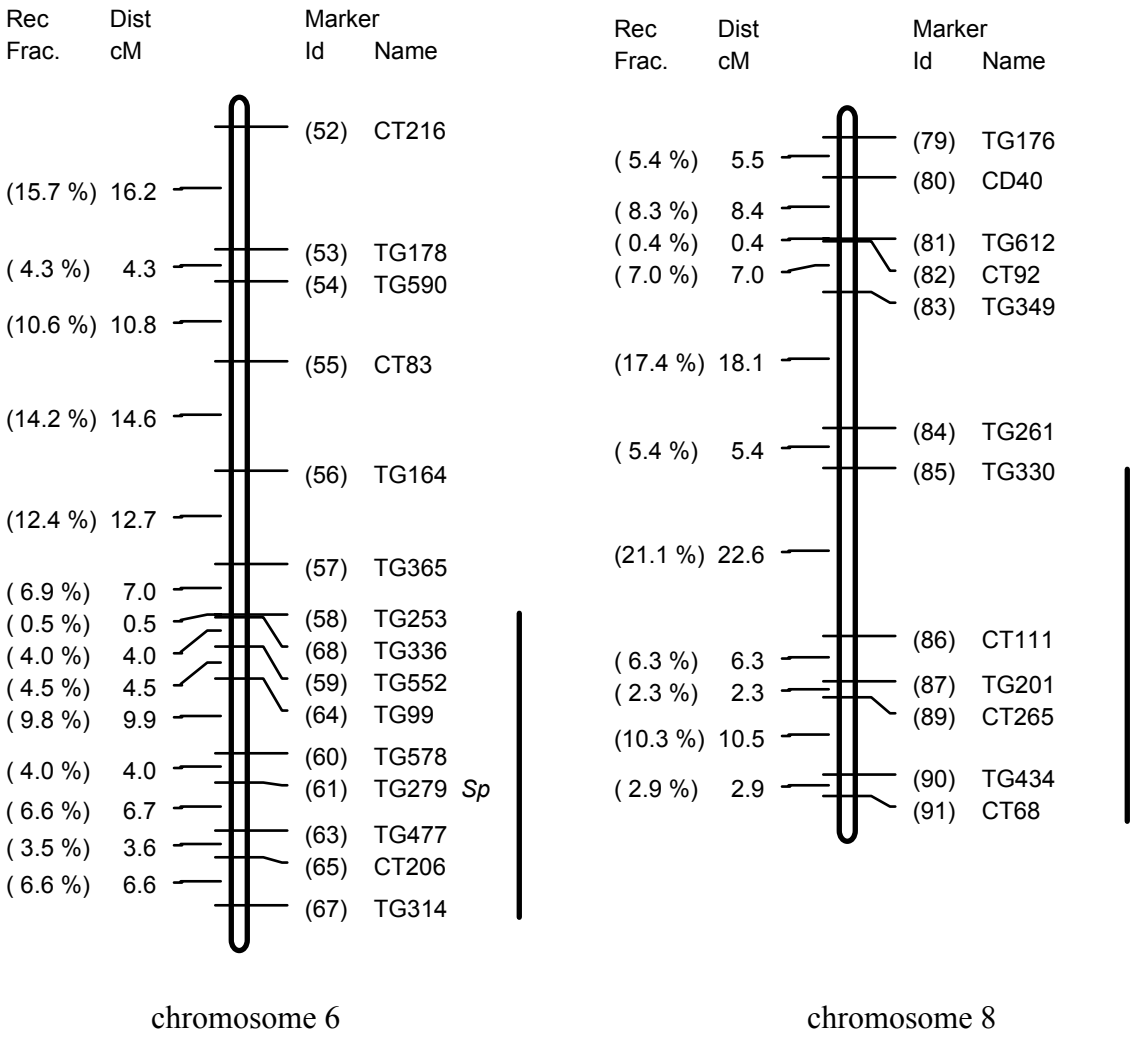

Figure 2. Maps of RFLP markers located on chromosome 6 and 8. The black bars indicate the regions with significant markers for late blight resistance.

Mapping of tomato genes associated with sugar metabolism

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Introduction:
Increased soluble solids is an important breeding target for the tomato processing industry.
A number of studies have been aimed at understanding the factors underlying quantitative traits for this character (QTLs)(1, 2); mostly relying on populations into which genes from high solids wild *Lycopersicon* species had been introgressed. Since soluble solids in tomato are primarily sugars (largely glucose and fructose) it was of interest to map a number of genes involved in sugar metabolism to see if these might be candidate genes underlying QTLs giving elevated soluble solids. If this proved to be the case these genes might enable more targeted introgressions to be produced from high solids wild species(3). Alternatively these genes might be targets for genetic manipulation (4). Here we describe the mapping studies of a number of genes thought to be involved in sugar metabolism.

Materials and Methods:
Probes: In general probes for specific genes were isolated by PCR.The available protein sequence from a number of plant sources were aligned and degenerate PCR primers designed corresponding to regions of amino acid sequence conservation. Templates were either genomic or cDNA. The resulting PCR fragment was sequenced to confirm its identity.

Sucrose Phosphate synthase (SPS): Based on the published sequences of SPS proteins and cDNAs (from maize, spinach and potato) the following primers were designed 5'GATTCTGATAC(A/T)GGTTGG(C/T)CAGGT3' and 5'TGG(A/C)CG(A/G)CA(C/T)TC(T/A)CCAA3'. A fragment of 1700bp was amplified from tomato genomic DNA and contained both intron and exon sequences. The exon sequences had 94% homology to potato SPS sequence and so probably correspond to a tomato SPS gene.

Hexose transporter: primers 5'TTGGTCATGGGGCCCAGACGGATGG3' and 5'GGACTCTGATTCCCATAGG3' amplified a 236bp fragment of a tomato hexose transporter from tomato cDNA.

Sucrose transporter: Primers were designed from the sequence of the sucrose transporter in the data base (Accession no. X82275) 5'CTTGATGTTGCTAATAACATG3' and 5'AAAGGGAAACCACGCAATCCA3' which amplified a 420bb fragment from cDNA.

Apoplastic Invertase: a tomato apoplastic invertase was identified in our laboratory using RT-PCR (primers 5'AGCCATTTGATTCTAGTTGGGCTG3' and 5'TCATAGTTGTTTCATTCTTTAGG3' amplify a 230bp fragment), it corresponds to gene lin6 recently reported by others(5) (accession X91390).

Vacuolar invertase: this gene has been mapped by others (6), we have confirmed its location close to TG102.

Invertase inhibitor: primers were designed using published sequences (7) 5'AAGAACACACCGAATTACCATTTCTG3' and 5'ACCTGATGATCCGACCATTCCATCTT3' and amplified a fragment of 306bp using cDNA as a template.

Sucrose synthase: Two tomato sucrose synthase genes have been identified. The sequences of the coding regions of these genes are highly homologous so allele specific probes were designed from the 3'untranslated (3'UT). One gene corresponds to that described previously (8), (GenBank accession L19762), this sequence is

very similar to a sucrose synthase (sus4) isolated from potato (9). Tomato genomic DNA was used as a PCR template using primers designed from the 3'UT region of this gene (5'GAAAGACTACTGACATCGGCTG3' and 5' CAATGTTTCTTGATACAAAA3'). A second sucrose synthase in tomato was identified (highly homologous to potato sus3) (9) (S.Chengappa in preparation) and a gene specific probe isolated using 3'RACE using the 5' specific primer 5'GACCGGCTGTTGACACTAG3' and fruit cDNA as a template.

Mapping: the position of the genes on the tomato map was determined using a population of chromosome segment introgressions of *L.pennellii* in an *L.esculentum* background (10). Briefly, restriction enzymes were identified that revealed an RFLP between *L.esculentum* (M82)and *L.pennellii* (LA 716) for each of the probes. This enzyme was used to digest DNA from each member of the mapping population which were electrophore-sed and Southern blotted. Map position could easily be identified from a polymorphism arising from a particular *L.pennellii* segment. In the case of the hexose transporter where the identified *L.pennellii* segment was rather large further mapping was carried out with a small F2 population from *a L.chmielewskii x L.esculentum* cross.

Results and discussion:

Mapping: The segmental introgressions containing the gene are given in the table:

Gene	*L.pennellii* segment	note
Sucrose synthase (sus3)	IL 12-3, IL 12-4	Accession AJ011319
Sucrose synthase (sus4)	IL 7-4	Accession L19762
SPS	IL 7-4, IL 7-5	Centromeric to TG 13A
Sucrose Transporter	IL 11-2, IL 11-3	Accession X82275
Hexose Transporter	IL 9-3	Accession AJ010942
Apoplastic invertase	IL 10-2, IL 10-3	Accession X91390
Invertase inhibitor	IL 12-4	Accession AJ010943
Vacuolar invertase	IL 3-2	Close to TG102

In the majority of cases it is possible to map the genes to a fairly small chromosomal segment with a resolution that exceeds most QTL mapping. The exception is the hexose transporter which lies on the large segment IL 9-3. This was positioned more accurately by mapping in a small *L.esculentumx L.chmielewskii* F2 population, the closest RFLP marker TG 348 is approximately 6cM away. The position of SPS could be refined more closely as the *L.chmielewskii* allele of SPS is not contained in line LA1501, which contains a telomeric region (7T) derived from *L.chmielewskii* (11) so the SPS gene lies centromeric to TG13A.
It remains to be seen if any of these genes could be candidate genes underlying QTLs for high soluble solids (1, 2, 12).

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Extreme segregation distortion seen in a *Lycopersicon pennellii* backcross

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Segregation distortion favoring the wild allele has been previously noted in interspecific crosses of tomato, including those involving *Lycopersicon pennellii* (Zamir and Tadmor, 1986; Chetelat and DeVerna, 1991 among others). We found an extreme case in a BC2 of *L. pennellii* LA1657 to *L.esculentum* E6203. The population was developed as part of our advanced backcross program. Molecular markers were scored at approximately 10 cM intervals to enable us to place QTLs on the map later (unpublished data). In a BC2, the expected level of heterozygosity at any locus is 25%. In this population, markers in the upper telomeric region of chromosome 12 were as much as 93% heterozygous. Five markers covering more than 30 cM were significantly ($p < 0.0001$) skewed toward the *pennellii* allele with a gradual decrease in percent heterozygosity the closer they were to the centromere (Figure 1). The other markers (CT99 and beyond) on this chromosome did not show significant skewing.

A similar phenomenon was seen previously in a *L. peruvianum* LA1708 BC3 at the upper telomeric region of chromosome 9 where the maximum heterozygosity was 88% at GP39 and also decreased as markers neared the centromere (Fulton et al. 1997). This region had been noted much earlier and postulated to contain a gamete promoter gene (*Gp*) (Pelham 1968).

Non-Mendelian segregations such as these bring up many interesting questions about meiotic drive and preferable inheritance. In these two cases it is also interesting to note that both regions are located at telomeres, perhaps signifying something functionally related to telomeres. In any case, for practical uses of interspecific crosses where breaking linkages may be necessary (eg. introgressing a small region only), skewed segregations can mean extra backcrossing and therefore need to be considered in such a breeding program.

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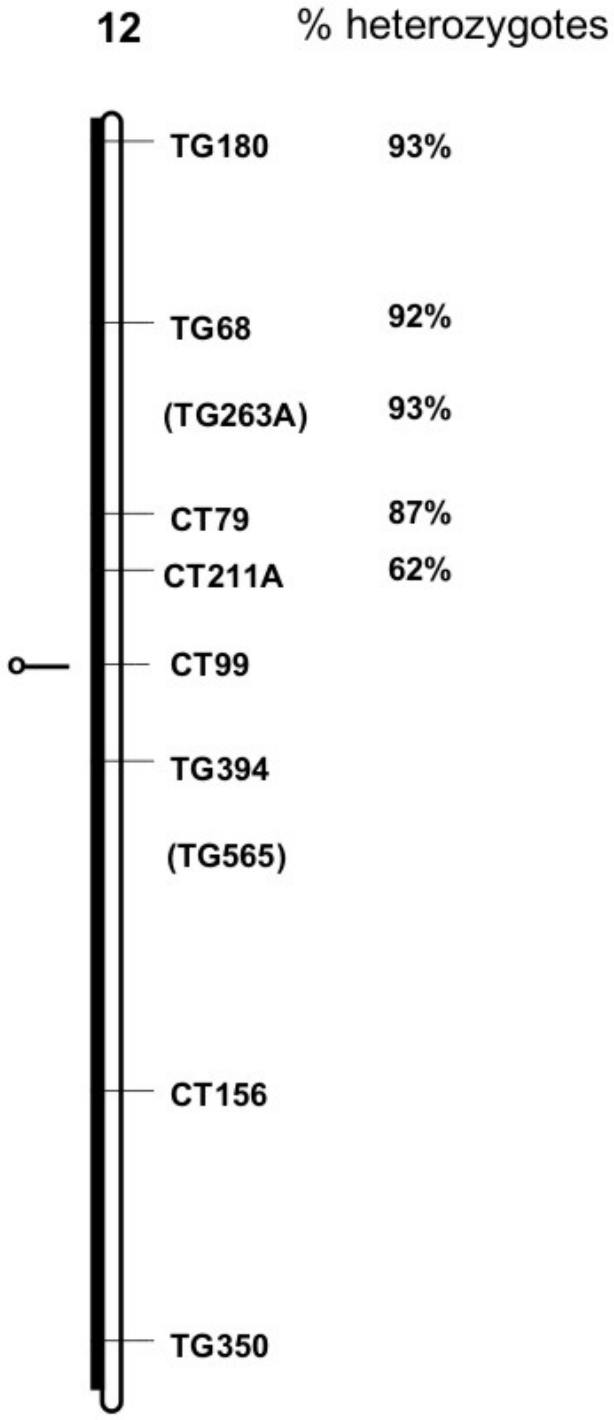

Figure 1. Percent of heterozygous plants in markers showing significant ($p < 0.0001$) skewing toward *L.pennellii* alleles.

Metabolic changes in tomato pollen after infection with *Clavibacter michiganensis* subsp. *michiganensis*

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The bacterial canker of tomato caused by *Clavibacter michiganensis* subsp. *michiganensis* is tracheobacterioses. The development of bacterium promote the appearance of plugs in xylem vessels and the death of tracheas and their neighboring cells (Sotirova, Bogatsevska, 1990). Symptoms include decreased vigor, stunting, leaf yellowing and wilt. The disturbances in reproductive organs are not well understood. It was reported that inoculation with *C. michiganensis* is associated with abnormalities in meiosis and decrease in pollen viability (Sotirova, Beliva, 1978). In the present study the changes in activities of some oxireductases in pollen grains of infected tomato plants were investigated cytochemically to reveal the response of tomato pollen to the bacterial invasion.

Material and methods. For the investigation, pollen of tomato (*Lycopersicon esculentum* cv. *Ideal*) plants, which are susceptible to *C.michiganensis*, was used. The inoculation was carried out with a 10 CFU/ml bacterial suspension made by 36h purified culture of *C.michiganensis* subsp. *michiganensis* grown on PDA at a temperature of 24C. The pollen was collected from plants with severely expressed symptoms. Pollen from healthy plants was investigated as a control.

The dehydrogenases isocitrate dehydrogenase (IDH), glutamate dehidrogenase (GDH), alcohol dehydrogenase (ADH), lactate dehydrogenase (LDH and glucose-6-phosphate dehidrogenase (G6PDH) were determined by the method of tetrazolium reductases. The cytochrome oxidase (CO) was proved by NADI reaction (Lojda et al, 1979). The peroxidase (POD) was detected by the method of Craham and Karnovski, modification of Roels et al (1975). The pollen viability was assessed using the fluorochromatic (FCR) test (Heslop-Harrison et al, 1984).

The effect of the bacterial infection was estimated by determination of the percentage of pollen grains with positive cytochemical reactions. For each sample a minimum 300 pollen grains were counted. The statistical significance of the differences was estimated by Student's t-test.

Results and discussion. The cytochemical investigation showed that the bacterial infection was associated with a decrease in the percentage of the pollen grains with IDH and GDT activities. At the same time the activity of CO in pollen grains of infected plants did not change (Fig 1). Obviously, the pathogen invasion provoked disturbances in the oxidative intramitochondrial metabolism decreasing the activities of dehydrogenases taking part in the Krebs' tricarbolic acid cycle. The activity of ADH involved in the pathway of anaerobic glucose degradation dropped too. However, LDH activity was enhanced which indicated activation of another anaerobic pathway of the anaerobic glycolysis. (Fig. 1). These data support the suggestion that the energetic metabolism of pollen of tomato plants infected with *C.michiganensis* became more dependent on anaerobic glucose degradation. The decreased percentage of viable pollen grains (Fig. 1) may be the result of changes in these metabolic pathways, connected with energy production. We have found similar disturbances in the pollen glucose catabolism in tomato plants infected with CMV (Georgieva, Stoimenova, 1998).

The activity of G6PDH in pollen of *C.michiganensis* inoculated plants fell drastically (Fig. 1) as opposed to the pollen of the CMV infected tomatoes (Georgieva, Stoimenova, 1998), where the activation of the oxidate pentose phosphate pathway is associated presumably with formation of pentosophosphates for biosynthesis of virus RNA.

The increase of percentage of pollen grains with POD activity after the bacterial infection (Fig. 1) is probably a response reaction of pollen to the biotic stress caused by the bacterial invasion. Besides, it was proposed that *C.michiganenses* infected tomato plants are under constant water stress (Van Alfan, 1989). Our data are in agreement with this statement and the findings of Zhang and Kirkham (1994) which established increased POD activity in drought stressed wheat plants.

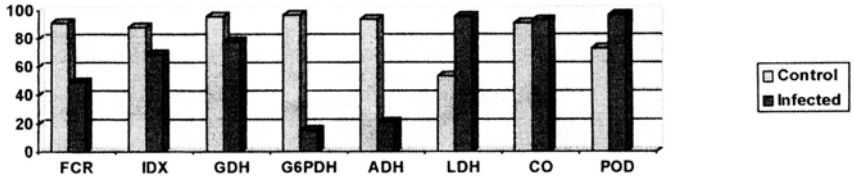

Figure 1. Changes of viability and activities of the oxireductases in pollen of C. michiganensis subsp. Michiganensis infected tomato plants expressed as percentage of pollen grains with positive cytochemical reaction.

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Round fruit allele of *fs8.1* is associated with reduced incidence of blossom-end rot in tomato fruit

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Blossom-end rot in tomato fruit is affected by several factors including inorganic nutrient status (Wui and Takano 1995), cultivar and temperature (Ho et al 1995).

fs8.1 is a major fruit shape QTL found to be highly conserved across different species, for example, *Lycopersicon esculentum* and *L. pimpinellifolium* (Grandillo et al., 1996), *L. pervianum* (Fulton et al., 1997) and *L. hirsutum* (Bernacchi et., 1997). In a BC4F2 population derived from a cross between *L. esculentum* and *L. pimpinellifolium*, most round fruit showed reduced incidence of blossom-end rot (BER) compared to the elongated fruit in the fine mapping study of *fs8.1* (Ku et al, unpublished data). A total of 25 BC4F3 homozygous recombinants between the interval of *fs8.1* region (TG176-CT92) were selected and evaluated for fruit shape and the level of BER. As shown in Table 1, TG45 was the most tightly linked marker to the fruit shape (R-square = 90%, P < 0.0001); however, blossom-end rot was shown to be more significant at CD40 (R-square = 37%, P= 0.0001) than TG45 (R-square = 30%, P=0.0006). To test whether fs8.1 has pleiotropic effects or another gene affecting BER is tightly linked to *fs8.1*, NILs containing only CD40 or TG45 BER will be developed and evaluated for fruit shape and BER differences.

Table 1. ANOVA test for the FS (fruit shape index = a ratio of fruit length to diameter) and BER at (A) TG45 and (B) CD40

(A) TG45

	R-square	P value	EE mean	PP mean
FS	90%	<0.0001	1.141	0.806
BER	30%	0.0006	2.341	1.792

(B) CD40

FS	77%	<0.0001	1.149	0.853
BER	47%	0.0001	2.521	1.822

EE mean: mean value of the trait when homozygous for *L. esculentum* alleles at the locus
PP: mean value of the trait when homozygous for *L. pimpinellifolium* alleles at the locus

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Molecular mapping of eight tomato *myb*-related genes in an interspecific cross (*L. esculentum* x *L. pennellii*)

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Abstract
Eight *myb*-related genes were further characterized by determining their map positions on a tomato molecular marker map. The eight genes map to a total of five different chromosomes.

Introduction
Transcription factors regulate the expression of genes during development and differentiation and are involved in the maintenance of cellular functions. Myb transcription factors bind to specific DNA sequences and activate or inactivate the promoters containing its binding sites (Biedenkapp et al., 1988; Nakagoshi et al., 1989; Sakura et al., 1989; Weston and Bishop, 1989). A large number of *myb*-related genes have been isolated from several plant species and are involved in a wide range of functions, such as flavonoid biosynthesis (Paz-Ares et al., 1987; Grotewold et al., 1994), trichome development (Oppenheimer et al., 1991; Larkin et al., 1994), dehydration stress (Urao et al., 1993) and cell shape determination (Noda et al., 1994). We have previously isolated 14 *myb*-related genes from tomato (Lin et al., 1996) and in this study we were interested to see where these genes are located on the genetic map of tomato. The determination of map position may aid further investigation of the Myb-related protein function(s) in cases where they are found to map close to known mutants or quantitative trait loci. Furthermore, the determination of map positions allows the *myb* genes themselves to be used as markers in breeding.

