

Diallel analysis of the resistance to powdery mildew (*Erysiphe graminis* f.sp. *hordei*) in barley (*Hordeum vulgare* L.)

M. A. BARGOUGUI

School of Higher Education in Agriculture of Mateur

*Corresponding author: Bargouguedali@yahoo.fr

Abstract - A complete diallel cross including five barley genotypes was analyzed to study the genetic interaction between barley "*Hordeum vulgare*" and powdery mildew "*Erysiphe graminis* f. sp. *hordei*". The results revealed an important genetic variability. The resistance is expressed as a dominant character. The genetic decomposition of variance showed preponderant general combining ability, which let us to expect good selection efficiency. Such assumption is sustained by very high narrow sense heritability equal to 0.70. This study highlights that the varieties Manel and INAT102 as good sources of genetic resistance to barley powdery mildew. These two lines and their hybrids may be used as good head. According to the results, a genetic improvement program on the resistance to barley powdery mildew can be based on pedigree method to take advantage of the transgression within progenies.

Keywords: Combining ability, Breeding program, *Erysiphe graminis* f. sp. *Hordei*, genetic resistance resources, *Hordeum vulgare* L., narrow sense heritability.

1. Introduction

Barley is among the most widely grown annual crop in North Africa, but foliar diseases are major factors affecting its productivity and aggravating the effects of the inherent drought of the region. Powdery mildew, net blotch, scalds and barley yellow dwarf virus are the most encountered diseases (Amril 2002)

In Tunisia, Barley powdery mildew is considered among the main fungal diseases. Chalghaf et al. (1993) considered it as the most frequent disease with net blotch and scald. However, Harrabi et al. (1991) and ElFelah (1998) situated the powdery mildew in a second place after the net blotch. Yahyaoui et al. (1993) reported that barley powdery mildew causes equivalent loss as that caused by barley stripe.

Barley Powdery mildew causes about 30% yield loss (Cherif and Harrabi 1990). Moreover, Kamel et al. (1987) noted that about 10 to 66% of Tunisian fields are often infected by powdery mildew and indicated that this disease has the most widespread distribution spectrum.

In fact, *Erysiphe graminis* f.sp *hordei* has a worldwide distribution and it is found mainly in all continents, but the yield reductions vary according to the infection's beginning and the severity of the epidemic that can run to so much as 30 % (Cook and King 1984). In addition, James and Sicksen (1956), Sloomaker and Van Essen (1969) and Sutton (1996) established this and indicated 15 to 20% yield reduction. The infection begins at the lower leaves then moves to the higher leaves and may reach all parts of the plant when the infection is heavy. The infection manifests itself very clearly in the formation of whitish to pale grey flour-like pustules on the assimilation organ. It appears in winter as little white pustules scattered on the leaf surface. The pustules are made of mycelium and conidial chains that extend and turn to yellowish and grayish color. After that a black scattering punctuation made of cleistothecia appears (Nasraoui 2000).

Several genetic studies of pathogenicity of the powdery mildew have been done and led to different results. Indeed, Moseman (1959) has demonstrated the oligogenic determinism of the host resistance, indicating functional alleles generally inherited as dominant character.

Afterward, Moseman (1966) concluded for the existence of an almost unlimited number of barley variety having genes conditioning resistance to *E. graminis hordei*, located on chromosome 5 and distributed at complex loci such as the Mla locus. He also, assessed for the existence of numerous physiologic races of powdery mildew that can be distinguished with different barley varieties. Both

resistance and virulence genes control the host-parasite interactions, and when corresponding virulence genes come up against dominant resistance genes, the result is an incompatibility reaction. For Gallais and Bannerot (1997) and Hafidi et al. (1996), a major recessive gene, induced by artificial mutagenesis “Mlo” controls a full resistance to barley powdery mildew. However, Maroof et al (1997) demonstrated that resistance reaction involves many genes with a main additive action, particularly when the varieties are devoid of major genes, but according to Brown and Wolfe (1990), single barley genotype cannot include all these genes. The host-specificity of the parasitism is the basis for the differentiation of “special forms” within which numerous physiological strains have been identified on test assortment (Menzies and Mac-Neill 1986). This specificity allowed differentiating numerous barley genotypes containing resistance genes, commonly named: differential series, such as the European differential series containing 25 near-isogenics barley genotypes (Kolster et al. 1986) and the American differential series containing 13 genotypes (Hafidi et al. 1996).

