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Genetic Stability of Wheat Stem Rust Resistance Genes under Egyptian Field Conditions

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Forty six stem rust resistance monogenic lines (*Sr's*) were evaluated for their resistance stability to wheat stem rust at five different environmentally diverse locations *i.e.,* Itay El-Baroud, Minia, Sakha, Sharkyia and Beni Suef, during three successive growing seasons *i.e.,* 2015/16, 2016/17 and 2017/18 under Egyptian field conditions. According to the stem rust response of the tested monogenic lines, *Sr* genes were divided into three main categories. The first category included the highest effective stem rust resistance genes; *Sr*2, *Sr*24, *Sr*32, *Sr*33, *Sr*36, *Sr*38 and *Sr*39. The second category contained *Sr* genes that showed different disease reaction (susceptible and/or resistance). While, the third category included ineffective Sr genes (susceptible) under different environments in three growing seasons. Due to the highly significance interaction between genotypes and environments, stability analysis was carried out in this study. Stability parameters show clearly that the three stem rust monogenic lines; *Sr*14, *Sr*17 and *Sr*37 exhibited high stability to stem rust under a wide range of environmental conditions. Eventually, the current study aimed to facilitate important information that helps plant breeders to make suitable decision in the use of these genotypes (monogenic lines) as a good source of resistance in wheat breeding program for rust resistance, especially stem rust.

Keywords: AMMI Model, Stem rust, Stability of resistance, Monogenic lines, and Wheat.

Wheat stem rust caused by *Puccinia graminis* f.sp. *tritici*, is the most potentially destructive wheat disease that, seriously threaten grain yield production, worldwide (Kokhmetova *et al*., 2011). Breeding for wheat stem rust resistance is the most efficient, economic and environmental friendly approach to control stem rust (Line and Chen 1995). In many wheat producing areas, the disease has been effectively controlled through the growing of wheat cultivars having sustainable stem rust resistance.

However, there are two main types of resistance which have been identified in wheat to stem rust *i.e.,* qualitative (race-specific), and quantitative (race-non-specific) resistance. Deployment of race specific resistance genes ensures effective and complete protection against the disease (Shah *et al*., 2010). Conversely, race-non-specific resistance is mainly polygenic and quantitatively inherited. This type of resistance has also been described as slow-rusting or partial resistance (Parlevliet 1979) and is known to be long-lasting and more durable (Herrera-Fossel *et al.,* 2007).

In 1999, a new race (Ug99) of *Puccinia graminis* f.sp. *tritici* was identified in Uganda, which has unique virulent to stem rust resistance gene; *Sr* 31 (Pretorius *et al*., 2000). This aggressive race was designated as TTKSK using the North American nomenclature system (Roelfs and Martens 1988 and Jin *et al*., 2008). This race has been rapidly spread to more than 13 countries worldwide and more recently in Egypt (Singh *et al*., 2015 and Patpour *et al*., 2016). The original race TTKSK and it's variants, were virulent to stem rust resistance genes; *Sr*5, *Sr*6, *Sr*7b, *Sr*8a, *Sr*8b, *Sr*9b, *Sr*9e, *Sr*9h, *Sr*9g, *Sr*11, *Sr*15, *Sr*17, *Sr*24, *Sr*30, *Sr*36, *Sr*31, *Sr*38 and *Sr*Tmp (Jin *et al*., 2008; Pretorius *et al*., 2012; Rouse *et al*., 2014 and Patpour *et al*., 2016).

At present, more than eighty stem rust resistance genes have been described (McIntosh *et al*., 2017), Some of them were resistant at adult plant stage so called adult plant resistance genes (APR). The effectiveness of these resistance genes depend to a large extend, in an interaction between it's genetic make-up and environmental conditions. The dramatic change in gene behavior is mainly due to the dynamic nature of the target pathogen which led to the appearance of new virulent pathotypes, so the resistance of a variety is not a constant trait. Where, any wheat variety carrying a single resistance gene with major effect may become susceptible within a short time (Kolmer *et al.*, 2008).

Different methods of stability analysis have been previously described in some studies (Becker and Léon, 1988; Lin *et al.*, 1986 and Li *et al.*, 2003). The concept "stability" is useful for many agronomic traits including disease resistance. Out of the stability methods previously used, AMMI analysis method was used in this study to analyse genotype versus environment interaction and to detect environmental stability for each genotype under different environmental conditions (Gauch 1992).

The present study aimed to evaluate and characterize adult plant resistance (APR) to stem rust in 46 monogenic lines (Sr's). Stability analysis of resistance to these tested lines at adult plant stage at different environments under Egyptian filed conditions was conducted in this study.

Materials and Methods

Evaluation of wheat stem rust monogenic lines at adult plant stage under field conditions:

Forty-six stem rust resistance genes (Sr's) were used in this study (Table 1). The experiments were carried out under field conditions at different five locations in Egypt *i.e.,* Itay El-Baroud, Minia, Sakha, Sharkyia and Beni Suef, during three successive growing seasons *i.e.,* 2015/16, 2016/17 and 2017/18. These monogenic lines were planted in randomized complete block design (RCBD) with three replicates, with each three rows (3 m long and 30 cm apart). Each row was sown with 5 g of given tested monogenic line. The experiment was surrounded by spreader area planted with mixtures of the highly susceptible varieties; Morocco, Thatcher and *Triticum spelta sahariensis*. The spreader plants were artificially inoculated using a

mixture of urediniospores of the prevalent stem rust races (BBBGC, JTTTC, KTLRC and TTKTS) mixed with talcum powder at a rate of 1: 20 (v:v) (spores: talcum powder), according to the method described by Tervet and Cassell (1951), during late tillering and late elongating stage. The urediniospores of stem rust were received from Wheat Dis. Res. Dept., Plant Pathol. Res. Inst., Agric. Res. Center, Egypt. To maintain crop stand/vigor normal agronomic practices including recommended fertilization dose and irrigation schedule were followed.