Materials and methods
Mapping population. A mapping population was obtained by selfing one individual F1 plant from an interspecific cross between individuals of *L. esculentum* cv Allround and *L. pennellii LA716* (Odinot et al., 1992).
RFLP analyses and linkage map. Fifty-one probes were used as RFLP markers for the evaluation of 84 plants. Based on RFLP-analyses of the 84 plants an RFLP-linkage map was calculated using JoinMap 1.4 (Stam 1993). The map positions of eight of the *myb*-related genes were calculated with the segregation data of 51 individuals which were randomly chosen from the 84 plants that were used to generate the RFLP-linkage map.
Results
None of the *myb*-related RFLPs showed a significantly distorted segregation. All eight *myb*-related genes were significantly linked with at least one TG-probe. The closest linkages and map order of the five chromosomes with at least one *myb*-related gene are given in Tables 1 and 2.

Table 1. Chromosome assignment of the THM probes and LOD-scores for the TG-probes showing the highest linkage

	LOD	Chromosome
pTHM03-TG259	9.8	1
pTHM05-TG275	13.7	6
pTHM06-TG234	3.7	2
pTHM13-TG154	13.7	2
pTHM16-TG523	15.6	11
pTHM18-TG25	11.5	6
pTHM24-TG178a	10.8	6
pTHM26-TG342	21.0	7

Table 2. Map positions of RFLPs and the eight *myb*-related genes on the relevant chromosomes. The corresponding map positions on the RFLP-linkage map of Tanksley et al. (1992) are given in parentheses.

Chromosome 1			Chromosome 2			Chromosome 6			Chromosome 7			Chromosome 11		
TG184	0	(9)	TG189	0	(2)	pTHM24	0		TG20A	0	(20)	TG523	0	(27)
TG71	34	(58)	pTHM06	24		TG178	8	(9)	TG170	17	(42)	pTHM16	5	
TG465	70	(90)	TG234	43	(41)	pTHM18	21		TG252	34	(61)	TG47	18	(45)
TG430B	82	(104)	TG337	78	(79)	TG25	28	(32)	pTHM26	68		TG46	31	(59)
TG258	110	(129)	pTHM13	99		TG151	32		TG342	69	(91)	TG30	41	(81)
pTHM03	113		TG154	104	(124)	TG275	57	(65)						
TG259	115	(133)				pTHM05	61							
						TG314	77	(97)						

Discussion

We compared the map positions of morphological mutants (Solgenes Genome Database) and the *myb*-related genes, but no obvious morphological mutation that might have been caused with changes in *myb* transcription factors mapped close to one of the eight mapped *myb*-related genes. However, the mapped genes in this report can be used as probes in further mapping studies, especially those involving mutations that might be due to changes in Myb transcription factors.

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Abbreviation: THM Tomato Hypocotyl Myb

Expression of the *pat* syndrome and other floral traits in a tomato BC_1 population after an interspecific cross with ***L. pennellii***

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The *pat* mutation, the first gene for parthenocarpy discovered in tomato (Bianchi and Soressi, 1969), has recently been described as causing a complex syndrome involving different aspects of flower and fruit development. This single recessive mutation was shown to affect stamen (reduced length and carpelloidy), ovule (arrested integument growth and inviability) and ovary (autonomous growth, i.e. parthenocarpy) development (Mazzucato et al., 1998). With the long term objective of positional cloning, we have obtained a large BC_1 seed stock after an interspecific cross between a tomato line homozygous for the *pat* allele (Chico III background) and *L. pennellii* (LA716). A single F_1 plant was used as staminate parent in the backcross to Chico III-*pat* plants. A first population of 68 BC_1 plants has been characterized in relation to the segregation and the expression of *pat* and other floral traits. On a single plant basis, sepal (SN), petal (PN) and stamen (StN) number, stigma exsertion (SE) and type (ST), percentage of aberrant anthers (AA) and ovules (AO), fruit set (FS), fruit weight (FW) and number of seeds per fruit (SpF) were recorded. For comparison, *pat* expression was studied under the same environmental conditions on a small population of Chico III-*pat* plants.

The *pat* genotype was assigned to plants concurrently showing an aberrant phenotype on anther and ovule development. Allowance was made for a maximum 2% aberrancy in wild type (WT) plants. On the whole the BC_1 population showed undistorted segregation at the *pat* locus; 30 WT versus 34 *pat* plants ((^{2}=0.25, P>0.5). Four plants remained unscored; two showed WT anthers with *pat* ovules and two had *pat* anthers and WT ovules. The distribution of *pat* expressivity on anthers and ovules was different in the BC_1 population from that detected on plants expressing *pat* in a totally *esculentum* background (table 1). BC_1 *pat* plants were highly variable for anther aberrancy, ranging from 3 to 100%, with a concentration in the extreme classes, while *esculentum pat* plants had a distribution skewed towards the low frequency class. Ovule aberrancy grouped BC_1 plants towards low-frequency classes, while *esculentum* plants were normally distributed around a mean of 49% (table 1). Different than these floral traits, parthenocarpy (precocious ovary development and high fruit set) was not clearly expressed in BC_1 plants; out of 34 *pat* segregants, only three were parthenocarpic since the first inflorescences and four expressed the character later. Moreover, fruitfulness was not related to the *pat* phenotype; on the whole population, 8 *pat* and 9 WT plants were able to set fruits.

Table 1. Distribution of *pat* expressivity on anther and ovule development in the BC_1-*pennellii* and in the *esculentum* background.

Trait	Genetic background	Frequency classes				
		> 2-20%	21-40%	41-60%	61-80%	81-100%
Aberrant anthers	BC_1	11	5	4	6	10
	esculentum	13	5	2	0	0
Aberrant ovules	BC_1	16	10	6	3	1
	esculentum	3	7	12	6	3

Correlation analysis was carried out independently for WT and *pat* BC_1 plants (table 2): correlation coefficients involving variables FS, FW and SpF were calculated only for plants that set fruits. Highly significant correlations were found among organ numbers (SN, PN and StN) and between these variables and FS, both in the WT and *pat* group. Higher organ number is a trait associated with *pat* expression (e.g. on average 6.0 stamens per flower in *pat* and 5.5 in WT *esculentum* plants) and therefore derived from the *esculentum* genome in our material. Its correlation with FS and its expression in BC_1 was however unrelated to *pat*. Organ numbers were inversely related to the *pennellii*-type stigma (ST) also in both the series of plants. Higher fruit set therefore appears, independently from the expression of the *pat* allele, related to the inheritance of *esculentum* traits. This behavior supports the hypothesis of flower morphology traits being linked together in *Lycopersicon* species (Bernacchi and Tanksley, 1997). Exserted stigma was inversely related to fruit weight in the WT plants which in turn was associated with seed set: in *pat* plants, SE did not affect FW, since parthenocarpic fruit development does not necessarily need pollination, but the eventual seed set was also strongly dependent on the degree of exsertion (table 2). While AA and AO were not correlated in *pat* plants, they were strongly associated in the WT group. Although the latter relationship is obvious (most WT plants have no aberrant anther or ovule at all), lack of correlation between AA and AO in *pat* plants strengthens the belief that the common genetic determinant of these traits (Philouze and Pecaut, 1986) has downstream consequences that are very different in the development of male and female organs. Moreover, the distribution of *pat* expression (table 1) also suggests that a 25%-*pennellii* genetic background quantitatively affects organ formation in mutant tomato flowers. Therefore *pat* expression is affected to a different extent by genetic modifiers and external factors (our unpublished results).

Table 2. Significant correlations among floral traits independently calculated for *pat* (top-right) and WT (bottom-left) BC_1 plants.

	SN	PN	StN	SE(1)	ST(2)	AA(3)	AO(4)	FS(5)	FW	SpF
SN	-	0.90***	0.79***		-0.39*			0.86**		
PN	0.70***	-	0.65***					0.80*		
StN	0.48***	0.83***	-		-0.37*			0.91**		
SE				-		0.55***				-0.93**
ST		-0.41*	-0.43**		-					
AA						-				
AO						0.91***	-			
FS		0.69*	0.77*					-		
FW				-0.82*					-	
SpF									0.76*	-

(1) Scored on a scale 1-4: 1, inserted; 2, flush with the anther cone tip; 3, 1-2 mm exserted; 4, 3-4 mm exserted.
(2) Scored: 1, *esculentum*-type; 2, *pennellii*-type.
0) Percentage calculated on at least 4 flowers per plant.
1) Percentage calculated on at least 3 flowers per plant.
(5) Recorded on 3 to 5 fruiting inflorescences per plant.

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Morphological, cytological, biochemical and molecular analyses of cytoplasmic male sterile form in genus *Lycopersicon*

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In this study we investigate the morphological, cytological, biochemical and molecular characteristics of the CMS in the late backcross BC10 - P2 (*L. peruvianum* x *L. pennellii*) (CMS-*pennellii*) originating from the described CMS line (Vulkova Z. 1980). The comparative study of the phenotype of the CMS-*pennellii* and the nuclear donor *L. pennellii* showed that the only phenotypic difference is established in the flowers of CMS-*pennellii*. They differ from those of the recurrent parent by the following features: the corolla size is smaller, pale yellow in color, and the petals are not well opened; the anthers are reduced in size with the mean anther length 2.3 mm, pale green, and are not coalesced laterally to form a normal staminal cone. The anthers do not shed pollen. CMS-*pennellii* plants are female fertile. The fruit and seed set are similar to the fertile analogue. The meiotic division in pollen mother cells of CMS-*pennellii* is regular including the tetrad stage. The microspore degeneration takes place after the tetrad disintegration and the pollen stainability in acetocarmine is 0 %.

A comparative study between CMS-*pennellii* and *L. pennellii* have been carried out for the following enzymes: malate dehydrogenase (MDH), maleic enzyme (ME), peroxidase (PRX), superoxide dismutase (SOD), glutamate oxaloacetate transaminase (GOT) and esterase (EST) extracted from young leaf tissue. Allozymes of CMS-*pennellii* could be distinguished by PAGE from the fertile analogue by the pattern of the isoforms for MDH, ME, EST. New fractions appear in the electrophoretic spectrum of SOD and MDH extracted from anthers of CMS-*pennellii* in comparison with *L. pennellii*.

The study of the mitochondrial genome has been carried out on total DNA samples from CMS-*pennellii* and *L. peruvianum* digested with different restriction enzymes. As probes, cloned heterologous mitochondrial genes have been used: atp A , atp 6 , 18S+5S rRNA , nad 3 , cob , cox , cox III and clone 2 (Petunia S-Pcf locus including: cox II gene, urf-s, nad 3 gene, rps 12). RFLP between the CMS-*pennellii* and L. *peruvianum* cytoplasms has been established with: atp A (*Dra I*, *EcoRI/HindIII*), nad 3 (*EcoRI/HindIII*) and clone 2 (*HindIII*). The results demonstrate that atp A and nad 3 are proximal and situated on the same 3.6 kb *EcoRI/HindIII* fragment. Additional fragments are revealed in the hybridization profile of individual plants of *L. peruvianum* with atp A and clone 2 in comparison with CMS-*pennellii* plants, which indicates the presence of recombinant mitochondrial DNA molecules with low stoichiometry in the progenitor cytoplasm. The absence of these fragments from the RFLP pattern of CMS-*pennellii* can be considered as an evidence for the generation of diversity in the mitochondrial genome due to the interaction of the cytoplasm of *L. peruvianum* and the nuclear genome of *L. pennellii*.

The long-term aim of our study is to develop a CMS system for cultivated tomato. Because the direct hybridization of CMS-*pennellii* with *L. esculentum* is impossible due to the unilateral incompatibility barriers, we have used the hybrid F1 (*L. esculentum* x *L. pennellii*) as pollinator as described in Stoeva P. (1980). Via the obtained complex hybrids we have been able to perform direct crosses with *L. esculentum*. These unique hybrids are the base for the development of maintainer and restorer lines.

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ASSAYING EXTRACTION PROCEDURES FOR RAPID AND RELIABLE DOT BLOT TYLCV DIAGNOSIS

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Tomato Yellow Leaf Curl Virus (TYLCV) is a whitefly-transmitted geminivirus which causes serious economic losses to tomato crops all over the world (Picó *et al*., 1996). Early detection of TYLCV is necessary to prevent field epidemics. Traditional diagnosis which relied on symptoms produced on indicator plants by grafting or by vector transmission is time-consuming and nonaccurate. More recently, molecular hybridization (squash, dot or southern blotting) using radioactive probes has been suggested. However, the alkaline transfer blotting analysis is time-consuming and the simpler direct tissue-squash blotting is unsuitable for quantitative comparisons. Dot blot analysis has been succesfully used as an appropriate and sensitive technique for qualitative and quantitative detection of TYLCV (Czosneck *et al*., 1988; Crespi *et al*., 1991; Rom *et al*., 1993; Hajimorad *et al*., 1996). However, DNA extraction procedure can be a major bottleneck when using this technique for large scale analysis.

We have compare three extraction procedures of TYLCV (EP1-EP2-EP3) from fresh leaf samples of susceptible and partially resistant tomato varieties and wild tomato accessions (Table1). Plants at the fourth-leaf stage were exposed to 15 viruliferous whiteflies/plant for 4 days in whitefly-protected cages. Samples of fresh tissue from top leaves were collected at 15 days post inoculation. Symptom development was monitored at the same time. Sap and DNA extraction were performed as described (Table 1). 5 ul of each sample and three 1/10 serially diluted extracts (corresponding to 5, 0.5, 0.05, 0.005 mg of fresh leaf tissue) were spotted onto Nylon membranes for dot spot hybridization assays. Membranes were hybridized overnight at 65ºC and washed once in 2x SSC-2% SDS at room temperature and twice in 0.1x SSC-0.1%SDS at 60ºC. The DNA probe employed, spanning the entire genome of a TYLCV-isolate (belonging to the TYLCV-Sar subgroup), was radiolabelled by random priming. The amount of viral DNA in each sample was calculated according to a standard curve of known concentration of TYLCV dotted onto the same membrane.

The limit of detection using this probe was about 5 pg of viral DNA. Differences in TYLCV accumulation were observed among susceptible and partially resistant material (Table1). Relative TYLCV accumulation in different materials was consistent using the three EP (*L. esculentum* FC> F1-*L.peruvianum* 143679> BC-*L.chilense* 1938> *L.peruvianum* 126935> *Lchilense* LA 1932). The three EP proved to be effective for successful detection of TYLCV in spots corresponding to 10 ug of leaf tissue of susceptible material FC. However, the amount of viral DNA obtained by the EP1 procedure was approximately 2-10 times lower than that obtained with the other EP. Thus, EP1 failed to extract detectable amount of viral DNA from the partially resistant *L.peruvianum* PI-126935.

Results indicated a lower accumulation of viral DNA in nonsymptomatic materials than in symptomatic. However, significant differences in DNA accumulation are not directly correlated with symptom severity in all cases (F1-*L.peruvianum* PI-143679 and *L.peruvianum* PI-126935) suggesting that symptom expression could depend more on the genetic background than on viral accumulation in some wild or wild-derived partially resistant materials.

Both EP2 and EP3 procedures detected TYLCV DNA in all the samples except for *L.chilense* LA 1932. EP2, a Dellaporta-based extraction method, proved to be useful for detecting the presence of TYLCV using very small amounts of tissue. Thus, this method provided viral DNA suitable to be used as template for PCR, a more sensitive technique. EP3 extracted TYLCV DNA slightly less efficiently than EP2 in most cases, but this procedure is simpler and more rapid and could be very useful for large scale analysis.

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Table 1. Detectable DNA amounts extracted from fresh leaf tissues using three extraction procedures

Accession	Characteristics and source	EP1*-[a]	EP2*-[b]	EP3*-[c]	Symptoms severity-[d]
***L.esculentum* FC**	Susceptible control UPV Genebank	0,4	3	2,8	4
F1 *L.peruvianum* PI-143679	Interspecific hybrid FCx *L.peruvianum* PI 143679 (an asymptomatic TYLCV carrier accession) UPV Genebank	0,1	1,1	0,7	1
BC-*L.chilense* 1938	Second backcross to FC from the cross *L.esculentum x L chilense* LA 1938 Provided by Dr. J.W.Scott University of Florida	0,12	0,6	0,2	2
***L.peruvianum* PI-129365**	Asymptomatic carrier of TYLCV. Source of resistance of the TY-hybrid series obtained in Israel (Pilowsky and Cohen,1990) Provided by M.Pilowsky. The Volcani Center, Israel	ND	0,1	0,12	0,5
***L.chilense* LA 1932**	Provided by C.M. Rick. The Tomato Genetics Stock Center. USA.	ND	ND	ND	0

* Viral DNA pgr/ fresh tissue ugr.
Data are means of three replicates
ND: Non Detected

In all cases fresh tissue was frozen in liquid nitrogen and ground to a fine powder

a) EP1: Leaf lysate (Czosneck *et al*., 1988): Crush tissue in boiling extraction buffer (2% CTAB, 100mM TrisHCL (pH 8), 10mM EDTA, 1,4M NaCl-1)-Add 1% 2-mercaptoethanol Boil for 1 min and incubate for 5 min at 55ºC. Extract the aqueous phase after washing with phenol:chloroform-isoamyl 24:1.

b) EP2: DNA extraction (modified from Dellaporta *et al* 1983 according to Crespi *et al*., 1991): Crush tissue in extraction buffer (100mM Tris-HCL pH 8, 50 mM EDTA, 500mM NaCl, 10mM B-mercaptoethanol, 1% SDS). Incubate at 65 ºC for 5 min. Add 5 M potassium acetate. Incubate on ice for 10 min. Centrifuge 10min. Precipitate with isopropanol. Wash with 70% and 100% ethanol, dry and resuspend in TE.
c) EP3: Sap extract (Hajimorad *et al*., 1996). Crush tissue in extraction buffer (50mM Tris- HCL, 1 mM EDTA pH 8). Add 1M NaOH. Incubate at room temperature for 30 min. Centrifuge for 10 min.

d) 0: no symptoms; 1:slight symptoms; 2: moderate symptoms; 3: severe symptoms; 4: very severe symptoms.

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Obtaining transgenic tomato (*Lycopersicon esculentum* Mill.) and potato (*Solanum tuberosum* L.) by transfer of the *ugt* gene from corn

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The general purpose of this project was the creation of transgenic forms of plants, in which the transfer of genes coding basic enzymes of IAA metabolism results in the increase of biosynthesis of indoleacetic acid (IAA) and its conjugation. As a result, the content of IAA will be increased, supported by these enzymes and balanced at a high physiological level. Previous work on transgenic plants, involving genes for modification of the hormonal status of plants and, hence, varying their growth and development, is practically nonexistent.

The *ugt* gene in endosperm of developing corn kernel encodes UDPG-transferase which conjugates IAA to glucose, transforming it into an alkali labile stored form. Due to high velocities of synthesis and effective linkage in the corn kernel there is an adjustment to a high concentration of IAA.

The creation of transgenic fast-growing fruitful forms of tomato or potato is a significant task not only for unfavourable growing season regions, like East Siberia, but also for agriculture in environmental stressed conditions and vegetable manufacturing factories using both tomato and potato as basic foods.

For producing transgenic plants, tomato varieties Ventura (Italian origin) and Verlioka (Russian origin) were used. In seedlings and cuttings of tomato the gene *ugt* was transferred by using the agrobacterial vector with the transconjugant of triparental mating: *A. tumefaciens* 699 (*gus-int, nptII*, mod35S), *E. coli* DH5alfa (*ugt* gene encoding UDPG-transferase), *E. coli* K802 (*gus*, 35S, *ugt* of bacterial origin). Infected seedlings and cuttings were cultivated on MS agar medium with 0.6 mg/l indolebutyric acid for a few days and then transferred to auxin free media for acclimatization before put them in soil. Higher rooting capacity was observed in the transgenics and the length and weight of roots were 2-4 times more than in the control plants (Table 1).

Table 1. Amount of roots and root weight of transformed and control tomato plants *Lycopersicon esculentum* Mill. var. Ventura

Roots	Transformed	Control
	After 5 days of cuttings transfer to fresh medium	
Number*	19.7±3.8	8.9±4.2
	After 3 weeks of growth in vitro	
Number	62.8±11.0	27.6±11.0
Total weight (g)	0.95±0.33	0.21±0.17
1 root weight (mg)	15.1±5.0	7.6±6.0

* The length of roots of transformed plants are twice as long than that of control plants.
The data are the average of 4 vessels with 4 plants each.

The transformed tomatoes were transferred to the ground and morphometric analysis was carried out. Growth and development of transformed tomatoes occurred almost 2 times faster (Fig 1). The transgenic had more leaf area (11-12 and 3-4 cm^2 in the transgenic and in the control, respectively), and amount of flowers and fruits exceeded the control by 2-3 times. Another characteristic of the transformed tomatoes was the formation of root initials which were wide spread on all the stems. Expression of the target gene has been found in genomic DNA from roots and stem with root initials by Southern blot (Table 2). In transgenic tomatoes the activity of marker enzymes GUS and NPTII has been revealed. Specific activity of UDPG-transferase in transgenic plants reached 514 nmol / mg of protein, in the control 148 nmol / mg of protein. Seeds of transgenic tomato plants grown in the greenhouse were obtained. The germination of these seeds occurred 2-3 days earlier in comparison with the control. The tomato plants are under study to evaluate the capacity to maintain the *ugt* gene expression.

