Report of the Tomato Genetics Cooperative

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Report

of the

Tomato Genetics Cooperative

Number 58- September 2008

University of Florida

Gulf Coast Research and Education Center

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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 is published annually and contains reports of work in progress by members, announcements and updates on linkage maps and materials available. The research reports include work on diverse topics such as new traits or mutants isolated, new cultivars or germplasm developed, interspecific transfer of traits, studies of gene function or control or tissue culture. Relevant work on the Solanaceous species is encouraged as well.

Paid memberships currently stand at approximately 91 from 19 countries. Requests for membership (per year) at US$20 to addresses in the US and US$25 if shipped to addresses outside of the United States should be sent to Dr. J.W. Scott, jwsc@ufl.edu. Please send only checks or money orders. Make checks payable to the **University of Florida**. We are sorry but we are **NOT** able to accept credit cards. If you have a problem with sending a check or money order, contact J.W. Scott.

Cover:
The cover shows a tomato leaf infected by *Pseudomonas syringae* pv. *tomato* and exhibiting the symptoms of bacterial speck disease. The leaf is superimposed over a gel blot showing degradation of the tomato resistance protein, Fen, caused by the pathogen effector protein, AvrPtoB (see feature article by G. Martin for more details; photo by Kent Loeffler and Tracy Rosebrock, Department of Plant Pathology and Plant-Microbe Biology, Cornell University).

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From the editor:

A fond hello to the TGC membership. Be sure to check out our "Cover Story", the feature article by Greg Martin summarizing some of the elegant work his group has done in elucidating the mechanism of resistance to the bacterial speck pathogen. Also in this issue is a memoriam for Ernie Kerr who passed away on August 30 at the age of 91. Ernie was a tomato pioneer on the gene list committee who contributed much to the establishment of tomato as a model crop that is now being sequenced. In 1959 he volunteered to do linkage work on chromosome 10 as well as screen unlocated genes and test for linkage of disease resistance genes (TGC 9:5). He authored numerous linkage reports in TGC over the next 20 years. Among his many contributions, was his extensive work on cladosporium leaf mold resistance with some key work published in TGC.

For those of you keeping track of our numbers in the Forward it appears that we had a precipitous drop from last year. However, the 2007 figure was not accurate as it included some people who were no longer current members, this year's figure is correct. My thanks go to Dolly Cummings who does most of the work in preparation of the TGC report and keeping our spreadsheets and mailings in order. Christine Cooley and Dolly have been helping with the website updates. My contact information remains the same:

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Do not hesitate to contact me if you have any questions or concerns. Also be sure to check our website for additional TGC information: **http://tgc.ifas.ufl.edu/.** All volumes are electronically available online and fairly complete searches can be done by keyword. Thanks to all who have submitted reports this year and I hope everyone will consider submitting reports in the future. If there has been a change in your contact information please email me about it. Good luck in your future tomato pursuits.

Jay W. Scott
Managing Editor

2008 and 2009 Tomato Meetings

- 5th Solanaceae Genome Workshop 2008, Oct 1-8, 2008, Cologne, Germany.
- 23rd Annual Tomato Disease Workshop, October 15-16, 2008, Raymond, Mississippi
- 43rd Tomato Breeders Round Table, June 28- July 1, 2009, Sacramento, California

Dear fellow tomato scientists,

The venue and dates have been selected for the 43rd Tomato Breeders Round Table. The 2009 meeting will be held June 28th through July 1st, 2009 at the Embassy Suites, Sacramento, California. This TBRT is being jointly hosted by several processing tomato breeding programs – Campbell's, Nunhems, HeinzSeed, Harris Moran, and Seminis - as well as the Center of Fruit and Vegetable Quality at UC Davis.

Embassy Suites is located at the south end of "Old Sacramento", a state historic park known for its shops, train museum and wide variety of restaurants. There are many more activities available within a few-block walking distance from this riverfront hotel (art museum, mall, even a baseball stadium). The conference room rate will be $179 per night (mention 2009 TBRT) with a limited number of rooms available the nights before and after the conference at this same rate. The rate includes complementary cocktails at happy hour, airport shuttle and cooked-to-order breakfast. Room reservations can be made at http://embassysuites.hilton.com/en/es/groups/personalized/SACESES-TOM-20090628/index.jhtml

A complementary shuttle is available from the Sacramento Airport to the hotel (about 10 miles) so a car may not be necessary even if you or a guest have some extra time to fill. If you do rent a car, UC Davis is located about 30 minutes west of the hotel. San Francisco is about 100 miles away. The Sacramento Airport is serviced by most major domestic airlines. Driving time from the San Francisco airport can range from 1.5 to 3 hours depending on traffic generally making it less convenient than Sacramento for all but international travelers.

It is our intention to steer the meeting to a more discussion-oriented model, perhaps with shorter, less formal presentations; a return to our "round table" roots. The Center at UC Davis will be contributing to the program and including a tour of their new sensory laboratory. If you have ideas for speakers or presentations, please let pass them to one of the organizers (Dawn Adams, Diane Barrett, Steve Schroeder, Mike Kuehn, Teresa Beck-Bunn or myself). A website and mailing will be forthcoming with more details and program information.

PLEASE NOTE: If you are interested in receiving more information on this meeting and you DID NOT receive a "Save-The-Date" e-mail in June, we may not have your current contact information. Please forward your e-mail and mailing addresses to Rich Ozminkowski at the e-mail address below with TBRT in the subject line.

See you there.

The 2009 TBRT Organizing Committee.

Rich Ozminkowski *(rich.ozminkowski@us.hjheinz.com)*
Dawn Adams *(dawn_adams@campbellsoup.com)*
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Theresa Beck-Bunn *(teresa.beck.bunn@seminis.com)*

Grant Opportunity: Request for Proposals for Tomato Germplasm Evaluation

Funding is expected to be available again in fiscal year 2009 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 by November 7, 2008 so that reviews and rankings can be forwarded to the USDA in Beltsville.

Evaluation priorities established by the CGC will provide review criteria. These criteria were revised in 2006, and applicants are encouraged to review the URL (http://www.ars-grin.gov/npgs/cgc_reports/tomatocgc2006evalpriorities.html). **The Tomato CGC is placing the highest emphasis on Genetic analysis to define core collections**. Our aim is to collect objective data that can be leveraged to expand the knowledge of genetic diversity within the tomato collections. Although there are several proposed core collections (http://tgrc.ucdavis.edu) and (https://www.msu.edu/~douchesd/SolResources.html), the CGC will consider new proposals for core collections drawing from NPGS supported centers. If molecular data are to be collected, marker systems and allele calling that are unambiguous and can be linked to existing data are strongly encouraged.

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 David Francis, CGC Chair, francis.77@osu.edu by November 7.

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).

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Use of tomato as a model system to understand the molecular basis of plant disease resistance

Gregory Martin, Boyce Thompson Institute for Plant Research and Department of Plant Pathology and Plant-Microbe Biology, Cornell University (Email: gbm7@cornell.edu)

There is now substantial evidence that plants use two successive surveillance systems to defend themselves against pathogens. In the first system, plants detect various pathogen-associated molecular patterns (PAMPs) by using pattern recognition receptors (PRRs). These PRRs, in concert with other host proteins, then activate signaling pathways leading to what is now referred to as PAMP-triggered immunity (PTI). A common counter-strategy of many pathogens is the production and delivery into the plant cell of various virulence proteins (or 'effectors') many of which act to suppress PTI. In response to pathogen effector proteins, plants have evolved a second defense mechanism that relies on resistance (R) proteins. R proteins detect the presence of specific effector proteins and activate a strong immune response typically associated with rapid localized cell death (the hypersensitive response, HR). This second defense system, which has been relied upon for many years by plant breeders to develop disease-resistant crops, is now often referred to as effector-triggered immunity (ETI). To counter ETI, some pathogen effectors have further evolved to interdict either R protein recognition or downstream signaling events.

This integrated view of the evolutionary 'arms race' between plants and pathogens has emerged just recently and is due largely to the discovery of the central role of PTI in plant disease resistance. Despite this remarkable advance, we still have much to learn about the molecular basis of PTI, ETI, and the way in which effectors act in both of these processes. For example, we know relatively little about the number or types of PRRs and the PAMPs they recognize or the structural basis by which R proteins recognize specific effector proteins to activate ETI. Correspondingly, little is known about the mechanisms effector proteins use to interfere with PRR function and how, in some cases, effectors are also able to overcome ETI. Future progress on these questions is important for both our understanding of plant immunity and for providing new approaches for generating durable, broad-spectrum disease resistance in crop plants. This short article summarizes some recent advances in our understanding of the molecular basis of plant-pathogen interactions, with a focus on

the use of tomato in these studies and on the interaction of the bacterial pathogen *Pseudomonas syringae* pv. *tomato* with tomato.

Tomato has developed into an excellent model system for studying responses to various pathogens and in particular for investigating aspects of both PTI and ETI. Tomato is a host to many well-characterized and economically important pathogens (viruses, bacteria, fungi, oomycetes, and nematodes). The relatively large size of tomato leaves (and especially leaves of the related model plant, *Nicotiana benthamiana*) permits facile quantification of pathogen populations and of subtle aspects of disease and provides sufficient host tissue for biochemical analyses. The natural genetic diversity of tomato has been key to the identification of many *R*) genes. Tomato was the first plant species from which a 'gene-for-gene' type of *R* gene was cloned, and more than 25 *R* genes have now been isolated from tomato and other solanaceous species. The influential ‘guard hypothesis’ of R protein function arose from observations in tomato and the many cloned solanaceous *R* genes and abundance of information and resources related to diverse plant defense responses provide an unparalleled foundation for using tomato as a model system to increase our understanding of both PTI and ETI. Finally, it is important to note that potato, pepper, or tobacco are susceptible to many of the same pathogens as tomato (e.g. *Phytophthora* spp., *Pseudomonas*, *Fusarium* spp.). Thus, discoveries made using tomato are likely to benefit our understanding of many economically-important plant species.

The many experimental and bioinformatics resources available for tomato add to its usefulness for studying plant-pathogen biology. Extensive genetic and genomic resources are now available for tomato and its genome is currently being sequenced and analyzed (see the Solanaceae Genomics Network (SGN) website: http://www.sgn.cornell.edu/). Tomato and *N. benthamiana* are amenable to cellular-level experiments using protoplasts, to RNAi-based gene silencing by using virus-induced gene silencing (VIGS), and to stable and transient transformation by using *Agrobacterium*. Finally, wide crosses are feasible between tomato and many of its wild relatives allowing access to exceptional natural variation.

Many of the advances in understanding the response of tomato to pathogens have come from studying its interaction with *Pseudomonas syringae* pv. *tomato (Pst)*, the causative agent of bacterial speck disease. Tomato is the natural host for *Pst* and wild relatives of tomato appear to have co-evolved with this pathogen in the species’ center of origin in South America. Bacterial speck is an

economically important disease throughout the world where tomatoes are grown in cool, wet climates (Europe, northern California, northeast U.S, and South America). Where adapted varieties lack genetic resistance (see below), bacterial speck is controlled by application of copper-based pesticides. Aside from this practical reason for studying the disease, this pathosystem offers many experimental advantages for studying fundamental aspects of plant-pathogen interactions. These include the advantages of tomato described above and the fact that the complete genome sequence of *Pst* is available and along with many experimental and web-based resources for *Pst* and for related bacterial pathogens (see the *Pseudomonas*-Plant Interaction (PPI) website: http://pseudomonas-syringae.org/).

Much has been learned about the molecular basis of the tomato-*Pst* interaction in the past 15 years. As part of its infection process, *Pst* uses its type III secretion system to inject ~30 effector proteins into the plant cell. Two of these effectors, AvrPto and AvrPtoB, have been intensively studied by many labs. These effectors both contribute to bacterial virulence and recently they have been shown to interfere with host proteins required for PTI. Probably due to their important role in bacterial virulence, tomato has evolved genes whose proteins specifically target the parts of these effectors that are required for their virulence activity. This recognition leads to ETI involving complex defense responses including generation of reactive oxygen species, increased expression of many defense-associated genes, production of anti-microbial proteins, and localized cell death (the HR).

Recent evidence from tomato suggests that ETI directed against *Pst* evolved in at least two steps (see Figure 1). First, a host protein kinase called Fen arose in order to detect the presence of the N-terminal region of AvrPtoB. However, over time genome rearrangements in *Pst* led to the acquisition by AvrPtoB of a C-terminal domain. This domain is a molecular mimic of a eukaryotic E3 ubiquitin ligase and it acts to facilitate the targeting and ultimate degradation of the Fen kinase. Secondly, in response to this subversion of Fen, tomato appears to have evolved (probably by gene duplication) another protein kinase gene, *Pto*. The Pto kinase is able to detect AvrPtoB despite the presence of the E3 ligase and therefore re-establishes effective ETI. The *Pto* gene has been in use for over 20 years and, although there are occasional reports that it is losing its effectiveness, it remains the best source of genetic resistance to bacterial speck disease. The relative stability of Pto-mediated resistance may be due to the fact that the two effectors it recognizes both play important roles in bacterial virulence. It is interesting to speculate about how the pathogen will eventually evolve to

respond to the presence of Pto. Deletion of the *avrPto* or *avrPtoB* genes entirely from the genome or simple mutations of the genes that would allow avoidance of recognition by Pto reduces bacterial virulence and would seemingly be detrimental to bacterial fitness. It is possible that a new effector gene with activity similar to *avrPto* and *avrPtoB* but unable to be recognized by Pto or Fen, will eventually be introduced into the *Pst* genome by horizontal gene transfer.

One of the lessons of the *Pst*-tomato system for plant breeders appears to be that the most durable and effective *R* genes will be those that detect effector proteins that have the greatest fitness advantage for the pathogen. This highlights the importance of understanding fundamental aspects about virulence activities in order to identify the "Achilles heel" of the pathogen. This knowledge will then allow directed efforts to seek *R* genes that target these specific virulence determinants.

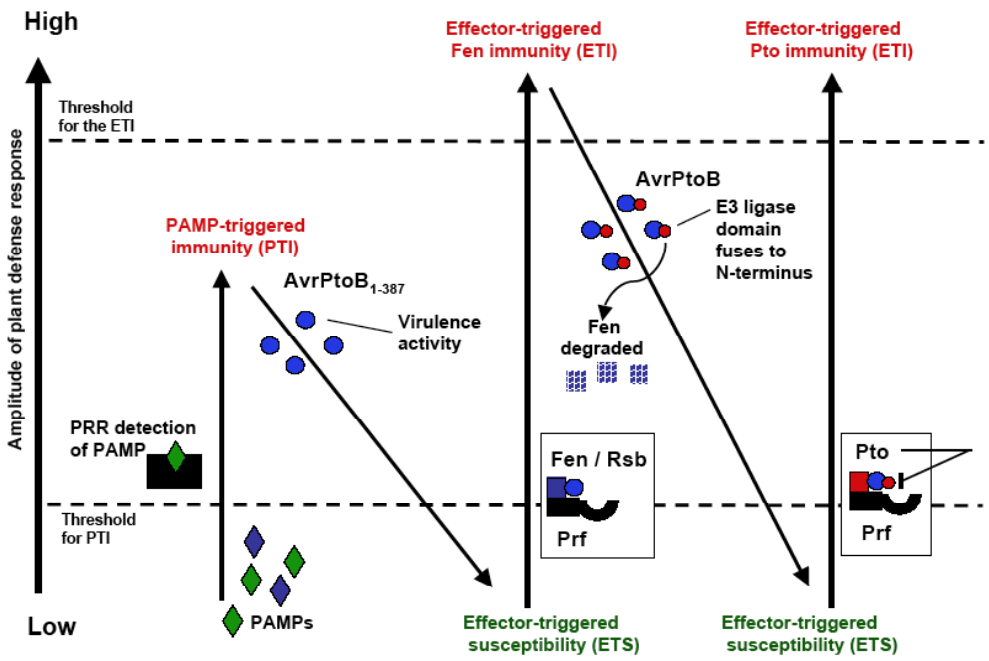

Figure 1: A model of the 'evolutionary arms race' between tomato and *Pseudomonas syringae*.

Additional reading on this topic

Abramovitch RB, Anderson JC, Martin GB: Bacterial elicitation and evasion of plant innate immunity. *Nature Reviews Molecular Cell Biology* 2006, 7:601-611.

Jones JD, Dangl JL: The plant immune system. *Nature* 2006, 444:323-329.

Leach JE, Vera Cruz CM, Bai J, Leung H: Pathogen fitness penalty as a predictor of durability of disease resistance genes. *Annual Review of Phytopathology* 2001, 39:187-224.

Pedley KF and Martin GB: Molecular basis of *Pto*-mediated resistance to bacterial speck disease in tomato. *Annual Review of PhytoPathology* 2003, 41:215-43.

Rosebrock TR, Zeng L, Brady JJ, Abramovitch RB, Xiao F, and Martin GB: A bacterial E3 ubiquitin ligase targets a host protein kinase to disrupt plant immunity. *Nature* 2007, 448:370-374.

Zipfel C: Pattern-recognition receptors in plant innate immunity. *Current Opinions in Immunology* 2008, 20:10-16.

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CAPS and SCAR markers for detection of *I-3* gene introgression for resistance to *Fusarium oxysporum* f. sp. *lycopersici* race 3

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Introduction

Fusarium oxysporum f. sp. *lycopersici* (FOL) race 3 is responsible for Fusarium wilt of tomatoes. It was first detected in Australia in 1979 (Grattidge et al., 1982) and then found in Florida in 1982 (Volin and Jones, 1982). It has also been reported as an important disease in Mexico (Valenzuela-Ureta et al., 1996) and Brazil (Reis et al., 2005). Resistance to FOL race 3 was discovered in an accession from *Lycopersicon pennellii* LA716 (Scott et al., 1989). Mapping of the *I-3* gene on chromosome 7 indicated that it was contained within a 0.3-cM interval between markers CT226 and TG572 (Hemming et al., 2004). Lim et al. (2006) reported the use of the CAPS marker CT226 to detect the *I-3* gene in breeding of FOL race 3-resistant tomato hybrids for Australia.

The goal for this research was to evaluate CAPS markers and to develop a co-dominant SCAR marker for the *I-3* gene introgression. Molecular markers for the chromosome 7 region between the markers TG183 (42 cM) and TG639 (43.3 cM) (Hemming et al., 2004) were evaluated on homozygous susceptible and homozygous resistant tomato inbred lines as well as on heterozygous F1 hybrids. This report evaluates two CAPS markers and a co-dominant SCAR marker linked to the *I-3* gene introgression, which could be used in marker-assisted selection for tomato breeding lines.

Materials and Methods

Primers: Forward and reverse primers were designed from sequences of markers in the chromosomal region between the molecular markers TG183 (42 cM) and TG639 (43.3 cM) (Hemming et al., 2004). Marker sequences (SGN, www.sgn.cornell.edu) were compared with genomic sequences at GenBank and primers were designed from putative exon regions to amplify a genomic region that includes at least one intron.

Primers, P7-43BF1 and P7-43BR1, for a CAPS marker were designed from marker C2_At2g20830 (43 cM), which matched a sequence of *Vitis vinifera*, AM476255.

Forward primer, P7-43BF1: 5'- CAGTCATTATTAACAAATTTCAGGATC G -3'

Reverse primer, P7-43BR1: 5'- TCTGAGCAATACGTCTAGCAGC -3'.

Primers, PTG190F1 and PTG190R1, for a CAPS marker were designed from the RFLP marker, TG190 (36.3 cM for the potato-TXB 1992 map).

Forward primer, PTG190F1: 5'-GCAGTACACTTCTCCTTATCATGTG-3'

Reverse primer, PTG190R1: 5'- AGTTTCAGTAGTTGTTCCAAATTCC-3'

Primers, P7-43DF1 and P7-43DR1, for a co-dominant SCAR marker were designed from marker cTOF-21-J12 (SGN-U321614, mRNA, BT014299, 43 cM), which matched exons in a sequence of *Vitis vinifera*, AM427259.

Forward primer, P7-43DF1: 5'- GGTAAAGAGATGCGATGATTATGTGGAG -3'

Forward primer, P7-43DF3: 5'- CACGGGATATGTTRTTGATAAGCATGT-3'

Reverse primer, P7-43DR1: 5'- GTCTTTACCACAGGAACTTTATCACC -3'.

PCR protocol: DNA was extracted from fresh leaves of plants with MasterPure™ Plant Leaf DNA Purification Kit (EPICENTRE® Biotechnologies, Madison WI), and DNA adjusted to approximately 15 ng/µl. PCR was carried out in 25-µl reactions containing 2.5 µl 2.5 mM dNTPs, 5 µl 10X buffer, 2.5 µl 25 mM MgCl2, 0.1 µl *Taq* polymerase (Promega Corp., Madison WI), 2.5 µl each forward and reverse primer at 10 µM, 2.5 µl of 15 ng/µl DNA extract and H2O. PCR cycler parameters were as follows: denaturation at 94ºC for 3 min, then 35 cycles at 94ºC for 30 sec, annealing at 53ºC for 1 min, and extension at 72ºC for 1 min, followed by 72ºC for 10 min, then the reaction was held at 4ºC. PCR reactions were performed in the MJ DNA Engine PT200 Thermocycler™ (MJ Research Inc., Waltham MA). Amplified fragments were separated by electrophoresis through 1.5% or 2.0% agarose in 0.5X TBE buffer, then stained with ethidium bromide, and visualized with UV light. For sequencing, ssDNA was digested in the PCR reactions with shrimp alkaline phosphatase (Promega Corp.) and exonuclease I (EPICENTRE® Biotechnologies), and the PCR fragments were directly sequenced with Big Dye Sequencing Kit™ and analyzed by the Biotechnology Center, University of Wisconsin-Madison.

