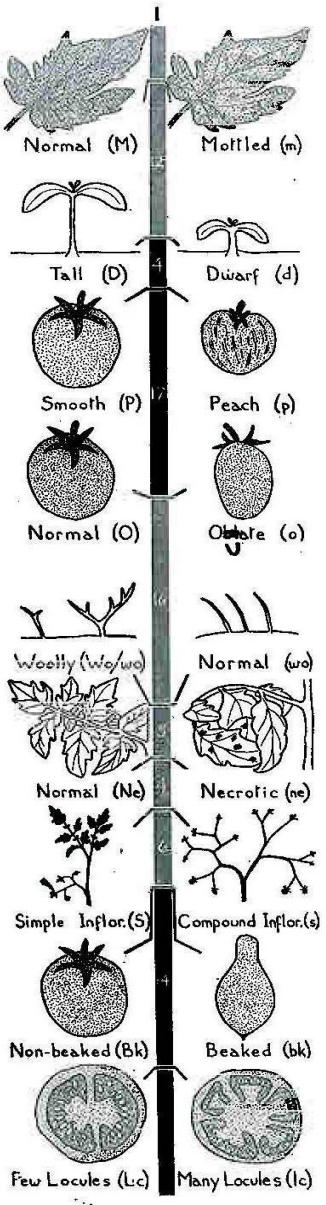
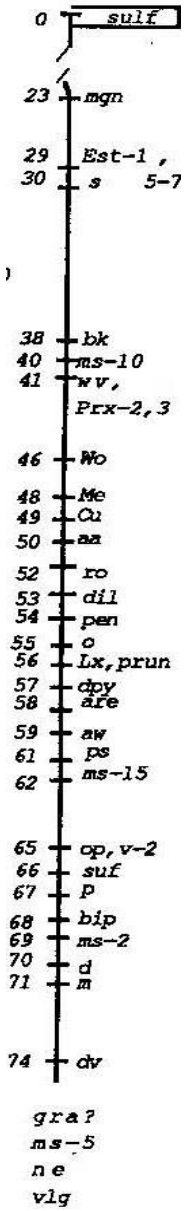
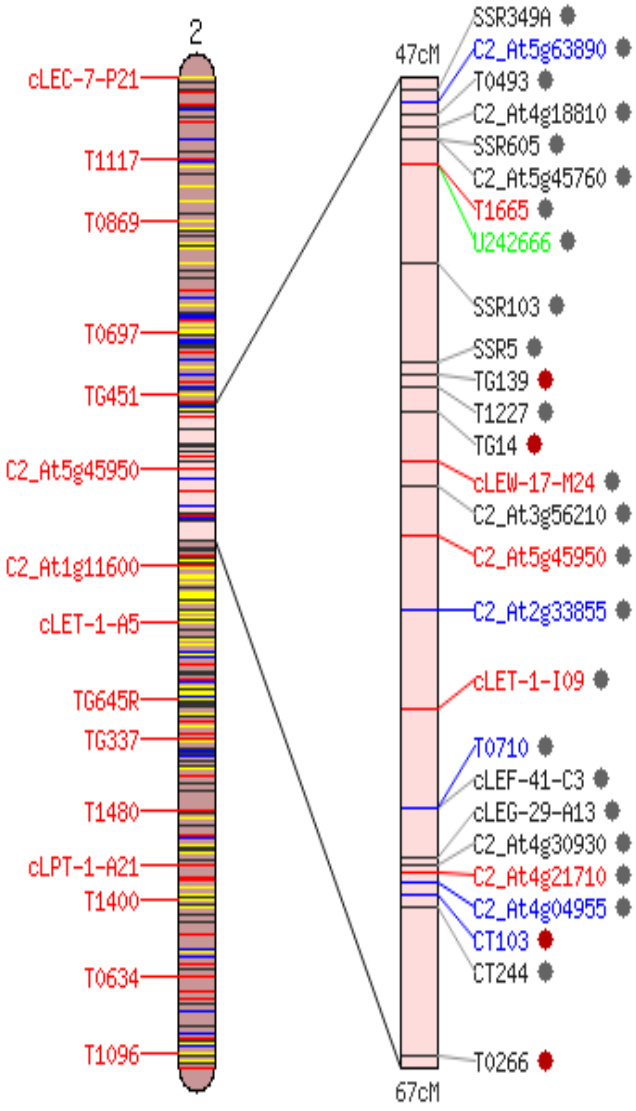
Report of the Tomato Genetics Cooperative







Volume 59 September 2009

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Report

of the

Tomato Genetics Cooperative

Number 59- September 2009

University of Florida

Gulf Coast Research and Education Center

Wimauma, FL 33598 USA

Foreword

The Tomato Genetics Cooperative, initiated in 1951, is a group of researchers who share and interest in tomato genetics, and who have organized informally for the purpose of exchanging information, germplasm, and genetic stocks. The Report of the Tomato Genetics Cooperative has been 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 or tissue culture. Relevant work on the Solanaceous species is encouraged as well. This will be the last Report published on hard copy and future reports will be only in electronic format on the TGC website: http://tgc.ifas.ufl.edu . See the website to request membership.

Paid memberships currently stand at approximately 83 from 16 countries.

Cover: This may well be the last of the TGC covers as we move to electronic only publishing. Little was known about linkage in tomato when the TGC began in 1951. On the left is the linkage group with the most linked genes that was published by Leonard Butler in the 1952 Journal of Heredity 43(1):25-35. This turned out to be chromosome 2. In the middle is the last morphological marker linkage map of chromosome 2 that was published in the l987 TGC (Vol. 37). On the right is a present day depiction of chromosome 2 with a 20cM interval highlighted showing numerous molecular markers mapped in that region. Most of the morphological markers on this and other chromosomes have not been precisely mapped because of other research priorities and a lack of funding for such work. Be sure to read the Feature Article about the formation of Tomato Genetics Cooperative by one of the two founders, Allan Burdick.

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From the editor:

Welcome to the last printing of the Tomato Genetics Cooperative. In case you haven't heard, the TGC will be available only in electronic format after 2009 due to changes in the academic world that have resulted in the submission of too few research reports. The legacy of the TGC has been impressive; in large part this journal has laid the foundation for tomato becoming a model crop for genetic studies. However, in today's world there are many other outlets for the publishing of tomato research and the storage of genetic information. In my as usual outspoken opinion, there is too much administrative pressure for public scientists to publish in "high impact" [sic] journals which in reality is mostly smoke and mirrors for a bean counting process. For whatever reason, time is limited for tomato researchers and it seems the value of publishing here is not what it used to be. But all is not lost, it is just time to adapt and move ahead. I have enjoyed being editor and look forward to keeping the electronic version of TGC alive on the TGC website http://tgc.ifas.ufl.edu/. I encourage all to submit reports and varietal pedigrees in future years! Thanks to those who submitted reports for this volume.

In commemoration of this final printing, it seemed appropriate that we look back to the beginning of the Tomato Genetics Cooperative. You can do this by reading the entertaining Feature Article that was written by one of the founders and the longest standing member of the TGC, Dr. Allan Burdick. It was he and fellow graduate student Don Barton who went to Charley Rick with the concept of forming a Tomato Genetics Cooperative….and the rest is history!

Finally, I want to express my appreciation to Dolly Cummings who skillfully keeps track of all our records and who puts the TGC Volume together. Also thanks go to Christine Cooley who, along with Dolly, works on the website and who helped with the cover of this issue. Your input on any TGC matters is welcome.

Jay W. Scott
Managing Editor

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Upcoming Tomato Related Meetings

24th Annual Tomato Disease Workshop, Nov. 3-5, 2009 State College, Pennsylvania,USA

http://guest.cvent.com/EVENTS/Info/Summary.aspx?e=dcf7cea9-4cc3-47da-ba05-2dec9005bc44

Sixth Solanaceae Genome Workshop, Nov. 8-13, 2009 New Delhi, India
http://www.sol2009.org/

3rd International Symposium on Tomato Diseases, July 25-30, 2010 Ischia, Naples Italy http://www.3istd.com

Grant Opportunity:

Request for Proposals for Tomato Germplasm Evaluation

Funding is expected to be available again in fiscal year 2010 for evaluation of tomato germplasm. Proposals must be submitted through the Tomato Crop Germplasm Committee (CGC). All proposals will be evaluated according to the national need for evaluation data, the likelihood of success, and the likelihood that the data will be entered into GRIN and shared with the user community. When all other factors are equal, preference for funding will be given to supporting those proposals forwarded by CGCs that have not received prior funding. Proposals will be reviewed by the CGC and forwarded to the USDA for consideration. Proposals must be returned to the CGC Chair (Majid Foolad) by October 30, 2009 so that reviews and rankings can be forwarded to the USDA in Beltsville on time. Evaluation priorities established by the CGC will provide review criteria.
These criteria were revised in 2006, and applicants are encouraged to review (http://www.ars-grin.gov/npgs/cgc_reports/tomatocgc2006evalpriorities.html). Because of limited funds, the USDA cannot support all proposals submitted. Consequently, please be very frugal in your request for funds. In recent years, the USDA has limited budget allocations to $15,000-$18,000 per project annually.
The proposal format is outlined below. Please submit proposals **electronically as a PDF file** to Majid Foolad, CGC Chair, mrf5@psu.edu by October 30, 2009.

I. Project title and name, title of evaluators.

II. Significance of the proposal to U.S. agriculture.

III. Outline of specific research to be conducted including the time frame involved – include the number of accessions to be evaluated.

IV. Funding requested, broken down item by item. Budgets should follow USDA form ARS454 as funding will be in the form of a specific cooperative agreement. No overhead charges are permitted.

V. Personnel:

   A. What type of personnel will perform the research (e.g. ARS, State, or industry scientist; postdoc; grad student, or other temporary help).

   B. Where will personnel work and under whose supervision.

VI. Approximate resources contributed to the project by the cooperating institution (e.g. facilities, equipment, and funds for salaries).

Roots of the Tomato Genetics Cooperative from the Graduate Student Underground*

Allan Burdick, Professor Emeritus, 3000 Woodkirk Drive, Columbia, MO 65203, USA

Tomato lovers, let us call them Lycopersiphiles. They have affection for this once thought to be poisonous fruit. They are scientists, and in a sense artists, as they correspond with one another in the annual Report of the Tomato Genetics Cooperative, now in its 59th edition.

The Editor and CEO of the TGC, Professor Jay W. Scott, of Florida has called on me, Allan Burdick - probably the oldest member - to serve as ad hoc Historian for the 59th edition.

Early History

Don Barton, now deceased, and I figure in this as graduate students working with tomatoes in the Genetics Department of the University of California, located in Berkeley, CA (at that time there was no need to add Berkeley, but if you did be sure to use three e's). The time was 1947-49.

In 1949, Barton was finishing his PhD. He had discovered and mapped the structure of the twelve pairs of pachytene chromosomes of the tomato - the first to do that.

I had been, at the same time and place, looking for hybrid vigor in the tomato. Hybrid vigor had long been known in Maize, a cross fertilizing species. The question was "Does hybrid vigor exist in the tomato, a self fertilizing species?"

Hybrid vigor did show up in the Berkeley fields used by the Genetics faculty along Shattuck Avenue next to the campus. We had planted 864 plants from crosses made in the greenhouse the season before. These plants were examined in unimaginable detail.

To reduce the results to understandable data we used a Constant Parent Regression Analysis devised by Fred Hull of the University of Florida.

All this appeared in publications, along with Barton's karyotypes.

A seed company in Arizona, as I remember it was the Lagomarsino Brothers, took note of the thesis and asked for seeds of the cherry types. I think this went on to be developed as varieties like Sweet 100.

_____

*Title by J.W. Scott, Dr. Burdick should not be blamed

Don Barton and I received our PhDs at the same ceremony in 1949. General Marshall spoke; we did not attend. Instead we went to Davis to see Charlie Rick and discuss starting a TGC. He agreed reluctantly to think about it. Destiny lay in the hands of Charlie.

We went off to faculty positions, at Cornell for Don who took Bill Meshanic with him. I went to the University of Arkansas in the Agronomy Department to work on grain sorghum and corn. I played with tomatoes in the greenhouse on the side.

While at Berkeley Don had a close relationship with Erney Jund, the cytology technician for Ernest Babcock, the Chairman and originator of the Genetics Department. (He and Clausen of Stanford wrote a genetics textbook.)

The department was made up of seven or eight faculty members, all but one of whom were Babcock's students. The one who was not his student was Ledyard Stebbins.

At that time Davis was not yet a campus of the University System. Charles Rick was there in what seemed like an experiment station, breeding tomatoes and other horticultural plants.

The "Departmental Plan" for PhD students was intended to give them really broad experience outside their thesis topic. We, as graduate students were not enthusiastic about it - sort of, but we went along.

It included taking classes in the graduate department of zoology with Curt Stern (Drosophila -ugh!) and Goldstein, the philosopher of the Natural Sciences.

The plan included field trips with Stebbins in the high Sierras - like Mt. Whitney at 14,494 feet high - and in the Central Valley. Ledyard, we called him, was tall, skinny and energetic. He climbed the mountains with three foot strides; hard for us to keep up.

Suddenly he would stop to point about 50 feet away and shout "There's an Elymus, get it!" I recall going what seemed like half way up Mt. Whitney.

Also among our field trips were two to Davis to talk to Charles Rick. He breathed and respired enthusiasm and knowledge of genetics and breeding, especially tomatoes. He was nothing like our professors at Berkeley - sedate and proper. Charlie was easy to talk to; he listened. We felt like he was a friend. Actually, he sat on my final PhD committee.

Jay, you posed a question in your letter: "What have your graduate students done for you lately?" President John F. Kennedy: "Ask not what your country can do for you; ask what you can do for your country." I said we did not think much of the "Plan" at the time. Now it comes back to me over and over. It gave us a real education!

About the Author-Curriculum vita-Allan Burdick

Born:	Cincinnati, OH, 16 August 1920
Brought up:	Ft. Worth, TX (drug up, as they said)
High School:	last two years, Flushing, NY
New Business Department, Wall Street Bank: 9 months	
University:	Iowa State College 1938, class of '42
WWII:	Enlisted 12 February 1942, Army Air Corp sent to RAF for flight training; various commands, then 9th Air Force, fighter pilot in England and France, then North Africa, finally home March 1946, rank major. Iowa State awarded 12 credits for Air Force service, enough to graduate me, BS in Animal Husbandry, but my interest was in Genetics the last two years before service.
Graduate work:	Iowa State with Gene Lindstrom on the inheritance of kernel row number in Maize. Three publications. MS 1947. (Lindstrom had an interest in tomatoes also.)
Graduate work:	Berkeley, Genetics Department, PhD 1949.

Overview of Career

University of Arkansas, 3 years to 1952
Purdue University, 1952 - 1960, Drosophila, gene structure, tomato mutations by thermal neutrons, Brookhaven National Laboratory, Upton, NY
1959-60 Guggenheim Foundation, Kyoto University, Japan
1960-63 Dean of Science, American University of Beirut
1963-66 Chairman, Biology Department, Adelphi University, Garden City, NY
1966-69 Chairman, Genetics Department, University of Missouri, Columbia, MO
1969-73 Professor of Genetics, University of Missouri
1976 Professor of Medical Genetics, clinical studies, University of Missouri
1986 Professor Emeritus, about 119 publications

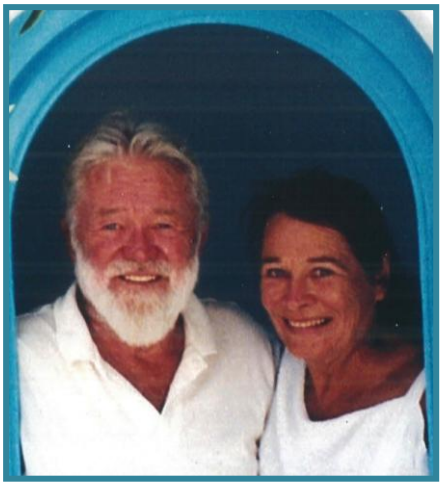
Research

- Mammalian, including human, genetics and cytogenetics
- A possible relationship between X-linked dominant orofacialdigital-I syndrome and anhidrotic ectodermal dysplasia (30510)
- Certain cryptic human bilateral asymmetries as potential polymorphic genetic markers
- Genetics of flaccid periodic paralysis
- Familial Spastic Paraplegia
- van der Woude Syndrome
- Carrier status risk determinations in certain late-onset human genetic disease

Family

Married Sally Cummins, 1943,
Four children: Mike, Nancy, Stephen and Lindy
Sally died April 1983
Married Elizabeth (Betty) W. Revington, November 1983
Enjoying a 25-year marriage.

Picture titled “Halcyon Days”



Allan and Betty

Addendum

The following information was sent via email from Roger Chetelat who has a large file of Charley Rick's correspondence at the time of the TGC's formation:

In Charley's correspondence with Burdick, I found this in a letter recommending him for a position at Purdue:

"...he [Burdick] and Dr. D. W. Barton, a fellow graduate student of his period, conceived the idea of a Tomato Genetics Cooperative and successfully organized the group. Although they preferred to have me coordinate the cooperative, they both maintain a very active interest in it."

I have a thick file of the letters from interested researchers indicating their support for the formation of the TGC and listing their research areas. I don't have a copy of the original form letter Charley distributed to the tomato community soliciting this feedback, unfortunately.

The Burdick correspondence file is interesting. He was at Arkansas, then Purdue, and starting in 1963 he was Dean at the American Univ. of Beirut (that was apparently the end of his tomato research).

I also found a file with the records of who had paid their dues in each year. In those days the charge was $1.00, which some of the foreign members paid via UNESCO dollars (which I'd never heard of). Many others sent Charley postal stamps (he accumulated a fairly massive collection of foreign stamps this way).

The story of the Stubbe mutants is also interesting, and might be worth mentioning. Much of this work was conducted by Hans Stubbe in East Germany during the Lysenko period, but he managed to pursue Mendelian genetics nonetheless. We owe a very large share of our mutant stocks to Stubbe.

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Nuclear DNA content in *Solanum* sect. *Juglandifolia* and *Solanum* sect. *Lycopersicoides*

Roger T. Chetelat

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Comparisons of the nuclear DNA content by flow cytometry have revealed interesting differences in the genome sizes of the tomato species. Estimates for cultivated tomato range from 1.88-2.07 pg/2C, or approximately 907-1000 Mbp/C (Arumuganathan and Earle, 1991; Bennett and Smith 1976; Michaelson et al. 1991). Similar values were obtained for *S. habrochaites* and *S. cheesmanii*. Somewhat larger genomes were detected in *S. pennellii* (2.47-2.77 pg/2C) and *S. peruvianum* (2.27 pg/2C) (Arumuganathan and Earle, 1991). The larger genome size of *S. pennellii* is consistent with cytological observations of meiosis in the F_1 *S. lycopersicum* × *S. pennellii* hybrid which indicated that several *pennellii* chromosomes have longer heterochromatic regions (Khush and Rick, 1963). This could indicate that the difference in genome size is due mostly to variation in the repetitive (i.e. non-coding) DNA fraction.

Until now, there was no data available on the genome sizes of the four tomato-allied species in *Solanum* section *Juglandifolia* and *Solanum* sect. *Lycopersicoides*. Previous studies of meiosis in *S. lycopersicum* × *S. lycopersicoides* hybrids had shown that chromosomes of the wild parent were substantially larger at diakinesis and metaphase than corresponding chromosomes of cultivated tomato (Ji et al. 2004). At pachytene, the total length of *S. lycopersicoides* chromosomes was 1.5 fold higher than in *S. lycopersicum* (Menzel 1962). Furthermore, pollen grain size – sometimes an indirect indicator of relative genome size – is higher in *S. lycopersicoides* and *S. sitiens* than in members of the tomato clade (Carrizo Garcia 2007; Chetelat et al. 2009). All these observations pointed to a significantly larger genome in *S. lycopersicoides* and *S. sitiens*.

To obtain direct measurements, we sent leaf samples of the four tomato-like nightshade species to Dr. K. Arumuganathan at the Virginia Mason Research Center in Seattle, Washington. DNA content was determined by flow cytometry. The results confirmed our expectation that *S. lycopersicoides* and *S. sitiens* have larger genomes than cultivated tomato. Nuclei of *S. ochranthum* contain approximately the same amount of DNA as cultivated tomato, while the *S. juglandifolium* genome is a bit smaller.

The significance of these results is unclear. There seems to be little correlation with phylogenetic distance, since *S. pennellii*, a member of the tomato clade, has a

greater difference in genome size relative to cultivated tomato than the members of the more distantly related section *Juglandifolia*. The difference in genome size between *S. juglandifolium* and *S. ochranthum* is consistent with recent findings that their genomes differ by a large chromosomal rearrangement (Albrecht and Chetelat 2009). Yet there was a similar difference in DNA content between *S. lycopersicoides* and *S. sitiens*, and their genomes appear to be colinear (Pertuze et al. 2002). There is no correlation with ploidy level, since all species are diploids with the same number of chromosomes (2n=24). In other plants, genome size variation has been attributed to differences in the rates of amplification and removal of LTR-retrotransposons (Bennetzen et al. 2005).

Table 1. Nuclear DNA content of some tomato-like nightshade species. Values are from flow cytometry of isolated nuclei. Published results for three tomato species are shown for comparison purposes.

