PHOTOMORPHOGENIC MUTANTS OF TOMATO

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I. Abstract

Tomato (Lycopersicon esculentum Mill.) has been chosen as a model species for the study of photomorphogenesis. The aurea (au) and yellow-green-2 (yg-2) mutants which are severely phytochrome deficient appear to be phytochrome chromophore mutants. Mutants modified with respect to specific members of the phytochrome gene family: the far-red light-insensitive mutant (fri, for phytochrome A) and the temporarily red light-insensitive mutant (tri, for phytochrome B1) have been identified. Mutants that exhibit an exaggerated phytochrome response are putative transduction-chain mutants affecting an amplification step in phytochrome signal transduction. These mutants are being used to understand the complexities of juvenile anthocyanin in the hypocotyl during seedling de-etiolation.

II. Photomorphogenesis

The word photomorphogenesis is used as a collective term for the way plants process the information content of the light environment (e.g. light quantity, quality, duration and direction) and modify their growth and development accordingly (Kendrick and Kronenberg 1994). Processes are controlled at all stages of the life cycle of a plant, from germination to the induction of flowering. One stage in the life cycle of a plant where light plays a critical role is at the time of seedling establishment after germination, initiating the transition from heterotrophic growth utilizing stored seed reserves to a green autotrophic self-sufficient photosynthetic plant (deetiolation). Once established many seedlings exhibit the so called shadevoidance' response. Seedlings growing in the shade of

other plants are clearly disadvantaged, since the photosynthetic pigments of the leaves above ill absorb much of the available light. The early detection of the shade of ther plants, gives the opportunity to a plant to modify its growth by more apid elongation and so compete with its neighbours for the available hotosynthetic light. To achieve this process the red light (R)/far-red light (FR)-reversible photoreceptor phytochrome has evolved enabling a plant to ontinuously and accurately monitor the spectral quality of the light. Shade light has more FR than R, whereas open sunlight has slightly more R than FR (Smith 1994). The photoreceptor phytochrome is not only used in this response, but also at the time of de-etiolation and in many species functions in concert with UV-B, UV-A and blue light (B) photoreceptors. The most abundant phytochrome in plants accumulates in dark-grown seedlings and it was this phytochrome that was initiall y isolated and has been most exten sively studied. However the development of molecular biological approaches has led to the discovery that there is a small gene family of phytochromes, which in Arabidopsis have been called phytochromes A, B, C, D and E (phyA, phyB, phyC, phyD and phyE) and are encoded by the genes PHYA, PHYB, PHYC, PHYD and PHYE, respectively (Sharrock and Quail 1989; Clack *et al.*, 1994; Quail *et al.*, 1994). The tetrapyrrole chromophore gives phytochrome its capacity to absorb in the R and FR regions of the spectrum and the apoprotein part of the molecule to which it is attached is designated PHYA, PHYB, PHYC, PHYD and PHYE. Antibodies have been raised that can discriminate between some of these phytochrome apoproteins. Molecular and genetic approaches have provided evidence that these phytochromes can have both discrete and overlapping functions (Quail 1994; Koornneef and Kendrick 1994).

Phytochrome responses can be subdivided into response modes on the basis of the amount of light required: very-low fluence responses (VLFRs); low fluence responses (LFRs); high irradiance responses (HIRs). There is also evidence of interactions among the different classes of photoreceptors. To unravel this complexity of photomorphogenesis during de-etiolation, mutants are being studied in which elements of the system are modified (hopefully creating a more simplified photomorphogenesis). In this article progress using tomato as a model plant species is outlined

III. Tomato as a Model Plant Species

Tomato (Lycopersicon esculentum Mill.) has been chosen as an alternative model

plant species to the almost universally adopted Arabidopsis (Table 1). Tomato compares quite favourably in many respects and has the advantage that the seeds are relatively large and develop into seedlings which are more amenable to physiological and biochemical analysis. Perhaps most important from the point of view of our understanding of the control of plant growth and development in general is that we do not forget that the growth of a cruciferous rosette plant, such as Arabidopsis, may not be typical of all plants within the plant kingdom. Therefore parallel studies on other species are of great potential value. In addition, being an economically important crop, tomato is genetically well characterized and many mutants already exist, some of which have been shown to be photomorphogenic mutants. There are at least five phytochrome genes in tomato which have been designated PHYA, PHYB1, PHYB2, PHYX and PHYZ to which specific DNA probes have been produced (Cordonnier-Pratt *et al.*, 1994)

