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*Full Length Research Paper*

# Genotype × environment interactions and heritability of quantitative resistance to net blotch in Tunisian barley

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**A doubled-haploid barley population derived from a cross between the Tunisian cultivar 'Roho' and the local line '90' was used to assess the genotype x environment interaction, heritability estimates and correlations between disease parameters of net blotch resistance at adult growth stage in three environments. Net blotch reaction was evaluated using the mass disease index, the area under the disease progress curve and the apparent infection rate. The DH lines showed different levels of quantitative resistance to *Pyrenophora teres* under low, moderate and high epidemic conditions. Large variation of the mass disease index, the area under the disease progress curve and the apparent infection rate was obtained under high-pressure conditions that facilitate identifying tolerant lines. Significant genotypic differences were noted, however preponderant genotype x environment interactions were the major sources of variation. Broad sense heritability values were large for all parameters using data from single environment and low for the mass disease index, the area under disease progress curve and the apparent infection rate using estimates from different growing conditions. In a single environment, resistance related parameters were found to be genetically associated. Excepting mass disease index, lack of associations between area under the disease progress curve and apparent infection rate measured on the three field conditions were obtained. The results obtained suggest that loci for mass disease index may be pleiotropic or linked and loci for areas under the disease progress curve and apparent infection rate may be independent. Multi-location screening for quantitative resistance to *P. teres* should be considered in a breeding program.**

**Key words:** Heritability, genotype x environment interactions, genetic correlation, disease parameter, *Hordeum vulgare, Pyrenophora teres.*

## INTRODUCTION

Net blotch of barley (*Hordeum vulgare* L.) caused by *Pyrenophora teres* (Died), is a major disease in most barley growing areas around the world. In Tunisia, Cherif et al. (1994) reported that net blotch is the most common disease of barley that was associated with high severity levels (70 - 80%) in some regions. Both net and spot forms are prevailing in Tunisia. Management practices used, the intensification of barley cultivation and the absence of resistant varieties are among the factors favouring net blotch incidence. Thus, selecting for quantitative resistance to *P. teres* could be efficient. Quantitative or partial resistance as a reduction in the percentage of leaf tissue affected was reported in this pathosystem (Douglas and Gordon, 1985; Robinson and Jalli, 1997; Steffenson and Webster, 1992; Steffenson et al., 1996). It was termed adult or field resistance since it reduces the rate of disease development in the field. Screening for quantitative resistance in the field is often

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**Abbreviations: AUDPC,** Area under the disease progress curve; **$AUDPC_{MDI}$,** AUDPC calculated for MDI in 2003-2004; **$AUDPC_S$,** AUDPC calculated for S; **DH,** doubled-haploid, **ESA-Mograne**, ecole supérieur agricole de mograne, **INAT,** institut national agronomique de Tunisie; **MDI,** mass disease index; $MDI_1$, MDI calculated in 2002 – 2003; **$MDI_2$**, final MDI calculated in 2003-2004; **r,** apparent infection rate; **$r_{MDI}$,** apparent infection rate calculated for MDI in 2003 – 2004; **$r_S$,** apparent infection rate calculated for S; **S,** final disease severity.

difficult. For net blotch resistance, Cakir et al. (2003) and Spaner et al. (1998) used the scale of Tekauz (1985). Others as El Yousfi and Ezzahiri (2001), Steffenson and Webster (1992) and Steffenson et al. (1996) used the percent disease severity developed by Burleigh and Loubane (1984). Area under the disease progress curve (AUDPC) describes disease development in the field and can differentiate, at a critical point, between two epidemics with equal final disease severities. This parameter was used by Burleigh and Loubane (1984), El Yousfi and Ezzahiri (2001), Robinson and Jalli (1997) and Steffenson and Webster (1992) to estimate resistance to net blotch. Apparent infection rate (r) was first proposed by Vanderplank (1963) and was later applied by many authors for several pathosystems (Burleigh and Loubane, 1984; El Yousfi and Ezzahiri, 2001; Robinson and Jalli, 1997; Steffenson and Webster, 1992). Information given by the AUDPC and the r is different when disease increases rapidly within a short period. The environment may alter the expression of quantitative resistance (El Yousfi and Ezzahiri, 2001). Little attention has, however, been directed toward the analysis of the genotype x environment interactions and the heritability estimates of these parameters. Doubled-haploid (DH) lines are suited for this analysis since these lines present an unlimited number of individuals facilitating the estimation of the genotype x environment interactions by multiplying replicates (Choo et al., 1985) and thus estimating heritability (Marwede et al., 2004; Spaner et al., 1998). The objectives of this study were:

(1) To investigate quantitative expression of resistance to *P. teres* in different environments using a DH population of barley.
(2) To evaluate genotype x environment components.
(3) To estimate heritability.
(4) To determine potential associations between disease related parameters.

## MATERIALS AND METHODS

### Plant material

A total of fifty-nine DH barley lines were obtained at Florimond Desprez, using anther culture and the *Hordeum bulbosum* method (Devaux and Pickering, 2005). These DH lines were developed from F1 plants of the cross between the Tunisian cultivar 'Roho' and the local 'line 90' which was carried out at INAT (Institut National Agronomique de Tunisie). 'Roho' is a widely grown two-row barley cultivar susceptible to net blotch. 'Line 90' is a six-row fixed line selected from the Tunisian national breeding program for its resistance to net blotch in the field. This line was selected from a cross between Local Cap-Bon and Jérusalem à barbes lisses/CI 10836 ICB 77-319-1AP-0SH-2AP-1AP-0AP.

### Field experiments

The experiments were conducted during two consecutive growing seasons 2002 - 2003 and 2003 - 2004 at the experimental station of ESA-Mograne (Ecole Supérieur Agricole de Mograne) associate which is, with, considered a particularly hot spot area for net blotch disease and during 2004 at the experimental station of INAT (Tunis) corresponding to a moderate site for net blotch infection.

### Mograne trials 2002-2003 and 2003-2004

Parents and DH lines were sown in single rows on 5 December, 2002 and in two rows on 17 November, 2003 using a randomised block design with three replications. Row-lines were 1 m long and spaced 0.5 m apart. The Tunisian commercial cultivar 'Martin' was planted as a susceptible check every 14 entries. In 2002 - 2003, plants were inoculated at the mid-tillering stage of growth (GS 22 - 26) and again at the early stem elongation (GS 30 - 33) stage (Zadoks et al., 1974) using infected barley seeds with a mixture of local isolates including both net and spot forms of *P. teres* according to Onfroy (1997). In 2003 - 2004, inoculations were made twice: at early tillering stage of growth (GS 20 - 23) with barley straw collected from infected volunteer barley plants in neighbouring fields and at the end of tillering stage (GS 28 - 29) with infected barley seeds prepared as in 2002 - 2003. Disease incidence (percentage of plants having at least one lesion) and severity (average percent of leaf area affected by the disease) were assessed on 10 randomly selected plants per line according to Yahyaoui et al. (2003). Both disease incidence and severity were estimated only once in 2002 - 2003 at the mid-dough growth stage (GS 85 - 87) and four times in 2003 – 2004 starting at stem elongation growth stage (GS 35 - 37) to the dough development stage (GS 85 - 87).

### Tunis trial 2004

Five plants of each parent and DH lines were sown on 15 January, 2004 in plastic pots (0.25 m diameter) filled with loamy-clay soil and grown at INAT station. A completely randomised design with three replications was adopted. Plants were inoculated three times every 15 days from mid-tillering growth stage (GS 22 - 26) by spraying a mono-conidial suspension of 'Bir Mcharga' isolate of *P. teres* f. *teres* adjusted to $10^4$ conidia/ml for which parents present differential reaction (unpublished data). Pathogen culture and inoculum preparation were done as described by Steffenson et al. (1996). Inoculated plants were then covered with plastic sheets to insure a high level of humidity. Disease reactions were recorded three times after symptoms apparition from ear emergence stage (GS 53 - 58) to milk development stage (GS 72 - 76) using the percent net blotch severity (including both chlorotic and necrotic areas) according to the scale devised by Burleigh and Loubane (1984).