Species	Accession	Sample	DNA Content (pg/2C +/- S.D.)
S. lycopersicoides	LA2951	08L9904	2.43 +/- 0.028
S. sitiens	LA4331	05L5033	2.69 +/- 0.013
S. juglandifolium	LA3322	07L7984	1.75 +/- 0.006
S. ochranthum	LA3649	07L7977	1.96 +/- 0.013
S. pennellii	--	--	2.47-2.77[a]
S. peruvianum	--	--	2.27[a]
S. lycopersicum	--	--	1.88-2.07[a], 1.9[b]

[a]from Arumuganathan and Earle, 1991; [b]from Michaelson et al, 1991.

Reference

Albrecht E, Chetelat RT (2009) Comparative genetic linkage map of *Solanum* sect. *Juglandifolia*: evidence of chromosomal rearrangements and overall synteny with the tomatoes and related nightshades. Theor. Appl. Genet. 118:831-847

Arumuganathan K, Earle ED (1991) Nuclear DNA content of some important plant species. Plant Molec. Biol. Rep. 9:208-218

Bennett MD, Smith JD (1976) Nuclear DNA amounts in angiosperms. Phil. Trans. Royal Soc. London, Series B Biol. Sci. 181:109-135

Bennetzen JL, Ma J, Devos KM (2005) Mechanisms of recent genome size variation in flowering plants. Ann. Bot. 95:127-132

Carrizo Garcia C (2007) Pollen Starch Reserves in Tomato Relatives: Ecophysiological Implications. Grana 46:13-19

Chetelat RT, Pertuze RA, Faundez L, Graham EB, Jones CM (2009) Distribution, ecology and reproductive biology of wild tomatoes and related nightshades from the Atacama Desert region of northern Chile. Euphytica 167:77-93

Ji Y, Pertuze RA, Chetelat RT (2004) Genome differentiation by GISH in interspecific and intergeneric hybrids of tomato and related nightshades. Chrom. Res. 12:107-116

Menzel MY (1962) Pachytene chromosomes of the intergeneric hybrid *Lycopersicon esculentum* x *Solanum lycopersicoides*. Amer. J. Bot. 49:605-615

Michaelson MJ, Price HJ, Ellison JR, Johnston JS (1991) Coomparison of plant DNA contents determined by Fuelgen microspectrophotometry and laser flow cytometry. Amer. J. Bot. 78:183-188

Pertuze RA, Ji Y, Chetelat RT (2002) Comparative linkage map of the *Solanum lycopersicoides* and *S. sitiens* genomes and their differentiation from tomato. Genome 45:1003-1012

***entire-2*, a mutation on chromosome 10S conferring reduced leaf serration and subdivision**

Roger T. Chetelat and Charles M. Rick

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In 1983, Campbell's R & D in Davis grew an EMS-treated M2 mutagenesis population in the variety CX8012 (equivalent to UC204C). One of us (CMR) surveyed the M2 families and discovered a mutant whose phenotype was similar to *entire* (*e*): broad leaves with reduced subdivision. Our studies of this mutant, conducted many years ago but never published, demonstrated the gene is not an allele of *e,* but represent a new locus, denoted *entire-2* (*e-2*). Recent interest in this mutant for studies of compound leaf development highlighted the need to describe its phenotype, transmission, and map location, hence the present research note.

The phenotype of *e-2* is superficially similar to the original *e*, but with some unique features. Cotyledons are often subnormal or fused. The first true leaves have fewer lateral segments than wild type and incompletely separated lobes. The leaf margins are undulate and irregularly lobed. On older leaves the leaf rachis is elongated and angled towards the tip of the leaf. Interstitial leaflets are elongated and enlarged. Mature plants produce only a few scattered flowers, with slender parts and other abnormalities. The calyx is enlarged, and anthers are often deformed and sometimes adnate to the pistil. Fertility is low, but sufficient for propagation via homozygotes.

Allele tests with *e* indicated that the new mutation represents a different gene. From the cross of *e-2* × *e* (in a stock of *ful, e, a, hl*), all eight F_1 plants had the wild type phenotype, with leaf segments well separated, but slightly broader than normal. In the F_2 progeny of this allele test, segregation was consistent with the expected 9:3:3:1 ratio (χ^2 = 2.23, Table 1). Classification of some of the *e/e-2* combinations was arbitrary, but there was no question that the majority of progeny were wild type for both genes. Allele tests with other leaf shape mutants, such as *c,* and *sf,* were not conducted.

Transmission of *e-2* is relatively normal, with a slight deficiency of mutant phenotypes. Our most reliable segregation data came from a BC of the hybrid with *S. pimpinellifolium* (Table 2). Out of 115 total progeny, only 47 were *e-2*, less than the expected 50%, but just shy significance at the P<0.05 level (χ^2 = 3.83, df = 1). In the BC of a similar cross to *S. pennellii*, segregation fit Mendelian expectations (Table 3).

Our tests for linkage of *e-2* with known markers indicated a map location on the short arm of chromosome 10. The search for linkage was first conducted by crossing *e-*

2 to *S. pennellii* LA0716, the latter providing abundant marker polymorphisms. The results indicated independence vis-à-vis markers on chromosomes 1 (*Prx-1, Dia-2*), 2 (*Est-1, Prx-2, Fdh-1*), 3 (*Prx*-7), 4 (*Pgm-2*), 6 (*Aps-1*), 7 (*Got-2*), and 8 (*Aps-2*). The chromosome 10S marker, *Prx-4* (Rick and Fobes 1977), on the other hand, showed a clear association with *e-2*, despite the small population size (χ^2 = 12.1, P<0.001, Table 3). From the co-segregation data, the map distance between *e-2* and *Prx-4* was estimated at 27.5 cM (r=0.25, Figure 1). Fortuitously, the cultivar in which the mutation appeared also carried the chromosome 10S marker gene *u,* for uniform ripening of the fruit (i.e. absence of a green shoulder). The *u – Prx-4* distance was approx. 19.7cM (r=0.19), and the *u – e-2* distance was 24.9 cM (r=0.23), suggesting *e-2* could be located distal to *u* and *Prx-4*. However, the map distances were not additive in this population, possibly because some *e-2* genotypes were scored incorrectly (e.g. the *S. pennellii* parent also contains genes for broad, undivided leaves).

A more robust test of gene order was obtained by the test cross of *e-2, u* × *S. pimpinellifolium* LA1575. This wild species not only provided the dominant alleles for *e-2* and *u*, but also carries a third chromosome 10 marker, gene *h* for hairless stems. Luckily, *h* is incompletely dominant over wild type, and can be scored in the heterozygous state, as necessitated by this backcross to *e-2, u*. The results (Table 2) of this three point cross indicated *e-2* is located between *u* and *h*, at a distance of 14.3 cM (r=0.139) from the former and 34.0 cM (r=0.3) from the latter (Figure 1). The distance between the outer markers, *u* and *h*, was 46.5 cM (r=0.365), close to the sum of the internal distances. Thus, the second linkage test is probably more accurate regarding the relative positions of *e-2* and *u.* The two tests do agree in placing *e-2* on the short arm of chromosome 10. Seeds of *e-2* (accession 3-705) and its non-mutant control (LA3130) are available through the TGRC.

Reference

Rick, C.M., and J. Fobes (1977) Linkage relations of some isozymic loci. TGC 27: 22-24.

Figure 1. Genetic map of the short arm and centromeric region of chromosome 10, showing the position of *e-2* relative to *u* (*uniform ripening*), *Prx-4* (*Peroxidase-4*), and *h* (*hairs absent*). The black area indicates the approximate location of the centromere, with the short arm above, and long arm (only partially shown) below. Distances are in centiMorgans.

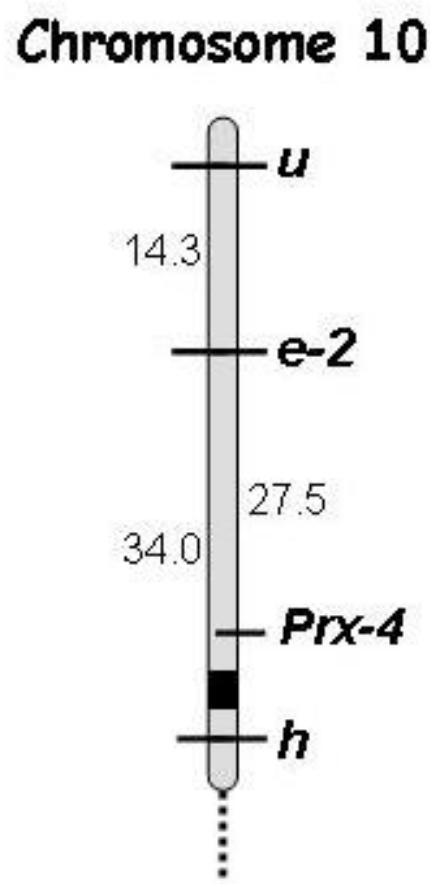

Table 1. Segregation in F_2 *e-2* × *a-hl, clau-e*. Data are from family 92L6207.

Phenotype	Observed	Expected
+/+	48	48.9
+/e	18	16.3
e-2/+	19	16.3
e-2/e	2	5.5

Table 2. Segregation in BC (*u, e-2*, + × +, +, *h* in *S. pimpinellifolium*) ×*u, e-2*, +. The mutants *u* (*uniform ripening*) and *e-2* are linked in coupling phase, with *h* (*hairs absent*) in repulsion. SCO = single crossover genotype, DCO = double crossover genotype. Data are from family 93L9449.

Type	***u***	***e-2***	***h***	**# Plants**
Parental	+	+	*+/h*	38
Parental	*u*	*e-2*	+	31
SCO	+	+	+	18
SCO	*u*	*e-2*	*+/h*	12
SCO	*u*	+	*+/h*	9
DCO	+	*e-2*	+	3
DCO	*u*	+	+	3
DCO	+	*e-2*	*+/h*	1
Total	60 + : 55 *u*	68 + : 47 *e-2*	55 + : 60 *h*	115

Table 3. Segregation of *e-2, u,* and *Prx-4* in BC *e-2, u* × (F_1 *e-2, u* × *S. pennellii* LA0716). Data are from family 92L8063.

Type	***e-2***	***u***	***Prx-4***	**# Plants**
Parental	*e-2*	*u*	+	18
Parental	+	+	+/*p*	14
SCO	+	*u*	+	4
SCO	*e-2*	*u*	+/*p*	4
DCO	+	*u*	+/*p*	3
SCO	*e-2*	+	+/*p*	3
SCO	+	+	+	1
DCO	*e-2*	+	+	1
Total	**22 + : 26 *e-2***	**19 + : 29 *u***	**24 + : 24 +/*p***	**48**

Study of the effect of cytoplasmic male sterility on the expression of B and C class floral-identity genes in tomato species and hybrids

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Introduction

Cytoplasmic male sterility (CMS) is a phenomenon documented in more than 150 plant species. It had been determined that CMS results from a mitochondrial dysfunction that leads to the formation of abnormal anthers, pollen abortion or no pollen formation (Schnable, Wise 1998) and is inherited through the female parent. CMS plants usually appear normal, vigorous, and indistinguishable from the fertile homolog. CMS often affects the size and color of the petals (Andersen 1963, 1964; Petrova et al. 1999; Farbos et al. 2001; Leino et al. 2003) and may cause homeotic changes in floral structures (Kitagawa et al. 1994; Linke et al. 1999; Farbos et al. 2001).

According to the well accepted "floral quartet" model the four consecutive whorls of plant floral organs (sepals, petals, stamens, and carpel) develop in response to the expression of four key classes of floral-identity genes designated as A, B, C and E (Coen and Meyerowitz 1991; Theissen 2001). A and E classes of genes specify sepals; A, B and E - petals; B, C and E – stamens; and C and D – carpels. Many of these genes are highly conserved among dicotyledonous and monocotyledonous species (Chase 2006).

AP3 and *PI* subfamilies of genes are members of the B class of genes and are required for petal and stamen identity. The cultivated tomato *Solanum lycopersicum* has two *AP3* genes: *TAP3* and *TM6,* which demonstrate functional diversification in their roles in the development of the flower. *TAP3* loss-of-function mutants produce complete transformation of the petals into sepal-like structures and of stamens into carpel-like organs. The reduction in *TM6* function in transgenic plants results in homeotic defects primarily in the stamen, and reduced size of the petals. *TAP3* is highly expressed in the petals and stamens of developing buds, while *TM6* is transcribed mainly in stamens and carpels. The tomato *PI* gene (*TPI*) expression is confined to the stamens and petals of buds and is not affected by the down-regulation of *TAP3* and *TM6* (De Martino et al. 2006). The study of Mazzucato et al. (2008) demonstrated that the two *AP3* genes are actively transcribed in mature stamens and their expression is not affected by the homeotic phenotype of *pat-2* stamens. The same study indicated that *TPI* expression is more pronounced in the wild type mature stamens in comparison to the mutant. Four C class genes had been identified in tomato, among which *TAG1* is detected at high levels during flower development and its expression increases at anthesis (Pnueli et al. 1994; Busi et al. 2003). According to Hileman et al. (2006) *TAG1* is expressed in the stamens

and the petals, while *TAGL1* is transcribed in the stamens. Lozano et al. (1998) reported that *TM6* and *TAG1* expression in buds is up-regulated under low temperature stress which causes flower aberrations.

Recent studies had shown that the CMS phenotype is correlated with the disturbance in the expression of the nuclear genes that play a role in the development of male reproductive organs, particularly the B-class genes involved in the specification of petals and stamens (reviewed in Chase 2006). For example, studies in CMS lines of tobacco, carrots, wheat and *Brassica napus* had found correlations between the down-regulation of B class genes and the CMS phenotype (Zubko et al. 2001; Linke et al. 2003; Murai et al. 2002; Teixeira et al. 2005). Interestingly, a comparative global gene expression profiling of flower formation in a CMS line of *B. napus* and its fertile homolog revealed that CMS affected not only the expression of floral organ identity genes, but also genes implicated in energy production and metabolism; genes, whose products are targeted to the mitochondria and genes implicated in the cell-wall remodeling (Carlsson et al. 2007). The results of these studies emphasize that the mitochondrial genome of the CMS plants strongly influences nuclear gene expression, thus underlining the retrograde regulation between the mitochondria and the nucleus.

In tomato, CMS does not occur naturally. Few studies had reported CMS plants, all resulting from interspecific crosses (Andersen 1963; 1964; Valkova-Atchkova 1980). The CMS line created by Vulkova (termed CMS-pennellii) is comprised of the cytoplasm of *S. peruvianum* and the nuclear genome of *S. pennellii*. Our studies had shown that the maternal inheritance of the male sterility in CMS-*pennellii* has been stable over many generations, have manifested normal female fertility when pollinated by *S. pennellii* (Vulkova et al. 1997; Petrova et al. 1999). Recently we have conducted a comparative study correlating the bud and anther sizes with the development of pollen mother cells (PMCs) in the CMS-*pennellii* and *S. pennellii* (Stoeva unpublished). The study clearly indicated that CMS affects the development of the flower. The buds, corolla and anthers of the CMS line are smaller in comparison to *S. pennellii*, while the filament is of several magnitudes longer. The anthers are not coalesced laterally and do not form a staminal cone. Anthers do not form an apical pore and don't shed pollen. PMCs undergo normal meiotic division, but the produced pollen grains degenerate after the disintegration of the tetrads (Petrova et al. 1999; Stoeva et al. 2007; Stoeva unpublished). Initial microscopic analysis revealed that although the CMS anthers are reduced in size, the structure of their locules are of identical type and have similar tissue developmental patterns as the fertile homologue *S. pennellii* (Radkova 2002). Segregation studies in crosses between CMS-*pennellii* and the cultivated tomato *S. lycopersicum* indicated that at least one dominant nuclear restorer-of-fertility gene from the cultivated tomato acts in the restoration of male fertility (Petrova et al. 1999; Radkova 2002; Stoeva et al. 2007). Hybrid plants with varying percentages of restored male fertility carrying the CMS cytoplasm were produced from such crosses. These

hybrid plants also have a variable degree of restored "male fertile" flower characteristics.

Presently there are no studies that have investigated the effect of CMS on the expression of tomato nuclear genes. Our unique CMS system offers an excellent model for comparative research of the retrograde regulation of nuclear genes, and particularly the genes involved in development of stamens and petals, which are most affected by the CMS phenotype in tomatoes. The goal of this research is to study and compare the expression of B class genes *TM6*, *TAP3* and *TPI*, and C class floral organ identity genes *TAG1* and *TAGL1* in stamens, petals and buds of tomato species and hybrids with pollen sterility ranging from zero to 100%.

Materials and methods

All plants used in the study: *CMS-pennellii*, *S. pennellii*, hybrid plants H1 (76% viable pollen) and H8 (21% viable pollen) and *S. lycopersicum* line used in the hybrid crosses, were grown in one environmental chamber with a temperature of 20°C for 8 hours of darkness and 25°C for 16 hours of light. Stamens and petals were collected from mature flowers. Bud material was collected from buds with anthers sizes with PMC undergoing meiosis (Stoeva P., unpublished data), corresponding to stages 9-11 as defined by Brukhin et al. (2003). The bud sepals were removed at the time of the collection of material. All plant material was kept on ice and was either immediately used for RNA extraction or frozen at -80°C until used. RNA was extracted using the RNeasy Plant Mini Kit (Qiagen), followed by DNase treatment (Ambion TURBO DNA-*free* Kit). The reverse transcription (RT)-PCR reactions were carried out using illustra Ready-To-Go RT-PCR Beads (0.5-ml tubes) (GE Healthcare) with 100 ng of total RNA as template. RT reactions were carried out according to the manufacturer protocol at 42°C for 30 min followed by 5 min at 95°C. The PCR step for B class genes: *TAP3*, *TM6*, and *TPI* and the C class genes *TAG1*, *TAGL1* as well as the *ACTIN* gene was carried out using primarily published primer sequences (De Martino et al. 2006; Hileman et al. 2006). For each gene, the optimal annealing temperature conditions were determined using touchdown PCR with genomic DNA. The linear range of amplification of the RT product was determined for each pair of gene primers (Table 1). All reactions were carried out in an Eppendorf Mastercycler.

The expression of genes was assessed by gel electrophoresis and spot densitometry. Twenty-five micro liters from the RT-PCR products were run on 1.2 - 1.5% agarose gels and the gels were stained with ethidium bromide. The bands were viewed and spot densitometry was carried out with ChemiImager 4000 system. The expression of each gene was normalized to the expression of *ACTIN* (*ACT)* amplified from the same amounts and sources of RNA by dividing the Integrated Density Values (IDV) of the

nuclear floral-identity genes by the IDV for *ACT.* The obtained ratio was used as a measure of the level of expression of the gene.

Results and discussion

To test the hypothesis that CMS affects the expression of floral identity genes we studied the expression of B and C class genes in the sterile line CMS-*pennellii*, the fertile homolog *S. pennellii* , S. lycopersicum line that was used in crosses with the CMS line and two hybrids H1 and H8 with 76 and 21 percent pollen viability, respectively.

TM6 Our data supports previous studies that indicate the ubiquitous expression of *TM6* in developing buds and mature flower structures in the cultivated tomato (De Martino et al. 2006; Hileman et al. 2006; Mazzucato et al. 2008). The expression of *TM6* (Fig.1.) was detected in all studied tissues and genotypes (with one exception: no expression was detected in the buds of the H1 hybrid). The expression of *TM6* was lower in stamens in comparison to the petals in the two fertile species *S. pennellii* and *S. lycopersicum*. The gene was up-regulated in the stamens of CMS-*pennellii* in comparison to both the fertile homolog *S. pennellii* and the cultivated tomato, which may be an indication of the involvement of *TM6* with male sterility phenotype. On the other hand there was no difference between the level of expression of *TM6* in the stamens of the semi-sterile hybrids (76% and 21% pollen fertility) and the cultivated tomato. Considering these results, it is difficult to associate the expression of *TM6* in CMS-*pennellii* stamens with the CMS phenotype. The transcription of the gene was lower in the petals of the sterile line, which may be explained by the smaller size of petals in the CMS plant that resembles the effect of TM6i loss-of-function transgenic lines described by De Martino et al. (2006). The transcription of *TM6* in *S. lycopersicum* and *S. pennellii* was higher in the petals and lower in the stamens of fully expanded flower. These results differ from the data reported by the same authors for petals and stamens of pre-anthesis buds of *S. lycopersicum* and indicate dynamic changes in the spatial expression of this gene.

TAP3 *TAP3* expression was established in buds, petals and stamens of all genotypes (Fig.1.) which supports previous studies (Mazzucato et al 2008; Hileman et al. 2006; Xiao et al. 2009). Our results showed that *TAP3* gene is down-regulated in the buds of the CMS line and the semi-fertile hybrids in comparison to *S. pennellii* and *S. lycopersicum*, which could be explained with developmental differences between the buds of the studied genotypes. The transcription of the gene in the petals of the CMS line and the fertile isonuclear form was the same and higher in comparison to the other genotypes. This result may be an indication of genotypic differences in the tissue specific expression of the gene since the comparison involves nuclear genomes of two different species *S. pennellii* and the cultivated tomato. In the stamens of sterile line and even more in *S. pennellii*, *TAP3* expression decreased and was closer to the level of expression of the gene in the stamens of the semi-fertile hybrids and the cultivated

tomato. This result shows that *TAP3* expression is not affected by the CMS phenotype. In the cultivated tomato the expression of *TAP3* in the buds, stamens and petals was similar.