Disease assessment:

Disease responses to stem rust pathogen was recorded after heading stage as a disease severity (%) expresses in the tested genotypes using modified Cobb's scale, adopted by Peterson *et al.* (1948). Infection types *i.e.,* highly resistant (0), resistant (R), moderately resistant (MR), moderately susceptible (MS) and susceptible (S) were recorded according to Roelfs *et al*. (1992).

Stability analysis:

Disease severity data for each genotype under study were subjected to analysis of variance (ANOVA). Bartlett test was used to determine the homogeneity of error variances between environments to determine the validity of the combined analysis of variance on the data as described by Gomez and Gomez (1984). Combined analysis of variance was done using the mean data of each location in each year to create data means for the stability analysis model. Additive main effect and multiplicative interaction (AMMI) analyse used to analyse the genotype x environment interaction, and to define stability for each genotype according to Gauch (1992). This approach used the analysis of variance (ANOVA) to study the main effects of genotypes and environments and the principal component analysis (PCA) for the residual multiplicative interaction between genotypes and environments forming different interactive principal component axes (PCA). AMMI analysis was presented in the form of biplot, which allowing to visualize clear relationships between the Eigen values of IPCA, means of environments and genotypes. Also, both genotypes and environments were occurred on the same scatter plot (Gauch and Zobel 1996).

Table 1. The tested wheat stem rust monogenic lines (46 Sr's) carrying single gene for resistance

No.	Sr. gene	Chromosome. location	Original source	Tester
1	*Sr*2	3BS	*T. turgidum (Yaroslav emmer*)	CnS(Hope3B)
2	*Sr*5	6DS	Reliance	ISr 5-Ra
3	*Sr* 6	2DS	Red Egyptian	ISr 6-Ra
4	*Sr*7a	4BL	Kenya117A	Line G sel
5	*Sr*7b	5BL	Kenya B	ISr 7b-Ra
6	*Sr*8a	6AS	Red Egyptian	ISr 8-Ra
7	*Sr*8b	2BL	Red Egyptian	ISr 8b-Ra
8	*Sr*9a	2BL	Red Egyptian	ISr9a-Ra
9	*Sr*9b	2BL	Red Egyptian	W2691Sr9b
10	*Sr*9d	2BL	*Kenya117A*	ISr9d Ra
11	*Sr*9e	2BL	*T. turgidum (Yaroslav emmer)*	Vernstein
12	*Sr*9g	2BL	*T. turgidum*	CnSSr9g
13	*Sr*10	2B	Egypt NA95	W2691Sr10
14	*Sr*11	6BL	Isr H-Ra	ISrl1-Ra
15	*Sr*12	3BS	*T. turgidum (Iumillo durum)*	ISrl1-Ra
16	*S* 13	6AL	*T. turgidum (Kaphli emmer)*	W2691 Sr 13
17	*Sr*14	1BL	*T. turgidum (Kaphli emmer)*	Line A sel
18	*Sr*15	7AL	Norka	W2691 Sr 15
19	*Sr*16	2BL	Thatcher	ISr 16-Ra
20	*Sr*17	7BL	*T. turgidum*? (Yaroslav emmer)	CS (Hope7B)
21	*Sr*18	1D	Marquis	
22	*Sr*19	2BL	*Marquis*	LCSr19Mq
23	*Sr*20	2AL	*T. monococcum*	LC
24	*Sr*21	2BS	*Exchange*	Einkorn
25	*Sr*22	3DL	*Thinopyron ponticum*	SwSr22T.B.
26	*Sr*23	7DL	*Thinopyron ponticum*	BtSr24Agt
27	*Sr*24	2B	Egypt NA95	LCSr25Ars
28	*Sr*25	6BL	Isr H-Ra	ISrl1-Ra
29	*Sr*26	6AL	Lee	CnSSr-9g
30	*Sr*27	3A	Secalis cereale (Imperial rye)	W2691Sr27
31	*Sr*28	2BL	Kota	W2691Sr28Kt
32	*Sr*29	6DL	Etiole de Choisy	PusaSr29Edch
33	*Sr*30	5DL	Webster	Bt sr 30 Wst
34	*Sr*31	1BL	*Secalis cereale (Imperial rye)*	Line Esr 31 Kvz
35	*Sr*32	2A, 2B	*T. speltoides*	ER 5155
36	*Sr*33	1DL	*T. tauschii*	Tetracan Thatch *T. tauschii*
37	*Sr*34	2A, 2B	*T. comosa*	Compare
38	*Sr*35	3AL	*T. monococcum*	Mg (2) 5xG 2919
39	*Sr*36	2BS	*T. timopheevi*	W2691SrTt-1
40	*Sr*37	4BL	*T. timopheevi*	W2691SrTt-2
41	*Sr*38	-	*VPM-1*	13 Pullman, W
42	*Sr*39	2B	*Aegilops speltoides*	W2691Sr39
43	*Sr*40	2BS	*T. araraticum*	RL6087
44	*Sr*Tmp	4B	*Triumph 64*	Triumph 64
45	*Sr*Wld-1	-	Waldron	BF Sr WLd WLd
46	*Sr* McN	-	*McNair 701*	Griffey2011

Results

Evaluation of tested wheat monogenic lines to stem rust disease at adult stage under field conditions, during three successive seasons:

Forty six stem rust resistance genes (Sr,s) were evaluated against stem rust infection, to study the efficacy and stability of these monogenic lines under Egyptian field conditions at five different locations i.e., Itay El-Baroud, Minia, Sakha, Sharkyia and Beni Suef, during three successive growing seasons; 2015/16, 2016/17 and 2017/18 (Tables 2, 3 and 4).

