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Screening Bread Wheat Germplasm for Resistance to Take-all Disease (*Gaeumannomyces graminis* var. *tritici*) in Greenhouse Conditions

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ABSTRACT

Root and crown rot of wheat is caused by the fungus *Gaeumannomyces graminis* var. *tritici*. "Take-all" is an important disease affecting wheat, and its incidence has been reported in several provinces of Iran. To identify resistant cultivars, bread wheat germplasm should be evaluated. To evaluate bread wheat germplasm in response to Iranian isolate fungus (T-41) of *Gaeumannomyces graminis* var. *tritici*, 333 genotype of bread wheat, collected from different locations of Iran and other countries were evaluated to take-all in greenhouse conditions. Two experiments were conducted, the first with 89 and the second with 244 genotypes. The measured traits were amount of root and crown infection, disease intensity, wet and dry biomass, and height of shoots. Analysis of variance and means comparison for the parameters indicated that in the first experiment, two genotypes were resistant to the disease, and the rates of disease intensity in these genotypes were 0.13 and 0.06. In the second experiment, five completely resistant genotypes were identified with disease intensity ratings of '0'. The identified resistant genotypes screened from both experiments were re-evaluated, and the results were the same. Mean comparison between winter and spring types for dry weight and disease intensity showed that winter wheat is more resistant than spring type. The results of this research showed that there is resistance resource to take-all (T-41 isolate), in this germplasm. Since the experiment was conducted in greenhouse conditions, these genotypes should be tested against this disease in infected conditions at field.

Keywords: Evaluation, Root rot, Spring and winter wheat, T-41 isolate.

INTRODUCTION

Wheat (*Triticum aestivum* L.) is an important food source that provides 20% of the calories and 60-65% of the protein in the world's food supply. It is the staple food for about 40% of the world's population (Karov *et al*., 2008). From a total of 164.8 million hectares of land in Iran, more than 14.3 million hectares were planted in 1388. Statistics show that 12.34 million hectares were planted crops, of which 7.51 million hectares were planted with wheat (Radmehr, 2007-2008).

About 200 different diseases affecting wheat have been detected worldwide, and take-all disease, caused by *Gaeumannomyces graminis* var. *tritici*, is one of the most important (Karov *et al*., 2008). This fungus has a wide host range, especially in Poaceae family and over 350 species of plants and grasses are infected or

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parasitized (Fassihiani and Zare, 2010). Worldwide, this disease is highly intense in temperate climates in which wheat is cultivated also in the tropics (Nasraoui *et al*., 2007). Take-all disease has been reported in the Iranian provinces of Golestan, Mazandaran, West Azerbaijan, and Kurdistan (Sadravi, 2008), and effects of the disease have been seen in some fields in Zanjan Province (Joolideh *et al*., 2011). Spreading and contamination of roots occur with fungus mycelia which measure 6.3 micrometers in diameter, and the roots are brown to dark brown (McMillan, 2012). The fungal hyphae invade the cortex, penetrate the roots, and then destroy the vascular tissue (Freeman and Ward, 2004). Take-all is identified by the dark brown to black rotten roots or stolons in the early stages of seedling growth (Clarkson and Polly, 1981). Plants may be infected at any stage of growth, and infection worsens at 12-20°C (Huber and McCay-Buis, 1993). Heavily rotted roots are very friable, and much of the root system remains in the soil when plants are pulled up. When the leaves are pulled away from the stem, the shiny black discoloration of the basal stem is highly diagnostic for take-all. Under a microscope, dark brown "runner hyphae" (or necrotrophic growth) and mycelial mats on roots or stolon surfaces are easily seen and are also helpful in diagnosing this root disease (Karov *et al*., 2008). Other symptoms of take-all are stunting, reduced tillering and incomplete seed maturation (Liatukas *et al*., 2010). Various methods for controlling and managing the disease, such as fallow crops, crop rotation, delaying planting, use of nitrogen fertilizers in favor of ammonium, planting in acidic soil, planting non-host plants of take-all disease of wheat in the rotation, somewhat reduces the disease (Asher and Shipton, 1981). The use of any of these methods has disadvantages; for example, delaying winter wheat sowing leads to a reduction in crop yield (Darwinkle *et al*., 1977). A natural control to take-all can build up in the soil in a monoculture after several years, and fields automatically show "take-all decline". Therefore over the long term, the continuous production of hosts can show suppressiveness. Biological control research using seed treatments with suppressive bacteria on small grains or applying biological agents to established plants has shown promise (Raaijmakers *et al*., 1997). One most important method for controlling this disease is the use of resistant cultivars. Some small grain and bent grass cultivars have a slight level of resistance to take-all, but no highly-resistant cultivars are available. Some level of resistance has been identified in wild grass species that may be transferable into cultivated species.