TPI *TPI* gene was expressed in both stamens and petals with the exception of the CMS petals (Fig.1). Since the data was not replicated the absence of *TPI* expression in CMS petals will not be interpreted. *TPI* transcription in the two fertile species was similar, decreasing in the stamens, considerably in *S. pennellii.* The expression of *TPI* in the semi-fertile hybrids was similar. The comparison of the transcription of *TPI* in the stamens across the studied genotypes does not reveal an association with the CMS phenotype.

TAG1 Our data determined that *TAG1* was expressed across all tissue sources and genotypes, supporting published data for stamens and petals of cultivated tomato (Hileman et al. 2006) and the data of Xiao et.al (2009) for buds and flowers of *S. pimpinellifolium*. Our results demonstrated that *TAG1* was differentially expressed in the buds, stamens and petals of most of the studied genotypes. The gene was down-regulated in the buds and up-regulated in the petals and stamens of the H1 and H8 hybrids. In the buds of CMS line and *S. pennellii* the level of expression of *TAG1* was similar, while comparison between the differential expression of the gene in their stamens indicated down-regulation in the male sterile form. In *S. pennellii* and *S. lycopersicum* the dynamics of the expression in the different flower parts is similar with higher expression in the buds and stamens and lower expression in the petals.

TAGL1 *TAGL1* was expressed in buds, petals and stamens (Fig. 1). Its pattern of expression was similar in *CMS-pennellii*, *S. pennellii* and the hybrids with the lowest expression in the buds and the highest expression in the petals. In *S. lycopersicum* the highest expression was detected in the stamens. The gene was down-regulated in the stamens of the male sterile line and the H8 hybrid (27% male sterility) which may be an indication of the involvement of this gene in the male sterile phenotype. The data of Hileman et al. (2006) confined the expression of the gene to the stamens but not to the petals of developing buds. On the contrary, our data shows that its expression is relatively high in the petals of all studied genotypes including the cultivated tomato and *S. pennellii.* Our results also indicated the *TAGL1* expression in the petals is genotype dependent and its induction may differ even between closely related species.

Conclusion

In this study we tested the hypothesis that anther and petal modifications and the viability of the pollen grains in the CMS tomato plants and hybrids are driven by signals coming from the altered expression of B and C class floral identity genes. Our first results showed that all studied genes were expressed in the petals and stamens of mature flowers of all studied genotypes. All genes were expressed in the developing

bud tissues of the CMS plants, the fertile homologue and the cultivated tomato. The present data does not support our hypothesis that the altered expression of *TM6, TAP3, TPI* and *TAG1* genes is correlated to the male sterility phenotype. The differential expression of *TAGL1* in buds, petals, stamens of the CMS-*pennellii*, H8 hybrid and *S. pennellii* are in support to our hypothesis and indicate that the expression *TAGL1* gene may be affected by the mitochondrial-nuclear interaction and could be implicated in the development of male sterile phenotype in tomatoes.

Acknowledgements

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Table 1.

Gene	Annealing temperature range	Number of PCR cycles used to amplify RT products
TM-6	55-58	26
TAP3	56	25
TPI	51-58	26
TAGL	55-58	25
TAGL1	55-58	25
ACT	55-58	23

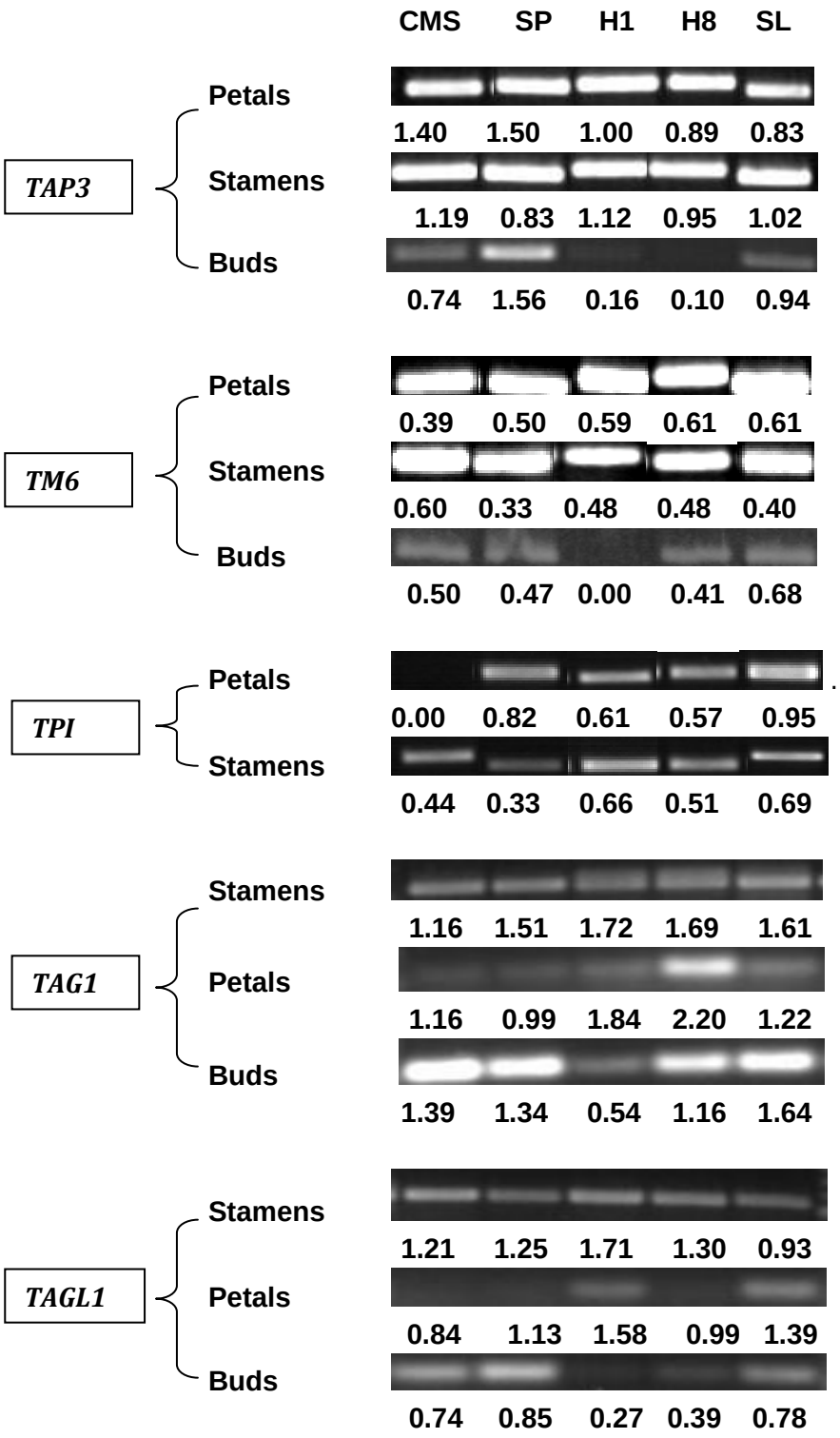

Fig.1. RT-PCR of B class (*TAP3, TM6* and *TPI*), and C class (*TAG1* and *TAGL1*) floral organ identity genes in various tissues in CMS-*pennellii* (CMS), *S.pennellii* (SP), H1 hybrid (H1), H8 hybrid (H8) and *S. lycopersicum* (SL). Data was obtained from one RT-PCR analysis. Values were obtained using ChemiImager 4000 system and were normalized to the expression of *ACTIN* amplified from the same amounts and sources of RNA.

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A CAPS Marker linked to the Tomato gray leafspot (*Stemphyllium* sp.) Resistance Gene *Sm*

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Introduction: Tomato gray leafspot is an important foliar disease in warm growing regions caused by four *Stemphylium* species. It has been controlled by resistance conferred by a single incompletely dominant gene, *Sm* (Bashi et al.,1973). RFLP markers such as T10 and TG110 were showed to be linked to *Sm* (Behare et al., 1991). In the present study, we report on a recessive CAPS marker, CT55, which is linked to *Sm* and can likely be used in marker-assisted selection for gray leafspot resistance.

Plant Materials: Eight inbred lines developed from crosses with begomovirus tolerant breeding lines derived from *S. chilense* accession LA2779 were grown in the field at the Gulf Coast Research & Education Center in fall 2006. These were supplied by Jean-Claude Mercier of Clause Seed Company. Four lines; 06CH3604.02, 06CH3604.05, 06CH3604.10, and 06CH3604.ML were homozygous susceptible to gray leafspot and four lines; 06CH3605.02, 06CH3605.07, 06CH3605.10, and 06CH3605.ML were resistant to gray leafspot. The lines were grown in a completely randomized block design with 3 blocks and 10 plants per plot and were inoculated with TYLCV to see if there was a difference in resistance to TYLCV that was associated with gray leafspot resistance. The TYLCV work is not the subject of this report, but we did not find an association of TYLCV resistance with gray leafspot susceptibility which was our hypothesis at the time.

PCR Methods: Total genomic DNA was isolated from young leaves of the plants 3 weeks after transplanting to the field, as described previously (Fulton et al., 1995). PCR reactions were performed in a Perkin-Elmer GeneAmp PCR 9700 Thermal Cycler and included 94^{o}C for 2 min, followed by 35 cycles of 30 s at 94^{o}C, 60 s at 55^{o}C and 60 s at

72°C. These cycles were followed by 72°C for 7 min, and the reaction was held at 15°C. The PCR products were separated on a 1.5% agarose gel stained with ethidium bromide and visualized under ultraviolet (UV) light. The primer sequences for marker CT55 are: forward, CATCTGGTGAGGCGGTGAAGTA, and reverse, TCCGCCCAAACAAAACAGTAATA.

Results and Discussion: A PCR fragment of ~400 bp was generated for both *Stemphyllium*-resistant and *Stemphyllium*–susceptible materials with marker CT55 (data not shown). After digestion of the PCR product with enzyme *Dde*I, the susceptible genotypes produced a fragment of ~330 bp. Two other bands, of sizes ~200 bp and ~140 bp, respectively, were generated from both resistant and susceptible genotypes (Fig. 1). Therefore, CT55 is a recessive marker - heterozygous and homozygous susceptible plants share the same band pattern; i.e., all three bands, while the homozygous resistant plants have only the two lower bands. CT55 distinguished the genotypes tested here, but it has yet to be tested for its utility on a broader range of germplasm.

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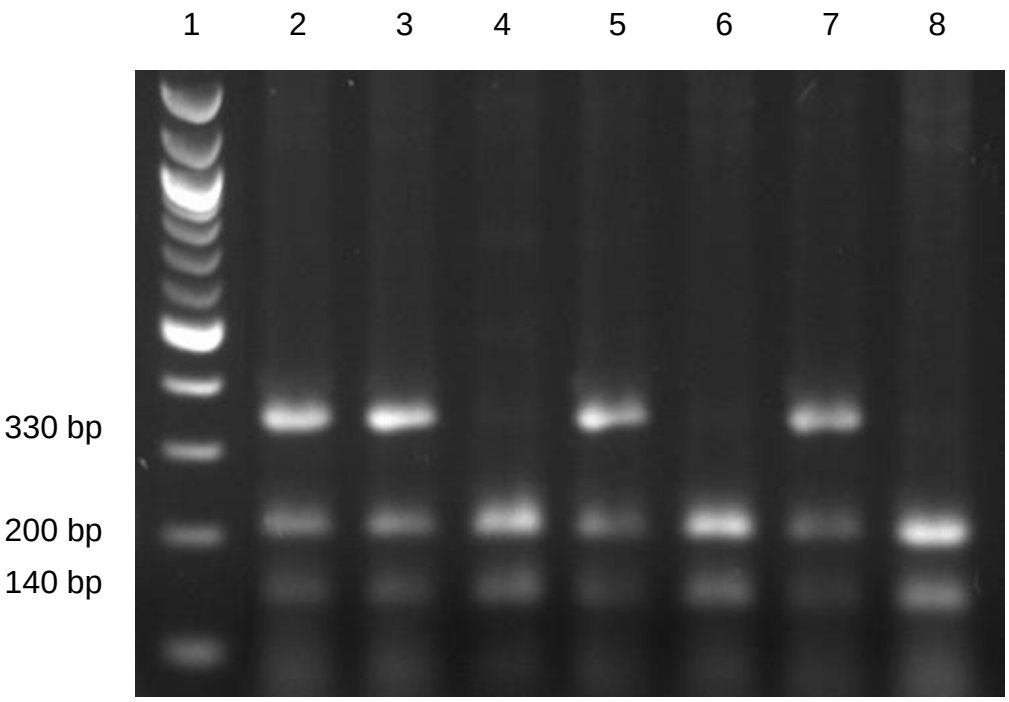

Fig.1. DNA fragments after *Dde*I digestion of PCR products amplified with CT55 primer set. Lane 1, 100-bp DNA ladder; lanes 2, 3, 5 and 7 are *Stemphyllium* susceptible lines (*Sm*-)- 06CH3604.ML, 06CH3604.02, 06CH3604.05, and 06CH3604.10; lanes 4, 6 and 8 are *Stemphyllium* resistant lines (*Sm*+)- 06CH3605.02, 06CH3605.07, and 06CH3605.10. Resistant line 06CH3605.ML is not shown.

Evaluation of Recombinant Inbred Lines for Resistance to *Ralstonia solanacearum* in Guatemala and Preliminary Data on PCR-based Tagging of Introgressions Associated with Bacterial Wilt-Resistant Line, Hawaii 7996

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Bacterial wilt (BW) caused by *Ralstonia solanacearum* (Rs) is a serious disease of tomato in southeastern Guatemala. Sánchez-Pérez et al. (2008) found that the strain of *R. solanacearum* on tomato in the lowlands in Guatemala was phylotype I sequevar 14 (race 1, biovar 3), which is the phylotype most prevalent in Taiwan (J.-F. Wang, pers. com.).

All tomato hybrids currently grown in Guatemala are susceptible to Rs, and thus, the development of hybrids with resistance to Rs would provide a major management option for growers. Breeding for resistance to Rs in tomatoes has been difficult (Scott et al. 2005). *Solanum lycopersicum* cv. Hawaii 7996 (H7996) is resistant to Rs race 1 phylotype I and molecular studies have shown that resistance to be controlled by several QTLs against Rs Pss4 (Wang et al., 2000) associated with chromosomes 2, 6 and 12 near markers GP504, TG73, and TG564, respectively. Thoquet et al. (1996a,b) found markers linked to resistance from H7996 on chromosomes 3, 4, 6 and 8 and possibly chromosomes 10 and 11. Scott et al. (2005) stated that "it is possible that few major genes together with several minor genes condition resistance to bacterial wilt in tomato."

This research reports the use of RFLP or COSII markers associated with the QTL regions for chromosomes 6 and 12 (Wang et al., 2000) to develop PCR-based protocols for detection of sequence differences between the resistant line, H7996, and susceptible line, *Solanum pimpinellifolium* cv. West Virginia 700 (WVa700). These sequence differences were further evaluated in selected recombinant inbred lines (RIL) and other breeding lines provided by AVRDC. The RILs were developed from a cross

of the resistant line, H7996, with the susceptible line, WVa700 (Thoquet et al., 1996a,b). J.-F. Wang (AVDRC) provided F_8 seeds of RILs that had been scored as either resistant or susceptible in greenhouse and field trials at several locations in Taiwan.

Methods:

Evaluation of the RILs: Four-week-old seedlings of thirty RILs were transplanted on March 13, 2008 into a Rs-infested field in Agua Blanca, Jutiapa, Guatemala. The resistant line, H7996, and two susceptible lines, WVa700 and L390, were included as controls. The 33 entries were grown in a completely randomized block design with three blocks and eight plants per entry. A susceptible commercial hybrid grown in the region was interspersed as follows: three plants of the susceptible commercial hybrid, then 8 plants of an entry, then 8 plants of another entry followed by 3 plants of the susceptible commercial hybrid and this was continued throughout. In this way, the susceptible hybrid was located next to all entries. Two teams evaluated the RILs on June 4, 2008 (75-days-after transplanting) at which time most of the susceptible commercial hybrid and L390 plants were dead. The presence of Rs was confirmed from randomly selected wilted plants using immuno-strip test of Agdia. The mean of the three replications for each entry is presented as percentage of surviving plants.

PCR Primer Design: The general strategy for designing PCR primers for markers between 32 (T0834) to 43 cM (TG73) on chromosome 6 and between 30 (CT120) and 65 cM on chromosome 12 (markers listed on the SGN site) was to do a Blast search at GenBank and then to use the sequences that corresponded to exons for the primer design. Primers were designed so that the amplified PCR fragment would include one or more introns. If no matches were obtained with the Blast search, then the primers were designed form the SGN sequences. Primers for five markers on chromosome 6 and two on chromosome 12 were evaluated. The PCR primers were synthesized by Integrated DNA Technologies, Coralville, IA and diluted with HPLC-purified water (Fisher Scientific).

PCR methods: Total DNA was extracted from fresh leaves with the Puregene® DNA Purification Kit (Gentra Systems, Inc., Minneapolis MN) following the manufacturer's instructions. The DNA extract was adjusted to approximately 15 ng/µl or until PCR fragments were obtained. The 25-µl PCR reaction mixture contained: 2.5 µl 2.5 mM dNTP, 2.5 µl buffer 10X, 2.5 µl 25 mM $MgCl_2$, 0.1 µl *Taq* polymerase (Promega Corp., Madison WI), 2.5 µl each primer at 10 µM, 2.5 µl diluted DNA extract, and HPLC water (Fisher Scientific). The parameters for the thermal cycler (MJ DNA Engine PT200 Thermocylcer™, MJ Research Inc., Waltham MA) were as follows: denaturation at 94 C for 3 min, then 35 cycles at 94 C for 30 sec, annealing at 53 C for 1 min and extension at 72 C for 1 min, followed by 72 C for 10 min, then the reaction was maintained at 4 C.

The PCR fragments were separated by gel electrophoresis using 1.5% agarose and 0.5X TBE buffer, stained with ethidium bromide and observed with UV light.

Sequencing methods: PCR primer pairs that produced strong single bands were used to amplify DNA from the different germplasms. These PCR fragments were then sequenced. The PCR reaction mixture was treated with shrimp alkaline phosphatase (Promega Corp.) and exo-nuclease I (EpiCentre Biotechnologies) to remove residual PCR primers. Samples were then sequenced with the Big Dye protocols and electrophoresis was performed at the University of Wisconsin-Madison Biotechnology Center. The CHROMAS and DNAMAN sequence analysis software were used. Sequences are available by contacting D. P. Maxwell.

Results and Discussion:

Evaluation of RILs: The general response of the RILs to Rs in Guatemala was similar to that in Taiwan (Table 2). The percentage survival for the resistant parent, H7996, was 84% and 88% in Guatemala and Taiwan, respectively. For the susceptible parent, WVa700, the survival was 22% and 31% in Guatemala and Taiwan, respectively. Nine RILs (GT-susceptible group) had survival rates ≤ 35% in Guatemala and five of these RILs also had less than 35% survival in Taiwan. Nineteen RILs (GT-resistant group) had survival rates ≥ 75% in Guatemala and only nine RILs had this survival rate in Taiwan. Of these nine Taiwan RILs, eight were in the GT-resistant group. The exception was RIL 46, which had 79% survival in Taiwan and 69% in Guatemala. Because of the similarity of survival rates for the RILs in Taiwan and Guatemala, it is expected that tomato hybrids with bacterial wilt resistance derived from the resistance source, H7996, would be useful in both Guatemala and Southeast Asia, where Rs phylotype I is prevalent.

Detection of Introgressions: The QTL associated with chromosome 6 was shown in Wang et al. (2000) to be between CP18 and TG240 (35 cM and 37.9 cM, respectively, for the Tomato-EXPEN 1992). Another marker, TG73, which is present on the maps for Tomato-EXPEN 1992 and 2000, is mapped to 37.9 cM and 43.3 cM, respectively. Primers were designed for five markers from the EXPEN-2000 map from 32 cM to 43.3 cM. For the marker T0834 at 32 cM, the PCR primers produced a 400-bp fragment for all samples tested and there were no sequence differences between H7996 and the sequences from three susceptible lines. The primers for C2_At1g2164 (37 cM, P6-37F1/R1) gave a PCR fragment of 850 bp for H7996, WVa700 and a susceptible line, M82-1-8. The sequences from these fragments were identical. P6-38.3F1/R1 primer pair for marker C2_At1g44835 (38.3 cM) yielded a 590-bp fragment for H7996 and WVa700. There was a SNP at nt 258; a G for H7996 and a C for

WVa700. For the COSII marker at 41.5 cM (At1g03150), a large PCR fragment (about 1,000 bp) was obtained from H7996 and M82-1-8. Partial sequences were compared and four SNPs and two indels were detected. The primers for PTG73F1/R1 (TG73, 43.3 cM) gave a large fragment (about 1,100 bp) for H7996 and WVa700; and one SNP was detected between the sequences for these lines. H7996 had a G and WVa700 had a C. These data indicate that the primer pairs for locations at 38.3 and 41.5 cM could be used to evaluate additional germplasm for the occurrence of an introgression associated with H7996.

The location of the QTL on chromosome 12 was predicted to occur from 30 to 65 cM (Tomato-EXPEN 1992 map) by Wang et al. (2000) (see Fig. 4). Marker T1667 (39 cM, EXPEN 2000 map) was used to design primers P12-39F1/R1, which yielded a PCR fragment of ca. 500 bp. The PCR fragments were sequenced and H7996 and WVa700 were 462 and 461 nt, respectively. There were 6 SNPs and two indels (one nt and two nt indels) between these two sequences. For the marker at 54.5 cM, the sequences of the PCR fragments were 674 bp and there was one SNP between H7996 and WVa700. The primers for the marker T1667 would be the most useful for evaluating the presence of the H7996 introgression in other resistant germplasm.