Data in Tables 2, 3 and 4 indicate that the tested monogenic lines could be divided into the three main groups according to their performance under field conditions at all locations and three years under study; Group I: Effective genes (resistant): This group includes seven Sr genes with high degree or level of resistance to stem rust at all five locations and three years of the study. These genes are Sr2, Sr24, Sr32, Sr33, Sr36, Sr38 and Sr39. The genes in this group showed the lowest stem rust response which ranged from 0 to 10MR. Group II: this group includes Sr genes that showed different disease response (susceptible and/or resistant): This group mainly includes 15 Sr genes with high degree of resistance to stem rust at some locations, as well as susceptible reaction at other locations. These genes are Sr11, Sr12, Sr13, Sr15, Sr23, Sr25, Sr26, Sr27, Sr28, Sr29, Sr31, Sr34, Sr37, Sr40 and SrWld-1. The tested genes present in this group showed a broad spectrum range of rust reaction (ranged from 0 to 60S). Group III: Ineffective Sr genes to stem rust at adult plant stage (susceptible), such as Sr5, Sr6, Sr7a, Sr7b, Sr8a, Sr8b, Sr9a, Sr9b, Sr9d, Sr9e, Sr9g, Sr10, Sr14, Sr16, Sr17, Sr18, Sr19, Sr20, Sr21, Sr22, Sr30, Sr35, SrTmp and SrMcN. All of these genes showed susceptible rust response that reached to 90S at all locations and years under study.

Stability of stem rust resistance in the tested Sr genes under different environmental conditions:

The combined analysis of variance presented in Table 6 indicate that the main effects of both environments (E) and genotypes (G) and their interactions (G×E) were highly significant for stem rust resistance genes, that indicating to the presence of a wide variation among the tested stem rust resistance genes (Sr's) and a relatively high diversity of growing conditions at different environments. The significant effects of genotype × environment (GE) interaction reflected the differential response of stem rust resistance genes in the various environments. This demonstrates that GE interaction was highly significant and had remarkable effect on genotypic performance in different environments. As GE was significant, it was possible to proceed and calculate phenotypic stability using AMMI model.

Table 2. Stem rust response of 46 wheat monogenic lines to stem rust at five locations under Egyptian field conditions during 2015/16 growing season

Sr gene	Location / Rust response[a]				
	Itay El-Baroud	Minia	Sakha	Sharkyia	Beni Suef
Group I: Effective genes					
*Sr*2	0	0	0	10 MR	0
*Sr*24	0	0	0	0	0
*Sr*32	Tr S	0	Tr R	5 MR	0
*Sr*33	5 MR	0	0	5 MR	0
*Sr*36	Tr R	5 MR	5 R	5 MR	5 R
*Sr*38	0	Tr MR	0	Tr MR	0
*Sr*39	0	0	Tr R	5 MR	0
Group II: Differentiated genes					
*Sr*11	0	10 S	20 S	0	30 S
*Sr*12	10 S	0	10 S	5 S	0
*Sr*13	20 S	Tr S	10 MR	30 S	Tr S
*Sr*15	5 S	10 MR	5 MR	30 S	0
*Sr*23	Tr S	10 MR	10 S	Tr S	5 S
*Sr*25	Tr S	20 MR	Tr MS	5 MR	0
*Sr*26	5 S	10 R	0	0	Tr MS
*Sr*27	Tr MR	0	5 MS	0	0
*Sr*28	Tr MR	0	30S	Tr S	Tr MS
*Sr*29	Tr MS	0	40 S	10 S	0
*Sr*31	0	5 S	0	Tr R	30 MS
*Sr*34	Tr S	0	30 S	Tr MR	0
*Sr*37	10 S	0	30 S	10 S	Tr R
*Sr*40	0	0	5 MS	5 MS	Tr MR
*Sr*Wld-1	5 MR	10 MS	Tr MR	5 S	5 MR
Group III: Ineffective genes					
*Sr*5	10 S	30 S	20 S	30 S	40S
*Sr*6	10 S	10 S	30 S	50 S	30 S
*Sr*7a	Tr S	5 S	5 S	20 S	Tr S
*Sr*7b	5 S	30 S	20 S	20 S	10 S
*Sr*8a	20 S	30 S	40 S	5 S	20 S
*Sr*8b	20 S	30 S	30 S	60 S	30 S
*Sr*9a	Tr S	5 S	30 S	40 S	30 S
*Sr*9b	10 S	10 S	30 S	80 S	5 S
*Sr*9d	Tr S	40 S	20 S	30 S	40 S
*Sr*9e	Tr S	10 S	Tr S	20 S	Tr S
*Sr*9g	Tr S	10 S	10 MS	10 S	Tr MS
*Sr*10	20 S	10 S	10 S	60 S	40 S
*Sr*14	5 S	20 S	Tr S	40 S	Tr S
*Sr*16	Tr S	Tr S	5 MS	5 S	Tr S
*Sr*17	10 MS	20 S	10 S	20 S	Tr S
*Sr*18	Tr MS	5 MS	20 S	50 S	Tr S
*Sr*19	40 S	20 S	40 S	80 S	Tr S
*Sr*20	Tr S	5 S	50 S	20 S	20 S
*Sr*21	50 S	10 S	30 S	5 S	20 S
*Sr*22	Tr S	5 S	10 S	20 S	Tr S
*Sr*30	Tr MS	10 MS	20 S	40 S	20 S
*Sr*35	10 S	10 S	10 S	50 S	30 S
*Sr*Tmp	20 S	0	50 S	40 S	10 MS
*Sr*McN	60 S	40 S	50 S	70 S	50 S

[a]Rust response includes two components: disease severity (%) based on modified Cobb's scale (Peterson *et al*., 1948), infection type based on the scale described by Roelfs *et al*. (1992), where R = resistant, MR = moderately resistant, MS = moderately susceptible and S = susceptible.