The restriction enzyme digestion for the P7-43F1/R1 CAPS marker was a 20 µl reaction mixture containing 13 µl water, 3 µl buffer D, 0.25 µl BSA, 1 µl *Nsi*I (Promega Corp.), and 8 µl PCR reaction mixture. The digestion for the PTG190F1/R1 CAPS marker was similar, using buffer B and *Alu*I (Promega Corp.). The reaction mixture was placed in a 37ºC water bath overnight. Analysis of digestion was completed by electrophoresis through 1.5% agarose in 0.5X TBE buffer, then stained with ethidium bromide, and visualized with UV light.

Germplasm: The cultivars M82-1-8 (H. Czosnek, Hebrew University of Jerusalem) and Purple Russian (a heritage tomato, Seed Savers Exchange, Decorah IA) were the susceptible genotype (*i-3*/*i-3*). L40, an F2 plant from Llanero F1 (resistant to begomoviruses, GenTropic Seeds, *i-3*/*i-3*) and GMh6330 (*i-3*/*i-3*), a begomovirus-resistant inbred line from San Carlos University, were also used as

susceptible controls. Three inbreds, homozygous for resistance to FOL race 3, that have the *I-3* introgression from *S. pennellii* LA716, were used as positive controls: Fla7547 (J. W. Scott, University of Florida), NC-EBR-8 and NC123S (R. Gardner, North Carolina State University). The *I-3* introgression in NC123S was derived from Fla7481, which can be traced to the same F6 family as Fla7547 (J. W. Scott, pers. com.). The commercial F1 hybrid, Plum Crimson (Harris Moran), and NC07196, an indeterminate Roma type hybrid (R. Gardner), were used as known heterozygous genotypes (*I-3*/*i-3*). Experimental F1 hybrids designated as A, B, and C were also used as known heterozygous genotypes for evaluation. Three commercial hybrids, which were listed as being resistant to FOL race 3 by the seed companies, were also tested, as well as eleven inbreds or hybrids, which were reported as either resistant to FOL race 2 or known to have the *I-2* introgression (El Mohatar et al., 2007).

Results and discussion

CAPS markers: The susceptible genotypes (i3/i3), Purple Russian, L40 and M82, and the resistant genotypes (*I-3*/*I-3*), Fla7547, NC-EBR-8, and NC123S, gave PCR fragments of approximately 765 bp with primer pair PTG190F1/R1. The sequences of the PCR fragments from Purple Russian (EU926659) and NC-EBR-8 (EU926658) were compared, and differences of 12 SNPs and three indels were found between the susceptible and the resistant genotypes. A restriction site was identified near one of the indels, corresponding to the enzyme *Alu*I, which would result in fragments of 400, 249, 90 and 60 bp for the resistant genotypes and 401, 165, 143, and 90 bp for the susceptible genotypes. The 249-bp fragment from the resistant and the 165-bp fragment from the susceptible were indicative of the two genotypes (see Fig. 1); the other fragments were not informative. For this reason, this CAPS marker, PTG190F1/R1, was not an improvement over the previously reported CT226 CAPS marker (Lim et al., 2006).

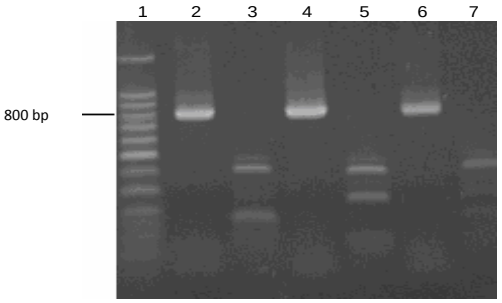

Fig. 1. CAPS marker PTG190F1/R1. PCR fragments digested with *Alu*I for lanes 3 ,5 and 7. Lanes: 1) 100-bp DNA Ladder (Promega Corp.), 2) Purple Russian (*i-3*/*i-3*) -no digestion-, 3) Purple Russian digested, 4) NC-EBR-8 (*I-3*/*I-3*) -no digestion-, 5) NC-EBR-8 digested, 6) F1 hybrid A (*I-3*/*i-3*)-no digestion-, 6) F1 hybrid A digested. Note that the 400-bp fragment of Purple Russian and the 401-bp fragment of NC-EBR-8 are indistinguishable. The 165-bp and 143-bp fragments of Purple Russian appear as one band, and the smaller bands are not distinguishable. Faint bands of all the different sizes were detectable with F1 hybrid A.

Another CAPS marker, P7-43BF1/R1, was evaluated on the same germplasm. The susceptible genotypes, Purple Russian and M82, and the resistant genotypes, NC-EBR-8 and NC123S, gave PCR fragments of approximately 730 bp with primer pair P7-43BF1/R1. The PCR fragments were sequenced and a Blast search was performed at NCBI and SGN. The sequence of M82 (EU926651) matched the chromosome 7 BAC clone C07HBa0045O10 (141,097 nt, AC212615) with 100% nt identity, as did the sequence for NC-EBR-8 (EU926652) with 97% nt identity.

When the sequences of M82 and NC-EBR-8 were compared, there were differences of 16 SNPs and one indel. An *Nsi*I restriction site was associated with one of the SNPs. When PCR fragments were digested with *Nsi*I, the resistant genotype would give two fragments, 362 and 367 bp, and susceptible genotype would not be digested (730-bp fragment). PCR fragments for the heterozygous genotypes were evaluated, and the CAPS marker gave two fragments, approximately 365 bp and about 730 bp, as expected. This CAPS marker was effective for detecting the three different *I-3* genotypes (Fig. 2).

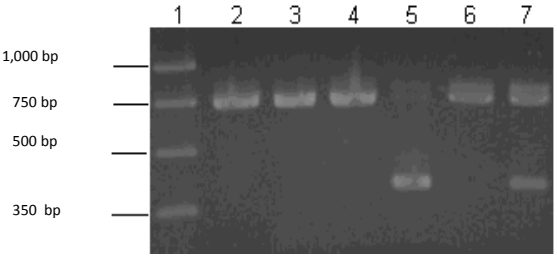

Fig. 2. CAPS marker P7-43BF1/R1. PCR fragments digested with *Nsi*I for lanes 3, 5, and 7. Lanes: 1) PCR Marker (Promega Corp.), 2) Purple Russian (*i-3*/*i-3*) –no digestion-, 3) Purple Russian digested, 4) NC-EBR-8 (*I-3*/*I-3*) -no digestion-, 5) NC-EBR-8 digested, 6) F1 hybrid A (*I-3*/*i-3*) –no digestion-, 7) F1 hybrid A digested. The heterozygous has two bands of 365 bp and 730 bp, as expected.

SCAR marker: The primer pair PTG183F1/R2, which was reported by Hemming et al. (2004), as a co-dominant SCAR marker, was initially evaluated. These primers were useful on those resistant lines that have the longer introgression from *S. pennellii* (Hemming et al., 2004). Results indicated that Fla7547 and NC-EBR-8 have the short introgression, while NC123S has the longer introgression. The sequence for the 1.1-kb PCR fragment from NC-EBR-8 (*S. lycopersicum* sequence, EU926656) was identical to the sequence from the susceptible heritage tomato, Purple Russian (EU926657). The PCR fragment for NC123S was about 800 bp (FJ004839). These results are consistent with those reported by Hemming et al. (see Fig. 1, 2004) in that the NC123S line (origin Fla7481) has the *I-3* introgression. Evaluations of the experimental F1 hybrids A, B, and C, which are heterozygous for *I-3*, resulted in single bands of about 1,100 bp, the same as M82, Fla7547 and NC-EBR-8. Therefore, these primers would not be generally useful in breeding programs using marker-assisted selection for the *I-3* genotypes.

Other primers were designed from additional markers between 42 and 43.3 cM with the objective of continuing the search for a co-dominant SCAR marker. Primer pair P7-43DF1/R1 gave a band of 1,060 bp for the susceptible genotypes and 1,270 bp for the resistant genotypes. Comparison of the sequences of the PCR fragments from the resistant genotype, NC123S (EU926654), and the susceptible genotype, M82 (EU926653), revealed 28 SNPs and six indels, one of which was 215 bp. Primer P7-43DF3 was designed from the sequences upstream of this large indel to give PCR fragment sizes of 875 bp for the resistant and 650 bp for the susceptible genotypes (Fig. 3), when used with P7-43DR1. The sequence from M82 for the P7-43DF1/R1 fragment had 100% nt identity with the chromosome 7 BAC clone C07HBa0045O10 and matches nt 114,068-115,118, which is part of a predicted gene location (gene_16_AGS_323, SGN-E745364).

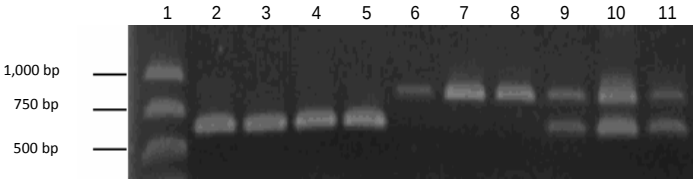

Fig. 3. Co-dominant SCAR marker P7-43DF3/R1. Lanes: 1) PCR Marker (Promega Corp.), 2) M82 (*i-3*/*i-3*), 3) GMh6330 (*i-3*/*i-3*), 4) L40 (*i-3*/*i-3*), 5) Purple Russian (*i-3*/*i-3*), 6) Fla7547 (*I-3*/*I-3*), 7) NC-EBR-8 (*I-3*/*I-3*), 7) NC123S (*I-3*/*I-3*), 8) NC07196 (*I-3*/*i-3*), 9) Plum Crimson (*I-3*/*i-3*), 10) F1 hybrid A (*I-3*/*i-3*).

The primer pair P7-43DF3/R1 was tested on different genotypes, including *I-2*-resistant hybrids and inbred lines, and commercial hybrids with and without resistance to FOL race 3. The 10 inbred lines and hybrids known to have the *I-2* introgression (El Mohatar et al., 2007) all gave the susceptible size fragment for the *I-3* introgression, as was expected. Commercial hybrids, Plum Crimson, Amelia, Crista, and Solar Fire, which are reported to have resistance to FOL race 3, and three experimental hybrids (*I-3*/*i-3*) gave the two fragment sizes indicative of the heterozygous genotype (Fig. 4).

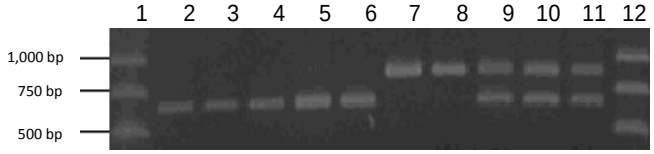

Fig. 4. Evaluation of inbred lines and commercial hybrids with P7-43DF3/R1 SCAR marker. Lanes: 1) PCR Marker (Promega Corp.), 2) inbred G-38 (*i-3*/*i-3*), 3) Gc171-1 (*i-3*/*i-3*), 4) Gc143-2 (*i-3*/*i-3*), 5) Marina (*i-3*/*i-3*), 6) Don Raul (*i-3*/*i-3*), 7) NC-EBR-8 (*I-3*/*I-3*), 8) NC123S (*I-3*/*I-3*), 9) Plum Crimson (*I-3*/*i-3*), 10) Amelia (*I-3*/*i-3*), 11) F1 hybrid A (*I-3*/*i-3*), 12) PCR Marker.

Of particular interest was the relationship of this SCAR marker (P7-43DF3/R1) to the location of the *I-3* gene. Dr. David Jones (Hemming et al., 2004; Lim et al., 2006) was provided with the sequence of the introgression from NC123S before it was available at GenBank, as his research team had published a simplified diagram of the *I-3*-introgression region (see Fig. 3, Lim et al., 2006). His response was as follows: "The P7-43DF3/R1 SCAR marker is outside of the BAC contig covering *I-3*, which Dr. David Jones and his research team at The Australian National University, Canberra has developed, but is estimated to be less than 1 cM from *I-3*" (David Jones, pers. com.). Thus, our evaluations of this SCAR marker with various inbreds and hybrids along with the information from Dr. Jones indicate that this marker, P7-43DF3/R1, is linked to the *I-3* introgression, and can be used as a co-dominant SCAR marker for selection of inbreds with FOL race 3 resistance in tomato breeding programs.

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Genetic analysis of an F_2 population for the segregation of two introgressions associated with the begomovirus-resistant parent, Gc171

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Introduction

Several wild tomato species and their accessions have been used in developing tomato inbred lines with resistance to begomoviruses (see review, Ji et al., 2007c). Resistance in one of these begomovirus-resistant inbred lines, Gc171 (selected from a Florida University line in Guatemala by L. Mejía and D. P. Maxwell, see Scott and Schuster, 2007), was derived from *Solanum chilense*. Two *S. chilense* accessions, LA2779 and LA1932, are in its background. A co-dominant SCAR marker, P6-25F2/R5, for the *Ty-3* introgression on chromosome 6 at 25 cM can distinguish between the introgressions from these two accessions (Ji et al., 2007a). Inbred line Gc171 has the introgression from LA1932. This introgression was given the tentative designation *Ty-3a* (Ji et al., 2007a, 2007b) and the *Solanum lycopersicum* locus was designated *ty-3*. Gc171 has another introgression from *S. chilense* on chromosome 3, designated *Ty-4*, which can be detected with another co-dominant SCAR marker, P3-Ty4F1/R1 (Y. Ji, D. P. Maxwell, and J. W. Scott, unpublished data).

This inbred, Gc171, was selected for at least six generations in Sanarate, Guatemala for resistance to bipartite begomoviruses (Nakhla et al., 2005) and has generally had a very high level of resistance in field trials in Guatemala (L. Mejía and D. P. Maxwell, unpublished data). Since there were two introgressions associated with Gc171, a project was initiated to develop F_3 families that would have only one of the introgressions in order to evaluate the contribution of each introgression to begomovirus resistance.

Materials and Methods

PCR protocol for the co-dominant SCAR markers: The *Ty-3a* introgression on chromosome 6 was detected with the PCR primer pair P6-25F2 and P6-25R5 (Ji et al., 2007). The introgression on chromosome 3, tentatively designated *Ty-4*, could be detected with the PCR primers P3-Ty4F1 and

P3-Ty4R1 (contact J. W. Scott, University of Florida, for the protocol). The different genotypes could easily be distinguished with these two co-dominant SCAR markers.

Germplasm: An F_1 population was created by crossing Gc171 (both introgressions) x Gh44 (neither introgression), and F_2 plants were analyzed for the genotype of the introgressions.

Results and discussion

The 77 F_2 plants were analyzed for each introgression (Table 1). For the *Ty-3a* introgression, the numbers of plants for the genotypes *Ty-3a/Ty-3a*, *Ty-3a/ty-3*, and *ty-3/ty-3* was an acceptable fit to the expected 1:2:1 ratio (X^2 = 5.42, p = 0.05-0.1). The *Ty-4* introgression segregation for genotypes *Ty-4/Ty-4*, *Ty-4/ty-4*, and *ty-4/ty-4* was unacceptable for the fit to the expected ratio of 1:2:1 (X^2 = 38.37, p = <0.005). The numbers of plants in the classes with the *Ty-4* introgression were greatly reduced and the numbers of plants without the *Ty-4* introgression (*ty-4/ty-4*) were greatly increased.

Table 1. Segregation of *Ty-3a* and *Ty-4* introgressions in an F_2 population.

Introgression	Homozygous dominant	Heterozygous	Homozygous recessive	Total	X^2	p value
Ty-3a	17	48	12	77	5.42	0.05-0.1-
Ty-4	10	24	43	77	38.37	<0.005
Expected	19	38	19	1:2:1		

The number of plants for the 9 genotypes for inheritance of two independent markers did not fit the expected ratio (Table 2). The numbers of plants in all genotypes that had the *Ty-4* introgression were fewer than would be expected and the numbers of plants in all genotypes homozygous for *ty-4* were greater than expected. For example, from the 77 plants the genotype *Ty-3a/ty-3*, *ty-4/ty-4* would be expected to have 10 plants, however, there were 25 plants with this genotype.

Table 2. Number of plants for each genotype in the F_2 population.

	AA BB	**AA Bb**	**AA bb**	**Aa BB**	**Aa Bb**	**Aa bb**	**aa BB**	**aa Bb**	**aa bb**	**Total**	**X^2**	**p value**
Observed	2	4	11	5	18	25	4	2	6	77	50.5	<0.005
Expected	5	10	5	10	20	10	5	10	5	75		

A = *Ty-3a*, a = *ty-3*, B = *Ty-4*, b = *ty-4*

In conclusion, it appears that the *Ty-4* introgression decreases the number of viable seedlings and thus must also carry deleterious alleles for gamete viability, seed set or seed germination. Two additional observations support this view: i) Gc171 fruits have few seeds and these seeds have a low percentage of germination (approximately, 60%). ii) The F_1 seeds from the above cross had about 70% germination and few seeds were produced when either parent was used as the female. Since the use of this *Ty-4* introgression can result in problems with seed production and germination, it is therefore critical to determine its contribution to begomovirus resistance.

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Effectiveness of the *Ty-3* introgression for conferring resistance in F3 families of tomato to bipartite begomoviruses in Guatemala

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Introduction

Begomoviruses, whitefly-transmitted geminiviruses, have been the main cause of losses in tomato production in many subtropical and tropical regions. The management of this disease in Guatemala has been difficult because of the high diversity of begomoviruses (Nakhla et al., 2005) and an increase in the vector population, the whitefly, *Bemisia tabaci*. In the last decade, many management practices have been introduced. These include the use of virus-free seedlings and new insecticides. Breeding begomovirus-resistant tomato hybrids has become one of the goals for several seed companies.

Solanum lycopersicum is susceptible to begomoviruses, and resistance has been associated with wild tomato species (Ji et al., 2007c). Resistance genes from *Solanum chilense* and *Solanum habrochaites* have been introgressed into *S. lycopersicum* (Hanson et al., 2000; Scott et al., 1995; Vidavsky and Czosnek, 1998). Ji et al. (2007b) described the begomovirus-resistance locus, *Ty-3*, on chromosome 6 near 25 cM, and a co-dominant SCAR marker (P6-25F2/R5) has been developed for this introgression (Ji et al., 2007a). In Guatemala, a program was initiated at San Carlos University in 1998 to develop tomato breeding lines with resistance to begomoviruses (Mejía et al., 2005). One breeding line, Gh13, was selected from the hybrid, FAVI 9, which was provided by F. Vidavsky and H. Czosnek, Hebrew University of Jerusalem. Molecular marker studies showed that Gh13 had an introgression in chromosome 6 from 20 cM to 32 cM (C. Martin and D. P. Maxwell, unpublished data; Martin et al., 2007) and that this introgression was identical to that for *Ty-3* introgression (Ji et al., 2007b). The introgression associated with the *Ty-1* region was not present in Gh13. Thus, this

begomovirus-resistant inbred, Gh13, provided an opportunity to evaluate the effectiveness of the *Ty-3* introgression to predict resistance to begomoviruses in Guatemala in a field situation were at least seven different bipartite begomoviruses have been identified (Nakhla et al., 2005). Gh13 was crossed with the susceptible genotype M82, and F3 families developed that were homozygous for either *Ty-3* or *ty-3*. These F3 families were generated to reduce the amount of variation, which can be associated with using F2 populations in field experiments.

Materials and Methods

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. The PCR primers for the co-dominant SCAR marker (P6-25F2/R5) were (Ji et al., 2007a):

Forward primer: P6-25F2, 5' - GGTAGTGGAAATGATGCTGCTC - 3'

Reverse primer: P6-25R5, 5' - GCTCTGCCTATTGTCCATATATAAC - 3'

Generation of F3 families: Gh13 (*Ty-3/Ty-3*) was crossed with the susceptible inbred, M82 (*ty-3/ty-3*), and the genotype for the *Ty-3* introgression of the F2 plants determined with the PCR primer pair P6-25F2/F5. Homozygous *Ty-3* and *ty-3* plants were selfed to create F3 families. The phenotype for each F3 family and the parents was evaluated in a field trial in Sanarate, Guatemala. Four-week-old seedlings were transplanted on December 13, 2007, which is near the beginning of the dry season in Guatemala. The *Bemisia tabaci* population in the area was extremely high because of the presence of an old sweet pepper field adjacent to the tomato trial. The symptom incidence for begomoviruses was 100% in the peppers. The bipartite begomoviruses that infect sweet peppers in Guatemala also infect tomatoes (Nakhla et al., 2005). Each plant was evaluated with a disease severity index (DSI) from zero to six at 30 and 42 days after transplanting. 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 ≤ 2.5 were considered resistant, as these would yield marketable fruit. The experimental design was a randomized complete block with five plants per family and three blocks. All families were coded before transplanting to eliminate any bias during scoring each plant by at least two individuals. Eleven F3 families with the homozygous *Ty-3* introgression and 15 families without the introgression (*S. lycopersicum* sequence at the marker site) were transplanted. Also, Gh13 and M82 were coded and included in each block. Susceptible (cv. Silverado) and resistant (cv. Llanero) control hybrids were randomly planted throughout the blocks. Begomoviral symptoms were observed on the susceptible hybrid, Silverado, as early as 10 days after transplanting.

