Journal of Plant Physiology and Breeding

2021, 11(1): 109-121 ISSN: 2008-5168

Research paper

Unraveling genotype-isolate interaction in sunflower (*Helianthus annuus* L.)-*Sclerotinia* pathosystem using GGE biplot method

Khadijeh Musa-Khalifani[1], Reza Darvishzadeh[1*], Masoud Abrinbana[2], and Hadi Alipour[1]

Received: March 24, 2021 Accepted: June 26, 2021
[1]Department of Plant Production and Genetics, Faculty of Agriculture and Natural Resources, Urmia University, PO Box 165, Urmia, Iran
[2]Department of Plant Protection, Faculty of Agriculture and Natural Resources, Urmia University, PO Box 165, Urmia, Iran
*Corresponding author; Email: r.darvishzadeh@urmia.ac.ir

Abstract
Sunflower (*Helianthus annuus* L.) is one of the most important oilseed crops in the world. Basal stem rot, caused by *Sclerotinia* spp., is an important disease of sunflower causing considerable yield losses worldwide. Effective improvement for disease resistance relies on the understanding of the interaction between pathogen and host. A total of 100 sunflower genotypes from different worldwide agricultural research institutions were evaluated for their responses to three isolates of each of the *S. sclerotiorum* and *S. minor* at the seedling stage in the controlled conditions. Remarkable significant host-pathogen isolate interaction indicates the existence of vertical or isolate-specific resistance in the studied sunflower germplasm against *Sclerotinia* spp. Genotype-by-pathogen biplot analysis was performed to observe the pathogenicity of the two fungi on host genotypes and facilitate the simultaneous visualization of the relationship among the pathogens and genotypes. The first two principal components accounted for 95.86% and 79.77% of the total variation of the genotype-isolate interaction of *S. sclerotrium* and *S. minor*, respectively. The GGE biplot related to *S. Sclerotiorum* isolates depicted that out of the studied genotypes, "H100A/LC1064" was resistant against the A37 isolate of *S. Sclerotiorum*. Among the examined germplasm, the genotype "1059" was identified as the resistant genotype against the J2 isolate of *S. Sclerotiorum*. None of the genotypes were resistant to the J1 isolate of *S. Sclerotiorum*. Regarding the generated biplot for *S. minor*, "8A*/LC1064C" was the most resistant sunflower genotype against the M1 isolate of *S. minor*. The genotype "H205A/83HR4" was located in vertex near to A1 and G2 isolates and, therefore, was resistant to these isolates of *S. minor*. The genetic variation detected within the sunflower collections can be utilized for the selection of diverse parents in the resistant breeding programs as well as the development of mapping populations for the QTL analysis of resistance to *S. sclerotiorum* and *S. minor*.

Keywords: Biplot analysis; Disease resistance; Genotype-by-pathogen interaction; Isolate specific resistance; Sclerotinia basal stem rot; Sunflower

Citation: Musa-Khalifani Kh, Darvishzadeh R, Abrinbana M, and Alipour H, 2021. Unraveling genotype-isolate interaction in sunflower (*Helianthus annuus* L.)- *Sclerotinia* pathosystem using GGE biplot method. Journal of Plant Physiology and Breeding 11(1): 109-121.

Introduction

Sunflower (*Helianthus annuus* L.) is one of the most important oilseed crops in the world which, due to its high nutritional value (Tabrizi *et al.* 2012) and lack of anti-nutritional factors in its oil (Sosulski 1979), is useful for human nutrition. Sunflower seed contains high oil content ranging from 35 to 50% (Skoric and Marinkovic 1986), about 20% protein (Dorrell and Vick, 1997), and a high percentage (60%) of polyunsaturated fatty acids including oleic acid and linoleic acid, which control cholesterol in the blood (Satyabrata *et al.* 1988). This plant is native to North America (Putt 1997).