Table 2. **Intensity of the ^{32}P-radioactivity spots of Southern blotting of genomic DNAs from *L. esculentum* probed with *ugt* gene.**

Plants	**^{32}P intensity pixel unit***		
	***L. esculentum* Mill. var. Ventura**		
	Leaves	Stem	Roots
Control	0	0	906
Transformed	1025	3876	1028

* The intensity of spots has been integrated with Jandel SigmaScan software

Several varieties of *S. tuberosum* L. were also transformed by insertion of the *ugt* gene from corn using the agrobacterial vector in the same way as for tomato. The transgenic potatoes showed rapid rooting and faster plant growth. Furthermore, enzyme activities and IAA content have been assayed in this species. Activity of non specific UDPG-transferase was found in both types of plants, but in transgenic plants the conjugation of IAA to glucose in vitro was highly effective. Also, the contents of free and bound IAA were higher in transgenic plants (Table 3). Characteristic of *ugt* transgenic plants, both tomato and potato, was the lowered % of dry matter in comparison to control. This is consistent with the general point of view about the physiological role of endogenous IAA in stimulation of growth by cell enlargement acting as sink for the increase of water content. The harvest of transgenic potato was 1.5-3 times higher than in the control potato plants.

Table 3. IAA content, free fraction and after NaOH hydrolysis, in *Solanum tuberosum* L. plants, after two weeks of in vitro growth (ng/g)

	free	ester bound	tissue bound	total
		var. Borodianskiy		
control	0.9801	0.000	174.173	113.849
ugt transformed	1.8285	3.999	237.920	134.061
		var. Puschkinetz		
control	0.7936	0.000	144.225	54.772
ugt transformed	1.9761	2.199	177.564	63.736

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Figure 1. Control and *ugt* transformed tomato plants. Left: control and right: *ugt* transformed, after 1 month in soil.

New alleles at old loci.

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In 1997 Dr. Neelima Sinha reported to us her observation that the leaf morphology of *ics* (incisifolia) resembles that of *clau* (clausa). Since stocks of each were growing at the time, crosses were attempted between LA1166 (multiple marker stock with *clau*, *su*^{3}, *ag*, *icn*) _ and LA3713 (*ics* in 'Ailsa Craig' background) as _. The resultant F_1 seeds were sown and all the 10 progeny displayed *clau* morphology. The absence of the other markers of LA1166 proved that they were not accidental selfs. Since *clau* has priority, it is suggested that the symbol of *ics* be changed to *clau*^{ics}.

In autumn, 1997, one of our single-plant progenies of cv. Rheinlands Ruhm (LA535) segregated for a mutant with the typical dwarf (*d*) syndrome except that it was extremely modified, even more stunted than *d*^{x}, scarcely growing 20cm tall in the course of a year in the greenhouse. Leaves are reduced to small, dark green globs of highly curled, rugose tissue. Flowers are functional but very reduced with tiny, scale-like corolla segments. Hand-selfing or crossing induces the setting of small 3-4cm fruit with seeds of normal size and morphology. Reciprocal crosses of this line with LA1700 (*wv*, *aa*, *d*) yielded progenies of 15 and 25 plants of uniform *d* morphology, none with other markers of LA1700. The new mutant is therefore an allele of *d*.

In April, 1998, plants of this new dwarf were exhibited at the Cal Aggie Picnic Day, a traditional campus event, and visitors were invited to suggest names for it. The many outlandish suggested names included 'Tomacolly', 'Tomabrocugly', 'Bromato', 'Spinocoli', and 'Wannabe Broccoli'. The resemblance to broccoli thereby manifested prompts us to propose the name of broccoli allele of dwarf with the symbol *d*^{b}. Although it can be reproduced by selfing at the expense of considerable time and effort, we shall maintain it via heterozygotes in 'Rheinlands Ruhm' background.

Efficiency of AFLP markers to saturate a tomato intraspecific map

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In order to map QTLs controlling fruit quality components of fresh market tomato, we have constructed a molecular linkage map using RFLP, RAPD and AFLP markers. To efficiently scan the genome for QTLs, we needed a core map with markers regularly distributed. We thus compared the efficiency of RFLP, RAPD, and AFLP markers to reach this objective.

*A population of 144 recombinant inbred lines (F_7 plants) was developed from a cross between an elite line for fresh market tomato (*L. esculentum*) and a cherry tomato line (*L. esculentum *var.* cerasiforme*) with good chemical and aroma characteristics.*
We first selected 600 RFLP probes regularly dispersed on the reference tomato map (Tanksley et al, 1992) and screened the 2 parental lines for polymorphism, using 6 restriction enzymes. About 30% of the probes revealed polymorphism with at least one enzyme and 136 were mapped on the RIL population. No major differences in locus order were observed with previous maps, although differences in recombination fractions were frequently observed, with two regions where recombination frequencies are much higher than in the reference tomato map. A few gaps remained on the map.
We then screened the parental lines for polymorphism with 126 RAPD primers, among which 48 revealed at least one polymorphic band. On a subset of the population, we mapped 28 RAPD loci to 9 linkage groups. These groups appeared mainly located around centromeric regions. The largest cluster contained 12 loci within 10 cM. This result is consistent with Grandillo and Tanksley (1996). Finally, only 3 RAPD loci were kept on the core map.

To test AFLP markers, the parental lines were screened with 46 HindIII-MseI *primer combinations (PC), and 20 PCs were selected (based on the number of polymorphic bands and the ease in scoring the profiles), which allowed us to score a total of 231 polymorphic bands (between 8 and 22 bands per PC). We looked for allelic and cosegregating bands within PCs. After discarding the uninformative and distorted markers, the number of loci was reduced to 174. These loci were frequently clustered: the 174 loci were grouped in 47 5 cM-clusters (containing 1 to 34 loci), or 82 1 cM-clusters (containing 1 to 27 loci). The distribution of AFLP markers compared to RFLP markers across the chromosomes is shown figure 1. The main clusters were observed in centromeric regions of chromosomes 2, 4, 5, 8 and 9, with clusters of 8, 22, 18, 18 and 31 loci, respectively. As PCs were cross products of 4* Hind*III and 8* Mse*I primers, we tested the homogeneity of the locus distributions across the chromosomes, according to the primers. Distributions were homogeneous between* Hind*III primers ($P<0.30$), but different* Mse*I primers revealed different chromosome distributions of the loci ($P<0.01$).*

The present core map comprises 94 RFLP, 3 RAPD, 1 morphological and 30 AFLP markers, that span 1265 Haldane cM, with an average distance between markers of 9.7 cM. It covers about 85% of the tomato genome, as fragments of 50, 40 and 60 cM remain uncovered on chromosomes 5, 9, and 11, respectively (distances based on the map developed by Tanksley et al, 1992).
With a high number of polymorphic bands revealed per PC, AFLPs may be the most efficient markers to rapidly saturate a map. In tomato, this efficiency is limited in regard to the non-random distribution of markers across the chromosomes and their preferential localization around putative centromeres. If no RFLP markers had been mapped in our experiment, about 57 loci would have been useful for the core map construction (figure 1), leading to an average efficient locus number per PC of 2.8. Nevertheless, even if the apparent marker cost is increased, AFLP is much faster than RFLP. Finally, the non-random distribution of AFLP markers needs to be confirmed with other primer x enzyme combinations. The use of enzymes which preferentially cut in coding regions may be helpful in improving the distribution of AFLP markers in tomato. A methylation sensitive enzyme, like Pst*I, or enzymes with cutting sequences enriched in GC, like* Taq*I or* Sac*I, should be compared to* Eco*RI-*Mse*I.*

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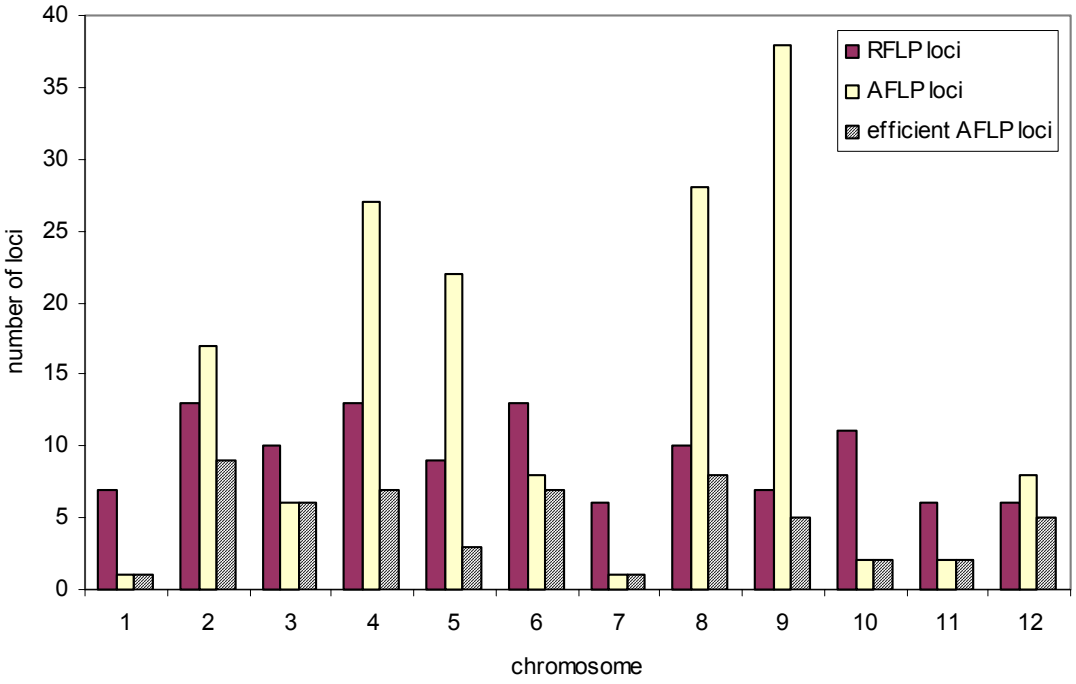

Figure 1: *Distributions of the number of AFLP and RFLP loci across the chromosomes. Efficient AFLP represent the number of AFLP loci which could have been useful for the construction of a core map if no RFLP markers were available.*

Apparent recovery in two *Lycopersicon hirsutum* accessions infected by tomato spotted wilt virus (TSWV).

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Tomato spotted wilt virus (TSWV) is one of the most polyphagous plant viruses. It can induce a wide variety of symptoms including chlorotic and necrotic spots, stunting and malformation in its hosts, and can cause serious diseases.

This virus causes significant losses of yield and quality in tomato production in all producing areas of Spain. At present, all available forms of chemical or cultural control are either ineffective or not economically viable. Genetic resistance is one of the most promising alternatives to control the disease caused by this virus (Roselló ***et al***., 1996).

Wild *Lycopersicon* spp. have gradually increased their importance as a source of genetic variability for tomato improvement. Resistance to TSWV has been reported in accessions of *L. peruvianum* and *L. hirsutum* (Maluf et al., 1991). In this work we describe the response to TSWV infection in *Lycopersicon hirsutum* accessions ECU-434 y ECU-436.

Plants of both accessions were maintained in a growth chamber with a 14 h light cycle (25ºC/18ºC) and (60-65%/95-100%) relative humidity day/night. Grolux fluorescent tubes (Sylvania) providing 65-85 mol photons m^{-2}!s^{-1} (at a wave-length between 400 and 700 nm) were used. Inoculum of TSWV isolate HA-931100 was maintained on susceptible plants of tomato line NE-1 and the susceptible pepper cultivar Negral. Virus transmission was carried out by thrips to avoid the appearance of viral defective forms. Inoculum was prepared by grinding infected leaves of susceptible NE-1 tomato line in cold 0.1 M phosphate buffer, pH 7.0, containing 0.2% sodium sulfite and 0.2% sodium diethyldithiocarbamate in a proportion of 1:5 (w:v). Carborundum (600 mesh) was added at a concentration of 1%. A cotton-tipped applicator dipped in inoculum and lightly rubbed on the third expanded leaf of the fourth leaf stage plants delivered inoculum. Inoculation was performed on sixteen plants of ECU-434 and nineteen plants of ECU-436. Twelve plants of line NE-1 were included as control plants. Plants were scored visually for TSWV symptoms and tested using DAS-ELISA with BR-01 antiserum at 4, 8, 14, 24, 36 and 51 days post inoculation (DPI). The absorbance was measured by a Titertek Multiskan MCC/340 photometer (405 nm). Two plants of accession ECU-434 died and were discarded in the experiment.

First symptoms appeared at 8 DPI and became progressively more severe. Control plants showed visible symptoms after 14 DPI, consisting of distorted and curled leaves. In the resistant accessions, symptoms consisted of distorted and puckered leaves and curling at the apex. From 14 DPI on, symptoms became less severe or disappeared entirely in some plants. The apex of the plants recovered and new healthy leaves emerged. In the end, all plants recovered from infection and showed no symptoms of infection. At 4 DPI all control plants were ELISA-positive. After that, absorbance values increased up to a value of 2.5 (Table 1). At this same date, some resistant plants gave DAS-ELISA- positive values. The mean absorbance values increased to 14 DPI. From this moment, absorbance values decreased and several plants were ELISA-negative. For ECU-434 accession, only one plant gave positive values of absorbance at 51 DPI. In ECU-436 plants, four of them were ELISA-positive but showed no symptoms of disease. The genetic basis of this recovery phenomenon has not yet been elucidated. The recovery of *L. hirsutum* plants may occur as a result of some active mechanism of the plant, exhaustion of compounds essential for virus multiplication, plant developmental factors or a combination of them (Al-Kaff and Covey, 1995). Additional studies involving a greater number of TSWV isolates will be necessary to determine if the observed resistance of these *L. hirsutum* lines is isolate-specific. Additional studies are also needed to elucidate their genetic control and the resistance mechanism involved in this recovery phenomenon.

Table 1: Number of plants of the accessions showing TSWV infection on tested plants as determined by DAS-ELISA tests at various times after inoculation.

Accession	Days Post Inoculation (DPI)					
	4	8	14	24	36	51
NE-1	12/12 (1.037)*	12/12 (2.448)	12/12 (2.615)	12/12 (2.515)	12/12 (2.492)	12/12 (2.521)
ECU-434	4/14 (0.760)	10/14 (2.609)	14/14 (2.389)	6/14 (2.066)	6/14 (0.978)	1/14 (0.522)
ECU-436	3/19 (1.711)	16/19 (2.177)	18/19 (2.766)	13/19 (2.265)	5/19 (2.108)	4/19 (0.438)

* values between brackets are mean of absorbance of ELISA-positive plants.

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The response of tomato lines to tomato and pepper-tomato pathotypes of Xanthomonas vesicatoria

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Bacterial spot of tomato (Lycopersicon esculentum) incited by Xanthomonas vesicatoria = Xanthomonas campestris pv. vesicatoria (Xv) causes significant losses reducing both yield and grade of fruit (Sotirova and Beleva, 1975; Goode and Sasser, 1980; Gitaitis et al 1992). Three pathotypes of Xv have been distinguished on the basis of virulence of tomato and pepper host plants namely tomato (XvT), pepper (XvP) and pepper-tomato (XvPT) pathotypes (Reifschneider et al., 1985; Minsavage et al., 1990). Two pathotypes were discovered in Bulgaria- tomato XvT and pepper-tomato XvPT (Bogatzevska and Sotirova, 1992).

The response of 40 lines obtained from a three genome hybrid (L. esculentum- L. chilense- L. peruvianum var. humifusum) to tomato and pepper-tomato pathotypes of bacterial spot was studied. Plants in the phase of 5-6 true leaves were inoculated by the vacuum infiltration method (Bogatzevska, 1988). The hypersensitive reaction (HR) was recorded 24h after infiltration of bacteria. The evaluation of disease severity was made by the Sotirova and Beleva scale (1975). The average number of spots per plant was calculated also.

The response of lines to both pathotypes of bacterial spot is given in Table 1. Lines LCH: 36/1; 44/1; 165/2 were symptomless. They manifested HR 24h after infiltration of bacteria to both pathotypes of Xv. Some lines (LCH: 168/1; 168/3; 168/4) were symptomless and manifested HR to the tomato pathotype, and LCH: 161/5; 166/4; 167/1; 169/1; 171/4; 172/2- to the pepper-tomato pathotype. The number of plants with low degree of disease severity (0,01- 0.99) was higher with inoculation of the tomato rather than the pepper-tomato pathotype. Lines LCH: 36/3; 166/6; 17/2 had low degree of disease severity to both pathotypes.

TABLE 1. Reaction of lines from three genome hybrid (L. esculentum- L. chilense- L. peruvianum var. humifusum) to tomato and pepper-tomato pathotypes of Xanthomnas vesicatoria.

Lines	XvT					XvPT				
	T	SI	HR	x	s	T	SI	HR	x	s
36/1	25	0	25	0	0	25	0	25	0	0
36/3	20	0	15	0.70	3.4	30	0	27	0.20	1.8
36/5	25	0	12	1.30	15	25	0	10	1.56	14
44/1	25	0	5	0	0	0	0	25	0	0
44/4	32	0	27	0.30	2	20	0	9	1.15	14
44/6	20	0	8	1.00	16	20	0	7	1.80	21
47/3	25	0	11	1.30	11	25	0	9	1.48	18
50/1	20	0	9	1.35	15	32	0	7	1.47	14
62/2	25	0	12	1.38	17	22	0	9	1.27	13
160/1	20	0	16	0.40	3	25	0	11	1.24	14
160/2	20	0	4	1.80	20	30	0	7	1.43	13
161/5	25	0	18	0.48	3	25	0	25	0	0
161/7	30	0	21	1.56	13	26	0	8	1.73	21
163/2	25	0	16	0.70	10	20	0	8	1.15	11
163/3	30	0	24	0.30	3	30	0	13	1.40	17
163/4	30	0	27	0.10	1	25	0	8	1.48	116
164/6	20	0	6	1.90	19	20	0	10	1.25	14
164/7	30	0	18	0.70	8	21	0	11	1.05	13
165/2	25	0	25	0	0	25	0	25	0	1.96
165/3	40	0	22	0.90	3	26	0	6		19
166/4	25	0	24	0.08	0.4	20	0	20	0	0
166/5	25	0	21	0.32	2.1	25	0	0	2.40	17
166/6	25	0	22	0.20	1.9	20	0	19	0.10	0.3
167/1	25	0	22	0.24	0.3	20	0	20	0	0
167/2	250	0	24	0.04	0.7	25	0	19	0.46	2.8
167/3	25	0	20	0.30	2.6	25	0	0	2.36	19
168/1	25	0	25	0	0	25	0	22	0.30	1.3
168/3	20	0	20	0	0	25	0	23	0.12	0.8
168/4	25	0	25	0	0	25	0	23	0.16	0.8
169/2	25	0	23	0.16	1	25	0	25	0	0
170/3	25	0	14	0.92	10	30	0	10	1.37	15
171/4	25	0	22	0.16	1	20	0	20	0	0
171/5	25	0	13	1.13	4	25	0	25	0	0
172/2	25	0	18	0.48	1	25	0	25	0	0
172/4	30	0	16	0.96	9.5	26	0	11	1.16	11
173/3	25	0	8	1.30	5	20	0	12	0.95	11
174/2	20	0	7	1.30	11	30	0	13	1.13	11
175/1	30	0	6	1.80	24	20	0	10	0.95	4
176/3	35	0	13	1.34	14	35	0	11	1.43	13
177/1	30	0	14	1.30	13	30	0	21	0.43	3.1

T - total number of plants; SI - number of symptomless plants; HR - plants with hypersensitive reaction; x - average grade of disease; s - average number of spots per plant.

Our investigation indicates that some of the lines are resistant to the tomato pathotype and others to the pepper-tomato pathotype. The lines of most practical importance are LCH: 36/1; 44/1; 165/2 which are resistant to both pathotypes. These lines are distinguished by abundant pollen production and high percentage of fruit set under unfavorable climatic conditions (Sotirova et al., 1990). They can be used for breeding resistance programs in tomato.

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Cmr*, a gene controlling resistance to cucumber mosaic virus (CMV) in *L. chilense

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Cucumber mosaic is one of the most devastating virus diseases affecting commercial tomato cultivation in temperate regions, such as the northern Mediterranean. Natural sources of resistance have been found amongst wild *Lycopersicon* species (Laterrot, 1980; Ciccaresse et al., 1987, Stamova et al., 1990; Stoimenova et al., 1992) but their incorporation into cultivated tomato has been slow, and so far no varieties with such resistance are available. Gebre-Selassie et al. (1990) revealed *L. hirsutum* PI 247087 to be more resistant than tested accessions of *L. pimpinellifolium*, *L. chmielewskii*, *L. pennellii*, *L. hirsutum* and *L. peruvianum*. Parella et al. (1997) suggested one gene was involved in this late-expressing resistance and described special conditions for screening. Stamova et al. (1990a), searching for resistance to Bulgarian and Hungarian isolates of CMV, found a high level of resistance in BC_1-inbred progenies from the cross *L. esculentum* 'Ace' x *L. chilense* LA0458. Some plants were virus-free according to ELISA tests. We subsequently found these BC_1 progenies were also resistant to Californian isolate 113A, a shoestring strain of CMV subgroup I from the Huron area. Some plants had undetectable CMV RNA levels, tested by high stringency Dot Blot hybridization.