Table 1. Comparison oftomato and Arabidopsis as model plant (modified from Kendrick et al. 1994b)

	Arabidopsis	Tomato
Haploid chromosome No.	5	12
Haploid genome (kbp)	7×10^4	7.1×10^5
Genetic maps available	+	+
Generation time (months)	2	6
Transformation possible	+	+
Tagging techniques available	+	+
Seeds and seedlings	small	large

IV. Photomorphogenic Mutants

Deficiency mutants. The aurea (au) and yellow green-2 (yg-2) mutants were the first tomato mutants demonstrated to be severely phytochrome deficient in spectrophotometrically active phytochrome and to have the corresponding pleiotropic phenotype expected for such a mutant at the time of de-etiolation: taller and paler green than the corresponding wild type (WT) (Koornneef *et al.*, 1985). However, despite being essentially blind to R at the seedling stage de-etiolation does take place, although with difficulty, in white light (WL) and it has been argued that this is facilitated by the co-action of a small residual phytochrome pool and one or more B/UV-A photoreceptors (Oelmler and Kendrick 1991). In addition, WL-grown plants, despite being paler in colour than WT do exhibit response to

vegetational shade and in the laboratory exhibit a strong, related response to daily end-of-day FR (EODFR) (Adamse *et al.*, 1988; Peters 1992b). It is therefore quite clear that the au mutant can synthesis some spectrophotometrically active phytochrome in older plants since such responses are R/FR reversible. All the evidence to date (see Kendrick *et al.*, 1994a for review), which is largely circumstantial, points to these mutants being modified with respect to biosynthesis of the tetrapyrrole chromophore (phytochromobilin) which is common to all phytochromes. Since the au mutant is likely to be deficient in all phytochromes it cannot be used to assign function to any individual member of the phytochrome gene family in tomato. At least five phytochrome genes have been shown to be expressed in tomato (Hauser *et al.*, 1994): PHYA, PHYB1, PHYB2, PHYX (which might be equivalent to Arabidopsis PHYE), and PHYZ (which is distinct from any previously described phytochrome). These genes show their own discrete pattern of expression throughout the life cycle of the plant (Hauser *et al.*, 1994). Southern analysis indicates further phytochrome-like sequences in the tomato genome anntherefore there is a high probability that additional phytochrome genes are expressed.

In a search for type-specific phytochrome mutants several new long-hypocotyl mutants have been selected under low fluence rate B and R. Two of these mutants which are allelic were subsequently shown to be more or less completely blind to FR. This locus has been named FR insensitive (fri) (van Tuinen *et al.*, 1994). Mutants at this recessive locus have been shown to lack the bulk pool of phytochrome in etiolated seedlings (predominantly phyA) and immunologically detectable PHYA (van Tuinen *et al.*, 1994). In addition, Northern analysis shows the PHYA mRNA is modified in the fri mutants (L.H.J. Kerckhoffs pers. comm.). The fri locus has been mapped to chromosome 10, as has the PHYA gene (A. van Tuinen pers. comm.). Since the au and yg-2 mutants map to chromosomes 1 and 12, respectively, this is an additional piece of evidence pointing to them not being specifically phyA deficient. Young WL- grown fri-mutant plants are almost indistinguishable from the WT, but one interesting observation is that on sunny days in the greenhouse older fri- mutant plants are prone to wilting, which results in retardation of growth. Provisional experiments suggest that this is not due to abnormal behaviour of stomata (van Tuinen *et al.*, 1994).

Another group of recessive long-hypocotyl mutants selected under WL or low-fluence rate R were shown to be temporarily R insensitive (tri), being essentially blind to R during the first two days after transfer from darkness irrespective of

