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Research Article

Inheritance of Resistance to Stripe Rust (*Puccinia Striiformis* f.sp. *Tritici*) Race 198E154A+ In Wheat cv. Morvarid

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Abstract

To study the heritability of resistance to stripe rust in bread wheat, F1, F2, BC1 and BC2 generations, derived from a cross between two wheat cultivars named Morvarid (resistant) and Bolani (susceptible) along with the parents, were evaluated under greenhouse condition, in a Randomized Complete Block Design (RCBD) with three replications. The seedlings were inoculated using the race 198E154A+ urediniospores. Components of resistance including infection type and latent period on single plants of generations were recorded. The results of weighted ANOVA showed that there were significant differences ($p < 0.01$) between the generations for two traits. Results of the generations mean analysis showed that additive; dominance and epistasis (especially additive × dominance and dominance × dominance) gene actions have a significant role in control of the traits. Furthermore, high broad-sense heritability was observed for these traits. The narrow-sense heritability was in an average range for infection type and was low for latent period. The number of segregating genes was estimated 1 - 2 for infection type and 1 - 3 for latent period.

Keywords: Heritability; Yellow Rust; Resistance Components; Generations Mean Analysis; Wheat

Introduction

Wheat is one of the most important cereals and cultivated in large areas around the world. Yellow (strip) rust, caused by *Puccinia striiformis* f. sp. *tritici* is the most important fungal disease of wheat, especially in cold areas [1]. It is reported that in epidemic conditions, seed yield will decrease up to 75 percent [2]. Yellow rust is the most common wheat rust in Iran which a major epidemic occurs every few years in different regions of Iran. The main approach for control of yellow rust is proper using of genetic resistance and cultivation of resistant cultivars [3]. To date, more than 70 stripe rust resistance genes with official and provisional designations have been reported in wheat [4]. However, many rust resistance genes are affective for a short period of time, their resistance become ineffective with appearance of new races. Therefore, producing new resistance cultivars needs continuous efforts [5].

Since resistance components demonstrating quantitatively and single gene effects are so little, we can't identify them with Mendelian analysis. Thus, genes specifications should be examined by genetic analysis such as generation mean analysis and generation variance analysis [6,7].

Information of these analyses is useful for breeding methods of wheat rust resistance selection. Also, for determining the number of genes that control attributes which have quantitative variety statistical methods can be used such as generations means analysis and QTL analysis. Nowadays, by creating genetic maps that have been made by molecular techniques, it is possible to determine quantitative traits controlling gene locus (QTL) on chromosome and their contribution to the appearance of phonotype should be determined [2]. Lots of researches have been done in order to find out inheritance of resistance components. Asad identified and mapped a stripe rust resistance gene in wheat line Shaannong 104 using SSR (simple sequence repeat) markers. F_1, F_2 and F_3 populations from Shaannong 104/Mingxian 169 were inoculated with Chinese Pst race CYR32 in a greenhouse. Pedigree analysis, pathogenicity tests using 26 Pst races, haplotyping of associated markers on isogenic lines carrying known stripe rust resistance genes, and associations with markers suggested that YrSN104 was a new resistance gene or an allele at the Yr24/Yr26 locus on chromosome 1BS. Deployment of YrSN104 singly or in combination to elite genotypes could play an effective role to lessen yield losses caused by stripe rust [8]. Moghadam and colleague, by using generations mean analysis in yellow rust resistance evaluations, have observed resistance return phenomenon in two different crosses for two different yellow rust race and have reported mean high broad-sense heritability and narrow-sense heritability 69 and 48 percent respectively [9].

Khodarahmi., *et al.* [10] studied the inheritance of stripe rust resistance and estimated the genetic components of resistance in wheat. The generation mean analysis of F_1, F_2, BC_1 and BC_2 generations derived from a cross between MV17 as resistant and Bolani susceptible parents with pathotypes 134E134A$^+$ and 166E134A$^+$ of stripe rust in two different experiments, revealed that additive, dominance and epistasis (especially [j] and [l] components) play a major role in increasing and decreasing latent period and infection type, respectively. In spite of significant additive effect, dominance gene effect was the most important component in controlling these two characteristics. Estimates of degree of dominance were very close to unity for the two concerned traits in response to both pathotypes which indicates a complete dominance resistance. Heritability ranged from moderate to high and number of segregating genes governing resistance ranged from 1 to 3. Bihamta., *et al.* [11] have studied five wheat cultivars and ten F_1 breed of their confluence with two yellow rust race that Kotari has the high broad-sense heritability in order to increase resistance between other parents. Also, for reducing infection type, additive amount has a mean roll in comparison of non-additive. Ma for identifying the resistance gene(s) against stripe rust, crossed Zhongliang 12 with stripe rust susceptible genotype Mingxian169, and F_1, F_2, $F_{2:3}$ and BC_1 progenies were tested with Chinese Pst race CYR30 and CYR31 at seedling stage under greenhouse conditions and reported resistance gene effects with linkage maps on chromosomes 7 AL and 1 AL [12].

