African Journal of Biotechnology Vol. 10(21), pp. 4400-4404, 23 May, 2011
Available online at http://www.academicjournals.org/AJB
DOI: 10.5897/AJB11.426
ISSN 1684–5315 © 2011 Academic Journals

Full Length Research Paper

Genetic adaptability of durum wheat to salinity level at germination stage

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Accepted 29 April, 2011

Evaluation of genetic adaptability of durum wheat to different levels of salt was conducted at the Faculty of Sciences, University of Tunis El Manar, Tunis from April to May 2010 to improve salt tolerance during germination stage. For this purpose, two crosses and their progenies (F1, F2, BC1Ps and BC1Pr) were used based on shoot length at different salinity levels (0, 50, 75, 100, 150 and 200 mmol/L). Significant differences for salt tolerance between means of generations were observed in all the treatments. Separate generation means analysis indicated that inheritance of resistance to salt at germination stage was dependent upon the level of salinity. With low salinity level (50 and 75 mmol/L), only additive and dominance effects were implicated in the genetic control of this trait. For moderate salinity level (100 and 150 mmol/L) in the two crosses, genetic interactions were solicited and the di-genic epistatic model was sufficient to explain variation in generation means. However, for the 200 mmol/L treatment, none of these models explained the variations in generation means and probably, higher order interactions or genes linkage were solicited. The estimated values of narrow-sense heritability were dependent on the cross and the salinity level and ranged between 29 and 90%. The results of this study indicated that selection in specific environments is useful for enhancing resistance to salt, but it may not be effective in providing resistance across a wide range of environments.

Keys words: Durum wheat, genetic-adaptability, salinity level.

INTRODUCTION

Durum wheat is a typical Mediterranean species, 60% of which is produced in Southern Europe, Northern Africa and the Middle East (Royo and Abio, 2003). In Tunisia, durum wheat (*Triticum durum* Desf.) is the most important cereal crop and is used primarily for couscous, macaroni and various types of bread (Bnejdi and El Gazzah, 2008). Soil salinity is one of the major problems in agriculture in arid and semi-arid regions (Epstein et al., 1980). This abiotic stress causes significant losses in crop production throughout the world (Flowers et al., 1997). In the Mediterranean area, the percentage of irrigated soils affected by salinity reached about 20%, depending on the country, and it varied from 7 to 40% (Hamdy et al., 1995). Tunisia is one of the countries that suffer from severe salinity problems. Improving the salt tolerance of crop proves the first way to overcome the limitation of crops production in a salinized area. Salt tolerance during germination stage is important because salinity is usually higher in the soil surface at high groundwater levels. Boubaker (1996) showed that germination and seedling characteristics are also viable criteria for selecting salt tolerance in durum wheat in a screening experiment with eight durum wheat cultivars.

The genetic mechanism controlling quantitative traits is very complicated and heredity can be changed with the variation of environment (Perez de la Vega, 1996). Therefore, individual gene effect or different gene interactions were solicited in an environment and were not solicited in others. Epistasis is one of the mechanisms adapted by the plant in such non-favourable environment (Allard. 1996). Recently, Bnejdi et al. (2010) found that the presence and importance of genetic interactions in

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resistance of pepper to *Phytophthora nicotianae* are dependent upon the aggressiveness of the isolates. The variation of the heredity of quantitative traits with environment has new axes that need more studies from geneticists. Allard reported this in 1960, but for the simplicity of studies and the choice of selection scheme, breeders considered only the additive model. Information regarding genetic adaptation to different salt concentrations in durum wheat species is not well known. This study was undertaken to determine the mode of inheritance for salt tolerance under different salinity levels at the germination stage of durum wheat.

MATERIALS AND METHODS

The study of genetic adaptability to salinity level at the germination stage of durum wheat was carried out at the Faculty of Sciences, University of Tunis El Manar, Tunisia from April to May 2010. For this purpose, two crosses and their progenies (F1, F2, BC1Ps and BC1Pr) were used to study the genetic basis of tolerance to different salinity level based on shoot length. Durum parental lines were selected based on preliminary observation of their salt tolerance; the resistant parent (Pr) was Chili and the susceptible parents (Ps) were the two first cultivated varieties in Tunisia, Karim 80 and Razzak. Crosses were made as follows: Chili × Karim 80 and Chili × Razzak. The two crosses were controlled under pollinations in a greenhouse. Seeds of each population were germinated in two layers of filter paper moistened in water with different salinity level of NaCl (control (0 mmol/L) 50, 75, 100, 150 and 200 mmol/L) in 5 cm dish at 25°C. Each day, 5 ml of water was added to replace the loss of water by evaporation. The experiment was laid out following complete randomized design with three replications for the treatment as well as the control. Shoot length was measured after 15 days. Indices of resistance of salt were measured as: Relatives salt tolerance = shoot length/mean of shoot length of control.

