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Genetic analysis of resistance to stripe rust in cross of commercial bread wheat cv. Aflak × Avocet

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ABSTRACT

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Stripe rust caused by *Puccinia striiformis* f. sp. *tritici* (*Pst*) is one of the most devastating wheat diseases worldwide. Identification of virulence factors of the pathogen as well as using resistant cultivars are effective approaches in controlling wheat strip rust. Knowledge of genetic basis of resistance to stripe rust in commercial wheat cultivars is an important objective to ensure that diverse resistance genes are deployed in breeding programs. Five bread wheat commercial cultivars were evaluated for stripe rust using 40 *Pst* races that were collected from different locations in Iran. Among them cultivar Aflak showed seedling resistance to all races and the other cultivars displayed field resistance at the adult plant stage. To study the heritability and genetic basis of resistance to stripe rust in bread wheat cv. Aflak, it was crossed with susceptible cultivar Avocet's' and F_1, F_2, BC_1 and BC_2 generations were developed. The generations along with their parents were planted in Randomized Complete Block Design (RCBD) with three replications under greenhouse conditions. The seedlings were inoculated using the race 230E158A+, *Yr27+* urediniospores. The resistance components including infection type, latent period, pustule size and pustule density were recorded on single plants of each generation. The results of the weighted analysis of variance showed that there were significant ($p \leq 0.01$) differences among generations for each of the four traits. Generations mean analysis showed that in addition to additive and dominance effects, epistasis, particularly the additive × dominance epistasis also played a significant role in controlling resistance to stripe rust. For all traits, high broad-sense heritability was observed. Moderate to high narrow-sense heritability was estimated for resistance components

Keywords: resistance components, epistasis, generations mean analysis, heritability

INTRODUCTION

Wheat, as the most important crop, is the leading source of human food in many developing countries. Wheat rusts are the major biotic constraint to sustainable wheat production worldwide **.**Rusts may occur wherever wheat is grown (Ali *et al*., 2017).

Stripe rust or yellow rust caused by *Puccinia striiformis* f. sp *tritici* (*Pst*) is the most economically important wheat's fungal disease, especially in cold and temperate areas (Chen *et al*., 2014). Currently, 88% of the world's wheat genotypes are susceptible to stripe rust, leading to global losses of over 5 million tons of wheat with an estimated market value of $USD 1 billion annually (Beddow *et al*., 2015).

Stripe rust has numerous physiological races (Wellings, 2011). The sexual cycle of this fungus occurs on barberry (*Berberis spp*) plant. Sexual recombination plays a very important role in pathogenic diversity for new virulence (Jin *et al*., 2010; Hovmøller *et al*., 2016). The emergence and spread of new races of stripe rust (through high pathogenicity and variability, breakdown of important resistance genes) have emerged as a great concern for global wheat community with estimated yield losses from 30-70% (Chen, 2005). The emergence of new races can severe epidemics within a few years (Sharma *et al*., 2016).

Under favorable conditions (susceptible varieties and favorable environmental conditions), wheat rusts may occur as

epidemic in different parts of the world every few years, which may cause severe losses to wheat production (Singh *et al*., 2004). Recent severe stripe rust epidemics occurred in the USA in 2003, South Africa in late 1990s, China in 2002, Central Asia in late 1990s and again in 2009 and 2010, and Australia in 2003 (Chen, 2005; Hovmøller *et al*., 2008; Milus *et al*., 2009; Wellings, 2011). Additionally, several new stripe rust races have been identified in Europe in recent years, overcoming many resistance genes in European wheat varieties (Hubbard *et al*., 2015). The latest intermediate epidemics in Iran occurred in 2010 and to a lesser extent in 2013.

Breeding for the disease resistance in wheat cultivars has the greatest effect and management strategies in reducing the disease losses (Singh *et al*., 2016). The use of resistant varieties is preferred to other control and management measures for this disease due to diversity in genetic resistance, ease of deployment in commercial cultivars, cost-effectiveness and environmental safety (Steuernagel *et al*., 2016). Currently, more than 150 resistance genes have been confirmed for three brown rust, yellow rust, and stem rust in wheat or grasses related to wheat. So far, 84 *Yr* (Yellow rust) genes have been designated in wheat out of which 36 genes have temporary designations. Additionally, 327 Yr and 249 LR QTLs have also been identified as conferring resistance to stripe rust and leaf rust in bread and durum wheats (Gebrewahid *et al*., 2020).