### Evaluation criteria and statistical analysis

The data collected from Mograne trials were transformed in to mass disease index (MDI) following the method of Ding et al. (1993):

$$MDI = (DI \times DS) / 100$$

Where: DI is the disease incidence and DS is the disease severity. The area under the disease progress curve (AUDPC) and the apparent infection rate (r) were then estimated. The AUDPC was calculated using the equation:

$$AUDPC = \Sigma^{n}_{i=1} [(Y_{i+1} + Y_i) \times 0.5] [T_{i+1} - T_i]$$

Where: $Y_i$ is the MDI at the $i^{th}$ observation, $T_i$ the time (in day) at the $i^{th}$ observation and n the total number of observations. The r was estimated by a linear regression on time (T) of the natural logarithm

**Table 1.** Variation of disease parameters[a] for net blotch reaction in 59 doubled-haploid lines of barley under three environments.

| | $MDI_1$ | $MDI_2$ | $AUDPC_{MDI}$ | $r_{MDI}$ | S | $AUDPC_S$ | $r_S$ |
|---|---|---|---|---|---|---|---|
| Mean | 3.150 | 22.091 | 947.522 | 0.094 | 13.852 | 260.864 | 0.404 |
| Min | 0.080 | 1.500 | 33.700 | 0.068 | 5.000 | 82.670 | 0.381 |
| Max | 10.000 | 58.333 | 2441.000 | 0.109 | 28.333 | 542.080 | 0.427 |
| LSD 0.05 | 1.990 | 9.306 | 452.720 | 0.007 | 4.905 | 63.787 | 0.007 |
| 'Martin' | 5.003 | 59.000 | 2133.760 | 0.111 | - | - | - |

[a] **$MDI_1$,** mass disease index calculated in Mograne during 2002 - 2003; **$MDI_2$,** mass disease index calculated on the basis of data obtained from the last rating date in Mograne during 2003-2004; **$AUDPC_{MDI}$,** area under the disease progress curve calculated from mass disease index estimated in Mograne during 2003 - 2004, **$r_{MDI}$,** apparent infection rate calculated from mass disease index estimated in Mograne during 2003 – 2004; **S,** final disease severity estimated in Tunis 2004; **$AUDPC_S$,** area under the disease progress curve calculated from disease severity estimated in Tunis 2004; **$r_S$,** apparent infection rate calculated from disease severity estimated in Tunis 2004.

(ln) of the diseased tissue proportion (x) divided by the proportion of non-diseased tissue (1-x). The slope of the regression line is then taken as an estimate of r.

For Tunis trial, AUDPC and r were calculated using disease severity. In this trial, the severity was considered equivalent to the mass disease index because controlled conditions have induced a disease incidence of 100%.

Components of variance were estimated for each of the seven evaluation parameters in a single environment, while the analysis for the MDI, the AUDPC and the r was carried out across environments. These analyses were achieved using REML of PROC MIXED of SAS (SAS Institute, 1988).

The models I and II were used for parameters estimated at Mograne (2002 - 2003 and 2003 - 2004) and at Tunis 2004, respectively.

Model I:

$$Y_{ij} = \mu + B_i + G_j + \varepsilon_{ij}$$

with $Y_{ij}$ = observation of genotype j in block i, $\mu$ = general mean, $B_i$ = effect of block i, $G_j$ = effect of genotype j and $\varepsilon_{ij}$ = residual error.

Model II:

$$Y_{ij} = \mu + G_i + \varepsilon_{i(j)}$$

with $Y_{ij}$ = observation of genotype i in replication j, $\mu$ = general mean, $G_i$ = effect of genotype i and $\varepsilon_{i(j)}$ = residual error.