Statistical analysis

Analysis of variance using GLM procedures (SAS, 1990) were used for analysis of the replication and generation x replication effects.

Genetic model

Weighted least squares regression analyses were used to solve for mid-parent [m], pooled additive [a], pooled dominance [d] and pooled di-genic epistatic ([aa], [dd] and [ad]) genetic effects. Following the models and assumptions described in Mather and Jinks (1982), a simple additive dominance genetic model containing only the m, a and d effects was tested first. Using the joint scaling test described in Rowe and Alexander (1980), adequacy of the genetic model was assessed using a chi-square goodness-of-fit statistic derived from di-genic epistatic effects which were then tested until the chi-square; statistic was non-significant.

Variance components and heritability estimates

Homogeneity of variances of non-segregating generations was tested by using Bartlett's test (Bartlett, 1937), and when the variances were heterogeneous, the environmental variance (VE) was replaced by an adequate number of separate parameters in the model fitting and pooled to produce a single environmental variance. Additive, dominance and environmental variance components were estimated using the maximum likelihood method with the observed variances of the six basic generations used as the initial weights ($df/2 \times S^2$) until the χ^2 test values reached a minimum (Lynch and Walsh, 1998). Narrow-sense heritability (h^2n) was calculated as follows: $h^2n = VA/VA + VD + VE$. Where, VA is the additive genetic component of variance, VD is the dominance or non-additive genetic component of variance, and VE is the environmental variance (Kearsey and Pooni, 1996). The dominance variance (VD) was negative and was set at zero.

RESULTS

The generation means of different crosses for salt tolerance of the five salt treatments are presented in Table 1. F1 means were higher than the mid-parent value and the F2 means for all majorities of treatments, and tended towards tolerant parent (Ps). Means for back-crosses (BC1Pr and BC1Ps) tended towards their respective parents in the two crosses.

The results of separate generation means analysis are presented in Table 2. For treatments 50 and 75 mmol/L, the additive-dominance model (m + a + d) was showed appropriate in the two crosses. The additive effect was significant and negative in the two crosses and for all the treatments. The dominance effect was not significant in the three cases, with additive effect been more important than the dominance effect in all the majorities of combination treatment-cross. For treatments 100 and 150 mmol/L, the additive dominance effect failed to explain variation in generation means in the two crosses. Therefore, a di-genic epistatic models was applied and found to be adequate (*m + a + d + aa +dd; m + a + d + aa +ad*). For treatment 200 mmol/L, both the additive-dominance and di-genic epistatic models failed to explain variation in generation means in the two crosses. The estimates of the different variance components and narrow-sense heritability (h^2n) are shown in Table 3. The additive and dominance variance components estimates were inconsistent between crosses and across the test treatments. The values of narrow-sense heritability (h^2n) varied depending on treatment and ranged from 60 to 82% in the cross Chili × Karim and 29 to 90% in the cross Chili × Razzek.

DISCUSSION

In the two crosses, the means of the parents (Pr and Ps) showed a tendency to be extreme and contrasted than the means of the F1 and F2 generations. As expected, the backcrosses, BC1Pr and BC1Ps, showed means that had a tendency to be located close to those of their respective recurrent parents. These results confirmed the choice of parents for this study and validated the genetic analysis of the trait according to the method of Mather and Jinks (1982).

Table 1. Shoot length mean ± SE (×100) for salt tolerance in parents and offspring of two crosses of resistant par susceptible parent.