Genetic diversity is necessary for the production of take-all disease-resistant wheat varieties. Reports have shown some difference, albeit negligible, among wheat cultivars for resistance to take-all. Bread wheat is one of the most sensitive to this disease (Scott, 1981; Rothrock, 1988). In one study, a number of small grain cereals, such as barley, wheat, triticale, rye, and oats, were evaluated with respect to take-all disease. Results showed that wheat had the greatest sensitivity, barley and triticale had moderate sensitivity, and oat proved to be resistant (Zare and Fassihiani, 2008). Another study evaluated the responses of 244 bread wheat genotypes, 56 six-row barley genotypes, 50 lines of naked and 36 two-row barley genotypes to take-all. Results showed that barley and wheat were resistant to take-all disease in different manners. Wheat genotypes were the most sensitive, and six-row and two-row barley genotypes were more tolerant (Oyanagi *et al*., 1990). Low levels of take-all disease resistance have been reported in some isolates of *Aegilops tauschii*, rye (*Secale cereal* L.), and oat (*Avena*s pp.) (Linde-Laursen *et al*., 1973; Eastwood *et al*., 1993).

Some durum wheat varieties have been determined to be resistant to take-all disease. An amphidiploid between durum and *Haynoldia villosa* (2n= 42, AABBVV) (*TH3*) has been identified as a resistant-to-take-all genotype. One derivative of TH3

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(HW918-5) was more resistant, which indicated that the resistance gene was located on chromosome 3V of *H. villosa*; this gene was studied by molecular analysis based on PCR (Da-hui *et al*., 2007).

Success in breeding for resistance to disease depends on the nature of the pathogens and pathogenic diversity in the population, availability of diversity, mechanisms of genetic resistance, screening methods, and the environment in which the selection is made (Singh and Rajaram, 1998). Genetical resistance is the most economical way to control take-all disease. No significant levels of resistance or tolerance to this disease have been identified in wheat (Eastwood *et al*., 1993; Kim *et al*., 2003). There is evidence of partial tolerance to take-all in older, hard, red winter wheat varieties compared with the newer, soft, white winter wheat varieties (Huber and McCay-Buis, 1993). In the past thirty years, attempts to identify sources of resistance to take-all in wheat germplasm have been done. Resistance genes for take-all disease exist in bread wheat, rye, and some wild ancestors. Crosses made in order to transfer rye resistance into bread wheat were not successful (Marshal, 2014: Un–published).

It seems that full screening of the primary gene pool of wheat to find quite resistant accessions and quite sensitive genotypes has not yet been done, particularly in Iran. If resistance genes in the primary gene pool of wheat are identified, their use and transfer would be easier in wheat breeding programs. To this end, the current study was designed and implemented.

MATERIALS AND METHODS

Fungus Resource

In a research several isolates were isolated from infected samples to take-all disease from different provinces of Iran, including, Esfahan, Markazi, Mazandaran, Tehran, East Azerbaijan, Western Azerbaijan, Ardabil, Qazvin and Golestan. Pathogenicity tests indicated that all isolates were pathogenic on wheat and one of them 'T-41' which was collected from Mazandaran has strong Pathogenicity (Sadeghi *et al.,* 2012; Sadeghi *et al*, 2012). Therefore T-41 isolate was selected for our research (This isolate was obtained from mycological collection of Vali-e-Asr University of Rafsanjan). Figure 1, shows middle simple hyphopodia of *Ggt* that have formed aggregation.