Evaluation of RILs for introgressions from H7996: Susceptible and resistant RILs were evaluated for the presence of the three markers associated with H7996 (Table 3). The sequences associated with WVa700 for these markers were also the sequences in the susceptible line, L390, from AVRDC (Wang et al., 2000) and one other BW-susceptible line, Gh13. All three susceptible RILs had the sequence from WVa700 for P6-38.3F1/R1 (chr. 6) and two had the WVa700 sequence at P12-39F1/R1 (chr. 12). Unfortunately, sequence was not obtained for susceptible RIL-183 for this marker on chr. 12. Two of the susceptible RILs had the WVa700 sequence for P6-41F4/R4 (chr. 6), but the susceptible RIL-170 had the H7996 sequence at this marker. Sequences were obtained for these markers for six resistant RILs. All six RILs had the H7996 sequence for the two markers on chr. 6. For the marker on chr. 12 only four resistant RILs were tested and they all had the H7996 sequence. Unfortunately no data are available for the chr. 12 marker for the other two RILs. The sequence associated with H7996 was also found in H7997 (see Scott et al., 2005, for information on H7997) for markers P6-41F4/F5 (chr. 6) and P12-39F1R1 (chr. 12). The P6-38.3F1/R1 (chr.6) marker was not tested with H7997.

Thus from this limited data, it seems that the presence of the introgressions (H7996 sequence) on chr. 6 and chr. 12 are associated with a resistant phenotype in these RILs. It would be very interesting to test the RILs with moderate levels of resistance for these markers.

Evaluation of BW-resistant breeding lines for H7996 introgressions: Three BW-resistant breeding lines provided by P. Hanson (World Vegetable Center, AVRDC) were tested for the presence of H7996 sequences for markers P6-41F4/R4 and P12-39F1/R1. Only line CLN2413L had H7996 sequences on chr. 6 and 12. Line CLN2418A had the H7996 sequence for the marker on chr. 6 and the sequence for WVa700 for the marker on 12. In contrast, line CLN1466EA had the H7996 sequence for marker on chr. 12 and the WVa700 sequence for marker on chr. 6. All resistant inbreds had at least one of the markers on chromosome 6 or 12. These results are consistent with a quantitative inheritance for BW resistance (Scott et al., 2005), but also indicate that these markers might be of value for pyramiding resistance loci.

Conclusions:

Resistance to BW in tomato is controlled by several QTLs (Thoquet et al., 1996a,b; Wang et al., 2000) and in this report, sequence data are provided for introgressions from the resistant line, H7996, on chromosome 6 at 38.3 and 41.5 cM and on chromosome 12 at 39 cM. From this limited study of RILs and BW-resistant inbred lines, it is evident that when both introgressions were present, the RILs or inbred breeding lines were resistant (unpublished data). Unfortunately the phenotype could not be predicted if only the introgression in chromosome 6 or 12 was present. One RIL had the introgression on chromosome 6 and was susceptible. For two BW-resistant inbreds from the World Vegetable Center, each had only one introgression from H7996. Because of the complex nature of resistance to BW, these results are only a beginning in the development of markers for use in a tomato breeding program.

H7996 was found to be highly resistant to Rs race 3 phylotype II in greenhouse studies and a QTL *Bwr-6* was mapped to TG73 (43.3 cM) on chromosome 6 (Carmeille et al., 2006), which corresponds to the region of the marker for race 1 phylotype I detected by Wang et al. (2000). This QTL near TG73 could be detected by the PCR-based marker (P6-41F4/R5) developed in this study and should have relevance for resistance to race 1 phylotype I and race 3 phylotype II.

Recently Miao et al. (2009) reported the development of SCAR markers for detection of BW resistance in tomato. One marker ($TSCAR_{AAT/CGA}$) was present in 159 of 171 resistant F_2 plants and in 2 of 129 susceptible F_2 plants.

With the development of high-throughput methods for SNPs genotyping (Pick, 2009), the markers reported here on chromosome 6 and 12 could easily be converted to SNP technologies and used to pyramid BW-resistance loci in tomato breeding programs.

Table 1. Primers sequences and markers on chromosome 6 and 12.

Marker	cM	PCR Primer	Primer pair
Chromosome 6			
T0384	32 cM	P6-32F1	CATTGTTGTTGCTCCTCAG
" "		P6-32R2	CTG CTC CTT CCA CTA AAT ATA ACT G
C2_At1g21640	37 cM	P6-37F1	CCCAAGAGAAGATGACTGTTCT
" "		P6-37R1	GTGGCCACAATGACACCATCACCTTGC
C2_At1g44835	38.3 cM	P6-38.3F1	GAGCTTCAAATTGATTTCACCAAACATG
" "		P6-38.3R1	GAGCCATTCACCCTCCTTTTCC
C2_At1g03150	41.5 cM	P6-41F1	GATTATTTCCATGTTGCAAAAGCTCC
" "		P6-41R1	GATTCACCTTGCCCTTCAACTTTTCC
" "		P6-41F4	CAAATATAAGCTTGAAGGTAGGAC
" "		P6-41R4	CACGGAAGGGAGTATAAGAGAATG
" "		P6-41F5	GAAATAATATGCCTAAAGCTCTCC
" "		P6-41R5	CATGAAGAGGCCAGAATACACC
TG73	43.3 cM	PTG73F1	GTAGTACGAGCTATTGTGTCTCAGC
" "		PTG73R1	CAGAACAGAGAAATCCTAGCCACTGATG

Table 1 (continued). Primers sequences and markers on chromosome 6 and 12.

Marker	cM	PCR Primer	Primer pair
Chromosome 12			
T1667	39 cM	P12-39F1	GATTCAACTTATGCAGAGAGGG
" "		P12-39R1	CCTCTCTCGGAATTTTGTAAC
C2_At5g42740	54.5 cM	P12-54.5F2	CAGCACAGAAAACAGACCCG
		P12-54.5R2	GGCTACATCAATTGGATCAACATTCG
TG564	57.6 cM	PTG564F1	CAACTCATGGTGCTTATCTTACTGACCTTAG
" "		PTG564R1	CTTATGTGAGATGTTGAAAACTGGAAAGAAG
TG564		PTG564F2	CACCGCCAAATTTAACTTTAATCAACTG
		PTG564R2	CCATAGTGTTCATCATTCAAGATCTGTCC

Table 2. Percentage of surviving plants for Hawaii7996, WVa700, L390, and 30 RILs in Guatemala.

	Mean % survival[2]	
Code[1] (RILs)	**GT**	**Taiwan**
170	0	25
158	1	35
183	11	40
79	18	39
30	21	35
83	21	35
182	28	25
6	33	40
100	37	21
38	45	25
89	64	59
46	69	79
150	76	67
13	78	70
12	80	89
70	81	89
95	83	55
RILs	GT	Taiwan
18	85	85
130	88	79
23	89	70
26	91	59
41	94	71
74	94	75
200	94	89
32	95	55
162	95	89
92	96	63
39	100	70
128	100	67
154	100	100
H7996	84	88
L390	11	8
WVa700	22	31

1) Identification (ID) code for the RILs (F8 families), H7996 (resistant parent for RILs) and WVa700 (susceptible parent for RILs). Line L390 was the susceptible control.

2) Mean percentage of plants surviving from three replication

Table 3. Sequences for two markers on chromosome 6 and one marker on chromosome 12 associated with the susceptible line, L390, the parents of the RILs, H7996 and WVa700, and nine RILs.

		Introgression[3)]		
Line[1)]	**% Survival[2)]**	**Chr. 6 38.3 cM**	**Chr. 6 41.5cM**	**Chr. 12 39 cM**
H7996	84	H	H	H
WVa700	22	W	W	W
L390	11	W	W	W
RIL-158	1	W	W	W
RIL-170	0	W	H	W
RIL-183	11	W	W	nd[4)]
RIL-26	91	H	H	H
RIL-32	95	H	H	H
RIL-41	94	H	H	H
RIL-74	94	H	H	H
RIL-162	95	H	H	nd
RIL-200	94	H	H	nd

[1)] RIL's are F8's of the cross of H7996 by WVa700 and supplied by J.-F. Wang.

[2)] Mean per cent survival for three replications in the BW plot at Agua Blanca, Guatemala.

[3)] Introgression codes: H = sequence for H7996; W = sequence for WVa700. Primers used: P6-38.3F1/R1, P6-41F4/R4 or P6-41F5/R5, P12-39F1/R1.

[4)] nd = no data.

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Effectiveness of the *Ty-3* introgression for conferring resistance in recombinant inbred lines of tomato to bipartite begomoviruses in Guatemala

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Management of begomovirus-incited diseases on tomatoes in Guatemala continues to be an expensive practice. Currently, in one of the main tomato growing regions, the Salama Valley, the growers have started using whitefly-proof polypropylene fabric (AGRIBON™) to prevent the inoculation of tomato transplants for the first 4-6 weeks; and in some cases, fabric (AGRIBON™) macro-tunnels are used for the entire growing season. Thus, there continues to be a need to better understand the genetics of resistance to begomoviruses (Vidvaski, 2007) and the development of horticulturally acceptable hybrids.

A begomovirus-resistant inbred line, Gh13, had been selected from the hybrid, FAVI 9 (Vidavsky and Czosnek, 1998), in plots that had multiple begomoviruses (Mejía et al. 2005; Nakhla et al., 2005). Ji et al. (2007b) reported that the *Ty-3* introgression associated with chromosome 6 was a major contributor to resistance to begomoviruses. This introgression can be detected by a co-dominant SCAR marker P6-25F2/R5 at 25 cM (Ji et al., 2007a). This *Ty-3* introgression was present in Gh13 from marker C2_At3g56040 (19 cM) to T0834 (32 cM) and the *Ty-1* introgression was absent (Martin et al., 2007; unpublished data). Garcia et al. (2008) found that the *Ty-3* introgression significantly explained the resistant genotypes in an experiment with F_3 families generated using Gh13 as the resistant parent and M82 as the susceptible parent.

In this study, the resistant line, Gh13, was crossed with the susceptible line, HUJ-VF, that lacked the *Ty-1* and *Ty-3* introgressions; and about 100 F_2 plants were selfed for 3 or 4 generations to create the recombinant inbred lines (RILs). These RILs were scored for the presence of the *Ty-3* introgression with PCR primers P6-25F2/R5 for the marker at 25 cM on chromosome 6 (Ji et al., 2007a).

Materials and Methods:

PCR methods: Plant extraction and PCR protocols were the same as reported in Garcia et al. (2008) and involved the PCR primers P6-25F2/R5. Five seedlings were extracted together for each RIL and this DNA extract was used in the PCR reactions to determine the presence of the *Ty-3* introgression.

Generation of the RILs: The begomovirus-resistant inbred, Gh13 (*Ty-3/Ty-3*), was crossed with the susceptible inbred, HUJ-VF (*ty-3/ty-3*), which was provided by F. Vidavsky (Hebrew University of Jerusalem). F_2 plants were grown in a greenhouse and F_3 seeds collected from 100 randomly selected plants. Each generation was grown in a greenhouse and self-pollinated; and seeds were collected from individual fruit. The F_4 and F_5 generation RILs were used for the field experiment. The genotype of each RIL was determined as described above.

Field evaluation of disease severity for the RILs: The experimental design was a randomized complete block with five plants per plot and three blocks. The resistant parent, Gh13, and the susceptible inbred, HUJ-VF, were coded and included in each block. Four-week-old seedlings were transplanted on 17 Dec. 2008 into a field near Sanarate, Guatemala where high levels of viruliferous whiteflies were present. All entries were coded before transplanting to eliminate any bias during scoring. Each plant was scored on 28 Jan. 2009 (42 days after transplanting) using a disease severity index (DSI) from zero to six. The DSI descriptions are: 0, no virus symptoms; 1, extremely slight symptoms; 2, slight symptoms; 3, moderate symptoms; 4, severe symptoms with deformed leaves; 5, severe symptoms and stunted plant; 6, very severe symptoms, no marketable fruit and very stunted plant. Plants with DSI ≤ 3.0 were considered resistant, as these would yield marketable fruit. The disease responses of the three blocks were uniform; therefore a mean was calculated for each entry of 15 plants.

Results and Discussion:

The genotype of 88 RILs was determined for the *Ty-3* introgression. Forty-six were *ty-3/ty-3*, 41 were *Ty-3/Ty-3*, and one was *Ty-3/ty-3*. The homozygous RILs were planted and the DSI determined for each plant at 42-days-after transplanting. The resistant parent, Gh13, had a DSI = 1.8 sd 0.7 and all 15 plants of the susceptible parent, HUJ-VF, had a DSI = 6.0. All 46 of the ty-3/ty-3 introgression RILs had DSI's ≥4.1 (Table 1). The 41 RILs with the Ty-3/Ty3 introgression were divided into three groups: resistant (DSI ≤3.0, 9 RILs), moderately resistant (DSI 3.1 – 4.0, 7 RILs), and susceptible (DSI ≥4.1, 25 RILs). As expected, only RILs that had the *Ty-3* introgression

were resistant or moderately resistant. However, the low number of highly resistant RILs was unexpected, since in the previous experiments with F3 families, the presence of the *Ty-3* introgression generally predicted the resistant phenotype. Thus, it is suggested that resistance loci in addition to *Ty-3* are necessary for expression of high levels of resistance. This agrees with the observation by Vidavsky and Czosnek (1998) who separated resistance into two categories: tolerance (virus present and modest symptoms) and resistant (no virus present and no symptoms). Tolerance was conditioned by one dominant major gene and resistance was controlled by multiple recessive genes. The resistant inbred, Gh13, was developed from a hybrid, FAVI 9, that was known to have the dominant and recessive genes as discussed by Vidavsky and Czosnek (2008).

In recent years, there has been considerable interest in understanding the genetic bases of resistance to begomoviruses in tomatoes. In all cases, resistance loci have been introgressed from wild species. Several accessions of *Solanum chilense* have contributed resistance loci located on chromosomes 3 and 6 (Zamir et al. 1994; Ji et al. 2007b, 2008). *Ty-1* locus (accession LA1969) was the first resistance locus to be mapped and is located on chromosome 6 near 8 cM (Zamir et al., 1994). Ji et al. (2007b, 2008) described the *Ty-3* and *Ty-3a* resistance loci from LA2779 and LA1932, respectively. Both loci are located on chromosome 6 near 25 cM. *Ty-4* from LA1932 was located on chromosome 3 near 81 cM (Ji et al., 2009). In a study by Ji et al. (2009) *Ty-3a* explained more of the variance in resistance than *Ty-4*. Four accessions of *S. peruvianum* contributed to the *Tomato yellow leaf curl virus*-resistant line TY172 (Anbinder et al., 2009), and similar lines (TY197 and TY198) to TY172 were highly resistant to bipartite begomoviruses in Guatemala (Mejía et al., 2005). In a mapping study with TY172 as the resistance source Anbiner et al. (2009) found that resistance is controlled by one major QTL (*Ty-5*) on chromosome 4 near 46 cM and four minor QTLs. In these mapping populations the minor QTLs came from either the resistant or the susceptible parents. Since *S. chilense* and *S. peruvianum* are phylogenetically closely related (Peralta et al., 2008), it is suggested that these *S. peruvianum* QTLs may also be present in inbreds with resistance derived from *S. chilense*. The genetic studies reported by Ji et al. (2007b, 2009) and Anbinder et al. (2009) as well as our results on the RILs reported here stress the complex nature of resistance to begomoviruses. Thus, molecular markers may be useful in development of begomovirus-resistant inbreds, but good field testing for begomovirus-resistant inbreds remains essential.

Table 1. Disease severity index (DSI) for the 87 RILs with *Ty-3/Ty-3* or *ty-3/ty-3* genotype.

	Number of RILs	
DSI's	*ty-3/ty-3*	*Ty-3/Ty-3*
0 – 2.0	0	0
2.1 – 3.0	0	9
3.1 – 4.0	0	7
4.1 – 5.0	26	16
5.1 – 6.0	20	9

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Response of tomato lines (*Solanum lycopersicum x Solanum pennellii*) and their parental genotypes toward high temperatures and drought

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Introduction

Most commercial tomato varieties are sensitive to unfavorable environmental factors during different stages of plant development. A complementary approach of agriculture methods currently followed is to minimize losses by stress factors and to develop, via genetic means, high temperature and drought tolerant cultivars with the ability to escape or tolerate effects of the stress. Potential sources of genes for drought tolerance in tomato have been identified within the related wild species *S. pennellii* (Rick and Chetelat, 1995).

Under stress conditions the photosynthetic apparatus (PSA) is one of the most sensitive systems. High temperature and drought strongly influence parameters of the photosystem II (PSII); fast fluorescence emissions which are successfully used as criteria of assessment to stress tolerance (Goltsev et al., 1994; Stirbet et al., 2001; Petkova et al., 2007; Jing Yang et al., 2009).

The aim of this work is to study the effect of high temperature and drought stress on PSA in hybrid tomato lines (*S. lycopersicum x S. pennellii*) and their parental genotypes.

Materials and methods

The PSA efficiency of 21 F_4 hybrid lines (*S. lycopersicum x S. pennellii*), 3 varieties and accession 964750063 (*S. pennellii*), grown under field conditions, were evaluated by chlorophyll fluorescence parameters – initial (Fo), variable (Fv), maximum (Fm) and the ratios between them. The experiments were conducted in 2006-2008 during the reproductive period of the plants (July-August) under ambient temperature of about 25^{o}C (control) and high temperature of 37-39^{o}C. Before the measurements plants were not irrigated for a week.

Fluorescence parameters were registered in 10 replications, on intact, 30-min dark adapted, fully developed leaves, illuminated with actinic light (>650 nm) with

photon flux 1500 µmol.m^{-2}.sec^{-1}. A fluorimeter Plant Efficiency Analyzer (PEA MK2, Hansatech, UK) was used.

Values of chlorophyll fluorescence parameters and their ratios in tomato plants at high temperature and drought are expressed as a percentage to the values measured at 23-25^{o} C.

The data were statistically processed by the common MS Excel software.

Results and Discussion

The changes in values of chlorophyll fluorescence parameters (expressed in percentages) of the stressed plants compared to the plants under normal conditions are presented in Table 1. It was established that the loss of the excitation energy during its transfer from the pigment bed to reaction centre (RC) of PSII, expressed by Fo, increased under high temperature stress (Briantais et al., 1996; Yordanov et al., 1997). Considerable differences between the initial fluorescence values in high temperatures compared to the controls are observed in variety Milyana, and the hybrid lines 1842 and1838. The Fo values of these genotypes exceeded the controls by 16 - 20%. The lowest deviation of this parameter is registered in hybrid lines 1852, 1848 and 1855 (Table. 1).

The reduced potential of PSII activity (Fv/Fo) under the stress conditions proved to have a higher level of sensitivity to the stress than the maximum quantum efficiency of PSII primary photochemistry, expressed by the ratio Fv/Fm. According to Bolhar-Nordenkampf and Oquist (1993) the variable/maximum fluorescence ratio (Fv/Fm) in the plants with normal physiological status is from 0.75 to 0.85. Compared to the controls, it was considerably reduced in the line 1842 and variety Pautalya (7.33 % and 7.31%, respectively). Values of Fv/Fm under stress conditions, close to the biological minimum, are registered in lines 1842, 1849 and 1840 (0.764, 0.766, and 0.767, respectively) (Fig.1).

In most of the studied genotypes the ratio Fv/Fm was slightly reduced. It remains almost unaltered in hybrid lines 1848, 1855, 1851, 1852 and 1844. The plants from these genotypes also showed a slight temperature-induced reduction in Fm values.

On the basis of summarized analysis of the changes in different chlorophyll fluorescence parameters, high tolerance to the studied abiotic factors is registered in the hybrid lines 1848, 1852 and 1844. From the parents participating in the hybrid combinations accession 964750063 expressed the highest tolerance to the studied stress factors. Probably it is due to the intermediate morphological leaf type in hybrids coming from *S. pennellii*.

Conclusions

Exposure of tomato plants to high temperature and water deficit beyond their biological requirements results in alteration of photosynthetic activity, particularly on PSII efficiency. Although there were differences in the values of the chlorophyll fluorescence parameters in the studied tomato genotypes, the level of tolerance is comparatively high. The highest temperature and drought stress tolerance is established in hybrid lines 1848, 1852 and 1844 – these have potential for breeding purposes.

