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The determination of the resistance inheritance against common bunt in wheat and half-diallel hybrids

Gülçin Akgören Palabiyik[1], İsmail Poyraz[2,3], Ahmet Umay[4]*

[1]*Medicinal and Aromatics Plants Department, Pazaryeri Vocational School, Bilecik Seyh Edebali University, Bilecik, Turkey*

[2]*Molecular Biology and Genetics Department, Faculty of Science and Letters, Bilecik Seyh Edebali University, Bilecik, Turkey*

[3]*Biotechnology Application and Research Centre, Bilecik Seyh Edebali University, Bilecik, Turkey*

[4]*Test Research Unit Department, Distance Education Faculty, Anadolu University, Eskisehir, Turkey*

**Corresponding author: gulcin.akgoren@bilecik.edu.tr*

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Abstract: This study was conducted to determine the inheritance of common bunt resistance in twelve bread wheat varieties and their half-diallel hybrids in Turkey. The disease ratings were performed on the F_2 generations of the hybrids in field conditions. The obtained data were analysed by the χ^2 test to determine the effective gene numbers and inheritance type in the disease resistance. In addition, the data were evaluated according to the Jinks-Hayman diallel analyses. In conclusion, it was found that of the twelve wheat parents, four contained three resistance genes and four of them contain two resistance genes. The dominant genes were prominent in the population and complete dominance was present. Therefore, the selection for disease resistance should be delayed until the following generations.

Keywords: chi-squared test; *Tilletia* sp.; inheritable resistance; resistance genes; breed wheat

The common bunt (*Tilletia*) is one of the most important diseases that reduces the wheat yield. *Tilletia* (*T. tiritici* and *T. foetida*) causes a decrease in the product's quality and yield (Ciucă 2011; Elyasi-Gomari & Farrokhi-Nejad 2013). *Tilletia* sp. causes disease in almost all wheat cultivation areas. Approximately 150 million spores of the common bunt can be found in a diseased wheat spike and these spores contaminate three million seeds on average. It has been reported that if the plants are not protected against this disease, damage could range from 15–20%. When the seed is planted without any chemical treatment for a few years, this damage could reach 75–90% (Kochanová *et al*. 2004; Akan *et al*. 2014).

This disease is also quite widespread in Turkey. The common bunt can be controlled by seeds being treated with chemical agents; however, the development of disease resistant varieties is seen as the best protection method because the chemical agents cause environmental pollution (Dumalasová & Bartoš 2008; Oncică & Săulescu 2008; Furan & Yüce 2009; Knox *et al*. 2013; Yarullina *et al*. 2014).

Resistance against the common bunt disease is expressed with the *Bt* genes. The *Bt* genes are determined by using the race differential set (Matanguihan 2011). Resistant varieties can be developed by crossing the genotypes with the *Bt*. Moreover, understanding the genetic mechanism that is effective in the in-

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heritance of the disease resistance and selecting the appropriate parents will increase the success of the breeding studies. To this aim, the diallel method is reliable (Şener *et al*. 2000; Kutlu & Olgun 2015). This study aimed to investigate the inheritance of the common bunt resistance in twelve bread wheat varieties and their half diallel in the field conditions.

MATERIAL AND METHODS

The experimental area and material. This experiment was conducted between 2012 and 2014 in the field of the Transitional Zone Agricultural Research Institute (TZARI), in a randomised complete block design. The pedigrees and disease reaction types of the twelve bread wheat varieties used as the material in this study are presented in Table 1.

The PI178383 and M732154 wheat varieties were used in the hybridisation. These varieties are in the differential set that contains the bunt resistance genes. PI178383 carries the *Bt*-8-9-10 genes while M732154 carries the *Bt*-3-7-8 genes (Matanguihan 2011). *Tilletia* isolates (*T. foetida*) used for the inoculation were obtained from TZARI.

The hybridisation and disease inoculation. In 2012, hybrids were obtained from a half-diallel mating design. In October of the second year, the seeds were contaminated with spores before sowing. The disease rating of the F_2 hybrids and their parents were performed in May 2014 and the disease discrimination was observed macroscopically (Figure 1). According to the disease rating scale, the disease reactions were determined as follows: 41% and above is considered susceptible (S), 11–40% is considered medium resistant (MR) and 0–10% is considered resistant (R) (Mirza & Khan 1983; Tuncel & Boyraz 2006).