their physiological age (Kerckhoffs *et al.*, 1994). Four alleles have been isolated and when examined by Western analysis one of these had none (below detection limit) and one had a reduced amount of a PHYB-like protein as compared to the WT (Kerckhoffs *et al.*, 1994). The other two alleles had polypeptides recognized by the PHYB antibody, but were of lower molecular masses than the PHYB-like protein in the WT. Northern analysis shows that two of the alleles have a modified PHYB1 mRNA (L.H.J. Kerckhoffs pers. comm.). The tri locus has been mapped to chromosome 1 (A. van Tuinen pers comm.). The WL-grown plants of this mutant are slightly taller than the WT, but otherwise very similar. Furthermore, the fri, tri double mutant looks essentially the same as the tri mutant in WL, demonstrating that residual phytochromes can sustain a relatively normal photomorphogenesis. One interesting observation is the fact that these phyB1-deficient tri mutants exhibit a normal EODFR response (Kerckhoffs *et al.*, 1994). This result is in contrast to phyB deficient mutants described so far in other species (Koornneef and Kendrick 1994). Since phyB1 and phyB2 are closely related we hypothesize that they might both be able to regulate the EODFR response in tomato. Such a redundancy in the phytochrome system would explain why, despite considerable effort, no constitutively tall tomato mutants have so far been found. The fri,tri double mutant is an ideal launch point for further mutagenesis and the selection of tall mutants deficient in the residual phytochromes.

Some properties of these phytochrome deficiency mutants are summarized in Table 2.

Table 2. Summary of photomorphogenic mutants tomato

Genotype	WT	*au*	*yg-2*	*fri*	*tri*	*PHYA3*$^+$	*hp-1*	*hp-2*	*Ip*	*atv*
Chromosome No.		1	12	10	1	?	?	?	?	?
Hypocotyl inhibition										
FR	+[a]	−[a]	−[a]	−[d]	+[e]	++[f]	−/+[h]	?	?	?
R	+[a]	−[a]	−[a]	+[d]	−/+[e]	++[f]	++[h]	−/+[f]	++[f]	++[f]
Adult plants										
Chlorophyll	++[a]	+[a]	+[a]	++[d]	++[e]	+++[g]	+++[e]	−/+++[f]	++[f]	++[i]
EODFR	+[b]	+[b]	+[c]	+[d]	+[e]	?	+[b]	+[c]	+[c]	+[c]
Mutant type (putative)		(Chr$^-$)	(Chr$^-$)	phyA	phyB1$^-$	phyA3$^-$	(Resp$^-$)	(Resp$^+$)	(Resp$^-$)	(Resp$^-$)

WT=wild type; EODFR=end-of-day FR response; FR=far-red light; R=red light; ?=not determined; Chr$^-$=chromophore biosynthesis deficiency; Resp$^-$=response amplification. References: [a]Koornneef *et al.* 1985; [b]Adamse *et al.* 1988; [c]L.H.J. Kerckhoffs unpublished; [d]van Tuinen *et al.* 1994; [e]Kerckhoffs *et al.* 1994; [i]A. van Tuinen unpubllished; [g]Boylan and Quail 1989; [f]Peters *et al.* 1989.

Exaggerated response mutants. A spontaneous mutant at a high pigment (hp)

locus was found as early as 1917 (Adamse *et al.*, 1989; Peters *et al.*, 1989). The monogenic recessive hp-1 mutants are characterized by features, such as, a short hypocotyl with high anthocyanin pigmentation and adult plants with dark-green foliage and immature fruit colour due to high chlorophyll levels, higher lycopene and carotene content resulting in deep-red fruits. Using continuous R at the de-etiolation stage several new hp mutants have been selected (Peters *et al.*, 1989; A. van Tuinen pers. comm.). Plant height is also somewhat reduced in hp-1 mutants. The pleiotropic nature of the hp-1 mutant suggests that it has a modification of a basic process affecting plant morphogenesis rather than being a specific response mutant affecting pigment synthesis only.