Two types of resistance have been identified in rusts; race-specific and non-race-specific resistance. The race-specific type of resistance is controlled by a single gene and is usually effective in the seedling stage and the majority of the genes are short-lived as the races acquire virulence in a short period, leading to boom and bust cycles (Singh *et al*., 2004). Most of the known resistance genes considered as race-specific resistance (Singh *et al*., 2011). Virulence to *Yr9* and more recently to *Yr27* resistance genes in the most important commercial cultivars such as ‘Chamran’ and ‘Shiroudi’ in the Fars province of Iran, are examples of ineffectiveness of the race-specific resistance genes in Iran (Afshari *et al*., 2003; Afshari *et al*., 2014). In resistance improvement programs, if selection favors a specific gene, that gene will be fixed in the improved materials, and as a result, the varieties will become susceptible to a specific race of the pathogen, which has pathogenicity to that gene (Singh *et al*., 2004). Non-race-specific resistance that is effective against all races of the pathogen is often controlled by a few minor genes, especially in adult plant stage. This type of resistance has different characteristics such as slow rusting, partial resistance, with minor to moderate additive impacts (Singh *et al*., 2011).

To study the inheritance, because resistance components such as infection type, latent period, pustule size, and pustule density manifest themselves quantitatively, and individual genetic effects are so rare, they cannot be identified by Mendelian analysis. For this reason, traits of these genes should be studied through genetic analysis methods, including generations mean and variance analysis (Falconer and Mackay, 1996; Hill *et al*., 1998). Information resulting from such analyses has an important role in the selection of breeding scheme and selection method for resistance to wheat rust. Many researches have been interested to understand the inheritance of resistance components. Rutkoski *et al.* (2011) reported the segregation of a single recessive gene and Mendelian inheritance in F2, F3 generations. Inheritance of resistance to rust can be qualitative or quantitative. In their research, they demonstrated the recessive inheritance of the trait. Quantitative resistance is more durable. However, it’s more difficult to evaluate because it is expressed only in adult plant stage.

To study the inheritance of resistance to yellow rust as well as to determine the genetic components of resistance, Khodarahmi *et al.* (2007) analyzed F_1, F_2, BC_1 and BC_2 generations, resulting from a cross between resistant variety MV17 and susceptible variety Bolani, along with the parents by two races of the pathogen of yellow rust in two separate experiments. Based on the results of

generation mean analysis employing the weighted scale test, additive effect, dominance effect, and epistasis (especially additive × dominance and dominance ×dominance interactions) effects was observed, and significant reduction of infection type and increase in the duration of latent period. Estimation of dominance was almost equal to the unit for both traits, which indicates the complete dominance of resistance. Estimation of heritability for resistance components varied from average to high. Number of genes controlling resistance varied from 1 to 3.

Khodarahmi *et al.* (2009) studied the genetic parameters of several genes as well as the performance of wheat rust resistance genes. In this study, F_1, F_2, F_3, BC_1, BC_1S_1, BC_2S_1 generations were devloped from a cross between MV17 (resistant) and Bolani (susceptible) were planted, and three traits including FCI, AUDPC and rAUDPC were evaluated by generation mean analysis. The dominance degree showed complete dominance of the three traits. Broad-sense heritability was high for resistance components, however, they reported average heritability for narrow-sense heritability.

Spring bread wheat cv. Aflak has shown complete seedling resistance to most races of yellow rust in different regions in Iran. A heritability study of resistance components for this cultivar seems to be necessary. The objectives of this research were to study heritability of resistance to wheat yellow rust in cv. Aflak using generations mean analysis under greenhouse conditions.

MATERIALS AND METHODS

This research was carried out in yellow rust greenhouses of the cereal pathology unit of cereal research department Seed and Plant improvement Institute (SPII), Karaj, Iran. A highly virulent isolate of stripe rust collected from the Sari region (Mazandaran province, Iran) was used. The race analysis of Sari isolate was also conducted employing the method proposed by Johnson *et al.* (1972). For race identification, 45 standard set of differential wheat genotypes and isogenic lines including lines derived from backcross of Avocet's' variety with *YrA, YrSP, Yr32, Yr27, Yr26, Yr24, Yr18, Yr17, Yr15, Yr10, Yr9, Yr7, Yr6, Yr5* and *Yr1* genes were used.

After purification and multiplication of isolate, the genotypes of differential set were inoculated in the greenhouse. Seedlings were inoculated at 12 stage in Zadoks (Zadoks *et al*., 1974) scale. A mixture of urediniospore and talcum powder in 4:1 ratio was used, by employing the powder injection method.

Inoculated seedlings were maintained under full dark conditions at 11 ˚C at saturated humidity (above 95%) for 24 hours. Then, the pots moved to greenhouse with 60-70% humidity, 15 ˚C temperature and light of 16000 Lux produced by a combination of lights of florescent and sodium lamps for 16 hours per day. Pots were held in these conditions for 20 days. Infection type of standard and segregating generations were recorded over latent period based on 0-9 scale, following Mc Neal *et al.* (1971). Scores 0-6 indicated resistance, whereas scores 7-9 indicated susceptibility.

