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Genetic analysis of salt tolerance during germination in *Lycopersicon*

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Summary. The salt-tolerant cultivated tomato (*Lycopersicon esculentum*) accession, 'PI174263', and a sensitive cv, 'UCT5', were crossed to develop reciprocal F_1, F_2 and BC_1 populations for genetic analysis of salt tolerance in tomatoes during seed germination. Variation was partitioned into embryo, endosperm and maternal (testa and cytoplasmic) components. Generation means analysis indicated that there were no significant embryo (additive, dominance or epistatic) effects on germination performance under salt stress. Highly significant endosperm additive and testa dominance effects were detected. The proportion of the total variance explained by the model containing these two components was $R^2 = 98.2\%$. Variance component analysis indicated a large genetic variance with additive gene action as the predominant component. Furhter inspection indicated that this variance was attributable to endosperm additive effects on germinability under salt stress. Narrow-sense heritability was estimated as moderately high. Implications for breeding procedures are discussed.

Key words: *Lycopersicon esculentum* – Salt tolerance – Seed germination – Maternal effects – Tomato improvement – Gene action

Introduction

Salinity stress represents a ubiquitous constraint to crop productivity under irrigated agriculture. Within the cultivated tomato, *Lycopersicon esculentum* Mill., most cultivars are relatively salt sensitive in that their growth and economic yields are diminished by mildly saline conditions in the rhizosphere (Maas and Hoffman 1977). In contrast, several wild *Lycopersicon* species have demonstrated high phenotypic variability in salt responsiveness (Rush and Epstein 1976; Jones 1986; Tal and Shannon 1983; Jones et al. 1988). To increase the salt tolerance of the cultivated tomato, Rush and Epstein (1976) and Tal and Shannon (1983) have proposed the development of salt-tolerant cultivars by exploiting the potential gene resources within these wild *Lycopersicon* accessions. Unfortunately, there is insufficient information concerning the genetic control of salt tolerance in *Lycopersicon* or other crop species to warrant such endeavors. The sensitivity of seed germination (Jones 1986), seedling survival and growth (Rush and Epstein 1976; Jones et al. 1988), and vegetative and reproductive growth (Maas and Hoffman 1977; Tal and Shannon 1983; Jones 1987) to salinity stress suggests that salinity responsiveness in *Lycopersicon* is likely to be controlled by a number of genes that may be stage specific (Jones and Qualset 1984; Shannon 1982; Jones 1987). Hence, component analysis, by examining stage-specific variability, may aid in the identification of highly heritable variation in stress characters and facilitate their transfer into improved backgrounds (Jones and Qualset 1984).

Crop ontogeny is most vulnerable to salinity stress at the seed germination and early seedling growth stages (Cook 1979; Jones et al. 1988). Salinity stress greatly delays the onset, reduces the rate and increases the dispersion of germination events in tomatoes (Jones et al. 1988). This sensitivity has important biological and applied significance. The dependence upon mechanization in modern plant cultivation systems requires rapid, uniform and complete germination. In many salt-affected soils the dispersion of salt along the soil horizon is not uniform (Richards 1983). In others, improper irrigation practices, irrigation with water sources carrying some

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burden of salt and evaporation from the soil surface would further concentrate soluble salts in the top of the soil bed and have an adverse impact upon seed performance. In either case, for direct seeded crops like the tomato (El-Sayed and John 1973), many of the seeds may not germinate, or germinate so sporadically that plants grow differentially and crop development is extremely variable and delayed. The development of tomato cultivars with the ability to germinate rapidly and emerge reliably from saline-afflicted soild would contribute significantly to the efficiency of stand establishment. This report assesses the potential of a primitive *L. esculentum* accession ('PI174263') to contribute useful genes for the improvement of salt tolerance in modern tomato cultivars.

Materials and methods

Genetic material and germination conditions

Highly inbred sources of 'PI174263' (P_1) and 'UCT5' (P_2) were crossed to develop reciprocal F_1 progeny. Ten plants of each parent were used to generate F_1 seed. 'UCT5' was selected as a suceptible parent because it exhibits a high degree of salt sensitivity during germination (Jones 1986) and it represents a horticulturally superior, multiple disease-resistant breeding line. The salt-tolerant accession, 'PI174263', is a primitive cultivar from an arid region in Turkey, and exhibits rapid germination under high salinity (Jones 1986). Seed of both inbred parents, their reciprocal F_1, F_2 and backcross generations were produced by controlled pollination under greenhouse conditions.