Caldo et al. (2006) elucidated how basal defense responses influence the onset of *Mla* (mildew resistance locus *a*)-specified resistance. They assumed that the regulation of basal defense influences host-cell accessibility to the fungal pathogen and drives allelic diversification of gene-specific resistance phenotypes particularly in barley genotypes containing the *Mla1*, *Mla6*, or *Mla13* alleles.

Pavan et al (2010) reported that many recent studies on plant immunity have suggested that a pathogen should suppress induced plant defense in order to infect a plant species, which otherwise would have been a non-host to the pathogen. They proposed a novel breeding strategy called disabling plant disease susceptibility genes (S-genes) to achieve durable and broad-spectrum resistance.

Repeated cultivation of barley varieties with specific resistance genes over many years leads to a gradual increase in adaptation of strains with appropriate virulence because of selection pressure. If the frequency of the pathogen’s virulence compared with the host’s resistance increases, the incompatibility becomes less and less effective, and vice versa (Menzies and Mac-Neill 1986). Wolfe (1987) reported a similar selection pressure and the associated loss of sensitivity to fungicides on the part of the mildew population caused by huge use of fungicides.

The data reported in the present paper deal with a genetic study of barley and powdery mildew interaction. Indeed, the main purpose of our present work is to investigate resistance resources that could be used to produce new genetic variability, required to carry out a breeding program of the resistance to barley powdery mildew.

2. Materials and methods

2.1 Plant material

Five barley parental varieties, described in the table 1, have been systematically crossed, generating 20 hybrid progenies. As *Hordeum vulgare* is normally self-fertilized species, so, artificial hybridization was needed to cross it by emasculation and four dates of seeding, 10 to 15 days separated, have been considered to get enough time to do all crosses. Indeed seven ears for every cross were artificially hybridized.

Table 1: Barley parental varieties

Varieties	Origin	Characteristics
Rihane	ICARDA / 1992	Six-rowed, early line, susceptible to powdery mildew and to net blotch, good yield potential.
Manel	INRAT and ICARDA / 1996	Six-rowed, early line, high level of resistance to diseases and to lodging, good yield potential.
Martin	Cultivated since 1931	Six-rowed, susceptible to powdery mildew, to barley stripe, to brown rust and susceptible to lodging.
INAT-102	INAT / Genes Bank	Six-rowed, early line, good yield, tolerant to most important fungal diseases.
Souihli	Landrace / Genes Bank	Six-rowed, relatively late line, susceptible to most fungal diseases and lodging.

The five parental varieties and their twenty F1 hybrids progenies were grown the same year in the field and under the same crop condition, according to a completely randomized design, in which the experimental unit is represented by a row of 125 cm length for each genotype, containing twenty plants.

2.2 Assessment of the infection level

The evaluation of barley powdery mildew infection of all genotypes had been realized in the same crop conditions, under the same environment affect and at the most favorable infection stage. Barley plants infection were evaluated under natural epidemics. For each parent and each hybrid, twenty observations were done according to a five notes scale, established by Bargougui and Chalbi (2005), presented in table 2.

Table 2: Scale for appraising the infection level

Note	Infection level	Reaction
0	No visible infection	Highly resistant (HR)
1	Few lesions only on the lower leaves	Resistant (R)
2	Intense infection of the lower leaves but light on the median leaves	Moderately resistant (MR)
3	Intense infection of the half lower of the plant but light on the higher leaves	Moderately susceptible (MS)
4	All the leaves are severely infected.	Susceptible (S)

2.3 Statistical analysis

2.3.1 Analysis of the variance

A complete analysis of variance has been conducted according to the adopted randomized design experiment considering the linear model:

$$y_{ijk} = \mu + g_{ij} + r_k + \varepsilon_{ijk}$$

Where:

y_{ijk} = k^{th} observation of the cross between the i^{th} and j^{th} parents.

μ = mean

g_{ij} = effect of the ij^{th} genotype

b_k = effect of the k^{th} repetition.

ε_{ijk} = a random effect

The variability among the parental varieties and their hybrid progenies has been afterward submitted to a complete diallel cross analysis to throw light on combining ability study.

2.3.2 Combining ability analysis

The major utility of diallel cross analysis is the evaluation of general and specific combining ability. Another use of the diallel cross technique is the early generations evaluation of parental material in breeding programs. In our study, the five parental genotypes are deliberately chosen and intercrossed. All p^2 combinations were tested and all the genotypes are assumed a fixed set. In fact, our aims are firstly, to acquire information about the genetic system governing resistance to powdery mildew, secondly, to identify parents whose hybrids are most likely to respond to selection and thirdly, to predict segregation in later generations. Consequently, the data analysis was made according to Griffing's Model I (Griffing, 1956), in which we are particularly interested in estimating combining ability effects and computing appropriate standard errors for differences between effects.

