Cytoplasmic Effects on Grain Resistance to Yellowberry in Durum Wheat

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Abstract: Parental, F_1, reciprocal F_1 (RF_1), F_2, reciprocal F_2 (RF_2), BC_1P_1 and BC_1P_2 generations of four crosses involving four cultivars of durum wheat (*Triticum durum* Desf.) were evaluated for grain resistance to yellowberry. Significant differences were reported for F_1, F_2 and their reciprocals in all crosses. A generation means analysis indicated the inadequacy of additive-dominance model and additive-dominance model considering maternal effects. However, the variation in generation means in the four crosses could be explained by a digenic epistatic model with cytoplasmic effects. Cytoplasmic effects were significant and consistent in all the crosses. Dominance effects and additive × dominance epistasis were more important than additive effects and other epistatic components. The choice of a female parent possessing grain resistance to yellowberry appeared to be decisive in durum wheat breeding for resistance to this serious seed disorder.

Keywords: cytoplasmic effects; resistance to yellowberry; *Triticum durum*

Yellowberry is a serious seed disorder in durum wheat and triticale (Ammiraju *et al*. 2002). In durum wheat, yellowberry manifests itself as the presence of farinaceous (blotchy) areas in a usually vitreous grain (Valdeyron & Seguela 1958). Finding sources of resistance and utilizing them to improve yellowberry resistance are high priorities of wheat breeding programmes in many regions of the world. Variation in individual phenotype may be determined not only by the genotype and environment but also by maternal effect. There are essentially three routes by which the mother can influence her progeny. The first is through cytoplasmically inherited factors, such as mitochondria or chloroplasts. The second involves the effects of the mother's own genes on the progeny. Third, the mother's environment may affect the phenotype of the progeny (Kearsey & Pooni 1996). Maternal effect and the cytoplasmic inheritance of quantitative traits have been widely studied in cereal crops. Millet *et al*. (1984) reported upon maternal effects on grain protein content in wheat and in rice. Shi and Zhu (1998) detected effects of cytoplasm on milling quality traits. The cytoplasm evidently contributed to virulence of *Mycosphaerella graminicola* (Fuckel) Shroeter in wheat (Mazouz *et al*. 2002) and also to wheat resistance to stripe rust (Chen & Line 1993). This paper brings results of experiments designed to determine the effects of cytoplasm on grain resistance to yellowberry in durum wheat.

MATERIALS AND METHODS

Four durum wheat genotypes of Tunisian origin were selected on the basis of their differential reaction to yellowberry. The yellowberry resist-

ant parent (P_r) was the cultivar OmRabï, the intermediate resistant parent (P_i) was Ben Bachir and the susceptible parents (P_s) were Cocorit 71 and Karim. Four crosses were made as follows: OmRabï (P_r) × Cocorit 71 (P_s), Ben Bachir (P_i) × Karim (P_s), Ben Bachir (P_i) × Cocorit 71 (P_s) and OmRabï (P_r) × Karim (P_s). F_1 and RF_1 of direct and reciprocal crosses were self-pollinated to produce F_2 and RF_2, respectively. F_1's of direct crosses were backcrossed to both parents using the F_1 plants as females. Backcrosses of F_1 to resistant and susceptible parents were noted BC_1P_1 and BC_1P_2, respectively.

This study was carried out at the El Kef site located in Tunisia under rainfed conditions without additional application of fertilisers in the 2006/2007 growing season. This area is characterized by loamy soil and sub-humid climate with rainfall of about 700 mm. Experiments with 28 populations including parental lines (P_r or $_i$ and P_s), F_1, RF_1 (reciprocal F_1), F_2, RF_2 (reciprocal F_2) and backcrosses (BC_1P_1 and BC_1P_2) were grown in a randomised complete block design with two replications. The number of evaluated plants, equal for each replication, was significantly higher in segregating populations (for the number of evaluated plants see Table 1).

The yellowberry percentage was evaluated by visual observation of each individual plant as follows: two random kernels were phenotyped using 0–5 scale: 0 – grain unaffected, 1 – farinaceous part < 25%, 2 – 25%< farinaceous part < 50%, 3 – 50% < farinaceous part < 75%, 4 – 75% < farinaceous part < 100% and 5 – grain 100% affected. Transforming the data by log, square root, arc-sine and arc-sine of squared root had no effect on data distribution or on removing epistatic effects. Analysis of variance using GLM procedures (SAS Institute 1990) indicated the absence of blocking effects on generation means (data not presented here). Therefore, a generation means analysis was conducted without adjusting data for replication.