Results and Discussion:

The genotype for the *Ty-3* introgression of the 64 F2 plants was determined. Homozygous *ty-3* and *Ty-3* plants yielded 320-bp and 450-bp fragments, respectively, and heterozygous plants had these two fragments (Fig. 1). The ratio of *ty3/ty3*:*Ty3/ty3*:*Ty3/Ty3* was 18:29:17, which fits the expected ratio of 1:2:1 (x^2=0.59, p= 0.1-0.5). Homozygous plants were allowed to self either in a greenhouse or in a field in Wisconsin. Before transplanting the seedlings to the field, the genotype of the F3 family was confirmed by extracting together 5 plants per family and then completing PCR with the co-dominant SCAR marker. Only those F3 families were transplanted that were homozygous for either *Ty3* or *ty3* genotypes.

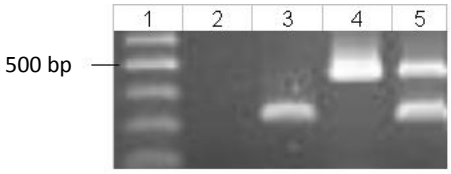

Fig 1. PCR fragments for the P6-25F2/R5 primers. Lane 1) Promega 100-bp marker; 2) No target DNA; 3) M82 (*ty-3/ty-3*); 4) Gh13 (*Ty-3/Ty-3*); 5) heterozygous line (*Ty-3/ty-3*).

The DSI scores at 42 days after transplanting were averaged for each entry for each block and analyzed with the least squares estimation of the means was obtained using SAS Software version 9.1.3 (SAS Institute Inc., Cary NC). The least square means for the DSI's for those F3 families with the *Ty-3* introgression and those without the *Ty-3* introgression were 2.0 and 4.5, respectively (Table 1, Fig. 2). One F3 family, number 3, with an introgression had a mean DSI of 3.2 and had both resistant and susceptible plants. The mean DSI for the families with an introgression (2.0) was greater than the mean for the resistant parent, Gh13 (0.9), and the mean DSI for the families without the introgression (4.5) was less than the mean for the susceptible parent, M82 (6.0). This indicates

that other genes besides those of the *Ty-3* introgression were influencing the expression of the phenotypes.

Table1. Least squares means for the F3 families with the homozygous genotypes for the introgression (*Ty-3/Ty-3*) and no introgression (*ty-3/ty-3*).

Marker Genotype	LS Mean	Standard Error	P value of the difference between means
Ty-3/Ty-3	1.96	0.13	< 0.0001
ty-3/ty-3	4.49	0.11	

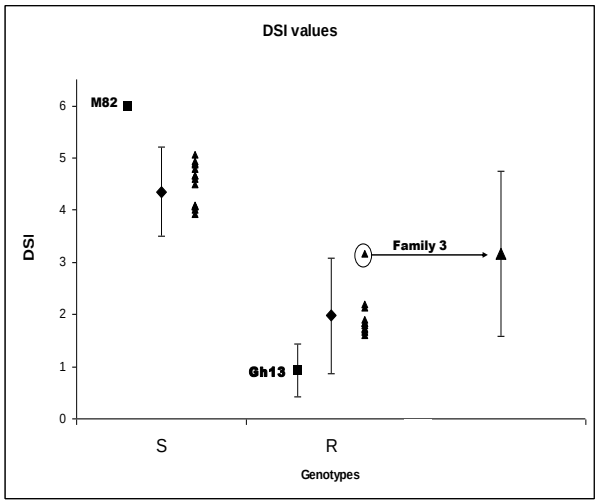

Fig. 2. Disease severity index (DSI) values (0 to 6) of the means of each family (▲), all families (♦) in a class, and resistant (Gh13) and susceptible (M82) parents (■). Bars indicate one standard deviation around the mean. All plants for M82 had a DSI = 6, so there is no bar for the mean. R = 11 F3 families with *Ty-3* introgression and S = 15 F3 families without the introgression for *Ty-3*. Family 3 = one F3 family, number 3, which had the introgression, but also had resistant and susceptible plants.

The means were also analyzed with a two-way ANOVA that allowed for different variances for each genotype, and included effects of genotype, block, and the block by genotype interaction. The model was fit using PROC MIXED of the SAS Software. The ANOVA test indicates that the *Ty-3* introgression (P6-25F2/R5 marker) genotype significantly explains the DSI values for the resistant or susceptible phenotypes ($p<0.0001$). There was no significant contribution of the block ($p=0.0953$) or the genotype by block interaction ($p=0.9291$).

Since this experiment was conducted in the field in Sanarate, Guatemala during the dry season, there are many factors that can influence the DSI for each plant. Some of these factors are

the time of infection of the plants, the possibility for infection of plants by different begomoviruses (Nakhla et al., 2005) or by mixed begomoviral infections, and the variation in the field conditions associated with each plant and block. Regardless of these factors, which are expected to cause variation in the DSI's for each plant, 85% of the variation among the F3 families was explained by this SCAR marker (Table 2). This number also includes a family, number 3, with a putative recombination, which decreases the percentage of variation explained by the marker. These results are consistent with the similar observation by Ji et al. (2007b), where the *Ty-3* introgression was a major contributor to resistance to *Tomato yellow leaf curl virus* in an F2 mapping population. Thus, this introgression has a major effect on begomovirus-resistance, and the molecular SCAR marker (P6-25F2/R5) can be used for predicting begomovirus-resistance in a tomato breeding program.

Table 2. Influence of different factors on the observed phenotypic variance.

Source	Variance	Percentage of variance
Marker Genotype	3.18	85.1%
Block	0.05	1.2%
Interaction Genotype*Block	-0.05	-1.2%*
Error	0.55	14.9%

Since the field in Guatemala can have more than seven bipartite begomoviruses present Nakhla et al., 2005), it is expected that the resistance locus/loci that are associated with the *Ty-3* introgression may be effective in other regions of the world. This is supported by the observation that FAVI 9, which has the *Ty-3* introgression, exhibited resistance to monopartite begomoviruses in Israel, India (Maruthi et al., 2003) and South Africa (Pietersen and Smith, 2002) and that breeding lines with begomovirus-resistance traced to the same source of resistance as Gh13 were resistant in Jordan, Lebanon, Egypt and Morocco (D. P. Maxwell, unpublished data). Also, this co-dominant SCAR marker for the *Ty-3* introgression will facilitate the pyramiding of begomovirus-resistance genes from different sources (Favi, 2007).

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***Ty-4*, a *Tomato Yellow Leaf Curl Virus* Resistance Gene on Chromosome 3 of Tomato**

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Introduction

Genes resistant to *Tomato yellow leaf curl viruses* (TYLCV), such as *Ty-1* and *Ty-2*, have been used for marker-assisted selection in tomato breeding programs worldwide. We recently mapped a third begomovirus resistance gene *Ty-3*, which originated from several *S. chilense* accessions, on the long arm of chromosome 6 (Ji *et al.* 2007a). *Ty-3* contributes a large portion of the begomovirus resistance, but additional gene(s) are required to obtain the highest levels of resistance. Efforts have been taken to search for other potential begomovirus resistance loci by screening advanced resistant breeding lines derived from various *S. chilense* accessions with molecular markers dispersed in the whole tomato genome. Two introgressions were found in the majority of advanced resistant breeding lines derived from crosses of lines with resistance from *Solanum chilense* accessions LA2799 combined with LA1932 (LA2779/LA1932), one on chromosome 6 carrying *Ty-3* and the other on chromosome 3 spanning markers from C2_At1g02140 to TG599, which was also associated with TYLCV resistance. A new TYLCV resistance locus, herein designated as *Ty-4*, was mapped to the marker interval between C2_At4g17300 and Ct_At5g60160 in the introgression on chromosome 3 (Ji et al. submitted).

Material and Methods

Advanced breeding lines derived from *S. chilense* accessions LA2779, LA1932, LA2779/LA1932, LA1938 combined with 'Tyking' (a TYLCV resistant hybrid probably derived from *S. peruvianum (Ji et al. 2007b)*; designated as LA1938/Tyking), that displayed a high level of resistance to both TYLCV and tomato mottle virus (ToMoV) were screened with PCR-based markers on the 12 tomato chromosomes to identify *S. chilense* introgressions in tomato genome. An F_7 line, 040980-3, derived from a cross between susceptible *S. lycopersicum* lines 7655B and a begomovirus resistant line 000529, has both accessions LA2779 and LA1932 in its pedigree. Line 040983-3 was heterozygous for *S. chilense* introgressed segments on chromosomes 6 and 3, respectively. A segregating population from this heterozygous plant was employed to map the resistance loci. The wild *S. chilense* accessions were obtained from the Tomato Genetics Resource Center at UC-Davis, California.

Results and Discussion

PCR-based markers were used to screen breeding lines derived from three *S. chilense* accessions, which confer high levels of resistance to both TYLCV and ToMoV. Previous studies on chromosome 6 found a large introgressed segment (approx. 27 cM in length) in the LA2779-derived lines, but a smaller introgression about ~6 cM in LA1932-derived lines (Ji *et al.* 2007a). The present study showed that early (less advanced lines with fewer backcrosses from *S.* chilense) breeding lines such as 960719 and 960744 derived from LA1932 carry two additional introgressions: one is ~35 cM spanning markers from TG472 to the *sp* gene and TG275 on the long arm of chromosome 6, and the other is ~14 cM spanning markers from C2_At1g02140 to TG599 on the long arm of chromosome 3, while the early LA1932-derived line 960729 carries only the latter additional introgression. Advanced breeding lines derived from LA2779/LA1932, including line 040980 used for segregation analysis, carry the same introgressions on chromosomes 3 and 6 as did its ancestor line 960729. Both introgressed segments in these lines originated from LA1932.

The segregating population derived from 040983-3 was used to investigate the association of resistance with the introgressions. General linear model analysis indicated that all the markers contained within the *S. chilense* introgressions on both chromosomes 3 and 6 showed significant association with the mean disease severity ratings for the segregating population. Molecular linkage maps of these PCR-based markers in the introgression regions on chromosome 3 and 6 were constructed using MapMaker v3.0 with a threshold LOD score of 3.0. *Ty-3* was mapped to the marker interval between cLEG-31-P16 (20 cM) and C2_At5g41480 (26 cM) on the long arm of chromosome 6, which is consistent with the previous report (Ji *et al.* 2007a). A new locus, designated as *Ty-4*, was mapped to the marker interval between C2_At4g17300 (81 cM) and C2_At5g60610 (83.3 cM) on the long arm of chromosome 3. Approximately 60% of the variance in the TYLCV resistance in the segregating progeny was explained by the *Ty-3* locus, while the *Ty-4* locus accounted for only ~16% of the variance, suggesting *Ty-3* had a major effect on resistance, while *Ty-4* had a lesser effect. We will be monitoring the detrimental fecundity effects that have been reported with *Ty-4* (Garcia et al., 2008) as we work with this germplasm. With the population derived from 040984-3, a more recently derived line, we did not find a deficiency of *Ty-4* plants (Ji et al., submitted). We will compare introgression of Gc 171 with this material.

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Pollen production and efficiency of pollination and fertilization in tomato.

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Studies are limited on the efficiency of pollination and fertilization in many crops including tomato. I spent considerable time on this topic as part of my Ph.D. dissertation but never did get around to publishing the information in a refereed journal. With this report I hope to make some amends for this. Although the information was obtained about 30 years ago there has not been a lot of new information in this area. Zamir and Jones, (1981) pollinated male-sterile line (*ms-10*^{35}) and using two different methods estimated that there were about 5,000 or 10,000 pollen grains per stigma. The seed produced per fruit was 90, thus about 2% of the pollen grains per stigma effected fertilization. They pointed out that gametes may not effect fertilization randomly and that natural selection during pollen germination and tube growth may favor particular pollen genotypes. The present work examined total pollen produced-both dehisced and not dehisced, pollen per stigma, and seed production for several genotypes to estimate the efficiency of the entire process.

Materials and Methods

Details of the procedures used are given elsewhere (Scott, 1978) but the main points will be summarized here. Data from two greenhouse experiments will be presented. In one the genotypes were Ex-3, an inbred with the stigma exerted beyond the anther cone by 3mm, InA-3.5 with stigma within the anther cone in the sterile tissue area, and InA-5.5 with a short style and the stigma about 3mm proximal to the sterile anther tissue. In the second experiment an inbred selected for improved fruit set with minimal pollination 1811-2 , ‘Ohio MR13’, and reciprocal hybrids between these two were grown. There were two pollination treatments in this experiment. For treatment 1 flowers were pollinated by vibration with an electric vibrator while in treatment flowers were pollinated by tapping the trellis wire with a metal rod three times at 5 M intervals in the row. Vibration treatments were done before the tapping to prevent pollen loss due to tapping. All the genotypes had 4 plants each planted in completely randomized design within each experiment. The indeterminate plants were pruned to a single stem and tied to cord attached to a trellis wire. Two flowers per plant were tagged at 1 day before anthesis and pollinated with a vibrator, or by tapping the trellis wires for treatment 2 in experiment 2, over the next 3 days. Pollen was collected in gelatin capsules. These were attached to

the plants with small gauge wires when not the first flower per plant vibrated or in the case of tapping the trellis wire. Twenty-six hours after the last pollination day, the anthers and styles were excised. Seed was extracted from ripe fruit and counted. To determine pollen not dehisced anthers were placed in well plates with 1ml of dH_2O containing 0.10% Tween 20. A rubber piston of a syringe was used to crush the anthers and release the pollen (Nitsch, 1977). Pollen dehisced was rinsed from the gelatin capsules into small bottles using 1ml of the above solution. Samples were all stored in a refrigerator until ready for observation. To do this the vials were shaken to suspend the pollen and the suspension was immediately pipetted to a hemacytometer where 5 $0.1mm^3$ squares were counted for each of the two grids. This procedure was repeated twice so 20 $0.1mm^3$ squares total were counted for each flower using a light microscope at 43x. The total pollen counted ($2mm^3$) was divided by 2 and multiplied by 1000 to give pollen/ml and thus pollen/sample. To obtain pollen per stigma the excised styles were placed in vials and a procedure adapted from Martin (1959) and Kho (1968) was used to observe the pollen per stigma. The cleared, softened, aniline blue stained styles placed on a slide in a drop of aniline blue and a cover glass was placed on top. The style was squashed by gently tapping on the cover glass with the blunt end of a dissecting needle. Observations were made at 100x with a Leitz microscope equipped with a mercury burner HBO 200 type bulb and a blue exciter filter and with reflected light. Pollen grains on the stigma were counted by focusing up and down while scanning slowly.

Results and Discussion

In this type of experimentation small errors in pollen counts can be amplified as mm^3 fields are extrapolated to the total sample. Despite this great care was used to minimize the experimental error and considerable sampling was done to provide data that was realistic. For this presentation the focus will be on overall values and not so much on individual genotypic differences. For the heterostyly experiment approximately 180,000 pollen per flower were produced with nearly 60% being dehisced (Table 1). An average of 245 pollen reached the stigma which is 0.14% and 0.23 % of the pollen produced and dehisced, respectively. The seeds per fruit for the three lines averaged 106 so about 43% of the pollen per stigma resulted in seed. Results for the vibration treatment of the second study are in Table 2. These genotypes averaged 197,000 pollen per flower with about 67% being dehisced. An average of 387 pollen grains reached the stigma which is 0.2% and 0.3% of the pollen produced and dehisced, respectively. Overall there were 124 seeds per fruit so approximately 32% of the pollen per stigma resulted in seed. ‘Ohio MR-13’ had less seed per fruit than the hybrid with 1811-2 as the seed parent.

In contrast, Zamir and Jones (1981) estimated between 5,000 and 10,000 pollen per stigma when they actually dipped the stigmas into capsules of pollen. Part of this discrepancy could relate to

greater stigma coverage when dipping as opposed to landing by gravity with vibration. There also may be more of a tendency for pollen to clump to each other with dipping as opposed to being vibrated. In my work some pollen per stigma may have been lost in the style fixing treatments although when some vial solutions were checked such pollen was not found. Other differences could be due to the different genotypes used where the shape and size of different stigmatic surfaces could have a large effect on the amount of pollen that sticks to them. Given the large differences in pollen per stigma from the two studies it logically follows that Zamir and Jones (1981) found only 2% of the pollen was able to effect fertilization while I found more like 30-40%. When pollination and fertilization conditions are good the efficiency of pollination and fertilization is probably not too critical. However, the efficiency may be more important under stress conditions. Table 3 is a comparison of the vibration and tapping trellis wire pollination treatments. Much less pollen was dehisced with tapping than with vibration. Thereafter, tapping resulted in 121 pollen per stigma, 51 seeds per fruit and 94g fruit size all of which were significantly less respectively than the 387 pollen per stigma, 124 seeds per fruit and134g fruit size for the vibration treatment. Interestingly, with vibration 32% of the pollen per stigma resulted in seed while with tapping 41% of the pollen per stigma resulted in seed. Although fertilization with tapping may have been a little more efficient, it was not nearly enough to result in seed production and fruit size equal to the vibration treatment. Thus, the fertilization process seems rather inefficient based on these results.

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Table 1. Pollen production and efficiency of pollination and fertilization for heterostylous tomato lines from the greenhouse pollination study in Columbus, Ohio.

	1) Pollen/flower	2) Pollen dehisced/flower		3) Pollen not dehisced/flower		4) Pollen/stigma			5) Seed/fruit			
Line[z]	#[y]	#[y]	% of (1)	#[x]	% of (1)	#[w]	% of (1)	% of (2)	#[w]	% of (1)	% of (2)	% of (4)
1	171,890	120,438	70.1	51,188	29.8	301.6	0.16	0.25	123.5	0.07	0.10	41.0
2	206,102	118,125	57.3	87,750	42.6	259.3	0.13	0.22	118.0	0.06	0.09	45.0
3	165,900	83,937	50.6	81,750	49.3	173.9	0.10	0.21	76.5	0.05	0.09	44.0
$\bar{X}$	181,276	107,500	59.3	73,563	40.6	244.9	0.14	0.23	106.0	0.06	0.10	43.3

[z] Line 1 = Ex-3, Line 2 = InA-3.5, and Line 3 = InA-5.5.
[y] Means in column not significantly different by direct comparisons with Student's t test at the 5% level.
[x] Line 1 significantly different from lines 2 and 3 by direct comparisons with Student's t test at the 1 and 5% levels respectively.
Lines 2 and 3 not significantly different at the 5% level.
[w] Line 1 significantly different from line 3 by direct comparison with Student's t test at the 5% level.
No other lines were significant, at the 5% level.

Table 2. Pollen production and efficiency of pollination and fertilization for parents and hybrids of greenhouse tomatoes pollinate by vibrating the clusters Columbus, Ohio.

	1) Pollen/flower	2) Pollen dehisced/flower		3) Pollen not dehisced/flower		4) Pollen/stigma			5) Seed/fruit			
Line[z]	#[y]	#[y]	% of (1)	#[x]	% of (1)	#[w]	% of (1)	% of (2)	#[w]	% of (1)	% of (2)	% of (4)
1	177,024	111,563	63.0	65,125	36.8	384.0	0.22	0.34	120.8 ab	0.07	0.11	31.5
2	187,830	124,400	66.2	63,100	33.6	329.8	0.18	0.27	93.4 b	0.05	0.08	28.3
3	219,673	155,250	70.7	64,000	29.1	422.5	0.19	0.27	153.0 a	0.07	0.10	36.2
4	205,260	138,833	67.6	66,083	32.2	412.2	0.21	0.31	126.7 ab	0.06	0.09	30.7
$\overline{X}$	197,447	132,512	66.8	64,577	32.9	387.1	0.20	0.30	123.5	0.06	0.095	31.7

z Line: 1) = 1811-2, 2) = Ohio MR-13, 3) = 1811-2 x Ohio MR-13, and 4) = Ohio MR-13 x 1811-2.
y Means in column not significantly different by direct comparisons with Student's t test at the 5% level.
x Mean separation in column by direct comparison with Student's t test at the 5% level.

Table 3. Comparison of pollination method on pollen, seed and fruit size parameters in greenhouse parent and hybrid lines (Columbus).[z]

Pollination method	Total pollen/flower	Pollen dehisced/flower	Pollen not dehisced/flower	Pollen/stigma	Seed/fruit	Fruit size (g)
Vibration of clusters	197,446 ± 11,144	132,511 ± 8,849	64,577 ± 8,140	387.1 ± 27.5	123.5 ± 9.5	134.2 ± 7.1
Tapping trellis wires	166,316 ± 10,891	5,964 ± 8,647	160, 250 ± 7,955	121.3 ± 29.6	50.6 ± 9.3	93.5 ± 6.2

[z]All means in columns significantly different by the F test at the 5% level except for the Total pollen/flower column which is significant at the 6% level.

A new source of resistance to *Tomato spotted wilt virus* (TSWV) from *Solanum habrochaites*

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Introduction

Tomato spotted wilt virus (TSWV) is still causing serious losses on tomato crops in several continents, mainly in North America (USA) (Riley and Pappu, 2004), in South America (Argentina) (Borbon *et al*. 2006), in Asia (Gera *et al*. 2000), and in Europe, in particular in Mediterranean countries. In this last region, the disease causes important losses in Spain (Roselló *et al*., 1999), Italy (Parrella and Crescenzi, 2005), France (Marchoux *et al*., 2000) and Greece (Chatzivassiliou *et al*., 2000).