Seed of each generation was surface sterilized with 0.5% sodium hypochlorite solution for 10 min, rinsed with sterile distilled water several times, and briefly blotted on paper towels. Twenty seeds of each generation were aseptically plated in petri dishes on salinized media containing 0.8% (w/v) agar. Generation responses were compared in two saline treatments, one at 0 m*M* and the other 150 m*M* synthetic sea salt (with a Na^+/Ca^{++} ratio equal to 5; Rila-Mix, N.J.). The water potentials for the control and saline treatments were approximately -1 and -8 bars, respectively. Six replicates for the nonsaline (control) treatment and 7–10 replicates for each generation in the saline treatment were randomized in an incubator maintained at $20° \pm 0.5$ °C in the dark. Germination responses were visually scored as radicle protrusion at 6-h intervals for 10 days in the control (16 days in the saline treatment), and subsequently at 12-h intervals for an additional 19 days in the saline treatment. Seeds germinating within an interval were presumed, on average, to have germinated at the midpoint of the interval. Seeds that failed to germinate in the salt treatment, above the percentage which failed to germinate in the control treatment (presumably were initially nonviable), were included in the analysis as right-censored observations (Gehan 1969). Hence, the sample size for each experimental unit was the number of viable seeds, not the number observed to germinate.

Statistical and genetic analyses

The distributions of germination responses for each generation (individual and pooled replicates) were analyzed by survival analysis with life tables (Scott and Jones 1982). This is a nonparametric procedure that measures time responses as well as the distribution of several descriptive parameters over time (Lee 1980). The time, in days, to 50% germination estimated from life table analysis was used as an estimate of the generation mean. Generation means and variances were determined for samples within each experimental unit (e.g., within plot means and variances) and pooled over replicates. Variances for each generation were pooled among the replications as $s^2 = \Sigma (n_i - 1) s_i^2$ divided by $\Sigma (n_i - 1)$, where n_i is the sample size and s_i^2 is the variance for each replicate.

The presence or absence of maternal effects was tested by analyzing the variance (ANOVA) of the parental and reciprocal F_1 data and comparing the appropriate means with linear contrasts (Dixon 1985). The parental and reciprocal F_1, F_2 and backcross means (a total of ten generations) were analyzed according to the weighted generation means analysis of Mather and Jinks (1971) to estimate parameters for the genetic model containing additive and dominance effects of the embryo, endosperm and maternal components as well as the digenic interaction effects of the embryo. Maternal components were further partitioned into seed coat (testa) and cytoplasmic effects. Coefficients for the genetic parameters tested are shown in Table 1. Each generation mean was weighted by the inverse of the variance of the mean for that generation (Mather and Jinks 1977). Suitability of genetic models was tested by a weighted least squares regression technique and the goodness-of-fit of each model was tested by a weighted χ^2 (Cavalli-Sforza 1952; Hayman 1958; Mather and Jinks 1971, 1977). Significance tests for the genetic parameters were constructed by dividing each estimate obtained from the regression analysis by its standard deviation and comparing the result with the *t*-value for the appropriate number of degrees of freedom and significance.

Variance component analysis and heritability estimations

Variance components, V_E, V_A, and V_D, representing the environmental, additive genetic and dominance genetic variances, respectively, were estimated as follows:

$$V_E = \tfrac{1}{4}(V_{P_1}) + \tfrac{1}{4}(V_{P_2}) + \tfrac{1}{2}(V_{F_1})$$
$$V_A = 2(V_{F_2}) - V_{BC_1} - V_{BC_2}$$
$$V_D = V_{BC_1} + V_{BC_2} - V_{F_2} - V_E$$

where V_{P_1}, V_{P_2}, V_{F_1}, V_{F_2}, V_{BC_1} and V_{BC_2} represent the variances of the parental, F_1, F_2, and the backcrosses to P_1 and P_2, respectively. Standard errors of the variance components were estimated according to Mather and Junks (1977). Broad-sense heritability was estimated as $H^2 = (V_A + V_D)/V_{F2}$, where $V_A + V_D$ represents the genetic variance of the F_2 population (Hanson 1963). Narrow-sense heritability was calculated as $h^2 = V_A/V_{F2}$. Standard errors of the heritability estimates were calculated according to the formula provided by Ginkel and Scharen (1987).