The means of different generations were analyzed by a joint scaling test as described by Rowe and Alexander (1980) using the weighted least-squares method (Mather & Jinks 1982; Kearsey & Pooni 1996; Lynch & Walsh 1998). The observed generation means were used to estimate the parameters of a model comprising only mean, additive and dominance genetic effects. The estimated parameters were used in turn to calculate the expected generation means, a significant chi-squared value indicating a significant difference between the observed and expected generation means, which implied that a simple additive model was insufficient to explain the genetic variance. When the addition of maternal parameter to the three-parameter model was not satisfactory, then the six-parameter model with maternal effect was applied. The significance of each parameter was determined by *t*-test.

Table 1. Estimates of grain resistance to yellowberry (mean symptom scores ± standard errors) in the parents and six hybrids coming from four durum wheat crosses

Generation	OmRabï × Cocorit 71	Ben Bachir × Karim	Ben Bachir × Cocorit 71	OmRabï × Karim
$P_{r(i)}(20)^Y$	0.25 ± 0.44^a	0.75 ± 0.83^a	0.75 ± 0.83^a	0.25 ± 0.44^a
BC_1P_1: $(P_{r(i)} \times P_s) \times P_r{}^Z$ (70)	1.53 ± 1.41^c	1.96 ± 1.64^b	1.55 ± 0.84^{bc}	1.49 ± 1.31^c
F_1: $(P_{r(i)} \times P_s)^Z$ (50)	1.05 ± 1.06^b	0.93 ± 0.79^a	1.08 ± 1.03^{ab}	0.88 ± 0.80^b
RF_1: $(P_s \times P_{r(i)})$ (50)	3.3 ± 1.82^d	2.25 ± 1.76^b	2.56 ± 1.60^d	2.36 ± 1.46^d
F_2: $((P_{r(i)} \times P_s) \times (P_{r(i)} \times P_s))$ (100)	1.25 ± 1.27^b	3.15 ± 1.83^c	1.66 ± 1.28^c	2.46 ± 1.42^d
RF_2: $((P_s \times P_{r(i)}) \times (P_s \times P_{r(i)}))$ (100)	2.95 ± 1.69^d	4.09 ± 1.45^d	3.28 ± 1.81^e	3.17 ± 1.72^e
BC_1P_2: $(P_{r(i)} \times P_s) \times P_s$ (70)	1.25 ± 1.27^c	2.36 ± 1.40^b	2.68 ± 1.67^d	2.28 ± 1.27^d
PS (20)	3.90 ± 1.25^e	4.09 ± 1.45^d	3.90 ± 1.25^f	4.09 ± 1.45^f

Means followed by different letters within each column are significantly different based on Duncan's test ($P < 0.05$)
^{Z}female listed first in each cross; ^{Y}in parentheses is the number of plants evaluated in each generation
Indices $_{r(i)}$ and $_s$ are used to mark resistant (intermediate resistant) and susceptible parents, respectively

RESULTS AND DISCUSSION

Mean symptom scores and their corresponding standard errors indicating the level of resistance to yellowberry in the different generations of four crosses are shown in Table 1. Significant differences between generation means were detected in all four crosses, showing genetic diversity in resistance to yellowberry in the materials studied. In all cases, the means of the parents in each cross tended to be more extreme. Differences between mean disease scores from direct and reciprocal crosses were significant in all F_1 and F_2 populations, which may indicate the presence of maternal effect.

To determine the direction of the maternal effect, a generation means analysis was applied. Estimates of different types of gene effect in individual crosses (Table 2) clearly illustrate the variation between populations. The joint scaling test with three-parameter model and three-parameter model with maternal parameter showed a significant chi-square (χ^2) value or $P < 0.01$, indicating that the additive-dominance model and additive-dominance model with maternal effect were not satisfactory for the explanation of variation in resistance to yellowberry. A digenic parameter model with cytoplasmic effect was applied and it was found adequate ($P > 0.01$) in all four crosses (Table 2). The highest probability of goodness of fit of the model (69%) was detected

Table 2. Estimates of gene effects ± SE (standard error) for grain resistance to yellowberry in four crosses of durum wheat