Genes from *Solanum peruvianum* offer the highest protection level against TSWV. *Sw-5* gene (Stevens *et al*. 1992) has shown a high resistance level in artificial transmission, both mechanically and by thrips, and in field trials (Díez *et al*. 1995, Moury *et al*. 1997). However, this resistance is partially overcome when a high inoculation pressure occurs with thrips transmission (Díez *et al*. 1995), or completely broken down by highly virulent isolates (Latham and Jones 1998). This type of isolates has been identified in Catalonia (Spain) (Aramburu and Martí, 2003). Consequently, it is very important to identify new resistance sources to TSWV.

Material and methods

Some accessions resistant to isolates of *Tomato spotted wilt virus* (TSWV), that did not overcome the resistance of the Sw-5 gene, were identified in previous experiments (Roselló *et al*. 1999). These accessions were UPV-32 and Uco Plata from *S. lycopersicum*, ECU-523 from *S. habrochaites*, and PI-126944 and PI-126935 from *S. peruvianum*. Twenty-four plants of each accession plants were tested against the isolate 'Grau', which overcomes the Sw-5 resistance. Inoculum was prepared by grinding infected tissue of susceptible infected NE-1 tomato plants in 0.1 M phosphate buffer, pH 7.0, containing 0.2% sodium sulfite and 0.2% sodium diethyldithiocarbamate in a proportion of 1:5 (w:v). Carborundum (600 mesh) was added at a concentration of 1%. A cotton-tipped applicator dipped in inoculum and lightly rubbed on the third expanded leaf of the fourth leaf stage plants delivered the inoculum. Plants were scored visually for TSWV symptoms and tested using DAS-ELISA with BR-01 antiserum at 15, 30, 45 and 60 days post inoculation (DPI). The absorbance, measured by a Titertek Multiskan MCC/340 photometer (405 nm), was considered as an estimator of viral accumulation. Fortuna-C and NE-1 from *S. lycopersicum* were included as susceptible control.

In order to study the genetic control of the ECU-523 resistance to TSWV, four generations were obtained between the resistant accession and the susceptible control NE-1: F1 (NE-1xECU-

523), BC1 (NE-1x(NE-1xECU-523)), BC2 ((NE-1xECU-523)xECU-523) and F2 (NE-1xECU-523). Forty-four plants of NE-1, 25 of ECU-523, 30 of the F1, 145 of the BC1, 100 of the BC2 and 101 of the F2 were tested.

Results and discussion

Yellow spots were observed at 15 DPI in the inoculated leaves of the controls NE-1 and Fortuna-C. At 30-45 DPI, bronzing and curling of the leaflets were observed in the newly developed leaves. Throughout the assay the symptoms became more acute, particularly bronzing, and all susceptible control plants became systemically infected (Table 1).

All plants from the accession Uco Plata showed systemic infection (Table 1). Nevertheless, at 60 DPI, only 31.6% of the Uco Plata plants were systemically infected. Viral accumulation in these materials, measured by the maximum absorbance, was 48% of the accumulation of the susceptible control NE-1. In line UPV-32, 16.7% of the plants were not infected. Systemically infected plants of this accession showed 86% of the NE-1 viral accumulation. Accessions PI-126944 and PI-126935 showed 10% and 20% of resistant plants, respectively. Susceptible plants of these accessions showed similar viral accumulations than the susceptible control Fortuna-C. At 15 DPI, ECU-523 showed 40% of systemically infected plants. Nevertheless, at 30 DPI these plants recovered and did not show systemic infection and no symptom, similarly to what was reported by Soler *et al*. (1998) in *S. habrochaites*.

In the genetic control assay, all NE-1 plants were susceptible (Table 2). ECU-523 showed the same performance that in previous assay. Although, at 15 DPI some plant showed systemic infection, subsequently, these plants recovered and did not present symptoms. All F1 plants became infected at 15 DPI. Nevertheless, at 60 DPI, only 26.7 % of the plant showed systemic infection, and no plant had symptoms. In 32 plants (31.7%) of the F2, a susceptible reaction to mechanical inoculation was observed. The other plants did not show systemic infection (resistant), or recovered at the end of the assay. These data, taken together, indicate that the ECU-523 resistance is monogenic and dominant. The results in BC1 and BC2 (Table 2) confirm this suggested genetic control for the resistance in this accession of *S. habrochaites*.

Table 1- Response to mechanical inoculation with isolate 'Grau' of different accessions resistant to isolates of TSWV that not overcome the *Sw-5* resistance gene.

Accession	Mean max. absorbance of positive plants[1]	Mean max. symptoms index of positive plants[2]	Absorbance index[3]	Percentage of systemically infected plants[4]
ECU-523	0.197	0.25	0.07	40
Uco Plata	1.306	2.28	0.48	100
PI-126935	1.84	1.31	0.68	80
Fortuna-C	1.86	2.31	0.69	100
PI-126944	1.882	1.11	0.7	90
UPV-32	2.336	2.0	0.86	83.3
NE-1	2.7	3.2	1.0	100

[1]Mean of the maximum DAS-ELISA absorbance (405 nm) shown by plant with systemic infection during all the assay.
[2]Mean of the maximum index of symptoms (range: 0, symptomless plant; 4, dead plant) shown by the plant with systemic infection during all the assay.
[3]Calculated as (mean max. absorbance of accession / mean max. absorbance of NE-1)
[4]Plants considered DAS-ELISA positive or with systemic infection when absorbance of sample from the youngest leaf was higher than the mean absorbance of non inoculated plants plus three times its standard deviation.

Table 2- Response of different generations of NE-1 x ECU-523 family to mechanical inoculation with the 'Grau' isolate, which overcomes the Sw-5 resistance gene.

Generation	Number of inoculated plants	Resistant plants[1]	Susceptible plants[2]	Expected (R:S) ratio[3]	Prob. χ^2
NE-1	44	0	44	0 : 1	-
ECU-523	25	25	0	1 : 0	-
F1 (NE-1 x ECU-523)	30	30	0	1 : 0	-
F2 (F1 X F1)	101	69	32	3 : 1	0.121
BC1 (NE-1 x F1)	145	73	72	1 : 1	0.933
BC2 (F1 x ECU-523)	100	100	0	1 : 0	-

[1]Symptomless plant.
[2]Plant DAS-ELISA positive with symptoms.
[3]R=resistant; S=susceptible.

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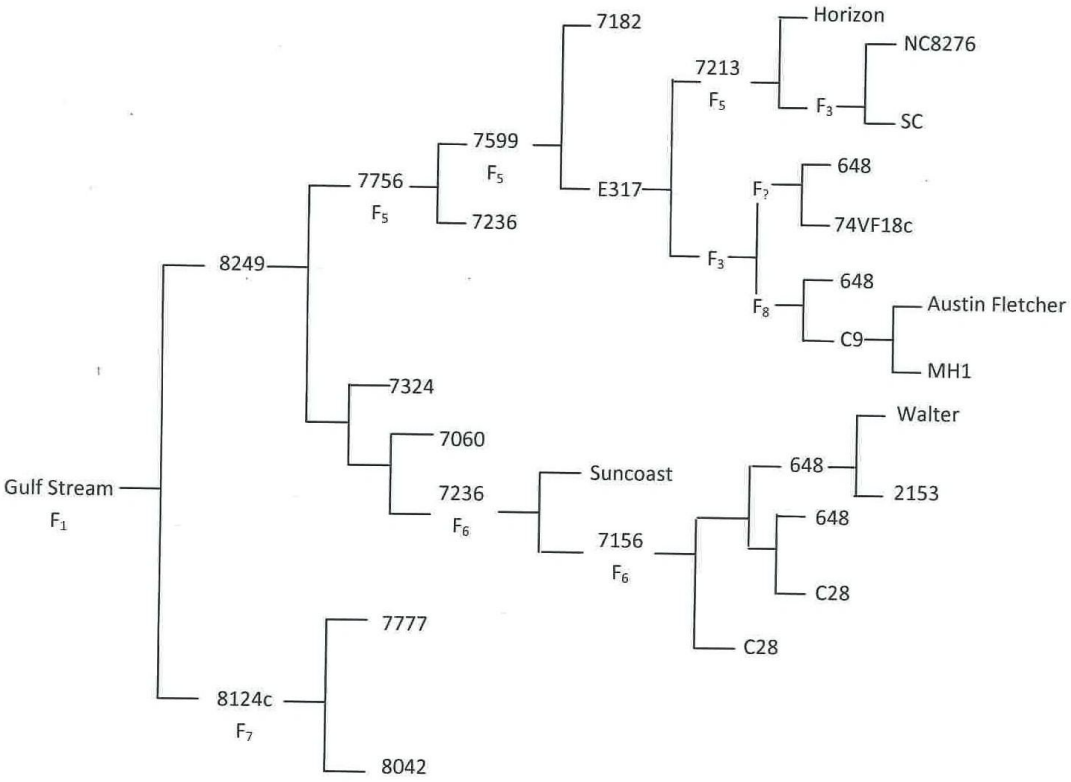
Varietal Pedigrees

Gulf Stream hybrid tomato; Fla. 8124C and Fla. 8249 breeding lines.
J.W. Scott, S.M. Olson, and J.A Bartz. 2007.

Pedigree:



Characteristics:

Fruit: Medium-large, deep flat round shape, light green shoulders, smooth blossom scar, firm. Fla. 8124 has large globe shaped fruit, Fla. 8249 has medium sized, flat round fruit with nippled blossom scars (*n-4*)

Plant: *sp, I, I-2, Ve/+, Sm, Sw-5*, medium tall vine with good leaf cover.

Utility and maturity: Fresh market hybrid with tomato spotted wilt virus resistance and heat-tolerant fruit setting (>32^{o}C day/>21^{o}C night), adapted to SE USA and Turkey, early production under high temperatures, early-midseason under lower temperatures. Fla. 8124 is the source of *Sw-5* and *Ve* and has moderate heat-tolerance. Fla. 8249 is the main source of heat-tolerance

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Revised List of Monogenic Stocks

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The following catalogue lists 1,023 monogenic mutants, allozyme markers, disease resistance genes and other types of stocks at 625 putative genetic loci maintained by the TGRC at UC-Davis. This is a revision of the previous list, issued in TGC 55. Lists of available wild species and miscellaneous genetic stocks were last updated in TGC 56 and TGC 57, respectively. Certain obsolete or inactive items have been deleted, newly acquired stocks have been added, inaccuracies corrected, and gene symbols revised to reflect allele tests or other information. This stock list includes only accessions we consider to be the primary sources for individual mutations: usually the original stock in which the mutation was first described, as well as any nearly isogenic lines into which it has been bred. Most mutant stocks are homozygous and true-breeding. However, seed of the male-steriles, homozygous-inviable mutants, and other stocks that are difficult or impossible to maintain as homozygotes, must be propagated via heterozygotes. In these cases, seed are provided in the form of segregating F_2 or BC populations. Note: some accessions may be temporarily unavailable during seed regeneration.

Monogenic mutants acquired since the last edition of this stock list included the phytochrome mutant *phyB2* and cryptochrome mutant *cry-1*, both donated by Maarten Koornneef, and *ec* (exserted carpels) donated by Ernie Kerr.

Documented cases of allelism between mutants are incorporated into this list, and gene symbols revised accordingly. The mutant *Nr-2* (Neverripe-2) was determined by Cornelius Barry (Pl. Physiol. 138: 267-275) to be allelic to *Gr* (Green-ripe), thus is herein designated $Gr^{Nr\text{-}2}$.

Additional information on individual stocks, including phenotypes, references, images, chromosomal locations, etc., can be obtained through our website (http://tgrc.ucdavis.edu). We ask that users report any problems they detect in our lines, such as aberrant segregation, incorrect phenotypes, unexpected variability, etc. TGC members are also encouraged to submit stocks of verified monogenic mutants not listed here to the TGRC for maintenance and distribution.

Table 1. List of monogenic stocks, ordered by gene symbol. For each locus, stocks containing the original mutant allele are listed first, followed by any additional alleles at the same locus ('prov' indicates a provisional allele). Older gene symbols (synonyms) for each allele are listed ('^' indicates superscript). Each mutant is assigned to one or more phenotypic categories (Class), defined in Table 2 ('*' indicates the primary category for each allele). Background genotypes (Back.) of each stock are listed in abbreviated form, with full names given in Table 3. The origin of each mutation is specified as either spontaneous ('SPON'), or induced by chemical treatment ('CHEM') or irradiation ('RAD'). Isogenicity (Iso.) indicates whether the nonmutant control is available as an isogenic ('IL') or nearly isogenic ('NIL') line, or is nonisogenic ('NON').

Gene	Allele	Locus name	Synonyms	Class	Origin	Back.	Iso.	Accession
a		anthocyaninless	*a1*	A*	SPON	AC	NIL	LA3263
a		anthocyaninless	*a1*	A*	SPON	X	NON	LA0291
a	*prov2*	anthocyaninless	*a*	A*	CHEM	VF36	IL	3-414
a	*prov3*	anthocyaninless	*a*	A*	CHEM	VF36	IL	3-415
aa		anthocyanin absent		A*	SPON	MD	IL	LA1194
aa		anthocyanin absent		A*	SPON	AC	NIL	LA3617

Gene	Allele	Locus name	Synonyms	Class	Origin	Back.	Iso.	Accession
Abg		Aubergine		P*	SPON	X	NON	LA3668
abi		aborted inflorescence		M*	CHEM	CSM	NON	3-803
Aco-1	*1*	Aconitase-1		V*	SPON	pen	NON	LA2901
Aco-1	*3*	Aconitase-1		V*	SPON	pim	NON	LA2903
Aco-2	*2*	Aconitase-2		V*	SPON	chm	NON	LA2905
acr		acroxantha	*acr1*	D*JK	RAD	CR	IL	LA0933
ad		Alternaria alternata resistance		Q*	SPON	X	NON	LA1783
Adh-1	*1*	Alcohol dehydrogenase-1		V*	SPON	VCH	NON	LA2416
Adh-1	*2*	Alcohol dehydrogenase-1		V*	SPON	par	NON	LA2417
Adh-1	*n*	Alcohol dehydrogenase-1		V*	CHEM	MM	IL	LA3150
Adh-2	*1*	Alcohol dehydrogenase-2		V*	SPON	hir	NON	LA2985
adp		adpressa		K*J	RAD	CR	IL	LA0661
adp		adpressa		K*J	RAD	AC	NIL	LA3763
adu		adusta	*adu1*	H*K	RAD	CR	IL	LA0934
ae		entirely anthocyaninless	*a332*	A*	RAD	AC	NIL	LA3612
ae		entirely anthocyaninless	*a332*	A*	RAD	KK	IL	LA1048
ae		entirely anthocyaninless	*a332*	A*	RAD	CG	NIL	LA3018
ae	*2*	entirely anthocyaninless		A*	CHEM	UC82B	IL	3-706
ae	*afr*	entirely anthocyaninless	*afr, ap*	A*	RAD	CT	IL	LA2442
ae	*prov3*	entirely anthocyaninless	*ae*	A*	CHEM	VCH	IL	3-620
aeg		aegrota		H*	RAD	CR	IL	LA0537
aer		aerial roots		R*	SPON	X	NON	LA3205
aer-2		aerial roots-2		R*	SPON	X	NON	LA2464A
af		anthocyanin free	*a325*	A*I	RAD	AC	NIL	LA3610
af		anthocyanin free	*a325*	A*I	RAD	RCH	IL	LA1049
afe		afertilis	*afe1*	N*CJK	RAD	RR	IL	LA0935
afl		albifolium	*af*	B*G	SPON	XLP	IL	2-367
afl		albifolium	*af*	B*G	SPON	AC	NIL	LA3572
Aft		Anthocyanin fruit	*Af*	P*	SPON	X	NON	LA1996
ag		anthocyanin gainer		A*	SPON	GS5	NON	LA0177
ag		anthocyanin gainer		A*	SPON	AC	NIL	LA3163
ag	*2*	anthocyanin gainer		A*	SPON	AC	NIL	LA3164
ag	*2*	anthocyanin gainer		A*	SPON	che	NON	LA0422
ag	*k*	anthocyanin gainer		A*	SPON	T5	IL	LA3149
ag	*s*	anthocyanin gainer		A*	SPON	X	NON	LA4425
ag-2		anthocyanin gainer-2		A*	SPON	AC	NIL	LA3711
ah		Hoffman's anthocyaninless	*ao, a337*	A*	SPON	OGA	IL	LA0260
ah	*prov3*	Hoffman's anthocyaninless	*ah*	A*	CHEM	VCH	IL	3-607
ah	*prov4*	Hoffman's anthocyaninless	*ah*	A*	CHEM	VCH	IL	3-628
ah	*prov5*	Hoffman's anthocyaninless	*ah*	A*	CHEM	VCH	IL	3-629
ah	*prov6*	Hoffman's anthocyaninless	*ah*	A*	SPON	PSN	IL	LA0352
ah	*prov7*	Hoffman's anthocyaninless	*ah*	A*	CHEM	MM	IL	3-343
ai		incomplete anthocyanin	*a342*	A*	RAD	KK	IL	LA1484
ai		incomplete anthocyanin	*a342*	A*	RAD	AC	NIL	LA3611
ai	*2*	incomplete anthocyanin	*am, a340*	A*	RAD	KK	IL	LA1485
al		anthocyanin loser	*a2*	A*	SPON	AC	NIL	LA3576
alb		albescent		G*C	SPON	AC	NIL	LA3729
alb	*prov2*	albescent	*alb*	G*C	CHEM	VCH	IL	3-625
alc		alcobaca		P*	SPON	X	NON	LA2529
alc		alcobaca		P*	SPON	RU	NIL	LA3134
alu		alutacea	*alu1*	C*K	RAD	CR	IL	LA0838
an		anantha	*an^1, an^2, ca*	L*N	RAD	CR	IL	LA0536
ap		apetalous		L*N	SPON	ESC	IL	2-009
ap		apetalous		L*N	SPON	AC	NIL	LA3673

Gene	Allele	Locus name	Synonyms	Class	Origin	Back.	Iso.	Accession
apl		applanata		J*K	RAD	LU	IL	LA0662
apn		albo-punctata		G*BJK	CHEM	VF36	IL	3-105
Aps-1	*1*	Acid phosphatase-1		V*	SPON	VCH	NIL	LA1811
Aps-1	*2*	Acid phosphatase-1		V*	SPON	chm	NON	LA1812
Aps-1	*n*	Acid phosphatase-1		V*	SPON	pim	NON	LA1810
Aps-2	*1*	Acid phosphatase-2		V*	SPON	SM	NON	LA1814
Aps-2	*2*	Acid phosphatase-2		V*	SPON	che	NON	LA1815
Aps-2	*3*	Acid phosphatase-2		V*	SPON	par	NON	LA1816
Aps-2	*n*	Acid phosphatase-2		V*	SPON	che	NON	LA1813
are		anthocyanin reduced		A*	CHEM	VF36	NON	3-073
Asc		Alternaria stem canker resistance		Q*	SPON	X	NON	LA3528
at		apricot		P*L	SPON	AC	NIL	LA3535
at		apricot		P*L	SPON	X	NON	LA0215
at		apricot		P*L	SPON	RU	NIL	LA2998
atn		attenuata	*at*	E*AJK	RAD	RR	IL	LA0587
atn		attenuata	*at*	E*AJK	RAD	AC	NIL	LA3829
atv		atroviolacium		A*	SPON	VF36	NON	LA0797
atv		atroviolacium		A*	SPON	AC	NIL	LA3736
au		aurea		C*B	RAD	AC	NIL	LA3280
au	*(1s)*	aurea	*au^2, au, brac*	C*B	RAD	CR	IL	LA0538
au	*6*	aurea	*yg^6, yg-6, au^yg-6, yo*	C*B	SPON	RCH	IL	LA1486
au	*6*	aurea	*yg^6, yg-6, au^yg-6, yo*	C*B	SPON	AC	NIL	LA2929
au	*tl*	aurea		C*B	SPON	VF145	IL	2-655A
au	*w*	aurea	*w616*	C*B	CHEM	MM	IL	LA2837
aus		austera		J*KT	RAD	LU	IL	LA2023
aut		aureata		C*F	SPON	AC	NIL	LA3166
aut		aureata		C*F	SPON	X	NON	LA1067
auv		aureate virescent		F*C	CHEM	VF36	IL	3-075
avi		albovirens	*avi1*	C*BGN	RAD	CR	IL	LA0936
aw		without anthocyanin	*aba, ab, a179*	A*	SPON	X	NON	LA0271
aw		without anthocyanin	*aba, ab, a179*	A*	SPON	AC	NIL	LA3281
aw	*prov3*	without anthocyanin	*aw*	A*	CHEM	VF36	IL	3-121
aw	*prov4*	without anthocyanin	*aw*	A*	CHEM	VCH	NON	3-603
aw	*prov5*	without anthocyanin	*aw*	A*	CHEM	VCH	NON	3-627
B		Beta-carotene		P*	SPON	X	NON	LA2374
B		Beta-carotene		P*	SPON	O8245	NON	LA3899
B		Beta-carotene		P*	SPON	E6203	NIL	LA3898
B		Beta-carotene		P*	SPON	RU	NIL	LA3000
B	*c*	Beta-carotene	*og^c,Crn,Cr,crn-2,cr-2*	P*L	SPON	PCV	NON	LA0806
B	*c*	Beta-carotene	*og^c,Crn,Cr,crn-2,cr-2*	P*L	SPON	AC	NIL	LA3179
B	*og*	Beta-carotene	*og*	L*P	SPON	PSN	NIL	LA0348
B	*og*	Beta-carotene	*og*	L*P	SPON	X	NON	LA0500
B	*og*	Beta-carotene	*og*	L*P	SPON	X	NON	LA4025
B	*og*	Beta-carotene	*og*	L*P	SPON	X	NON	LA4026
bc		bicolor	*bi*	U*JKT	RAD	CR	IL	LA0588
Bco		Brilliant corolla		L*	SPON	VF36	NON	LA4261
bi		bifurcate inflorescence		M*	SPON	X	NON	LA1786
bip		bipinnata		J*	RAD	LU	IL	LA0663
bip		bipinnata		J*	RAD	AC	NIL	LA3765
bip	*prov2*	bipinnata	*bip*	J*	CHEM	VCH	IL	3-602
bk		beaked		O*	SPON	X	NON	LA0330