The model III was used for parameters estimated across environments (MDI, AUDPC and r)

Model III:

$$Y_{ijk} = \mu + E_i + R_j(E_i) + G_k + E_iG_k + \varepsilon_{ijk}$$

with $Y_{ijk}$ = observation of genotype k in environment i in replication j, $\mu$ = general mean, $E_i$ = effect of environment i, $R_j(E_i)$ = effect of replication j in environment i, $G_k$ = effect of genotype k, $E_iG_k$ = genotype x environment interaction of genotype k with environment i and $\varepsilon_{ijk}$ = residual error. .

Broad sense heritability ($H^2$) estimates were calculated from variance components. For a single environment, heritability on a plot level was estimated from the following equation:

$$H^2 = \sigma^2g / (\sigma^2g + \sigma^2\varepsilon),$$

and for MDI, AUDPC and r, heritability for mean values across environments was estimated according to the formula proposed by Marwede et al. (2004):

$$H^2 = \sigma^2g / (\sigma^2g + \sigma^2ge/E + \sigma^2\varepsilon/ER)$$

where $\sigma^2g$, $\sigma^2ge$ and $\sigma^2\varepsilon$ are the genotypic variance, the genotype x environment interaction variance and the environmental variance respectively. E and R are number of environments and replicates, respectively.

Genetic correlation coefficients were estimated between parameters investigated in each environment and between the same parameters evaluated in different environments. Variance and covariance components were estimated by the REML method of SAS PROC MIXED (SAS INSTITUTE, 1988).

## RESULTS

Table 1 showed that MDI (or S for Tunis 2004) ranged from 0.08 to 10.00 at Mograne 2002 - 2003, from 5.00 to 28.33 at Tunis 2004 and from 1.50 to 58.33 at Mograne 2003 - 2004, with mean values of 3.15, 13.85 and 22.09, respectively. The magnitude of variations and the mean values of MDI (or S) would suggest that the prevailing growing conditions represent a major component of the disease inducing factors. Thus, lower mean with a limited range values indicate a low epidemic of net blotch, while intermediate mean and range and superior mean with higher range represent moderate and high epidemic of net blotch respectively. Moreover, for the same growing season (2003 - 2004), AUDPC estimates were higher at Mograne (ranging from 33.70 to 2441.00 with a mean of 947.52) than at Tunis (ranging from 82.67 to 542.08 with a mean of 260.86). However, r were lower at Mograne (ranging from 0.068 to 0.109 with a mean of 0.094) than at Tunis (ranging from 0.381 to 0.427 with a mean of 0.404). At Mograne, infection responses of most of the DH lines were significantly lower than that of the susceptible check 'Martin' suggesting quantitative resistance to *P. teres* of these lines. At Tunis, the susceptible check was not used; however, the DH lines showed a relatively moderate degree of resistance, as measured by final net blotch severities (S). Selection, for quantitative resistance to *P. teres* could be more appropriate under high epidemic conditions as those prevailed at Mograne 2003 - 2004 because the DH lines exhibited a large variation in their reaction as described by $MDI_2$, $AUDPC_{MDI}$ and $r_{MDI}$ (Table 1).

**Table 2.** Variance components and heritability estimates of disease parameters for net blotch reaction in 59 doubled-haploid lines of barley in single environment.

| Disease parameter [a] | $MDI_1$ | $MDI_2$ | $AUDPC_{MDI}$ | $r_{MDI}$ | S | $AUDPC_S$ | $r_S$ |
|---|---|---|---|---|---|---|---|
| $\sigma^2_g$ [b] | 8.89** | 214.94** | 44,89 x $10^{4**}$ | 7.49 x $10^{-5**}$ | 26.54** | 1.11 x $10^{4**}$ | 9.94 x $10^{-5**}$ |
| $\sigma^2_\varepsilon$ [c] | 1.82 | 40.15 | 9.43 x $10^4$ | 2.40 x $10^{-5}$ | 10.44 | 0.19 x $10^4$ | 2.19 x $10^{-5}$ |
| $H^2$ [d] | 0.83 | 0.84 | 0.82 | 0.76 | 0.72 | 0.85 | 0.82 |