Treatment	200 mmol/L	150 mmol/L	100 mmol/L	75 mmol/L	50 mmol/L
Chili × Karim 80					
Pr (20)	7.62 ±3.10^{ab}	24.10 ±6.9^{a}	34.48 ±9.5^{a}	59.71 ±9.8^{a}	86.66 ±14.6^{a}
BC1Pr(100)	7.74 ±4.14^{ab}	21.20 ±6.1^{b}	30.44 ±8.1^{ab}	54.93 ±6.4^{b}	80.55 ±11.9^{b}
F1(50)	8.63 ±2.87^{a}	15.64 ±3.8^{e}	33.01 ±10^{ab}	50.99 ±6.1^{c}	76.02 ±12b^{c}
F2(200)	7.66 ±5.45^{ab}	20.22 ±7.8b^{c}	29.74 ±13^{b}	50.47 ±12^{c}	71.75 ±16^{c}
BC1Ps(100)	6.03 ±2.65^{c}	17.26 ±5.3d^{e}	22.85 ±12^{c}	43.47 ±8.7^{d}	64.83 ±11^{d}
Ps(20)	6.73 ±3.21b^{c}	18.67 ±7.4^{cd}	25.34 ±12^{c}	40.53 ±11^{d}	57.55 ±13^{e}
Chili × Razzek					
Pr(20)	7.62 ±3.1^{a}	24.10 ±6.9^{a}	34.48 ±9.5^{a}	59.71 ±9.8^{a}	86.66 ±14^{a}
BC1Pr(100)	7.16 ±3.6^{b}	18.08 ±5.3^{b}	29.06 ±9.0^{b}	50.95 ±12^{b}	72.84 ±19^{b}
F1(50)	8.29 ±3.0^{bc}	15.93 ±4.1^{c}	27.53 ±6.5^{bc}	42.18 ±6.7^{c}	59.01 ±11^{c}
F2(200)	6.18 ±3.8^{c}	15.38 ±6.6^{cd}	24.59 ±11^{cd}	39.88 ±19^{c}	55.16 ±30^{c}
BC1Ps(100)	4.68 ±3.4^{d}	13.77 ±4.7^{d}	24.12 ±7.5^{d}	33.42 ±11^{d}	45.19 ±18^{d}
Ps(20)	5.27 ±3.1^{e}	11.61 ±4.3^{e}	17.95 ±6.9^{e}	24.66 ±6.5^{e}	31.37 ±13^{e}

Means followed by different letter within each column for each population and treatment are significantly different based on Duncan's test ($P < 0.05$).

Table 2. Estimates of gene effects ± SE (×100) for salt tolerance in two crosses (Karim 80 × Chili, Razzek × Chili).

Model	200 mmol/L	150 mmol/L	100 mmol/L	75 mmol/L	50 mmol/L
Karim 80 (s) × Chili (r)					
Three-parameter model					
m	6.71 ±0.27**	22.22 ±0.55**	27.90 ±0.96**	49.28 ±0.82**	71.12 ±1.20**
a	-0.99 ±0.26**	-3.22 ±0.54**	-5.36 ±0.89**	-10.40 ±0.76**	-15.01 ±1.09**
d	1.18 ±0.50**	-6.08 ±0.88**	2.06 ±1.76*	1.22 ±1.36	3.44 ±2.21*
p	< 0.001	< 0.005	< 0.005	0.28	0.49
Best-fit model					
m	34.38 ±1.86**	24.69 ±1.16**	40.37 ±4.92**		
a	-0.45 ±0.32*	-2.72 ±0.72**	-5.66 ±0.90**		
d	-8.79 ±4.39**	-9.08 ±1.53**	-35.18 ±11.97**		
aa	-3.09 ±1.83**	-3.37 ±1.42**	-10.78 ±4.83**		
dd	7.16 ±2.70**	…..	27.82 ±7.68**		
ad	-2.52 ±1.17**	-2.47 ±2.17*	…..		
p	…..	0.80	0.10		
Razzek (s) × Chili (r)					
Three-parameter model					
m	5.77 ±0.31**	16.91 ±0.47**	24.87 ±0.71**	41.94 ±0.74**	58.59 ±1.24**
a	-1.52 ±0.30**	-5.31 ±0.45**	-6.43 ±0.69**	-17.44 ±0.74**	-27.60 ±1.23**
d	1.34 ±0.57**	-1.67 ±0.81**	2.45 ±1.24*	-0.07 ±1.25	-0.15 ±2.11
p	< 0.001	< 0.005	< 0.005	0.46	0.41
Best-fit model					
m	-33.67±1.52**	14.72 ±1.07**	22.40 ±1.76**		
a	-1.22 ±0.39**	-6.21 ±0.58**	-7.51 ±0.85**		
d	-6.14 ±3.88**	1.12 ±0.50*	5.51 ±2.43**		
aa	-1.03 ±0.47**	3.06 ±1.25**	3.28 ±2.01**		
dd	6.91 ±2.54**	…..	…..		
ad	-2.52 ±1.26*	3.73 ±1.83**	5.60 ±2.87**		
p	…..	0.65	0.16		

Mean (m), additive (a), dominance (d), additive × additive (aa), dominance × dominance (dd), and additive × dominance (ad) genetic effects. *, **, indicates that means and gene effects are statistically different from zero at $P < 0.05$, $P < 0.01$, respectively. (p) = Probability of adequateness of model.