Results

Germination responses of the ten generations in the presence and absence of salt stress are presented in Table 2. Seeds of the salt-tolerant parent ('PI174263') germinated significantly faster than those of the salt-sensitive parent in both treatments; the magnitude of this difference however, was considerably greater in the presence of salt. Analysis of variance indicated that there were highly significant differences among generations in both treatments. A linear contrast comparing reciprocal F_1s showed a significant difference in germinability in the salt treatment, indicating maternal effects. A linear contrast

Table 1. Coefficient of effects in the genetic model for generation means analysis

Generation		Genetic effects[a]									
		m	[d]	[h]	[i]	[j]	[l]	[de]	[he]	[dt]	[ht]	[dc]
P_1	(PI174263)	1	−1	0	1	0	0	−1	0	−1	0	−1
P_2	(T5)	1	1	0	1	0	0	1	0	1	0	1
F_1	$P_1 \times P_2$	1	0	1	0	0	1	−1/3	1	−1	0	−1
	$P_2 \times P_1$	1	0	1	0	0	1	1/3	1	1	0	1
F_2	$(P_1 \times P_2)$ selfed	1	0	1/2	0	0	1/4	0	3/4	0	1	−1
	$(P_2 \times P_1)$ selfed	1	0	1/2	0	0	1/4	0	3/4	0	1	1
BC_1	$(P_1 \times P_2) \times P_1$	1	−1/2	1/2	1/4	−1/4	1/4	−1/3	3/4	0	1	−1
	$(P_2 \times P_1) \times P_1$	1	−1/2	1/2	1/4	−1/4	1/4	−1/3	3/4	0	1	1
BC_2	$(P_1 \times P_2) \times P_2$	1	1/2	1/2	1/4	1/4	1/4	1/3	3/4	0	1	−1
	$(P_2 \times P_1) \times P_2$	1	1/2	1/2	1/4	1/4	1/4	1/3	3/4	0	1	1

[a] m, Mean effects; [d], embryo additive; [h], embryo dominance; [i], additive × additive (embryo); [j], additive × dominance (embryo); [l], dominance × dominance (embryo); [dt], testa additive; [de], endosperm additive; [dc], cytoplasm additive; [ht], testa dominance; [he], endosperm dominance

Table 2. Days to 50% germination under control (0 m*M*) and salt-stress (150 m*M* synthetic sea salt) treatments for the ten generation means

Generation		Median response time (±SE)[a]	
		0 m*M*	150 m*M*
P_1	(PI174263)	3.31±0.19	7.24±0.34
P_2	(T5)	3.99±0.18	13.49±0.81
F_1	$(P_1 \times P_2)$	3.82±0.17	8.89±0.30
	$(P_2 \times P_1)$	6.26±0.28	12.06±0.67
F_2	$(P_1 \times P_2)$ selfed	4.56±0.45	13.94±0.55
	$(P_2 \times P_1)$ selfed	4.93±0.41	13.76±0.73
BC_1	$(P_1 \times P_2) \times P_1$	4.23±0.38	12.56±0.61
	$(P_2 \times P_1) \times P_1$	3.51±0.28	11.64±0.55
BC_2	$(P_1 \times P_2) \times P_2$	4.50±0.40	14.31±0.91
	$(P_2 \times P_1) \times P_2$	4.22±0.30	14.19±0.35

[a] SE = standard error between means of replicates

Table 3. Tomato seed germination responses for the ten generations tested under control (0 m*M*) and salt (150 m*M* synthetic sea salt) treatments[a]