For p parents and n observations for each genotype, the mathematical linear model for the combining ability analysis is assumed to be

$$y_{ij} = \mu + g_i + g_j + s_{ij} + r_{ij} + \frac{1}{n} \sum_k \varepsilon_{ijk}$$

Where

μ = General mean value.

g_i = Additive mean effect of female parent i , ($i = 1 \dots p$)

g_j = Additive mean effect of male parent j , ($j = 1 \dots p$)

s_{ij} = Specific effect of the (i,j) cross.

r_{ij} = Reciprocal effect,

ε_{ijk} = Residual error associated to the observation.

To classify the different genotypes experimented according to their combining abilities we use appropriate Student t tests based on their own computed least squares and considering corresponding degrees of freedom.

3. Results and discussion

The obtained data are summarized and presented in the table 3.

Table 3: Means value and their variances of all parents and hybrids

	Rihane		Manel		Martin		INAT-102		Souihli	
	M	σ^2	M	σ^2	M	σ^2	μ	σ^2	μ	σ^2
Rihane	3.20	0.48	0.55	0.58	2.25	1.36	1.70	0.54	2.30	0.43
Manel	1.60	1.09	0.30	0.33	0.55	0.69	0.00	0.00	0.10	0.09
Martin	2.10	0.41	0.10	0.09	2.70	0.75	1.35	0.35	1.50	0.47
INAT-102	1.90	0.41	0.50	0.37	1.60	0.99	0.80	0.59	1.00	0.58
Souihli	2.05	0.83	0.25	0.30	1.80	0.48	2.05	0.89	1.65	0.45

3.1 Analysis of the genetic variability

The homogeneity of data variances has been checked, by using Bartlett's criteria. The calculated χ^2 is equal to 36.29 that prove likelihood of homogeneity hypothesis at 0.05. As it can be noticed in table 3, hybrids' mean values are often less than their parents' mean values, showing a significant higher level of resistance.

The first stage of the analysis is to test the null hypothesis that there are no genotypic differences between all genotypes and between all replicates. The analysis of variance for infection level among all genotypes and all replicates is given in table 4.

Table 4: analysis of variance for infection level

Source	d.f.	Mean Score	Variance ratio	Pr>F
Total	499			
Genotypes	24	15.876	29.97	0.0001
Replicates	19	0.6985	1.34	0.1551
Error	456	0.52265		

A high significant genotypic variability is observed. As a result, parents and their hybrids react in different ways to powdery mildew. No replicate effects are noticed, which is probably due to the homogeneity of the experimental conditions. The importance of main genetic effects allows us to conclude for a genetic determinism of barley reaction to powdery mildew. Indeed, the Duncan t test confirms such hypothesis, and reveals different genotypic susceptibility levels. Table 5 gives the classification of genotypes according to their infection level.

We can notice, in table 5, the least susceptible genotypes are Manel's progenies. Therefore, the resistance within Manel is probably a complete dominant character. The progenies of INAT-102 and Souihli are moderately resistant; the dominance of their resistance may be incomplete. The progenies of Martin and Rihane are the most susceptible. In the last case the progenies are less susceptible than their parents. Therefore, the susceptibility is probably a recessive character.

Comparing Manel's reaction with that of its progenies, we notice an enhanced level of the resistance, probably due to an additive effect of minor genes beside the assumed major genes in Manel.

3.2 Diallel cross analysis

The previous analysis of the variance showed that there is a main genotypic effect but replicate effect is not significant. Hence, the replicate variance has been included within the error variance and, genotypic variance was decomposed into general combining ability effects of parents, specific combining ability effects of crosses and reciprocal effects.

The analysis of variance, according to Griffing's Model I of the genotypic infection level is given in table 6 F ratios calculated underline a highly significant main general combining ability effect. Likewise, all other effects are significant.