The wheat cultivar Aflak with pedigree: HD160/5/Tob/Cno/23854/3/Nai60//Tit/-Son64/4/LR/Son64, in previous experiments, showed resistance to strip rust races from different regions in Iran. Therefore, F_1, F_2, BC_1 and BC_2 generations developed from cross between cv. Aflak (resistant) with variety Avocet's' (susceptible) along with two parents (P_1, P_2) were evaluated under greenhouse conditions. Fifteen seeds of P_1, P_2 and F_1 as well as 240, 60 and 60 plants of F_2, BC_1 and BC_2, respectively, were evaluated in each replication.

The scores recorded on 8^{th} day following the inoculation to measure the duration of the latent period (LP, the number of days from inoculation to the appearance of the first pustule). Seedling leaves on which pustules developed were marked with a plastic ring. Different colored rings employed to mark successive days. Then, the number of the days from inoculation to the appearance of first pustule on the first and second leaf appeared on individual plants, was recorded in replications. This was continued until 20^{th} day after inoculation. On the 20^{th} day, the infection

type was recorded based on 0-9 scale following Mc Neal *et al.* (1971). Where no pustule was formed, number 20 was recorded for the latent period.

To measure the pustule size and pustule density (number of pustules in one square millimeter), samples of infected leaves with rust were prepared. Samples were placed in lacto phenol solution, which fixes pustules on the leaf and discolors the leaf. For samples taken from each plot in replications, length and width of elliptical pustules were measured using a microscope equipped with an ocular micrometer and lens No. 10×4. The size of the pustule for each treatment in each replication was calculated based on the average size of a single pustule. The pustule size was calculatedfollowing Lee and Shaner 1985.

Pustule size (cm^2) = Major axis (length) × minor axis (width) × π/4

To measure the pustule density, a microscope was employed. The number of pustules on each cut sample was counted by counting the number of pustules in 10 microscopic fields, then the number of pustules in one square millimeter was obtained.

Generations mean analysis was performed using inverse variance for each generation as the weight of each generation following Mather and Jinks (1982).

$$Y = m + a[d] + \beta[h] + a^2[i] + 2a\beta[j] + \beta^2[l]$$

where, Y = mean of a generation, m = mean of total generations, d = sum of additive effects, h = sum of dominance effects, i = sum of reciprocal effect among additive effects, j = sum of reciprocal effect among additive and dominance effects and l = sum of reciprocal effect among dominant effects. α, β, α^2, 2αβ, β^2 are coefficients for the model's parameters.

The standard method includes the estimation of genetic effects by comparing the mean of observed and expected generations that is estimated based on the six parameters (Mather and Jinks, 1982). Using the least weighted squares as the number of the variances of individuals that were different in each generation, six parameters, or less than six, were estimated. In this study, all seven generations were tested with scale tests' two, three, four, five, and six parameters, and the best model was selected. The best model for each traits was identified based on the $\chi 2$ test (Mather and Jinks, 1982). Employing MATLAB, the matrices were inversed and multiplied. Variance Components were estimated Mather and Jinks (1971).

$$E = (\hat{\sigma}^2_{P1} + \hat{\sigma}^2_{P2} + 2\hat{\sigma}^2_{F1}) / 4$$
$$A = 2\hat{\sigma}^2_{F2} - \hat{\sigma}^2_{BC1} - \hat{\sigma}^2_{BC2})$$
$$D = \hat{\sigma}^2_{BC1} + \hat{\sigma}^2_{BC2} - \hat{\sigma}^2_{F2} - \hat{\sigma}^2_{E}$$
$$F = (\hat{\sigma}^2_{BC2} - \hat{\sigma}^2_{BC1}) / 2$$

where, E = non-inheritable components or environmental variance, A = additive variance component, D = dominance variance component and F = additive variance in dominance. Furthermore, A and D under Falconer method (Falconer, 1960) are equivalent of H and D in Mather and Jinks (1982) method, respectively.