The aim of this research was to evaluation of inheritance and genetic control of resistance to wheat yellow rust disease in Morvarid cultivar, which cultivated in great scale in north of Iran, and also determining the number of resistance genes by generation mean analysis in greenhouse condition.

Materials and Methods

This research was done in yellow rust greenhouses of cereal research department of Seed and Plant Improvement Institute (SPII) in Karaj in 2015. Morvarid cultivar with Milah/sha7 pedigree showed resistance to all of races from different areas in previous experiments and also this cultivar is culture in large areas of north of Iran and has a significant resistance to yellow rust.

In this research, P_1, P_2, F_1, F_2, BC_1 and BC_2, and Morvarid as resistance cultivar and Boloni as susceptible cultivar in greenhouse condition were planted. The generations were inoculated with 198E154A$^+$ yellow rust race in seedling stage. The inoculation was done with spraying mixture of urediniospores and tween 20 (industrial oil) on surface of plants. 20 seeds of P_1 and P_2 and F_1 and 240, 60, 60 seeds for F_2, BC_1, BC_2 planted in all replicate respectively. Inoculation was done in 12 steps of Zadoks scale [13]. Inoculated plants placed in cold room for 24 hour and 11^{0}c temperature with 95 percent humidity in dark condition, then pots transfer to greenhouse with 60 to 70 percent humidity, 18^{0}c temperature, 16000 lux lights provided by mixture of sodium and fleurcent lights, 16 hours day long and stayed in this situation for 20 days. Seedling irrigation was performed using flooding method. Infection type scoring was recorded in scale 0 to 9 by MacNeal method [14].

Generation mean analysis was done using Mather and Jinks method [15]. Broad-sense heritability (H^2) and narrow-sense heritability (h^2) was estimated by Warnner [16] and Mather and Jinks [15] methods. The least number of gene number or effective factor were calculated as below (Cockerham, 1988, Wright, 1968 and Lande, 1981):

GNF_1: $$n = \frac{(\bar{P}_1 - \bar{P}_2)^2}{\left[8(\hat{\sigma}^2_{F2} - \hat{\sigma}^2_{F1})\right]}$$

GNF_2: $$n = \frac{(\bar{P}_1 - \bar{P}_2)^2}{8\left[\left(\hat{\sigma}^2_{F2} - (0.5\hat{\sigma}^2_{F1} + 0.25\hat{\sigma}^2_{P1} + 0.25\hat{\sigma}^2_{P2})\right)\right]}$$

GNF_3: $$n = \frac{(\bar{P}_1 - \bar{P}_2)^2}{8\left[2\hat{\sigma}^2_{F2} - (\hat{\sigma}^2_{BC1} + \hat{\sigma}^2_{BC2})\right]}$$

GNF_4: $$n = \frac{(\bar{P}_1 - \bar{P}_2)^2}{8\left[(\hat{\sigma}^2_{BC1} + \hat{\sigma}^2_{BC2}) - (\hat{\sigma}^2_{F1} + 0.5\hat{\sigma}^2_{P1} + 0.5\hat{\sigma}^2_{P2})\right]}$$

GNF_5: $$n = \frac{(\bar{F}_1 - \bar{P}_1)^2}{4\left[\hat{\sigma}^2_{BC1} - 0.5(\hat{\sigma}^2_{F1} + \hat{\sigma}^2_{P1})\right]}$$

Results and Discussion

To evaluate the differences between different generations, traits latent period and infection type, weighted ANOVA was conducted. The results of this variance analysis are presented in table 1.

S.O.V	df	MS	
		IT	LP
Rep	2	1.67^{ns}	1.00^{ns}
Genotype	5	145.10**	474.492**
Error	10	1.02	1.76
CV %		38.16	5.25

Table 1: Weighted ANOVA for Infection Type (IT) and Latent period in Wheat.