Table 3. Stem rust response of 46 wheat monogenic lines to stem rust at five locations under Egyptian field conditions during 2016/17 growing season

Sr gene	Location / Rust response[a]				
	Itay El-Baroud	Minia	Sakha	Sharkyia	Beni Suef
Group I: Effective genes					
*Sr*2	Tr MR	0	0	5 MR	0
*Sr*24	0	0	0	Tr MR	0
*Sr*32	0	0	5 MR	Tr MR	10 R
*Sr*33	0	Tr MR	0	Tr MR	0
*Sr*36	Tr MR	10 MR	10 MR	Tr MR	Tr R
*Sr*38	0	Tr MR	0	5 MR	0
*Sr*39	0	0	Tr R	Tr MR	0
Group II: Differentiated genes					
*Sr*11	5 S	5 S	20 S	Tr MS	10 S
*Sr*12	10 S	Tr MR	10 S	10 S	20 R
*Sr*13	Tr S	Tr S	10 MR	10 S	10 R
*Sr*15	5 S	5 S	5 S	10 S	10 MR
*Sr*23	Tr MR	Tr S	30 S	5 S	0
*Sr*25	10 S	10 MR	Tr MS	10 MR	Tr MS
*Sr*26	0	Tr MS	Tr MR	5 MS	10 MR
*Sr*27	Tr MR	5 MR	0	5 S	0
*Sr*28	Tr S	0	40 S	5 S	0
*Sr*29	Tr S	Tr S	50 S	10 S	0
*Sr*31	0	5 S	Tr MR	0	5 S
*Sr*34	Tr S	0	40 S	5 MR	0
*Sr*37	5 S	0	40 S	10 S	10 R
*Sr*40	Tr R	0	10 MS	5 S	0
*Sr*Wld-1	5 MR	5 S	5 MR	5 S	0
Group III: Ineffective genes					
*Sr*5	Tr S	5 S	40 S	30 S	20 S
*Sr*6	5 S	20 S	30 S	40 S	20 S
*Sr*7a	5 S	5 S	20 S	30 S	10 S
*Sr*7b	Tr S	30 S	20 S	30 S	5 S
*Sr*8a	5 S	20 S	30 S	20 S	30MS
*Sr*8b	5 S	30 S	40 S	60 S	20 S
*Sr*9a	5 S	40 S	50 S	50 S	Tr MS
*Sr*9b	10 S	20 S	30 S	70 S	Tr MS
*Sr*9d	20 S	30 S	20 S	20 S	40 MS
*Sr*9e	10 S	10 S	Tr S	30 S	20 MS
S 9g	20 S	30 S	20 MS	10 S	5 MS
*Sr*10	10 S	10 S	10 S	70 S	5 MS
*Sr*14	Tr S	10 S	5 S	50 S	40 S
*Sr*16	5 S	5 S	20 S	20 S	10 MS
*Sr*17	Tr S	10 S	10 S	30 S	20 S
*Sr*18	Tr S	20 S	30 S	60 S	40 S
*Sr*19	30 S	60 S	50 S	70 S	5 S
*Sr*20	5 S	30 S	60 S	20 S	10 S
*Sr*21	20 S	5 S	10 S	10 S	Tr S
*Sr*22	Tr S	20 S	20 S	30 S	20 MS
*Sr*30	Tr S	Tr S	30 S	40 S	5 S
*Sr*35	Tr S	40 S	20 S	70 S	5S
*Sr*Tmp	5 S	20 S	70 S	60 S	Tr MS
*Sr*McN	50 S	50 S	70 S	50 S	70 S

[a]Rust response includes two components: disease severity (%) based on modified Cobb's scale (Peterson *et al*., 1948), infection type based on the scale described by Roelfs *et al*. (1992), where R = resistant, MR = moderately resistant, MS = moderately susceptible and S = susceptible.

Table 4. Stem rust response of 46 wheat monogenic lines to stem rust at five locations under Egyptian field conditions during 2017/18 growing season