There are also other mutants which have a similar phenotype to hp-1, such as hp-2 (Soressi and Salamini 1975), atroviolatia (atv) (Rick *et al.*, 1968) and intensive pigment (Ip) (Rick 1974). Furthermore, plants with hp-1-like characteristics at their seedling stage were obtained when high levels of the oat PHYA3 gene were expressed in tomato (Boylan and Quail 1989). The hp-1 mutant of tomato exhibits exaggerated phytochrome responses, whereas the phytochrome content of etiolated seedlings and the characteristics of the phytochrome system are similar to that in WT (Adamse *et al.*, 1989; Peters *et al.*, 1989). Therefore there is so far no evidence to suggest that the hp-1 mutant is a photoreceptor mutant. In contrast to WT, the hp-1 mutant does not require co-action of the B photoreceptor and phytochrome for normal development and exhibits maximum anthocyanin synthesis and hypocotyl growth inhibition in R alone i.e. the mutation mimics the action of B. On the basis of its recessive (loss-of-function) nature it is proposed that the phytochrome action in etiolated seedlings is under the constraint of the hp-1-gene product (HP-1) (Peters *et al.*, 1992a). Both exposure to B and the hp-1 mutation appear to result in reduction of HP-1 or its effectiveness. The exaggerated response of the hp-1 mutant compared to WT fits the definition of responsiveness amplification' proposed by Mohr (1994) to describe the amplification of a phytochrome response as a result of pre-irradiation which excites either the B photoreceptor or phytochrome. It was proposed that the hp-1 mutation is associated with this amplification step in the phytochrome transduction chain (Peters *et al.*, 1992a). A study of the photoregulation of phenylalanine ammonia lyase (PAL), a key enzyme in flavonoid biosynthesis, showed a higher level in the hp-1 mutant when compared to the WT level (Goud *et al.*, 1991). Goud and Sharma (1994) demonstrated that pulses of R are effective in the induction of amylase and nitrate reductase (NR) activity in the WT and that the hp-1 mutant again exhibits an amplified response. This

amplification of synthesis of enzymes in unrelated biochemical pathways points to action of the hp-1 gene product at some fundamental point in the photo-induction transduction chain leading to modification of gene expression.

Adult plants of the hp-1 and the au,hp-1 double mutant show a quantitatively similar elongation response to reduction in R:FR photon ratio during the daily photoperiod (Kerckhoffs *et al.*, 1992) and EODFR treatments (Peters *et al.*, 1992b). However, the presence of the hp-1 gene does result in a dwarfing effect in WL-grown plants, particularly when they are grown under light sources poor in FR such as white fluorescent tubes. In addition the hp-1 and hp-2 mutations have rather similar phenotypes resulting in a very tissue specific effect in immature fruits, where large amounts of chlorophyll accumulate. In the au,hp-1 double mutant the au mutation is not completely epistatic to hp-1 in the case of WL-grown plants.

Some of the properties of these exaggerated-response mutants are summarized in Table 2.

V. Anthocyanin Biosynthesis

The au-mutant seedling has been used as a highly phytochrome-deficient starting material for investigation of phytochrome signal transduction. Neuhaus *et al.* (1993) micro-injected phyA into hypocotyl cells and elicited anthocyanin biosynthesis, as well as partial plastid development. The lack of phytochrome in the au mutant enables these manipulative experiments to be carried out in the light. Their studies revealed evidence for two parallel pathways which were both induced by activation of one or more trimeric G-proteins. The pathway within a single cell type leading to partial plastid development was also induced by injection of calcium and calmodulin, whereas the pathway to anthocyanin biosynthesis was independent of calcium. Bowler *et al.*, (1994) extended this work and provided evidence for cyclic GMP (cGMP) being an important intermediate. The anthocyanin response could be induced by micro-injection of cGMP alone and in the presence of calcium could lead to the development of fully functional plastids indicating some cross talk between the signal transduction pathways involved in the regulation of gene expression during de-etiolation.

The responses taking place during de-etiolation show a strong tissue specificity in the hypocotyl. The anthocyanin production is restricted to the single subepidermal layer of cells, whereas all cells throughout the cortex have the capacity for plastid development into chloroplasts. Apart from guard cells, which produce chloroplasts,

the epidermal cells show neither response. An extensive study of anthocyanin biosynthesis under a 24-h irradiation schedule with different fluence rates of R has been carried out (L.H.J. Kerckhoffs and M.E. Schreuder unpublished data). The WT response shows two components: a low fluence rate response and a HIR response at higher fluence rates. The fri and tri mutants essentially lack the low fluence rate and the HIR response components, respectively. Since the low fluence rate response, regulated by phyA, is only revealed at the very low light levels, anthocyanin accumulation under high fluence rate R, appears to be solely regulated by the phyB1, which is deficient in the tri mutant. Both response modes result in accumulation of anthocyanin in the same sub-epidermal cells. Overexpression of the PHYA3 gene results in an increase in anthocyanin which is predominantly located in the same tissue-specific manner. Therefore in this transgenic line overexpressed PHYA3 presumably maintains a higher than normal phyA pool, enhanced by the slower degradation of phyA3 than the endogenous tomato phyA. However, at medium to high R fluence rates the endogenous phyA in the WT is degraded before it has time to act.