Generation		Germination (%)		Variances	
		Control	Salt	Control	Salt
P_1	(PI174263)	95.8	94.4	4.45	8.63
P_2	(T5)	93.3	91.9	4.12	4.82
F_1	$(P_1 \times P_2)$	89.3	87.5	2.86	4.45
	$(P_2 \times P_1)$	72.5	76.1	5.21	8.76
F_2	$(P_1 \times P_2)$ selfed	97.5	93.8	2.43	28.49
	$(P_2 \times P_1)$ selfed	88.3	89.4	4.46	21.85
BC_1	$(P_1 \times P_2) \times P_1$	90.0	76.7	3.45	18.67
	$(P_2 \times P_1) \times P_1$	97.5	94.5	1.16	6.73
BC_2	$(P_1 \times P_2) \times P_2$	95.0	95.6	1.73	19.18
	$(P_2 \times P_1) \times P_2$	98.3	97.1	1.80	18.25

[a] Germination and variances of salt treatment after correction for right censoring

comparing the pooled reciprocal F_1 mean (10.48) with the parental midpoint (10.37) showed no significant difference, indicating the absence of embryo or endosperm dominance effect. Median response times for all generations under salt stress (150 m*M*) with those at 0 m*M* salt were not correlated ($r=0.44$, $P>0.05$). However, excluding the $P_2 \times P_1$, which showed unexpectedly late germination responses in the control treatment, raised the correlation coefficient to $r=0.78$ ($P<0.01$). The F_2 population ($n=311$) did not segregate into distinct categories, indicating that salt germinability is inherited as a quantitative trait.

An examination of the generation means and the pooled variances (Tables 2 and 3) at high salinity indicated that they were intermediately correlated ($r=0.61$, $P<0.05$). Square-root and logarithmic transformations of the data homogenized the variances and eliminated the correlation between means and variances ($r=0.38$ and 0.21, respectively). The transformations did not, however, alter the results of the generation means analysis (not shown). The weighted least squares regression analysis indicated that estimates of nonallelic interaction effects were consistently nonsignificant suggesting that the genes involved in germination responses under salinity stress were independent of each other in producing their effects. Intra-allelic (dominance) interactions, corresponding to the embryo and endosperm genotypes, were also not significant; however, additive and maternal components had significant effects on the performance of different generations under high salinity treatment.

The relative contribution of individual genetic factors from embryo, endosperm and maternal tissues were ini-

tially evaluated by simple weighted linear regression analysis. The proportion of the total variance among the generation means explained by additive or dominance contributions arising from embryo (i.e., [d] or [h]), endosperm (i.e., [de] or [he]) or maternal tissues (i.e., [dt] or [ht] for the testa and [dc] for the cytoplasm) provided the following ranked order of importance when each factor was individually entered into the model: [de] > [ht] > [dt] > [d] > [dc] > [he] > [h] (Table 4). Clearly, the endosperm additive and testa dominance contributions accounted for a significantly greater proportion of the total variance (73.8% and 70.5%, respectively) than the additive and dominance contributions of the embryo (53.0% and 0.0%, respectively). Paired combinations of these genetic components also were tested to determine the most suitable bifactor genetic model. The model which accounted for the largest proportion of the total variance among generation means contained endosperm additive and testa dominance genetic components (R^2 = 98.2%; Table 4). The joint scaling test (Mather and Jinks 1971) indicated that this model was adequate ($\chi^2_{(7)} = 6.05$, $P > 0.50$; Model 1, Table 5). The weighted least squares estimates of both genetic parameters in the model were highly significant (Model 1, Table 6). The endosperm additive genetic effects accounted for a significantly larger percentage of the total genotype sums of squares in the ANOVA than did the testa dominance genetic effects (Model 1, Table 6). Models containing trifactor combinations of the genetic parameters did not significantly increase the adequacy of the model over that obtained with the most appropriate bifactor (i.e., [de] + [ht]) genetic model (comparisons not shown).