Sr gene	Location / Rust response[a]				
	Itay El-Baroud	Minia	Sakha	Sharkyia	Beni Suef
Group I: Effective genes					
*Sr*2	0	0	0	10 MR	5 MR
*Sr*24	Tr R	5 R	0	0	0
*Sr*32	Tr MR	0	5 MR	5 MR	Tr MR
*Sr*33	Tr MR	Tr R	0	5 MR	0
*Sr*36	0	10 MR	0	5 MR	10 R
*Sr*38	0	Tr MR	0	5 MR	0
*Sr*39	0	0	5 R	5 MR	0
Group II: Differentiated genes					
*Sr*11	Tr S	10 S	10 MR	5 MS	Tr MR
*Sr*12	Tr S	Tr MR	20 MS	10 S	Tr MS
*Sr*13	10 MS	5 S	20 MR	20 S	Tr MR
*Sr*15	5 MR	5 S	10 S	20 S	5 R
*Sr*23	Tr MS	Tr S	20 S	5 S	Tr MS
*Sr*25	5 S	0	5 MS	5 MS	10 MS
*Sr*26	Tr MS	0	0	5 MR	20 MS
*Sr*27	10 MR	0	5 MR	10 MS	0
*Sr*28	Tr S	0	40 S	Tr S	5 S
*Sr*29	5 S	0	60 S	5 S	0
*Sr*31	0	Tr S	5 MS	5 MR	Tr S
*Sr*34	10 S	0	40 S	10 MR	Tr S
*Sr*37	20 S	0	50 S	5 S	10 R
*Sr*40	0	0	Tr MS	10 MS	5 MR
*Sr*Wld-1	10 MR	5 S	5 S	10 S	Tr S
Group III: Ineffective genes					
*Sr*5	Tr S	30 S	10 S	40 S	5MS
*Sr*6	5 S	10 S	20 S	60 S	5MS
*Sr*7a	Tr S	30 S	40 S	70 S	5 S
*Sr*7b	10 S	5 S	10 S	30 S	20 S
*Sr*8a	40 S	50 S	40 S	20 S	40 S
*Sr*8b	5 S	40 S	50 S	80 S	10 S
Sr 9a	5 S	40 S	50 S	60 S	10 MS
*Sr*9b	40 S	50 S	40 S	70 S	40 S
Sr 9d	30 S	10 S	30 S	40 S	10 S
*Sr*9e	5 S	30 S	10 S	50 S	Tr S
*Sr*9g	10 S	30 S	20 S	20 S	30 S
*Sr*10	Tr S	70 S	20 S	90 S	5 S
*Sr*14	Tr S	40 S	10 S	30 S	10 S
*Sr*16	5 S	30 S	20 S	10 S	Tr MS
*Sr*17	5 S	40 S	30 S	40 S	10 S
*Sr*18	Tr S	30 S	20 S	50 S	40 S
*Sr*19	40 S	50 S	60 S	80 S	40 S
*Sr*20	Tr S	40 S	60 S	10 S	5 S
*Sr*21	20 S	50 S	40 S	5 S	20 S
*Sr*22	Tr MS	30 S	10 S	20 S	20 MS
*Sr*30	5 S	20 S	30 S	30 S	10 S
*Sr*35	5 S	60 S	30 S	80 S	10 S
*Sr*Tmp	10 S	50 S	80 S	70 S	10 MS
*Sr*McN	50 S	70 S	60 S	60 S	70 S

[a]Rust response includes two components: disease severity (%) based on modified Cobb,s scale (Peterson et al., 1948), infection type based on the scale described by Roelfs et al. (1992), where R = resistant, MR = moderately resistant, MS = moderately susceptible and S = susceptible.

Table 5. Combined analysis of variance and AMMI stability analysis for the studied stem rust monogenic lines under five environments during three growing seasons

S.O.V	D.F.	Mean sum of squares (M.S.)	Variance ratio	
Blocks	2	653.021	0.190%TSS	
Years (Y)	2	1291.745**	0.376%TSS	
Environments (E)	4	26192.004**	15.267%TSS	
Genotypes (G)	45	5798.643**	38.023%TSS	
Y x E	8	539.918**	0.629%TSS	
Y x G	90	180.266**	2.364%TSS	
G x E	180	1170.973**	30.714%TSS	
Y x E x G	360	130.326**	6.837%TSS	
IPCA1	48	1.456	0.010%TSS	69.911%GESS
IPCA2	46	0.381	0.003%TSS	17.545%GESS
Residual	86	0.146	0.002%TSS	12.635%GESS
Error	1378	27.887		

* and ** Significant at 0.05 and 0.01 probability levels, respectively.

The AMMI analysis of stability for 46 stem rust resistance genes in the five environments under study show that only 15.267% of the total sum of squares was attributable to environmental effects, while 38.023% to genotypic effects and 30.714% to the interaction between genotypes and environment effects (GE) as clear in Table (5). A large sum of square (SS) for G indicates that the stem rust resistance genes were diverse with high differences among the means. Also, most of the variations in the level of their disease reactions were due to the genetic structure of the tested genotypes. The small proportion of SS for (E) indicates that the difference among the environmental conditions was not very high. The magnitude of G x E SS was smaller than that for the SS for (G). This result indicates that the differences in the disease response of the tested stem rust resistance genes across different environments were not affected by the slight changes in environment conditions.

Also, data in Table 5 indicate that the first two principle components (PC1 and PC2) of the GE interaction explained 87.456 of the sum of squares (GESS). The first interaction principal component (PCA1) accounted for 69.911% of the GESS. While, PCA2 explained further 17.545% of the GESS. The mean sum of squares (MS) for both PCA1 and PCA2 was significant, at $P = 0.01$ level and cumulatively contributed to 87.456% of the total GESS. Thus, the interaction of the 46 stem rust resistance genes across five environments was best predictable by the first two principal components. Genotypes with PCA1 scores close to zero have small interactions and hence show wider adaptation or high stability under the tested environments. On the other hand, a large host genotypic PCA1 score have high interactions and reflects more specific adaptation to the environments with PCA1 values of the same sign (either positive or negative).

Data in Table 6 and Fig.1 show that, the first two principal components for the tested stem rust resistance genes and mean of two PCs. The PC scores of a genotype in the AMMI analysis are an indication of the stability or adaptation over environments. The lowest PC1 value was observed for each of the three stem rust resistance genes; *Sr*14, *Sr*17 and *Sr*37, as they showed PCA1 close to zero (0.7, 1.1 and 0.8, respectively). Therefore, they were recognized as the most stable genes across the five environments under study. These results indicate that the AMMI stability parameters helped breeders to have an overall picture in the behaviour of the stem rust resistance genes under different environmental conditions.

The two environments E3 and E5 had longer vector than vector for each of the other three environments under study *i.e*., E1, E2 and E4 (Fig. 1). Thus, these two environments were the best discriminative environments for investigating stem rust resistance genes. On the other hand, the other three environments with shorter vectors (E1, E2 and E4) are not discriminative ones for the stem rust resistance genes. Acute angle between vectors of Minia (E1), Itay El-Baroud (E2), Sakha (E3) and Beni Suef (E4) environments indicate that these two environments were similar for gene determination. Yet, environments with obtuse angle were different *i.e*., Sakha (E3) and Sharkyia (E5). The biplot analysis visualizes the best genotype for each environment. Stem rust resistance genes; *Sr*7a, *Sr*10, *Sr*18 and *Sr*25 showed a degree of positive relationship to Sharkyia (E5). While, Stem rust resistance genes; *Sr*8a, *Sr*9a, *Sr*20 and *Sr*21 are the best relationship for environment Sakha (E3).