Broad-sense heritability is calculated based on different methods for estimation of environmental variance following Mather and Jinks (1982).

$$(1) h^2_{Bs} = \left[\hat{\sigma}^2_{F2} - \left(\frac{(\hat{\sigma}^2_{P1} + \hat{\sigma}^2_{P2})}{2} \right) \right] / \hat{\sigma}^2_{F2}$$
$$(2) h^2_{BS} = \left[\hat{\sigma}^2_{F2} - \left(\sqrt{\hat{\sigma}^2_{P1} \times \hat{\sigma}^2_{P2}} \right) \right] / \hat{\sigma}^2_{F2}$$
$$(3) h^2_{BS} = \left[\hat{\sigma}^2_{F2} - \left(\hat{\sigma}^2_{F1} \right) \right] / \hat{\sigma}^2_{F2}$$
$$(4) h^2_{BS} = \left[\hat{\sigma}^2_{F2} - \left(\sqrt[3]{\hat{\sigma}^2_{P1} \times \hat{\sigma}^2_{P2} \times \hat{\sigma}^2_{F1}} \right) \right] / \hat{\sigma}^2_{F2}$$
$$(5) h^2_{BS} = \left[\hat{\sigma}^2_{F2} - \left(\frac{(\hat{\sigma}^2_{P1} + \hat{\sigma}^2_{P2} + \hat{\sigma}^2_{F1})}{3} \right) \right] / \hat{\sigma}^2_{F2}$$
$$(6) h^2_{BS} = \left[\hat{\sigma}^2_{F2} - \left(\frac{(\hat{\sigma}^2_{P1} + \hat{\sigma}^2_{P2} + 2\hat{\sigma}^2_{F1})}{4} \right) \right] / \hat{\sigma}^2_{F2}$$

Narrow-sense heritability is calculated following Warnner (1952).

$$h^2_{NS} = \frac{\left[2\hat{\sigma}^2_{F2} - (\hat{\sigma}^2_{BC1} + \hat{\sigma}^2_{BC2}) \right]}{\hat{\sigma}^2_{F2}}$$

Genetic progress was calculated for all traits following Jain and Allard (1960).

$$G_S = K \sqrt{\hat{\sigma}^2_{F2}} \times h^2_{NS}$$

To study the differences between various generations for the duration of latent period,

infection type, pustule size, and pustule density, a weighted analysis of variances were performed. Moreover, the assumptions of analysis of variance regarding homogeneity of variance across studied treatments was considered. Since this is not true for different generations, balanced variance analysis was applied to address the homogeneity of variances.

RESULTS AND DISCUSSION

Based on scores assigned to each of the standard and differential set varieties following the method proposed by Johnson *et al.* (1972), this isolate was designated 134 230E158A+, *Yr27+*. The designation of this race, and virulent and non-virulent gene formula is presented in Table 1.

Table 1. Virulent and non-virulent gene formula of isolate of wheat yellow rust used in greenhouse test

Isolate	Yr virulent genes	Yr non-virulent genes
230E158A+	*Yr1, 3, 4, 5, 10, 15, 24, 32, CV, SP*	*Yr2, 2+, 6, 6+, 7, 7+, 8, 9, 9+, 17, 18, 22, 23, 25, 26, 27, ND, SD, SU, A*

Analysis of variance for the four traits are presented i Table 2. A significant difference between generations indicates the feasibility of genetic analysis and the study of their inheritance. The results of mean comparison for traits by Duncan's Multiple Range Test showed that the susceptible parent (Avocet's') has higher values of infection type, pustule size, and pustule density and lower ones for the latent period of the disease (Table 3). Generally, susceptible varieties have a shorter latent period, compared to the resistant varieties. Resistance variety of Aflak, along with F1 generation has also lower values for infection type, pustule size, and pustule density and higher ones for the latent period of the disease. In addition, F2, BC2 generations together, and BC1 generations have a close amount of infection type, pustule size, pustule density, and latent period.

Table 2. Weighted analysis of variance for latent period (LP), infection type (IT), pustule Size (PS) and pustule density (PD) traits in wheat

		Mean square			
S.O.V.	df	IT	LP	PS	PD
Replication	2	47.15^{ns}	272.31^{ns}	256.7	73.56
Generation	6	95393.2^{**}	173401.1^{**}	16143.42^{**}	75369.78^{**}
Within	3722	369.8	2931.08	2929.57	2923.9
Error	12	190.2	115.26	127.06	136.32
CV%		9.06	3.31	7.09	4.48

* and ** : Significant at the 5% and 1% probability levels, respectively.

Table 3. Mean and standard error of infection type (IT), latent period (LP), pustule size (PS) and pustule density (PD) traits for parents and four generations

Trait	Aflak	Avocet's'	F_1	F_2	BC_1	BC_2
IT	0±0.1d	7.69±0.97a	0±0.1d	1.93±3.56c	4.51±4.27b	1.75±3.29c
LP	20±0.1a	10.91±0.82d	20±0.1a	17.84±5.95b	15.1±4.61c	18.22±3.68b
PS	0±0.1d	2.06±0.69a	0±0.1d	0.54±1.02c	1.3±1.26b	0.46±0.96c
PD	0±0.1d	5.47±1.63a	0±0.1d	1.37±2.55c	3.26±3.06b	1.08±2.29c

Means, in each column, followed by similar letter are not significantly different at the 5% probability level-using Duncan's Multiple Range Test.