Table 5: Genotypes classification according to their infection level

Genotype	Means value of infection level								
Rihane	3.20	a							
Martin	2.70	b							
Souihli x Rihane	2.30	b	c						
Martin x Rihane	2.25	b	c						
Rihane x Martin	2.10	c	d						
Rihane x Souihli	2.05	c	d						
INAT-102 x Souihli	2.05	c	d						
Rihane x INAT-102	1.90	c	d	e					
Martin x Souihli	1.80	c	d	e	f				
INAT-102 x Rihane	1.70		d	e	f				
Souihli	1.65		d	e	f				
Martin x INAT-102	1.60		d	e	f				
Rihane x Manel	1.60		d	e	f				
Souihli x Martin	1.50			e	f				
INAT-102 x Martin	1.35				f	g			
Souihli x INAT102	1.00					g	h		
INAT-102	0.80						h	i	
Martin x Manel	0.55						h	i	j
Manel x Rihane	0.55						h	i	j
Manel x INAT102	0.50							i	j
Manel	0.30								j
Manel x Souihli	0.25								j
Manel x Martin	0.10								j
Souihli x Manel	0.10								j
INAT-102 x Manel	0.00								j

Two genotypes followed by the same letter are not significantly different.

Table 6: Analysis of variance according to Griffing's Model I

Sources	d.f	Mean Score	Variance ratio	Pr>F
General combining ability	4	3.8365	144.86	0.0001
Specific combining ability	10	0.22279	8.412	0.0001
Reciprocal maternal effect	4	0.1085	4.097	0.0001
Reciprocal specific affect	6	0.17412	6.574	0.0001
Error	475	0.02648		

** The difference is highly significant at 0.01

NS: The difference is not significant

Our assumption here is that genetic determinism of the interaction between the host and the parasite includes concurrently an additive and non-additive genetics effects. However, the additive contribution seems to be predominant. In fact, using the appropriate t tests all parents are significantly different according to their general combining abilities, as it appears from the classification presented in table 7.

Table 7: Classification of parents according to their general combining abilities

Parent	General combining ability	Classification for $\alpha = 5\%$
Rihane	0.729	A
Martin	0.309	b
Souhli	0.079	c
INAT-102	- 0.186	d
Manel	- 0.931	e

$$\text{Var}(g_i) = 0.0021 \text{ et } \text{Var}(g_i - g_j) = 0.00532$$

Manel has the most significantly suitable general combining ability, followed by INAT-102. Such result reveals once again simultaneous dominance and additive actions in the genetic control of the resistance. Considering the general combining abilities, the prediction of each genotype's mean value (α_{ij}) can be given by the following expression: $\alpha_{ij} = \mu + g_i + g_j$

Where: μ = general means value,

g_i = general combining ability of parent i.

g_j = general combining ability of parent j.

Then, correlation between the predicted and the observed values is established. The coefficient of correlation is equal to 89.758 %, highly significant. There is a very important additive action in the genetic determinism of the barley reaction to the powdery mildew.

The Specific combining abilities of all genotypes are computed and presented in table 8.

Table 8: The specific combining abilities

	Rihane	Manel	Martin	INAT-102	Souihli
Rihane	0.386	- 0.079	- 0.219	- 0.099	0.011
Manel	- 0.079	0.806	- 0.409	0.011	- 0.329
Martin	- 0.219	- 0.409	0.726	- 0.004	- 0.094
INAT-102	- 0.099	0.011	- 0.004	- 0.184	0.276
Souihli	0.011	- 0.329	- 0.094	0.276	0.136

$$\text{var}(s_{ii}) = 0.017 ; \text{var}(s_{ij}) = 0.009 \text{ et } \text{var}(s_{ij} - s_{ki}) = 0.01596$$

Most of the hybrids have negative values concerning their specific combining abilities. There is probably a genetic interaction favorable to the resistance. This is obvious especially among Manel's progenies.

Once more, considering general and specific combining abilities, the predicted genotype's mean value (β_{ij}) may be as follow:

$$\beta_{ij} = \mu + g_i + g_j + s_{ij}$$

Where:

μ = General mean value for all 25 genotypes.

g_i = General combining ability of parent i

g_j = General combining ability of parent j

s_{ij} = Specific combining ability of hybrid (i,j)

The correlation between observed and predicted values becomes more significant, with a coefficient equal to 95.53%. Once more, the genetic determinism of the resistance seems to include additive and non-additive effects.

This analysis gives an idea about the genetic determinism of the Barley-powdery mildew interaction. The resistance seems to be dominant but not monogenic. It is more likely that there are minor genes adding their effects to the major genes action. In deed according to Aghnoum et al. (2009), the basal resistance of barley to powdery mildew (*Blumeria graminis* f. sp. *hordei*) is a quantitatively inherited trait that is based on nonhypersensitive mechanisms of defense.