The statistical analysis. The χ^2 test was used to determine the rate of the resistance and susceptible plants in the F_2 generation, the gene numbers controlling the common bunt, the inheritance type of these genes, and the relationship among the different genes.

In some combinations, since the seed could not be obtained due to a hybrid mismatch, the statistical analysis was performed according to the 8 × 8 half-diallel mating design. The genetic parameters and the ratios between these parameters proposed by

Figure 1. The disease discrimination of the Alpu01 × Karahan99 cross: bunt infected head **(A)**, healthy head **(B)**

Table 1. The names, pedigrees, disease reactions and origins of the wheat varieties

Variety	Pedigree	Disease reaction	Origin
Alpu01	ID800994.W/VEE	susceptible	TZARI
Altay2000	ES14//YKT/BLUEBOY2		TZARI
Karahan99	C126-15/COFN/3/N10B/P14//P101/4/KRC		BDIARI
Ikizce	ATR*2/7C//BI		FCCRI
Mufitbey	NGDA146/4/YMH/TOB//MCD/3/LIRA/5/F130L1.12		TZARI
Yayla305	Mixture of lines 1705, 505, 157		TZARI
Kirac66	Floransa171/Yayla305	resistant	TZARI
Soyer02	ATAY/GALVEZ87		TZARI
Sultan95	AGRI/NAC		TZARI
Nacibey	F900K/3/EGL//BUC/PVN		TZARI
PI178383	Domestic variety		Turkey
M732154	Domestic variety		ICARDA

TZARI – Transitional Zone Agricultural Research Institute; BDIARI – Bahri Dagdas International Agricultural Research Institute; FCCRI – Field Crops Central Research Institute; ICARDA – International Center for Agricultural Research in the Dry Areas

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Jinks and Hayman (1953) were performed using the TarPopGen Statistical Package Program developed by Ozcan (1999).

RESULTS

The disease rates of the parents examined in the study were presented in Table 2. Alpu01 was detected as the most susceptible variety at a 71.34% disease rate, while the Karahan99, Kirac66, Mufitbey, Nacibey, PI178383 and M732154 varieties were completely resistant to the disease. In addition, a medium resistance was observed in the Yayla305, Altay2000, Soyer02, Ikizce and Sultan95 varieties.

The disease and segregation ratio, the inheritance form, and the χ^2 values of the hybrids between the susceptible variety Alpu01 and some resistant varieties are presented in Table 3. The disease rates changed from 1.5 to 22.5%. The Alpu01 × Yayla305 hybrid showed the highest resistance to disease. The Alpu01 × Karahan99 hybrid showed high susceptibility (its segregation ratio was 3R : 1S), and it was determined that the resistance property was managed by a single gene pair.

Table 2. The disease rates of the parental varieties

Variety	Total No. of plants	No. of bunted plants	Infected plants (%)	Disease reaction
Alpu01	178	127	71.34	S
Altay2000	168	47	27.97	MR
Karahan99	155	0	0	R
Ikizce	210	71	33.8	MR
Mufitbey	169	3	1.7	R
Yayla305	198	35	17.68	MR
Kirac66	196	2	1	R
Soyer02	185	49	35	MR
Sultan95	136	27	19.85	MR
Nacibey	118	0	0	R
PI178383	127	0	0	R
M732154	118	0	0	R

S – susceptible; MR – moderately resistant; R – resistant

We determined that the hybrids of Alpu01 with Altay2000, Kirac66, Nacibey, and Sultan95 had a 15R : 1S segregation ratio. The segregation in these hybrids showed that the common bunt resistance was controlled by two gene pairs. The segregation rate in Alpu01 × Mufitbey was 57 : 7 while in Alpu01 × Yayla305 it was 63 : 1.