Model	OmRabï ×Cocorit 71	Ben Bachir × Karim	Ben Bachir × Cocorit 71	OmRabï × Karim
Three-parameter model				
Mean	2.53 ± 0.05**	4.93 ± 0.09**	3.19 ± 0.77**	3.05 ± 0.06**
Additive	–1.96 ± 0.04**	–2.20 ± 0.07**	–1.60 ± 0.10**	–2.24 ± 0.07**
Dominance	–1.35 ± 0.13**	–3.88 ± 0.13**	–1.82 ± 0.17**	–1.77 ± 0.14**
^{A}P	$P < 0.01$	$P < 0.01$	$P < 0.01$	$P < 0.01$
Three-parameter model with maternal effect				
Mean	1.77 ± 0.07**	2.28 ± 0.09**	2.30 ± 0.13**	2.11 ± 0.11**
Additive	–2.6 ± 0.19**	–1.85 ± 0.19**	–1.17 ± 0.27**	–2.50 ± 0.24**
Dominance	0.39 ± 0.15*	–0.47 ± 0.16*	–0.27 ± 0.21^{ns}	–0.42 ± 0.17*
Maternal additive	1.31 ± 0.11**	0.72 ± 0.11**	0.44 ± 0.11**	0.77 ± 0.11**
Maternal dominance	–0.03 ± 0.12^{ns}	1.64 ± 0.08**	0.24 ± 0.12*	0.86 ± 0.12**
Cytoplasmic genetic effects	–0.18 ± 0.07*	–0.29 ± 0.05**	–0.57 ± 0.08**	–0.09 ± 0.08^{ns}
^{A}P	$P < 0.01$	$P < 0.01$	$P < 0.01$	$P < 0.01$
Best fit-model				
Mean	3.54 ± 0.56**	8.35 ± 0.43**	3.73 ± 0.58**	6.14 ± 0.59**
Additive	–0.86 ± 0.12**	–1.28 ± 0.13**	–0.78 ± 0.18**	–1.51 ± 0.16**
Dominance	–4.14 ± 1.42*	–12.08 ± 1.17**	–3.18 ± 1.49*	–8.54 ± 1.48**
Additive × additive	–1.46 ± 0.55*	–5.79 ± 0.41**	–1.40 ± 0.55*	–3.84 ± 0.57**
Dominance × dominance	2.67 ± 0.91*	5.21 ± 0.77**	1.29 ± 0.66*	3.91 ± 0.93**
Additive × dominance	6.50 ± 0.44**	3.78 ± 0.42**	2.46 ± 0.53**	3.59 ± 0.49**
Cytoplasmic genetic effects	–0.94 ± 0.08**	–0.50 ± 0.05**	–0.78 ± 0.08**	–0.53 ± 0.08**
^{A}P	0.12	0.20	0.69	0.02

*, **indicates means and gene effects statistically different from zero at $P < 0.05$ and $P < 0.01$, respectively
^{ns}not significant; ^{A}P – probability of adequateness of the model

in the cross Ben Bachir (P_i) × Cocorit 71 (P_s) and relatively lower values (2% and 12%) in the crosses between resistant OmRabï and susceptible parents Karim and Cocorit 71.

All types of gene effects (additive, dominance, additive × additive, dominance × dominance, dominance × additive and cytoplasmic) were significant in the examined crosses. Dominance type effect accounted for a particularly large portion of genetic variance. Cytoplasmic effect was negative and consistent in all crosses.

Additive, dominance and cytoplasmic effects were negative, which may indicate a higher contribution to resistance than to susceptibility. The negative estimates of additive × additive variance show that the gene pairs responsible for resistance to yellowberry are in dispersive form (Mather & Jinks 1977). This means that both parents contributed the genes for resistance to yellowberry. The presence of epistatic effects has been reported for grain resistance to yellowberry by Bnejdi and El Gazzah (2008). A new important finding obtained in this study is the significance of cytoplasmic gene effects, which could be exploited in breeding wheat for resistance to yellowberry. The obtained results indicate that in durum wheat breeding programmes the choice of the female parent resistant to yellowberry could significantly contribute to an increase in resistance level as an additional source of resistance. However, a better understanding of the relationship between nuclear and organellar genomes is undoubtedly needed for efficient use of cytoplasmic resistance.

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