Likewise, the present diallel cross analysis let us thinking subsequent genetic increase of the resistance to barley powdery mildew by selection for genetic gain. In fact, according to the general and specific combining abilities, the genotypes Manel and INAT-102 may be considered as good genotypes to improve the resistance to barley powdery mildew. Their crosses can be retained as heads of lines in genetic improvement programs. Finally, as it is obvious in table 9, we notice that the resistance of Manel and INAT-102 depends on reciprocal effect and the crossing way must be considered.

Table 9: Reciprocal effects

	Rihane	Manel	Martin	INAT-102	Souihli
Rihane		- 1.05	0.15	- 0.20	0.25
Manel	1.05		0.45	- 0.50	- 0.15
Martin	- 0.15	- 0.45		- 0.25	- 0.30
INAT-102	0.20	0.50	0.25		- 1.05
Souihli	- 0.25	0.25	0.30	1.05	

$$\text{var}(r_{ij}) = 0.0133, \text{var}(r_{ij} - r_{kl}) = 0.0266$$

3.3 Heritability analysis

The heritability of a character expresses the proportion of total variance that is attributable to the average effects of genes, and determines the degree of resemblance between relatives. Therefore, the heritability is usually estimated from the degree of resemblance between relatives. If b_{EP} is the coefficient of regression of progenies' values on those of their parents, then heritability is expressed as follow: $h^2 = 2b_{EP}$, (Falconer, 1974).

In this study, the regression of hybrids' infection level (IL_H) on their parents' values (IL_P) led to linear model highly significant at a probability equal to 0.0014.

$$IL_H = 0.66639 + 0.346 IL_P$$

Taking into account this model, the heritability is equal to 0.70 and such value indicates that barley-powdery mildew interaction is genetically determined and we have there an almost genetic improvement progress, to get by selection.

4. Conclusion

Irrelevant with conclusion, the study reveals that estimate of heritability is 70%, and indicates that major part of the total phenotypic variability can be attributed to genetic rather than environmental causes. Sustaining evidence for this conclusion was obtained from statistical tests, displaying non-significant replicates' variance and preponderant genotypic effect. These results are an indication that correspondence between genotype and phenotype is satisfying. It is therefore expected that effective selection should be possible to improve the resistance to barley powdery mildew.

Based on the diallel analysis results, it was possible to make inferences about the genetic component of the total variability. Major genes exhibit a complete dominance in the direction of the resistance, particularly in Manel genotype. Adding to this, the genetic variability is probably associated to some minor genes with an additive action. The revealed high significant general combining ability underlines this assumption.

It is therefore expected that progress under selection may occur by crosses between parents that carry different genes. Rapid progress toward homozygous types equaling or perhaps slightly transgressing the resistance level of the parents can be expected from selection. The cross between Manel and INAT-102 can be regarded as the most promising head of lines to progress in the direction of resistance. In fact, the cross between Manel and INAT-102 provided a highly resistant hybrid, better than its two parents. In this case, the pedigree selection method seems to be the more adequate.

In crosses between parents carrying different minor genes, progress depends entirely on the polygenic system. If that is the case, a recurrent selection program should be necessary to combine progressively most genes within improved genotypes. This may be suggested to use many other crosses, particularly those including Manel or INAT-102 as one of the parents.

5. References

- Aghnoum R, Marcel TC, Johrde A, Pecchioni N, Schweizer P, Niks RE (2010)** Basal Resistance of barley to powdery mildew: connecting Quantitative Trait Loci and Candidate Genes. *Mol. Plant-Microbe Interactions*. 23: 91–102.
- Amril A, Yahyaoui A, El-Moustafa T (2002)** Reaction of barley landraces to major foliar diseases. 2nd International workshop on barley leaf blight, ICARDA: 43
- Bargougui MA, Chalbi N (2005)** Système de notation pour une évaluation quantitative de l'interaction génétique orge/oïdium «*Blumeria (Erysiphe) graminis* F. sp. *Hordei*» *Revue Soc. Sci. Nat. de Tunisie* T: 31 : 9-7.
- Brown JKM., Wolfe MS (1990)** Structure and evolution of population of *Erysiphe graminis* f. sp. *hordei*. *Plant Pathology* (39): 376–390.
- Caldo RA, Nettleton D, Peng J, Wise RP (2006)** Stage-Specific Suppression of Basal Defense Discriminates Barley Plants Containing Fast- and delayed-Acting *Mla* Powdery Mildew Resistance Alleles. *Mol. Plant-Microbe Interactions*. 19 : 939–947.
- Chalghaf E, Harrabi M, Yahyaoui O, Cherif M, Abed M, Sbei A, Snoussi N (1993)** Surveillance des maladies des céréales en Tunisie 92/93 et leur évolution durant 5 années. *Rapport Annuel sur la surveillance des maladies et amélioration du germoplasme des céréales et des légumineuses alimentaires* : 129-132.
- Cherif M, Harrabi M (1990)** Diagnostique des maladies des grandes cultures, Institut National Agronomique de Tunis 3–13.