Stalk and head rot, caused by *Sclerotinia sclerotiorum* (Lib) de Barry and *S. minor* Jagger, are important diseases of sunflower causing considerable yield losses worldwide (Anonymous

2010). The two fungal species are also devastating soil-borne pathogens of some other crops (Clarke *et al.* 1990). All known hosts of these pathogens belong to angiosperms which a vast majority of these plants are dicotyledonous, with only a small number of monocots reported as being hosts (Melzer 1997). The *S. sclerotiorum* has a broader host range than *S. minor* infecting more than 400 plant species (Boland and Hall 1994; Melzer *et al.* 1997). It initiates the disease by myceliogenic or carpogenic germination of sclerotia producing mycelia and ascospores, respectively (Abawi and Grogan 1979). However, sclerotia of *S. minor* primarily germinate myceliogenically and carpogenic germination rarely occurs under natural conditions (Abawi and Grogan 1979).

Control of diseases caused by *Sclerotinia* species is difficult since the fungi persist in soil for long periods (Smolińska and Kowalska 2018). Employment of resistant cultivars has been considered as the most effective, economic, and environmentally-safe strategy to manage *Sclerotinia* spp., but due to the unavailability of commercial cultivars with effective resistance, fungicide application is currently the common method to control the two pathogens in various crops. Resistance to *Sclerotinia* spp. is under polygenic control (Talukder *et al.* 2014) and, thus, breeding for resistance to the pathogen relies on incorporating genetic factors from different partially resistant genotypes. In this regard, identification of novel sources of resistant genotypes is necessary to provide genetic materials required for improving crop resistance. Genetic variability for partial resistance to *S. sclerotiorum* in sunflower has been reported in both fields (Vear *et al.* 2004; Godoy *et al.* 2005) and controlled conditions (Davar *et al.* 2011) studies, however, limited information is available on the genetic variability of resistance to *S. minor* in sunflower.

Effective improvement for disease resistance relies on the understanding of pathogen × host interaction. To evaluate interactions between host genotypes and pathogen isolates, in addition to common methods including analysis of variance and mean comparisons, the biplot method (Yan and Falk 2002) can be used. GGE biplot is the abbreviation of the main effect of genotype (G) plus genotype × environment interaction (GE) which in the case of evaluating host genotype-pathogen isolate means pathogen interaction. This method shows host genotypes and pathogen isolates simultaneously in a scatter plot in which each genotype or pathogen is considered as a single point according to their scores in terms of the first and second principal components. The genotypes located near the vertices of the polygons are resistant against the isolates falling in the same sector (Yan and Tinker 2006). The present study aimed to evaluate the interactions of 100 sunflower lines with the isolates of *S. sclerotiorum* and *S. minor* using the GGE biplot method.

Materials and Methods

Sunflower germplasm and *Sclerotinia* spp. isolates

A total of 100 sunflower genotypes (Supplement Table 1), kindly provided by agricultural research institutions worldwide, were used to evaluate their responses to *S. sclerotiorum* and *S. minor* isolates

at the seedling stage in controlled conditions. The isolates have previously been isolated from symptomatic sunflower plants collected from naturally-infected fields located in Urmia and Khoy in the West Azarbaijan province, Iran (Mousa Khalifani *et al.* 2018). Three isolates from each species including the isolates A37, J1, and J2 of *S. sclerotiorum* and isolates A1, G1, and M1 of *S. minor* were selected based on their appropriate but various levels of aggressiveness on sunflower cultivar Farrokh in the previous study (Mousa Khalifani *et al.* 2018).

Host-pathogen experiment

Seeds of sunflower genotypes were sterilized for 5 min in 0.5% sodium hypochlorite solution and then sown in 20 × 60 cm rectangular pots filled with Peat moss. Plants were grown in a controlled environment with a 12 h light, 65–70% relative humidity, and 25±1°C temperature for 6 weeks until they reached the growth stage V6–V8 (Schneiter and Miller 1981). After two irrigation cycles with normal water, one irrigation cycle was performed with water containing 0.5 grams per liter of 20-20-20 (NPK) fertilizer. Factorial experiment (Factor A: sunflower genotypes and Factor B: *Sclerotinia* spp. isolates) was conducted in a completely randomized design with three replications (pots) and six plantlets in each replication.