*, **: Significant at 5% and 1% probability level respectively ns: Non-significant.

Significant differences between generations (genotypes) demonstrated that it is possible to apply Generation Mean Analysis (GMA) and study their inheritance. Results of mean comparison in different generations using Duncan test showed that susceptible parent (Bolani) as P_1 has the most infection type and had the lowest latent period as we expected (table 2). The least value of in-

fection type and the most value of latent period were observed in Morvarid cultivar (P_2) and F_1. Also, F_2, BC_1 and BC_2 had same value for infection type and latent period.

Trait	Bolani P_1	Morvarid P_2	F_1	F_2	BC_1	BC_2
IT	7.81 A	0.24 D	0.67 C	2.11 B	2.23 B	2.31 B
LP	9.98 C	19.99 A	19.96 A	13.24 B	13.39 B	13.99 B

Table 2: Mean error of infection type (IT) and latent period (LP) traits for six generations in wheat and comparison of means by Duncan method.

Inheritance estimations by using of different method and estimating degree of dominance (h/d) for latent period and infection type are presented in table 3. There are some difficulties in explanations results of generations mean analysis. The parameters which define the gene effects are in facts balanced the effects of all loci are segregating and the effect of genes in different directions, causing the effect is less than the real value estimate.

Trait		h^2							H^2
	h/d	1	2	3	4	5	6	Means	
IT	-0.73	0.79	0.91	0.52	0.84	0.70	0.66	0.74	0.52
LP	0.78	0.85	0.85	0.41	0.76	0.70	0.63	0.70	0.24

Table 3: Estimates of heritability by different methods. Degree of dominance for infection type (IT) and latent period (LP).

The number of dominance degree for both traits was less than one that shows genetically control of incomplete dominance. The negative dominance degree showed for less infection type (high resistance) there is incomplete dominance. The positive number of resistance shows that there is an incomplete dominance for high resistance (highest latent period). Broers and Jacobs [17] reported similar results. We should pay attention that (h/d) rate doesn't have value in determine gene function. Especially when more than one gene is involved in control of trait [15].

In this case, Ratio (h/d) may become very small because different sign dominant genes controlling traits (and as a result, the miniaturize of the h) or due the distribution of increasing and decreasing trait genes between parents and eliminate the effects of each other (and as a result, the miniaturize of the d) this ratio is very large [15].

Due to this reason, the parameter $\sqrt{H/D}$ used instead ratio (h/d) as estimation of average degree of dominance. The $\sqrt{H/D}$ value was less than one for infection type which indicate genetic control of this trait is the incomplete dominance (table 5). For latent period the $\sqrt{H/D}$ was more than one which indicates there is over dominance in controlling this trait.

Trait	Number of Segregating genes					
	1	2	3	4	5	6
IT	1.95	1.55	1.98	1.29	2.88	0.13
LP	2.06	1.35	3.55	0.83	1.61	0.11

Table 4: Estimates of the segregating genes number for infection type(IT) and latent period(LP).

	Components of Variation					
Trait	D	H	F	Ew	$\sqrt{H/D}$	$F/\sqrt{HD}$
IT	0.8	0.42	-0.08	0.26	0.73	-0.13
LP	6.22	20.3	-8.74	4.75	1.81	-0.78

Table 5: The components of variation for infection type(IT) and latent period(LP) in six generations.

The broad sense heritability (h^2) range for infection type with using the different formula was 52 - 91% and the average broad sense heritability for this trait was 74%. (table3). In general, broad sense heritability for this trait was high. The narrow sense heritability was estimated as 52%. The difference of broad sense heritability and narrow sense heritability shows the greater role of dominance variance in genetically control of this treat and additive variance (Heritable genetic variance) has a less importance.

The broad sense heritability for latent period was in a range of 41 - 85% and the average broad sense heritability for this trait was 70%. There is high broad sense heritability for latent period. The narrow sense heritability for latent period was 24% indicated that the narrow sense heritability is low for this trait. Should pay attention that don't including epistasis in estimating heritability, it may effect in estimating additive genetically variance and predicting of progress of selection result. This genetic data is important for breeders in breeding programs. Although should pay attention that estimation epistasis in heritability may have effect on additive variance estimation and also may have effect on predicting genetic gain of selection. Heritability estimations help breeders to predict genetic gain with different intensity of selection. Knowledge of the genetic control (Monogenic or multigenic) of trait is important for choosing breeding method. Based on five different method, minimum controlling genes for infection type was estimated between one or two segregating gene (table 4). Other researcher has reported some gene number for this treat in wheat [17,18]. For the latent period was estimated at one to three segregating genes. In estimating the number of genes, it should be noted that it is assumed to be: estimating non-existence relation between mean and variance, absent of genes adherence linkage, absent of epistasis, equal gene effects in all different genes loci, existence of positive alleles in one parent and negative alleles in other parent and equal degree of dominance for positive alleles. If these assumptions are not considered, the number of segregating genes estimation will be different from actual number. Another limitation in the estimated number of genes is the number of plants sampled. The number