To study the inheritance of this resistance, we made crosses between a resistant line, 97L6824, and the susceptible cvs. 'VF36' and 'Vendor Tm-2^a'. Plants of the original resistant line, F_1 and F_2 populations were grown under greenhouse conditions. They were mechanically inoculated at the two-leaf stage with isolate 113A. To avoid escapes the plants were re-inoculated 10 days later. 'VF36' and 'Vendor Tm-2^a' were used as susceptible controls. All plants were scored for visual symptoms 20 days after the first inoculation using a scale of 0 - 4 (0 = no symptoms and 4 = severe symptoms, including shoestring leaves). Plants with scores of 0 and 1 (very mild mosaic symptoms) were considered resistant, while plants with scores of 2, 3 and 4 were considered susceptible.

'VF36' and 'Vendor Tm-2^a' developed systemic symptoms within 2-4 weeks (e.g. mosaic, leaf wrinkling and curling, fern leaves and shoestrings) (Table 1). Some of the resistant F_2 plants appeared to be virus-free after performing Compound Direct ELISA tests. Others were symptomless despite having a virus titer. Some plants developed symptoms 2 months after inoculation, whereas other plants, originally showing symptoms, seemed to recover. All F_1 plants were considered resistant (Table1), showing only mild symptoms on a few plants. We conclude the resistance is dominant, and provides effective protection in the heterozygous condition. In the F_2 population, segregation was consistent ($X^2_{(3:1)} = 2.668$, not significant) with a 3 resistant: 1 susceptible ratio at the 95% confidence level (Table1), suggesting a major gene is involved with resistance to CMV. We therefore propose the symbol *Cmr* (cucumber mosaic resistance) for this apparently monogenic trait.

There are indications that the gene we are working with differs from the resistance in *L. hirsutum*. Unlike *L. hirsutum* PI 247087 whose resistance was reported to be expressed most clearly in 45 day old plants grown at 20°C (Parella et al., 1997), our plants resist infection in the juvenile stage under normal greenhouse conditions.

Table 1. Reaction to CMV inoculation in susceptible controls, resistant line 97L6824, F_1 and F_2 populations.

Genotypes	Segregation				$\chi^2_{(3:1)}$
	Observed		Expected		
	R	S	R	S	
'VF36'	0	25	0	25	
'Vendor Tm-2[a]'	0	25	0	25	
97L6824	17	0	17	0	
F_1 (6824 x 'VF36')	20	0	20	0	
F_2 (6824 x 'VF36')	288	77	274	91	2.668

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Lycopene content in some Bulgarian tomato lines

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Recent studies have focused on the role of carotenoids and especially on lycopene as the major carotenoid in tomato and tomato products and its role in prevention of some human diseases such as cancer and cardiovascular diseases ((Lowe et al., 1998).

The natural abundance of lycopene in fresh tomato fruits varies on a large scale and it is influenced considerably by environment (Daskaloff et al., 1990). Tomes (1963) has shown the inhibition of lycopene synthesis under temperatures above 30° C. There are not many reports on the inheritance of lycopene and different genetic controls of lycopene synthesis have been suggested (Wann and Mc Ferran, 1960; Thomson and Helper, 1962; Daskaloff et al., 1976; Hirota et al., 1994).

For many years in "Maritsa" Institute considerable efforts were ongoing to enhance mainly β- carotene content in some of our breeding materials. From segregating populations of the cross Ace x (Ace x *L .chilense*) lines were stabilized with β-carotene accounting for over 90% of the total pigments, as well as lines with a high content of lycopene and vitamin C (Manuelyan et al., 1975, 1993).

During the summer of 1998 we have evaluated 35 determinate tomato lines for their total carotenoids, β-carotene and lycopene content. The lines were grown in the field as a middle season crop. They are without *L. chilense* in their pedigrees and also do not have *hp* character. The first 24 numbers under study (Table 1) represent breeding lines with round, middle or big sized fruits and the other numbers are peeled-type tomatoes. We used as a control UC 82A, a variety that is still in use in Bulgaria. Tomato fruits were harvested at full red stage and the content of carotenoids were determined using spectrophotometric method (Manuelyan, 1967). The data (Table 1) represent the average carotenoid contents of tomato samples from two replications, each one of 50 fruits.

The concentration of total carotenoids, including β-carotene and lycopene is not very high and is quite variable: - 2.79 -8.10 mg % for total carotenoids, 0.24 - 1.26 mg % for β-carotene and lycopene ranges from 2.10 - 6.95 mg %. It is worth mentioning that we have had very high temperatures for two months this summer (30-40° C). Compared with UC 82A (3.83 mg % lycopene) eight lines have shown much larger amounts of lycopene - higher than 6.00 mg %. Four of them are from the round fruited group - 146, 216, 171 and 531 with 6.03, 6.12, 6.48 and 6.95 mg % respectively. Another four lines - 459, 460, 472 and 523 from the peeled tomato group, also had high lycopene concentrations - 6.66, 6.92, 6.54 and 6.54 respectively. It should be mentioned also that there were six other lines with a lycopene content higher than 5.5 mg %, especially lines 589 and 481, both with 5.88 mg % lycopene.

All lines that have been evaluated for carotenoids content have resistance to verticillium wilt and fusarium wilt (races 1 or 1 and 2) and some lines have other resistances in addition. We intend to study the inheritance of lycopene in some of our lines showing relatively higher amounts of lycopene.

Table 1. Total carotenoids, β-carotene and lycopene content in some tomato lines

Lines	Total carotenoids, mg%	β-carotene, mg%	Lycopene, mg%	Lines	Total carotenoids, mg%	β-carotene, mg%	Lycopene, mg%
15	4.04	0.24	3.54	510	6.83	0.72	5.63
146	7.12	0.59	6.03	501	6.22	0.59	5.19
242	6.00	0.90	4.68	350	5.57	0.39	4.79
216	7.51	0.86	6.12	481	6.92	0.55	5.88
100	4.05	0.28	3.48	592	5.42	0.38	4.66
152	5.33	0.65	4.30	462	6.04	0.54	5.07
150	4.46	0.40	3.74	250	7.01	0.80	5.71
168	3.42	0.46	2.72	300	5.50	0.40	4.72
198	2.79	0.49	2.10	549	6.21	0.65	5.12
200	5.42	0.52	4.51	658	5.43	-	5.26
311	6.98	0.94	5.55	638	5.46	0.98	4.10
262	4.82	0.39	4.08	459	7.66	0.46	6.66
171	7.81	0.78	6.48	460	7.96	0.48	6.92
240	6.80	0.67	5.66	472	7.93	0.83	6.54
243	6.04	1.26	4.36	523	7.98	0.88	6.54
531	8.10	0.58	6.95	589	7.17	0.79	5.88
476	5.40	0.32	4.70	UC 82A	4.63	0.47	3.83
664	6.16	0.25	5.48				

The authors would like to thank Prof. Dr. Sci. St. Stamov from the Higher Institute for Food and Flavor Industries (Plovdiv) for his assistance and support.

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Resistance to TSWV in transgenic tomato varieties

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The objective of the study was to introduce by genetic engineering genes conferring resistance to TSWV in commercial tomato genotypes.

On the basis of the developed genotype independent protocol for genetic transformation of tomato, the nucleoprotein (Np) gene of TSWV (Stoeva et al. 1998) and *Mn SOD* gene from *N. plumbaginifolia* (Bowler et al. 1991) were introduced in different tomato cultivars and lines. Primary transgenic plants were obtained with both constructs. All transgenic plants were selected for resistance to TSWV after mechanical inoculation with the TSWV Bulgarian tomato greenhouse isolate 1D-94. Resistant plants were selected from the primary transformants carrying the Np gene or the *Mn SOD* gene. *See errata* [1] Studies were carried out on derived R1-R3 progenies from the selected resistant primary transgenic plants carrying the *Mn SOD* gene and R1-R2 progenies carrying the N- gene from TSWV for:

- the expression of the NPT II selectable marker gene by studying the rooting ability of in vitro plants on kanamycin;
- PCR and Southern blot analysis for the NPT II selectable marker gene;
- PAGE analysis of the expression of *Mn SOD* gene;
- immunoprotein detection of the expression of Np-gene and (or) the viral accumulation by TAS or DAS-ELISA;
- resistance to TSWV by mechanical inoculation of transgenic plants.

The established resistance or tolerance to TSWV in the R1 progenies of transgenic plants carrying the *Mn SOD* gene varied from 54 to 80%. In R2 and in R3 100% TSWV tolerant lines were selected. The established resistance to TSWV in the R1 progenies of transgenic plants carrying the Np gene varied from 50 to 71%. In R2 progeny lines the resistance varied from 25 to 93%. Further studies are in progress to study the inheritance of the resistant and the tolerant phenotypes.

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[1] From Vol. 49, page 37. In TGC Report v. 48, p. 55: in Stoeva P., et al. "Resistance to TSWV in transgenic tomato varieties" we reported that full or partial resistance to TSWV was established in R1-R3 progenies from selected resistant primary tomato transformants carrying the *MnSOD* gene from *N. plumbaginifolia* and in R1 -R2 progenies from selected resistant primary transformants carrying the TSWV nucleoprotein (Np) gene. Further analysis (PCR analysis with specific primers for both transgenes and Southern blot analysis with the Np gene probe) has demonstrated the presence of the TSWV Np gene in all studied resistant transgenic plants. A technical mistake in primary seed material is assumed.

A novel tomato mutant with changed flower and fruit morphology (Giant)

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A tomato plant with unusual flower and fruit morphology has been observed in the R3 progeny of a transformant produced after Agrobacterium mediated transformation of a tomato plant from the Bulgarian cultivar Helios with a plant transformation vector carrying a manganese superoxide dismutase gene from *N. plumbaginifolia* (Bowler et al. 1991). The mutant phenotype is characterized by a change in the form, size and color of the petals and sepals and fruit shape. For all other characteristics the mutant is like cv. Helios.

The flowers have seven sepals, and petals that are leaf-like and with green color. The average length of the petal at anthesis is about 2.5 cm. At anthesis the sepals turn green yellow and are about 2.0 cm long. The green buds are normal and coalesced to form the characteristic staminal cone. The development of the ovary is accelerated in comparison to the anther development and determines the formation of parthenocarpic fruits. Due to the accelerated growth of the ovary the stamens at anthesis are separated. The plant is a diploid and the meiotic division in pollen mother cells is normal; the pollen is abundant and 100% stainable in 1% acetocarmine. The parthenocarpic fruits are smaller in size in comparison to the progenitor variety Helios and the rest of the sib plants from the R3 progeny line, usually with radish-like form with well preserved style. Some fasciated fruits have an unusual flower-like form and instead of the style inflorescence-like structures have been developed. The plant is proved to be transgenic by PCR and Southern blot analysis. Further studies are in progress to study the inheritance of the mutant phenotype and to study the pattern of transgene integration in the mutant and the rest of the transgenic plants from the R3 progeny line.

The GIANT mutant is a novel mutation for tomatoes - in the latest published list of tomato mutants no data is available for similar or comparable changes in the flower and fruit morphology phenotype.

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Bowler C., Slooten L. Vandenbranden S., Rycke R de, Botterman J., Sybesma C., Montague M. van, Inze D. 1991, EMBO J,10,1723-1732.

A gene for reduced lycopene on chromosome 12 of *Lycopersicon parviflorum*, possibly allelic to *Del*

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A gene causing yellowish fruit was found in a *L.parviflorum* LA2133 backcross population. The population was derived from a cross between LA2133 and *L. esculentum cv.* E6203 (the recurrent parent) as part of our advanced backcross program searching for new QTLs from wild species of tomato. The BC2, which was in the field in California (and other locations) in 1996, was segregating for yellow fruit. We scored this characteristic as a nominal trait where a score of 2 signified yellow fruit and a score of 1 signified red (not yellow) fruit. Chi-squared analysis showed a significant effect ($p < 0.0001$) at only one region, on chromosome 12 at TG565/TG111 (which are <1 cM apart in this population) (Figure 1). Yellow fruit were associated with the allele from the wild donor parent.

No other region had a significant effect on this trait, so it appears to be a single gene. Other data taken on this same population shows this same region to be also associated with a decrease in lycopene, but with no significant effect on beta-carotene. It is interesting to note that the same region of chromosome 12 was associated with a decrease in fruit color in another interspecific backcross using *L.peruvianum* as LA1708 the donor parent (Fulton et al. 1997), but in *L.peruvianum* this effect was seen as a quantitative decrease in red color, not as a yellow color.

Delta, a dominant mutation also causing yellowish fruit color and associated with a decrease in lycopene, has also been localized to this region of chromosome 12 (Zamir, personal comm) raising the possibility that the *parviflorum* gene reported here is a new allele of *Delta*..

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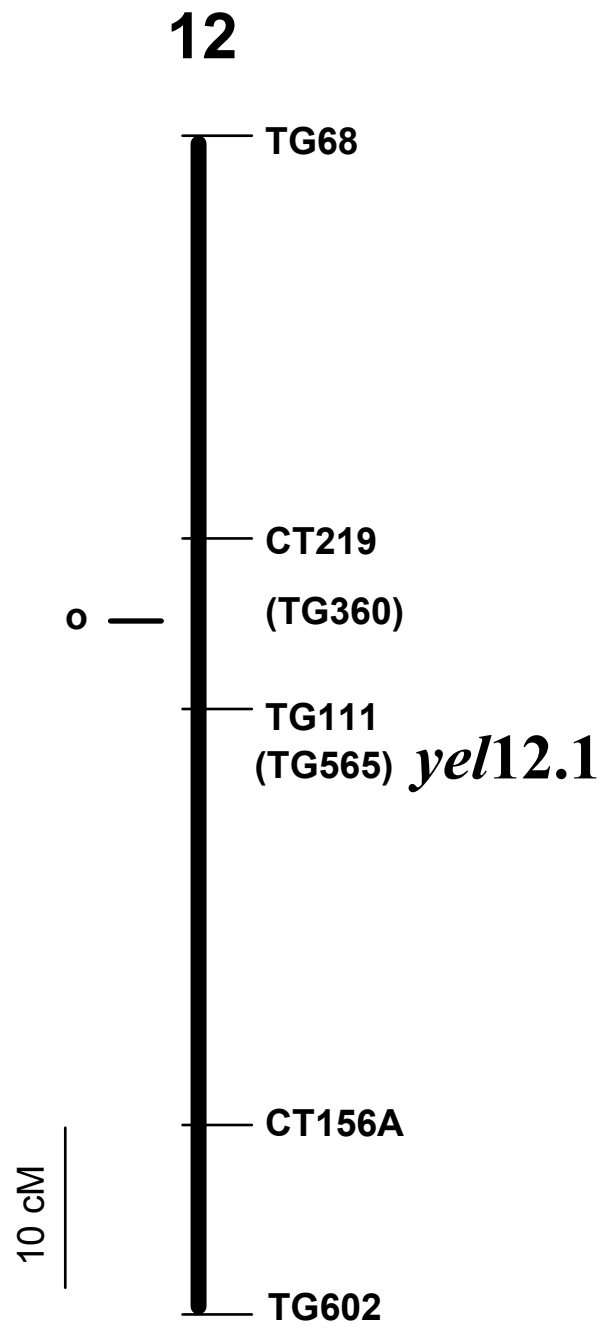

Figure 1. Ch12 of the *L.parviflorum* BC2 map showing the location of the yellow gene (*yel*12.1).

Comparing the performance of a set of processing lines nearly isogenic for the *rin* and *nor* mutations

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The *rin* (*ripening-inhibitor)* and *nor* (*non-ripening*) mutations both affect fruit ripening by apparently blocking steps prior to ethylene biosynthesis (Giovannoni et al., 1995). *rin* (when deployed in the heterozygous state) has proven very useful in prolonging the shelf life of fresh market tomatoes. However, thus far, to our knowledge, neither gene has been exploited for the development of processing tomato varieties. One possible benefit of using genes that inhibit ripening would be to increase the firmness and field holding capabilities of processing tomatoes.

To test the effects of *rin* and *nor* on the performance of processing tomatoes, we transferred each gene into the processing variety E6203 through 6 sequential backcross generations. Homozygous BC6S1 NILs were then established and each was crossed with TA496 (E6203 + Tm2a gene). The control was E6203 x TA496. The *rin*/+ NIL, *nor*/+ NIL and +/+ controls were transplanted into field plots of 20 plants each at standard densities in the summer of 1997. The test sites were in Akko, Israel (IS), Badajoz, Spain (SP), Woodland, CA (CS) and Numata, Japan (JP). There were 3 reps of the heterozygous NIL and 9 reps of the control at each site. The only exception was JP where there were 2 reps of each NIL and 6 reps of the control. Details of the field conditions and cultural practices can be found in Tanksley et al. (1997a). All plots were harvested at the end of the season and evaluated for plant growth/cover, maturity, stem scar size, fruit color (internal and external), fruit firmness, puffiness, pH, soluble solids, yield (total, red and green), brix*yield, fruit weight and viscosity. The methods used for these evaluations are described in Tanksley et al. (1997a). Analysis of variance was used to compare the performance of the control to each of the heterozygous NILs (*rin/*+ and *nor/+)* with respect to each of the measured traits. Table 1 summarizes those traits for which significant differences ($P< 0.1$) was observed.

Table 1. p values derived from the ANOVAs comparing the control (E6203, +/+) to each heterozygous NIL (*+/rin* and *+/nor*) over all 4 test sites (all sites) as well as individual sites for which additional traits were measured (single sites). The genotype with the higher value for the trait is listed in parenthesis beside the p value.

Trait	+/*rin*	+/*nor*
all sites:		
firmness.field	ns	<0.01 (+/*nor*)
stem scar	ns	0.04 (+/+)
external color	0.02 (+/+)	<0.01 (+/+)
internal color	ns	0.10 (+/+)
lab color	<0.01(+/+)	0.05 (+/+)
puffiness	ns	0.02 (+/+)
fruit weight	ns	0.02 (+/*nor*)
green yield	0.10 (+/*rin*)	0.05 (+/*nor*)
single sites:		
overmature (SP)	ns	0.05 (+/+)
firmness.lab (SP)	ns	0.02 (+/*nor*)
lycopene (CS)	ns	0.02 (+/+)
B-carotene (CS)	(0.2)(+/*rin*)	0.03 (+/*nor*)
total acidity (CS)	ns	0.04 (+/*nor*)
titratable acidity (CS)	ns	<0.01 (+/*nor*)
pH (SP)	0.01 (+/*rin*)	ns

+/rin versus +/+

The *rin* heterozygous NIL (+/*rin*) performed remarkably like the E6203 control (+/+) except for color and pH (one location, SP). Based on visual ratings, the external color of mature fruit was significantly diminished for *+/rin* compared with the control. Also, when measured in the lab (Agtron or La/b) the puree derived from +/*rin*. fruit had reduced color readings. However, the internal color of +/*rin* (rated by eye from sliced fruit) was not significantly different than the control. In other words, from an external view, the *rin* fruit was less red, but when one slices into the fruit, the internal portions have color of similar intensity to the wild type. Lycopene levels measured on puree of +/*rin* were not significantly different from the wildtype; however, the B-carotene levels of +/*rin* tended to be higher than the control, adding a slight orange cast to the fruit color.

Firmness was not enhanced by *rin*, either based on hand squeezing (firmness.field) or by measurements taken by an instrument in the lab (firmness.lab). *rin* also did not have any significant effect on field holding capacity as measured by the "overmature" ratings taken in Spain. *rin* significantly increased the fruit pH in one location (Spain).

+nor versus +/+

The effects of *nor* were much more noticeable. Firmness was significantly improved in all locations, whether measured by hand or by lab instrument. *nor* also improved the field holding capacity as measured by the "overmature" ratings in Spain. The percentage of diseased or rotten fruit was also generally decreased for the *+/nor* NIL in the two sites where it was measured (Japan and Spain), but did not reach the cut off significance level for the ANOVA.

External fruit color was drastically reduced by *nor*, but internal color was much less affected. Fruit that appeared greenish on the outside usually had red pigmentation developed on the inside. Unlike *rin*, *nor* reduced lycopene levels by approximately 35% compared with the control. At the same time, B-carotene levels were increased by approximately 50% over the control, giving the internal color an orangish appearance. Total acidity and titratable acidity were both increased by *nor*, but, surprisingly, the pH was not significantly different than the control. *nor* also decreased puffiness. This may be due to the fact that *nor* fruit do not soften as much as non-*nor* fruit and therefore the internal gel is much less likely to liquefy and create air pockets as can occur during ripening of non-*nor* fruit.