Acknowledgement:

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Table 1. Chlorophyll fluorescence parameters and their ratios in tomato plants at stress conditions – means from the period 2006-2008

Genotype	Fo	Fm	Fv	Fv/Fm	Fv/Fo	Fm/Fo
Milyana	120.09	93.70	88.59	94.63	76.42	80.25
Jaklin	110.21	86.75	82.35	94.64	77.28	80.87
Pautalya	112.72	89.63	84.69	92.69	79.42	83.02
221	111.94	93.60	89.66	94.30	82.10	85.24
964750063	111.36	99.25	96.59	96.32	88.46	90.47
1837	109.82	88.58	84.25	96.59	78.61	82.16
1838	116.36	86.00	80.08	93.75	70.41	75.18
1839	110.87	94.23	90.39	95.52	83.77	86.80
1840	115.78	95.08	90.38	95.15	82.63	85.82
1841	106.40	90.21	86.64	95.99	82.44	85.60
1842	118.50	92.51	86.75	92.67	75.28	79.73
1843	115.35	94.49	89.72	94.33	78.80	82.72
1844	106.59	100.10	98.57	97.90	93.49	94.73
1845	108.94	94.24	90.80	95.00	85.55	88.27
1846	115.86	92.40	87.59	95.28	76.75	80.69
1847	107.32	91.73	88.42	96.71	83.57	86.43
1848	102.95	95.13	93.50	98.55	92.03	93.40
1849	108.77	84.75	79.66	94.21	74.49	78.88
1850	105.36	81.91	77.18	94.33	73.31	77.74
1851	105.98	93.15	90.47	96.60	86.55	88.84
1852	100.07	90.94	88.92	98.10	86.79	89.11
1853	112.05	87.50	82.61	94.14	76.08	80.06
1854	115.42	86.69	80.88	93.21	73.11	77.63
1855	103.45	90.79	87.77	97.62	86.11	88.47
1856	109.07	86.56	81.85	94.77	75.73	79.61

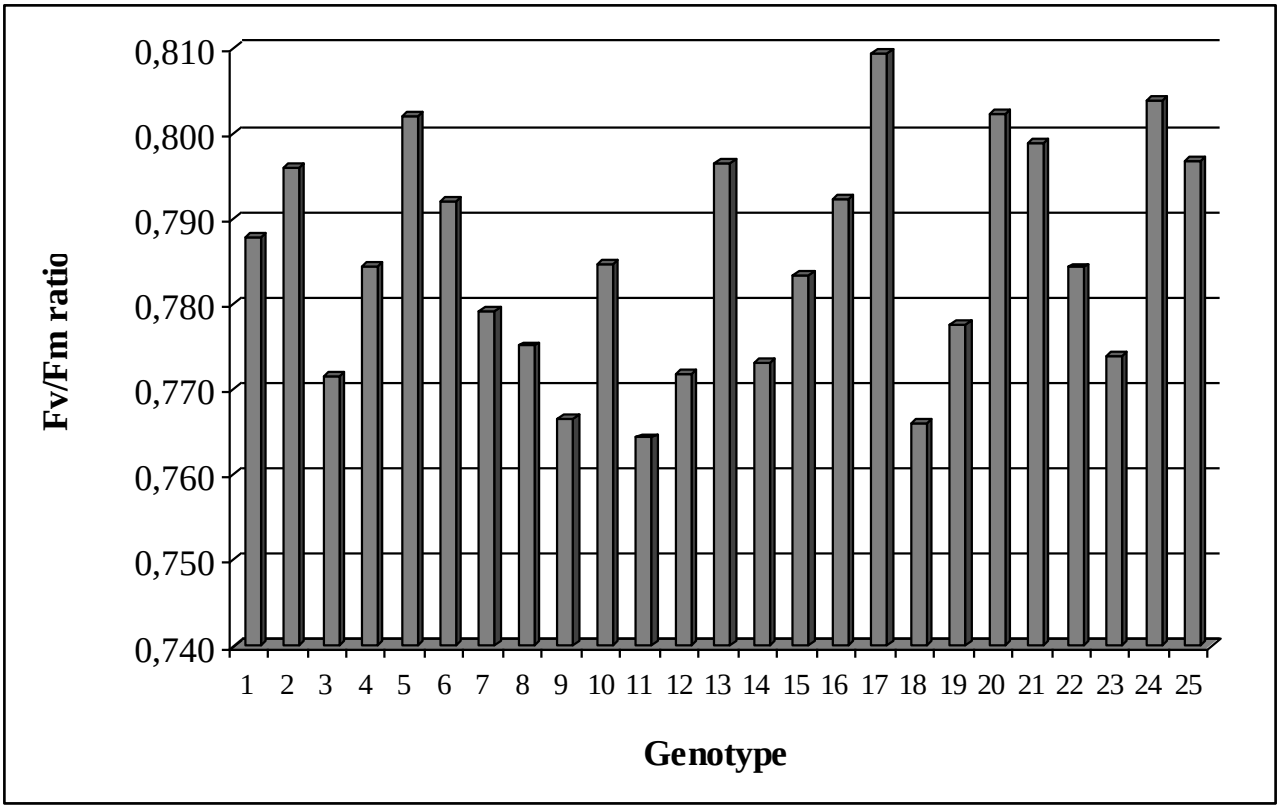

Fig. 1. Values of Fv/Fm at high temperature and drought; 1. Milyana; 2. Jaklin; 3. Pautalya; 4. 221; 5. 964750063; 6. 1837; 7. 1838; 8. 1839; 9. 1840; 10. 1841; 11. 1842; 12. 1843 13. 1844; 14. 1845; 15. 1846; 16. 1847; 17. 1848; 18. 1849; 19. 1850; 20. 1851; 21. 1852; 22. 1853; 23. 1854; 24. 1855; 25. 1856.

Genotypic differences seen for possible carbon monoxide damage might relate to bacterial wilt resistance in tomato.

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Breeding for resistance to bacterial wilt (*Ralstonia solanacearum*) in tomato has been challenging for a number of reasons including variable pathogen strains, environmental influences on disease expression-especially temperature and soil moisture, and linkage of resistance with undesirable characteristics such as small fruit size (Scott et al., 2007). Not only has it been difficult to develop resistant varieties, but the variable response to environmental conditions prevented our development of a reliable seedling screening procedure despite numerous experiments to develop one. In this brief report I present an observation that might be of use to someone studying biochemical growth responses.

Last winter we were growing our tomato seedlings in a plastic greenhouse with roll up sides. The greenhouse has two overhead propane heaters to provide heat which is blown over the seedlings on cool nights. Apparently on some nights the burning of propane produced carbon monoxide rather than carbon dioxide and this resulted in distorted plant growth in some areas of the greenhouse where one would generally see some distorted plants mixed in with normal growing plants within genotypes. Our bacterial wilt resistant material was in a corner of the greenhouse where we had in previous years seen a greater amount of this type of damage. What was seen this time was striking; bacterial wilt resistant lines Hawaii 7997, Fla. 8109 (Scott et al., 2009), and 'Neptune' sustained no damage while our susceptible control 'Florida MH-1' was severely affected (Figures 1,2).

Of course this may have nothing to do with bacterial wilt resistance, but instead could just mean that 'Florida MH-1' is extremely susceptible to CO damage. The MH-1 plants ultimately grew out of the problem so they could be inoculated, planted and ultimately killed by the bacterial wilt pathogen.

The CO effects on tomato may interfere with the electron transport system and oxidative phosphorylation of membranes where cytochrome oxidase is blocked. Cyanide and azide can also block the oxidation process (Salisbury and Ross, 1978). Inbreds such as Hawaii 7997 and Fla. 8109 would appear to have a CO-resistant oxidation pathway whereas 'Florida MH-1' does not. However, I do not know for sure that the damage

seen was due to CO as it was not measured. It just seemed like the most likely explanation given that the time the damage occurred was when the heaters were used and that the plants then grew out of the problem once they were no longer turned on.

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Fig. 1. Tomato seedlings after possible exposure to carbon monoxide; rows to left of the small stake at the bottom of the tray are bacterial wilt resistant Hawaii 7997 showing no symptoms B) 'Florida MH-1' plants to behind and to the right of the stake showing malformed growth. Note that all 'Florida MH-1' plants were affected, when this picture was taken the 5th plant in the second row from the left has grown out of the problem already.

Fig. 2. Tomato seedlings after possible exposure to carbon monoxide; in the left flat are normal looking, bacterial wilt resistant Fla. 8109 plants that were grown adjacent to the flat on the right of 'Florida MH-1' whose plants have distorted growth.

Cytogenetic Characterization of Species Hybrids in the Tomato Clade

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Introduction

The tomato clade consists of twelve species and subspecies (Fig. 1) (Spooner et al. 2005; Moyle 2008). Members of the clade share the same diploid chromosome number ($2n = 2x = 24$), and interspecies hybrids are more or less fertile (our observations).

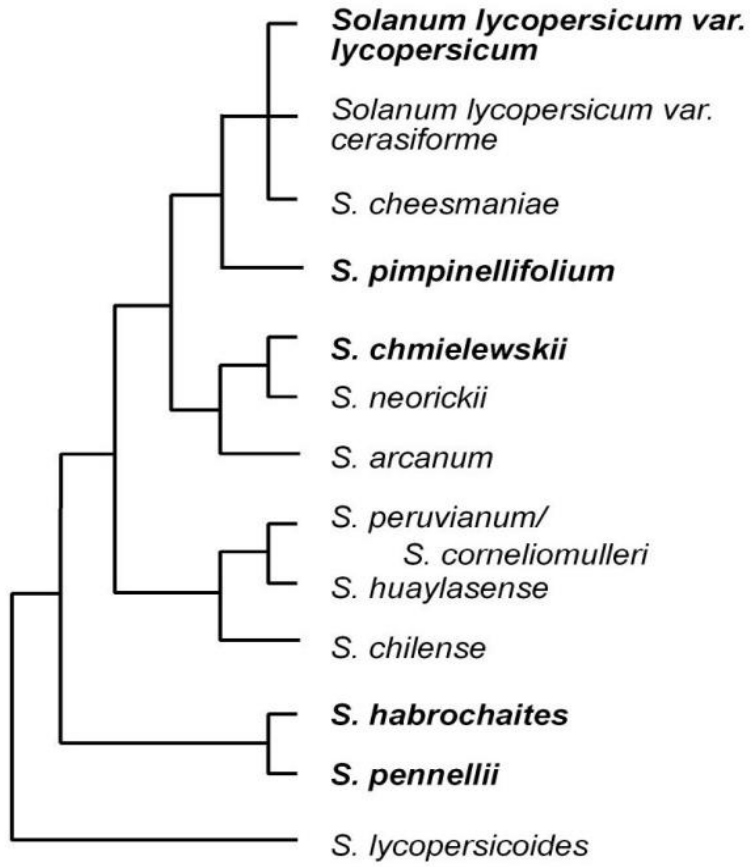

Figure 1. Phylogenetic tree of the tomato clade redrawn from Moyle (2008). Tomato (*S. lycopersicum* var. *lycopersicum*) and the species we have hybridized with tomato are in bold type. *S. lycopersicoides* is included as an out group.

The chromosomes are small, but they are individually identifiable in pachytene chromosome squashes (Brown 1949; Barton 1950). Hybrids between tomato and its close relative *S. pimpinellifolium* have been reported to behave cytologically like

intraspecific tomato hybrids, *i.e.*, there is no evidence of chromosomal differentiation between these species. On the other hand, tomato hybrids with other members of the clade, *e.g.*, *S. cheesemanii, S. peruvianum, and S. habrochaites* show some sterility, segregation distortion, and reduced recombination, suggesting structural differences in chromosomes (Quiros 1991). Here we examine spreads of synaptonemal complexes (SCs) from hybrids between tomato and other members of the tomato clade by electron microscopy and describe various structural rearrangements that have occurred.

Materials and Methods

Using tomato as the female parent, hybrids were made between tomato (*S. lycopersicum*) and *S. pimpinellifolium*, *S. chmielewskii*, *S. habrochaites*, and *S. pennellii*. SC spreads were prepared as described by Chang et al. (2007) and in detail by Stack and Anderson (2009). Briefly, the cell walls were enzymatically removed from primary microsporocytes, and the protoplasts were burst hypotonically and allowed to air dry on a glass microscope slide covered with a thin plastic film. DNA was removed enzymatically from the spreads. SC spreads were fixed with formaldehyde and glutaraldehyde, and the spreads were stained with phosphotungstic acid. The plastic with SC spreads was lifted onto grids, and the SC spreads were examined and photographed in an electron microscope.

Results and Discussion

Changes in chromosome structure have long been recognized as useful taxonomic characters (*e.g.*, Swanson 1957). Observations of the small mitotic metaphase chromosomes in the tomato clade show no differences in ploidy or structure that would be useful in defining the phylogeny of the group. In comparison, long pachytene bivalents are more revealing, but even so, overlapping bivalents and the resolution of the light microscope limit interpreting the details of synapsis in cases of structural heterozygosity.

Here we show that by examining well-spread sets of SCs (= pachytene chromosomes) from hybrids with the superior resolution of electron microscopy, a variety of structural differences between species becomes apparent. The most common and obvious structural irregularity observed in the hybrid SCs is mismatched kinetochores (Fig. 2). The probable basis for mismatched kinetochores is heterozygosity for pericentric inversions with nonhomologous synapsis through these inverted segments. This interpretation is supported by observations of inversion loops in early pachytene (Fig. 3), which are subsequently adjusted to straight nonhomologous synapsis by late pachytene.

Because all tomato chromosomes have pericentric heterochromatin, these inversions may primarily involved heterochromatin. Considering that crossing over is rare in pericentric heterochromatin (Sherman et al. 1995), these inversions may not

have much effect on segregation or genetic linkage maps (except for genes located in pericentric heterochromatin).

Other irregularities observed include fold back synapsis, asynapsis, mismatched ends, and a whole arm translocation (not illustrated, but observed in the *S. chmielewskii* hybrid).

It is interesting that regardless of what synaptic irregularities are observed, generally on each bivalent there is at least one synapsed arm with a late recombination nodule (LN). Since LNs occur at sites of crossing over, chiasmate bivalents will be formed, leading to proper segregation of the homeologues. This indicates that the partial fertility observed for the hybrids is unlikely to be due primarily to errors in meiotic segregation (see Quiros 1991. pp. 131-132).

So far our cytogenetic results generally support the phylogenetic tree (Fig. 1) in that the further species are separated from tomato on the phylogenetic tree, the more numerous and severe the synaptic irregularities observed in the hybrids. For example, only two mismatched kinetochores were observed in the tomato X *S. pimpinellifolium* hybrid, while at least five are visible in the tomato X *S. pennellii* hybrid and in the tomato X *S. habrochaites* hybrid. On the other hand, *S. chmielewskii* is located much closer to tomato on the phylogenetic tree than *S. pennellii* and *S. habrochaites*, but the tomato X *S. chmielewskii* hybrid also has at least five mismatched kinetochores as well as a translocation. We have not yet determined whether the mismatched kinetochores occur on the same five bivalents in the hybrids. Examination of additional hybrids and a quantitative comparison of synaptic irregularities among hybrids should aid in defining relationships within the tomato clade.

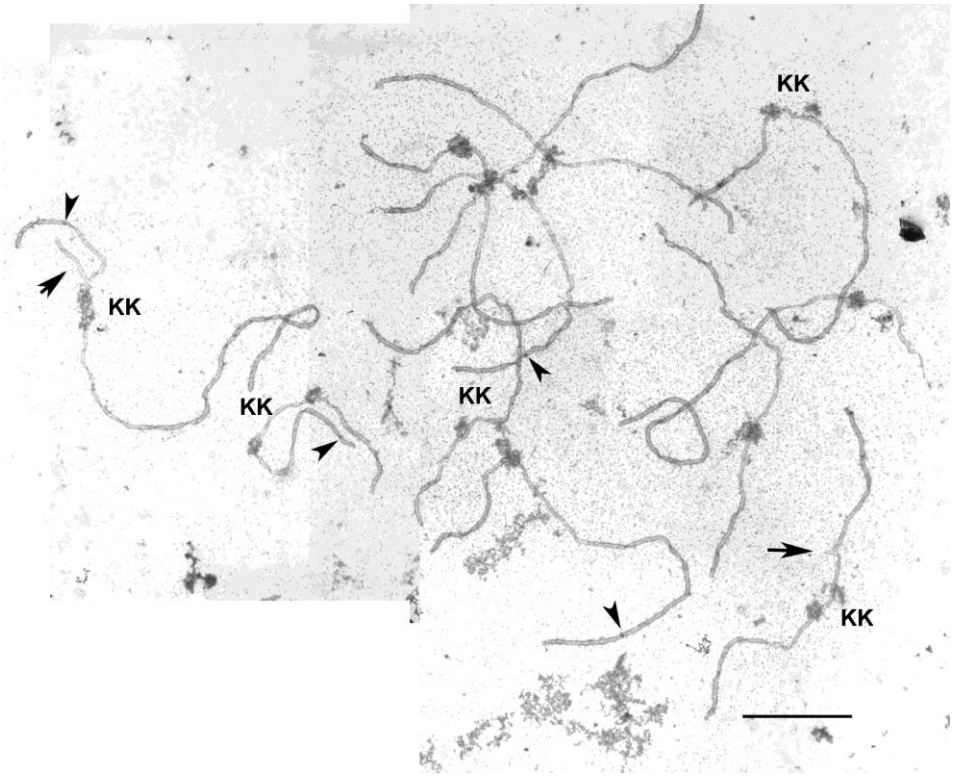

Figure 2. SC spread from a tomato X *S. pennellii* F_1 hybrid. Note mismatched kinetochores on at least five bivalents (KK). Fold-back synapsis is also visible (arrows), as well as RNs on every bivalent (*e.g*., arrowheads). The bar represents 5 µm.

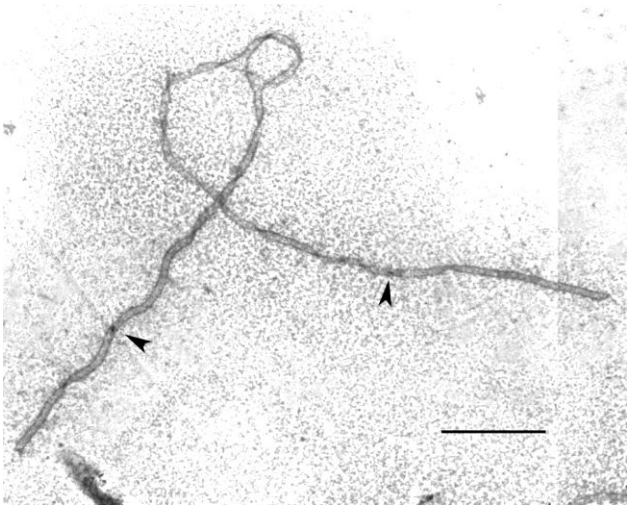

Figure 3. Early pachytene SC from a tomato X *S. pennellii* F_1 hybrid. Note the inversion loop and RNs in both arms (arrows).The bar represents 2 µm.

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Revised List of Miscellaneous Stocks

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This list of approx. 1,560 miscellaneous genetic stocks is a revision of the previous one issued in TGC 56 (2006). Extinct, obsolete, or faulty accessions have been dropped. New stocks include two historical cultivars (Red River, Bryan Self Topper) that were important in the development of determinate processing types. Two other processing types (UC204B, Heinz 1706) used for sequencing and genomics projects, fresh market inbreds or varieties (NC84173, Gold Nugget), and a Spanish landrace with long storage capabilities (B-L-35), were also added. The list also incorporates new double mutant combinations synthesized by E. Kerr or R. Robinson, and a large number of multiple marker stocks developed by A. Kuzemenskiy in the Ukraine. Prebred lines containing morphological traits introgressed from *S. lycopersicoides* by C.M. Rick are listed here for the first time. Other new introgressants include recombinant sub-ILs of *S. pennellii* isolated by C. Jones or C. Tauer and associates.

We attempt to maintain all listed accessions in adequate seed supply for distribution. However, some stocks, such as certain multiple marker combinations, aneuploids, or prebreds, are weak and require special cultural care; consequently, seed supplies may at times be too low to permit distribution. Other accessions may be temporarily unavailable during seed regeneration or for other reasons.

Names and phenotypic classes of individual mutations are given in the last Monogenic Stocks List (see TGC 58). More detailed information on these stocks is available through our website (http://tgrc.ucdavis.edu).

see also:
Wild Species Stocks (1,190 accessions total) are listed in TGC 57 (2007)
Monogenic Stocks (1,023 accessions total) are listed in TGC 58 (2008)

Types of Stocks on this List

1. Cultivars and Landraces
   - 1.1. Modern and Vintage Cultivars
   - 1.2. Latin American Cultivars
2. Prebred Lines
   - 2.1. Introgression Lines
   - 2.2. Backcross Recombinant Inbreds
   - 2.3. Alien Substitution Lines
   - 2.4. Monosomic Alien Addition Lines
   - 2.5. Other Prebred Lines
   - 2.6. Interspecific Hybrids
3. Stress Tolerant Stocks
4. Cytogenetic Stocks
   - 4.1. Translocations
   - 4.2. Trisomics
   - 4.3. Autotetraploids
5. Cytoplasmic Variants
6. Genetic Marker Combinations
   - 6.1. Chromosome Marker Stocks
   - 6.2. Linkage Screening Testers
   - 6.3. Miscellaneous Marker Combinations
7. Provisional mutants

1. CULTIVARS AND LANDRACES

1.1. Modern and Vintage Cultivars (211)

We maintain the following set of cultivars, inbreds, and breeding lines for various purposes, mainly as isogenic (or nearly isogenic) stocks for specific mutants, standards for genetic comparison, sources of disease resistances, or other purposes. Marglobe is considered the standard for tomato gene (mutant) nomenclature. Most lines have been maintained by selfing for many generations.