Estimations of dominance (h/d), broad sense heritability by different methods, narrow sense heritability and genetic advance for the four traits are presented in Table 4. The influences of genes in different ways may cause related impacts to be estimated less than the true value. The dominance of the four traits was equal to one that indicates a

complete dominance. Negative dominance of infection type, pustule size, and pustule density shows that there exists a full dominance of low infection type (higher resistance). For the latent period, the dominance shifted towards longer latent period (higher resistance).

The estimations for broad-sense heritability of the four traits varied between 84% to 99% (see Table 4). Narrow-sense heritability for four traits of latent period, infection type, pustule size, pustule density was estimated 57, 51, 54, and 62%, respectively. The broad-sense heritability was high for the four traits and moderate to narrow-sense heritability was obtained for all the resistance components. The difference between broad-sense and narrow-sense heritability indicated the role played by the dominance variance in genetic control of all the four traits. Besides, the additive variance (heritable and stabilized genetic variance) has lower importance. It must be noted that by ignoring the epistasis effect in the estimation of the heritability, may also affect both the estimations of genetic diversity and the prediction of the selection. This type of genetic data is very useful for breeders in applied breeding programs.

Genetic advance for the latent period, infection type, pustule size, and pustule density were 4.49, 3.91, 1.48, and 3.76, respectively (Table 4). The success in selection depends on the narrow-sense heritability (Mather and Jinks, 1982). The high narrow-sense heritability can speed up the selection process of high resistance (Chen and Line, 1995). On the other hand, heritability provides the required information for the transfer of traits to progeny. Therefore, it facilitates the estimation of genetic and environmental contributions to phenotypic diversity. However, it should be noted that considering epistasis in the estimation of heritability may affect estimations of additive variance and also the prediction of genetic advances resulting from the selection. The estimations of heritability allow improvers to predict genetic advance under selected conditions through different selection methods at different pressures. Knowledge of manner of genetic control (single-gene and/or multiple genes) of traits is very important in determining improvement methods.

Table 4. Estimates of degree of dominance (h/d), broad- sense hereditability (h^2_{BS}) using different methods, narrow- sense heritability (h^2_{NS}), and genetic advance (GA) for latent period (LP), infection type (IT), pustule size (PS) and pustule density (PD) traits

		h^2_{BS}								
Trait	h/d	1	2	3	4	5	6	Means	h^2_{NS}	GA
IT	-1	0.96	0.99	0.99	0.99	0.97	0.98	0.98	0.51	3.91
LP	1	0.97	0.99	0.99	0.99	0.98	0.98	0.99	0.57	4.49
PS	-1	0.86	0.98	0.99	0.99	0.9	0.93	0.94	0.54	1.48
PD	-1	0.84	0.99	0.99	0.99	0.89	0.92	0.94	0.62	3.76

Jacob and Broers (1989) and Khodarahmi *et al.* (2007) also reported similar results. One should remember that the ratio (h/d) for determining the validity of gene action is not considered a valid index. It is especially true when more than one gene is involved in controlling the trait (Mather and Jinks, 1971). In this case, the ratio (h/d) which is due to the marked difference of dominant of very small genes that control the traits (and thus, shrinking the section h) may be very large, because of the distribution of additive genes or gene subtraction between parents, and the elimination of all other gens effects (resulting in shrinking section d) (Mather and Jinks, 1971).

Therefore, the parameter √H/D is used rather than (h/d) as an estimate of the average degree of dominance. The √H/D was greater than one for all traits, except for the pustule density (Table 5). It indicates the existence of over dominance genetic control of these traits, however, the √H/D was close to one for pustule density which indicates the existence of complete dominant genetic control for this trait (Table 5).

Table 5. Variance components for latent period (LP), infection type (IT), pustule size (PS) and pustule density (PD) traits in parents and four generations

	Variance components					
Trait	D	H	F	Ew	√H/D	F/√HD
IT	14.59	28.72	-0.38	0.24	1.4	-0.02
LP	16.7	24.44	-1.7	0.17	1.21	-0.08
PS	1.9	2.68	-0.67	0.12	1.19	-0.3
PD	10.65	10.04	-1.15	0.67	0.97	-0.11

D: Additive, H: Dominance, F: Covariance of additive components of dominance, Ew: Environmental variance.