Table 4. Proportion of the total genetic variance accounted for by individual and paired combinations of genetic factors in the model

Monofactor[a]	R^2	Bifactor[a]	R^2
[de]	73.8	[de] + [ht]	98.2
[ht]	70.5	[d] + [ht]	92.0
[dt]	66.8	[dt] + [ht]	89.6
[d]	53.0	[dt] + [he]	78.8
[dc]	43.8	[de] + [dt]	78.7
[he]	32.4	[d] + [dt]	78.7
[h]	0.00	[d] + [de]	78.6

[a] For definition of the factors see the footnote in Table 2

The pooled germination variances for all generations were uniformly small under nonstress conditions and were comparable between segregating and nonsegregating generations (Table 3). In comparison, under salt stress the germination variances were generally larger in magnitude, and were greater in segregating than in nonsegregating generations (Table 3). This might partly be due to differences in the scale of measurements between control and salt treatments. The traditional variance component analysis (i.e., including only additive and dominance variances) gave the following estimates for the different variance components: $V_P = 25.17 \pm 2.05$ ($t_{(309)} = 12.30$, $P < 0.001$), $V_G = 18.50 \pm 2.08$ ($t_{(916)} = 8.87$, $P < 0.001$), $V_A = 19.92 \pm 4.54$ ($t_{(921)} = 4.17$, $P < 0.001$),

Table 5. Joint scaling test to determine the suitability of selected genetic models[a] for the inheritance of days to 50% germination of *L. esculentum* PI174263 and T5 and their reciprocal F_1, F_2 and backcross progeny at 150 m*M* salt

Generation		Replicates number	Weight	Observed mean[b]	Model 1		Model 2	
					Expected mean[b]	O−E	Expected mean[b]	O−E
P_1	(PI174263)	9	8.46	7.24	7.05	0.19	7.15	0.09
P_2	(T5)	9	1.52	13.49	13.45	0.04	13.71	−0.22
F_1	$(P_1 \times P_2)$	8	11.02	8.89	9.19	−0.30	8.96	−0.07
	$(P_2 \times P_1)$	9	2.25	12.06	11.31	0.75	11.90	0.16
F_2	$(P_1 \times P_2)$ selfed	8	3.31	13.94	13.32	0.62	13.35	0.59
	$(P_2 \times P_1)$ selfed	9	1.89	13.76	13.32	0.44	13.35	0.41
BC_1	$(P_1 \times P_2) \times P_1$	9	2.66	12.56	12.26	0.30	12.47	0.09
	$(P_2 \times P_1) \times P_1$	10	3.30	11.64	12.26	−0.62	12.47	−0.83
BC_2	$(P_1 \times P_2) \times P_2$	8	1.21	14.31	14.38	−0.07	14.23	0.08
	$(P_2 \times P_1) \times P_2$	7	8.30	14.19	14.38	−0.19	14.23	−0.04
R^2					0.982		0.988	
χ^2 7; 6					6.050		4.030	
Probability					0.50–0.75		0.50–0.75	

[a] Model 1 = m + [de] + [ht]; Model 2 = m + [de] + [d] + [ht]
[b] Observed means, expected means from the model and the difference between the respective means (O−E)

Table 6. Comparison of the weighted least square estimates of the genetic parameters fitted to two genetic models and their relative contribution to the genotype sums of squares in the analysis of variance

Genetic parameter[a]	Model 1[b]		Model 2[b]	
	Estimate	% Genotype SS	Estimate	% Genotype SS
m	10.25±0.23		10.43±0.23	
[de]	3.20±0.31*	75.2	4.46±0.77*	74.7
[d]	–		-1.17±0.68	4.9
[ht]	3.07±0.32*	24.8	2.91±0.29*	20.4

* Significant at the 1% probability level

[a] m, Mean effect; [de], additive endosperm; [d], additive embryo; [ht], dominance testa

[b] Model 1 = m + [de] + [hp]; Model 2 = m + [de] + [d] + [hp]

$V_D = -0.42 \pm 2.86$ ($t_{(1528)} = -0.15$, $P > 0.5$), $V_E = 6.68 \pm 0.40$ ($t_{(607)} = 16.67$, $P < 0.001$). Thus, most of the total genetic variance is due to additive gene action. Broad- and narrow-sense heritabilities for the mean germination time under salt stress were calculated to be 0.74 ± 0.03 and 0.75 ± 0.03, respectively.