Accession	Cultivar
LA0818	A-1
LA0516	Ace
LA2838A	Ailsa Craig
LA2529	Alcobaca
LA2463	Allround
LA4403	Amiko
LA1995	Angela
LA3244	Antimold-B
LA3527	Apex 1000
LA4406	Asgerocu
LA4402	Atlasiyj
LA0657	Beaverlodge
LA2973	Big Rainbow
LA2972	Big Yellow Red Ctr.
LA4347	B-L-35
LA1499	Break O'Day
LA4346	Bryan Self-Topper
LA3341	C5
LA0198	Cal 255
LA2414	Cal Ace
LA1439	Calmart
LA3316	Campbell 24
LA3317	Campbell 28
LA3228	Canary Export
LA2374	Caro Red
LA2400	Castlemart
LA3121	Chico Grande
LA4407	Cit
LA4285	CLN2264F
LA4286	CLN2264G
LA3213	Columbian
LA0533	Condine Red
LA0817	CP-2
LA3247	Craigella
LA1162	Cuba Plum
LA1219	Dwarf San Marzano
LA0313	Dwarf Stone
LA3245	E.S.1
LA4024	E-6203
LA3238	Earliana
LA2006	Earlinorth
LA3010	Earlipak

Accession	Cultivar
LA0266	Earlipak
LA0517	Early Santa Clara
LA2711	Edkawi
LA3800	Fargo Self-pruning
LA4415	Farshirovochniyj
LA3801	Farthest North
LA3024	Fireball
LA3840	FLA 7060
LA4404	Flora
LA3242	Flora-Dade
LA4026	Florida 7481
LA4025	Florida 7547
LA3030	Gardener
LA2969	Georgia Streak
LA2802	Globonnie
LA4355	Gold Nugget
LA4011	GT
LA3231	Gulf State Market
LA0314	Hardin Miniature
LA3202	Hawaii 7997
LA3856	Hawaii 7998
LA4345	Heinz 1706-BG
LA0806	High Crimson
LA3237	Homestead 24
LA3320	Hotset
LA3144	Hunt 100
LA2805	Indehiscent Currant
LA3201	IRB 301
LA4408	Irska
LA4409	Iskorka
LA1089	John Baer
LA1131	Kallio's Alaskan Dwarf
LA0025	King Humbert #1
LA3240	Kokomo
LA3526	L04012
LA4410	Lagidnyj
LA4405	Lajka
LA0505	Laketa
LA3203	Large Plum
LA3118	Laurica
LA0791	Long John
LA0534	Lukullus

Accession	Cultivar
LA3475	M-82
LA3120	Malintka 101
LA3007	Manapal
LA0502	Marglobe
LA1504	Marmande
LA0278	Marzano Grande
LA3151	Mecline
LA0011	Michigan State Forcing
LA3911	Micro-Tom
LA2825	Mobaci
LA2824	Moboglan
LA3152	Moboline
LA2821	Mobox
LA2830	Mocimor
LA3471	Mogeor
LA2828	Momor
LA2829	Momor Verte
LA2818	Monalbo
LA2706	Moneymaker
LA2819	Monita
LA2713	Montfavet 167
LA2714	Montfavet 168
LA2827	Moperou
LA2822	Mossol
LA2820	Motabo
LA2826	Motaci
LA2823	Motelle
LA3472	Movione
LA2661	Nagcarlang
LA4354	NC 84173
LA3845	NC EBR-5
LA3846	NC EBR-6
LA3847	NC HS-1
LA3625	NC265-1 (93)-3-3
LA3802	New Hampshire Victor
LA2009	New Yorker
LA3321	Ohio 7663
LA1088	Ohio Globe A
LA2447	Ontario 717
LA2449	Ontario 7517
LA2396	Ontario 7710
LA2448	Ontario 7818
LA2970	Orange, Red Ctr.
LA0012	Pearson
LA0020	Pennheart
LA3528	Peto 95-43
LA3243	Platense
LA3125	Pomodorini Napolitan
LA2715	Porphyre
LA3820	Potentate

Accession	Cultivar
LA3903	Primabel
LA0089	Prince Borghese
LA3233	Pritchard
LA3229	Prospero
LA2446	Purdue 135
LA2377	Purple Calabash
LA2378	Purple Smudge
LA0337	Red Cherry
LA4350	Red River
LA0276	Red Top VF
LA3129	Rehovot 13
LA2356	Rey de Los Tempranos
LA0535	Rheinlands Ruhm
LA3343	Rio Grande
LA3145	Rockingham
LA0503	Roumanian Sweet
LA3214	Rowpac
LA2088	Royal Red Cherry
LA3215	Roza
LA1090	Rutgers
LA2662	Saladette
LA3216	Saladmaster
2-297	San Marzano
LA3008	San Marzano
LA0180	San Marzano
LA1021	Santa Cruz B (Gigante)
LA2413	Severianin
LA2912	Short Red Cherry
LA3234	Sioux
LA3221	Slender Pear
LA3632	Start 24
LA0030	Stemless Pennorange
LA2443	Stirling Castle
LA1091	Stokesdale
LA1506	Stone
LA4432	Sunseeds 1642
LA0164	Sutton's Best of All
LA2399	T-5
LA2590	T-9
LA0154	Tiny Tim
LA1714	UC-134
LA4437	UC-204B
LA3130	UC-204C
LA1706	UC-82
LA2937	UC-MR20
LA2938	UC-N28
LA2939	UC-T338
LA2940	UC-TR44
LA2941	UC-TR51
LA0021	Uniform Globe

Accession	Cultivar
LA2445	V-121
LA0745	V-9 Red Top
LA3246	Vagabond
LA3905	Vantage
LA3122	Vendor
LA2968	Vendor (Tm-2a)
LA2971	Verna Orange
LA2444	Vetomold K10
LA0744	VF-11
LA1023	VF-13L
LA1507	VF-145 21-4
LA0816	VF-145 22-8
LA1222	VF145 78-79
LA0742	VF-34

Accession	Cultivar
LA0490	VF-36
LA0743	VF-6
LA2086	VFN Hi Sugar
LA0815	VFN-14
LA1022	VFN-8
LA1221	VFNT Cherry
LA3630	Vrbikanske nizke
LA3465	Walter
LA0279	Webb Special
LA2464A	White Beauty
LA2804	Yellow Currant
LA2357	Yellow Peach
LA3148	Zemer Kau

1.2. Latin American Cultivars (225)

This collection of Latin-American cultivars has been assembled from various sources but principally from our collecting trips, often at local markets. With a few exceptions they are indigenous in the sense that they are not recently introduced lines. Many of them are extinct in the source region, having been replaced by modern cultivars.

Country	LA	Collection Site
Bolivia	LA0172	Santa Cruz
Bolivia	LA2699	Coroica
Bolivia	LA2871	Chamaca
Bolivia	LA2873	Lote Pablo Luna #2
Bolivia	LA2874	Playa Ancha
Chile	LA0466	Hacienda Rosario
Chile	LA0467	Lluta Valley
Chile	LA0468	Iquique
Colombia	LA0356	Buenaventura
Colombia	LA0357	Buenaventura
Colombia	LA0358	Buenaventura
Colombia	LA1539	Cali to Popayan
Costa Rica	LA1215	
Costa Rica	LA3453A	Turrialba
Costa Rica	LA3453B	Turrialba
Costa Rica	LA3453C	Turrialba
Costa Rica	LA3453D	Turrialba
Ecuador	LA0126	Quito
Ecuador	LA0292	Santa Cruz
Ecuador	LA0408	Guayaquil
Ecuador	LA0409	Guayaquil
Ecuador	LA0410	Guayaquil
Ecuador	LA0415	Daular
Ecuador	LA0416	Puna
Ecuador	LA0423	Wreck Bay: Cristobal
Ecuador	LA1224	Puyo
Ecuador	LA1238	Viche

Country	LA	Collection Site
Ecuador	LA1239	Esmeraldas
Ecuador	LA1240	Esmeraldas
Ecuador	LA1241	Esmeraldas
Ecuador	LA1244	Coop Carmela, Los Sapos
Ecuador	LA1249	Loja
Ecuador	LA1250	Loja
Ecuador	LA1251	Loja
Ecuador	LA2094	El Naranjo
Ecuador	LA2132	Chuchumbetza
Ecuador	LA2381	Malacatos
Ecuador	LA2382	Malacatos
Ecuador	LA2383	Malacatos
Ecuador	LA2384	Malacatos
Ecuador	LA3126	Malacatos
Ecuador	LA3624	Santa Rosa
El Salvador	LA1210	San Salvador
El Salvador	LA1211	San Salvador
Guatemala	LA1460	Antigua
Honduras	LA0147	Tegucigalpa
Honduras	LA0148	Tegucigalpa
Mexico	LA0146	Mexico City
Mexico	LA1218	Vera Cruz
Mexico	LA1459	Huachinango
Mexico	LA1462	Merida
Mexico	LA1544	Xol Laguna
Mexico	LA1564	Culiacan
Mexico	LA1565	Val. nacionale

Country	LA	Collection Site
Mexico	LA1566	Val. nacionale
Mexico	LA1567	Sinaloa
Mexico	LA1568	Yucatan
Mexico	LA1702	Sinaloa
Mexico	LA1703	Rio Tamesi
Mexico	LA1704	Rio Tamesi
Mexico	LA1994	Tamaulipas
Mexico	LA2083	Guaco, Culiacan
Mexico	LA2084	Comala, Culiacan
Nicaragua	LA1212	
Nicaragua	LA1213	
Panama	LA1216	
Panama	LA1217	
Panama	LA1570	Cerro Azul
Peru	LA0113	Hacienda Calera
Peru	LA0116	Chiclayo
Peru	LA0117	Piura
Peru	LA0125D	Trujillo
Peru	LA0131H	Arequipa
Peru	LA0134C	Ayacucho
Peru	LA0393-LA0396	Chiclayo
Peru	LA0401-LA0405	Piura
Peru	LA0457	Tacna
Peru	LA0472	Tacna
Peru	LA0473	Calana
Peru	LA0477	Chincha
Peru	LA0478	Chincha
Peru	LA0721	Chiclayo
Peru	LA1313	Convento de Sivia, Pichari
Peru	LA1315	Ayna, San Francisco
Peru	LA1390	La Molina
Peru	LA1397	Iquitos
Peru	LA1398	Iquitos
Peru	LA1650	Fundo Bogotalla
Peru	LA1655	Tarapoto
Peru	LA1669	Jahuay
Peru	LA1698	Kradolfer Chacra
Peru	LA1701	Trujillo
Peru	LA1976A	Calana
Peru	LA1976B	Calana
Peru	LA1976C	Calana
Peru	LA1988	Iquitos
Peru	LA2207-LA2212	Bajo Naranjillo
Peru	LA2213-LA2220	Nueva Cajamarca

Country	LA	Collection Site
Peru	LA2221-LA2235	Moyobamba
Peru	LA2237	La Habana
Peru	LA2238	La Habana
Peru	LA2239	La Habana
Peru	LA2240	La Habana
Peru	LA2241	La Habana
Peru	LA2242	La Habana
Peru	LA2243	La Habana
Peru	LA2244	La Habana
Peru	LA2245-LA2253	Soritor
Peru	LA2254-LA2256	Puerto Moyobamba
Peru	LA2257	Hotel Abricias, Moyobamba
Peru	LA2258	Fundo Conovista
Peru	LA2259A-2259D	Moyobamba
Peru	LA2260-LA2264	La Huarpia
Peru	LA2265-LA2268	Casaria de Pacaisapa
Peru	LA2269-LA2276	Km 57 from Tarapoto
Peru	LA2278-LA2282	Tabalosas
Peru	LA2283-LA2307	Tarapoto
Peru	LA2309-LA2311	Punto Santa Cruz
Peru	LA2316	Sargento
Peru	LA2622	Mangual Pucallpa
Peru	LA2623	Pucalepillo Pucallpa
Peru	LA2676	San Juan del Oro, Basura
Peru	LA2841	Chinuna
Peru	LA2842	Santa Rita
Peru	LA2843	Moyobamba
Peru	LA2844	Shanhao
Peru	LA2845	Moyobamba
Peru	LA3222-LA3326	San Isidro
Peru	LA3646	Puente Tincoj
Sri Lanka	LA2703	Kandy #2

2. PREBRED STOCKS

2.1. Introgression Lines (ILs)

2.1.1. *S. pennellii* ILs (89)

The following group of introgression lines (ILs) was developed by Y. Eshed and D. Zamir (Eshed 1994 Euphytica 79:175; Liu 1999 TGC 49:26). Each IL (except IL 8-1) is homozygous for a single introgression from *S. pennellii* (LA0716) in the background of cv. M-82 (LA3475). The entire *pennellii* genome is thereby represented by 50 lines with overlapping introgressions. Recombinant sublines provide increased mapping resolution in some regions (the IL 5-4 sublines are described in Jones 2007 Amer. J. Bot. 94: 935, and . The IL # indicates the *pennellii* chromosome and introgressed segment number in each.

Accession	Line
LA4028	IL 1-1
LA4029	IL 1-1-2
LA4030	IL 1-1-3
LA4031	IL 1-2
LA4032	IL 1-3
LA4033	IL 1-4
LA4034	IL 1-4-18
LA4035	IL 2-1
LA3480	IL 2-1
LA4036	IL 2-1-1
LA4037	IL 2-2
LA4038	IL 2-3
LA4039	IL 2-4
LA4040	IL 2-5
LA4041	IL 2-6
LA4042	IL 2-6-5
LA4043	IL 3-1
LA4044	IL 3-2
LA3488	IL 3-3
LA4046	IL 3-4
LA4047	IL 3-5
LA4048	IL 4-1
LA4049	IL 4-1-1
LA4050	IL 4-2
LA3492	IL 4-2
LA4051	IL 4-3
LA4052	IL 4-3-2
LA4053	IL 4-4
LA4054	IL 5-1
LA4055	IL 5-2
LA4056	IL 5-3

Accession	Line
LA4057	IL 5-4
LA4434	IL 5-4-1
LA4435	IL 5-4-2
LA4436	IL 5-4-4
LA4439	IL 5-4-5-137
LA4429	IL 5-4-5-44
LA4430	IL 5-4-5-49
LA4438	IL 5-4-8
LA4058	IL 5-5
LA4059	IL 6-1
LA4060	IL 6-2
LA4061	IL 6-2-2
LA3502	IL 6-3
LA4062	IL 6-3
LA4063	IL 6-4
LA4064	IL 7-1
LA4065	IL 7-2
LA4066	IL 7-3
LA4067	IL 7-4
LA4068	IL 7-4-1
LA4069	IL 7-5
LA4070	IL 7-5-5
LA4071	IL 8-1
LA4072	IL 8-1-1
LA4073	IL 8-1-3
LA4074	IL 8-2
LA4075	IL 8-2-1
LA4076	IL 8-3
LA4077	IL 8-3-1
LA4078	IL 9-1
LA4079	IL 9-1-2

Accession	Line
LA4080	IL 9-1-3
LA4081	IL 9-2
LA4082	IL 9-2-5
LA4083	IL 9-2-6
LA4084	IL 9-3
LA4085	IL 9-3-1
LA4086	IL 9-3-2
LA4087	IL 10-1
LA4088	IL 10-1-1
LA4089	IL 10-2
LA3516	IL 10-2
LA4090	IL 10-2-2
LA4091	IL 10-3
LA3517	IL 10-3
LA4092	IL 11-1
LA4093	IL 11-2
LA4094	IL 11-3
LA4095	IL 11-4
LA4096	IL 11-4-1
LA4097	IL 12-1
LA4098	IL 12-1-1
LA4099	IL 12-2
LA3524	IL 12-3
LA4100	IL 12-3
LA4101	IL 12-3-1
LA4102	IL 12-4
LA4103	IL 12-4-1

2.1.2. *S. habrochaites* ILs (93)

The following group of introgression lines represent the genome of *S. habrochaites* (*L. hirsutum*) LA1777 in the background of cv. E-6203 (LA4024) via homozygous chromosome segments (Monforte 2000 Genome 43:803). The first 57 lines (LA3913 - LA3969) represent approximately 85% of the donor genome, while the remaining lines (LA3970 - LA4010) contain different introgressions, mostly derivatives of the first group. Unlike the *pennellii* ILs above, each *habrochaites* IL may contain more than one introgression, representing one to several chromosomes, as indicated below.

LA	Line	Chrom.
LA3913	TA1258	1
LA3914	TA523	1
LA3915	TA1229	1
LA3916	TA1223	1
LA3917	TA1535	1
LA3918	TA1127	1
LA3919	TA1128	1
LA3920	TA1536	1
LA3921	TA1105	2
LA3922	TA1266	2
LA3923	TA1537	2
LA3924	TA1538	2
LA3925	TA1111	3
LA3926	TA1276	3
LA3927	TA1277	3
LA3928	TA1540	3
LA3929	TA1541	3
LA3930	TA1133	4
LA3931	TA1280	4
LA3932	TA1562	4
LA3933	TA1542	4
LA3934	TA1459	4
LA3935	TA517	4
LA3936	TA1475	4
LA3937	TA1473	4
LA3938	TA1287	5
LA3939	TA1293	5
LA3940	TA1112	5
LA3941	TA1543	5
LA3942	TA1117	5
LA3943	TA1544	5
LA3944	TA1539	6
LA3945	TA1545	6
LA3946	TA1546	6
LA3947	TA1559	6
LA3948	TA1303	7
LA3949	TA1304	7
LA3950	TA1547	7
LA3951	TA1312	7
LA3952	TA1315	8
LA3953	TA1316	8
LA3954	TA1548	8
LA3955	TA1320	8
LA3956	TA1324	9
LA3957	TA1325	9
LA3958	TA1330	9
LA3959	TA1331	9
LA3960	TA1550	10
LA3961	TA1551	10
LA3962	TA1552	10
LA3963	TA1337	10
LA3964	TA1339	10
LA3965	TA1555	11
LA3966	TA1554	11
LA3967	TA1342	11
LA3968	TA1350	12
LA3969	TA1121	12
LA3970	TA1219	1
LA3971	TA1218	2
LA3972	TA1173	2
LA3975	TA1629	3
LA3976	TA1138	4
LA3977	TA1467	4
LA3978	TA1468	4
LA3979	TA1630	4
LA3980	TA1290	5
LA3981	TA1116	5
LA3983	TA1631	5
LA3984	TA1632	5
LA3985	TA1306	7
LA3986	TA1309	7
LA3988	TA1318	8
LA3989	TA1319	8
LA3990	TA1560	8
LA3991	TA1326	9
LA3993	TA1549	10
LA3994	TA1635	10
LA3995	TA1553	11
LA3996	TA1120	11
LA3997	TA1563	1, 10
LA3998	TA1637	1, 11, 12
LA3999	TA1638	1, 12
LA4000	TA1557	1, 4
LA4001	TA1644	1, 7, 12
LA4002	TA1645	1, 8, 12
LA4003	TA1648	2, 11
LA4004	TA1649	2, 3, 6
LA4005	TA1652	3, 5
LA4006	TA1654	4, 10, 11
LA4007	TA1655	4, 12
LA4008	TA1656	5, 6, 9
LA4009	TA1564	5, 7, 10
LA4010	TA1561	8, 12

2.1.3. *S. lycopersicoides* ILs (101)

The following group of ILs have been bred from *S. lycopersicoides* into the background of cv. VF36. These lines represent ~96% of the donor genome and are described in Canady 2005 Genome 48: 685, and Rick 1988 Theor. Appl. Genet. 76: 647. While some lines are available in the homozygous condition, others are partially or completely sterile as homozygotes, thus are maintained via heterozygotes. In this case, marker analysis is required to identify the desired genotypes in segregating progenies. Seed of some lines may be limited or temporarily unavailable.

LA	Line	Chr.
LA3866	LS1-1	1
LA3867	LS11-9	1
LA4230	LS15-2H	1
LA4231	LS15-2B	1
LA4232	LS11-11A	1
LA4233	LS20-9	1
LA4234	LS21-2	1
LA4235	LS10-2	1
LA4293	LS5-8	1
LA4294	LS15-2AD	1
LA4295	LS15-2A	1
LA4296	LS15-2AA	1
LA4297	LS15-2AAA	1
LA4298	LS15-2BA	1
LA3869	LS42-4	2
LA3870	LS38-10	2
LA3871	LS41-3	2
LA4236	LS49-8A	2
LA4237	LS40-8	2
LA4238	LS5-1	2
LA4239	LS41-20	2
LA4420	C2S	2
LA3882	LS43-14	2, 6
LA3344	Mdh-1	3
LA3874	LS20-9	3
LA4240	LS1-13	3
LA4241	LS40-2	3
LA4242	LS14-8	3
LA4243	LS1-3	3
LA4244	LS10-9	4
LA4245	LS10-11A	4
LA4246	LS49-8B	4
LA4247	LS12-9	4
LA4314	LS12-9B	4, 10
LA3875	LS24-14	4, 12
LA3878	LS24-6	5

LA	Line	Chr.
LA4248	LS11-6	5
LA4249	LS9-1	5
LA4250	LS49-8C	5
LA4251	LS49-3	5
LA4252	LS32-11	5
LA4299	LS4-9	5
LA4426	ILX	5
LA3879	LS1-5	5, 11
LA3893	LS16-6	5, 12
LA4300	LS9-7B	5, 6
LA4253	LS11-11B	6
LA4254	LS32-14	6
LA4255	LS38-5	6
LA4256	LS9-22	6
LA4421	Lac	6
LA3886	LS48-5	7
LA4257	LS46-3	7
LA4258	LS19-7	7
LA4259	LS32-4	7
LA4260	SL-7F	7
LA4261	LS8-11	7
LA4301	SL-7A	7
LA4302	SL-7C	7
LA4303	SL-7D	7
LA4304	LS8-11A	7
LA3883	LS48-6	7, 11
LA4305	LS9-26C	7, 8
LA3876	LS29-1	8
LA3889	LS41-13	8
LA3906	Wa, Dl	8
LA4262	LS20-16	8
LA4263	LS46-6A	8
LA4264	LS9-26A	8
LA4265	LS9-26B	8
LA4266	SL-8A	8
LA4267	LS16-10	8

LA	Line	Chr.
LA4306	LS46-6	8
LA4307	SL-8	8
LA3345	Dia-3	9
LA4268	LS14-7	9
LA4269	LS12-2	9
LA4270	LS10-6	9
LA4271	LS49-5	9
LA4272	LS41-11	9
LA4308	LS32-10	9
LA4309	LS10-6D	9
LA4273	LS12-8	10
LA4274	LS4-14	10
LA4275	SL-10	10
LA4276	LS12-12	10
LA4425	Abg	10
LA3892	LS48-2	11
LA4277	LS24-11	11
LA4278	LS3-2	11
LA4279	LS19-11	11
LA4310	LS19-10A	11
LA4422	PROS	11
LA4280	LS1-5	11, 5
LA4281	LS13-13	12
LA4282	LS45-7	12
LA4283	LS8-9	12
LA4284	LS9-13	12
LA4311	LS14-2	12
LA4312	LS45-7C	12
LA4313	LS8-12A	12
LA4427	C12S	12

2.2. Backcross Recombinant Inbreds (90).

The following group of backcross recombinant inbred lines originated from the cross *S. lycopersicum* E6203 × *S. pimpinellifolium* LA1589 (Doganlar 2002 Genome 45: 1189). The result of 2 BC's and at least 6 generations of inbreeding via single seed descent, the lines are highly homozygous (residual heterozygosity ~3%). The population has been genotyped at 127 marker loci, and the corresponding maps, map files, and QTL data are available from the Solanaceae Genome Network (www.sgn.cornell.edu). This set of 90 lines has been selected for optimum mapping resolution using the MapPop software, and provide a permanent, high resolution mapping population.