Genetic Resources

Genetic resource was 333 genotype of bread wheat, collected and received from different locations of Iran and other countries. At first these genotypes were planted in one line at field of Vali-e-Asr University and a single plant selected from each line and their seeds were used in this screening for resistance and susceptibility to take-all in greenhouse. Genotypes accession numbers are shown in Table 8 (in the results and discussion section). These genotypes were maintained in the germplasm collections of Vali-e-Asr University and are available at any time to investigators for use or research.

Purification and Storage Fungus

The selective medium for fungal culturing was Potato Dextrose Agar (PDA) containing

Figure 1. Middle simple hyphopodia of *Ggt* that have formed aggregation.

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streptomycin (0.03 gram in 1,000 cc PDA). The fungus was purified once every 20 days; the border was growing in a Petri-dish, and the fully developed fungus was stored in the refrigerator at 4°C.

Fungus Inoculum Preparation

Because of a high colonization rate and the uniformity of propagules, millet was chosen for the prepared fungus inoculums**.** A mixture of 100 grams of cooked millet seed and 100 grams of wet sand was poured into a flask and autoclaved twice at 120°C for 20 minutes.

For fungus propagation, a few circles of mycelia with one centimeter in diameter from the edge of the growing colonies were inoculated into each of the flasks and incubated at 20-25°C for 15 days. The flasks were then removed and incubated for 15 days at a temperature of 20-28°C in a laboratory environment under natural and fluorescent light. The flasks were shaken several times for aeration and were avoided of being shot. They were then refrigerated until time of use.

Greenhouse Experiment

A suitable sieved soil (EC= 1.2-2 dS, pH= 7.5-8), was autoclaved at 121°C for one hour. Seeds were disinfected in a solution of 1% sodium hypochlorite for one minute and then planted in pots containing 800 grams of soil in the greenhouse. Experiments were conducted in a completely randomized design with 3 replications in two steps. In the first and second experiments, 89 and 244 genotypes were evaluated respectively. Plants were inoculated using a slightly modified Thomashow and Weller method (1988). Inoculation was performed 10 days after planting, when the seedlings were about 20 cm in size. Two grams of inoculum were dumped close to the crown of the plant and covered with sand (Figure 2) and one replicate was considered as control. Irrigation was done as required. Greenhouse temperatures ranged from 20-25°C. Six weeks after inoculation, the percentage of the crown that was blackened, its wet and dry weight (biomass), and height were measured and recorded. Contamination levels based on the percentage of necrosis in the roots and crowns were scored based on 0 as follows (Ownley *et al*., 2003):

0= Roots and crowns without necrotic spots;

1= Roots with one or more necrotic spots and crowns without symptoms;

2= Roots with continuous necrotic spots (more than 25% and less than 50% necrosis of roots) and crowns without symptoms;

3= More than 50% necrosis of the roots and blackened crowns;

4= Roots approximately black with 75% blackened crowns;

5= Roots and crowns black and drying.

Disease Intensity (DI) was calculated according to the following Formula (1):

$$\%DI = \frac{\text{Sum of scores in each pot}}{5 \times \text{Number of plant}} \times 100 \quad (1)$$

Statistical Analysis

ANOVA and logistic regression analyses were performed using MINITAB 14 statistical software. PLSD and mean comparison test was done by SAS statistical software.

Figure 2. Inoculated on wheat seedlings.

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RESULTS AND DISCUSSION

First Experiment

The analysis of variance on disease intensity and fresh dry weight of 89 genotypes showed that there is a significant difference between genotypes for disease intensity (Table 1). Genotypes 1879 and 1530 had the lowest severity rates (0.13 and 0.06) with no blackening on their crowns and a necrotic spot observed only on the roots. Contrarily, genotypes found in front of the fungus *Ggt* had become dehydrated (Figures 3-A and -B).

The PLSD mean comparison test was performed for disease intensity, and genotypes were sorted into 19 groups (results not shown). Analysis of variance showed a significant difference among genotype dry and fresh weights (Table 2).

Table 1. Analysis of variance for disease intensity in the first experiment.