Gene	Allele	Locus name	Synonyms	Class	Origin	Back.	Iso.	Accession
Bk-2		Beaked-2		O*	SPON	X	NON	LA1787
bks		black seed	*bks1-1*	S*A	RAD	X	NON	LA4290
bks	*2*	black seed	*bks1-2*	S*A	RAD	X	NON	LA4291
bl		blind		K*	SPON	X	NON	LA0059
bl		blind		K*	SPON	AC	NIL	LA3745
bl	*2*	blind	*to^2*	K*	RAD	LU	IL	LA0980
bl	*to*	blind	*to*	K*JLO	RAD	CR	IL	LA0709
bls		baby lea syndrome	*alm*	A*K	SPON	X	NON	LA1004
bls		baby lea syndrome	*alm*	A*K	SPON	AC	NIL	LA3167
bls	*prov2*	baby lea syndrome	*bls*	A*K	CHEM	VCH	IL	3-610
Bnag-1	*1*	Beta-N-acetyl-D-glucosaminidase-1		V*	SPON	pen	NON	LA2986
br		brachytic		K*	SPON	X	NON	LA2069
brt		bushy root		R*	SPON	X	NON	LA2816
brt-2		bushy root-2		R*	SPON	X	NON	LA3206
bs		brown seed		S*	CHEM	AC	NIL	LA2935
bs-2		brown seed-2		S*	SPON	PLB	IL	LA1788
bs-4		brown seed-4		S*	RAD	MM	IL	LA1998
btl		brittle stem		J*Y	SPON	X	NON	LA1999
bu		bushy	*fru*	K*JM	RAD	X	NON	LA0897
bu		bushy	*fru*	K*JM	RAD	AC	NIL	LA2918
bu	*ab*	bushy	*fru^ab*	K*JM	RAD	RR	IL	LA0549
bu	*cin*	bushy	*cin*	K*JM	SPON	HSD	IL	LA1437
bu	*cin-2*	bushy	*cin-2*	K*JM	SPON	HSD	IL	LA2450
bu	*hem*	bushy	*fru^hem*	K*JM	RAD	CR	IL	LA0604
bul		bullata		C*JK	RAD	CR	IL	LA0589
buo		bullosa	*buo1*	J*O	RAD	pim	IL	LA2000
c		potato leaf		J*	SPON	AC	NIL	LA3168
c	*int*	potato leaf	*int*	J*	RAD	CR	IL	LA0611
c	*int*	potato leaf	*int*	J*	RAD	AC	NIL	LA3728A
c	*prov2*	potato leaf	*c*	J*	CHEM	MM	IL	3-345
c	*prov3*	potato leaf	*c*	J*	CHEM	X	IL	3-604
c	*prov4*	potato leaf	*c*	J*	CHEM	VCH	IL	3-609
c	*prov5*	potato leaf	*c*	J*	CHEM	VCH	IL	3-626
c	*prov6*	potato leaf	*c*	J*	CHEM	VCH	IL	3-631
car		carinata		J*DLO	RAD	CR	IL	LA0539
car-2		carinata-2	*car2*	J*K	RAD	pim	IL	LA2001
cb		cabbage		J*K		AC	NIL	LA3819
cb-2		cabbage leaf-2		J*K	RAD	X	NON	LA2002
cb-2		cabbage leaf-2		J*K	RAD	AC	NIL	LA3169
ccf		cactiflora		N*LO	CHEM	CSM	IL	3-805
Cf-1		Cladosporium fulvum resist.-1	*Cf, Cf1, Cfsc*	Q*	SPON	X	NON	LA2443
Cf-1	*3*	Cladosporium fulvum resist.-1	*Cf-5, Cf5*	Q*	SPON	MM	NIL	LA3046
Cf-1	*3*	Cladosporium fulvum resist.-1	*Cf-5, Cf5*	Q*	SPON	X	NON	LA2447
Cf-2		Cladosporium fulvum resist.-2	*Cf2, Cfp1*	Q*	SPON	X	NON	LA2444
Cf-2		Cladosporium fulvum resist.-2	*Cf2, Cfp1*	Q*	SPON	MM	NIL	LA3043
Cf-3		Cladosporium fulvum resist.-3	*Cf3, Cfp2*	Q*	SPON	X	NON	LA2445
Cf-3		Cladosporium fulvum resist.-3	*Cf3, Cfp2*	Q*	SPON	MM	NIL	LA3044
Cf-4		Cladosporium fulvum resist.-4	*Cf-8, Cf4, Cf-1^2*	Q*	SPON	X	NON	LA2446
Cf-4		Cladosporium fulvum resist.-4	*Cf-8, Cf4, Cf-1^2*	Q*	SPON	AC	NIL	LA3267
Cf-4		Cladosporium fulvum resist.-4	*Cf-8, Cf4, Cf-1^2*	Q*	SPON	MM	NIL	LA3045
Cf-6		Cladosporium fulvum resist.-6		Q*	SPON	X	NON	LA2448
Cf-7		Cladosporium fulvum resist.-7		Q*	SPON	X	NON	LA2449
Cf-9		Cladosporium fulvum resist.-9		Q*	SPON	MM	NIL	LA3047

Gene	Allele	Locus name	Synonyms	Class	Origin	Back.	Iso.	Accession
cfa		conferta	*cfa1*	K*		LU	NON	LA0832
cg		congesta	*cg1*	K*J	RAD	RR	IL	LA0831
ch		chartreuse		L*	SPON	PSN	IL	2-253
ch		chartreuse		L*	SPON	AC	NIL	LA3720
ci		cincta	*ci1*	K*	RAD	CR	IL	LA0938
cit		citriformis		O*JK	RAD	RR	IL	LA2024
cjf		conjunctiflora		L*N	SPON	PTN	IL	LA1056
ck		corky fruit		O*	SPON	X	NON	LA2003
cl-2		cleistogamous-2	*cl2*	L*N	SPON	SM	IL	2-185
cla		clara		C*A	RAD	LU	IL	LA0540
clau		clausa	*ff, vc*	J*LO	RAD	LU	IL	LA0591
clau		clausa	*ff, vc*	J*LO	RAD	AC	NIL	LA3583
clau		clausa	*ff, vc*	J*LO	RAD	X	NON	LA0719
clau	*ff*	clausa		J*LO	SPON	VFSM	IL	2-505
clau	*ics*	clausa	*ics*	J*	SPON	PTN	IL	LA1054
clau	*ics*	clausa	*ics*	J*	SPON	AC	NIL	LA3713
clau	*prov2*	clausa	*clau*	J*LO	SPON	X	IL	LA0509
clau	*vc*	clausa		J*LO	SPON	X	NON	LA0896
cls		clarescens		C*K	RAD	RR	IL	LA2025
clt		coalita		J*	RAD	LU	IL	LA2026
cm		curly mottled		G*JNO	SPON	AC	NIL	LA2919
cm		curly mottled		G*JNO	SPON	PCV	NON	LA0272
cma		commutata		K*DHJ	RAD	RR	IL	LA2027
Cmr		Cucumber mosaic resistance		Q*	SPON	X	NON	LA3912
cn		cana	*ca*	D*K	RAD	RR	IL	LA0590
co		cochlearis		J*D	RAD	CR	IL	LA0592
coa		corrotundata	*coa1*	J*KLT	RAD	CR	IL	LA0940
com		complicata		K*J	RAD	CR	IL	LA0664
com	*in*	complicata	*in*	K*DJ	RAD	CR	IL	LA0610
com	*in*	complicata	*in*	K*DJ	RAD	AC	NIL	LA3715
con		convalescens		E*FK	RAD	CR	IL	LA0541
con		convalescens		E*FK	RAD	AC	NIL	LA3671
cor		coriacea		K*J	RAD	CR	IL	LA0666
cor		coriacea		K*J	RAD	AC	NIL	LA3743
cpa		composita	*cpa1*	M*K	RAD	RR	IL	LA0833
cpt		compact		K*EJ	SPON	XLP	IL	2-377
cpt		compact		K*EJ	SPON	AC	NIL	LA3723
Cri		Crispa		H*JU	RAD	CR	IL	LA0667
Crk		Crinkled		J*T	SPON	X	NON	LA1050
crt		cottony-root		R*	SPON	RCH	NON	LA2802
cru		corrupta	*cru1*	K*J		LU	IL	LA0941
cry-1		cryptochrome-1	*cyr1*	AE*	RAD	MM	IL	LA4359
cta		contaminata	*cta1*	K*HJN	RAD	RR	IL	LA0939
ctr	*1*	citrate concentration		V*	SPON	pim	NON	LA2904
ctt		contracta		K*J	RAD	LU	IL	LA2028
Cu		Curl		J*KT	SPON	STD	IL	LA0325
Cu		Curl		J*KT	SPON	AC	NIL	LA3740
cu-2		curl-2	*cu2*	J*	RAD	CT	IL	LA2004
cu-3		curl-3		J*KT	SPON	pim	NON	LA2398
cul		culcitula		K*U	RAD	RR	IL	LA2029
cur		curvifolia		J*EK	RAD	RR	IL	LA0668
cv		curvata	*cu*	K*JT	RAD	LU	IL	LA0593
cv	*2*	curvata	*acu*	K*JT	RAD	CR	IL	LA0660
cva		conversa		K*D	RAD	CR	IL	LA0665

Gene	Allele	Locus name	Synonyms	Class	Origin	Back.	Iso.	Accession
cvl		convoluta	*cvl1*	K*J	RAD	RR	IL	LA0830
Cvx		Convexa		J*	SPON	X	NON	LA1151
d		dwarf		K*JT	SPON	GRD	NIL	LA3031
d		dwarf		K*JT	SPON	STN	NIL	LA0313
d		dwarf		K*JT	SPON	FB	NIL	LA3022
d	*b*	dwarf		K*JTL	SPON	RR	IL	LA3865
d	*cr*	dwarf	*rob^crisp*	K*JT	RAD	CR	IL	LA0570
d	*im*	dwarf	*rob^imm*	K*JT	RAD	CR	IL	LA0571
d	*prov2*	dwarf	*d*	K*JT	CHEM	VCH	IL	3-623
d	*provcr-2*	dwarf	*d^cr*	K*JT	CHEM	VF36	IL	3-420
d	*provcr-3*	dwarf	*d^cr*	K*JT	CHEM	VF36	IL	3-422
d	*x*	dwarf		K*JT	SPON	PCV	NON	LA1052
d	*x*	dwarf		K*JT	SPON	AC	NIL	LA3615
d	*x*	dwarf		K*JT	SPON	SPZ	IL	LA0160
d	*x*	dwarf		K*JT	SPON	VAN	NIL	LA3902
d-2		dwarf-2	*rob2, rob II, d2*	K*N	RAD	RR	IL	LA0625
dc		decomposita	*dc1*	J*	RAD	RR	IL	LA0819
dd		double dwarf	*d^xx*	K*J	SPON	X	NON	LA0810
de		declinata		K*JU	RAD	RR	IL	LA0594
de		declinata		K*JU	RAD	AC	NIL	LA3742
deb		debilis		H*BCJ	RAD	CR	IL	LA0542
deb		debilis		H*BCJ	RAD	AC	NIL	LA3727
dec		decumbens		K*R	RAD	LU	IL	LA0669
def		deformis		J*LN	RAD	RR	IL	LA0543
def		deformis		J*LN	RAD	AC	NIL	LA3749
def	*2*	deformis	*vit*	J*	RAD	CR	IL	LA0634
def-2		deformis		J*LN	RAD	AC	NIL	LA2920
Del		Delta		P*	SPON	RU	NIL	LA2996A
Del		Delta		P*	SPON	M82	NON	LA4099
Del		Delta		P*	SPON	AC	NIL	LA2921
deli		deliquescens		K*CJ	RAD	RR	IL	LA0595
dep		deprimata		T*J	RAD	CR	IL	LA0544
depa		depauperata		K*CJ	RAD	RR	IL	LA0596
depa		depauperata		K*CJ	RAD	AC	NIL	LA3725
det		detrimentosa		C*KF	RAD	RR	IL	LA0670
det	*2*	detrimentosa		C*KF	RAD	RR	IL	LA0820
Df		Defoliator		Y*H	SPON	par	NON	LA0247
dgt		diageotropica	*lz-3*	K*R	SPON	VFN8	IL	LA1093
dgt	*dp*	diageotropica	*dp*	J*KT	RAD	CT	IL	LA2526
Dia-2	*1*	Diaphorase-2		V*	SPON	pen	NON	LA2987
Dia-2	*2*	Diaphorase-2		V*	SPON	VF36	NIL	LA4232
Dia-3	*1*	Diaphorase-3		V*	SPON	X	NON	LA3345
Dia-3	*1*	Diaphorase-3		V*	SPON	VF36	NIL	LA4269
Dia-4	*1*	Diaphorase-4		V*	SPON	VF36	NIL	LA4284
dil		diluta		D*JK	RAD	CR	IL	LA0545
dil		diluta		D*JK	RAD	AC	NIL	LA3728
dim		diminuta		A*DK	RAD	LU	IL	LA0597
dim-2		diminuta-2	*dim2*	A*K	RAD	AC	NIL	LA3170
dis		discolor		D*F	RAD	CR	IL	LA0598
div		divaricata		C*AJK	RAD	CR	NON	LA0671
div		divaricata		C*AJK	RAD	AC	NIL	LA3818
dl		dialytic		I*LN	SPON	AC	NIL	LA3724
dl		dialytic		I*LN	SPON	SM	IL	2-069
dl	*S*	dialytic	*Dl^s*	L*N	SPON	VF36	NIL	LA3906

Gene	Allele	Locus name	Synonyms	Class	Origin	Back.	Iso.	Accession
dlb		dilabens	*dlb1*	C*JK	RAD	CR	IL	LA0829
dm		dwarf modifier	*d2*	K*	SPON	X	NON	LA0014
dmd		dimidiata		K*JU	RAD	LU	IL	LA2033
dmt		diminutiva		K*	CHEM	VF36	IL	3-007
dps		diospyros		P*	SPON	X	NON	LA1016
dpy		dumpy		K*J	SPON	AC	NIL	LA3171
dpy		dumpy		K*J	SPON	X	NON	LA0811
dpy	*prov2*	dumpy	*dpy*	K*J	CHEM	VCH	IL	3-630
dpy	*prov3*	dumpy	*dpy*	K*J	SPON	ANU	IL	LA1053
drt		dwarf root		R*	CHEM	X	NON	LA3207
ds		dwarf sterile		N*K	SPON	EPK	IL	2-247
ds		dwarf sterile		N*K	SPON	AC	NIL	LA3767
dt		dilatata	*dt1*	C*JK	RAD	CR	IL	LA0828
dtt		detorta		J*K	RAD	LU	IL	LA2030
du		dupla		J*KU	RAD	LU	IL	LA2034
dv		dwarf virescent		F*D	SPON	X	NON	LA0155
e		entire	*b*	J*	SPON	AC	NIL	LA2922
e	*prov3*	entire	*e*	J*	CHEM	VCH	IL	3-616
e-2		entire-2		J*	CHEM		NON	3-705
ec		exserted carpels		O*		X	NON	LA4340
eca		echinata		K*	RAD	RR	IL	LA2035
el		elongated	*e*	O*	SPON	AC	NIL	LA3738
ele		elegans		E*JK	RAD	CR	IL	LA0546
ele		elegans		E*JK	RAD	AC	NIL	LA3825
ele	*2*	elegans	*ang*	E*JK	RAD	CR	IL	LA0586
elu		eluta		E*K	RAD	LU	IL	LA0547
em		emortua	*em1*	H*K	RAD	RR	IL	LA0827
em		emortua	*em1*	H*K	RAD	AC	NIL	LA3817
en		ensiform		J*	SPON	X	NON	LA1787
ep		easy peeling		O*	RAD	AC	NIL	LA3616
ep		easy peeling		O*	RAD	MM	IL	LA1158
Epi		Epinastic		J*K	SPON	VFN8	IL	LA2089
er		erecta		K*JT	RAD	CR	IL	LA0600
era		eramosa	*era1*	B*JK	RAD	CR	IL	LA0850
Est-1	*1*	Esterase-1		V*	SPON	pim	NON	LA1818
Est-1	*1*	Esterase-1		V*	SPON	cer	IL	LA2415
Est-1	*2*	Esterase-1		V*	SPON	pim	NON	LA1819
Est-1	*3*	Esterase-1		V*	SPON	pim	NON	LA1820
Est-1	*4*	Esterase-1		V*	SPON	par	NON	LA1821
Est-1	*5*	Esterase-1		V*	SPON	pen	NON	LA2419
Est-1	*n*	Esterase-1		V*	SPON	pim	NON	LA1817
Est-2	*1*	Esterase-2		V*	SPON	pen	NON	LA2420
Est-3	*1*	Esterase-3		V*	SPON	par	NON	LA2421
Est-4	*1*	Esterase-4		V*	SPON	par	NON	LA2422
Est-4	*2*	Esterase-4		V*	SPON	pim	NON	LA2423
Est-4	*4*	Esterase-4		V*	SPON	PCV	NON	LA2425
Est-4	*5*	Esterase-4		V*	SPON	pim	NON	LA2426
Est-4	*6*	Esterase-4		V*	SPON	pim	NON	LA2427
Est-4	*7*	Esterase-4		V*	SPON	cer	NON	LA2428
Est-4	*8*	Esterase-4		V*	SPON	pim	NON	LA2429
Est-5	*1*	Esterase-5		V*	SPON	pen	NON	LA2430
Est-6	*1*	Esterase-6		V*	SPON	pen	NON	LA2431
Est-7	*1*	Esterase-7		V*	SPON	par	NON	LA2432
Est-7	*2*	Esterase-7		V*	SPON	pen	NON	LA2433

Gene	Allele	Locus name	Synonyms	Class	Origin	Back.	Iso.	Accession
Est-8	*1*	Esterase-8		V*	SPON	pen	NON	LA2988
ete		extenuata	*ete1*	K*JN	RAD	CR	IL	LA0942
ex		exserted stigma		L*N	SPON	SM	IL	2-191
exl		exilis	*ex*	D*JK	RAD	CR	IL	LA0601
exs		excedens	*exs1*	K*J	RAD	CR	IL	LA0852
f		fasciated fruit		O*L	SPON	ESC	NON	LA0517
f	*D*	fasciated fruit		O*L	SPON	PCV	NON	LA0767
fa		falsiflora	*fa1*	M*N	RAD	RR	IL	LA0854
fcf		fucatifolia	*fcf1*	D*CK	RAD	CR	IL	LA0945
fd		flecked dwarf		G*DK	RAD	AC	NIL	LA3750
fd		flecked dwarf		G*DK	RAD	BK	NON	LA0873
Fdh-1	*1*	Formate dehydrogenase-1		V*	SPON	pen	IL	LA2989
Fdh-1	*2*	Formate dehydrogenase-1		V*	SPON	VF36	NIL	LA4238
fe		fertilis		J*LO	RAD	LU	IL	LA0672
fgv		fimbriate gold virescent		F*CJ	SPON	VF36	IL	LA1143
fir		firma		K*JM	RAD	CR	IL	LA0602
fl		fleshy calyx		O*	SPON	X	NON	LA2372
fla		flavescens		D*JK	RAD	LU	IL	LA0548
fla		flavescens		D*JK	RAD	AC	NIL	LA3565
flav		flavida		C*	RAD	LU	IL	LA0603
flc		flacca		W*HJY	RAD	RR	IL	LA0673
flc		flacca		W*HJY	RAD	AC	NIL	LA3613
fld		flaccida	*fld1*	K*HJT	RAD	RR	IL	LA0943
fle		flexifolia	*fle1*	A*J	RAD	AC	NIL	LA3764
fli		filiform inflorescence		M*LN	SPON	X	NON	LA1790
fn		finely-netted		D*	RAD	PSP	IL	LA2005
fr		frugalis		K*JT	RAD	CR	IL	LA0674
frg		fragilis	*frg1*	D*CJK	RAD	CR	IL	LA0864
fri	*1*	far red light insensitive	*phyA*	AY*	CHEM	MM	IL	LA3809
fri	*1*	far red light insensitive	*phyA*	AY*	CHEM	MM	IL	LA4356
Frl		FORL resistance	*Fr1, Fr-1*	Q*	SPON	AC	NIL	LA3273
Frl		FORL resistance	*Fr1, Fr-1*	Q*	SPON	VGB	NON	LA3841
Frs		Frosty spot	*Nec*	H*	SPON	X	NON	LA2070
frt		fracta		K*JT	RAD	LU	IL	LA2038
fsc		fuscatinervis	*dkv*	E*	SPON	VF145	IL	LA0872
ft		fruiting temperature		O*	SPON	X	NON	LA2006
fu		fusiformis		C*JK	RAD	CR	IL	LA0605
fu		fusiformis		C*JK	RAD	AC	NIL	LA3070
fua		fucata	*fua1*	E*K	RAD	CR	IL	LA0944
fug		fulgida	*fug1*	E*BK	RAD	RR	IL	LA0946
ful		fulgens		E*	RAD	CR	IL	LA0550
ful	*2*	fulgens	*ful1^2*	E*	RAD	RR	IL	LA0843
ful-3		fulgens-3		E*	SPON	VF36	IL	LA1495
fus		fulgescens		E*	RAD	LU	IL	LA2039
Fw		Furrowed		J*KN	SPON	PSN	IL	LA0192
Fw		Furrowed		J*KN	SPON	AC	NIL	LA3300
fx		flexa		K*	RAD	LU	IL	LA2037
fy		field yellow		E*	SPON	AC	NIL	LA3295
fy		field yellow		E*	SPON	VF36	IL	2-565
ga		galbina	*ga1*	D*BE	RAD	CR	IL	LA0836
ga		galbina	*ga1*	D*BE	RAD	AC	NIL	LA3828
gas		gamosepala	*gas1*	D*JL	RAD	RR	IL	LA0947
gbl		globula		K*JU	RAD	LU	IL	LA2032
Ge	*c*	Gamete eliminator		N*	SPON	CR	NON	LA0533