The hybrids between the resistant genotypes showed a different segregation ratio, and the heredity types changed according to the resistance genes they carried (Table 4). While 10 hybrids could not be evaluated due to the environmental conditions, such as the shortness of the hybridisation period and the incompatibility of the flowering, 32 hybrids were determined to be completely resistant. It was understood that the disease resistance in the resistant × resistant hybrids was controlled by three gene pairs. There were five hybrids showing a resistance of 60 with a susceptible segregation ratio of 4. The Altay2000 × Sultan95 and PI178383 × Sultan95 hybrids showed a segregation ratio of 57 : 7 while the Ikizce × PI178383 and Nacibey × Yayla305 hybrids showed a segregation ratio of 45 : 19. The Nacibey × Soyer02 hybrid showed a 48 : 16 segregation ratio (Table 4).

The variation of the genetic components, the ratios between the genetic parameters and the estimates of the heritability for the disease rates are presented in Table 5. The expected environmental effect and all the genetic parameters were found to be non-significant except for the complete dominance effect. The additive genetic component (*D*)

Table 3. The disease rate inheritance form and the χ^2 values of the susceptible × the resistance hybrids in the F_2 generation

Hybrid	Total No. of plants	No. of bunted plants	Infected plants (%)	Disease reaction	χ^2	Possibility rate	Segregation	Gene No.
Alpu01 × Altay2000	104	8	7.7	MR	0.369	0.50–0.70	15 : 1	2
Alpu01 × Karahan99	315	71	22.5	MR	1.017	0.30–0.50	3 : 1	1
Alpu01 × Kirac66	253	11	4.4	R	1.595	0.20–0.30	15 : 1	2
Alpu01 × Nacibey	211	13	6.2	R	0.003	0.95–0.99	15 : 1	2
Alpu01 × Mufitbey	228	30	13.2	MR	1.174	0.20–0.30	57 : 7	3
Alpu01 × Yayla305	195	3	1.5	R	0.000	0.99	63 : 1	3
Alpu01 × Sultan95	245	12	4.9	R	0.764	0.30–0.50	15 : 1	2

MR – moderately resistant; R – resistant

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Table 4. The allelism table of the resistant × resistant hybrids

Variety No.		Variety No.																			
		1		2		3		4		5		6		7		8		9		10	
		R	S	R	S	R	S	R	S	R	S	R	S	R	S	R	S	R	S	R	S
1	OV	141	0	137	19	226	0	184	0	246	0	157	0	217	0	147	0	251	0	253	17
	χ^2 value			0.237																0.0009	
	P			0.50–0.70																0.95–0.99	
	SR			57R : 7S																60R : 4S	
2	OV	227	0	183	0	160	0	132	46	–	–	–	–	296	20	144	0	245	5		
	χ^2 value							1.259						0.003				0.315			
	P							0.20–0.30						0.95–0.99				0.50–0.70			
	SR							45R : 19S						60R : 4S				63R : 1S			
3	OV	86	0	188	0	213	6	158	9	145	0	112	0	393	0	162	0				
	χ^2 value					1.977		0.212													
	P					0.05–0.20		0.50–0.70													
	SR					63R : 1S		60R : 4S													
4	OV	78	0	158	0	160	0	149	12	–	–	–	–	264	0						
	χ^2 value							0.399													
	P							0.50–0.70													
	SR							60R : 4S													
5	OV	178	0	282	13	164	0	–	–	142	0	–	–								
	χ^2 value			1.712																	
	P			0.05–0.20																	
	SR			60R : 4S																	
6	OV	–	–	190	0	180	0	166	0	136	0										
	χ^2 value																				
	P																				
	SR																				
7	OV	152	46	–	–	212	57	–	–												
	χ^2 value	3.952				2.083															
	P	0.01–0.05				0.05–0.20															
	SR	45R : 19S				48R : 16S															
8	OV	–	–	117	22	–	–														
	χ^2 value			3.416																	
	P			0.05–0.20																	
	SR			57R : 7S																	
9	OV	210	0	125	0																
	χ^2 value																				
	P																				
	SR																				
10	OV	210	0																		
	χ^2 value																				
	P																				
	SR																				