Table 6. Reaction of tested stem rust resistance genes (46 Sr,s) under five environments, during three growing seasons and AMMI stability values

Genotypes (Sr's)	E1	E2	E3	E4	E5	Mean	AMMI stability values	
							PCA1	PCA2
*Sr*5	6.2	5.0	21.1	19.1	27.7	15.8	-9.2	5.5
*Sr*6	3.9	5.9	26.7	15.3	38.2	18.0	-17.9	7.1
*Sr*7a	8.4	3.3	19.1	5.7	32.9	13.9	-7.3	8.2
S 7b	8.7	4.9	16.1	12.1	24.2	13.2	-2.3	6.1
*Sr*8a	27.8	19.8	37.8	25.1	20.0	26.1	-24.6	-11.7
*Sr*8b	17.8	7.7	38.9	17.2	52.1	26.7	-38.5	6.5
*Sr*9a	4.7	4.0	43.3	9.8	37.8	19.9	-25.3	-6.7
*Sr*9b	8.2	18.3	34.4	11.6	60.8	26.7	-40.6	14.3
*Sr*9d	15.1	13.4	22.2	22.0	22.5	19.1	-11.6	1.6
*Sr*9e	4.1	4.9	4.2	4.0	28.0	9.0	5.7	16.4
*Sr*9g	4.5	7.1	14.4	9.2	15.4	10.1	5.8	1.5
*Sr*10	9.3	7.9	14.2	13.1	54.6	19.8	-22.6	26.5
*Sr*11	1.7	2.2	14.4	12.1	1.9	6.5	15.8	-6.3
*Sr*12	3.7	5.4	10.0	1.6	6.2	5.4	17.4	-1.9
*Sr*13	2.0	7.8	4.7	1.6	13.5	5.9	15.4	6.6
*Sr*14	3.6	3.2	4.6	15.9	30.4	11.5	0.7	19.4
*Sr*15	5.8	3.0	4.6	1.6	9.5	4.9	18.6	4.3
*Sr*16	8.8	3.9	12.2	2.6	10.2	7.5	12.4	-1.0
*Sr*17	11.4	4.0	13.3	7.9	22.9	11.9	1.1	6.8
*Sr*18	3.1	2.2	21.1	25.1	47.5	19.8	-22.7	18.7
*Sr*19	19.8	32.2	50.0	12.9	64.2	35.8	-58.7	4.2

Continued

*Sr*20	5.4	2.8	56.7	10.0	11.7	17.3	-15.8	-33.2
*Sr*21	14.1	25.6	26.1	11.8	9.6	17.4	-5.2	-11.2
*Sr*22	2.7	2.9	11.7	6.0	17.0	8.0	8.8	4.2
*Sr*23	1.4	1.8	20.6	2.2	4.7	6.1	13.5	-10.8
*Sr*24	0.0	0.1	0.0	0.3	0.7	0.2	29.6	2.3
*Sr*25	1.4	4.6	1.8	2.8	4.0	2.9	24.0	3.2
*Sr*26	0.4	2.0	0.2	4.3	3.4	2.0	25.8	4.4
*Sr*27	0.7	1.4	3.6	0.0	3.1	1.8	25.5	0.9
*Sr*28	2.1	1.6	35.6	1.9	3.6	9.0	5.5	-23.1
*Sr*29	4.6	3.1	50.0	0.0	5.3	12.6	-4.4	-33.5
*Sr*30	4.1	2.6	26.7	10.6	29.6	14.7	-9.9	1.1
*Sr*31	0.0	0.0	1.2	11.8	1.2	2.8	25.0	3.4
*Sr*32	0.4	1.3	3.7	1.5	1.9	1.7	25.8	0.3
*Sr*33	0.0	0.9	0.0	0.0	1.3	0.4	29.1	2.6
*Sr*34	1.9	5.2	36.7	0.7	2.1	9.3	5.3	-25.2
*Sr*35	5.4	4.8	18.3	12.8	52.1	18.7	-21.5	21.8
*Sr*36	0.0	0.3	1.6	1.5	1.5	1.0	27.7	1.7
*Sr*37	3.3	9.6	40.0	1.3	5.9	12.0	-0.8	-25.4
*Sr*38	0.0	0.0	0.0	0.0	1.1	0.2	29.4	2.5
*Sr*39	0.0	0.0	0.9	0.0	1.2	0.4	28.8	1.9
*Sr*40	4.9	0.1	4.1	0.8	4.0	2.8	23.7	1.2
*Sr*Tmp	8.8	9.0	66.7	5.4	44.2	26.8	-44.2	-21.6
SrWld-1	0.0	2.3	2.8	1.6	5.9	2.5	23.5	3.5
*Sr*2	0.6	0.1	0.0	0.4	2.5	0.7	28.2	3.4
*Sr*McN	37.8	50.0	63.3	62.2	61.7	55.0	-89.0	-0.6
Overall mean	6.1	6.5	19.6	8.6	19.6	12.0		
Mina (E1)							-0.79	-0.01
Itay El-Baroud E2)							-0.76	-0.03
Sakha (E3)							-0.83	-0.55
Beni Suef (E4)							-0.76	0.19
Sharkyia (E5)							-0.89	0.42

E1, E2, E3, E4 and E5 = 5 different environments, PCA = Principle component axes and AMM = Additive main effect and multiplicative interaction analysis.