In this study, by selecting parents from two extremes phenotypic distribution of traits, the assumption of the distribution of alleles in parents of four generations was met. Variance components for four traits are presented in Table 5. According to estimates of variance components of three traits; infection types, latent period, pustule size, dominance component (H) are greater than the additive component (D). The dominance component is equal to the additive component in the pustule density. The narrow-sense heritability degree in all traits also confirmed it. The covariance of additive components of dominance (F) was negative in all the four traits. Therefore, dominant genes have added in a parent with a low value of the trait. This component (F) shows the correlation of d and h in the mean of all genetic loci (Table 5). The component F close to one indicates the fixed dominance (h/d) at all genetic loci, in respect to significance and magnitude. If the direction of dominance varies between loci controlling traits, then the F value will approximate zero.

The absolute value of the F/√DH component for all traits was estimated smaller than or equal to one (Table 5). It indicates that genes controlling these traits varied in respect to significance and magnitude. In this case, the ratio of h/d decreases and it cannot be used as a good estimate of dominance. Component √DH provides a good estimation of dominance. Generally, √DH is a more reliable estimation than [h] / [d] in determining the function of genes. This is because [h] / [d] is affected by the sign of the dominance of genes and the manner of distribution of increasing and decreasing alleles among parents. Environmental variance (Ew) suggests non-genetic variations. This may be due to different reasons as its nature largely depends on the trait and the plant used in the study. Generally, the environmental variance is a source of error that reduces the precision of genetic studies, therefore, the breeder aims to reduce this variance as much as possible. Nutritive and climatic factors are the most common factors of environmental variation which can be partly controlled by appropriate field management. In all four traits, environmental factors had quantitative effects on the expression of traits.

The estimation of mean genetic components using generation mean analysis employing the combined weight test showed that χ^2 of the three-parameter model of m, [d], [h] was significant for each of the four traits (Table 6), which at least suggest the presence of the interaction of two genes in controlling the resistance to yellow rust, besides the main effects. Thus, a simple dominance additive model to explain the genetic control of resistance to rust does not suffice in this crossbreeding.

Table 6. Estimates of genetic components for latent period (LP), infection type (IT), pustule size (PS) and pustule density (PD) traits using parents and four generations

	Genetic components						
Trait	*m*	*[d]*	*[h]*	*[i]*	*[j]*	*[l]*	χ^2
IT	3.84± 0.07**	-3.84 ± 0.07**	-2.85 ± 0.80**	-	12.38 ± 1.72**	-	5.96
LP	15.45 ± 0.06**	4.54 ± 0.06**	4.54± 0.06**	-	-14.55 ± 1.89**	-	5.13
PS	1.03 ± 0.05**	-1.03 ± 0.05**	-0.66 ± 0.27**	-	3.49 ± 0.51**	-	5.50
PD	2.73 ± 0.05**	-2.73 ± 0.05**	-2.07 ± 0.57**	-	9.21 ± 1.21**	-	5.22

* and ** : Significant at the 5% and 1% probability levels, respectively.

m: mean of total generations, *d* : sum of additive effects, *h:* sum of dominance effects, *i*: sum of reciprocal effect among additive effects, *j*: sum of reciprocal effect among additive and dominance effects and *l* : sum of reciprocal effect among dominant effects.

Mather and Jinks (1982) stated that data can be transformed as to find an appropriate scale. However, neither logarithmic nor the square root transformation of the data could reduce the chi-square test values for all models. Thus, all possible models including the six-parameter model were fitted to the observed means. Following Mather and Jinks Mather and Jinks (1982) method, non-significant components were removed from the six-parameter model, and the remaining components were fitted as the model could be better fitted. The Model's precision should increase with increase in the means and reduction in standard error. For all traits, the four-parameter model m, [d], [h], [j], which had smaller non-significant chi-square, was appropriate. In the 4-parameter model, all genetic components became significant at the 1% probability level (Table 6). All models were compared by the goodness of fit test and using the chi-square test with five, four, three, two and one degrees of freedom.

Having goodness of fit test for the four parametric models, in all studied traits, the possibility of the interaction of genotype × environment, triple interaction, and linkage among genes is very low. The significance of the additive interaction in dominance indicated that both additive and non-additive (dominance and epistasis) interactions had been effective in inheritance of resistance to yellow rust isolate. By observing epistasis, it is reasonable to assume that more genes control these traits. Epistatic gene action is not the common inheritance of qualitative traits, but it is common for quantitative traits. As the number of genes that control a trait increases, it is reasonable to assume that the number of factors that have interactions with each other increases. These results are consistent with the results reported by Milus and Line 1986.

The significance of [m] indicated that there was significant difference between the two parents. Also, [d] values in comparison with [h] were larger for infection type, pustule size, and the pustule density, and was equal to the latent period, which showed the correlation of genes (i.e. genes with reduced impact gathered in one parent). In other words, resistance genes were accumulated in one parent. Dominance effect [h] of infection type was negative for pustule size and pustule density which indicated the dominance effect in reducing these traits. Dominance effect [h] of latent period was positive which indicated the dominance effect in increasing the duration of the latent period.