Conclusions:

At least in the E6203 genetic background, heterozygosity for the *rin* mutation does not significantly improve fruit firmness or field holding capacity. It does, however, decrease external color and may slightly increase the B-carotene levels. Being heterozygous for *nor* dramatically improves fruit firmness and may also increase field holding ability. *nor* also appears to reduce fruit loss due to post-ripening disease and decay. Acids are increased by *nor* as are the levels of B-carotene. Color reductions for both mutations appear to be most dramatic in the external part of the fruit, with the internal parts being much less affected. Neither mutation reduced yield, brix or viscosity.

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Mapping of the *pro* gene and revision of the classical map of chromosome 11.

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The classical maps of tomato, based on morphological markers, are mainly dependent on relatively old data. These data were combined to construct linkage maps, without taking into account their statistical accuracy and reliability. Revised classical maps, based on critically evaluated previously published data in combination with recent linkage data and often integrated with molecular markers based on RFLPs, have been generated for the short arm of chromosome 1 (Balint-Kurti et al., 1995; Van Tuinen et al., 1997), for chromosome 3 (Koornneef et al., 1993; Van der Biezen et al., 1994), chromosome 6 (Van Wordragen et al., 1996; Weide et al., 1993) and chromosome 10 (Van Tuinen et al., 1997). However, no revised and updated map has been published for chromosome 11. Having generated additional linkage data for the *procera* (*pro*) locus we decided to re-evaluate all data available in literature for chromosome 11 to compose a more reliable classical map.

Table 1 comprises the data collected for *pro* and several other chromosome 11 morphological markers by the authors. Data from the literature have been verified (*e.g*. recalculated with the computer program RECF2 (Koornneef and Stam, 1992)) and, combined with the data from Table 1, give the recombination percentages as presented in Table 2. These have been used to construct a revised classical map of chromosome 11 (Fig. 1), using the computer program JOINMAP (Stam, 1993).

Some literature data that have been found were not used to construct the revised map. The *a-hl* (Repulsion phase; Butler, 1952), *a-f* (R phase; MacArthur 1928 and 1934) and *hl-j* (R phase; Butler, 1952) data have been omitted, because they are most certainly part of the pooled data published by Butler in 1959. The *a-hl* (Coupling phase), *g-hl* and *g-a* data presented by Rick et al. (1959) are identical to those published by Brauer and Rick (1956), the latter ones are mentioned in Table 2. The recombination percentages for *j -f* and *j -a* (Butler 1936) could not be verified, because the original data were not published. However, together with the *a-f* data from MacArthur (1934) they are probably part of Butlers data (1951), as concluded from a note of the author, and have therefore not been added to the data used for Table 2. The *a-hl* (C phase) data from Laterrot (1976) have been excluded because of the strong distorted segregation in the F_2 populations. The *pro-a* and *pro-hl* (both BackCross) data from Lachman (1973) have not been taken into account, as they are in conflict with our results (Table 1) and those of Rick (1991)(Table 2). From the Rick and Martin (1961) data on *sub-a* linkage the largest population exhibits an excess of double recessive plants and has been excluded from the pooled data used to calculate the recombination percentage given in Table 2. The literature data report *Cf-3* to be unlinked to *a* (Kanwar, 1980) or weakly linked to *a* and *f* (Langford, 1937). Stronger linkage was found with *j-2* (Kanwar, 1980) and *j* (Langford, 1937). Whereas Langford (1937) mapped *Cf-1* and *Cf-2* not only to the right chromosomes, but also to their appropriate positions (Balint-Kurti et al., 1995; Van Wordragen et al., 1994), his data concerning *Cf-3* are considered trustworthy. However, Kanwar (1980) mapped at least *Cf-9* (Van der Beek et al., 1992) and *Cf-8* (Gerlagh et al., 1989) to the wrong chromosomes and the linkage of *Cf-3* with *j-2* (which probably does not map to chromosome 11; see discussion of the revised map) is suspicious. Jones and Jones (1991) found *ne-2* to be allelic to *neg*. The data for *ne-2* by Kerr (1967) have therefore been included as data for *neg*.

Loci that have been added to the map.

Five loci (*pt-4*, *v*, *pet*, *uni* and *ds*) provisionally mapped on chromosome 11 (Tanksley, 1993) have now been placed on the revised classical map, based on the data shown in Table 2. The sixth new arrival on the map is the *pro* locus which, although mentioned to be linked to *a* (Rick and Martin, 1960), had not been allocated to chromosome 11 (Tanksley, 1993).

Loci omitted from the map, but probably located on chromosome 11.

The locus *wv-3* has been excluded from the revised map for the following reasons. The linkage data with *neg* and *ini* (Borgnino et al., 1974) are based on a population with strongly distorted segregation, the *neg-ini* control data are lacking and *wv-3* is only linked to *neg*. More data are needed before a map position can be allocated. The *ele* locus has not been placed, because the segregation from two of the three populations described by Reeves et al. (1966) is very distorted. However, the data do indicate linkage with *a* and *hl*. For *apn* the only data available are those of Opeña et al. (1972), which represent one F_2 in R phase. Whereas the segregation is distorted and no *a-hl* data are given as control, they are not used to calculate a map position for *apn*, although, on the basis of the linkage with *hl*, *apn* is expected to be on chromosome 11. Rick et al. (1973) found linkage of *pcv* with *a* and *hl*, but did not include the *a-hl* control data. Therefore, the *pcv* data have not been used for construction of the revised map. Given the fact that out of three detected *pcv-hl* recombinants two are triple recessives the map position distal to *hl* proposed by Rick et al. (1973) seems reasonable. Absolute linkage with *a* was found for *mnt* (Rick, 1966). This was based on a small population in R phase and since these were the only data found in literature, *mnt* has been omitted from the revised map. Whereas the only available data for *id* report linkage with *a* (Hansen et al., 1962), it could not be placed on the revised map also. For *ini* linkage with *neg* and *a* was described by Zobel et al. (1970), but the segregation of the populations is strongly distorted and the *neg-a* data are lacking. However, *ini* probably resides in the vicinity of *a*. The only data found for *bi* reveal a strong linkage with *f* (Mertens and Burdick, 1954), but were not sufficient for placing *bi*. Until now four genes conferring resistance to *Fusarium oxysporum* in tomato have been described, *I* (Bohn and Tucker, 1939), *I-1* (Sarfatti et al., 1991), *I-2* (Cirulli and Alexander, 1966; Stall and Walter, 1965) and *I-3* (McGrath et al., 1987), of which *I* and *I-2* are believed to map on chromosome 11. Mapping *I* has given difficulties, because of the deviation from the expected ratios in crosses between susceptible and resistant lines (Gilles and Hutton, 1958; Hutton et al., 1947; Retig et al., 1967). One explanation was the linkage of *I* to a gametophytic factor ***x*** (Bohn and Tucker, 1940). However, Kedar et al. (1967) postulate that *I* could have pleiotropic effects on both resistance to *Fusarium* wilt and the effectiveness of the pollen grains. However, based on F_2 and BC populations of six different parent lines Alexander (1972) concludes that the supposed gametophytic factor is linked to *a* and *hl*. Whereas these data are difficult to verify and interpret *x* has been excluded from the revised classical map. Regarding *I*, linkage with *j* has been described by Paddock (1950). This finding is reinforced by the results of Sarfatti et al. (1991), who found weak associations between TG194 and the disease response, while mapping the *I-1* gene on the RFLP map, and concluded that *I* may be linked to TG194. Wing et al. (1994) found linkage between TG194 and *j*, which further confirms the mapping by Paddock (1950). However, more morphological data are needed to determine a more precise position of *I* on the classical map. Laterrot (1976) mapped *I-2*, using the morphological markers *a* and *hl*. The segregation between *a* and *hl* in all populations used, is so distorted that these data were not used to position *I-2* on the revised classical map. However, its location distal to *a* (Laterrot, 1976) corresponds rather well to the position found on the RFLP map (Sarfatti et al., 1989; Segal et al., 1992). Two other resistance genes have been reported to map to chromosome 11. The gene for resistance to *Orabranche aegyptiaca* (*Ora*), was found to be closely linked to *j* (Avdeyev and Shcherbinin, 1980 and 1997), but whereas additional data are lacking it could not be placed on the map. The gene conferring resistance to *Stemphylium* (*Sm*) for which only linkage with *I* (Dennett, 1950) and RFLP data (Behare et al., 1991) are available could therefore not be positioned on the revised classical map either. The reported linkage of *ms-3* with *a* (Pratt, 1958) indicates that *ms-3* resides on chromosome 11, although its exact position remains unknown. Kerr (1961) reported linkage of *mn* (in old symbols *min*) with *a* (a_1) and *j* (j_1), but found no recombinants in the small populations used. The data were not used for construction of the revised map, although *mn* locates probably between *j* and *a*. Linkage for *fgv* with *a* and *hl* is based on two populations with a strong distorted segregation, for which the *a-hl* linkage data are lacking (Borgnino et al., 1974). The data allow the speculation that *fgv* lies above *hl*. Rick and Robinson (1952) described *ap*. On the basis of unpublished *f-ap* linkage data from Butler and McArthur, Rick and Butler (1956) reported *ap* to map on chromosome 11. Since the linkage data on *ap* have not been published, a place on chromosome 11 could not be reconfirmed and is therefore tentative. According to Tanksley (1993), based on his own unpublished results, *Sod-1* is on chromosome 11.

Loci omitted from the map and probably not located on chromosome 11.

For *mon* the only data found in the literature are linkage data with *j*, *a* and *f* (Kerr, 1973). Except for the *mon-a*

population in C phase (41.8 ± 5.6), segregation of all other populations is distorted. Recalculation of the data reveals that the linkage with *f* and *j* is very weak (43.4 ± 5.2 and 47.1 ± 5.0, respectively). It seems therefore that *mon* does not belong to chromosome 11.

Linkage data for *j-2* are more abundant. However, these do indicate that the mapping of *j-2* does not show convincingly that this locus resides on chromosome 11. Rick (1960) mentioned linkage of *j-2* to *a*. However, Kerr (1966) found *j-2* to be unlinked to *f* and *j* and, more importantly, unlinked to *a* in an F_2 population in C phase. Lachman and Tristan (1973) confirmed *j-2* to be unlinked to *a* and found very weak linkage with *hl*. Avdeyev and Shcherbinin (1982) reported linkage of *j-2* with *Ora*. Whereas the linkage of *Ora* to *j* , which they reported previously (1980) and confirmed recently (1997), is very tight and no linkage has been found between *j-2* and *j* , we conclude, based on the above mentioned data, that *j-2* probably does not map to chromosome 11. Close linkage of *j-2* with *up* has been reported by Gardner (1981) and Kerr (1987). Since *j-2* most likely does not map to chromosome 11, the same is implied for *up*.

Concluding remarks

All available literature data for linkage analyses of loci assumed to map on chromosome 11 were collected and critically re-evaluated. New linkage data obtained for the *procera* mutation were added. Linkage data based on linkage of any marker with at least two other markers were used to construct a revised classical map of chromosome 11, by combining all available data with the software package JOINMAP (Stam, 1993). The further integration of the classical map with the RFLP map of tomato will allow the future cloning of genes identified thus far only by mutations resulting in an altered phenotype.

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Table 1. Additional linkage data for several classical markers on chromosome 11

Markers	Phase[a]	Genotype classes				Total	χ^2_{ass}[b]	Rec. %[c]
		A.B.	*aaB.*	*A.bb*	*aabb*			
pro - j	R	321	136	148	0	605	11.0	0.0 ± 4.1
pro - hl	R	203	91	67	2	363	23.1	17.8 ± 5.0
pro - a	C	241	56	53	50	400	34.6	31.6 ± 2.9
pro - a	R	184	79	86	14	363	9.8	36.6 ± 4.5
hl - a	C	1413	185	227	296	2271	455.4	22.4 ± 1.1
j - a	R	208	89	93	10	400	16.9	31.2 ± 4.4

[a] C: coupling phase; R: repulsion phase
[b] All data are significant at 0.01 significance level
[c] Rec. % is recombination percentage ± SE calculated with RECF2

Table 2. Data on linkage between classical markers used to construct the revised genetic map of tomato chromosome 11

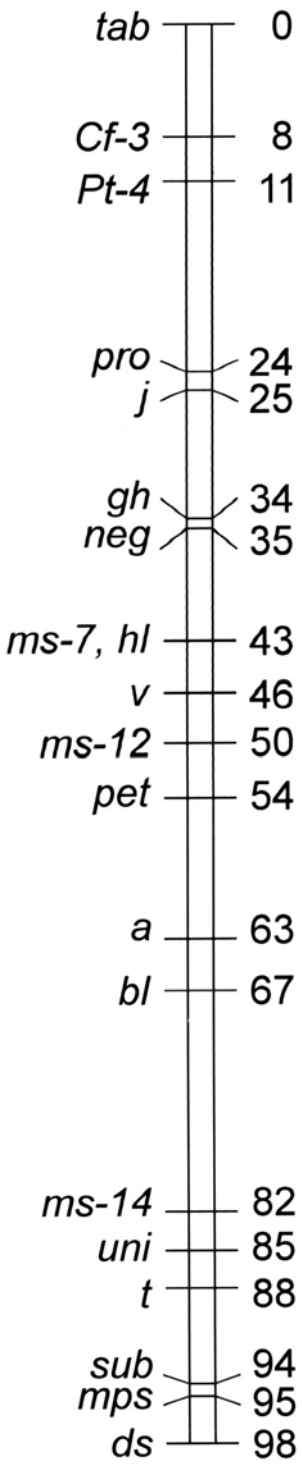
Markers	Phase[a]	Rec.%[b]	Literature reference
tab - hl	R	11.6 ± 4.0	Reeves et al., 1968
tab - hl	BC	39.1 ± 1.5	Lachman, 1972
tab - a	R	22.7 ± 3.5	Reeves et al., 1968
tab - a	BC	47.1 ± 1.5	Lachman, 1972
Cf-3 - j	C	17.4 ± 3.9	Langford, 1937
Cf-3 - a	C	44.2 ± 10.5	Langford, 1937
Cf-3 - f	C	42.1 ± 6.2	Langford, 1937
pt-4 - j	R	32.6 ± 3.5	pooled data: Rick et al., 1972
pt-4 - hl	R	27.5 ± 3.6	pooled data: Rick et al., 1972
pt-4 - a	R	35.6 ± 3.4	pooled data: Rick et al., 1972
pt-4 - f	R	35.2 ± 4.2	pooled data: Rick et al., 1972
pro - j	R	0 ± 4.1	Table 1
pro - hl	BC	16.7 ± 3.3	Rick, 1991

pro - hl	R	17.8 ± 5.0	Table 1
pro - a	BC	33.3 ± 4.2	Rick, 1991
pro - a	R	34.9 ± 2.4	pooled data: table 1; Rick and Martin, 1960
pro - a	C	31.6 ± 2.9	Table 1
gh - hl	R	8.8 ± 1.4	pooled data: Brauer and Rick, 1956; Burdick, 1961
gh - a	R	25.8 ± 1.2	pooled data: Brauer and Rick, 1956; Burdick, 1961
neg - j	R	10.3 ± 2.9	De la Roche and Lachman, 1967
neg - hl	R	11.5 ± 2.8	De la Roche and Lachman, 1967
neg - a	R	22.7 ± 2.7	pooled data: Kerr, 1967; Rick and Martin, 1960
hl - j	R	19.2 ± 3.4	Butler, 1959
hl - j	C	21.0 ± 1.0	pooled data: Butler, 1959; Clayberg, 1970
ms-7 - j	R	17.8 ± 4.7	Clayberg, 1970
ms-7 - hl	R	0 ± 4.9	Clayberg, 1970
ms-7 - a	R	20.2 ± 4.7	Clayberg, 1970
v - hl	R	6.1 ± 2.9	Whalen, 1972
v - a	R	17.3 ± 2.2	Whalen, 1972
ms-12 - j	R	29.4 ± 4.4	Clayberg, 1970
ms-12 - hl	R	0 ± 4.9	Clayberg, 1970
ms-12 - a	R	12.2 ± 4.8	Clayberg, 1970
pet - tab	R	23.5 ± 5.3	Quiros et al., 1974
pet - a	R	0 ± 6.0	Quiros et al., 1974
pet - bl	R	0 ± 6.0	Quiros et al., 1974
a - j	R	31.8 ± 3.4	pooled data: table 1; Butler, 1959
a - j	BC	31.7 ± 1.9	Butler, 1951
a - j	C	30.7 ± 1.0	pooled data: Butler, 1959; Clayberg, 1970
a - hl	R	25.0 ± 1.9	Butler, 1959
a - hl	BC	23.0 ± 0.8	pooled data: Lachman, 1972; Rick, 1991
a - hl	C	19.0 ± 0.4	pooled data: Table 1; Brauer and Rick, 1956; Burdick, 1961 Butler, 1959; Clayberg, 1970
a - f	R	30.9 ± 2.1	Butler, 1959
a - f	BC	22.1 ± 1.7	Butler, 1951
a - f	C	37.9 ± 2.3	Butler, 1959
bl - j	R	39.9 ± 5.1	Rick, 1963

bl - hl	R	25.1 ± 5.1	Rick, 1963
bl - a	R	11.3 ± 5.3	Rick, 1963
ms-14 - j	R	40.1 ± 4.0	Clayberg, 1970
ms-14 - hl	R	31.6 ± 4.3	Clayberg, 1970
ms-14 - a	R	19.5 ± 4.7	Clayberg, 1970
uni - hl	R	29.0 ± 6.2	Reeves et al., 1967
uni - a	R	26.3 ± 6.3	Reeves et al., 1967
f - j	R	47.8 ± 4.1	pooled data: Butler, 1959; Mertens and Burdick, 1954
f - j	C	37.5 ± 0.9	Butler, 1959
f - hl	R	45.2 ± 3.5	pooled data: Butler, 1959
sub - hl	R	34.2 ± 3.8	Rick and Boynton, 1963
sub - a	R	29.3 ± 2.6	pooled data: Rick and Boynton, 1963; Rick and Martin, 1960
mps - hl	R	40.7 ± 2.3	pooled data: Rick et al., 1970
mps - a	R	27.2 ± 2.6	pooled data: Rick et al., 1970
ds - hl	R	39.8 ± 4.1	Burdick, 1961
ds - a	R	30.4 ± 3.2	pooled data: Rick, 1958; Burdick, 1961

[a] C: coupling phase; R: repulsion phase; BC: backcross
[b] Rec %. is recombination percentage ± SE calculated with RECF2

Figure 1. Revised ‘classical’ morphological marker map of tomato chromosome 11.



TGRC Stock Lists

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1. MONOGENIC STOCKS (950 accessions listed): TGC 46 (1996)
2. MISCELLANEOUS STOCKS (1192 accessions listed): TGC 47 (1997)

3. WILD SPECIES STOCKS

The following list of 1,106 wild tomato species accessions is a revision of the previous list issued in TGC 45 (1995). Recent additions to this group include populations of several species, particularly *L. chmielewskii* and *L. parviflorum*, collected by Dr.s C.M. Rick, M. Holle, and C. Quiros during a 1995 trip to Depts. Apurimac and Cusco in Peru. Collections made the following year by Dr.s R. Chetelat, Holle, and Quiros in Depts. Arequipa (*L. pennellii*, *L. chilense* and *L. peruvianum*) and Ancash (*L. hirsutum*, *L. peruvianum*, and *L. pimpinellifolium*) are also included. Other noteworthy additions are collections of *L. pimpinellifolium*, *L. pennellii*, and *L. peruvianum* made by Holle in the Canete drainage of Peru, and populations of *S. juglandifolium* and *L. esculentum* var. *cerasiforme* collected by Dr. M. Hermann in Ecuador.

To facilitate choice of accessions, items which belong to our core subset for each species are highlighted by an asterisk after the accession number. Detailed passport information on each accession can be obtained from our website (http://tgrc.ucdavis.edu) or from the SolGenes database (http://probe.nalusda.gov). Additional information will be furnished on request.

As stated in previous lists, all accessions have been grown for seed increase at UC-Davis, and small seed samples are available for research purposes. Stocks of the outcrossing, polymorphic accessions are increased in a fashion to maintain diversity, and somewhat larger seed samples are generally distributed. Seed of certain recalcitrant populations, in particular the 4 *Solanum* spp., are provided in very small quantities as supplies permit.