LA	TA
LA4139	TA2874
LA4140	TA2875
LA4141	TA2876
LA4142	TA2877, TA2149
LA4143	TA2878
LA4144	TA2879
LA4145	TA2880
LA4146	TA2881
LA4147	TA2882
LA4148	TA2883
LA4149	TA2884
LA4150	TA2885
LA4151	TA2886
LA4152	TA2887
LA4153	TA2888
LA4154	TA2890
LA4155	TA2891
LA4156	TA2892
LA4157	TA2893
LA4158	TA2894
LA4159	TA2895
LA4160	TA2896
LA4161	TA2897

LA	TA
LA4162	TA2898
LA4163	TA2899
LA4164	TA2900
LA4165	TA2901
LA4166	TA2902
LA4167	TA2903
LA4168	TA2904
LA4169	TA2905
LA4170	TA2906
LA4171	TA2907
LA4172	TA2908
LA4173	TA2909
LA4174	TA2910
LA4175	TA2911
LA4176	TA2912
LA4177	TA2914
LA4178	TA2915
LA4179	TA2916
LA4180	TA2917
LA4181	TA2918
LA4182	TA2919
LA4183	TA2920
LA4184	TA2922
LA4185	TA2923

LA	TA
LA4186	TA2924
LA4187	TA2925
LA4188	TA2926
LA4189	TA2927
LA4190	TA2928
LA4191	TA2929
LA4192	TA2930
LA4193	TA2931
LA4194	TA2932
LA4195	TA2933
LA4196	TA2934
LA4197	TA2935
LA4198	TA2936
LA4199	TA2937
LA4200	TA2938
LA4201	TA2939
LA4202	TA2940
LA4203	TA2941
LA4204	TA2942
LA4205	TA2943
LA4206	TA2944
LA4207	TA2945
LA4208	TA2946
LA4210	TA2948

LA	TA
LA4211	TA2949
LA4212	TA2950
LA4213	TA2951
LA4214	TA2952
LA4215	TA2953
LA4216	TA2954
LA4217	TA2955
LA4218	TA2956
LA4219	TA2957
LA4220	TA2958
LA4221	TA2959
LA4222	TA2960
LA4223	TA2961
LA4224	TA2962
LA4225	TA2963
LA4226	TA2964
LA4227	TA2965
LA4228	TA2966
LA4229	TA2967

2.3. Alien Substitution Lines (7)

In the course of his study of segregation and recombination in *S. lycopersicum* x *S. pennellii* hybrids, Rick (Genetics 26:753-768, 1969; Biol. Zbl. 91:209-220, 1971) progressively backcrossed certain chromosomes of *S. pennellii* LA0716 into the background of several chromosome marker stocks in cultivated tomato. Selected heterozygotes of later generations were selfed and subsequent progenies containing the wild type alleles at the marker loci were selected. The chromosome 6 substitution (LA3142) was further selected with RFLP markers to eliminate residual heterozygosity (Weide 1993 Genetics 135:1175). The mutant loci used to select each substitution are indicated.

LA	Chrom.	Marker Loci
2091	1	*au, dgt, inv, scf*
1639	2	*Me, aw, m, d*
1640	3	*sy, bls, sf*
3469	4	*clau, ful, ra, e, su*[3]

LA	Chrom.	Marker Loci
3142	6	*yv, ndw, m-2, c*
1642	8	*l, bu, dl, al*
1643	11	*j, hl, a*

2.4. Monosomic Alien Addition Lines (10)

In the following group of monosomic additions (MA), each line contains a single extra chromosome from *S. lycopersicoides* LA1964 added to the genome of cultivated tomato (Chetelat 1998 Genome 41:40). The integrity of the *S. lycopersicoides* chromosomes in these stocks has been tested with a limited number of markers, hence some may be recombinant. For example, our stock of MA-8 lacks *S. lycopersicoides* markers distal to TG330 on the long arm. Furthermore, we were unable to maintain MA-1 and MA-6, both of which are now extinct.

Like other types of trisomics, progeny of the monosomic additions include both diploids and trisomics, the proportion of which varies between each chromosome group. Identification of monosomic additions in each generation is facilitated by their phenotypic resemblance to the corresponding primary trisomic. Therefore, the guidelines of Rick (TGC 37:60-61, 1987) for identifying trisomics in the seedling stage are useful for selecting monosomic additions as well. To further simplify this process, we have backcrossed some of the monosomic additions into the background of multiple marker stocks for the corresponding chromosomes. In this configuration, diploids are more easily distinguished from trisomics by the expression of recessive mutant alleles in the former, and dominant wild type in the latter. For example, in our stock of MA-2, the 2n progeny would have the phenotype *wv-aa-d*, whereas the 2n+1 plants would be wild type at these marker loci (as well showing the expected trisomic syndrome). In addition, some monosomic additions carry dominant morphological markers that can be used to distinguish them from 2n progeny. The marker genotypes of 2n+1 vs 2n progeny are listed below for each chromosome.

LA	Chrom.	2n+1	2n
3454	MA-2	+-+-+	*wv-aa-d*
3455	MA-3	+-+-+	*sy-bls-sf*
3456	MA-4	+	+
3457	MA-5	+	*obv*
3459	MA-7	*Bco*-+-+	+-*var-not*

LA	Chrom.	2n+1	2n
3460	MA-8	*Wa*	+
3461	MA-9	+	+
3462	MA-10	*Abg*-+-+-+-+	+-*u-t-nd-ag*
3463	MA-11	+	+
3464	MA-12	+	+

2.5. Other Prebreds (21). This group of prebreds contain selected morphological traits bred into cultivated tomato from related wild species. Some traits may be simply inherited, others likely involve multiple genetic loci.

Accession	Traits
LA0214	Dark anthers from S. peruvianum
LA1015	Compressed fruits from S. cheesmaniae
LA1016	Yellow green from S. cheesmaniae
LA1017	Pachypericarp from S. cheesmaniae
LA1018	Odorless from S. cheesmaniae
LA1019	Pachypericarp from S. cheesmaniae
LA1500	High solids, intense pigment from S. chmielewskii
LA1501	High solids from S. chmielewskii
LA1502	High solids from S. chmielewskii
LA1503	High solids from S. chmielewskii
LA1563	High solids from S. chmielewskii
LA1996	Anthocyanin fruit from S. chilense
LA2380	Exserted stigma from S. pimpinellifolium
LA3855	High 2-tridecanone from S. habrochaites
LA3897	High beta-carotene from S. galapagense
LA3898	High beta-carotene from S. galapagense
LA3899	High beta-carotene from S. galapagense
LA4104	High fruit sucrose from S. chmielewskii
LA4136	Regeneration ability from S. peruvianum
LA4424	Poodle syndrome from S. lycopersicoides
LA4428	Virescent leaves from S. lycopersicoides

2.6. Interspecific hybrids.

LA3857 F_1 cv. VF36 × *S. lycopersicoides* LA2951

A relatively male-fertile F_1 hybrid that is clonally propagated in vitro.

LA4135 F_1 cv. VF36 × *S. pennellii* LA0716

This hybrid is used as a rootstock for maintenance of *S. sitiens, S. juglandifolium,* and *S. ochranthum*.

3. STRESS TOLERANT STOCKS (50+)

We receive many requests for stocks with tolerances to environmental stresses (abiotic or biotic). Therefore, we chose this group of mostly wild species accessions based on our observations of plants in their native habitats and/or reports in the literature. If TGC members know of other accessions which should be added to this group, we would be grateful for the information and seed samples to accession in the TGRC.

Stress Tolerance	Species	Accessions
Drought	*S. pimpinellifolium*	LA1578, and others
Drought	*S. pennellii* (general feature)	LA0716, and others
Drought	*S. chilense* (general feature)	LA1958, LA1959, LA1972, and others
Drought	*S. sitiens* (general feature)	LA1974, LA2876, LA4105, etc.
Flooding	*S. lycopersicum* var. *cerasiforme* (wet tropics)	LA1421, and others
Flooding	*S. juglandifolium*, *S. ochranthum* (general feature)	LA2120, LA2682
High temperatures	*S. lycopersicum* cv.s	Nagcarlang LA2661 Saladette LA2662 Malintka-101 LA3120 Hotset LA3320
Low temperatures	*S. habrochaites*	LA1363, LA1393, LA1777, LA1778
Low temperatures	*S. chilense*	LA1969, LA1971, LA4117A
Low temperatures	*S. lycopersicoides*	LA1964, LA2408, LA2781
Aluminum toxicity	*S. lycopersicum* var. *cerasiforme*	LA2710 (suspected)
Salinity / alkalinity	*S. chilense*	LA1930, LA1932, LA1958, LA2747, LA2748, LA2880, LA2931
Salinity / alkalinity	*S. cheesmaniae* (from littoral habitats)	LA1401, LA1508, LA3124, LA3909
Salinity / alkalinity	*S. lycopersicum* cv.	Edkawi LA2711
Salinity / alkalinity	*S. lycopersicum* var. *cerasiforme*	LA2081, LA1310, LA2079, LA4133
Salinity / alkalinity	*S. pennellii*	LA0716, LA1809, LA1926, LA1940, LA2656
Salinity / alkalinity	*S. peruvianum*	LA0462, LA1278, LA2744
Salinity / alkalinity	*S. pimpinellifolium*	LA1579
Arthropods	*S. habrochaites*	LA0407 and many others
Arthropods	*S. pennellii*	LA0716, and others

4. CYTOGENETIC STOCKS

4.1. Translocations (37)

The following group of translocation stocks have been assembled from the collections of their originators - D.W. Barton, C.D. Clayberg, B.S. Gill, G.R. Stringham, B. Snoad, and G. Khush. As far as we know, they are all homozygous for the indicated structural changes. They are described by Gill *et al*. (TGC 23: 17-18; TGC 24:10-12). Accessions with an asterisk comprise the tester set.

Accession	Chrom.s
*LA1115	T9-12
*LA1119	T3-8
*LA1120	T6-12
*LA1876	T1-2
*LA1885	T5-7
*LA1898	T2-10a
*LA1899	T6-11
*LA1903	T4-7
LA1049	T1-9
LA1116	T1-11
LA1117	T5-7
LA1118	T7-11

Accession	Chrom.s
LA1121	T4-9
LA1122	T2-9
LA1123	T2-9
LA1124	T3-9
LA1125	T5-7
LA1126	T7-9
LA1127	T3-5
LA1129	T3-9
LA1877	T2-4
LA1878	T2-7
LA1879	T2-9
LA1880	T2-11
LA1881	T2-12

Accession	Chrom.s
LA1882	T12-3 or -8
LA1883	T3-7
LA1884	2 IV T3-8,9-12
LA1886	T12-3 or 8
LA1892	2 IV T9-12, ?-?
LA1894	T2-9a
LA1895	T2-9b
LA1896	T1-12
LA1897	T7-11?
LA1902	T2- ?
LA1904	T2-9d
LA1905	T1-3 or 8
LA1906	T2-10b

4.2. Trisomics (34)

The following series of trisomics contain various kinds of extra chromosomes. Since the extras are transmitted irregularly, each stock necessarily produce a majority of diploid progeny, the remainder aneuploid. Primary trisomics yield mostly 2n and 2n+1, and rarely tetrasomics (2n+2). Telotrisomics yield telos and an occasional rare tetratelosomic. Secondary, tertiary, and compensating trisomics transmit other trisomic types as expected. Because transmission is irregular and reproduction of stocks requires much labor, our stocks are limited. In requesting our aneuploids, researchers are asked to keep these points in mind. To assist in the identification of primary trisomics at the seedling stage, the key features of each have been summarized by Rick (TGC 37:60-61, 1987). Additional 2n+1 stocks are listed under Monosomic Alien Additions.

Accession	Genotype
Primary trisomics	
delta-10	Triplo-1
delta-06	Triplo-2
delta-08	Triplo-3
delta-02	Triplo-4
delta-04	Triplo-5
delta-12	Triplo-6
delta-07	Triplo-7
delta-03	Triplo-8
delta-05	Triplo-9
delta-01	Triplo-10
delta-40	Triplo-11
delta-09	Triplo-12

Accession	Genotype
Telo-trisomics	
delta-14	2n + 3S
delta-17	2n + 3L
delta-21	2n + 4L
delta-20	2n + 7L
delta-19	2n + 8L
delta-35	2n + 10S
Secondary trisomics	
delta-44	2n + 2S·2S
delta-43	2n + 5L·5L
delta-36	2n + 7S·7S
delta-26	2n + 9S·9S
delta-31	2n + 9L·9L

Accession	Genotype
delta-28	2n + 10L·10L
delta-41	2n + 11L·11L
delta-29	2n + 12L·12L
Tertiary trisomics	
delta-18	2n + 2L·10L
delta-16	2n + 4L·10L
delta-39	2n + 5L·7S

Accession	Genotype
delta-15	2n + 7S·11L
delta-25	2n + 9L·12L
delta-23	2n + 1L·11L
Compensating trisomics	
delta-32	2n - 3S·3L + 3S + 3L·3L
delta-33	2n - 3S·3L + 3S·3S + 3L·3L
delta-34	2n - 7S·7L + 7S·7S + 7L·7L

4.3. Autotetraploids (17)

We are currently maintaining only the following group of tetraploids. Whereas we formerly stocked many more lines, their rapid deterioration, low seed yields, and lack of demand required that we prune them to a smaller group of more frequently used genotypes. All are *L. esculentum* unless otherwise noted, and arose from either induced or spontaneous chromosome doubling.

Accession	Genotype
2-095	cv. San Marzano
2-483	cv. Red Cherry
LA0457	cv. from Tacna
LA0794	*ag, t^{v}*
LA1917	*L. chilense*
LA2335	*L. pimpinellifolium*
LA2337	cv. Stokesdale
LA2339	cv. Pearson
LA2340	*L. pimpinellifolium*
LA2342	cv. Danmark

Accession	Genotype
LA2343	cv. Waltham Fog
LA2581	*L. peruvianum*
LA2582	*L. peruvianum* var. *humifusum*
LA2583	*L. chilense*
LA2585	*L. pimpinellifolium*
LA2587	*L. esculentum* var. *cerasiforme*
LA3255	cv. Ailsa Craig

5. CYTOPLASMIC VARIANTS (3)

The following three lines are cytoplasmically-inherited chlorotic variants maintained by the TGRC and included in the miscellaneous group for want of better classification. They were induced by mutagens and are inherited in strictly maternal fashion. They are not transmitted by pollen but in reciprocal crosses -- no matter what male parents we have used -- the progeny are 100% variant.

LA1092 Uniform yellow, induced by fast neutrons in hybrid background (G.S. Khush)
LA1438 Light green, induced by X-rays in cv. Moneymaker (K. Kerkerk)
LA2979 Cyto-variegated, in cv. Glamour (R.W. Robinson)

6. GENETIC MARKER COMBINATIONS

6.1. Chromosome Marker Stocks (184)

This group consists of stocks in each of which has been assembled a series of marker genes for a single chromosome. In a few cases markers on other chromosomes are also present (listed in parentheses). Some of the more useful stocks have been combined with male steriles in order to make them useful for large scale test crossing. These stocks are listed below according to chromosome, and within each chromosome group by accession number. Asterisks indicate the preferred marker combination for each chromosome (i.e. that which provides the best map coverage).

Access.	Genotype

Access.	Genotype
Chromosome 1	
LA0910	*per, inv*
LA0984	*scf, inv*
LA0985	*inv, per*
LA1003	*scf, inv, per*
LA1082	*era, um*
LA1107	*inv, co*
LA1108	*inv, dgt*
LA1169	*scf, dgt*
LA1173	*gas, co*
LA1184	*au^{tl}, dgt*
LA1185	*au^{tl}, scf, inv*
LA1186	*au^{tl}, scf, inv, dgt*
LA1431	*au^{tl}, dgt*
LA1490	*au^{tl}, co, inv, dgt*
LA1492	*ms-32, bs*
LA1529*	*au^{tl}, co, scf, inv, dgt*
LA2354	*br, y* (*p, l*)
LA3209	*imb, irr, y*
LA3301	*fla, com^{in}*
LA3302	*imb, com^{in}*
LA3303	*imb, inv*
LA3305	*imb, Lpg*
LA3306	*com^{in}, inv*
LA3307	*com^{in}, Lpg*
LA3346	*au, bs*
LA3347	*au, ms-32*
LA3348	*au, com*
LA3349	*au, imb*
LA3350	*au, br*
LA3351	*imb, Lpg/+*
LA3352	*imb, au, Lpg/+*?
Chromosome 2	
LA0271	*aw, O*
LA0286	*d, m*
LA0310	*Wo^{m}, d*
LA0330	*bk, o, p, d, s* (*r, y*)
LA0342	*Wo^{m}, d* (*ms-17*)
LA0514	*aw, Wo^{m}, d*
LA0639	*Me, aw, d*
LA0650	*aw, d*
LA0715	*Wo^{m}, Me, aw, d*
LA0732	*suf, d*
LA0733	*Wo^{m}, d, ms-10*
LA0754	*aw, p, d, m, o*
LA0777	*dil, d*
LA0789	*Me, aw, d, m*
LA0790	*wv, Me, aw, d*
LA0986	*s, bk, Wo^{m}, o, aw, p, d*
LA1525	*aa, d*

Access.	Genotype
LA1526	*are, wv, d*
LA1699	*Wo^{m}, bip*
LA1700*	*wv, aa, d*
LA3132	*Prx-2^{1}, ms-10, aa*
Chromosome 3	
LA0644	*r, wf*
LA0782	*sy, sf*
LA0877	*pau, r*
LA0880	*sf, div*
LA0987	*pli, con*
LA0988	*ru, sf*
LA1070	*ru, sf, cur*
LA1071	*sy, bls, sf*
LA1101	*cn, sy, sf*
LA1175	*bls, aut*
LA1430*	*sy, Ln, bls, sf*
Chromosome 4	
LA0774	*ful, e*
LA0885	*ful, e, su^{3}*
LA0886	*ful, ra, e*
LA0888	*ful, ven, e*
LA0889	*ra, su^{3}*
LA0890	*ra, ven*
LA0902	*ful, ra^{2}, e* (*ms-31*)
LA0915	*clau, ful*
LA0916	*clau, ra, su^{3}*
LA0917*	*clau, ful, ra, e, su^{3}*
LA0920	*ful, ra, e, su^{3}*
LA0989	*afl, ful*
LA0990	*cm, ful, e, su^{3}*
LA0992	*clau, ra, su^{3}* (*com*)
LA0993	*ra, si*
LA0994	*cm, ver*
LA1073	*clau, afl*
LA1074	*clau, ver*
LA1075	*ver, e, su^{3}*
LA1536	*clau, su^{3}, ra; icn*
Chromosome 5	
LA0512	*mc, tf, wt, obv*
LA1188	*frg, tf*
LA3850*	*af, tf, obv*
Chromosome 6	
LA0336	*c, sp* (*a, y*)
LA0640	*yv, c*
LA0651	*m-2, c*
LA0773	*yv, m-2, c*
LA0802	*yv, m-2, c* (*ms-2*)
LA0879	*tl, yv*
LA1178	*yv, coa, c*
LA1189*	*pds, c*
LA1190	*pds, yv*
LA1489	*yv, ves-2, c*