Signs of parameters [d] and [j] depends on which parent is P1 or P2. Therefore, sign of [j] changes in most cases, however, the sign of other parameters remains unchanged. Reciprocal effect among additive and dominance effects [j] was positive for latent period, but it was negative for the other traits. This type of epistasis which is selected by selfing is not stable in population. The results of generations mean analysis showed that at least one or two significant two-way or epistatic interaction effects existed between genes, and this may resulted in higher estimates of the additive variance than the dominance variance.

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REFERENCES

Afshari, F., Nazari, K., Aghaei, M., Najafian, G., Esmaeilzadeh, M., Yazdansepas, A., Khodarahmi, M., Amini, A., Roohparvar, R., Hashamei, M. and Malihipour, A. 2014. The wheat stripe rust pathogen in Iran and achievement of resistant wheat cultivars over the last 10 years. pp. 5. In: Proceedings of the 2nd Int. Wheat Stripe Rust Symposium. Izmir, Turkey.

Afshari, F., Torabi, M. and Malihipour, A. 2003. Appearance of a new race of *Puccinia striiformis* f. sp. *tritici* in Iran. Seed and Plant J. 19: 543-546.

Ali, S., Rodriguez-Algaba, J., Thach, T., Sørensen, C. K., Hansen, J. G., Lassen, P., Nazari, K., Hodson, D. P., Justesen,

A. F. and Hovmøller, M. S. 2017. Yellow rust epidemics worldwide were caused by pathogen races from divergent genetic lineages. Front. Plant Sci. 8: 1057.

Beddow, J. M., Pardey, P. G., Chai, Y., Hurley, T. M., Kriticos, D. J., Braun, H. J., Park, R. F., Cuddy, W. S. and Yonow, T. 2015. Research investment implications of shifts in the global geography of wheat stripe rust. Nat. Plants 1: 15132.

Broers, L. H. M. and Jacobs, T. 1989. The inheritance of host plant effect on latency period of wheat leaf rust in spring wheat. II: number of segregating factors and evidence for transgressive segregation in F3 and F5 generations. Euphytica 44 (3): 207-214.

Chen, X. M. 2005. Epidemiology and control of stripe rust (*Puccinia striiformis* f. sp. *tritici*) on wheat. Can. J. Plant Pathol. 27: 314-337.

Chen, X. M. and Line, R. F. 1995. Gene number and heritability of wheat cultivars with durable, high-temperature, adult-plant (HTAP) resistance and interaction of HTAP and race-specific seedling resistance *Puccinia striiformis.* Phytopathol. 85: 573-578.

Chen, X. M. and Line, R. F. 1992. Identification of stripe rust resistance genes in wheat genotypes used to differentiate North American races of *Puccinia strirformis.* Phytopathol. 72: 1428-1434.

Chen, W., Wellings, C., Chen, X., Kang, Z. and Liu, T. 2014. Wheat stripe (yellow) rust caused by *Puccinia striiformis* f. sp. *tritici*. Mol. Plant Pathol. 15: 433–446.

Falconer, C. O. 1960. Introduction to quantitative genetics. Ronald Press. New York. 485 pp.

Falconer, D. S. and Mackay, T. F. C. 1996. Heritability. pp. 160-183. In: Falconer, D. S. and Mackay, T. F. (eds.) Introduction to quantitative genetics.

Gebrewahid, T. W., Zhang, P., Yue Zhou, Yan, X., Xia, X., He, Z., Liu, D. and Li, Z. 2020. QTL mapping of adult plant resistance to stripe rust and leaf rust in a Fuyu 3/Zhengzhou 5389 wheat population. The Crop J. 8: 655–665.

Hallauer, A. R. and Miranda, F. J. B. 1988. Quantitative genetics in maize breeding. 2nd edition. Iowa State University Press/Ames. 468 pp.

Hill, J., Becker, H. C. and Tigerstedt, P. M. A. 1998. Quantitative and ecological aspects of plant breeding. Chapman and Hall, London. 275 pp.

Hovmøller, M. S., Walter, S., Bayles, R. A., Hubbard, A., Flath, K., Sommerfeldt, N., Leconte, M., Czembor, P., Rodriguez-Algaba, J., Thach, T. and Hansen, J. G. 2016. Replacement of the European wheat yellow rust population by new races from the center of diversity in the near-Himalayan region. Plant Pathol. 65: 402–411.

Hovmøller, M. S., Yahyaoui, A. H., Milus, E. A. and Justesen, A. F. 2008. Rapid global spread of two aggressive strains of a wheat rust fungus. Mol. Ecol. 17: 3818–3826.