Discussion

Variance component analysis indicated that the superior germination performance exhibited by 'PI174263' in the presence of high salinity is under genetic control with the additive genetic component being the predominant gene action. Partitioning of the sources of genetic variance among generations into those attributed to the effects of embryo, endosperm, and maternal components suggested that most of this additive gene action was attributable to endosperm rather than embryo effects on germination performance under salt stress. The single-factor weighted least squares regression analysis indicated that the contribution of embryo additive ([d]) effects to the total variance among generations (53%, Table 4) was significantly smaller than that of endosperm additive ([de]) effects (73.8%, Table 4). Further inspection indicated that [d] and [de] were highly correlated and, therefore, the observed additive effects of the embryo must be due to the colinearity existing between these two components. The existence of colinearity can be seen from the contribution of a bifactor model (i.e., [d] + [de]) to the total variation (78.6%, Table 4) which is not significantly larger than the contribution of [de] alone (73.8%, Table 4). Furthermore, addition of [d] to the most suitable bifactor model (i.e., [de] + [ht]) in the order of [de] + [d] + [ht] indicated that [d] accounted for only 4.9% of the extra genetic sums of squares (Model 2, Table 6) and that R^2 increased only by 0.6% over that of the bifactor model (Table 5).

Maternal components were detected that significantly affected the germination responses under salinity stress. Reciprocal differences for germination performance under salinity were found in the F_1 generations. The response of the F_1 seed closely resembled that of the maternal inbred (i.e., maternal additive effects). In contrast, differences were not observed between the reciprocal BC or F_2 progenies (Table 2). If cytoplasmic factors were involved in germination performance, the reciprocal F_2s or BCs should have shifted towards the maternal parent. Clearly, then, the difference between reciprocal F_1s was not due to cytoplasmic effects. Results of the single-factor weighted least squares regression analysis indicated that following endosperm additive effects, testa dominance effects had the most significant effects on seed germinability under high salinity stress (Table 4). Our finding of a modest, but significant effect of the testa on germination performance is consistent with previous findings that the testa presented a small mechanical resistance to radicle protrusion (Groot and Karssen 1987). Furthermore, the combination of these two maternal components (i.e., endosperm additive and testa dominance effects) could most adequately explain the genetic variance among generations (Tables 4 and 5). We conclude from this analysis that the endosperm is the principal site of genetic determinants controlling germination performance. Such results provide genetic support to recent hypotheses that metabolic events leading to germination occur in the endosperm (Haigh and Barlow 1987; Groot and Karssen 1987; Groot et al. 1988). Conversely, our results indicate that the embryo, in contrast to earlier suggestions (Liptay and Schopfer 1983), plays a minor role in determining germination behavior. Experimental tests to further evaluate the endosperm's role as the physiologically active component in germination responsiveness are feasible.

Within a formal genetic array, we observed a highly significant correlation between mean time to germination in the presence of salinity stress and that observed in the absence of stress. This high correlation is consistent with the suggestion that the genetic parameters that facilitate rapid germination under stress conditions may also contribute to improved responses under nonstress conditions. This indicates that the genetic controls operating have no undesirable effects on performance in the absence of stress. Furthermore, in contrast to the uniformly small and comparable germination variances observed among the generations under non-stress, variances under salinity stress were generally much larger in magnitude and were also larger in segregating populations in comparison to nonsegregating generations (Table 3). It follows then that the two parents differed considerably in genes impacting upon favorable germination response under stress conditions where the expression of genotypic differences was more pronounced. Greater genetic vari-

ances in stress environments is one of the more favorable situations for plant breeders (Rosielle and Hamblin 1981), although it does not appear to be a common occurrence (Johnson and Frey 1967; Daday et al. 1973).

The results of this study clearly indicate that the major source of variation among generations were endosperm effects that were additively inherited. The involvement of mainly additive genetic components in germination performance under salt stress should facilitate selection efforts for improved salt tolerance in breeding programs. Narrow-sense heritability can be described to be moderately high and provides confidence that reasonably rapid progress can be expected from selection for this trait in early generations. Furthermore, the significant maternal effects suggest that in a breeding program to improve salt tolerance at the germination stage, the salt-tolerant parent and progeny should preferably be used as the maternal parent in crosses.

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