L. cheesmanii (39 accessions; all locations in the Galapagos Islands, Ecuador)

LA0166*	Santa Cruz: Barranco, North of Punta	Galapagos Islands	Ecuador
LA0421*	Cristobal: cliff East of Wreck Bay	Galapagos Islands	Ecuador
LA0422	Cristobal: Wreck Bay	Galapagos Islands	Ecuador
LA0428	Santa Cruz: Trail Bellavista to Miconia Zone	Galapagos Islands	Ecuador
LA0429*	Santa Cruz: Crater in highlands	Galapagos Islands	Ecuador
LA0434	Santa Cruz: Rambech Trail	Galapagos Islands	Ecuador
LA0437	Isabela: Ponds North of Villamil	Galapagos Islands	Ecuador
LA0521	Fernandina: Inside Crater	Galapagos Islands	Ecuador
LA0522	Fernandina: Outer slopes	Galapagos Islands	Ecuador
LA0524	Isabela: Punta Essex	Galapagos Islands	Ecuador
LA0528	Santa Cruz: Academy Bay	Galapagos Islands	Ecuador
LA0529	Fernandina: Crater	Galapagos Islands	Ecuador
LA0531*	Baltra	Galapagos Islands	Ecuador
LA0746*	Isabela: Punta Essex	Galapagos Islands	Ecuador
LA0749*	Fernandina: North side	Galapagos Islands	Ecuador
LA0927	Santa Cruz: Academy Bay	Galapagos Islands	Ecuador
LA0932	Isabela: Tagus Cove	Galapagos Islands	Ecuador
LA1035	Fernandina: Low elevation	Galapagos Islands	Ecuador
LA1036*	Isabela: far north end	Galapagos Islands	Ecuador
LA1037	Isabela: Alcedo East slope	Galapagos Islands	Ecuador
LA1040	Cristobal: Caleta Toruga	Galapagos Islands	Ecuador

LA1041	Santa Cruz: El Cascajo	Galapagos Islands	Ecuador
LA1042	Isabela San Tomas, 6 km from Villamil	Galapagos Islands	Ecuador
LA1043	Isabela San Tomas, 10 km from Villamil	Galapagos Islands	Ecuador
LA1138	Isabela: East of Cerro Azul	Galapagos Islands	Ecuador
LA1139	Isabela: West of Cerro Azul	Galapagos Islands	Ecuador
LA1402	Fernandina: West of Punta Espinoza	Galapagos Islands	Ecuador
LA1404	Fernandina: West Flank Caldera	Galapagos Islands	Ecuador
LA1406*	Fernandina: Southwest rim Caldera	Galapagos Islands	Ecuador
LA1407	Fernandina: Northwest Bench Caldera	Galapagos Islands	Ecuador
LA1409	Isabela: Punta Albermarle	Galapagos Islands	Ecuador
LA1412*	Cristobal: opposite Isla Lobos	Galapagos Islands	Ecuador
LA1414	Isabela: Cerro Azul	Galapagos Islands	Ecuador
LA1427	Fernandina	Galapagos Islands	Ecuador
LA1447	Santa Cruz: Charles Darwin Station-Punta	Galapagos Islands	Ecuador
LA1448	Santa Cruz: Puerta Ayora, Pelican Bay	Galapagos Islands	Ecuador
LA1449	Santa Cruz: Charles Darwin Station,	Galapagos Islands	Ecuador
LA1450*	Isabela: Bahia San Pedro	Galapagos Islands	Ecuador
LA3124	Barrington Island: near East landing	Galapagos Islands	Ecuador

L. cheesmanii* f. *minor (29 accessions; all locations in the Galapagos Islands, Ecuador)

LA0317*	Bartolome	Galapagos Islands	Ecuador
LA0426	Bartolome	Galapagos Islands	Ecuador
LA0436*	Isabela: Villamil	Galapagos Islands	Ecuador
LA0438	Isabela: Coast at Villamil	Galapagos Islands	Ecuador
LA0480	Isabela: Cowley Bay Santiago	Galapagos Islands	Ecuador
LA0483*	Fernandina: Inside Crater	Galapagos Islands	Ecuador
LA0526*	Abingdon: West Side	Galapagos Islands	Ecuador
LA0527	Bartolome	Galapagos Islands	Ecuador
LA0528	Santa Cruz: Academy Bay		
LA0530	Fernandina: Crater	Galapagos Islands	Ecuador
LA0532	Duncan	Galapagos Islands	Ecuador
LA0747	Santiago: Cape Trenton	Galapagos Islands	Ecuador
LA0748	Santiago: East Trenton Island	Galapagos Islands	Ecuador
LA0929	Isabela: Punta Flores	Galapagos Islands	Ecuador
LA0930	Isabela: Cabo Tortuga	Galapagos Islands	Ecuador
LA1039	Isabela: Cape Berkeley	Galapagos Islands	Ecuador
LA1044	Bartolome	Galapagos Islands	Ecuador
LA1136	Gardner, (near Floreana)	Galapagos Islands	Ecuador
LA1137*	Jervis	Galapagos Islands	Ecuador
LA1141*	Santiago: North Crater	Galapagos Islands	Ecuador
LA1400	Isabela: North of Punta Tortuga	Galapagos Islands	Ecuador
LA1401*	Isabela: North of Punta Tortuga	Galapagos Islands	Ecuador
LA1403	Fernandina: West of Punta Espinoza	Galapagos Islands	Ecuador
LA1408*	Isabela: Southwest volcano, Cape Berkeley	Galapagos Islands	Ecuador
LA1410*	Isabela: Punta Ecuador	Galapagos Islands	Ecuador
LA1411	Santiago: North James Bay	Galapagos Islands	Ecuador
LA1452	Isabela: East Slope Alcedo	Galapagos Islands	Ecuador
LA1508	Corona del Diablo (near Floreana)	Galapagos Islands	Ecuador
LA1627	Isabela: Tagus Cove	Galapagos Islands	Ecuador

L. chilense (83 accessions)

LA0130	Moquegua	Moquegua	Peru
LA0294	Tacna	Tacna	Peru
LA0456	Clemesi	Moquegua	Peru

LA0458	Tacna	Tacna	Peru
LA0460	Palca	Tacna	Peru
LA0470	Taltal	Antofagasta	Chile
LA1029	7 km North of Moquegua	Moquegua	Peru
LA1030	Tarata Rd.	Tacna	Peru
LA1782	Quebrada de Acari	Arequipa	Peru
LA1917	Llauta, Rio Palpa	Ica	Peru
LA1930	Quebrada Calapampa, Rio Acari	Arequipa	Peru
LA1932*	Minas de Acari	Arequipa	Peru
LA1938*	Quebrada Salsipuedes, Rio Chaparra	Arequipa	Peru
LA1958*	Pampa de la Clemesi	Arequipa	Peru
LA1959	Huaico Moquegua	Moquegua	Peru
LA1960*	Rio Osmore	Moquegua	Peru
LA1961	Toquepala	Tacna	Peru
LA1963*	Rio Caplina	Tacna	Peru
LA1965*	Causiri	Tacna	Peru
LA1967*	Pachia	Tacna	Peru
LA1968	Cause seco, road from Tacna to Tarata	Tacna	Peru
LA1969*	Estique Pampa	Tacna	Peru
LA1970	Tarata	Tacna	Peru
LA1971*	Palquilla	Tacna	Peru
LA1972	Rio Sama	Tacna	Peru
LA2404	Arica-Ticnamar	Tarapaca	Chile
LA2405	Ticnamar	Tarapaca	Chile
LA2406	Arica-Putre	Tarapaca	Chile
LA2731	Moquella	Tarapaca	Chile
LA2737	Yala-yala	Tarapaca	Chile
LA2739	Cruce Nama/Camina	Tarapaca	Chile
LA2746	Arenamiento-18/Azapa	Tarapaca	Chile
LA2747	Alta Azapa	Tarapaca	Chile
LA2748*	Soledad	Antofagasta	Chile
LA2749	Mina La Buena Esperanza	Antofagasta	Chile
LA2750*	Mina La Despreciada	Antofagasta	Chile
LA2751	Pachica	Tarapaca	Chile
LA2753	Laonzana	Tarapaca	Chile
LA2754	63-65 km East of Huara	Tarapaca	Chile
LA2755*	Chusmiza	Tarapaca	Chile
LA2757	66 km East of Huara	Tarapaca	Chile
LA2759*	2 km North of Mamina	Tarapaca	Chile
LA2762	Quebrada Mamina-Parca	Tarapaca	Chile
LA2764	Codpa	Tarapaca	Chile
LA2765	Timar	Tarapaca	Chile
LA2767*	Chitita	Tarapaca	Chile
LA2768	Empalme Codpa	Tarapaca	Chile
LA2771	Above Lluta	Tarapaca	Chile
LA2773*	Zapahuiri	Tarapaca	Chile
LA2774	Socorama	Tarapaca	Chile
LA2778*	Chaquepina	Tarapaca	Chile
LA2779*	Cimentario Belen	Tarapaca	Chile
LA2780	Belen-Lupica	Tarapaca	Chile
LA2879	San Roque de Peine	Antofagasta	Chile
LA2880	Quebrada Tilopozo	Antofagasta	Chile
LA2882	Camar	Antofagasta	Chile
LA2884*	Ayaviri	Antofagasta	Chile
LA2887	Quebrada Bandurria	Antofagasta	Chile

LA2888	Loma Paposo	Antofagasta	Chile
LA2891	Taltal	Antofagasta	Chile
LA2930*	Quebrada Taltal	Antofagasta	Chile
LA2931*	Huatacondo	Antofagasta	Chile
LA2932	Quebrada Gatico	Antofagasta	Chile
LA2946	Above Huatacondo	Antofagasta	Chile
LA2949	Chusmiza	Tarapaca	Chile
LA2952	Camina	Tarapaca	Chile
LA2955	Quistagama-Cuisama	Tarapaca	Chile
LA2980	Yacango	Moquegua	Peru
LA2981	Torata-Quilligue	Moquegua	Peru
LA3111	Tarata outskirts	Tacna	Peru
LA3112	Estique Pampa	Tacna	Peru
LA3113	Apacheta	Tacna	Peru
LA3114	Quilla	Tacna	Peru
LA3115	28 km East of Tacna	Tacna	Peru
LA3153	Torata-Omate	Moquegua	Peru
LA3155	Otora-Puente Jahuay	Moquegua	Peru
LA3355	Cacique de Ara	Tacna	Peru
LA3356	Cacique de Ara	Tacna	Peru
LA3357	Irrigation Magollo	Tacna	Peru
LA3358	Rio Arunta-cono sur	Tacna	Peru
LA3784	Rio Chaparra	Arequipa	Peru
LA3785	Terras Blancas, Chaparra	Arequipa	Peru
LA3786	Alta Chaparra	Arequipa	Peru

L. chmielewskii (28 accessions)

LA1028*	Casinchihua	Apurimac	Peru
LA1306*	Tambo	Ayacucho	Peru
LA1316*	Ocros	Ayacucho	Peru
LA1317*	Hacienda Pajonal	Ayacucho	Peru
LA1318*	Auquibamba	Apurimac	Peru
LA1325*	Puente Cunyac	Apurimac	Peru
LA1327	Soracata	Apurimac	Peru
LA1330	Hacienda Francisco	Apurimac	Peru
LA2639	Cunyac-Curahuasi	Apurimac	Peru
LA2663*	Tujtohaiya (Upper Apurimac)	Cusco	Peru
LA2677*	Huayapacha-1 (Upper Apurimac)	Cusco	Peru
LA2678	Huayapacha-2 (Upper Apurimac)	Cusco	Peru
LA2679	Huayapacha-3 (Upper Apurimac)	Cusco	Peru
LA2680*	Puente Apurimac-1 (Upper Apurimac)	Cusco	Peru
LA2681	Puente Apurimac-2 (Upper Apurimac)	Cusco	Peru
LA2695*	Chihuanpampa (Upper Apurimac)	Cusco	Peru
LA3642	Ankukunka	Cusco	Peru
LA3643	Colcha	Cusco	Peru
LA3644	Pte. Tincoj	Cusco	Peru
LA3645	Boca del Rio Velille	Cusco	Peru
LA3648	Huallapachaca	Apurimac	Peru

LA3653	Matara	Apurimac	Peru
LA3654	Casinchigua-Chacoche	Apurimac	Peru
LA3656	Chalhuani	Apurimac	Peru
LA3658	Occobamba	Apurimac	Peru
LA3661	Pampotampa (Rio Tintay)	Apurimac	Peru
LA3662	Huancarpuquio	Apurimac	Peru
LA3663	Desvio Capaya	Apurimac	Peru

L. esculentum* var*. cerasiforme (273 accessions)

LA0168		New Caledonia	Fr. Oceania
LA0292*	Santa Cruz	Galapagos	Ecuador
LA0349	Unknown Origin		Unknown
LA0475	Sucua	Morona-Santiago	Ecuador
LA0476	Sucua	Morona-Santiago	Ecuador
LA1025*	Oahu: Wahiawa	Hawaii	USA
LA1203	Ciudad Vieja		Guatemala
LA1204*	Quetzaltenango		Guatemala
LA1205	Copan		Honduras
LA1206*	Copan Ruins		Honduras
LA1207			Mexico
LA1208	Sierra Nevada		Colombia
LA1209			Colombia
LA1226	Sucua	Morona-Santiago	Ecuador
LA1227	Sucua	Morona-Santiago	Ecuador
LA1228*	Macas, San Jacinto de los Monos	Morona-Santiago	Ecuador
LA1229	Macas Plaza	Morona-Santiago	Ecuador
LA1230	Macas	Morona-Santiago	Ecuador
LA1231*	Tena	Napo	Ecuador
LA1247	La Toma	Loja	Ecuador
LA1268*	Chaclacayo	Lima	Peru
LA1286*	San Martin de Pangoa	Junin	Peru
LA1287	San Martin de Pangoa	Junin	Peru
LA1289	San Martin de Pangoa	Junin	Peru
LA1290	Mazamari	Junin	Peru
LA1291	Satipo Granja	Junin	Peru
LA1307*	Hotel Oasis, San Francisco	Ayacucho	Peru
LA1308	San Francisco	Ayacucho	Peru
LA1310	Hacienda Santa Rosa	Ayacucho	Peru
LA1311	Santa Rosa Puebla (16 subunits)	Ayacucho	Peru
LA1312	Paisanato (2 subunits)	Cusco	Peru
LA1314	Granja Pichari	Cusco	Peru
LA1320*	Hacienda Carmen	Apurimac	Peru
LA1323*	Pfacchayoc	Cusco	Peru
LA1324	Hacienda Potrero, Quillabamba	Cusco	Peru
LA1328	Rio Pachachaca	Apurimac	Peru
LA1334	Pescaderos	Arequipa	Peru
LA1338	Puyo	Napo	Ecuador
LA1372	Santa Eulalia	Lima	Peru
LA1385*	Quincemil	Cusco	Peru
LA1386	Balsas, Rio Maranon	Amazonas	Peru
LA1387	Quincemil	Cusco	Peru
LA1388*	San Ramon	Junin	Peru
LA1420*	Lago Agrio	Napo	Ecuador
LA1421	Santa Cecilia	Napo	Ecuador

LA1423	Near Santo Domingo	Pichincha	Ecuador
LA1425*	Villa Hermosa	Cauca	Colombia
LA1426	Cali	Cauca	Colombia
LA1428	La Estancilla	Manabi	Ecuador
LA1429*	La Estancilla	Manabi	Ecuador
LA1453	Kauai-Poipu	Hawaii	USA
LA1454			Mexico
LA1455	Gral Teran	Nuevo Leon	Mexico
LA1456	Papantla	Vera Cruz	Mexico
LA1457	Tehuacan	Puebla	Mexico
LA1458	Huachinango	Puebla	Mexico
LA1461*	University Philippines, Los Banos		Philippines
LA1464	El Progreso-Yoro		Honduras
LA1465	Taladro, Comayagua		Honduras
LA1467	Cali	Cauca	Colombia
LA1468	Between Cali and Fte. Casa	Cauca	Colombia
LA1479	Sucua	Morona-Santiago	Ecuador
LA1480	Sucua	Morona-Santiago	Ecuador
LA1481	Sucua	Morona-Santiago	Ecuador
LA1482*	Segamat		Malaysia
LA1483*	Trujillo		Saipan
LA1509	Tawan		Borneo
LA1510			Mexico
LA1511*	Sete Lagoas	Minas Gerais	Brazil
LA1512	Lago Llopango		San
LA1540	Between Cali and Popayan	Cauca	Colombia
LA1542*	Turrialba		Costa Rica
LA1543*	Upper Parana		Brazil
LA1545	Becan Ruins	Campeche	Mexico
LA1546	Papantla	Vera Cruz	Mexico
LA1548	Fundo Iliana, San Martin de Pangoa	Junin	Peru
LA1549	Chontabamba, Oxapampa	Junin	Peru
LA1569	Jalapa	Vera Cruz	Mexico
LA1574	Nana	Lima	Peru
LA1619	Pichanaki	Junin	Peru
LA1620	Castro Alves	Bahia	Brazil
LA1621	Rio Venados	Hidalgo	Mexico
LA1622	Lusaka		Zambia
LA1623	Muna	Yucatan	Mexico
LA1632	Puerto Maldonado	Madre de Dios	Peru
LA1654	Tarapoto	San Martin	Peru
LA1655	Tarapoto	San Martin	Peru
LA1662	El Ejido	Merida	Venezuela
LA1667	Cali	Cauca	Colombia
LA1668	Acapulco	Guerrero	Mexico
LA1673	Nana	Lima	Peru
LA1701	Trujillo	La Libertad	Peru
LA1703	Rio Tamesi	Tamaulipas	Mexico
LA1704	Rio Tamesi	Tamaulipas	Mexico
LA1709	Desvio Yojoa		Honduras
LA1710	Cariare	Limon	Costa Rica
LA1711	Zamorano		Honduras
LA1712	Pejibaye		Costa Rica
LA1713	CATIE, Turrialba		Costa Rica
LA1909	Quillabamba	Cusco	Peru

LA1953	La Curva	Arequipa	Peru
LA2076	Naranjitos		Bolivia
LA2077	Paco\Coroica		Bolivia
LA2078	Mosardas	Rio Grande de Sol	Brazil
LA2079	Maui: Kihei	Hawaii	USA
LA2080	Maui: Kihei	Hawaii	USA
LA2081	Maui: Kihei	Hawaii	USA
LA2082	Arenal Valley		Honduras
LA2085	Kempton Park		S. Africa
LA2095*	La Cidra	Loja	Ecuador
LA2121	Between Yacuambi and Guadalupe	Zamora-Chinchipe	Ecuador
LA2122	Yacuambi junction (4 subunits)	Zamora-Chinchipe	Ecuador
LA2123	La Saquea (2 subunits)	Zamora-Chinchipe	Ecuador
LA2126	El Dorado (4 subunits)	Zamora-Chinchipe	Ecuador
LA2127	Zumbi	Zamora-Chinchipe	Ecuador
LA2129	San Roque	Zamora-Chinchipe	Ecuador
LA2130	Gualaquiza	Zamora-Chinchipe	Ecuador
LA2131*	Bomboiza	Zamora-Chinchipe	Ecuador
LA2132	Chuchumbetza	Zamora-Chinchipe	Ecuador
LA2135	Limon	Santiago-Morona	Ecuador
LA2136	Bella Union	Santiago-Morona	Ecuador
LA2137	Tayusa	Santiago-Morona	Ecuador
LA2138	Chinimpini	Santiago-Morona	Ecuador
LA2138	Chinimpini	Santiago-Morona	Ecuador
LA2139	Logrono	Santiago-Morona	Ecuador
LA2139	Logrono	Santiago-Morona	Ecuador
LA2140	Huambi	Santiago-Morona	Ecuador
LA2140	Huambi	Santiago-Morona	Ecuador
LA2140	Huambi	Santiago-Morona	Ecuador
LA2141	Rio Blanco	Santiago-Morona	Ecuador
LA2142	Cambanaca	Santiago-Morona	Ecuador
LA2143	Nuevo Rosario	Santiago-Morona	Ecuador
LA2177	San Ignacio (6 subunits)	Cajamarca	Peru
LA2205	Santa Rosa de Mirador	San Martin	Peru
LA2205	Santa Rosa de Mirador	San Martin	Peru
LA2308*	San Francisco	San Martin	Peru
LA2312	Jumbilla #1	Amazonas	Peru
LA2313	Jumbilla #2	Amazonas	Peru
LA2392	Jakarta		Indonesia
LA2393	Mercedes Canton Hoja Ancha	Guanacaste	Costa Rica
LA2394	San Rafael de Hoja Ancha	Guanacaste	Costa Rica
LA2402*	Florianopolis	Santa Catarina	Brazil
LA2411	Yanamayo	Puno	Peru
LA2587	(4x of unknown origin)		
LA2616	Naranjillo	Huanuco	Peru
LA2617	El Oropel	Huanuco	Peru
LA2618	Santa Lucia-Tulumayo	Huanuco	Peru
LA2619	Caseria-San Augustin	Ucayali	Peru
LA2620	La Divisoria	Ucayali	Peru
LA2621	3 de Octubre	Ucayali	Peru
LA2624	Umashbamba (Urubamba)	Cusco	Peru
LA2625	Chilcachaca (Urubamba)	Cusco	Peru
LA2626	Santa Ana (Urubamba)	Cusco	Peru
LA2627	Pacchac-chico (Urubamba)	Cusco	Peru
LA2628	Echarate (Urubamba)	Cusco	Peru