1 – Altay; 2 – Ikizce; 3 – Karahan99; 4 – Kirac; 5 – Mufitbey; 6 – M732154; 7 – Nacibey; 8 – PI178383; 9 – Soyer02; 10 – Sultan95; OV – observed value; *P* – possibility rate; SR – segregation rate; R – resistant; S – susceptible

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Table 5. The genetic parameters and rates of the genetic parameters for the disease resistance

	Estimation ± SE
Genetic parameter	
E	0.009 ± 3.776
D	54.071 ± 11.329
F	75.935 ± 26.769
H_1	82.793 ± 26.043
H_2	59.523 ± 22.658
h^2	193.708 ± 15.195
D-H_1	–28.722 ± 22.346
Rates of the genetic parameters	
$H_1/D^{0.5}$	1.237
$H_2/4H_1$	0.180
KD/KR	3.624
K	3.254
BSH	0.988
NSH	0.887
r for Yr, Wr + Vr	0.945

E – expected environmental effect; D – additive genetic component; F – relative frequencies of dominant and recessive alleles; H_1 – dominance genetic variance; H_2 – adjusted dominance variance according to gene distributions; h^2 – complete dominance effect; D-H_1 – relative dominance to each other dominance and additive gene effects; $H_1/D^{0.5}$ – mean degree of dominance; $H_2/4H_1$ – value of the dominant and recessive alleles; KD/KR – dominant-recessive genes ratio; K – number of gene groups; BSH – broad sense heritability; NSH – narrow sense heritability; r – correlation coefficient; Yr – standardized mean for each parent; Wr –covariance; Vr – variance; SE – standard error

was smaller than the dominant genetic components (H_1 and H_2), and the D-H_1 value was negative. The value of the relative frequencies of the dominant and recessive alleles (F), which is the measure of the relative frequency of the dominant to recessive alleles in the parents, was positive. The mean degree of dominance $(H_1/D)^{0.5}$ for the rate of the disease was higher than 1. The value of the dominant and recessive alleles ($H_2/4H_{1)}$, which confirmed the asymmetrical distribution of the positive and negative alleles among the parents, deviated from 0.25 for the trait. The dominant-recessive genes ratio (KD/KR) was found to be 3.624. The number of gene groups (K) indicated that the trait was controlled by three gene pairs. The broad and narrow sense heritability was very high. When the covariance/variance (Wr/Vr) graph was examined, it was clearly seen that the y-axis of the regression line was very close to the origin point and in a positive direction. The order of the parents according to the distance from the origin along the regression line was as follows: Kirac66, Karahan99, Mufitbey, Yayla305, Sultan95, Altay2000, Ikizce, and Soyer02 (Figure 2).

DISCUSSION

In this study, the determined common bunt disease rates for 12 bread wheat varieties changed from 0 to 71.34% (Table 2), and only the Alpu01 variety was