[a] **$MDI_1$,** mass disease index calculated in Mograne during 2002-2003; **$MDI_2$,** mass disease index calculated on the basis of data obtained from the last rating date in Mograne during 2003-2004; **$AUDPC_{MDI}$,** area under the disease progress curve calculated from mass disease index estimated in Mograne during 2003-2004; **$r_{MDI}$,** apparent infection rate calculated from mass disease index estimated in Mograne during 2003-2004, **S,** final disease severity estimated in Tunis 2004; **$AUDPC_S$,** area under the disease progress curve calculated from disease severity estimated in Tunis 2004 and **$r_S$,** apparent infection rate calculated from disease severity estimated in Tunis 2004.
[b] $\sigma^2_g$, genetic variance.
[c] $\sigma^2_\varepsilon$, error variance.
[d] $H^2$, broad sense heritability estimates.

The variance components of the seven evaluation parameters in each environment were estimated from the analysis of variance (Table 2). All the parameters studied showed highly significant differences ($P < 0.01$) among the DH lines, indicating the presence of genetic variation ($\sigma^2_G$). Thus, broad-sense heritability ($H^2$) estimates on a plot level were high since it ranged from 0.72 to 0.85 for the different disease parameters investigated (Table 2). Similar values of heritability calculated in each of the three environments suggest that residual error variance represent almost 25%.

Pooled analysis of variance across environments indicated the presence of highly significant differences among genotype, environment and genotype x environment interactions for MDI, AUDPC and r (Table 3). Although the genotypic effects ($\sigma^2_G$) were highly significant, genotype x environment interaction effects ($\sigma^2_{GE}$) were larger than the genotypic variance for the three studied parameters. In fact, superiority of the interaction variation ($\sigma^2_{GE}$) relative to the genotypic variation ($\sigma^2_G$) was 513, 1803 and 435% for MDI, AUDPC and r, respectively. Moreover, a relatively high Experimental error was also observed for all parameters. These results affected the broad sense heritability ($H^2$) estimates for mean values across environments. These estimates were 0.35, 0.09 and 0.29 for MDI, AUDPC and r respectively (Table 3).

Genetic correlation coefficients were calculated between the parameters studied in the three environments (Table 4). Genetic correlation between parameters evaluated within each environment ranged from 0.75 to 0.95 and were all highly significant ($P < 0.01$). Mass disease index evaluated at Mograne 2002 - 2003 ($MDI_1$) was genetically associated with MDI evaluated at Mograne 2003 - 2004 ($MDI_2$) and with disease severity evaluated at Tunis 2004 (S). A lack of significant correlation between $MDI_2$ and S was noted. Similarly, no significant correlation was observed between AUDPC estimated at Mograne 2003 - 2004 ($AUDPC_{MDI}$) and at Tunis 2004 ($AUDPC_S$), as well as between r calculated in these two environments ($r_{MDI}$ and $r_S$).

## DISCUSSION

The DH population was screened under low, moderate and high net blotch pressure conditions respectively at Mograne 2002-2003, Tunis 2004 and Mograne 2003 - 2004. These differences noted on the epidemiological levels were mostly attributed to variable sowing dates and the effects of rainfall and temperature that prevailed at the three growing conditions. In the three environments, the disease level can be compared only on the basis of MDI because time between inoculation and the final observation was longer for Mograne trial 2003 - 2004 than for Tunis trial 2004. Therefore, AUDPC will be larger in the first trial even though the terminal MDI values were equal. Similar trend is noted for the apparent infection rate represented by the linear regression coefficient of disease proportion logit on time. For similar terminal disease proportions, the apparent