VI. Concluding Remarks

The process of de-etiolation is not the same in all species and is quite complex. Thus, comparative studies with different plant species are of great interest. The study of tomato photomorphogenesis, especially as assisted by photomorphogenic mutants will consequently make a valuable complement to parallel studies with Arabidopsis. Perhaps the complexity of photomorphogenesis during de-etiolation exists because the selection pressure for this critical process in the life of a plant was so strong that several different photoreceptors, functioning in concert, have evolved to control it.

In the laboratory, we have the opportunity to reveal response characteristics for excitation of selected photoreceptors which in nature never occurs. For example, the inhibition of elongation growth of a hypocotyl can be achieved by different wavelengths of light, in a number of different ways. The application of FR functions via the FR-HIR mode of phyA, whereas R functions via a R-LFR and a R-HIR in which both phyA and other phytochromes play a role. In nature none of these processes are saturated by the low light levels below the soil surface, but collectively they enable the selective advantage of perception of the light environment (soil surface) to be anticipated.

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References

1. Adamse, P., P.A.P.M. Jaspers, J.A. Bakker, J.C. Wesselius, G.H. Heeringa, R. E. Kendrick and M. Koornneef. 1988. Photophysiology of a tomato mutant deficient in labile phytochrome. *J. Plant Physiol.* 133: 436-17.
2. Adamse, P., J.L. Peters, P.A.P.M. Jaspers, A. van Tuinen, M. Koornneef and R.E. Kendrick. 1989. Photocontrol of anthocyanin synthesis in tomato seedlings: a genetic approach. *Photochem. Photobiol.* 50: 107-111.
3. Bowler, C., G. Neuhaus, H. Yamagata and N.-H. Chua. 1994. Cyclic GMP and calcium mediate phytochrome phototransduction. *Cell* 77: 73-81.
4. Boylan, M.T. and P.H. Quail. 1989. Oat phytochrome is biologically active in transgenic tomatoes. *The Plant Cell* 1: 765-773.
5. Clack, T., S. Mathews and R.A. Sharrock. 1994. The phytochrome apoprotein family in Arabidopsis is encoded by five genes: The sequences and expression of PHYD and PHYE. *Plant Mol. Biol.* (in press).
6. Cordonnier-Pratt, M.-M., L.H. Pratt, B. Hauser, G. Kockert and M. Caboche. 1994. Comparative analysis of the phytochrome gene family in tomato (Lycopersicon esculentum Mill.) and sorghum (Sorghum bicolor [L.] Moench). *Plant Physiol.* 105 (suppl.): 72.
7. Goud, K.V., R. Sharma, R.E. Kendrick and M. Furuya. 1991. Photoregulation of phenylalanine ammonia lyase is not correlated with anthocyanin induction in photomorphogenic mutants of tomato (Lycopersicon esculentum). *Plant Cell Physiol.* 32: 1251-1258.
8. Goud, K.V. and R. Sharma. 1994. Regulation of photoinduction of cytosolic enzymes in aurea mutant of tomato (Lycopersicon esculentum). *Plant Physiol.* 105: 643-650.
9. Hauser, B., M.-M. Cordonnier-Pratt and L.H. Pratt. 1994. Differential expression of five phytochrome genes in tomato (Lycopersicon esculentum Mill.). *Plant Physiology* 105 (suppl.): 72.