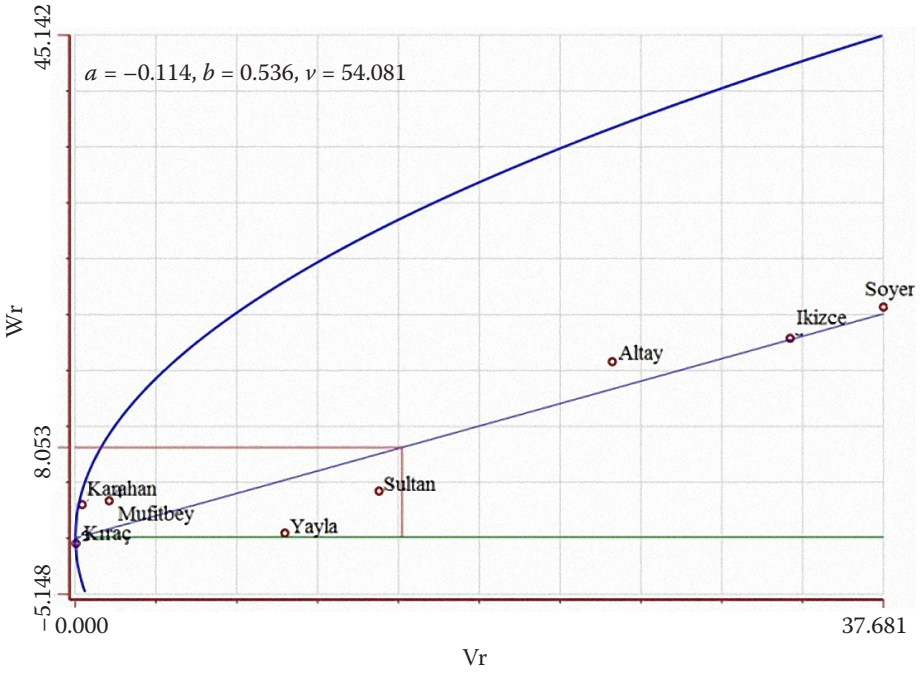

Figure 2. The covariance/variance (Wr/Vr) graph for the disease resistance of the parents

a – additive gene effect; b – non additive gene effect; v – variance

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found to be susceptible. When the results of other previous research were compared, we determined that the Kirac66 (Aktas & Tunali 1994; Akan *et al*. 2005; Tuncel & Boyraz 2006), Yayla305 (Aktas & Tunali 1994; Akan *et al*. 2005), and Karahan99 (Akan *et al*. 2005; Tuncel & Boyraz 2006) varieties showed a resistant reaction similarly to the results of these studies. In the hybrids made with Alpu01, which is a susceptible genotype, the medium or complete resistance was observed. While a double dominant epitasis was observed in the hybrids with two or three resistant genes, there was a complementary gene effect in the Alpu01 × Mufitbey hybrid. However, in the Mufitbey variety, the presence of two dominant genes with a double dominant epistatic effect may be accepted. Nevertheless, this issue should be examined in detail between the families and inter-family segregations in the F_3 generation.

The segregation was not observed in most of the hybrids between the resistant varieties (Table 4). This may be very normal when the pedigrees of the hybrids are examined since they have many common ancestors, and they have the same resistance genes. When the inheritance forms were examined in Table 4, five different segregation rates including 45R : 19S, 48R : 16S, 57R : 7S, 60R : 4S, and 63R : 1S were observed. It was understood that there are three resistance genes in all of the hybrids examined here, and the genes have epistatic effects on each other. Cota *et al*. (2009) determined that the disease rating was 18.4 to 63%. They identified the resistance gene by the χ^2 analysis result and with a 3 : 1 (resistant : susceptible) segregation ratio. In a similar study, Ulukan and Ozgen (1998) observed 3 : 1, 15 : 1, 57 : 7, and 55 : 9 segregation rates. The obtained inheritance forms are in parallel with the findings obtained from these studies.

According to the Jinks-Hayman diallel analyses, the dominant gene effects play an important role in the expression of the resistance to a disease due to the higher magnitude of the H_1 and H_2 components and the negative D-H_1 one. However, if a high narrow sense heritability degree was considered, an additive gene effect could be effective in this trait. The rate of a KD/KR higher than 1 and a positive F value indicated that the dominant alleles were more than recessive. According to the K value, this trait was controlled by three gene pairs. The correlation coefficient between the theoretical dominance order and the real values of the parents was positive and it indicated that the parents with low values had dominant alleles. The Wr/Vr graph reflected that the complete dominance was present in the examined trait. This finding did not match the ratio of $(H_1/D)^{0.5}$ which is greater than 1 and indicates the superior dominance. It is reported that these kinds of mismatches can be caused by the epistatic gene effects. Indeed, the fact that some genotypes were far away from the regression line reinforces the idea that epistatic gene effects may exist. It was understood that the genotypes of Kirac66, Karahan99 and Mufitbey, which were close to the starting point of the parabola, carried more dominant genes. The phenotypic observation values of these genotypes were low and the r value for Yr (standardized mean for each parent), Wr + Vr, which was positive, was consistent with this finding.

When these results are considered, it is seen that it is necessary to determine new resistance genes against the bunt disease in the case of a possible epidemic and to transfer these genes to the commonly planted varieties. As a result of this study, it is understood that if the resistance to disease is controlled by the three gene pairs, the higher resistance against the common bunt disease is provided. This study is a guideline for newly developed resistant varieties against the common bunt.

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