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Ge	*p*	Gamete eliminator		N*	SPON	PSN	NON	LA0012
gf		green flesh		P*	SPON	RU	NIL	LA2999
gf		green flesh		P*	SPON	AC	NIL	LA3534
gf		green flesh		P*	SPON	PCV	NON	LA2071
gfl		globular flower		L*	SPON	X	NON	LA2984
gh		ghost	*ab*	B*G	SPON	SM	IL	LA0295
gh-2		ghost-2		C*G	CHEM	SX	IL	LA2007
gi		gibberosa		J*K	RAD	RR	IL	LA2040
gib-1		gibberellin deficient-1		K*Y	CHEM	MM	IL	LA2893
gib-2		gibberellin deficient-2		K*Y	CHEM	MM	IL	LA2894
gib-3		gibberellin-deficient-3		K*Y	CHEM	MM	IL	LA2895
gib-3	*x*	gibberellin-deficient-3		K*Y	CHEM	X	NON	LA2993
gl		glauca		J*F	RAD	CR	IL	LA0675
glau		glaucescens		E*JK	RAD	CR	IL	LA0606
glb		globularis		K*CJ	RAD	RR	IL	LA0677
glc		glaucophylla		D*JK	RAD	RR	IL	LA0676
glf		globiformis	*glf1*	K*M	RAD	CR	IL	LA0948
glg		galapagos light green		D*	SPON	X	NON	LA1059
glm		glomerata		K*	RAD	LU	IL	LA2031
glo		globosa		K*	RAD	CR	IL	LA0551
glo	*2*	globosa	*inx, intro*	K*	RAD	LU	IL	LA0612
glo	*2*	globosa	*inx, intro*	K*	RAD	AC	NIL	LA3618
glu		glutinosa	*glu1*	O*P	RAD	RR	IL	LA0842
gm		gamosepalous		L*	RAD	SX	IL	LA2008
Got-1	*1*	Glutamate oxaloacetate transaminase-1		V*	SPON	pim	NON	LA1822
Got-1	*2*	Glutamate oxaloacetate trans.-1		V*	SPON	pim	NON	LA1823
Got-2	*1*	Glutamate oxaloacetate trans.-2		V*	SPON	pim	NON	LA1825
Got-2	*2*	Glutamate oxaloacetate trans.-2		V*	SPON	che	NON	LA1826
Got-2	*3*	Glutamate oxaloacetate trans.-2		V*	SPON	par	NON	LA1827
Got-2	*4*	Glutamate oxaloacetate trans.-2		V*	SPON	pim	NON	LA1828
Got-2	*n*	Glutamate oxaloacetate trans.-2		V*	SPON	pim	NON	LA1824
Got-3	*2*	Glutamate oxaloacetate trans.-3		V*	SPON	pim	NON	LA1831
Got-3	*3*	Glutamate oxaloacetate trans.-3		V*	SPON	par	NON	LA1832
Got-3	*n*	Glutamate oxaloacetate trans.-3		V*	SPON	che	NON	LA1829
Got-4	*1*	Glutamate oxaloacetate trans.-4		V*	SPON	par	NON	LA1834
Got-4	*2*	Glutamate oxaloacetate trans.-4		V*	SPON	pim	NON	LA1835
Got-4	*n*	Glutamate oxaloacetate trans.-4		V*	SPON	cer	NON	LA1833
Gp		Gamete promoter		N*	SPON	AC	NIL	LA3273
gq		grotesque		L*O	SPON	X	NON	LA0137
Gr		Green ripe	*gr*	P*	SPON	X	NON	LA2453
Gr	*Nr-2*	Green ripe	*Nr-2*	P*	SPON	X	NON	LA2455
gra		gracilis		K*J	RAD	CR	IL	LA0607
grc		gracillama	*grc1*	E*JK	RAD	RR	IL	LA0950
grf		grandifructa	*grf1*	K*O	RAD	LU	IL	LA0951
grl		gracilenta	*grl1*	E*JK	RAD	RR	IL	LA0949
grn		granulosa		I*	CHEM	CSM	IL	3-804
gro		grossa		J*DK	RAD	LU	IL	LA2041
gs		green stripe		P*	SPON	GSM	IL	LA0212
gs		green stripe		P*	SPON	AC	NIL	LA3530
h		hairs absent	*H*	I*	SPON	AC	NIL	LA3172
h		hairs absent	*H*	I*	SPON	X	NON	LA0154
he		heteroidea		D*JK	RAD	CR	IL	LA0679
Hero		*Heterodera rostochiensis* resis.		Q*	SPON	X	NON	LA1792

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hg		heterogemma	*hg1*	K*M	RAD	CR	IL	LA0837
hi		hilara		K*DJT	RAD	CR	IL	LA0952
hl		hairless		I*X	SPON	AC	NIL	LA3556
hl	*2*	hairless	*cal, cal1*	I*X	RAD	CR	IL	LA0937
hl	*prov3*	hairless	*hl*	I*X	CHEM	VCH	IL	3-095
hl	*prov4*	hairless	*hl*	I*X	CHEM	VCH	IL	3-126
hl	*prov5*	hairless	*hl*	I*X	CHEM	VCH	IL	3-605
hl-2		hairless-2	*hl^prov6*	I*X	CHEM	VF36	NON	3-417
hp-1		high pigment-1	*hp, hp1, hp2, bs, dr*	P*TA	SPON	X	NON	LA0279
hp-1		high pigment-1	*hp, hp1, hp2, bs, dr*	P*TA	SPON	RU	NIL	LA3004
hp-1		high pigment-1	*hp, hp1, hp2, bs, dr*	P*TA	SPON	AC	NIL	LA3538
hp-1	*w*	high pigment-1		P*TA	CHEM	GT	IL	LA4012
hp-2		high pigment-2	*hp*	P*TA	CHEM	MM	NON	LA4013
hp-2		high pigment-2	*hp*	P*TA	CHEM	SM	NIL	LA3006
hp-2	*dg*	high pigment-2	*dg*	P*AT	SPON	MP	NIL	LA3005
hp-2	*dg*	high pigment-2	*dg*	P*AT	SPON	MP	IL	LA2451
hp-2	*j*	high pigment-2	*hp*	P*T	SOMA	MM	NON	LA4014
Hr		Hirsute		I*	SPON	X	IL	LA0895
Hrt		Hirtum		I*	SPON	X	NON	LA0501
ht		hastate		J*L	SPON	SM	IL	2-295
hy		homogeneous yellow		E*	SPON	AC	NIL	LA3308
hy		homogeneous yellow		E*	SPON	cer	NON	LA1142
I		Immunity to Fusarium wilt		Q*	SPON	VD	NIL	LA3025
I		Immunity to Fusarium wilt		Q*	SPON	GRD	NIL	LA3042
I-2		Immunity to Fusarium wilt-2		Q*	SPON	MM	NIL	LA2821
I-3		Immunity to Fusarium wilt-3		Q*	SPON	X	NON	LA4025
I-3		Immunity to Fusarium wilt-3		Q*	SPON	X	NON	LA4026
ic		inclinata		J*CK	RAD	RR	IL	LA0682
ica		icana		B*JK	RAD	RR	IL	LA2042
icn		incana		B*F	SPON	X	NON	LA1009
icn		incana		B*F	SPON	AC	NIL	LA3173
id		indehiscens		L*JO	RAD	RR	IL	LA0684
ida		inordinata		K*JT	RAD	RR	IL	LA2043
Idh-1	*1*	Isocitrate dehydrogenase-1		V*	SPON	hir	NON	LA2906
ig		ignava		D*K	RAD	CR	IL	LA0608
ig		ignava		D*K	RAD	AC	NIL	LA3752
im		impatiens	*im1*	K*UW	RAD	RR	IL	LA0863
imb		imbecilla		E*DK	SPON	CR	IL	LA0552
imb		imbecilla		E*DK	SPON	AC	NIL	LA3566
imp	*dia*	impedita		E*K	SPON	CR	IL	LA0680
imp	*eg*	impedita		E*K	SPON	CR	IL	LA0681
ina		inflexa	*ina1*	K*	RAD	LU	IL	LA0840
ina		inflexa	*ina1*	K*	RAD	AC	NIL	LA3732
inc		incurva		K*J	RAD	CR	IL	LA0609
inc		incurva		K*J	RAD	AC	NIL	LA3730
inf		informa		J*K	RAD	CR	IL	LA0553
inf		informa		J*K	RAD	AC	NIL	LA3726
ini		inquieta	*ini1*	I*DJK	RAD	RR	IL	LA0953
ino		involuta	*ino1*	K*	RAD	CR	IL	LA0954
ins		inconstans	*ins1*	K*	RAD	RR	IL	LA0841
inv		invalida		F*EJK	RAD	CR	IL	LA0554

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inv		invalida		F*EJK	RAD	AC	NIL	LA3439
Ip		Intense pigment		P*	SPON	VF145	NIL	LA1563
Ip		Intense pigment		P*	SPON	VF145	NIL	LA1500
irr		irregularis		J*CT	RAD	CR	IL	LA0613
irr		irregularis		J*CT	RAD	AC	NIL	LA3747
ita		inquinata	*ita1*	H*G	RAD	RR	IL	LA0839
j		jointless	*lf*	M*	SPON	FB	NIL	LA3023
j		jointless	*lf*	M*	SPON	GRD	NIL	LA3033
j-2		jointless-2	*j2*	M*	SPON	PSN	NON	LA0315
j-2		jointless-2	*j2*	M*	SPON	O8245	NON	LA3899
j-2	*in*	jointless-2	*j2^in*	M*	SPON	X	NON	LA0756
Jau		Jaundiced		E*	SPON	AC	NIL	LA3174
jug		jugata		K*LO	RAD	CR	IL	LA0555
jug	*2*	jugata	*jug1^2*	K*LO	RAD	LU	IL	LA0834
l		lutescent	*g*	C*	SPON	AC	NIL	LA3717
l	*2*	lutescent	*rub*	C*	RAD	LU	IL	LA0572
l	*prov3*	lutescent	*l*	C*	SPON	ROMA	IL	2-491
l	*prov4*	lutescent	*l*	C*	SPON	EPK	NIL	LA3009
l-2		lutescent-2	*l-3, l2*	C*Y	SPON	LRD	IL	LA0643
l-2		lutescent-2	*l-3, l2*	C*Y	SPON	AC	NIL	LA3581
La		Lanceolate		J*	SPON	PCV	NON	LA0335
lae		laesa		H*JK	RAD	RR	IL	LA0685
lan		languida		D*F	RAD	RR	IL	LA2044
lap		lamprochlora	*lap1*	J*K	RAD	RR	IL	LA0955
lat		lata		K*	RAD	CR	IL	LA0556
le		lembiformis	*le1*	K*ACJR	RAD	RR	IL	LA0956
lep		leprosa	*lep1*	H*K	RAD	RR	IL	LA0957
lg		light-green	*lme*	D*	SPON	X	NON	LA1156
lg		light-green	*lme*	D*	SPON	AC	NIL	LA3175
lg-5		light green-5	*lg5, lm, fy, yt*	D*	SPON	X	NON	LA0757
lg-5		light green-5	*lg5, lm, fy, yt*	D*	SPON	AC	NIL	LA3176
li		limbrata		J*	RAD	LU	IL	LA2045
Ln		Lanata		I*	CHEM	VF36	IL	3-071
Ln	*G*	Lanata		I*	CHEM	FLD	IL	LA3127
lop		longipes	*lop1*	J*DK	RAD	CR	IL	LA0958
Lpg		Lapageria		J*LNT	SPON	VF36	IL	2-561
Lpg		Lapageria		J*LNT	SPON	AC	NIL	LA3739
ls		lateral suppresser		K*LN	SPON	AMB	NON	LA0329
ls		lateral suppresser		K*LN	SPON	X	NON	LA2892
ls		lateral suppresser		K*LN	SPON	AC	NIL	LA3761
ls	*2*	lateral suppresser		K*LN		PRI	NIL	LA3901
lt		laeta	*lt1*	E*DK	RAD	CR	IL	LA0835
ltf		latifolia		J*	CHEM	VF36	IL	3-035A
lu		luteola		L*	RAD	LU	IL	LA0686
luc		lucida		C*F	RAD	CR	IL	LA0557
lur		lurida	*lur1*	E*D	RAD	RR	IL	LA0959
lut		lutea		E*F	RAD	CR	IL	LA0558
lut		lutea		E*F	RAD	AC	NIL	LA3714
Lv		*Leveillula taurica* resistance		Q*	SPON	X	NON	LA3118
Lv		*Leveillula taurica* resistance		Q*	SPON	X	NON	LA3119
Lx		Lax		J*	SPON	LK	NON	LA0505
Lx		Lax		J*	SPON	AC	NIL	LA3177
lyr		lyrate		J*NO	SPON	AC	NIL	LA2923
lyr		lyrate		J*NO	SPON	PCV	NON	LA0763

Gene	Allele	Locus name	Synonyms	Class	Origin	Back.	Iso.	Accession
lz		lazy		K*	RAD	AC	NIL	LA3762
lz-2		lazy-2		K*	CHEM	SM	NIL	LA2924
lz-2		lazy-2		K*	CHEM	AC	NIL	LA3710
m		mottled		G*J	RAD	AC	NIL	LA3568
m-2		mottled-2	*m2, mo, md*	F*D	RAD	AC	NIL	LA3574
ma		macrocarpa		J*O	RAD	LU	IL	LA0687
mac		maculata	*mac1*	H*K	RAD	CR	IL	LA0960
mad		marcida	*mad1*	T*K	RAD	CR	IL	LA0961
Mae-1	*1*	Malic enzyme-1		V*	SPON	VF36	NIL	LA4251
mar		marcescens		T*K	RAD	LU	NON	LA0688
marm		marmorata		G*D	RAD	CR	IL	LA0559
marm	*2*	marmorata	*marm1^2*	G*D	RAD	CR	IL	LA0844
mc		macrocalyx		L*M	SPON	X	NON	LA0159
mcn		maculonecrotic		G*H*CF	CHEM	VF36	IL	3-045
mcr		multicolor		B*CH	RAD	LU	IL	LA2047
mcs		macrosepala		L*J	RAD	LU	IL	LA2046
Mdh-1	*1*	Malate dehydrogenase-1		V*	SPON	X	NON	LA3344
Mdh-1	*1*	Malate dehydrogenase-1		V*	SPON	VF36	NIL	LA4243
Mdh-4	*1*	Malate dehydrogenase-4		V*		pen	NON	LA2990
Mdh-4	*1*	Malate dehydrogenase-4		V*		VF36	NIL	LA4283
Me		Mouse ears		J*K	SPON	RU	IL	LA0324
Me		Mouse ears		J*K	SPON	AC	NIL	LA3552
med		mediocris	*med1*	K*	RAD	CR	IL	LA0962
mel		melongenoida	*mel1*	O*K	RAD	LU	IL	LA0963
mgn		marginal necrotic		H*C	CHEM	VF36	IL	3-025
Mi		Meloidogyne incognita resist.		Q*	SPON	VFN8	NON	LA1022
Mi		Meloidogyne incognita resist.		Q*	SPON	MM	NIL	LA2819
Mi-3		Meloidogyne incognita resist.-3		Q*	SPON	per	NON	LA3858
mic		microcarpa	*mic1*	D*GLO	RAD	CR	IL	LA0845
mn		minuta	*mi*	K*CJ	RAD	CR	IL	LA0614
mon		monstrosa		K*J	RAD	AC	NIL	LA3826
mon		monstrosa		K*J	RAD	CR	IL	LA0615
mor		morata	*mor1*	E*K	RAD	RR	IL	LA0848
ms-2		male-sterile-2	*ms2*	N*	SPON	PSN	IL	2-031
ms-3		male-sterile-3	*ms3*	N*	SPON	SM	IL	2-032
ms-5		male-sterile-5	*ms5*	N*	SPON	SM	IL	2-039
ms-6		male-sterile-6	*ms6*	N*	SPON	SM	IL	2-044
ms-7		male-sterile-7	*ms7*	N*	SPON	SM	IL	2-089
ms-9		male-sterile-9	*ms9*	N*	SPON	SM	IL	2-121
ms-10		male-sterile-10	*ms10*	N*	SPON	SM	IL	2-132
ms-10	*35*	male-sterile-10	*ms-35, ms35*	N*	SPON	VF11	IL	2-517
ms-10	*36*	male-sterile-10	*ms-36*	N*	SPON	VF36	IL	2-635
ms-11		male-sterile-11	*ms11*	N*	SPON	SM	IL	2-152
ms-12		male-sterile-12	*ms12*	N*	SPON	SM	IL	2-161
ms-13		male-sterile-13	*ms13*	N*	SPON	SM	IL	2-165
ms-14		male-sterile-14	*ms14*	N*	SPON	ERL	IL	2-175
ms-15		male-sterile-15	*ms15*	N*	SPON	SM	IL	2-193
ms-15	*26*	male-sterile-15	*ms26, ms-26*	N*	SPON	VE	IL	2-327
ms-15	*47*	male-sterile-15	*ms-47*	N*	SPON	UC82B	NIL	2-837
ms-16		male-sterile-16	*ms16*	N*	SPON	PRT	IL	LA0062
ms-17		male-sterile-17	*ms17*	N*	SPON	ACE	IL	2-225
ms-18		male-sterile-18	*ms18*	N*	SPON	C255	IL	2-233
ms-23		male-sterile-23	*ms23*	N*	SPON	EPK	IL	2-273
ms-24		male-sterile-24	*ms24*	N*	SPON	EPK	IL	2-277

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ms-25		male-sterile-25	*ms25*	N*	SPON	RTVF	IL	2-313
ms-27		male-sterile-27	*ms27*	N*	SPON	VE	IL	2-331
ms-28		male-sterile-28	*ms28*	N*	SPON	XLP	IL	2-355
ms-29		male-sterile-29	*ms29*	N*	SPON	CPC2	IL	2-423
ms-30		male-sterile-30	*ms30*	N*	SPON	SM	IL	2-455
ms-31		male-sterile-31	*ms31*	N*	SPON	VF6	IL	2-461
ms-32		male-sterile-32	*ms32*	N*	SPON	cer	NON	LA0359
ms-32		male-sterile-32	*ms32*	N*	SPON	POR	NIL	LA2715
ms-32		male-sterile-32	*ms32*	N*	SPON	M168	NIL	LA2714
ms-32		male-sterile-32	*ms32*	N*	SPON	MNB	NIL	LA2712
ms-32		male-sterile-32	*ms32*	N*	SPON	M167	NIL	LA2713
ms-33		male-sterile-33	*ms33*	N*	SPON	VF11	IL	2-511
ms-34		male-sterile-34	*ms34*	N*	SPON	VF11	IL	2-513
ms-38		male-sterile-38	*ms38*	N*	SPON	VF36	IL	2-539
ms-38	*40*	male-sterile-38	*ms-40*	N*	SPON	VF36	IL	2-553
ms-39		male-sterile-39		N*	SPON	VF36	IL	2-549
ms-44		male-sterile-44		N*J	CHEM	SM	IL	LA2090
ms-45		male-sterile-45		N*	SPON	VFN8	IL	2-659
ms-46		male-sterile-46		N*	SPON	VFN8	IL	2-681
Ms-48		Male-sterile-48		N*	CHEM	MR20	NIL	LA3193
Ms-48		Male-sterile-48		N*	CHEM	T5	NIL	LA3198
Ms-48		Male-sterile-48		N*	CHEM	TVD	NIL	LA3192
Ms-48		Male-sterile-48		N*	CHEM	VF36	NIL	LA3191
Ms-48		Male-sterile-48		N*	CHEM	CSM	IL	2-839
Ms-48		Male-sterile-48		N*	CHEM	VCH	NIL	LA3199
ms-49		male-sterile-49		N*	SPON	per	NON	LA1161
ms-50		male sterile-50		N*	RAD	T5	IL	LA3149
mt		midget		K*N	SPON	NRT	NON	LA0282
mta		mutata	*mta1*	K*EFJ	RAD	RR	IL	LA0965
mts		mortalis	*mts1*	K*JM	RAD	RR	IL	LA0849
mu		multinervis		D*J	RAD	CR	IL	LA0690
mu		multinervis		D*J	RAD	AC	NIL	LA3573
mu	*3*	multinervis	*rv-3*	D*J	CHEM	VF36	IL	3-033
mua		multifurcata	*mua1*	K*M	RAD	CR	IL	LA0851
muf		multifolia		J*DK	RAD	RR	IL	LA0689
mult		multiflora		M*	RAD	CR	IL	LA0560
mup		multiplicata	*mup1*	M*L	RAD	RR	IL	LA0846
mut		mutabilia	*mut1*	K*DT	RAD	RR	IL	LA0866
muv-2		multivalens-2	*mus1*	C*FJK	RAD	CR	IL	LA0964
muv-2		multivalens-2	*mus1*	C*FJK	RAD	AC	NIL	LA3758
mux		multiplex	*mux1*	L*KM	RAD	CR	IL	LA0847
n		nipple-tip	*nt*	O*	SPON	X	NON	LA2353
n		nipple-tip	*nt*	O*	SPON	X	NON	LA2370
na		nana		K*J	RAD	CR	IL	LA0561
nc		narrow cotyledons		J*	SPON	AC	NIL	LA3178
nd		netted	*m-4*	F*	RAD	AC	NIL	LA3584
ndw		necrotic dwarf		H*JK	SPON	X	NON	LA3142
ndw		necrotic dwarf		H*JK	SPON	M82	NIL	LA4061
ne		necrotic		H*	SPON	X	NON	LA2350
ne		necrotic		H*	SPON	AC	NIL	LA3084
neg		neglecta		H*DK	RAD	CR	IL	LA0562
neg		neglecta		H*DK	RAD	AC	NIL	LA3746
neg	*ne-2*	neglecta	*ne-2, ne2*	H*DK	RAD	AC	NIL	LA3621
neg	*ne-2*	neglecta	*ne-2, ne2*	H*DK	RAD	CT	IL	LA2454