SOV	df	MS	F	pr
Genotype	88	9.432	30.23***	0.0001
r(t)	178	0.591	1.89	0.0001
Sampling Error	266	0.312		
Total	532			

*** Significant at 0.001 level of probability.

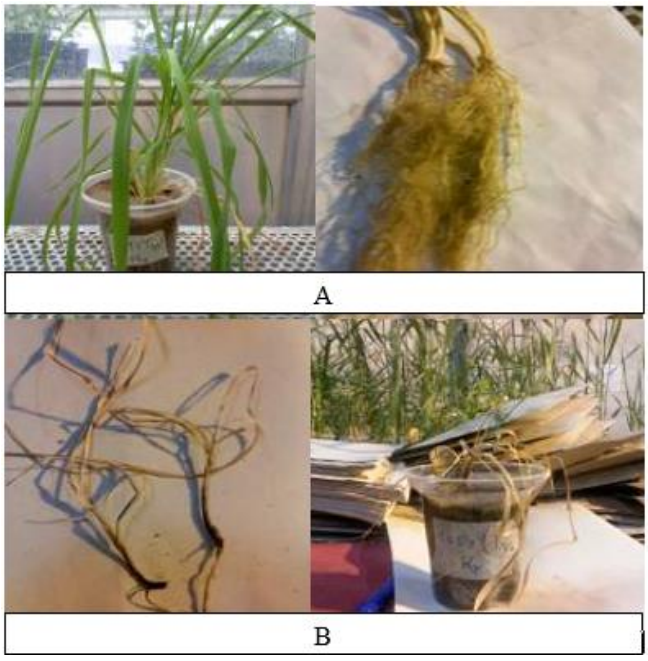

Figure 3. Infected plant with high resistance (A) and sensitive (B).

Table 2. Analysis of variance for fresh and dry weight in the first experiment.

SOV	df	MS	
		FW [a]	DW [b]
Genotype	88	19.51***	0.889***
Error	178	2.51	0.284
Total	266		

[a] Fresh Weight, [b] Dry Weight. *** Significant at 0.001 level of probability.

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Second Experiment

Analysis of variance showed significant differences in disease intensity among the genotypes (Table 3). Genotypes 1560, 729, 8031, 136, and 1637 scored zero in disease intensity. In 2013, resistance of 108 cultivars or lines of wheat (Zhengmai 3596, Zhaashi 2010-06, Zhongmai 9023-9, Yonang 211, Zhongmai 2, Yumai 49, Xinnong 19 and so on) in Henan Province to take all were evaluated under controlled conditions. The result showed that only one cultivar (Xinnong 19) was moderately resistant, also plant height, root dry weight and stem dry weight were positively correlated with the degree of resistance (Fei *et al.,* 2013). Furthermore, in Glasshouse screenings of 1,243 wheat varieties, there were significant differences in susceptibility to *Gaeumannomyces graminis* var. *tritici*. The best varieties were tested repeatedly against different isolates of the pathogen. Some were less susceptible; but none showed real resistance and in Germany, the screening of over 2000 wheat species and varieties at greenhouse conditions, found that all lines showed highly susceptible reaction except a few of *Triticum monococcum* lines showed a low susceptible (McMillan, 2012). An experiment were performed on *Pythium arrhenomanes* (root rot disease), seven wheat varieties were compared with TAM-101 variety against *P. arrhenomanes* at greenhouse, based on growth parameters, four days after inoculation. Among all varieties tested, Kenya-CI 12880 was the only one which consistently performed as well, or better than TAM-101 (Mojdehi and Singleton, 2000). Genotypes were sorted into 26 groups by PLSD comparison (results not shown). In some genotypes (33 genotypes), fresh and dry weight and height of the infected treatment were greater than that of the control plants (Table 4). At experiment it was found that the stimulation of root growth by disease may also explain higher plant weights of inoculated plants of Winalta - *Aegilops squarrosa* 6D (Winalta - *Aegilops squarrosa* chromosome substitution line) relative to the uninoculated check during the early stages of disease development (Conner *et al.,* 1988). Growth type of genotypes (spring and winter) was determined and plant height was measured. Some genotypes (182 genotypes) headed in the greenhouse, are considered as spring type; and the others remained in rosette which was due to their requirements to vernalization, these genotypes are considered as winter type. Therefore, separate analyses were conducted on the genotypes of spring and winter types. The results showed a large variation in the studied traits within and among types (Tables 5 and 6).