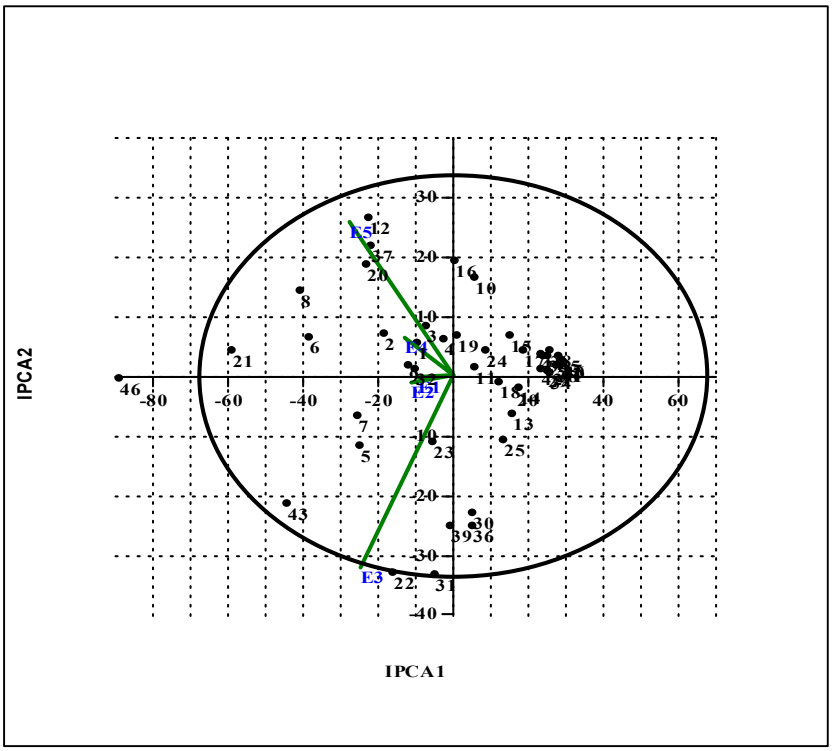

Fig. 1. AMMI model of stability analysis for 46 wheat stem rust monogenic lines evaluated at five different locations, during 2015/16, 2016/17 and 2017/18 growing seasons in Egypt.

Discussion

Two types of host-genetic resistance were previously identified and characterized in wheat genotypes against rust diseases *i.e.,* qualitative and quantitative resistance. Resistance to rust diseases especially stem rust can be qualitatively inherited, as it was governed by single gene or oligo genes. In this case, the resistance can be easily measured by infection types that produce resistance or susceptible host reaction (Abd-Alla and Hermsen, 1971 and Parlevliet and Zadoks, 1976). In contrast, quantitative resistance is usually governed by multiple minor genes with additive effect (Parlevliet 1978). Accumulation or pyramiding of minor genes each together in a particular genotype or one cultivar gives resistance more depth and more durable, which can be accurately measured by different quantitative disease parameters, such as disease severity and AUDPC etc. In both cases, stability analysis for disease

response is required to give more detailed impression or information and through lights on the genetic nature of such resistance. However, few or little studies have been previously carried out in Egypt in this concern. Therefore, available data to describe the stability of resistance genes to wheat stem rust are not sufficient.

The obtained results in this study indicated that, seven stem rust resistance genes; *Sr*2, *Sr*24, *Sr*32, *Sr*33, *Sr*36, *Sr*38 and *Sr*39, proved to be highly effective, where they showed an adequate level of resistance at different environments *i.e.,* Itay El-Baroud, Minia, Sakha, Sharkyia and Beni Suef, during 2015/16, 2016/17 and 2017/18 growing seasons. The stem rust resistance gene; *Sr*2 was determined on the short arm of chromosome 3B of wheat and located to hexaploid wheat in the 1920s from tetraploid emmer wheat cultivar 'Yaroslav'. This gene considered to be slow-rusting gene or adult plant resistance (APR) (Hare and McIntosh 1979 and McIntosh *et al.,* 1995). It was detected in several Kenyan varieties, including Kenya Plume and CIMMYT varieties; Pavon 76, Juchi 2000 and Kritati. These wheat varieties showed high and good level of stem rust resistance (Singh and McIntosh 1986). In addation, *Sr2* is closely linked to a minor genes; *Yr30* that conferring yellow rust resistance (Singh *et al.,* 2000).

Meanwhile, stem rust resistance gene; *Sr*24 is completely associated with leaf rust resistance gene; *Lr24*. It has been widely used in wheat breeding programs, worldwide. Since, it was introgressed into many wheat genotypes (McIntosh *et al.,* 1995). *Sr24* gene was ineffective to some variants in the lineage of Ug99, but it is effective to the new races; TKTTF, TTTTF, and many *Puccinia graminis* races in China (Bhattacharya 2017). As mentioned before in the previous studies, *Sr32* was derived from *Aegilops speltoides* and translocate to hexaploid wheat. It was effective against the original race; Ug99 and seven races out of the variants of Ug99 lineage, as well as it showed efficacy against some related races of *Puccinia graminis* f.sp. *tritici* (Jin *et al.,* 2008 and Abou-Zeid *et al.* 2014). However, this resistance gene has not been preferable for most of breeders, because it is tightly linked with some undesirable genes having deleterious effects an grain yield or present in genetic backgrounds unsuitable for breeding (McIntosh *et al.,* 1995 and Friebe *et al.,* 1996).