Hubbard, A., Lewis, C. M., Yoshida, K., Ramirez-Gonzalez, R. H., de Vallavieille-Pope, C., Thomas, J., Kamoun, S., Bayles, R., Uauy, C. and Saunders D. G. 2015. Field pathogenomics reveals the emergence of a diverse wheat yellow rust population. Genome Biol. 16: 23.

Hussain, F., Ashraf, M., Hameed, M. A., Hussain, N. and Sial, R. A. 2011. Genetic studies in wheat for leaf rust resistance (*Puccinia recondita*). Afr. J. Biotechnol. 10 (16): 3051-3054.

Jacobs, T. and Broers, L. H. M. 1989. The inheritance of host plant effect on latency period of wheat leaf rust in spring wheat. I: Estimation of gene action and number of effective factors in F_1, F_2 and backcross generations. Euphytica 44 (3): 197-206.

Jain, S. K. and Allard, R. W. 1960. Population studies in predominantly self-pollinated species, I. Evidence for heterozygote advantage in a closed population of barley. Proc. Natl. Acad. Sci. 46 (10): 1371-1377.

Jin, Y., Szabo, L. J. and Carson, M. 2010. Century-old mystery of *Puccinia striformis* life history solved with the identification of *Berberis spp.* as an alternate host.

Phytopathol. 100: 432-435.

Johnson, R., Stubbs, R. W., Fuchs, E. and Chambrlain, N. H. 1972. Nomenclature for physiologic race of *Puccinia striiformis* infecting wheat. Trans. Br. Mycol. Soc. 58: 475-480.

Khodarahmi, M., Bihamta, M. R., Mohammadi, S. A. and Majidi Hervan, E. 2007. Inheritance of stripe rust resistance in bread wheat. Iran. J. Crop Sci. 8 (4): 368-378.

Khodarahmi, M., Jalal Kamali, M. R. and Bihamta, M. R. 2009. Inheritance and gene action for resistance to stripe rust in bread wheat. pp. 13-16. In: Proceedings of 12th International Cereal Rust and Powdery Mildews Conference; Antalya, Turkey.

Lee, T. S. and Shaner, G. 1985. Oligogenic inheritance of length of latent period in six slow leaf rusting wheat cultivars. Phytopathol. 75: 636 -643.

Mather, K. and Jinks, J. L. 1982. Biometrical genetics. The Study of Continuous Variation. 3nd edition. Chapman and Hall. London. 412 pp.

McNeal, F. H., Konzak, C. F., Smith, E. P., Tate, W. S. and Russell, T. S. 1971. A uniform system for recording and processing cereal research data. Dept. Agric. Res. Serv. ARS. USA. 34: 121-43.

McIntosh, R. A., Dubcovsky, J., Rogers, W., Morris, C., Appels, R. and Xia, X. 2016. Catalogue of gene symbols for wheat: 2015–2016 supplement. 18 pp.

Milus, E. A., Kristensen, K. and Hovmøller, M. S. 2009. Evidence for increased aggressiveness in a recent widespread strain of *Puccinia striiformis* f. sp. *tritici* causing stripe rust of wheat. Phytopathol. 99: 89-94.

Rutkoski, J. E., Heffner, E. L. and Sorrells, M. E. 2010. Genomic selection for durable stem rust resistance. Euphytica 179: 161-173

Singh, R. P., Singh, P. K., Rutkoski, J., Hodson, D. P., He, X., Jørgensen, L. N., Hovmøller, M. S. and Huerta-Espino, J. 2016. Disease impact on wheat yield potential and prospects of genetic control. Annu. Review of Phytopathol. 54: 303-322.

Singh, R. P., William, H. M., Huerta-Espino, J. and Rosewarne, G. 2004. Wheat rust in Asia: meeting the challenges with old and new technologies. New directions for a diverse planet. pp. 1-13. In:Proceedings of the 4th Int. Crop Sci Cong. Brisbane, Australia.

Steuernagel, B., Periyannan, S. K., Hernandez-Pinzon, I., Witek, K., Rouse, M. N., Yu, G., Hatta, A., Ayliffe, M., Bariana, H., Jones, J. D. G. and Lagudah, E. S. 2016. Rapid cloning of disease resistance genes in plants using mutagenesis and sequence capture. Nat. Biotechnol. 34: 652-655.

Warner, J. N. 1952. A method for estimation heritability. Argon. J. 44: 427-430.

Wellings, C. R. 2011. Global status of stripe rust: a review of historical and current threats. Euphytica 179: 129-141.

Zadoks, J. C., Chang, T. T. and Konzak, C. F. 1974. A decimal code for the growth stages of cereals. Weed Res. 14: 415-21.