**Table 3.** Variance components and heritability estimates for the mass disease index (MDI), the area under the disease progress curve (AUDPC) and the apparent infection rate (r) in 59 doubled-haploid lines of barley evaluated under three environments.

| | MDI | AUDPC | r |
|---|---|---|---|
| $\sigma^2_g$ [a] | 13.61** | 12084.00** | 16.46 x $10^{-6}$** |
| $\sigma^2_e$ [b] | 89.03** | 225687.90** | 4.87x $10^{-2}$** |
| $\sigma^2_{ge}$ [c] | 69.85** | 217926.80** | 71.57 x $10^{-6}$** |
| $\sigma^2_\varepsilon$ [d] | 17.47 | 48098.90 | 22.48 x $10^{-6}$ |
| $H^2$ [e] | 0.35 | 0.09 | 0.29 |

**P<0.01. [a]$\sigma^2_g$: genetic variance, [b]$\sigma^2_e$: environmental variance, [c] $\sigma^2_{ge}$: variance of genotype × environment interaction, [d]$\sigma^2_\varepsilon$: error variance, [e]$H^2$: broad sense heritability estimates.

**Table 4.** Genetic correlation coefficients between disease parameters[a] for net blotch reaction in a doubled-haploid barley population under three environments.

| | | $MDI_2$ | $AUDPC_{MDI}$ | $r_{MDI}$ | S | $AUDPC_S$ | $r_S$ |
|---|---|---|---|---|---|---|---|
| Mograne 2002 - 2003 | $MDI_1$ | 0.48** | | | 0.28* | | |
| Mograne 2003 - 2004 | $MDI_2$ | | 0.95** | 0.91** | 0.20NS | | |
| | $AUDPC_{MDI}$ | | | 0.85** | | 0.17NS | |
| | $r_{MDI}$ | | | | | | 0.18NS |
| Tunis 2004 | S | | | | | 0.75** | 0.94** |
| | $AUDPC_S$ | | | | | | 0.86** |

* P<0.05, ** P<0.01, NS not significant at P<0.05.
[a] **$MDI_1$,** mass disease index calculated in Mograne during 2002 – 2003; **$MDI_2$,** mass disease index calculated on the basis of data obtained from the last rating date in Mograne during 2003 – 2004; **$AUDPC_{MDI}$,** area under the disease progress curve calculated from mass disease index estimated in Mograne during 2003 – 2004; **$r_{MDI}$,** apparent infection rate calculated from mass disease index estimated in Mograne during 2003 – 2004; **S,** final disease severity estimated in Tunis 2004; **$AUDPC_S$,** area under the disease progress curve calculated from disease severity estimated in Tunis 2004 and **$r_S$,** apparent infection rate calculated from disease severity estimated in Tunis 2004.

infection rate is superior when disease evaluation period is shorter explaining superior apparent infection rates found at Tunis than that noted at Mograne although the MDI is more significant in this latter environment.

DH lines produced from the cross: 'Roho' x 'Line 90' showed a better level of adult resistance in natural conditions (Mograne) especially in 2003 - 2004 than the susceptible check 'Martin'. Thus, these lines possess different levels of quantitative resistance to net blotch in the field. In controlled conditions (Tunis), the DH lines exhibited a relatively low degree of resistance with different reaction pattern. This result would assume that these DH lines at Tunis express partial resistance to *P. teres*. Quantitative resistance recognized by a reduction of percentage of tissue affected by net blotch was reported by Gupta et al. (2003), Steffenson and Webster (1992) and Tuohy et al. (2006). They consider the term quantitative resistance as a practical term to designate incomplete resistance which have no relation with quantitative genetic. Slow-scalding resistance was considered synonymous with quantitative resistance (Sorkhilalehloo et al., 2002). It exhibited a compatible reaction with *Rhynchosporium secalis* coupled with low to intermediate levels of disease incidence and severity. In this investigation, MDI is used to describe quantitative resistance since it combines disease severity and incidence. Moreover, the quantitative resistance observed in the three environments was characterized by the three following evaluation parameters: MDI, AUDPC and r. They were used to assess the relative reduction on the final infection level, the late symptoms apparition and slow development of the disease.