- Cook RJ, King JT (1984)** Loss caused by cereal diseases and the economics of fungicidal control. Plant Diseases: Infection, Damage, loss. Ed. Wood R. A. S., Jellis G. J., British Society for plant pathology 237-245.
- ElFelah M (1998)**. Evaluation de 423 éctypes d'orge collectés en Tunisie (Centre et Sud). Utilisation en alimentation variétale pour la sélection d'idéotypes adaptés à différentes régions bioclimatiques. Thèse de Doctorat en Biologie. Faculté des Sciences de Tunis
- Falconer DS (1974)** Introduction to quantitative genetics, ed: John Wiley & Sons, Inc., New York
- Gallais A, Bannerot H (1997)** Amélioration des espèces végétales cultivées, objectifs et critères de sélection. INRA ed: mieux comprendre p: 64
- Griffing B (1956)** Concept of general and specific combining ability in relation to diallel crossing systems. Austral. J. Biol. Sci 9: 463-493
- Hafidi M, Yahiaoui A, Amri A, Saadaoui M (1996)** Caractérisation du spectre de virulence des populations Marocaines d'*Erysiphe graminis* f. sp. *hordei* Dans Proceedings du Symposium Régional sur les Maladies des Céréales et des Légumineuses Alimentaires. Imprimerie El Maarif Al Jadida RABAT
- Harrabi M, Chérif M, Morjan H (1991)** Evaluation des dégats causés par les maladies cryptogamiques sur les grandes cultures.
- James G, Sickson P (1956)** Diseases of field crops. Mc Graw-hill book company, New York: 517
- Kamel AH, Harrabi M, Deghais M, Halila H, BenSalah M (1987)** Wheat and barley diseases in Tunisia. Rachis. 6(2): 24-29
- Kolster, Munk L, Stolen O, Lohde G (1986)** Near-isogenic barley lines with genes for resistance to powdery mildew. Crop Science. 2: 903-907
- Maroof MAS, Zhang Q, Biyashev RM (1997)** Molecular marker analyses of powdery mildew resistance in barley. Theoretical and Applied Genetics (1994) Vol: 88(6-7): 733-740. In Literature Update on Wheat, Barley and Critical. Vol: 3 No.5 ed: CIMMYT et ICARDA
- Menzies J. G. and B. H. MacNeil, 1986.** Virulence of *Erysiphe graminis* f. sp. *Tritici* in Southern Ontario in 1983, 1984 and 1985. Canadian Journal of Plant Pathology. 8: 338-341
- Moseman JG (1966)** Genetic of powdery mildews Crops Research Division Agriculture Research Service, United States Department of Agriculture, Beltsville Maryland. 269-290
- Moseman JG (1959)** Host-Pathogen interaction of the genes for resistance in *Hordeum vulgare* and for pathogenicity in *Erysiphe graminis* f. sp. *hordei*. Phytopathology 49: 469 – 472.
- Nasraoui B (2000)**. L'oïdium des céréales, Principales Maladies Fongiques des Céréales en Tunisie. Centre de publication Universitaire. 71
- Pavan S, Jacobsen E, Visser RGF, Bai Y (2010)** Loss of susceptibility as a novel breeding strategy for durable and broad-spectrum resistance. Molecular Breeding 25 : 1-12.
- Slootmaker LAJ, Van Essen A (1969)** Yield losses in barley caused by mildew attack. Reprint Netherlands J. agric. Sci 17: 279-282.
- Sutton JC (1996)** Maladies des feuilles et des épis de l'orge Fiche technique, ed: Ministère de l'Agriculture et des Affaires Rurales. L'Ontario. 96: 136
- Wolfe MS (1987)** Dynamics of the response of barley mildew « *Erysiphe graminis* f. sp. *hordei* » to the use of sterol synthesis inhibitors. In Wheat, Barley and Triticale Bibliography ed. CIMMYT, 3 (2): 37
- Yahyaoui AH, Cherif M, Harrabi M (1993)** Barley Disease Incidence in Tunisia. International Workshop on barley leaf blights. ICA