Access.	Genotype
LA1527	*d-2, c*
LA3805	*m-2, gib-1*
LA3806	*yv, Mi, B^{og}, sp, c*
LA3807	*tl, yv, c*
Chromosome 7	
LA0788	*La/+, deb*
LA0882	*La/+, deb, adp*
LA0923	*ig, La/+*
LA0924	*La/+, not*
LA1083	*ig, flc*
LA1103*	*var, not*
LA1104	*deb, not*
LA1172	*La/+, lg-5*
Chromosome 8	
LA0513	*l, bu, dl*
LA0712	*l, bu, dl; ms-2*
LA0776	*l, va^{virg}*
LA0897	*l, bu, dl, al*
LA0922	*bu, dl, spa*
LA0998	*l, bu, dl, Pn/+*
LA0999	*tp, dl*
LA1012	*dl, l*
LA1191	*spa, ae*
LA1442	*dl, glg, marm*
LA1666*	*l, bu, dl, ae*
Chromosome 9	
LA0883	*pum, ah*
LA0884	*wd, marm*
LA1000	*nv, ah*
LA1001	*pum, ah, marm*
LA1100	*ah, pla, marm*
LA1112	*marm, lut*
LA1176	*Crk, ah, marm*
LA3353*	*ah, marm, pct*
LA3841	*Tm-2^{a}, Frl, nv,* ™
Chromosome 10	
LA0158	*Xa/+, u, t* (*y*)
LA0339	*ag, u*
LA0341	*h, ag* (*ms-2*)
LA0643	*u, l-2*
LA0649	*t^{v}, ag*
LA0711	*t^{v}, ag* (*ms-2*)
LA1002	*h, u, l-2, t, ag* (*pe, lg*)
LA1085	*h, res*
LA1086	*h, ten*
LA1110	*icn, ag*
LA1192	*hy, ag*
LA1487	*icn, t^{v}*
LA2493	*Xa-2, hy, h, ag*
LA2495	*Xa-2, h, ten, ag, al*
LA2496	*Xa-2, h, l-2, t*
LA2497	*hy, u, icn, h, ag*

Access.	Genotype
LA2498	*u, Xa-3, h*
LA2499	*u, nor, t*
LA2500	*u, icn, h*
LA2501	*u, icn, h, ag*
LA2502	*u, h, auv, l-2, t^{v}*
LA2503	*u, h, l-2, t^{v}, ag*
LA2504*	*u, h, t, nd, ag*
LA2505	*u, l-2, t, ag, Xa*
LA2506	*ag, h, l-2, oli, t^{v}*
LA2507	*h, t, nd, ag*
LA2508	*h, t, ag, Xa*
LA2509	*oli, l-2, t^{v}, ag* (*wf*)
LA2591	*Xa-2, h, ag*
LA2592	*u, h, t, nd, ag*

Access.	Genotype
LA2593	*u, auv, ag*
LA4341	*h, hy, u*
Chromosome 11	
LA0259	*hl, a*
LA0291	*hl, a* (*ms-2*)
LA0729	*neg, a*
LA0730	*a, pro*
LA0761	*a, hl, j*
LA0798	*a, hl, j* (*ms-2*)
LA0803	*hl, a, pro* (*ms-2*)
LA0881	*neg, hl, a*
LA0925*	*j, hl, a, f*
LA1102	*a, hl, tab*
LA1109	*j, hl, mnt*

Access.	Genotype
LA1488	*neg, ini*
LA1786	*j, f, a, bi* (*c*)
LA2352	*j, f* (*p, c*)
LA2364	*j, a, f* (*y, wt, c, l, u*)
LA2489	*neg^{ne-2}, a*
LA4290	*a, bks*
LA4291	*a, bks^{2}*
LA4292	*j-2, up, wv-3*
LA4344	*a, mon*
Chromosome 12	
LA1111	*fd, alb*
LA1171	*yg-2^{aud}, fd*
LA1177*	*alb, mua*

6.2. Linkage Screening Testers (15)

The following set of linkage testers each combines two pairs of strategically situated markers on two different chromosomes (see TGC 22: 24). They are intended primarily for assigning new, unmapped markers to a chromosome. The more complete chromosome marker combinations (list 6.1 above) should be used for subsequent testing to delimit loci more accurately. Whereas six of these stocks should pretty well cover the tomato genome, we list below the entire series of the current available testers because alternative stocks differ in their usefulness, depending upon the phenotype of the new mutant to be located. The chromosomal location of each pair of markers is indicated in parentheses.

Access.	Genotype
LA0780	*yv, c* (chr 6)*; h, ag* (chr 10)
LA0781	*ful, e* (chr 4)*; neg, a* (chr 11)
LA0784	*ful, e* (chr 4)*; hl, a* (chr 11)
LA0982	*clau, e* (chr 4)*; hl, a* (chr 11)
LA0983	*l, dl* (chr 8)*; ah, marm* (chr 9)
LA1163	*d, wv* (chr. 2); *obv, tf* (chr. 5)
LA1164	*var, not* (chr 7)*; ah, marm* (chr 9)

Access.	Genotype
LA1166	*clau, su^{3}* (chr 4)*; icn, ag* (chr 10)
LA1182	*sy, sf* (chr 3)*; alb, mua* (chr 12)
LA1441	*coa, c* (chr 6)*; hl, a* (chr 11)
LA1443	*scf, dgt* (chr 1)*; l, al* (chr 8)
LA1444	*wv, d* (chr 2)*; af, tf* (chr 5)
LA1491	*scf, dgt* (chr 1)*; spa, ae* (chr 8)
LA1665	*scf, dgt* (chr 1)*; l, ae* (chr 8)

6.3. Miscellaneous Marker Combinations (321)

The following list groups stocks in which various mutant genes have been combined for various purposes. A few of these items include linked genes, but are classified here because other linkage testers provide the same combinations or because they are more useful as markers of several chromosomes. Some multiple marker combinations that are of limited usefulness, difficult to maintain, and/or redundant with other genotypes, have been dropped from the current list.

Access.	Genotype
LA0013	*a, c, d, l, r, y*
LA0014	*al, d, dm, f, j, wt, h*
LA0052	*j, wt, br*
LA0085	*Wo, d, h*
LA0137	*dl, wd, gq*
LA0154	*u, d, sp, h*
LA0158	*t, u, Xa, y*
LA0159	*a, e, mc, t, u, y, wf*
LA0169	*ps, wf, wt*
LA0189	*bl, cl-2*
LA0190	*wf, br, bk*
LA0215	*at, y, u*
LA0281	*e, t, u*
LA0296	*br, bk, wf*
LA0297	*tf, ug, Nr*
LA0299	*ag, rv*
LA0345	*ch, j-2*
LA0497	*ch, j-2, sf*
LA0499	*Od, sn, at, cm/+*
LA0508	*gf, d, c, a, r, y*
LA0638	*ht, d, r*
LA0648	*rv, e, Wo, wf, j, h*
LA0719	*Jau, clau*
LA0727	*wv, d, c, r*
LA0728	*a, lut*
LA0759	*lg, vi, pe, t*
LA0760	*lg, vi*
LA0770	*clau, pa*
LA0775	*tf, h, au, +/d*
LA0801	*atv, slx*
LA0875	*hp, u, sp*
LA0876	*hp, sp*
LA0895	*tp, sp, u, Hr*
LA0907	*lut, pr*
LA0908	*per, var*
LA0909	*con, sf*
LA0912	*ht, su^3*
LA0913	*ful, su^3, ht*
LA0914	*com, ful*
LA0991	*ful, e, com*
LA0995	*deb, um*
LA0996	*um, ig*
LA1018	*h, Od, ptb*
LA1038	*e, ht, su*

Access.	Genotype
LA1072	*sy, sf, um*
LA1078	*ria, ves-2*
LA1079	*c, ves-2*
LA1105	*con, cur*
LA1106	*fsc, ah*
LA1170	*cn, con*
LA1219	*d, u*
LA1663	*Ln, Wo^m*
LA1664	*hp, lp*
LA1783	*ad, sp*
LA1787	*Bk-2, en*
LA1789	*sl^{cs}, a*
LA1796	*Rs, d, h*
LA1804	*sr, sp, u*
LA1805	*sr, y*
LA1806	*ti, y, wf, al, j*
LA2350	*y, ne, p, c, sp, a*
LA2353	*y, wt, n*
LA2355	*sp, ug*
LA2360	*e, wt, l, u*
LA2363	*y, Wo, wt, c, t, j*
LA2369	*p, Tm-1*
LA2370	*wf, n, gs*
LA2372	*sp, fl*
LA2441	*d, m-2, mc, rvt, t, u*
LA2452	*B, f, gf, y*
LA2453	*Gr, u*
LA2454	*neg^{ne-2}, u*
LA2457	*u, so*
LA2458	*Pto, sp, u*
LA2461	*sp, stu, u*
LA2464	*aer-2, r, upg, y*
LA2465	*sp, u, v-2*
LA2466	*d, t, v-3*
LA2467	*pe, u, vi*
LA2473	*alb, c, gra, sft*
LA2477	*vo, cjf, wf, sp, l, u, h*
LA2478	*ae^{afr}, r, gs, h*
LA2486	*inc, pds, sp, u, t*
LA2490	*pdw, mc, pst, dl*
LA2492	*ti, wf, e, mc, u, a*
LA2524	*af, sd*
LA2526	*dp, sp, u*

Access.	Genotype
LA2527	*l allele, sp, u*
LA2595	*br, d, dm, wt, al, h, j, f*
LA2597	*y, r, wf, mc, m-2, c, gs, gf, marm, h*
LA2797	*bu, j*
LA3128	*Ln, t, up*
LA3212	*tmf, d, sp, u*
LA3217	*glg, Pts*
LA3251	*Del, y*
LA3252	*Del, t*
LA3254	*a, c, l, Ve*
LA3256	*at, t*
LA3257	*gf, gs, r*
LA3258	*u, Ve*
LA3261	*Del, gs*
LA3262	*Del, ug*
LA3267	*Cf-4, u*
LA3268	*Tm-2, nv, u*
LA3269	*Tm-1, u*
LA3271	*Cf-?, Tm-1, u*
LA3273	*Gp, $Tm\text{-}2^2$*
LA3274	*ah, Tm-2, nv, u*
LA3275	*ah, Gp, $Tm\text{-}2^2$*
LA3276	*Tm-1, u, Ve*
LA3279	*at, Del*
LA3284	*at, gf*
LA3286	*r, ug, y*
LA3287	*hp, r, ug*
LA3288	*hp, ug, y*
LA3289	*gf, r, y*
LA3290	*gf, hp, y*
LA3291	*at, hp, t*
LA3292	*Tm-2, u*
LA3294	*bl, d, u*
LA3297	*Tm-1, Tm-2, nv*
LA3299	*ep, u*
LA3311	*og^c, u*
LA3315	*sp, pst, u, j-2, up, vo*
LA3362	*gs, t*
LA3363	*at, gs*
LA3364	*gs, u*
LA3365	*gf, gs*

Access.	Genotype
LA3366	*t, y*
LA3367	*hp, t*
LA3368	*hp, y*
LA3369	*at, y*
LA3370	*at, hp*
LA3371	*hp, u*
LA3372	*gs, y*
LA3373	*at, u*
LA3374	*u, y*
LA3375	*gs, r*
LA3376	*Del, hp*
LA3381	*r, y*
LA3382	*r, u*
LA3383	*gs, hp*
LA3384	*gf, y*
LA3385	*gs, Nr*
LA3386	*gf, t*
LA3387	*Nr, t*
LA3389	*Nr, y*
LA3390	*Nr, ug*
LA3391	*gf, hp*
LA3393	*r, t*
LA3394	*at, ug*
LA3395	*gs, hp, y*
LA3396	*at, u, y*
LA3397	*gs, t, y*
LA3398	*gs, hp ,t*
LA3399	*at, gs, hp*
LA3400	*at, hp, u*
LA3401	*at, gs, y*
LA3403	*gf, gs, u*
LA3404	*hp, u, y*
LA3405	*gs, hp, u*
LA3406	*at, hp, y*
LA3407	*gs, u, y*
LA3408	*t, u, y*
LA3409	*gs, t, u*
LA3410	*at, gs, u*
LA3411	*gs, r, u*
LA3412	*gf, gs, hp, u*
LA3413	*at, gf*
LA3414	*t, ug*
LA3415	*ug, y*
LA3416	*hp, ug*
LA3417	*r, ug*
LA3418	*gf, gs, ug*
LA3419	*at, gf, gs*
LA3420	*gf, ug*
LA3421	*Nr, u*
LA3422	*at, gs, ug*
LA3423	*gf, gs, hp, u, y*
LA3424	*gs, hp, u, y*
LA3425	*gf, gs, hp, t, u*
LA3426	*gs, hp, t, u*
LA3427	*gf, gs, t, u*
LA3428	*I, u, Ve*
LA3429	*Del, gs, hp*
LA3432	*Tm-1, Tm-2, nv, u*
LA3433	*ah, Tm-2, nv, u*
LA3437	*at, Nr*
LA3442	*de, dil, u*
LA3443	*cor, de, u*
LA3444	*cor, dil, u*
LA3445	*cor, pum, u*
LA3446	*cor, sp, u*
LA3447	*dil, sp, u*
LA3448	*in, u*
LA3449	*d, sp, u*
LA3450	*bls, sp, u*
LA3451	*bl, sp, u*
LA3540	*I, u*
LA3541	*gs, r, ug*
LA3542	*u, ug*
LA3543	*bls, o, u*
LA3545	*Del, u, y*
LA3546	*bls, Cf-?, u*
LA3547	*ah, u*
LA3548	*pum, u*
LA3549	*bls, Gp, Tm-2^2, u*
LA3557	*Del, gf*
LA3558	*gf, Nr*
LA3559	*Del, gs, y*
LA3561	*gf, gs, hp, Nr, u*
LA3562	*gf, gs, u, y*
LA3563	*sp, u*
LA3585	*gf, u, ug*
LA3587	*r, u, ug*
LA3589	*u, ug, y*
LA3590	*Nr, gs, y*
LA3591	*Nr, u, y*
LA3593	*hp, u, ug*
LA3594	*gs, hp , ug*
LA3595	*gf, hp, ug*
LA3596	*hp, t, ug*
LA3597	*at, hp, ug*
LA3598	*r, t, ug*
LA3599	*at, t, ug*
LA3600	*t, ug, y*
LA3601	*gf, r, t*
LA3603	*at, gf, y*
LA3604	*hp, r, t*
LA3605	*at, ug, y*
LA3606	*r, t, y*
LA3607	*gs, hp, Nr*
LA3608	*hp, Nr, t*
LA3609	*hp, Nr, y*
LA3615	*d^x, u*
LA3675	*hp, Nr, u*
LA3676	*gf, hp, t*
LA3677	*gf, hp, r*
LA3678	*Nr, u, ug*
LA3679	*gs, Nr, ug*
LA3680	*Nr, t, u*
LA3682	*gs, t, ug*
LA3683	*gs, ug, y*
LA3684	*Nr, t, y*
LA3686	*gs, Nr, t*
LA3688	*gf, gs, hp*
LA3689	*gs, hp, r*
LA3691	*r, u, y*
LA3692	*at, r, y*
LA3693	*g, t, u*
LA3694	*Del, gs, u*
LA3695	*Del, hp, t*
LA3697	*gs, r, t*
LA3698	*gs, r, y*
LA3699	*gf, u, y*
LA3700	*at, gf, u*
LA3701	*at, t, u*
LA3702	*gf, gs, y*
LA3703	*gf, hp , u*
LA3704	*at, gf, hp*
LA3706	*at, gs, t*
LA3706A	*Del, t, y*
LA3709	*Del, gf, gs, hp, u*
LA3741	*pum, u*
LA3742	*de, u*
LA3743	*cor, u*
LA3744	*sph, u*
LA3745	*bl, u*
LA3771	*hp, B^c*
LA3811	*gf, r*
LA3812	*bls, Tm, Tm-2, nv*
LA3815	*Del, t, ug*
LA3821	*dil, pum, u*
LA3826	*mon, u*
LA3827	*dil, cor, sp, u*
LA3830	*ep, B^c, u*
LA4136	*Rg-1, r*
LA4342	*oli, u, y*
LA4343	*gq, h*
LA4348	*yg-2, c^{int}*
LA4361	*fri, tri*
LA4362	*fri, phyB2*
LA4363	*cry1, fri*
LA4364	*phyB2, tri*

Access.	Genotype
LA4365	*cry1, tri*
LA4366	*fri, phyB2, tri*
LA4367	*cry1, tri, fri*
LA4368	*fri, hp-1, tri*
LA4369	*fri, hp-1, tri, phyB2*
LA4372	*a, alc, gf, gs, u*
LA4373	*a, c, gf, gs, j-2, t, u*
LA4374	*alc, gf, gs, j-2, t, u, y, yg-2^{aud}*
LA4375	*alc, c, gs, j-2, r, u, yg-2^{aud}*
LA4376	*alc, c, gf, gs, j-2, r, u, y, yg-2^{aud}*
LA4377	*gs, hp-1, o, u*
LA4378	*B^{c}, gs, hp-1, u*
LA4379	*gs, u*

Access.	Genotype
LA4380	*B, gs, u*
LA4381	*gs, j-2, o, t, u*
LA4382	*alc, B, u*
LA4384	*B, gf, u*
LA4385	*ag, sp, t, u*
LA4386	*ag, hp-1, sp, t, u*
LA4389	*B, hp-2^{dg}, sp, u*
LA4390	*B, hp-2^{dg}, o, u*
LA4391	*hp-2^{dg}, o, u*
LA4392	*B^{og}, rin, hp-1, j-2, mc, o, sp, u*
LA4393	*el, hp-1, j-2, nor, sp, u*
LA4394	*alc, hp-2, u*
LA4395	*alc, rin, j-2, sp*

Access.	Genotype
LA4399	*alc, B^{c}, hp-1, j-2, u*
LA4401	*el, gs, hp-1, j-2, nor, u*
LA4411	*B, hp-2^{dg}, j-2, sp*
LA4412	*alc, B, u*
LA4413	*B, gs, u*
LA4414	*alc, hp-2^{dg}, u*
LA4415	*gs, Spf, u*
LA4417	*s, j-2, nor, o, u*
LA4418	*hp-1, j-2, nor, sp, u*
LA4419	*rin, j-2, o, u*

7. Provisional mutants (106).

The following group of provisional mutants are listed here, rather than with the monogenic stocks because they have not been fully characterized. For some, a monogenic segregation has not been verified, for others complementation tests were either not performed or did not detect allelism with existing mutants of similar phenotype. Most of these lines resulted from mutagenesis experiments, the remainder occurring spontaneously. Genetic background is indicated, if known. More information on these stocks is available at our website.

Access.	Traits
2-293	Snout
2-305	Broad
2-473	Yellow fruit, pale corolla
2-493	Purple tipped leaves, puny
2-575	Poxed fruit
2-585	Balloon
2-621	Turbinate
2-625	Prolific leaves
2-629	Me-oid
2-633	Hooded flowers
2-643	Yellow green
3-003	yv-oid
3-055	Round cotelydons and leaves
3-073	Abnormal flowers
3-077	Dwarf
3-082	Dwarf
3-083	Yellow virescent
3-084	Yellow green
3-088	Light green, dark veins
3-097	Yellow green
3-098	Slow chlorotic
3-101	tl mimic
3-106	Strong anthocyanin
3-107	Bright yellow virescent
3-112	Crippled
3-115	rv-oid
3-118	Rugose recurved leaves
3-127	Bright yellow
3-241-1	Yellow, anthocyanin
3-243	Long narrow
3-303	Slow, narrow leaves
3-305	La-mimic
3-307	Broad, grey green
3-309	Bunchy growth, mitten leaves
3-311	Slow, rugose
3-313	Acute, olive green
3-315	Glossy dwarf
3-317	ra-oid
3-319	Striated, divided
3-321	Narrow, dissected
3-323	Spirally coiled
3-325	Short, yv
3-329	Bronzing
3-331	Serrated leaves
3-335	Gold dust virescent
3-337	Glossy dwarf
3-341	Dwarf
3-403	Fimbriate leaves
3-404	Speckled white
3-405	Streaked virescent
3-406	Streaked variegated
3-408	bu mimic
3-411	Blue green; bushy roots

Access.	Traits
3-423	ra-oid
3-424	Extreme dwarf
3-434	d^cr like
3-436	Overall yellow
3-441	Singed hairs
3-601	clau mimic
3-612	wiry mimic
3-613	La mimic
3-614	pds-oid
3-617	Dwarf
3-618	mimic of a
3-619	wiry mimic
3-621	d mimic
3-622	d mimic
3-624B	Yellow virescent
LA0506	Triplo-8 mimic
LA0652	calycine poxed
LA0739	ag mimic
LA0765	Acute leaves
LA0791	Long John
LA0801	Pseudopolyploid
LA0870	frizzled virescent
LA0871	Calico
LA1012	Mottled, chlorotic petiole
LA1060	spl-oid
LA1065	Miniature
LA1066	Speckled
LA1095	fy-oid
LA1098	Multiple inflor.
LA1144	ful mimic
LA1148	Light green
LA1149	Xanthoid
LA1154	pale virescent, twisted leaves
LA1160	Fused cotyledons
LA1193	Yellow-sectored
LA1201	rv-oid
LA1202	Dirty orange cherry
LA1436	Withered cotyledons
LA1494	Adventitious roots
LA1532	rv-oid
LA1533	Purple stem
LA1707	Short stature
LA2018	Anthocyanin deficient
LA2019	Virescent tangerine mimic
LA2020	Dark green foliage
LA2021	Variegated yellow
LA2358	Marginal leaf chlorosis
LA2375	Lc- reduced locule
LA2806	Incomplete anthocyanin mutant
LA2817	lg mimic
LA2897	Virescent gold top
LA2899	Wrinkled fruit
LA3851	Virescent

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