10. Kendrick, R.E. and G.H.M. Kronenberg. 1994. Photomorphogenesis in Plants, Kluwer Academic Publishers, Dordrecht, The Netherlands.
11. Kendrick, R.E., L.H.J. Kerckhoffs, A.S. Pundsnes, A. van Tuinen, M. Koornneef, A. Nagatani, M.J. Terry, A. Tretyn, M.-M. Cordonnier-Pratt, B. Hauser and L.H. Pratt. 1994a. Photomorphogenic mutants of tomato. *Euphytica* (in press).
12. Kendrick, R.E., J.L. Peters, L.H.J. Kerckhoffs, A. van Tuinen and M. Koornneef. 1994b. Photomorphogenic mutants of tomato In. D. Bowles (Ed.), *Molecular Botany: Signals* and the Environment, pp xxx-xxx, Portland Press, London, UK (in press).
13. Kerckhoffs, L.H.J., R.E. Kendrick, G.C. Whitelam and H. Smith. 1992. Response of photomorphogenic tomato mutants to changes in the phytochrome photoequilibrium during the daily photoperiod. *Photochem. Photobiol.* 56: 611-616.
14. Kerckhoffs, L.H.J., A. van Tuinen, A. Nagatani, M.Koornneef and R.E. Kendrick. 1994. Temporarily red light insensitive mutants of tomato. *Plant Physiol.* 105 (suppl.): 105.
15. Koornneef, M., J.W. Cone, R.G. Dekens, E.G. O'Herne-Robers, C.J.P. Spruit and R.E. Kendrick. 1985. Photomorphogenic responses of long hypocotyl mutants of tomato. *J. Plant Physiol.* 120: 153-165.
16. Koornneef, M. and R.E. Kendrick. 1994. Photomorphogenic mutants of higher plants. In: R.E. Kendrick and G.H.M. Kronenberg (Eds.), Photomorphogenesis in Plants, pp. 601-628.
17. Kluwer Academic Publishers, Dordrecht, The Netherlands.
18. Mohr, H. 1994. Coaction between pigment systems. In: R.E. Kendrick. and G.H. M. Kronenberg (Ed.), Photomorphogenesis in Plants, pp. 353-373. Kluwer Academic Publishers, Dordrecht, The Netherlands.
19. Neuhaus, G., C. Bowler, R. Kern and N.-H. Chua. 1993. Calcium/calmodulin-dependent and- independent phytochrome signal transduction pathways. *Cell* 73: 937-952.
20. Oelmler, R. and R.E. Kendrick. 1991. Blue light is required for survival of the tomato phytochrome-deficient aurea mutant and the expression of four nuclear genes coding for plastidic proteins. *Plant Mol. Biol.* 16: 293-299.
21. Peters, J.L., A. van Tuinen, P. Adamse, R.E. Kendrick and M. Koornneef. 1989. *High pigment mutants of tomato* exhibit high sensitivity for phytochrome action. *J. Plant Physiol.* 134: 661-666.

22. Peters, J.L., M.E.L. Schreuder, S.J.W. Verduin and R.E. Kendrick. 1992a. Physiological characterization of a high pigment mutant of tomato. *Photochem. Photobiol.* 56: 75-82.
23. Peters, J.L., M.E.L. Schreuder, G.H. Heeringa, J.C. Wesselius, R.E. Kendrick and M. Koornneef. 1992b. Analysis of the response of photomorphogenetic tomato mutants to end-of-day far-red light. *Acta Hort.* 305: 67-77.
24. Quail, P.H. 1994. Phytochrome genes and their expression. In: R.E. Kendrick and G.H.M. Kronenberg (Eds.), Photomorphogenesis in Plants, pp. 71-104.
25. Kluwer Academic Publishers, Dordrecht, The Netherlands.
26. Quail, P.H., W.R. Briggs, J. Chory, R.P. Hangarter, N.P. Harberd, R.E. Kendrick, M. Koornneef, B. Parks, R.A. Sharrock, E. Scher, W.F. Thompson and G.C. Whitelam. 1994. Spotlight on phytochrome nomenclature. *Plant Cell* 6: 468-471.
27. Rick, C.M. 1974. High soluble-solids content in large-fruited tomato lines derived from a wild green-fruited species. *Hilgardia* 42: 493-510.
28. Rick, C.M. A.F. Reeves and R.W. Zobel. 1968. Inheritance and linkage relations of four new mutants. *Tomato Genet. Coop. Rpt.* 18: 34-35.
29. Sharrock R.A. and P.H. Quail. 1989. Novel phytochrome sequences in Arabidopsis thaliana: structure, evolution, and differential expression of a plant regulatory photoreceptor family. *Genes and Development* 3: 1745-1757.
30. Smith, H. 1994. Sensing the light environment: the functions of the phytochrome gene family. In, R.E. Kendrick and G.H.M. Kronenberg (Eds.), Photomorphogenesis in Plants, pp. 377-416. Kluwer Academic Publishers, Dordrecht, The Netherlands.
31. Soressi, G.P. and F. Salamini. 1975. New spontaneous or chemically-induced fruit ripening tomato mutants. *Tomato Genet. Coop. Rpt.* 25: 21-22.
32. Tuinen, A van, L.H.J. Kerckhoffs, A. Nagatani, R.E. Kendrick and M. Koornneef. 1994. Far-red light-insensitive mutants of tomato. *Mol. Gen. Genet.* (in press).