of plants sampled, particularly in the F_2 population must be large enough to include all modes of genes segregating [17,19]. In this study, Parents, were selected from both ends of the phenotypic distribution, so assuming the distribution of alleles in parents' generation was observed. Variation components for both traits are presented in table 5. To infection type trait, the amount of additive (D) is greater than the dominant component (H) that shows importance of additive effect in genetically control of this trait. Vice versa for latent period dominance component (H) was more important than additive component (D) in both traits, the narrow sense heritability emphasis of this covariance of additive-dominance components (F) for infection type was near zero and for latent period was negative. The negative sign of F shows dominant genes, mostly in the parent with the lower value of the trait is gathered. If the degree of dominance be different in between the locations trait controller, then the amount of F toward zero. The (F) shows correlation between d and h in all mean gene locus. Obsolete value of F/√DH estimated less than one that shows different genes controlling this traits in terms of sign and enlargement in different gene locos and dominance alleles are dispersed in both parents in this situation h/d rate will decrease and this rate can't be a good estimation of dominance. In this situation √H/D is acceptable estimation of dominance degree.

Generally, √H/D is a much more reliable than (h/d) for determining of genes actions. Because ratio of (h/d) is affected by genes dominance sign and distribution of alleles increasing and decreasing trait between parents. Environmental variance (EW) Show non-genetically changes and can have different causes, and it depends on trait and the related plant.

Generally (EW) is variations source that reduce accuracy of genetic studies and so that breeders aim is reduce this variance as much as possible. Nutritional and climate factors are the most common causes of environmental variation. And at least part of them can be controlled by experimentation. Environment had little effect on the infection type but had a greater effect to latent period than the in-faction type. The results of generation mean analysis (with 6 generation) using weighted least square (table 6) showed that for these traits X2 amount was significant for three parameters model m, [d], [h] that suggested in addition to the main genetic effect, there are at least the interaction of two genes in the control of yellow rust resistance. Thus, an additive-dominance simple model to explain the genetic control of rust resistance in this cross has no the necessary effectiveness.

Five parameter model m, [d], [h], [j] and [I], were suitable that it has nonsignificant and smaller K square ($\chi 2$). In five parameter model all genetic components were significant. With a goodness of fit of the 5 parameters model, the possibility of genotype × environment interaction, triple interaction and linkage between genes is very low. In the presence of epistasis, it is reasonable to assume that more genes that control traits. In fact, the genetic epistasis in the inheritance of quality traits not common but for quantitative traits are common.

As the number of genes controlling a trait increases it is reasonable to assume that the number of factors which has reaction increased. For both traits amount of dominance effect [h] and interaction of dominance -dominance effects [I] is less than additive effect[d] [20-24].

				Genetic components			
Trait	M	[d]	[h]	[i]	[j]	[l]	χ^2
IT	26.13 ± 1. 3**	20.81 ± 0.1**	8.29 ± 3.02**	-	-5.96 ± 0.5**	-7.65 ± 2.01**	0.80 ns
LP	74.13 ± .0.9**	-23.22 ± 0.4**	-6.74 ± 1.1**	-	4.09 ± 0.8**	6.63 ± 1.3**	0.89ns

Table 6: Estimates of genetic components estimates for infection type and latent period in six generations.

*,**: Significant at 5% and 1% probability level, respectively.
ns: Not significant.

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Conclusion

In this study the inheritance of stripe rust resistance, caused by *Puccinia striiformis* f. sp. *tritici*, genetic components of resistance in wheat was estimated/The results has shown that additive; dominance and epistasis (especially additive × dominance and dominance × dominance) gene actions have a significant role in control of the traits. The narrow-sense heritability was in an average range for infection type and was low for latent period. The number of segregating genes was estimated 1 - 2 for infection type and 1 - 3 for latent period. Information of these analyses is useful for breeding methods of wheat rust resistance selection.

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