Averages of dry weight and disease intensity for winter and spring genotypes were compared. Spring types had higher averages of disease intensity as well as lower mean dry weights than winter types (Table 7).

These results were generally consistent with those of studies in the USA (Huber and McCay Buis, 1993). Spearman correlation coefficient between dry weight and the disease score ($rs= -0.542^{**}$) showed that low levels of disease score significantly associated with high levels of dry weight.

Genotypes were classified in total by disease Scores (Sc) in the first and second experiments as follows: $Sc= 0$ (highly resistant), $0< Sc\leq 1$ (resistant), $1< Sc\leq 2$ (moderately resistant), $2< Sc\leq 3$ (moderately sensitive), $3< Sc\leq 4$ (sensitive) and $4< Sc\leq 5$ (highly sensitive) (Table 8).

Logistic Analysis

With regard to disease score as the ordinal dependent variable in six levels (0, 1, 2, 3, 4, and 5), growth habits in two levels (spring and winter) as a factor, and dry weight as a quantitative trait, logistic analyses were performed. Statistic $G= 174.84$ with $P= 0.000$ indicated that all coefficients are not zero; the test for Pearson goodness-of-fit ($P= 0.223$) and deviance ($P= 1.000$) indicated

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Table 3. Analysis of variance for disease intensity and dry weight in the second experiment.

SOV	DI [a]		SOV	DW [b]	
	df	MS		df	MS
Genotype	243	0.501***	Genotype	243	0.587***
Experimental Error	244	0.015	Experimental Error	244	0.057
Sampling Error	972	0.009	Total	587	
Total	1459				

[a] Disease Index, [b] There is not sampling for this trait. *** Significant at 0.001 level of probability.

Table 4. Comparison between infected and control treatment for fresh and dry weight by *T*-test.

Traits	Treatment	Number	Mean	*T*-test
Fresh weight	Infected	33	5.05a	2.14*
	Control	33	4.02b	
Dry weight	Infected	33	2.182a	5.85***
	Control	33	1.842b	

*** Significant at 0.001 level of probability.

Table 5. Analysis of variance for spring and winter variates separately in response to disease.

SOV	Winter		Spring	
	df	MS	df	MS
Genotype	48	0.1936***	194	0.4297**
r(t)	499	0.00936	195	0.0162
Sampling error	195	0.00779	775	0.0089
Total	292		1164	

*** Significant at 0.001 level of probability.

Table 6. Analysis of variance for height of spring genotype s in the second experiment.

SOV	df	MS
Genotype	176	102.556***
r(t)	174	32.038
Sampling error	649	17.556
Total	999	

*** Significant at 0.001 level of probability.

Table 7. Comparison between spring and winter types for disease intensity and dry weight by *T*-test.

Traits	Treatment	Number	Mean	*T*-test
Dry Weight	Winter	62	1.672a	10.3***
	Spring	182	0.987b	
Disease intensity	Winter	62	0.568a	12.17***
	Spring	182	0.205b	

*** Significant at 0.001 level of probability.

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Table 8. Number of genotypes and genotypes classification based on means of disease score.