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neg	*ne-2*	neglecta	*ne-2, ne2*	H*DK	RAD	X	NON	LA2489
nor		non-ripening		P*	SPON	AC	NIL	LA3770
nor		non-ripening		P*	SPON	X	NON	LA1793
nor		non-ripening		P*	SPON	RU	NIL	LA3013
not		notabilis		W*JY	RAD	LU	IL	LA0617
not		notabilis		W*JY	RAD	AC	NIL	LA3614
Nr		Never ripe		P*	SPON	AC	NIL	LA3537
Nr		Never ripe		P*	SPON	PSN	IL	LA0162
Nr		Never ripe		P*	SPON	RU	NIL	LA3001
nv		netted virescent		E*F	SPON	X	NON	LA0786
o		ovate		O*	SPON	AC	NIL	LA3543
o	*l*	ovate	*ol, O^1*	O*	SPON	X	NON	LA0271
ob		obscura		T*K	RAD	RR	IL	LA0691
obl		oblate fruit		O*	RAD	MM	NIL	LA1159
obv		obscuravenosa		U*X	SPON	M82	NON	LA3475
obv	+	obscuravenosa		U*X	SPON	M82	NON	LA4057
oc		ochroleuca		G*BK	RAD	RR	IL	LA0692
Od		Odorless		I*	SPON	PCV	NON	LA0292
oli		olivacea		K*U	RAD	AC	NIL	LA3722
op		opaca		D*CF	RAD	CR	IL	LA0618
op		opaca		D*CF	RAD	AC	NIL	LA3567
opa		opacata	*opa1*	E*K	RAD	CR	IL	LA0966
or		ordinata		D*F	RAD	RR	IL	LA2048
Ora		*Orobanche aegyptica* resistance		Q*	SPON	X	NON	LA2530
os		oligosperma	*os1*	K*JT	RAD	CR	IL	LA0868
ovi		oviformis	*ovi1*	J*O	RAD	LU	IL	LA0967
p		peach		O*I	SPON	X	NON	LA2357
pa-2		parva-2	*pa1, pa2*	K*J	RAD	CR	IL	LA0970
pal		pallida		D*L	RAD	CR	IL	LA0563
pap		paupercula		J*W	RAD	RR	IL	LA2050
pas		pallescens	*pas1*	D*K	RAD	CR	IL	LA0968
pat		parthenocarpic fruit		S*	CHEM	ROMA	IL	LA2013
pat-2		parthenocarpic fruit-2		S*	SPON	X	NON	LA2413
pau		pauper		K*	RAD	CR	NON	LA0877
pct		polycot		J*KLMS	SPON	MM	NON	LA2896
pcv		polychrome variegated		G*BDJ	SPON	X	NON	LA1199
pdc		pudica		K*JT	CHEM	VF36	IL	3-047
pds		phosphorus deficiency syndrome	*Ph-oid*	A*CY	SPON	X	NON	LA0813
pdw		pale dwarf		V*	SPON	X	NON	LA2457
pdw		pale dwarf		V*	SPON	X	NON	LA2490
pe		sticky peel		O*	SPON	X	NON	LA0759
pen		pendens		J*C	RAD	AC	NIL	LA3293
pen		pendens		J*C	RAD	CR	IL	LA0694
per		perviridis		A*KT	RAD	RR	IL	LA0564
pet		penetrabile	*pet-2, pet2*	K*J	RAD	CR	IL	LA0971
Pgdh-2	*1*	6-Phosphogluconate dehydrogenase-2		V*	SPON	pen	NON	LA2991
Pgdh-3	*1*	6-Phosphogluconate dehydrog.-3		V*	SPON	pen	NON	LA2434
Pgi-1	*1*	Phosphoglucoisomerase-1		V*	SPON	pen	NON	LA2435
Pgi-1	*2*	Phosphoglucoisomerase-1		V*	SPON	par	NON	LA2436
Pgm-1	*1*	Phosphoglucomutase-1		V*	SPON	hir	NON	LA2437
Pgm-2	*1*	Phosphoglucomutase-2		V*	SPON	pen	NON	LA2438
Ph		*Phytophthora infestans* resist.	*PiT, TR1*	Q*	SPON	X	NON	LA2009
Ph-2		*Phytophthora infestans* resist.-2		Q*	SPON	UC82	NIL	LA3151

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Ph-2		*Phytophthora infestans* resist.-2		Q*	SPON	MNB	NIL	LA3152
Ph-3		*Phytophthora infestans* resist.-3		Q		CLN2264F	NON	LA4285
Ph-3		*Phytophthora infestans* resist.-3		Q		CLN2264G	NON	LA4286
phyB2		phytochrome B2		AE*	RAD	MM	IL	LA4358
pi		pistillate		L*N	SPON	SM	IL	2-137
pi-2		pistillate-2		N*LM	CHEM	CSM	IL	3-802
pic		picta		H*C	RAD	CR	IL	LA0620
pl		perlucida	*pl1*	D*CJ	RAD	CR	IL	LA0867
pl		perlucida	*pl1*	D*CJ	RAD	AC	NIL	LA3296
pla		plana		D*CK	RAD	CR	IL	LA0695
pli		plicata		K*ABJ	RAD	AC	NIL	LA3672
pli		plicata		K*ABJ	RAD	LU	IL	LA0696
pm		praematura	*pm1*	Z*CJK	RAD	RR	IL	LA0855
Pn		Punctate		A*I	SPON	AC	NIL	LA3089
Pn		Punctate		A*I	SPON	X	NON	LA0812
pol		polylopha		K*JO	RAD	LU	IL	LA0697
pp		polyphylla	*pp1*	J*D	RAD	RR	IL	LA0860
ppa		purpurea		A*	RAD	LU	IL	LA2054
pr		propeller		J*	RAD	AC	NIL	LA2925
pr		propeller		J*	RAD	X	NON	LA0326
prc		procumbens		K*CJ	RAD	CR	IL	LA0698
pre		pressa		K*J	RAD	RR	IL	LA2053
pro		procera		J*Z	RAD	CR	IL	LA0565
pro		procera		J*Z	RAD	AC	NIL	LA3283
prt		protea	*prt1*	C*JK	RAD	CR	IL	LA0972
prun		prunoidea		O*J	RAD	LU	IL	LA0566
Prx-1	*1*	Peroxidase-1		V*	SPON	pim	NON	LA1837
Prx-1	*2*	Peroxidase-1		V*	SPON	pim	NON	LA1838
Prx-1	*3*	Peroxidase-1		V*	SPON	pim	NON	LA1839
Prx-1	*4*	Peroxidase-1		V*	SPON	chm	NON	LA1840
Prx-1	*5*	Peroxidase-1		V*	SPON	pim	NON	LA1841
Prx-1	*n*	Peroxidase-1		V*	SPON	pim	NON	LA1836
Prx-2	*1*	Peroxidase-2		V*	SPON	cer	NON	LA1843
Prx-2	*3*	Peroxidase-2		V*	SPON	pim	NON	LA1845
Prx-2	*n*	Peroxidase-2		V*	SPON	pim	NON	LA1842
Prx-3	*1*	Peroxidase-3		V*	SPON	pim	NON	LA1847
Prx-3	*2*	Peroxidase-3		V*	SPON	pim	NON	LA1848
Prx-3	*a1*	Peroxidase-3		V*	SPON	chm	NON	LA1849
Prx-3	*n*	Peroxidase-3		V*	SPON	pim	NON	LA1846
Prx-4	*1*	Peroxidase-4		V*	SPON	pim	NON	LA1850
Prx-4	*10*	Peroxidase-4		V*	SPON	cer	NON	LA1859
Prx-4	*11*	Peroxidase-4		V*	SPON	pim	NON	LA1860
Prx-4	*12*	Peroxidase-4		V*	SPON	pim	NON	LA1861
Prx-4	*13*	Peroxidase-4		V*	SPON	pim	NON	LA1862
Prx-4	*14*	Peroxidase-4		V*	SPON	pim	NON	LA1863
Prx-4	*15*	Peroxidase-4		V*	SPON	pim	NON	LA1864
Prx-4	*17*	Peroxidase-4		V*	SPON	pim	NON	LA1866
Prx-4	*18*	Peroxidase-4		V*	SPON	pim	NON	LA1867
Prx-4	*19*	Peroxidase-4		V*	SPON	pim	NON	LA1868
Prx-4	*2*	Peroxidase-4		V*	SPON	pim	NON	LA1851
Prx-4	*20*	Peroxidase-4		V*	SPON	cer	NON	LA1869
Prx-4	*21*	Peroxidase-4		V*	SPON	pim	NON	LA1870

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Prx-4	*23*	Peroxidase-4		V*	SPON	pim	NON	LA1872
Prx-4	*3*	Peroxidase-4		V*	SPON	pim	NON	LA1852
Prx-4	*4*	Peroxidase-4		V*	SPON	chm	NON	LA1853
Prx-4	*5*	Peroxidase-4		V*	SPON	chm	NON	LA1854
Prx-4	*6*	Peroxidase-4		V*	SPON	par	NON	LA1855
Prx-4	*7*	Peroxidase-4		V*	SPON	STN	NON	LA1856
Prx-4	*8*	Peroxidase-4		V*	SPON	pim	NON	LA1857
Prx-4	*9*	Peroxidase-4		V*	SPON	pim	NON	LA1858
Prx-7	*2*	Peroxidase-7		V*	SPON	pim	NON	LA1874
Prx-7	*n*	Peroxidase-7		V*	SPON	pim	NON	LA1875
ps		positional sterile	*va*	L*N	SPON	JBR	IL	LA0063
ps	*prov2*	positional sterile	*ps*	L*N	SPON	PSN	IL	2-303
ps-2		positional sterile-2		L*N	SPON	X	NON	LA2010
ps-2		positional sterile-2		L*N	SPON	VRB	IL	LA3631
ps-2		positional sterile-2		L*N	SPON	STR24	NON	LA3632
psa		perspicua		D*J	RAD	LU	IL	LA2051
pst		persistent style		O*	SPON	ESC	IL	2-005
pt		petite		D*J	RAD	AC	NIL	LA3768
pta		partiaria		J*	RAD	RR	IL	LA2049
ptb		protuberant		O*	SPON	X	NON	LA1017
ptb		protuberant		O*	SPON	X	NON	LA1018
Pto		*Pseudomonas syringae* pv *tomato* resistance		Q*	SPON	MM	NIL	LA3472
Pto		*Pseudomonas syringae* pv *tomato* resis.		Q*	SPON	X	NON	LA2396
Pto		*Pseudomonas syringae* pv *tomato* resis.		Q*	SPON	RG	NIL	LA3342
Pto	*2*	*Pseudomonas syringae* pv *tomato* resis.		Q*	SPON	RH13	NON	LA3129
Pto	*Pto-2*	*Pseudomonas syringae* pv *tomato* resis.	*Pto-2*	Q*	SPON	pim	NON	LA2934
Pts		Petroselinum		J*	SPON	VF36	NIL	LA2532
pu		pulvinata	*pul*	K*J	RAD	RR	IL	LA0621
pu	*2*	pulvinata	*pu2*	K*J	RAD	CR	IL	LA0973
pum		pumila		K*	RAD	CR	IL	LA0567
pum		pumila		K*	RAD	AC	NIL	LA3741
pun		punctata	*pun1*	J*DGKT	RAD	RR	IL	LA0974
pur		purilla		K*C	RAD	CR	NON	LA0568
px		praecox	*px1*	K*JOZ	RAD	LU	IL	LA0856
py		pyramidalis		K*CJT	RAD	RR	IL	LA2055
pyl		*Pyrenochaeta lycopersici* resist.	*py, py-1*	Q*	SPON	X	NON	LA2531A
r		yellow flesh		P*	SPON	RU	NIL	LA2997
r		yellow flesh		P*	SPON	C37	NIL	LA3003
r		yellow flesh		P*	SPON	AC	NIL	LA3532
r	*(2s)*	yellow flesh	*r^3, r-2, r2*	P*	RAD	RR	IL	LA2056
r	*prov4*	yellow flesh	*r*	P*	SPON	PSN	IL	2-141
r	*prov5*	yellow flesh	*r*	P*	SPON	EPK	IL	LA0353
ra		rava		D*CIJK	RAD	CR	IL	LA0569
ra	*2*	rava	*gri*	D*CIJK	RAD	RR	IL	LA0678
rd		reduced		K*	SPON	X	NON	LA2459B
re		reptans		K*	RAD	RR	IL	LA0624
rela		relaxata		K*D	RAD	AC	NIL	LA3757
rela		relaxata		K*D	RAD	CR	IL	LA0622
rep		repens		K*J	RAD	CR	IL	LA0623
rep-2		repens-2		K*J	RAD	LU	IL	LA2057

Gene	Allele	Locus name	Synonyms	Class	Origin	Back.	Iso.	Accession
res		restricta	*res1*	C*ADJK	RAD	RR	IL	LA1085
res		restricta	*res1*	C*ADJK	RAD	AC	NIL	LA3756
Rg-1		Regeneration-1			SPON	GT	NON	LA4136
ri		ridged	*rl*	J*R	RAD	X	NON	LA1794
ri		ridged	*rl*	J*R	RAD	AC	NIL	LA3180
ria		rigidula	*ria1*	C*JKT	RAD	CR	IL	LA0825
ria	*2*	rigidula	*ria1^2*	C*JKT	RAD	LU	IL	LA0975
rig		rigida		C*K	RAD	CR	IL	LA0699
rig	*2*	rigida	*pca, pca1*	C*K	RAD	LU	IL	LA0822
rig-2		rigida-2		C*K	RAD	AC	NIL	LA3716
rin		ripening inhibitor		P*	SPON	X	NON	LA1795
rin		ripening inhibitor		P*	SPON	RU	NIL	LA3012
rin		ripening inhibitor		P*	SPON	AC	NIL	LA3754
rl		radial cracking resistance	*ra*	O*	SPON	AC	NIL	LA3092
ro		rosette		K*	RAD	X	NON	LA0270
roa		rotundata	*roa1*	J*DK	RAD	CR	IL	LA0976
rot		rotundifolia		J*K	RAD	RR	IL	LA0700
rot		rotundifolia		J*K	RAD	AC	NIL	LA3751
Rs		Root suppressed		R*	RAD	X	NON	LA1796
rt		potato virus Y resistance		Q*	SPON	SCZ	IL	LA1995
rtd		retarded dwarf		J*K	SPON	X	NON	LA1058
ru		ruptilis		J*D	RAD	CR	IL	LA0626
ru		ruptilis		J*D	RAD	AC	NIL	LA3440
ru	*prov2*	ruptilis	*ru*	J*D	CHEM	VF36	IL	3-081
rust		rustica		K*J	RAD	LU	IL	LA0573
rust		rustica		K*J	RAD	AC	NIL	LA3766
rv-2		reticulate virescent-2		D*C	CHEM	SX	IL	LA2011
rvt		red vascular tissue		X*	SPON	X	NON	LA1799
s		compound inflorescence		M*	SPON	AC	NIL	LA3181
s		compound inflorescence		M*	SPON	X	NON	LA0330
sa		sphacelata	*sa1*	H*CK	RAD	CR	IL	LA0865
sar		squarrulosa	*sar1*	K*	RAD	CR	IL	LA0978
scf		scurfy		J*	SPON	PCV	NON	LA0767
scl		seasonal chlorotic lethal		C*	SPON	X	NON	LA1007
sd		sun dwarf		K*	SPON	X	NON	LA0015
sd		sun dwarf		K*	SPON	AC	NIL	LA3182
Se		*Septoria lycopersici* resistance		Q*	SPON	X	NON	LA1800
sem		semiglobosa		K*JT	RAD	CR	IL	LA0701
ses		semisterilis	*ses1*	C*DKN	RAD	LU	IL	LA0826
sf		solanifolia		J*LO	SPON	AC	NIL	LA3674
sf		solanifolia		J*LO	SPON	PSN	IL	2-311
sf	*wl*	solanifolia	*wl, wr*	J*LO	CHEM	ROMA	IL	LA2012
sfa		sufflaminata	*sfa1*	C*AEK	RAD	RR	IL	LA0862
sfa	*2*	sufflaminata	*par*	C*AEK	RAD	CR	IL	LA0969
sft		single flower truss		M*	SPON	PTN	IL	LA2460
sh		sherry		P*	RAD	CX	IL	LA2644
sha		short anthers		L*N	CHEM	ROMA	IL	LA2013
si		sinuata		E*JK	RAD	RR	IL	LA0993
si		sinuata		E*JK	RAD	AC	NIL	LA3728B
sig-1		signal transduction-1	*JL1*	Y*	CHEM	CSM	IL	LA3318
sig-2		signal transduction-2	*JL5*	Y*	CHEM	CSM	IL	LA3319
sit		sitiens		W*HJKY	RAD	RR	IL	LA0574
Skdh-1	*1*	Shikimic acid dehydrogenase-1		V*	SPON	pen	NON	LA2439
sl		stamenless		L*N	SPON	X	NON	LA0269

Gene	Allele	Locus name	Synonyms	Class	Origin	Back.	Iso.	Accession
sl		stamenless		L*N	SPON	AC	NIL	LA3816
sl	*cs*	stamenless	*cs, sl^5, sl5*	L*N	SPON	ONT	IL	LA1789
sl-2		stamenless-2	*sl2*	L*N	SPON	X	NON	LA1801
slx		serrate lax leaf		J*	SPON	PCV	NON	LA0503
Sm		*Stemphyllium* resistance		Q*	SPON	X	NON	LA1802
Sm		*Stemphyllium* resistance		Q*	SPON	MM	IL	LA2821
sn		singed		I*	SPON	CX	IL	LA2015
snt		Snout	*sn*	O*	SPON	X	NON	LA0499
so		soluta		J*	RAD	LU	IL	LA2058
sp		self-pruning		K*	SPON	X	NON	LA0154
sp		self-pruning		K*	SPON	X	NON	LA0490
sp		self-pruning		K*	SPON	GRD	NIL	LA3133
sp	*+*	self-pruning		K*	SPON	M-82	NIL	LA4287
sp	*prov2*	self-pruning		K*	RAD	spVCH	IL	LA2705
spa		sparsa		E*BK	RAD	CR	IL	LA0703
spe		splendida	*spe1*	C*K	RAD	RR	IL	LA0977
sph		sphaerica		K*T	RAD	CR	IL	LA0704
sph		sphaerica		K*T	RAD	AC	NIL	LA3744
Spi	*2*	Sympodial index		K*	SPON	pen	NON	LA0716
spl		splendens	*spl1*	C*DJ	RAD	LU	IL	LA0821
spl		splendens	*spl1*	C*DJ	RAD	AC	NIL	LA3282
squa		squarrosa		D*KU	RAD	LU	IL	LA0627
sr		slender stem	*sm*	J*KU	RAD	CT	IL	LA1803
ss		spongy seed		S*	RAD	AC	NIL	LA3619
sta		stabilis		K*	RAD	RR	IL	LA2060
ste		sterilis		J*DKN	RAD	CR	IL	LA0705
stri		stricta		J*K	RAD	LU	IL	LA0575
stu		stunted		J*	SPON	X	NON	LA2461
su		suffulta		C*JM	RAD	LU	IL	LA0628
su	*2*	suffulta	*exa*	C*JM	RAD	RR	IL	LA0853
su	*3*	suffulta	*di*	C*J	RAD	CR	IL	LA0599
su	*ni*	suffulta	*di^ni, ni*	C*J	RAD	CR	IL	LA0616
sua		suffusa		D*CK	RAD	RR	IL	LA0707
sub		subtilis		J*K	RAD	LU	IL	LA0576
suc		succedanea		C*JK	RAD	CR	IL	LA0706
sucr		sucrose accumulator	*TIV1*	P*	SPON	H100	NIL	LA4104
suf		sufflava		D*	RAD	CR	IL	LA0577
suf		sufflava		D*	RAD	AC	NIL	LA3569
sulf	*vag*	sulfurea		G*N	RAD	X	NON	LA4351
sup		superba		K*JT	RAD	RR	IL	LA2061
Sw-5		Spotted wilt resistance-5		Q*	SPON	X	NON	LA3667
sy		sunny	*ye*	F*CE	RAD	AC	NIL	LA3553
syv		spotted yellow virescent		F*CG	SPON	PCV	NON	LA1096
t		tangerine		P*L	SPON	X	NON	LA0030
t		tangerine		P*L	SPON	RU	NIL	LA3002
t		tangerine		P*L	SPON	AC	NIL	LA3183
t	*v*	tangerine		P*L	RAD	CX	IL	LA0351
ta		tarda		D*JK	RAD	CR	IL	LA0708
tab		tabescens		E*HJK	RAD	RR	IL	LA0629
tab		tabescens		E*HJK	RAD	AC	NIL	LA3734
tc		turbinate corolla		L*K	CHEM	SM	IL	LA2017
te		terminata	*te1*	K*LMO	RAD	LU	IL	LA0861
tem		tempestiva	*tem1*	K*DJ	RAD	CR	IL	LA0979
ten		tenuis		Y*DK	RAD	CR	IL	LA0578