LA2629	Echarate (Urubamba)	Cusco	Peru
LA2630	Calzana (Urubamba)	Cusco	Peru
LA2631	Chontachayoc (Urubamba)	Cusco	Peru
LA2632	Maranura (Urubamba)	Cusco	Peru
LA2633	Huayopata (Urubamba)	Cusco	Peru
LA2635	Huayopata (Urubamba)	Cusco	Peru
LA2636	Sicre (Urubamba)	Cusco	Peru
LA2637	Sicre (Urubamba)	Cusco	Peru
LA2640	Molinopata-Abancay (Pachachaca)	Apurimac	Peru
LA2642	Molinopata-Abancay (Pachachaca)	Apurimac	Peru
LA2643	Bellavista-Abancay (Pachachaca)	Apurimac	Peru
LA2660	San Ignacio de Moxos	Beni	Bolivia
LA2664	Yanahuana	Puno	Peru
LA2665	San Juan del Oro (Condori) (Tambopata)	Puno	Peru
LA2666	San Juan del Oro (Tambopata)	Puno	Peru
LA2667	Pajchani (Tambopata)	Puno	Peru
LA2668	Cruz Playa (Tambopata)	Puno	Peru
LA2669	Huayvaruni-1 (Tambopata)	Puno	Peru
LA2670*	Huayvaruni-2 (Tambopata)	Puno	Peru
LA2671	San Juan del Oro (Escuela, Tambopata)	Puno	Peru
LA2673	Chuntopata (Sangaban)	Puno	Peru
LA2674	Huairurune (Sangaban)	Puno	Peru
LA2675*	Casahuiri (Sangaban)	Puno	Peru
LA2683	Consuelo (Pilcopata)	Cusco	Peru
LA2684	Patria (Pilcopata)	Cusco	Peru
LA2685	Gavitana (Pilcopata)	Madre de Dios	Peru
LA2686	Yunguyo (Pilcopata)	Madre de Dios	Peru
LA2687	Mansilla (Pilcopata)	Madre de Dios	Peru
LA2688*	Santa Cruz, near Shintuyo-1 (Pilcopata)	Madre de Dios	Peru
LA2689	Santa Cruz near Shintuyo-2 (Pilcopata)	Madre de Dios	Peru
LA2690	Atalaya (Pilcopata)	Cusco	Peru
LA2691	Rio Pilcopata (Pilcopata)	Cusco	Peru
LA2692	Pilcopata-1 (Pilcopata)	Cusco	Peru
LA2693	Pilcopata-2 (Pilcopata)	Cusco	Peru
LA2694	Aguasantas (Pilcopata)	Cusco	Peru
LA2696	El Paramillo, La Union	Valle	Colombia
LA2697	Mata de Cana, El Dovio	Valle	Colombia
LA2698	La Esperanza de Belgica	Valle	Colombia
LA2700	Aoti, Satipo	Junin	Peru
LA2702	Kandy-1		Sri Lanka
LA2703*	Kandy-2		Sri Lanka
LA2709*	Bidadi, Bangalore	Karnataka	India
LA2710	Porto Firme		Brazil
LA2782	El Volcan	Antioquia	Colombia
LA2783	Titiribi	Antioquia	Colombia
LA2784	La Queronte	Antioquia	Colombia
LA2785	El Bosque	Antioquia	Colombia
LA2786	Andes #1	Antioquia	Colombia
LA2787	Andes #2	Antioquia	Colombia
LA2789	Canaveral	Antioquia	Colombia
LA2790	Buenos Aires	Antioquia	Colombia
LA2791	Rio Frio	Antioquia	Colombia
LA2792	Tamesis	Antioquia	Colombia
LA2793	La Mesa	Antioquia	Colombia
LA2794	El Libano	Antioquia	Colombia

LA2795	Camilo	Antioquia	Colombia
LA2807	Taypiplaya	Yungas	Bolivia
LA2811	Cerro Huayrapampa	Apurimac	Peru
LA2814	Ccascani, Sandia	Puno	Peru
LA2841	Chinuna	Amazonas	Peru
LA2842	Santa Rita	San Martin	Peru
LA2843	Moyobamba	San Martin	Peru
LA2844	Shanhoa	San Martin	Peru
LA2845*	Mercado Moyobamba	San Martin	Peru
LA2871*	Chamaca	Sud Yungas	Bolivia
LA2873	Lote Pablo Luna #2	Sud Yungas	Bolivia
LA2874	Playa Ancha	Sud Yungas	Bolivia
LA2933	Jipijapa	Manabi	Ecuador
LA2977	Belen	Beni	Bolivia
LA2978	Belen	Beni	Bolivia
LA3123	Summit, Santa Cruz	Galapagos Islands	Ecuador
LA3135	Pinal Del Jigue	Holguin	Cuba
LA3136	Arroyo Rico	Holguin	Cuba
LA3137	Pinares de Mayari	Holguin	Cuba
LA3138	El Quemada	Holguin	Cuba
LA3139	San Pedro de Cananova	Holguin	Cuba
LA3140	Los Platanos	Holguin	Cuba
LA3141	Guira de Melena	La Habana	Cuba
LA3158-61	Vicinity of Los Mochis	Sinaloa	Mexico
LA3162	North of Copan		Honduras/
LA3452	CATIE, Turrialba	Turrialba	Costa Rica
LA3623	Tablones	Manabi	Ecuador
LA3633			Ghana
LA3652	Matara	Apurimac	Peru
LA3844	Algarrobito	Guarico	Venezuela

L. hirsutum (74 accessions)

LA0094	Canta-Yangas	Lima	Peru
LA0361*	Canta	Lima	Peru
LA0386	Cajamarca	Cajamarca	Peru
LA0387	Santa Apolonia	Cajamarca	Peru
LA1033	Hacienda Taulis	Lambayeque	Peru
LA1295	Surco	Lima	Peru
LA1298	Yaso	Lima	Peru
LA1347*	Empalme Otusco	La Libertad	Peru
LA1352	Rupe	Cajamarca	Peru
LA1353*	Contumaza	Cajamarca	Peru
LA1354	Between Contumaza and Cascas	Cajamarca	Peru
LA1361*	Pariacoto	Ancash	Peru
LA1362	Chacchan	Ancash	Peru
LA1363*	Alta Fortaleza	Ancash	Peru
LA1366	Cajacay	Ancash	Peru
LA1378	Navan	Lima	Peru
LA1391	Bagua-Olmos	Cajamarca	Peru
LA1392	Huaraz-Casma	Ancash	Peru
LA1393	Huaraz-Casma	Ancash	Peru
LA1557	84 km East of Huaral	Lima	Peru
LA1559	Desvio Huamantanga	Lima	Peru
LA1560*	Matucana	Lima	Peru
LA1648	5 km above Yaso	Lima	Peru

LA1681	Mushka, Rio Canete	Lima	Peru
LA1691	Yauyos, Rio Canete, Canete	Lima	Peru
LA1695	Cacachuhuasin, Canete	Lima	Peru
LA1696	Huanchuy-Cacra, Rio Canete	Lima	Peru
LA1717	Sopalache, Huancabamba	Piura	Peru
LA1718	Huancabamba	Piura	Peru
LA1721*	Ticrapo Viejo	Huancavelica	Peru
LA1731*	Rio San Juan	Huancavelica	Peru
LA1736	Pucutay	Piura	Peru
LA1737	Cashacoto	Piura	Peru
LA1738	Desfiladero	Piura	Peru
LA1739	West of Canchaque	Piura	Peru
LA1740*	West of Huancabamba	Piura	Peru
LA1741	Sondorillo	Piura	Peru
LA1753	Surco	Lima	Peru
LA1764	West of Canta	Lima	Peru
LA1772	West of Canta	Lima	Peru
LA1775	Rio Casma	Ancash	Peru
LA1777*	Rio Casma	Ancash	Peru
LA1778	Rio Casma	Ancash	Peru
LA1779	Rio Casma	Ancash	Peru
LA1918*	Llauta, Rio Palpa	Ica	Peru
LA1927	Ocobamba	Ica	Peru
LA1978*	Colca, Rio Fortaleza	Ancash	Peru
LA1980	Desv. Huambo	Ancash	Peru
LA2155*	Maydasbamba	Cajamarca	Peru
LA2156	Ingenio Montan	Cajamarca	Peru
LA2158*	Along Rio Chotano	Cajamarca	Peru
LA2159	Atumpampa	Cajamarca	Peru
LA2167*	Cementerio Cajamarca	Cajamarca	Peru
LA2171	El Molino	Piura	Peru
LA2196	Caclic	Amazonas	Peru
LA2204*	Balsapata	Amazonas	Peru
LA2314	San Francisco	Amazonas	Peru
LA2321	Chirico	Amazonas	Peru
LA2324	Leimebamba	Amazonas	Peru
LA2329*	Aricapampa	La Libertad	Peru
LA2409*	Miraflores (Yauyos)	Lima	Peru
LA2552	Las Flores	Cajamarca	Peru
LA2556	Puente Moche	La Libertad	Peru
LA2567	Quita (Valle de Casma)	Ancash	Peru
LA2574	Cullaspungro (Valle de Casma)	Ancash	Peru
LA2648	Santo Domingo-Morropon	Piura	Peru
LA2650*	Ayabaca	Piura	Peru
LA2651	Puente Tordopa-Ayabaca	Piura	Peru
LA2722	Puente Auca, Rio Canete	Lima	Peru
LA2812	Lambayeque	Lambayeque	Peru
LA2975	Coltao	Ancash	Peru
LA2976	Huangra	Ancash	Peru
LA3794	Alta Fortaleza	Ancash	Peru
LA3796	Anca, Marca	Ancash	Peru

L. hirsutum* f. *glabratum (38 accessions)

LA0407*	Mirador, Guayaquil	Guayas	Ecuador
LA1223*	Alausi	Chimborazo	Ecuador
LA1252	Loja	Loja	Ecuador
LA1253	Pueblo Nuevo-Loja	Loja	Ecuador
LA1255	Pedistal	Loja	Ecuador
LA1264	Bucay	Chimborazo	Ecuador
LA1265	Rio Chimbo	Chimborazo	Ecuador
LA1266*	Pallatanga	Chimborazo	Ecuador
LA1624*	Jipijapa	Manabi	Ecuador
LA1625	South of Jipijapa	Manabi	Ecuador
LA2092	Chinuko	Chimborazo	Ecuador
LA2098*	Sabianga	Loja	Ecuador
LA2099	Between Sabianga and Sozorango	Loja	Ecuador
LA2100	Sozorango	Loja	Ecuador
LA2101	Cariamanga	Loja	Ecuador
LA2103*	Lansaca	Loja	Ecuador
LA2104	Pena Negra	Loja	Ecuador
LA2105	Jardin Botanico, Loja	Loja	Ecuador
LA2106	Yambra	Loja	Ecuador
LA2107	Los Lirios	Loja	Ecuador
LA2108	Anganumo	Loja	Ecuador
LA2109*	Yangana #1	Loja	Ecuador
LA2110	Yangana #2	Loja	Ecuador
LA2114	San Juan	Loja	Ecuador
LA2115	Pucala	Loja	Ecuador
LA2116	Las Juntas	Loja	Ecuador
LA2119*	Saraguro	Loja	Ecuador
LA2124	Cumbaratza	Zamora-Chinchipe	Ecuador
LA2128*	Zumbi	Zamora-Chinchipe	Ecuador
LA2144	Chanchan	Chimborazo	Ecuador
LA2174*	Rio Chinchipe	Cajamarca	Peru
LA2175	Timbaruca	Cajamarca	Peru
LA2855	Mollinomuna	Loja	Ecuador
LA2860*	Cariamanga	Loja	Ecuador
LA2861	Las Juntas	Loja	Ecuador
LA2863	Macara	Loja	Ecuador
LA2864	Sozorango	Loja	Ecuador
LA2869	Matola-La Toma	Loja	Ecuador

L. parviflorum (49 accessions)

LA0247*	Chavinillo	Huanuco	Peru
LA0735	Huanuco-Cerro de Pasco	Huanuco	Peru
LA1319	Abancay	Apurimac	Peru
LA1321	Curahuasi	Apurimac	Peru
LA1322*	Limatambo	Cusco	Peru
LA1326	Rio Pachachaca	Apurimac	Peru
LA1329*	Yaca	Apurimac	Peru
LA1626*	Mouth of Rio Rupac	Ancash	Peru
LA1716*	Huancabamba	Piura	Peru
LA2072	Huanuco	Huanuco	Peru
LA2073	Huanuco North of San Rafael	Huanuco	Peru

LA2074	Huanuco	Huanuco	Peru
LA2075	Huanuco	Huanuco	Peru
LA2113*	7 km above La Toma	Loja	Ecuador
LA2133*	Ona	Azuay	Ecuador
LA2190*	Tialango	Amazonas	Peru
LA2191	Campamento Ingenio	Amazonas	Peru
LA2192	Pedro Ruiz	Amazonas	Peru
LA2193	Churuja	Amazonas	Peru
LA2194	Chachapoyas West	Amazonas	Peru
LA2195	Caclic	Amazonas	Peru
LA2197	Luya	Amazonas	Peru
LA2198	Chachapoyas East	Amazonas	Peru
LA2200*	Choipiaco	Amazonas	Peru
LA2201	Pipus	Amazonas	Peru
LA2202	Tingobamba	Amazonas	Peru
LA2315	Sargento	Amazonas	Peru
LA2317	Zuta	Amazonas	Peru
LA2318	Tambo (Lima Tambo)	Amazonas	Peru
LA2319*	Chirico	Amazonas	Peru
LA2325*	Above Balsas	Amazonas	Peru
LA2403	Wandobamba	Huanuco	Peru
LA2613	Matichico-San Rafael	Huanuco	Peru
LA2614	San Rafael	Huanuco	Peru
LA2615	Ayancocho	Huanuco	Peru
LA2639	Cunyac-Curahuasi	Apurimac	Peru
LA2641	Nacchera-Abancay	Apurimac	Peru
LA2727	Ona	Azuay	Ecuador
LA2847	Suyubamba	Amazonas	Peru
LA2848	West of Pedro Ruiz	Amazonas	Peru
LA2862	Saraguro-Cuenca	Azuay	Ecuador
LA2865	Rio Leon	Azuay	Ecuador
LA2913	Uchucyaco	Huanuco	Peru
LA2917*	Chullchaca	Ancash	Peru
LA3651	Matara	Apurimac	Peru
LA3655	Casinchigua-Chacache	Apurimac	Peru
LA3657	Casinchihua-Pichirhua	Apurimac	Peru
LA3660	Murashaya	Apurimac	Peru
LA3793	Huariaca to San Rafael	Huanuco	Peru

L. pennellii (40 accessions)

LA0716*	Atico	Arequipa	Peru
LA0751	Sisacaya	Lima	Peru
LA1272*	Pisaquera	Lima	Peru
LA1273	Cayan	Lima	Peru
LA1275	Quilca road junction	Lima	Peru
LA1277*	Trapiche	Lima	Peru
LA1282*	Sisacaya	Lima	Peru
LA1297	Pucara	Lima	Peru
LA1299	Santa Rosa de Quives	Lima	Peru
LA1303	Pampano	Huancavelica	Peru
LA1340*	Capillucas	Lima	Peru
LA1356	Moro	Ancash	Peru
LA1367*	Santa Eulalia	Lima	Peru
LA1376	Sayan	Lima	Peru

LA1515	Huaura-Churin	Lima	Peru
LA1522*	Huaura-Churin	Lima	Peru
LA1649	Molina (Ingenio)	Ica	Peru
LA1656*	Between Marca and Chincha	Ica	Peru
LA1657	Between Buena Vista and Yautan	Ancash	Peru
LA1674*	Toparilla Canyon	Lima	Peru
LA1693	Quebrada Machurango (Zuniga)	Lima	Peru
LA1724	La Quinga (Rio Pisco)	Ica	Peru
LA1732*	Rio San Juan	Huancavelica	Peru
LA1733	Rio Canete	Lima	Peru
LA1734	Rio Canete	Lima	Peru
LA1735	Rio Canete	Lima	Peru
LA1809	El Horador	Piura	Peru
LA1940	Rio Atico	Arequipa	Peru
LA1941	Rio Atico	Arequipa	Peru
LA1942	Rio Atico	Arequipa	Peru
LA1943	Rio Atico	Arequipa	Peru
LA1946*	Caraveli	Arequipa	Peru
LA2560*	Santa-Huaraz	Ancash	Peru
LA2580*	Valle de Casma	Ancash	Peru
LA2657	Bayovar	Piura	Peru
LA2963*	Acoy (Rio Majes)	Arequipa	Peru
LA3635	Omas, Canete	Lima	Peru
LA3788	Rio Atico	Arequipa	Peru
LA3789	Rio Atico	Arequipa	Peru
LA3791	Caraveli	Arequipa	Peru

L. pennellii* var*. puberulum (8 accessions)

LA0750	Ica-Nazca	Ica	Peru
LA1302*	Quita Sol	Ica	Peru
LA1911	Locari (Rio Santa Cruz)	Ica	Peru
LA1912	Cerro Locari (Rio Santa Cruz)	Ica	Peru
LA1920*	Cachiruma (Rio Grande)	Ayacucho	Peru
LA1926	Agua Perdida (Rio Ingenio)	Ica	Peru
LA3665	Rio Santa Cruz	Ica	Peru
LA3778	Palpa to Nazca	Ica	Peru

L. peruvianum (151 accessions)

LA0098	Chilca	Lima	Peru
LA0103*	Cajamarquilla	Lima	Peru
LA0107*	Hacienda San Isidro	Lima	Peru
LA0110	Cajacay	Ancash	Peru
LA0111	Supe	Lima	Peru
LA0153*	Culebras	Ancash	Peru
LA0370	Huampani quebrada	Lima	Peru
LA0371	Supe	Lima	Peru
LA0372	Culebras #1	Ancash	Peru
LA0374	Culebras #2	Ancash	Peru
LA0378	Cascas	Cajamarca	Peru
LA0392	Llallan	Cajamarca	Peru
LA0441*	Cerro Campana	La Libertad	Peru
LA0444*	Chincha #1	Ica	Peru
LA0445	Chincha #2	Ica	Peru

LA0446*	Atiquipa	Arequipa	Peru
LA0448	Chala	Arequipa	Peru
LA0451	Arequipa	Arequipa	Peru
LA0453	Yura	Arequipa	Peru
LA0454	Tambo	Arequipa	Peru
LA0455	Tambo	Arequipa	Peru
LA0462	Azapa	Tarapaca	Chile
LA0464	Hacienda Rosario	Tarapaca	Chile
LA0752*	Sisacaya	Lima	Peru
LA1027		Cajamarca	Peru
LA1031	Balsas	Amazonas	Peru
LA1032	Aricapampa	La Libertad	Peru
LA1133	Huachipa	Lima	Peru
LA1161	Huachipa	Lima	Peru
LA1270	Pisiquillo	Lima	Peru
LA1271	Horcon	Lima	Peru
LA1274*	Pacaibamba	Lima	Peru
LA1278	Trapiche	Lima	Peru
LA1281	Sisacaya	Lima	Peru
LA1300	Santa Rosa de Quives	Lima	Peru
LA1304	Pampano	Huancavelica	Peru
LA1305*	Ticrapo	Huancavelica	Peru
LA1331*	Nazca	Ica	Peru
LA1333	Loma Camana	Arequipa	Peru
LA1336*	Atico	Arequipa	Peru
LA1337	Atiquipa	Arequipa	Peru
LA1339*	Capillucas	Lima	Peru
LA1346*	Casmiche	La Libertad	Peru
LA1350	Chauna	Cajamarca	Peru
LA1351	Rupe	Cajamarca	Peru
LA1358	Yautan	Ancash	Peru
LA1360*	Pariacoto	Ancash	Peru
LA1364*	Alta Fortaleza	Ancash	Peru
LA1365*	Caranquilloc	Ancash	Peru
LA1368	San Jose de Palla	Lima	Peru
LA1369	San Geronimo	Lima	Peru
LA1373	Asia	Lima	Peru
LA1377	Navan	Lima	Peru
LA1379	Caujul	Lima	Peru
LA1394	Balsas, Rio Utcabamba	Amazonas	Peru
LA1395	Chachapoyas	Amazonas	Peru
LA1396	Balsas	Amazonas	Peru
LA1473	Callahuanca, Santa Eulalia	Lima	Peru
LA1474*	Lomas de Camana	Arequipa	Peru
LA1475	Los Anitos, Barranca	Lima	Peru
LA1513	Atiquipa	Arequipa	Peru
LA1517	Santa Rosa	Lima	Peru
LA1537	(Self-fertile, from Hogenboom)		
LA1554	Rio Huaura, 85 km from Huaral	Lima	Peru
LA1556	Rimac Valley, Hacienda Higuereto, Rio	Lima	Peru
LA1609	Between Asia and El Pinon	Lima	Peru
LA1616	La Molina, La Rinconada	Lima	Peru
LA1626*	Mouth of Rio Rupac	Ancash	Peru
LA1647*	Huadquina, Topara	Ica	Peru
LA1653	Uchumayo-Arequipa	Arequipa	Peru