Table 3. Estimates of variance components with their SE (×100) and narrow-sense heritability (h^2n) for salt tolerance in two crosses of resistant (r) by susceptible (s) parents.

Variance component	200 mmol/L	150 mmol/L	100 mmol/L	75 mmol/L	50 mmol/L
Karim 80 (s) × Chili (r)					
VE	0.09 ± 0.01*	0.20 ± 0.02*	1.05 ± 0.12*	0.52 ± 0.03*	1.76 ± 0.20*
VA	0.42 ± 0.06*	0.60 ± 0.13*	2.20 ± 0.41*	2.32 ± 0.34*	2.72 ± 0.62*
VD	-0.21 ± 0.03*	-0.18 ± 0.09*	-1.34 ± 0.27*	-1.18 ± 0.20*	-1.71 ± 0.44*
X^2 (df)	(3) ns	(3) ns	(3) ns	(3) ns	(3) ns
Heritability (h^2n) (%)	82	75	67	81	60
Razzek (s) × Chili (r)					
VE	0.10 ± 0.01*	0.18 ± 0.01*	0.50 ± 0.05*	0.46 ± 0.03*	1.56 ± 0.20*
VA	0.04 ± 0.03*	0.37 ± 0.13*	1.24 ± 0.28*	4.71 ± 1.07*	11.26 ± 1.92*
VD	-0.006 ± 0.003*	-0.12 ± 0.08*	-0.47 ± 0.19*	-1.50 ± 0.58*	-3.69 ± 1.16*
X^2 (df)	(3) ns	(3) ns	(3) ns	(3) ns	(3) ns
Heritability (h^2n) (%)	29	67	71	90	87

VE, Environmental; VA, additive; VD, dominance variance components; df = degrees of freedom, calculated as the number of generations minus the number of estimated variance parameters; ns = non-significant.* indicates variance components were different from zero at $P < 0.05$.

The results of generation means analysis revealed that the mechanism of genetic control of salt tolerance at germination stage was dependant upon the concentration of salt. With lower salt concentrations (50 and 75 mmol/L) at germination stage, the variation in the generation means fitted an additive-dominance model in the two crosses. This situation is more favourable than the presence of di-genic or higher order interaction due to a greater chance of having a successful breeding. The validity of the additive-dominance model was reported by Zafar et al. (2008) in bread wheat and Ray and Islam (2008) in rice. With moderate levels of salt at germination (100 and 150 mmol/L), genetic interactions were solicited and the di-genic epistatic model was fitted. This situation was more complicated than the presence of additive and dominance effects from breeder's point of view. The presence of additive and non-additive effects is similar to those reported by Dehdari et al. (2007) and Dashti et al. (2010) at germination stage.

Therefore, with higher level of salt at germination stage, none of these models explained the variation in generation means and probably higher order interactions or gene linkage were implicated. To identify whether or not a cause of the model failure is as a result of the presence of higher order interactions or gene linkage, further analyses need to be carried out with enough generations to fit a full trigenic interaction and linkage model. Epistasis is one of the genetic mechanisms solicited in the presence of elevated concentration of salt at germination stage.

These results show that the mode of inheritance of salt tolerance at germination was dependent upon the treatment, stressing the importance of the appropriate selection. With low levels of salt concentrations, the mechanism of resistance was relatively simple. Only additive and dominance effects were implicated in the control of resistance to salt. Selection in environment with low level of salinity was very simple because only additive and dominance effects were implicated in the genetic control of this trait. Therefore, selection with higher levels of salt was complicated by the presence of epistasis effects, but was suggested for the stability of salt tolerance in durum. Inheritance of quantitative traits was very complicated and was dependent upon the environment in which the trait was evaluated. One of the mechanisms that can be solicited by plant in such an unfavourable environment is epistasis. The presence or absence of epistasis may depend on the environment in which the plant material was evaluated and thus may not always be related to the inherent capacity of a genotype (Sunil and Singh, 2003).

In this study, intermediate to high values of narrow-sense heritability revealed important participation of genetic effect on the expression of this trait and selection should be efficient. Differential salt modifications of the effects of genes on salt tolerance at germination, or of the effects of different gene systems, may be an advantage in the process of adaptation to different salt concentrations. In breeding, however, such modifications cause many unpredictable reactions. Breeding for stability of any important characters would be expected to reduce the interactions. Therefore, selection in specific environment was suggested for the stability of salt tolerance.

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