Stem rust resistance gene; *Sr*38 has been originated from *T. ventricosum*, and it is widely used due to its association with the stripe rust resistance gene; *Yr17* and the leaf rust resistance gene; *Lr37,* thus it confers multiple resistance to all the three rust diseases (Delibes *et al*., 1993). Although, *Sr38* has been susceptible to UG99, it can be displayed high level of resistance to all *Puccinia graminis* races detected in China (Cao *et al.,* 2007). Therefore, in China, it should be used in combination with other genes resistant to Ug99 through gene pyramiding. Another stem rust resistance gene; *Sr*39 was transferred from goatgrass (*Aegilops speltoides*) to wheat cultivar; Marquis and the original wheat line, carried both *Sr39* and leaf rust resistance gene; *Lr35* (Kerber and Dyck 1990; Friebe *et al.,* 1994 and Yu *et al.,* 2011). As indicated in the present study *Sr39* proved to be the most effective gene over the tested environments and during the years of the study. This could be facilitated the use of this gene as a good source of resistance in wheat breeding program.

The combined analysis of variance for the 46 wheat stem rust resistance genes (Sr's) which were evaluated for stem rust resistance at different environments under study (five locations and three years). Most of the tested wheat genotypes (monogenic lines) showed different levels of stem rust resistance. Since, they displayed different percentages of final rust severity (FRS %), during the five locations and three years of the study, as they affected by the slight changes in environmental conditions, in each location and growing season (Abou-Zeid and Elkot 2017 and Abu Aly *et al*., 2017).

The interaction genotype × environment can be studied temporally (two or more seasons testing at one location) or spatially (several locations) or a combination of these. In this work, the interaction between 46 G × 5 E was studied to investigate the role of genetic structure, environment or their interaction in the onset and development of stem rust infection under field conditions. The differences between genotype × environment interactions were highly significant, which justifies the use of AMMI stability analysis for the obtained data in this investigation. Therefore, the current investigation revealed that the variation in the level of adult plant response to stem rust infection between stem rust monogenic lines was consistently attributed to their genetic structure rather than the slight changes in environmental conditions over three years. This result was reported by Singh and Narayanan (2000) who found that GE interaction was significant, therefore stability analysis can be carried out.

Principle component analysis (PCA) scores approximate to zero was recorded in any genotype means that this genotype is the more stable or adapted over all the environments (Purchase *at al*., 2000). According to the statistical analysis and based on the stability parameters obtained in the study, stem rust resistance genes; *Sr*14, *Sr*17 and *Sr*37 were the most stable genotypes across different environments and years under evaluation. This finding is in agreement with those of Letta and Tilahun (2007) who found that the two durum wheat varieties; Ilani and Kilinto were the highest stable varieties to stem rust resistance under Ethiopian conditions. So, using stem rust resistance genes; *Sr*14, *Sr*17 and *Sr*37 are useful in breeding programs aimed to develop new wheat varieties with stable resistance to stem rust, under Egyptian field conditions.

The our knowledge, the present study is one of the few studies that deal with these kind of multi-locational tests of stem rust resistance genes in Egypt. Nevertheless, information on the genetic stability of the tested stem rust resistance genes is not fully elucidated in this work. But, further studies are needed to determine a genetic basis of existing resistance to wheat stem rust in Egyptian wheat breeding materials (genotypes). Knowledge of the stem rust resistance genes that are currently deployed in Egyptian wheat cultivars is essential to determine which genes or gene combinations are providing long lasting protection and achieving a more durable resistance in these wheat cultivars. This knowledge will also alert pathologists and breeders to genetic vulnerability in wheat cultivars if, for example, it is determined that the same resistance gene is being used in cultivars that are grown over a wide areas.

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الثبات الوراثى لجينات المقاومة لمرض صدأ الساق في القمح تحت ظروف الحقل المصرية

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تم تقييم ستة وأربعين سلالة نباتية من القمح كل منها يحمل جين مفرد لمرض صدأ الساق ودراسة مدي ثباتها الوراثي تحت ظروف بيئية مختلفة وذلك في خمسة مواقع جغرافية متباينة وهي إيتاي البارود و المنيا و سخا والشرقية وبني سويف خلال ثلاثة مواسم زراعية متتالية وهى ١٦/٢٠١٥، ١٧/٢٠١٦، ١٨/٢٠١٧ تحت ظروف الحقل. تم تقسيم هذة السلالات النباتية وفقاً لكفاءتها في مقاومة مرض صدأ الساق في طور النبات البالغ إلى ثلاث أقسام. القسم الأول و يشمل السلالات النباتية الفعالة في مقاومة المرض تحت كل الظروف البيئية المختبرة وهي *Sr2* ، *Sr24*، *Sr32* ، *Sr33* ، *Sr36* ، *Sr38* ، *Sr39*. بينما القسم الثاني فقد تضمن السلالات النباية ذات الفعل المتغير أو المتباين للإصابة (حساسة او مقاومة) بمرض صدأ الساق تحت الظروف البيئية المختلفة ومواسم الدراسة. اما القسم الثالث فقد احتوي علي السلالات النباتية غير الفعالة في مقاومة مرض صدأ الساق (الحساسة) في كل المواقع وسنوات الدراسة الثلاثة. أدي وجود إختلافات معنوية بين السلالات النباتية والظروف البيئية المختلفة تحت الدراسة الي أجراء تحليل الثبات الوراثي لهذة السلالات النباتية. وفقاً لمقاييس هذ التحليل، تبين أن الثلاث سلالات نباتية *Sr14* ، *Sr17* ، *Sr37* كانت أكثر ثباتاً في مقاومتها للمرض خلال مواسم النمو الثلاثة المتتالية و في كل المواقع تحت الدراسة. وفي النهاية فأن نتائج هذة الدراسة توفر معلومات مهمة ومفيدة لمربي النبات ، تساعده علي إتخاذ القرار المناسب في إستخدام هذة السلالات النباتية كمصادر جيدة لمقاومة مرض صدأ الساق خلال برامج التربية لمقاومة أمراض الأصداء بوجة عام وخاصة مرض صدأ الساق.