LA1675	Toparilla Canyon	Lima	Peru
LA1677*	Between Fundo Huadquina and Topara	Lima	Peru
LA1692	Putinza, (Rio Canete)	Lima	Peru
LA1694	Cacachuhuasin Canete	Lima	Peru
LA1708*	Between Chamaya and Jaen	Cajamarca	Peru
LA1910*	Tambillo, (Rio Ica)	Huancavelica	Peru
LA1913	Tinguiayog, (Rio Santa Cruz)	Ica	Peru
LA1929	La Yapana, Rio Ingenio	Ica	Peru
LA1935	Lomas de Atiquipa, Rio Yauca	Arequipa	Peru
LA1937*	Quebrada Torrecillas, (Rio Chaparra)	Arequipa	Peru
LA1944	Rio Atico	Arequipa	Peru
LA1945*	Caraveli	Arequipa	Peru
LA1947	Puerto Atico, (Rio Atico)	Arequipa	Peru
LA1949	Las Calaveritas	Arequipa	Peru
LA1951	Ocona, (Rio Ocona)	Arequipa	Peru
LA1954*	Mollendo	Arequipa	Peru
LA1955	Matarani	Arequipa	Peru
LA1973*	Yura	Arequipa	Peru
LA1975	Desvio Santo Domingo, (Rio Rimac)	Lima	Peru
LA1977	Orcococto	Lima	Peru
LA1981	Vocatoma, (Rio Santa)	Ancash	Peru
LA1982*	Huallanca, Rio Santa	Ancash	Peru
LA1983	Rio Manta, (Rio Santa)	Ancash	Peru
LA1984*	Otuzco (Rio Moche)	La Libertad	Peru
LA1985	Casmiche, (Rio Moche)	La Libertad	Peru
LA1989	(Self-fertile, bilaterally compatible with *L. esculentum*, from Hogenboom)		
LA2068	Chasquitambo	Ancash	Peru
LA2157	Tunel Chotano	Cajamarca	Peru
LA2163*	Between Cochabamba and Yamaluc	Cajamarca	Peru
LA2164	Yamaluc	Cajamarca	Peru
LA2172*	Cuyca	Cajamarca	Peru
LA2185*	Pongo de Rentema	Amazonas	Peru
LA2326*	10-17 km above Balsas	Amazonas	Peru
LA2327	Aguas Calientes	Cajamarca	Peru
LA2328*	Aricapampa	La Libertad	Peru
LA2330	Chagual	La Libertad	Peru
LA2331	Agallapampa	La Libertad	Peru
LA2333	Casmiche	La Libertad	Peru
LA2388	Cochabamba-Huambos	Cajamarca	Peru
LA2553*	Balconcillo-San Marcos	Cajamarca	Peru
LA2555	Mariscal Castilla (Moche)	La Libertad	Peru
LA2561	Huallanca (Santa)	Ancash	Peru
LA2562	Huallanca (Santa)	Ancash	Peru
LA2563	Canon del Pato (Santa)	Ancash	Peru
LA2565	Potrero de Pomacocha (Poscha)	Ancash	Peru
LA2566	Cullachaca Pomacocha-Llamellin (Poscha)	Ancash	Peru
LA2573	Valle de Casma	Ancash	Peru
LA2575	Valle de Casma	Ancash	Peru
LA2581	Chacasilla	Tarapaca	Chile
LA2717	5 km East of Chilca	Lima	Peru
LA2721	Putinza (Rio Canete)	Lima	Peru
LA2724	Huaynilla (Rio Canete)	Lima	Peru
LA2732*	Moquella	Tarapaca	Chile
LA2742	Camerones-Guancarana	Tarapaca	Chile
LA2744*	Sobraya, (Azapa)	Tarapaca	Chile

LA2745	Pan de Azucar, (Azapa)	Tarapaca	Chile
LA2770	Lluta	Tarapaca	Chile
LA2808*	Huaylas	Ancash	Peru
LA2809	Huaylas	Ancash	Peru
LA2834	Rio Aja, Hacienda Asiento	Ica	Peru
LA2900	Aphid resistant (from Walgenbach)		
LA2959	Chaca, (Vitor)	Tarapaca	Chile
LA2962	Echancay	Arequipa	Peru
LA2964	Quebrada de Burros	Tacna	Peru
LA2981	Torata-Chilligue	Moquegua	Peru
LA3154	Quinistaquillas	Moquegua	Peru
LA3156	Omate Agricultural Valley	Moquegua	Peru
LA3218	Quebrada Guerrero	Arequipa	Peru
LA3219	Catarindo	Arequipa	Peru
LA3220	Cocachacra -Quebrada Cachendo	Arequipa	Peru
LA3636	Coayllo, Canete	Lima	Peru
LA3637	Coayllo, Canete	Lima	Peru
LA3639	Ccatac, Canete	Lima	Peru
LA3640	Mexico City		Mexico
LA3664	Nazca grade	Ica	Peru
LA3666	Frontier Ica/Nazca	Ica	Peru
LA3790	Caraveli	Arequipa	Peru
LA3792	Quebrada Infirenillo	Arequipa	Peru
LA3795	Alta Fortaleza	Ancash	Peru
LA3797	Anca, Marca	Ancash	Peru
LA3799	Rio Pativilca	Ancash	Peru

L. peruvianum* f. *glandulosum (13 accessions)

LA0364	9 km West of Canta	Lima	Peru
LA0366	22 km West of Canta	Lima	Peru
LA1283	Santa Cruz de Laya	Lima	Peru
LA1284	Espiritu Santo	Lima	Peru
LA1292*	San Mateo	Lima	Peru
LA1293	Matucana	Lima	Peru
LA1294	Surco	Lima	Peru
LA1296	Tornamesa	Lima	Peru
LA1551	Rimac Valley, 71 km from Lima	Lima	Peru
LA1552	Rimac Valley, 93 km from Lima	Lima	Peru
LA1646	3 km above Yaso, Rio Chillon	Lima	Peru
LA1722	Ticrapo Viejo	Huancavelica	Peru
LA1723	La Quinga	Ica	Peru

L. peruvianum* var. *humifusum (11 accessions; all from drainage of Rio Jequetepeque)

LA0385	San Juan	Cajamarca	Peru
LA0389	Abra Gavilan	Cajamarca	Peru
LA2150	Puente Muyuno	Cajamarca	Peru
LA2151	Morochupa	Cajamarca	Peru
LA2152*	San Juan #1	Cajamarca	Peru
LA2153	San Juan #2	Cajamarca	Peru
LA2334	San Juan	Cajamarca	Peru
LA2548	La Muyuna	Cajamarca	Peru
LA2550	El Tingo, Chorpampa	Cajamarca	Peru

LA2582	(4x) San Juan	Cajamarca	Peru
LA2583	(4x)		

L. pimpinellifolium (245 accessions)

LA0100	La Cantuta	Lima	Peru
LA0114*	Pacasmayo	La Libertad	Peru
LA0121	Trujillo	La Libertad	Peru
LA0122	Poroto	La Libertad	Peru
LA0369	La Cantuta	Lima	Peru
LA0373*	Culebras	Ancash	Peru
LA0375	Culebras #2	Ancash	Peru
LA0376	Chiclin	La Libertad	Peru
LA0381	Pongo	La Libertad	Peru
LA0384	Chilete	Cajamarca	Peru
LA0391	Magdalena	Cajamarca	Peru
LA0397	Hacienda Tuman	Lambayeque	Peru
LA0398	Hacienda Carrizal	Piura	Peru
LA0400*	Hacienda Buenos Aires	Piura	Peru
LA0411*	Pichilingue	Los Rios	Ecuador
LA0412	Pichilingue	Los Rios	Ecuador
LA0413	Cerecita	Guayas	Ecuador
LA0417*	Puna	Guayas	Ecuador
LA0418	Daule	Guayas	Ecuador
LA0420	El Empalme	Guayas	Ecuador
LA0442*	Sechin	Ancash	Peru
LA0443	Pichilingue	Los Rios	Ecuador
LA0480	Hacienda Santa Inez	Ica	Peru
LA0722	Trujillo	La Libertad	Peru
LA0753	Lurin	Lima	Peru
LA1236	Tinelandia, Santo Domingo	Pichincha	Ecuador
LA1237*	Atacames	Esmeraldas	Ecuador
LA1242	Los Sapos	Guayas	Ecuador
LA1243	Co-op Carmela	Guayas	Ecuador
LA1245*	Santa Rosa	El Oro	Ecuador
LA1246*	La Toma	Loja	Ecuador
LA1248	Hacienda Monterrey	Loja	Ecuador
LA1256	Naranjal	Guayas	Ecuador
LA1257	Las Mercedes	Guayas	Ecuador
LA1258	Voluntario de Dios	Guayas	Ecuador
LA1259	Catarama	Los Rios	Ecuador
LA1260	Pueblo Viejo	Los Rios	Ecuador
LA1261*	Babahoyo	Los Rios	Ecuador
LA1262	Milagro junction on route 33	Los Rios	Ecuador
LA1263	Barranco Chico	Guayas	Ecuador
LA1269	Pisiquillo	Lima	Peru
LA1279*	Cieneguilla	Lima	Peru
LA1280	Chontay	Lima	Peru
LA1301*	Hacienda San Ignacio	Ica	Peru
LA1332	Nazca	Ica	Peru
LA1335*	Pescaderos	Arequipa	Peru
LA1341	Huampani	Lima	Peru
LA1342	Casma	Ancash	Peru
LA1343	Puente Chao	La Libertad	Peru

LA1344	Laredo	La Libertad	Peru
LA1345	Samne	La Libertad	Peru
LA1348	7 miles East of Pacasmayo	La Libertad	Peru
LA1349	Cuculi	Lambayeque	Peru
LA1355	Nepena	Ancash	Peru
LA1357	Jimbe	Ancash	Peru
LA1359	La Crau	Ancash	Peru
LA1370	San Jose de Palla	Lima	Peru
LA1371*	Santa Eulalia	Lima	Peru
LA1374	Ingenio	Ica	Peru
LA1375*	San Vicente de Canete	Lima	Peru
LA1380	Chanchape	Piura	Peru
LA1381	Naupe	Piura	Peru
LA1382	Chachapoyas-Balsas	Amazonas	Peru
LA1383	Chachapoyas-Bagua	Amazonas	Peru
LA1384	Quebrada Parca (Chilca)	Lima	Peru
LA1416	Las Delicias	Pichincha	Ecuador
LA1466	Chongoyape	Lambayeque	Peru
LA1469	El Pilar, Olmos	Lambayeque	Peru
LA1470	Between Motupe and Olmos	Lambayeque	Peru
LA1471	Between Motupe and Jayanca	Lambayeque	Peru
LA1472	Quebrada Topara	Lima	Peru
LA1478*	Santo Tome	Piura	Peru
LA1514	60 km Huaura-Churin	Lima	Peru
LA1519	Vitarte	Lima	Peru
LA1520	Between Huaura and Sayan	Lima	Peru
LA1521*	El Pinon, Asia	Lima	Peru
LA1547*	Chota-El Angel	Carchi	Ecuador
LA1561	Huaura, San Eusebio	Lima	Peru
LA1562	Cieneguilla	Lima	Peru
LA1571	San Jose de Palle	Lima	Peru
LA1572	Huampani	Lima	Peru
LA1573	Nana	Lima	Peru
LA1575	Huaycan	Lima	Peru
LA1576*	Manchay Alta	Lima	Peru
LA1577	Cartavio	La Libertad	Peru
LA1578*	Jequetepeque	La Libertad	Peru
LA1579	Colegio Punto Cuatro #1	Lambayeque	Peru
LA1580	Colegio Punto Cuatro #2	Lambayeque	Peru
LA1581	Punto Cuatro	Lambayeque	Peru
LA1582*	Motupe	Lambayeque	Peru
LA1583	Tierra de la Vieja	Lambayeque	Peru
LA1584*	Between Jayanca and La Vina	Lambayeque	Peru
LA1585	Cuculi	Lambayeque	Peru
LA1586*	Between Zana and San Nicolas	La Libertad	Peru
LA1587	San Pedro de Lloc	La Libertad	Peru
LA1588	Between Laredo and Barraza	La Libertad	Peru
LA1589	Between Viru and Calunga	La Libertad	Peru
LA1590*	Between Viru and Tomaval	La Libertad	Peru
LA1591	Ascope	La Libertad	Peru
LA1592	Moche	La Libertad	Peru
LA1593*	Puente Chao	La Libertad	Peru
LA1594	Cerro Sechin	Ancash	Peru
LA1595	Nepena-Samanco	Ancash	Peru
LA1596	Santa-La Rinconada	Ancash	Peru

LA1597	Rio Casma	Ancash	Peru
LA1598	Between Culebras and La Victoria	Ancash	Peru
LA1599*	Huarmey	Ancash	Peru
LA1600	Las Zorras	Ancash	Peru
LA1601	Between Rio Pativilca and La Providencia	Ancash	Peru
LA1602*	Between Rio Chillon and Punchauca	Lima	Peru
LA1603	Quilca	Lima	Peru
LA1604	Horcon	Lima	Peru
LA1605	Canete-San Antonio	Lima	Peru
LA1606*	Tambo de Mora	Ica	Peru
LA1607	Between Canete and La Victoria	Lima	Peru
LA1608	Canete-San Luis	Lima	Peru
LA1610	Between Asia and El Pinon	Lima	Peru
LA1611	Rio Mala	Lima	Peru
LA1612	Rio Chilca	Lima	Peru
LA1613	Rio Huaura at Santa Eusebia	Lima	Peru
LA1614	Rio Huaura at Pampa Chumbes	Lima	Peru
LA1615	Between Piura and Simbala	Piura	Peru
LA1617*	Tumbes South	Tumbes	Peru
LA1618	Tumbes North	Tumbes	Peru
LA1628	Between Huanchaco and Libertad	La Libertad	Peru
LA1629	Between Miraflores and Costa Verde	Lima	Peru
LA1630	Between Chincha and Fundo La Palma	Ica	Peru
LA1631	Between Moche and San Fernando	La Libertad	Peru
LA1633	Chincha, co-op Huayna Capac	Ica	Peru
LA1634	El Ingenio, Fundo Bogotalla #1	Ica	Peru
LA1635	El Ingenio, Fundo Bogotalla #2	Ica	Peru
LA1636	Chincha, Laran	Ica	Peru
LA1637	Chincha, La Calera	Ica	Peru
LA1638	Huachipa, Fundo El Portillo	Lima	Peru
LA1645	Between Miraflores and Armendariz	Lima	Peru
LA1651	La Molina	Lima	Peru
LA1652	Cienguilla	Lima	Peru
LA1659*	4 km East of Pariacoto	Ancash	Peru
LA1660	Between Yautan and Pariacoto	Ancash	Peru
LA1661	Esquina de Asia	Lima	Peru
LA1670	Rio Sama	Tacna	Peru
LA1676	Between Fundo Huadquina and Topara	Ica	Peru
LA1678	San Juan Lucumo de Topara	Ica	Peru
LA1679	Tambo de Mora	Ica	Peru
LA1680	Between Canete and La Encanada	Lima	Peru
LA1682	Canete-Montalban	Lima	Peru
LA1683*	Rio Chira-Miramar	Piura	Peru
LA1684	Chulucanas	Piura	Peru
LA1685	Marcavelica	Piura	Peru
LA1686	Valle Hermosa #1	Piura	Peru
LA1687	Valle Hermoso #2	Piura	Peru
LA1688	Pedregal	Piura	Peru
LA1689*	Piura at Castilla #1	Piura	Peru
LA1690	Piura at Castilla #2	Piura	Peru
LA1697	Hacienda Santa Anita, (Rio Huaura)	Lima	Peru
LA1719	East of Arenillas	El Oro	Ecuador
LA1720	Yautan	Ancash	Peru
LA1728	Rio San Juan	Ica	Peru
LA1729	Rio San Juan	Ica	Peru

LA1742	Between Olmos and Marquina	Lambayeque	Peru
LA1781	Bahia de Caraquez	Manabi	Ecuador
LA1921	Majarena	Ica	Peru
LA1923	Cabildo	Ica	Peru
LA1924*	Piedras Gordas	Ica	Peru
LA1925	Pangaravi	Ica	Peru
LA1933	Jaqui	Arequipa	Peru
LA1936	Huancalpa, (Rio Chaparra)	Arequipa	Peru
LA1950	Pescadores, (Rio Caraveli)	Arequipa	Peru
LA1987	Viru-Fundo Luis Enrique	La Libertad	Peru
LA1992	Pishicato, (Rio Lurin)	Lima	Peru
LA1993	Chicama Valley (?)	Lima	Peru
LA2093	La Union	El Oro	Ecuador
LA2096	Playa	Loja	Ecuador
LA2097	Macara	Loja	Ecuador
LA2102*	El Lucero	Loja	Ecuador
LA2112	Above Hacienda Monterrey	Loja	Ecuador
LA2145	Juan Montalvo	Los Rios	Ecuador
LA2146	Limoncarro	Lambayeque	Peru
LA2147	Yube	Lambayeque	Peru
LA2149	Puente Muyuno	Cajamarca	Peru
LA2170	Pai Pai	Lambayeque	Peru
LA2173*	Cruz de Huaiquillo	Cajamarca	Peru
LA2176	Timbaruca	Cajamarca	Peru
LA2178	Tororume	Cajamarca	Peru
LA2179	Tamboripa-La Manga	Cajamarca	Peru
LA2180	La Coipa	Cajamarca	Peru
LA2181*	Balsahuaico	Cajamarca	Peru
LA2182	Cumba	Amazonas	Peru
LA2183*	Corral Quemado	Amazonas	Peru
LA2184	Bagua (Casual)	Amazonas	Peru
LA2186	El Salao	Amazonas	Peru
LA2187	La Caldera	Amazonas	Peru
LA2188	Machugal #1	Amazonas	Peru
LA2189	Machugal #2	Amazonas	Peru
LA2335	(4x)		
LA2336	(*r*, 4x)		
LA2340	(4x)		
LA2341	(4x)		
LA2345	(autodiploid from 13 chrom. haploid)		
LA2346	(autodiploid)		
LA2347	(autodiploid)		
LA2348	(*l, x*)		
LA2389	Tembladera	Cajamarca	Peru
LA2390	Chungal	Cajamarca	Peru
LA2391	Between Chungal & Monte Grande	Cajamarca	Peru
LA2401*	Moxeque, Casma	Ancash	Peru
LA2412	Fundo Don Javier, Chilca	Lima	Peru
LA2533*	Lomas de Latillo	Lima	Peru
LA2576	Valle de Casma	Ancash	Peru
LA2578	Tuturo (Valle de Casma)	Ancash	Peru
LA2585	(4x, aka. Reg971)		
LA2645	Desvio Chulucanas-Morropon	Piura	Peru
LA2646	1 km to Chalaco, Morropon	Piura	Peru
LA2647	Morropon-Chalaco	Piura	Peru

LA2652	Sullana	Piura	Peru
LA2653	San Francisco de Chocan	Piura	Peru
LA2655	La Huaca-Sullana	Piura	Peru
LA2656	Suarez	Tumbes	Peru
LA2659	Campus of U.N. de Piura	Piura	Peru
LA2718	5 km East of Chilca	Lima	Peru
LA2725	Tambo Colorado	Ica	Peru
LA2831	5 km South of Nazca	Ica	Peru
LA2832	Chichictara, (Rio Palpa)	Ica	Peru
LA2833	Hacienda Asiento, Rio Aja	Ica	Peru
LA2836	Fundo Pongo, (Rio Aja)	Ica	Peru
LA2839	Tialango	Amazonas	Peru
LA2840	Santa Hilarion de Tomaque	Amazonas	Peru
LA2850	Santa Rosa	Manabi	Ecuador
LA2851	Carcel Montecristi	Manabi	Ecuador
LA2852*	Cristo Rey de Charapoto	Manabi	Ecuador
LA2853	Experiment Station, Portoviejo	Manabi	Ecuador
LA2854	Jipijapa	Manabi	Ecuador
LA2857	Villamil	Isabela,	Ecuador
LA2866	Via a Amaluza	Loja	Ecuador
LA2914	La Castellana	Lima	Peru
LA2914	La Castellana	Lima	Peru
LA2915	Remanso de Olmos	Lambayeque	Peru
LA2966	La Molina	Lima	Peru
LA2974	Huaca del Sol	La Libertad	Peru
LA2982	Chilca #1	Lima	Peru
LA2983	Chilca #2	Lima	Peru
LA3468	La Molina Vieja	Lima	Peru
LA3634	Santa Rosa de Asia	Lima	Peru
LA3638	Ccatac, Canete	Lima	Peru
LA3798	Rio Pativilca	Ancash	Peru

S. juglandifolium (4 accessions)

LA2120	Sabanilla	Zamora-Chinchipe	Ecuador
LA2134	Tinajillas	Zamora-Chinchipe	Ecuador
LA2788	Quebrada la Buena	Antioquia	Colombia
LA3622	Cosanga	Napo	Ecuador

S. lycopersicoides (13 accessions)

LA1964	Chupapalca	Tacna	Peru
LA1990	Palca	Tacna	Peru
LA2385	Chupapalca Ingenio	Tacna	Peru
LA2386	Chupapalca	Tacna	Peru
LA2387	Lago Aricota	Tacna	Peru
LA2407	Arica-Putre	Tarapaca	Chile
LA2408	Above Putre	Tarapaca	Chile
LA2730	Moquella	Tarapaca	Chile
LA2772	Zapahuiri	Tarapaca	Chile
LA2776	Catarata Perquejeque	Tarapaca	Chile
LA2777	Putre	Tarapaca	Chile
LA2781	Desvio Putre	Tarapaca	Chile
LA2951	Quistagama, Camina	Tarapaca	Chile

S. ochranthum (3 accessions)

LA2118	San Lucas		Ecuador
LA2166	Rocoto-Pacopampa	Cajamarca	Peru
LA2682	Chinchaypuyo	Cusco	Peru

S. sitiens (5 accessions)

LA1974	Chuquicamata	Antofagasta	Chile
LA2876	Chuquicamata	Antofagasta	Chile
LA2877	El Crucero	Antofagasta	Chile
LA2878	Mina La Exotica	Antofagasta	Chile
LA2885	Caracoles	Antofagasta	Chile

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