Broad sense heritability values were large for all parameters using data from single environment. This result supports the finding of Grewal et al. (2008b), Robinson (1999) and Spaner et al. (1998) in the field conditions

and those of Grewal et al. (2008a,b; 2010) in the growth chamber conditions. However, heritability estimates from different growing conditions were low to very low because of genotype x environment interactions as noted for the AUDPC. It is expected that the genotype x environment interactions were significant since the three trial growing conditions differed greatly from each other. Tunis experiment is carried out using a specific monoconidial isolate of *P. teres* to inoculate plants grown in pots; whereas trials at Mograne were conducted under natural growing conditions and inoculated with mixture of local isolate of *P. teres*. Moreover, sowing dates, climatic conditions and variable concentrations of inoculum may result in different levels of net blotch epidemics in the three trials associated with a significant genotype x environment interactions. The AUDPC is the most influenced parameter by the genotype x environment interactions. These results are expected because for each genotype, the disease development depends on the age of the plant, the environmental factors and the aggressivity of the inoculum. Similar genotypes x environment interactions for quantitative resistance to net blotch were obtained for terminal severity, AUDPC and r (Pinnschmidt and Hovmøller, 2002; Robinson and Jalli, 1999; Steffenson and Webster, 1992). These results were explained by the presence of multiple resistance loci in the two parents with environment-specific genetic resistance (Grewal et al., 2008b; Naz.et al., 2008). Earlier study demonstrated that interactions of QTL with environments were significant in the region on chromosome 6H contributing an additional proportion of phenotypic variance for net blotch resistance (Spaner et al., 1998). In addition, the advent of molecular markers has shown that quantitative resistance is due to the segregation of QTLs for pathotype-specific resistance of major effect, along with QTLs of minor effects (Jones et al., 1995).

In a single environment resistance related parameters were found to be associated. These results suggest that quantitative resistance to net blotch is equally described by any criteria. However, results of this investigation indicated that AUDPC would be a valuable tool to select for quantitative resistance to *P. teres* since it describe the pattern of disease across varying growth stages and its magnitude was usually greater than these of the two other parameters. Nevertheless, Steffenson and Webster (1992) found that relationship between the final disease severity and the AUDPC is highly influenced by the environment. In addition, they noted that high values of apparent infection rate could occur sometimes on genotypes with reduced disease severities when there is a rapid increase of net blotch attacks from a low to a moderate level within a short period. Thus, they proposed the AUDPC as the most reliable statistic for assessing quantitative resistance to *P. teres*. However, El Yousfi and Ezzahiri (2001), found that the environmental effect remained significant until the cultivars reached the flowering stage at which the environment have no effect on the linear relationship between disease severity and the AUDPC. It was thus suggested that, discrimination between genotypes for quantitative resistance is best expressed at this growth stage.

In this investigation, genetic correlations were detected only between MDI evaluated at Mograne 2002-2003 with those evaluated at Mograne 2003 - 2004 and at Tunis 2004. Li et al. (2009) and Mahmoud et al. (2006) explained the significant genetic correlations by genetic linkage and/or pleiotropic effects of loci affecting the considered traits. Thus, the results obtained suggest that loci for MDI may be pleiotropic or linked and loci for AUDPC and r may be independent. Further QTL-based analyses are required to elucidate the genetic correlation between net blotch resistance parameters and to well know whether these correlations were due to linkage among genetic factors or pleiotropy or both.

Results obtained from this research show that net blotch resistance is quantitative and those genotypes x environment interactions were the major sources of variation. In a single environment, resistance related parameters were found to be genetically associated. Excepting mass disease index, lack of associations between area under the disease progress curve and apparent infection rate measured on the three field conditions were observed.

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