Gene	Allele	Locus name	Synonyms	Class	Origin	Back.	Iso.	Accession
ten		tenuis		Y*DK	RAD	AC	NIL	LA3748
tf		trifoliate	*ct, tri*	J*KN	SPON	X	NON	LA0512
tf	*2*	trifoliate	*tri*	J*KN	RAD	CR	IL	LA0579
ti		tiny plant		K*	SPON	X	NON	LA1806
tl		thiaminless		Y*C	SPON	X	NON	LA0758
tl		thiaminless		Y*C	SPON	AC	NIL	LA3712
Tm		Tobacco mosaic virus resistance		Q*	SPON	X	NON	LA2369
Tm-2		Tobacco mosaic virus resist.-2	*Tm2*	Q*	SPON	VD	NIL	LA3027
Tm-2	*a*	Tobacco mosaic virus resist.-2	*Tm-2^2*	Q*	SPON	MM	NIL	LA3310
Tm-2	*a*	Tobacco mosaic virus resist.-2	*Tm-2^2*	Q*	SPON	AC	NIL	LA3769
Tm-2	*a*	Tobacco mosaic virus resist.-2	*Tm-2^2*	Q*	SPON	VD	NIL	LA3028
tmf		terminating flower		K*M	SPON	X	NON	LA2462
tn		tenera		K*U	RAD	LU	IL	LA2062
tp		tripinnate leaf		J*K	RAD	X	IL	LA0895
tp		tripinnate leaf		J*K	RAD	AC	NIL	LA3184
Tpi-2	*1*	Triosephosphate isomerase-2		V*	SPON	pen	NON	LA2440
tr		truncata	*tr1*	D*CJK	RAD	CR	IL	LA0710
tri	*1*	temporarily red light insensitive	*phyB1*	AKY*	CHEM	GT	IL	LA3808
tri	*1*	temporarily red light insensitive	*phyB1*	AKY*	CHEM	MM	NIL	LA4357
trs		tristis		J*	CHEM		NON	3-057
Ty-1		TYLCV resistance-1		Q*	SPON	X	NIL	LA3473
u		uniform ripening	*u1*	P*	SPON	LRD	IL	LA0643
u		uniform ripening	*u1*	P*	SPON	GRD	NIL	LA3035
u		uniform ripening	*u1*	P*	SPON	AC	NIL	LA3247
u	*G*	uniform ripening		P*	SPON	X	NON	LA1018
ub		umbraculiformis		J*K	RAD	LU	IL	LA2063
uf		uniflora		M*	SPON	PTN	IL	LA1200
uf		uniflora		M*	SPON	AC	NIL	LA2936
ug		uniform gray-green	*u2*	P*	SPON	OGA	IL	LA0021
ug		uniform gray-green	*u2*	P*	SPON	AC	NIL	LA3539
ul		upright leaf		K*	SPON	X	NON	LA2463
um		umbrosa		K*JRT	RAD	CR	IL	LA0630
um		umbrosa		K*JRT	RAD	AC	NIL	LA3733
uni		unicaulis		K*	RAD	CR	IL	LA0580
up		upright pedicel		L*	SPON	FLD	IL	LA2397
upg		upright growth		K*	SPON	X	NON	LA2464A
v-2		virescent-2	*v2*	F*D	SPON	X	NON	LA2465
v-2		virescent-2	*v2*	F*D	SPON	AC	NIL	LA3185
v-3		virescent-3	*V3*	F*B	RAD	X	NON	LA2707
va	*dec*	varia		F*E	RAD	CR	IL	LA0581
va	*dec*	varia		F*E	RAD	AC	NIL	LA3669
va	*virg*	varia		F*E	RAD	CR	IL	LA0582
var		variabilis		D*EK	RAD	CR	IL	LA0583
Ve		*Verticillium* resistance		Q*	SPON	GRD	NIL	LA3038
Ve		*Verticillium* resistance		Q*	SPON	AC	NIL	LA3277
Ve		*Verticillium* resistance		Q*	SPON	MM	NIL	LA2818
ven		venosa		J*BDK	RAD	X	NON	LA0888
ven		venosa		J*BDK	RAD	AC	NIL	LA3564
ver		versicolor	*yv-4, ver1*	G*C	RAD	CR	IL	LA0632
ves		versiformis	*ves1*	J*P		pim	IL	LA0859
ves-2		versiformis-2	*vf*	C*JK	RAD	LU	IL	LA1078
vg		vegetative		L*N	SPON	AC	NIL	LA2916
vga		virgulta	*vga1*	D*EFK	RAD	RR	IL	LA0858
vi		villous		I*	SPON	X	NON	LA0759

Gene	Allele	Locus name	Synonyms	Class	Origin	Back.	Iso.	Accession
vio		violacea		D*A	RAD	LU	IL	LA0633
vio		violacea		D*A	RAD	AC	NIL	LA3734A
vir		viridis		T*J	RAD	CR	IL	LA0585
vlg		virescent light green		F*D	CHEM	VF36	IL	3-128
vms		variable male-sterile		N*L	SPON	SM	IL	2-219
vo		virescent orange		F*CP	SPON	RU	NIL	LA2995
vo		virescent orange		F*CP	SPON	ROVF	IL	LA1435
vra		viridula	*vra1*	D*JK	RAD	CR	IL	LA0857
vt		vieta		J*CFK	RAD	LU	IL	LA2064
w		wiry		J*LN	RAD	CX	NON	LA0274
w-3		wiry-3	*w3, w2*	J*LN	RAD	FEY	NON	LA1498
w-4		wiry-4	*w4*	J*LN	SPON	PSN	IL	2-237
w-6		wiry-6		J*	RAD	RR	IL	LA2065
Wa		White anthers		L*	SPON	VF36	NIL	LA3906
wd		wilty dwarf		R*K	SPON	SM	IL	2-110
wf		white flower		L*	RAD	X	NON	LA0023
wf		white flower		L*	RAD	AC	NIL	LA3575
Wlt		Wilty		W*	SPON	LGPL	NON	LA3203
Wo		Wooly		I*	SPON	AC	NIL	LA3186
Wo		Wooly		I*	SPON	X	IL	LA0053
Wo	*m*	Wooly		I*	SPON	RU	IL	LA0258
Wo	*m*	Wooly		I*	SPON	AC	NIL	LA3718
Wo	*mz*	Wooly		I*	SPON	VF145	IL	LA1908
Wo	*v*	Wooly		I*	SPON	RU	IL	LA1531
Wo	*v*	Wooly		I*	SPON	AC	NIL	LA3560
wt		wilty		J*W	SPON	X	NON	LA0030
wv		white virescent		F*B	SPON	AC	NIL	LA3187
wv		white virescent		F*B	SPON	X	NON	LA0659
wv-2		white virescent-2		F*B	SPON	X	NON	LA1150
wv-3		white virescent-3		F*B	SPON	X	NON	LA1432
x		gametophytic factor		N*	SPON	X	NON	LA2348
Xa		Xanthophyllic		C*	SPON	X	NON	LA2470
Xa		Xanthophyllic		C*	SPON	AC	NIL	LA3579
Xa-2		Xanthophyllic-2	*Xa2, A*	C*	RAD	X	NON	LA4134
Xa-2		Xanthophyllic-2	*Xa2, A*	C*	RAD	X	NON	LA2471
Xa-2		Xanthophyllic-2	*Xa2, A*	C*	RAD	AC	NIL	LA3188
Xa-3		Xanthophyllic-3	*Xa3*	C*	RAD	CR	IL	LA2472
Xa-3		Xanthophyllic-3	*Xa3*	C*	RAD	AC	NIL	LA3430
xan-2		xantha-2	*xan2*	C*	RAD	AC	NIL	LA3759
xan-4		xantha-4	*xan4*	C*	RAD	AC	NIL	LA3760
y		colorless fruit epidermis		P*	SPON	OGA	NON	LA1088
y		colorless fruit epidermis		P*	SPON	AC	NIL	LA3189
yg-2		yellow-green-2	*yc, yg282, yg2*	E*	RAD	AC	NIL	LA3551
yg-2		yellow-green-2	*yc, yg282, yg2*	E*	RAD	KK	IL	LA2469A
yg-2	*aud*	yellow-green-2	*yg-2^r, aud*	E*	SPON	AC	NIL	LA3165
yg-2	*aud*	yellow-green-2	*yg-2^r, aud*	E*	SPON	X	NON	LA1008
yg-3		yellow-green-3	*yg3, yg330, ye*	E*	RAD	KK	NIL	LA2926
yg-4		yellow-green-4	*yg4, yl, yg333*	E*J	RAD	KK	NIL	LA2927
yg-4		yellow-green-4	*yg4, yl, yg333*	E*J	RAD	AC	NIL	LA3731
yg-5		yellow-green-5	*yw, yg388, yg5*	E*	RAD	AC		LA2928B
yg-5		yellow-green-5	*yw, yg388, yg5*	E*	RAD	RCH	NIL	LA2928
yg-5		yellow-green-5	*yw, yg388, yg5*	E*	RAD	AC	NIL	LA2928A
yg-9		yellow-green-9		E*	SPON	C28	IL	LA2708
yv		yellow virescent		E*	SPON	AC	NIL	LA3554

Gene	Allele	Locus name	Synonyms	Class	Origin	Back.	Iso.	Accession
yv		yellow virescent		E*	SPON	SM	IL	LA0055
yv	*2*	yellow virescent	*vel^2, vel1^2*	E*	RAD	CR	IL	LA0981
yv	*3*	yellow virescent	*vel*	E*	RAD	CR	IL	LA0631
yv	*ms*	yellow virescent		E*N		X		LA3907
yv-2		yellow virescent-2		E*	SPON	AC	NIL	LA3190
yv-4		yellow virescent-4		E*	SPON	AC	NIL	LA3570

Table 2. Definition of phenotypic class symbols listed in Table 1.

Class	Description
A	Anthocyanin modifications: intensification, reduction, elimination
B	Chlorophyll deficiency: white or whitish
C	Chlorophyll deficiency: yellow or yellowish
D	Chlorophyll deficiency: light, grey, or dull green
E	Chlorophyll deficiency: yellow-green
F	Virescent: chlorophyll deficiency localized at growing point
G	Variegation, flecking or striping
H	Leaf necrosis
I	Hair modifications: augmentation, reduction, distortion, elimination
J	Leaf form and size
K	Plant habit and size
L	Flower form and color
M	Inflorescence (exclusive of L)
N	Sterility: any condition leading to partial or complete unfruitfulness
O	Fruit form and surface texture
P	Fruit color and flavor, ripening modification
Q	Disease resistance
R	Root modification
S	Seed
T	Foliage color: dark
U	Foliage color, miscellaneous: olive, brown, blue-green
V	Allozyme variant
W	Overwilting stomatal defect
X	Vascular modification
Y	Nutritional or hormonal disorder
Z	Precocious development

Table 3. Definition of abbreviations used for background genotypes in Table 1, and their corresponding accession numbers (n/a = not available)

Back.	Genotype name	Acc.#
A-1	A-1	LA0818
AC	Ailsa Craig	LA2838A
ACE	Ace	LA0516
ALA	Alabama	n/a
AMB	Antimold-B	LA3244
ANU	Anahu	LA3143
BK	Budai Korai	n/a
BOD	Break O'Day	LA1499
C255	Cal 255	LA0198
C28	Campbell 28	LA3317
cer	*L. esc*. var. *cerasiforme*	many
CG	Chico Grande	LA3121
che	*L. cheesmanii*	many
chi	*L. chilense*	many
chm	*L. chmielewskii*	many
CR	Condine Red	LA0533
CRGL	Craigella	LA3247
CSM	Castlemart	LA2400
CT	Chatham	n/a
CX	Canary Export	LA3228
E6203	E-6203	LA4024
EPK	Earlipak	LA0266
ERL	Earliana	LA3238
ESC	Early Santa Clara	LA517
FB	Fireball	LA3024
FEY	First Early	n/a
FLD	Flora-Dade	LA3242
GRD	Gardener	LA3030
GSM	Gulf State Market	LA3231
H100	Hunt 100	LA3144
hir	*L. hirsutum*	many
HSD	Homestead 24	LA3237
JBR	John Baer	LA1089
KK	Kokomo	LA3240
LGPL	Large Plum	LA3203
LK	Laketa	LA0505
LRD	Long Red	LA3232
LU	Lukullus	LA0534
lyc	*S. lycopersicoides*	many
M167	Montfavet 167	LA2713
M82	M-82	LA3475
M168	Montfavet 168	LA2714
MD	Marmande	LA1504
MGB	Marglobe	LA0502
MM	Moneymaker	LA2706
MNB	Monalbo	LA2818
MP	Manapal	LA2451
NRT	Norton	n/a
O8245	Ohio 8245	n/a
OGA	Ohio Globe A	LA1088
ONT	Ontario	n/a
par	*L. parviflorum*	many
PCV	primitive cultivar	n/a
pen	*L. pennellii*	many

Back.	Genotype name	Acc.#
per	*L. peruvianum*	many
pim	*L. pimpinellifolium*	many
PLB	Pieralbo	n/a
POR	Porphyre	LA2715
PRI	Primabel	LA3903
PRN	Prairiana	LA3236
PRT	Pritchard	LA3233
PSN	Pearson	LA0012
PSP	Prospero	LA3229
PTN	Platense	LA3243
RCH	Red Cherry	LA0337
RG	Rio Grande	LA3343
RH13	Rehovot 13	LA3129
RNH	Rouge Naine Hative	n/a
ROMA	Roma	n/a
ROVF	Roma VF	n/a
RR	Rheinlands Ruhm	LA0535
RSWT	Roumanian Sweet	LA0503
RTVF	Red Top VF	LA0276
RU	Rutgers	LA1090
SCZ	Santa Cruz	LA1021
SM	San Marzano	LA0180
spVCH	VFNT Cherry (sp)	LA2705
SPZ	San Pancrazio	n/a
STD	Stokesdale	LA1091
STN	Stone	LA1506
STR24	Start 24	LA3632
SX	Sioux	LA3234
T338	UC-T338	LA2939
T-5	UC-T5	LA2399
TGR	Targinnie Red	LA3230
TVD	Vendor (Tm-2a)	LA2968
UC82	UC-82B	LA1706
VCH	VFNT Cherry	LA1221
VD	Vendor	LA3122
VE	Van's Early	n/a
VF11	VF-11	LA0744
VF145	VF-145 78-79	LA1222
VF36	VF-36	LA0490
VF6	VF-6	LA0743
VFN8	VFN-8	LA1022
VFSM	VF San Marzano	n/a
VGB	Vagabond	LA3246
VRB	Vrbikanske nizke	LA3630
VTG	Vantage	LA3905
WA	Walter	LA3465
X	unknown or hybrid	n/a
XLP	XL Pearson	n/a

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In Memoriam *Information for this memoriam has been drawn from the 1994 nomination to H.R. MacMillan Laureate in Agriculture, prepared by Long Point Branch, Ontario Institute of Agrologists on May 19, 1994.*

ERNEST ANDREW KERR, B.A., M.Sc., Ph.D., F.A.I.C., P.Ag.

ERNEST ANDREW KERR was born near Guelph, Ontario on 24 August 1917. He was educated at Morriston Public School and Guelph Collegiate Vocational Institute. In 1940, he gained his B.A. (Hons. Biology) from McMaster University and then started his life-time career in plant breeding. A year after graduation, Kerr was awarded a M.Sc. in genetics from McGill University with a thesis on aberrant wheats. He then went to the University of Wisconsin for a Ph.D. in genetics and plant pathology in 1944, and his thesis project concerned seed development in blackberries.

In spite of these minor diversions into cereals and fruit, vegetable breeding was to become his professional career. With the exception of a stint in the Canadian Army Medical Corps at the end of World War II, Dr. Kerr was employed by the Province of Ontario from 1944 to 1982. He was first appointed as Research Assistant in vegetable breeding at the then Horticultural Experiment Station at Vineland Station, Ontario. He advanced to become Chief Research Scientist in charge of all plant breeding at Vineland Station in 1954, and in the fall of 1970 was appointed Research Coordinator of Horticultural Production Systems - in addition to his already very heavy personal programs in breeding tomatoes and sweet corn.

On 1 July 1972, Dr. Kerr transferred to the Horticultural Experiment Station, Simcoe, Ontario to concentrate on breeding processing tomatoes, with minor projects in greenhouse tomatoes and sweet corn. He was subsequently promoted Research Scientist 5, at the time the only one in the Horticultural Research Institute of Ontario, in recognition of his world-wide reputation as a vegetable breeder.

During his 38 years of employment with the Ontario government, Dr. Ernie Kerr's productivity of new cultivars, research papers and other publications was nothing short of prolific. Over 50 named cultivars were released including the internationally accepted greenhouse tomato Vendor, field tomatoes Veepro, Veebrite, Basketvee, Wondervee and Veeking, and sweet corns Earlivee, Tastyvee and Flavorvee. In addition, many new sweet corn inbreds were released to other breeders.

Dr. Kerr was elected an associate member of the Canadian Seed Growers' Association in 1971. In 1980, he was presented with the "Man of the Year" Award by the Canadian Seed Trade Association - the first ever to a horticulturist. The Agricultural Institute of Canada conferred a Fellowship on Dr. Kerr in 1981.

Following his formal retirement in 1982, at age 65, Dr. Kerr began another full-time career, becoming the first research director appointed by Stokes Seeds Ltd. in St. Catharines, Ontario. His work with this large commercial vegetable seed company continues in plant breeding with a range of vegetables e.g. asparagus, peppers, sweet corn, and tomatoes. His managerial duties have involved the establishment of a completely new staff unit for Stokes Seeds in the area of plant breeding, seed

research and crop development, which has strengthened this company's position in the vegetable seed industry.

In spite of a very full professional life, Dr. Kerr still found time for outside activities:
member of the Agricultural Institute of Canada and past president of the Niagara Branch; charter member of the Ontario Institute of Agrologists; member of the Canadian Society for Horticultural Science and past chairman of the Ontario/Quebec section; past chairman of and prolific contributor to the Tomato Breeders Round Table; past chairman of the editorial board of the Canadian Journals of Plant, Animal and Soil Science; past associate editor of "Horticultural Research" (Scotland); member of The Rotary Club of Simcoe; faithful worker for the United Church and former Sunday School Superintendent, and currently attends Old Windham United Church, Simcoe, Ontario.

Ernie Kerr and Olive Gordon were married in Thombury, Ontario on 1 September 1945 and they have three children: Gordon has a Ph.D. in animal behaviour from the University of Toronto. Douglas is an electronics engineering technologist graduate from Niagara College, Betty Lou has an M.B.A. from Queens. Five grandchildren complete the Kerr family and kept Ernie and Olive occupied when they were not walking the tomato and sweet corn research plots together.

Dr. Kerr was indeed, a unique Canadian. Even at the age of 77 years, he continued to accomplish plant breeding feats that have escaped plant breeder colleagues world-wide. During the course of his long, active and prodigiously successful career, Ernie Kerr had steadfastly rejected all opportunities offered to direct his research efforts towards the improvement of vegetable producers outside of Canada. However, the influence of his research in Canada is significant and seriously pursued by plant scientists and food producers world-wide.

Food production is not perceived by many Canadians to be particularly exciting. A well authored account of the long, successful career of Dr. Kerr will impact very few people, compared to a sports celebrity, a medical scientist, a business mogul, a politician, an entertainer, etc. However, the result of Dr. Kerr's research must surely be ranked in the very top tier of Canadian scientific achievements. He has manipulated the characteristics of plants, through selective breeding, in order that they may be grown in the harsh environment of Canada and other countries in the world. The impact of his success in manipulating plants to be resistant to disease, insects, birds, etc., is not yet possible to measure in economic, health or ecological terms.