Scoring range	Collection number of genotypes
$Sc = 0$	8031, 729, 136, 1637, 1560
$0 < Sc \leq 1$	1879, 1530, 1795, 2104, 23, 71, 1580, 21, 726, 757, 2113; 2058, 2068, 2093, 150, 2125, 1800, 2024, 2109, 2097, 2095, 113, 2013, 2105, 1870, 483, 9040, 2054, 1644, 494, 102, 148, 1611, 630, 657, 9039, 105, 1402, 1642, 90, 1454, 1867, 142, 922, 620, 666, 117, 20, 2111, 1878, 2110, 1640, 2120, 9016, 612, 1508, 1437, 1902, 2071, 449, 1561, 54
$1 < Sc \leq 2$	92, 153, 569, 62, 645, 2180, 66, 2078, 2029, 2134, 647, 2027, 2072, 122, 1596, 9052, 1495, 1420, 693, 1497, 491, 1419, 604, 130, 8034, 447, 593, 171, 2137, 1436, 1533, 1458, 1452, 9003, 1899, 1801, 1901, 1493, 565, 758, 61, 888, 443, 414, 736, 738, 137, 1170, 583, 203, 911, 549, 202, 971, 892, 106, 641, 516, 410, 539, 2039, 1398, 562
$2 < Sc \leq 3$	76, 610, 113, 532, 155, 1424, 1592, 3787, 519, 972, 407, 594, 638, 1403, 30, 2053, 748, 717, 734, 3791, 1882, 140, 3785, 170, 552, 3801, 600, 1442, 1529, 9010, 2059, 9004, 893, 427, 1412, 749, 691, 2043, 898, 581, 1872, 415, 905, 1466, 517, 1897, 557, 3798, 783, 1447, 1576, 1887, 1524, 722, 1505, 623, 2133, 1507, 477, 667, 571, 943, 1888, 2101, 663, 500, 750, 1444, 553, 699, 457, 592, 665, 3792, 606, 556, 1389, 1488, 730, 596, 589, 1400, 3800, 2080, 416, 412, 1621, 731, 1891, 1470, 429
$3 < Sc \leq 4$	189, 114, 1515, 157, 167, 3794, 564, 132, 1638, 614, 536, 35, 681, 190, 161, 605, 622, 1542, 628, 912, 211, 625, 843, 3789, 1577, 835, 511, 2060, 528, 446, 2038, 1573, 935, 440, 1477, 723, 1866, 509, 1448, 1388
$4 < Sc \leq 5$	56, 710, 173, 713, 14, 578, 2019, 531, 2016, 177, 164, 1469, 1107, 608, 1410,1546, 1526, 580, 168, 181, 1554, 50, 3786, 716, 739, 1520, 699, 165, 585, 640, 9007, 534, 1472, 1532, 9035, 664, 704, 9019, 403, 554, 1874, 2062, 629, 1479, 707, 1396, 703, 747, 656, 609, 184, 454, 439, 162, 119, 576, 413, 501, 745, 9013, 659, 507, 594, 2061, 1438, 1883, 637, 199, 169, 1511, 425, 2042

that the model is in complete agreement with the data (Table 9). The logistic regression model showed a good correlation between disease intensity and type of growth. The regression coefficients (-3.57) for type of growth (w) and 1.36 for dry weight (dw) with $P = 0.000$ indicated that the replacement of winter type instead of spring type reduces the disease score (-3.57), and dry weight increases with replacing a winter type instead of a spring wheat. The results of logistic regressions are in complete agreement with ANOVA and mean comparisons between spring and winter types.

In our study, it was determined that winter wheat genotypes are more resistant to the disease. Also, Huber and McCay Buis (1993) reported that hard winter wheat has more resistance to the take-all and khanahmadi *et al.* (2016) reported that winter varieties Zarrin, Alvand and Pishtaz have less injury against this disease. Also 324 accessions (winter wheat) including standard cultivars were tested during vegetation seasons. Varieties Flair and Dream were the most resistant lines (Liatukas *et al.*, 2010). One explanation for differences in resistance of varieties is the various abilities of winter wheat genotypes in the use of manganese. Manganese might increase the biosynthesis of defence-related phenolic and lignin (a major part in the plants defence against pathogenes) and thus resistance to Take-all (Wilhelm *et al.*, 1987; Rengel *et al.*, 1993). Increasing Mn levels in solution culture have an effect on both phenol and lignin levels in wheat seedlings. Mn sufficiency reduces *Ggt* infection through strong constitutive plant defence mechanisms (Pedler, 1994).

Furthermore, our research showed the production of extra roots when attacked by *Ggt* or a high intrinsic rate of root production is suggested as partially offsetting the loss of root function in already infected roots so that the plant can better tolerate infection. Others have speculated that differences in resistance between cultivars may only reflect differences in the

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Table 9. Logistic regression table.[a]

Predictor	Coef	*SE* Coef	Z	P			
Const (1)	-1.87519	0.53754	-3.49	0.000			
Const (2)	-0.40829	0.54489	-0.75	0.454			
Const (3)	1.33794	0.56952	2.35	0.019			
Const (4)	2.87490	0.57097	5.04	0.000			
Const (5)	3.57170	0.57679	6.19	0.000		Confidence interval	
Type					Odds ratio	Lower	Upper
w	-3.57111	0.42577	-8.39	0.000	0.03	0.01	0.06
dw	1.36290	0.30239	4.51	0.000	3.91	2.16	7.07

[a] Type: Winter, spring; Log-likelihood= -335.291, Test that all slopes are zero: *G*= 174.847, *DF*= 2, *P*-value= 0.000.

Goodness of fit tests

Method	Chi-square	df	P
Pearson	347.177	328	0.223
Deviance	217.002	328	1

ability of different cultivars to replace damaged roots (Deacon and Henry, 1978; du Plessis and Nortje, 1951; Scott, 1981; Colbach *et al.*, 1997).

CONCLUSIONS

Wheat resistance against take-all disease is the inability of a disease agent to penetrate the root, thus preventing pathogens from developing on crowns. From 333 evaluated genotypes, 72 genotypes showed high sensitivity to the disease, 40 were sensitive, 91 were moderately sensitive, 63 were moderately resistant, 62 genotypes were resistant, and 5 genotypes were completely resistant (based on the average means of scores from 6 plants in each genotype). Genotypes 1560, 1637, 136, 8031, and 729 were identified as being completely resistant and showed no infection. These genotypes were retested and identical results were obtained. It was also observed that the 5 genotypes 1526, 164, 2019, 1546, and 1107 were highly sensitive to take-all disease, such that the plants became dehydrated. Winter wheat was generally more resistant than the spring accession. Results of the ordinal logistic regression analysis had a good fit with the data. The sensitive and resistant samples screened in this experiment are useful material for subsequent genetic studies of take-all disease.

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غربال‌سازی ژرم‌پلاسم گندم نان برای مقاومت به بیماری پاخوره (*Gaeumannomyces graminis* var. *tritici*) در شرایط گلخانه

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چکیده

پوسیدگی ریشه و طوقه گندم (پاخوره) با عامل *Gaeumannomyces graminis* var. *tritici* از مهم ترین بیماری‌هایی است که به گندم حمله می‌کند و تا به حال از چندین استان ایران گزارش شده است. به منظور شناسایی ارقام مقاوم، ژرم‌پلاسم گندم نان باید ارزیابی گردد. به منظور ارزیابی ژرم‌پلاسم گندم نان در پاسخ به جدایه ایرانی قارچ *Gaeumannomyces graminis* var. *tritici* (T-41)، ۳۳۳ ژنوتیپ گندم نان جمع‌آوری شده از ایران و دیگر کشورها در مقابل بیماری پاخوره در شرایط گلخانه ارزیابی شدند. به این منظور دو آزمایش انجام شد. در آزمایش اول ۸۹ و در آزمایش دوم ۲۴۴ ژنوتیپ مورد بررسی قرار گرفتند. میزان آلودگی ریشه و طوقه، شدت بیماری، وزن تر و خشک گیاه و

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ارتفاع اندام هوایی از صفات اندازه‌گیری شده بودند. تجزیه واریانس و مقایسه میانگین برای صفات آزمایش اول نشان داد که دو ژنوتیپ با شدت بیماری ۰/۱۳ و ۰/۰۶ از ژنوتیپ‌های مقاوم به این بیماری هستند. در آزمایش دوم پنج ژنوتیپ کاملا مقاوم شناسایی شدند که شدت بیماری برای آنها صفر بود. ژنوتیپ های مقاوم شناسایی شده از هر دو آزمایش مجدد ارزیابی شدند و نتایج همان بود. مقایسه میانگین بین تیپ‌های بهاره و پاییزه بر اساس صفات وزن خشک و شدت بیماری نشان داد که گندم پاییزه از گندم بهاره مقاوم‌تر است. نتایج این تحقیق نشان داد که منابع مقاومت به ایزوله T-41 در این ژرم پلاسم وجود دارد. از آنجایی که آزمایش در شرایط گلخانه انجام گرفت، لازم است این ژنوتیپ‌ها در شرایط آلوده در مزرعه انجام شود.

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