A mycelial plug (3 mm diameter) was cut from actively growing margins of the 3-day-old colony of each isolate and placed on the basal stems of the sunflower plants at the V6–V8 (Schneiter and Miller 1981) growth stage. The stem of inoculated plants and mycelial plugs were wrapped with parafilm for 48 h to provide humidity for infection following the method described by Price and Colhoun (1975). The pots were kept in a controlled environment with a 12 h light, 65–70% relative humidity, and 25±1°C temperature. For each plant, the percentage of the necrotic area on 1 cm of the stem base and all around it was assessed visually three days after inoculation.

Data analysis

To check the significance of genotype × isolate interaction, disease severity data were first transformed to arcsin square root to satisfy the assumption of normality and then subjected to analysis of variance (ANOVA) in the software Minitab 13.0. The GGE biplot method introduced by Yan and Falk (2002) was used to visualize the genotype by isolate interaction using genotype-focused back-transformed mean disease severity data.

In the GGE biplot analysis, genotypes and isolates were treated as entries and testers, respectively. To determine the resistance of the genotypes to the isolates, the biplot was constructed by reversed sign disease severity data. The analysis was performed using the GGEBiplotGUI (Frutos *et al.* 2014) in R software. The d3heatmap, dendextend, gplots, colorspace, and RColorBrewer R-packages were used for heatmap clustering of the genotypes and traits based on their mean disease severity using Euclidean distance and Ward's clustering algorithm. The percentage of disease severity of sunflower genotypes concerning each one of the fungi isolates was considered as a variable (trait).

Therefore, we had three variables for each one of the *S. sclerotrium* and *S. minor* fungi, respectively. The cutoff point was determined by the Elbow method of the R program based on the total within-cluster sum of squares, and one-way multivariate analysis of variance (MANOVA) was performed to confirm the cutoff point. MANOVA revealed that there was statistically significant difference among the clusters at $p \leq 0.001$.

Results

Results of ANOVA revealed significant ($p \leq 0.01$) genotype and isolate effects for both *Sclerotinia* species indicating that sunflower genotypes responded differently to the fungal isolates and the isolates differed in inciting disease severity on the genotypes. Genotype by isolate interaction was also significant ($p \leq 0.01$) suggesting the existence of the isolate-specific interactions between sunflower genotypes and the isolates of *S. sclerotiorum* and *S. minor* (Table 1).

In the GGE biplot of *S. sclerotiorum* isolates-sunflower genotypes data, the first two principal components of the biplot explained 95.86% of the total variation (Figure 1). The isolates fell into two sectors but the genotypes were dispersed in all six sectors indicating that the genotypes responded differently to the isolates. Several genotypes fell in the same sectors with the *S. sclerotiorum* isolates, however, the genotypes at or near the vertices were specifically highly resistant to the isolate in the sector. For instance, the genotype “H100A/LC1064” was placed at the vertex and was specifically resistant to the isolate A37 and exhibited a high level of resistance to this isolate. Furthermore, the two sunflower genotypes “1009370 3 (100K)” and “HAR4” which were placed near the vertex, were also specifically resistant to A37 but with slightly lower resistance levels. Similarly, the two genotypes “1059” and “110” at and near the vertex, respectively, were identified as resistant to the isolate J2. The isolate J1 was placed near the biplot origin and, thus, none of the genotypes was found to be resistant to this isolate (Figure 1).

In the GGE biplot of *S. minor* isolates-sunflower genotypes data, the first two principal components accounted for 79.77% of the total variation. GGE biplot for *S. minor* isolates depicted that the genotype "8A*/LC1064C" was the most resistant sunflower genotype against the M1 isolate. Genotype "H205A/83HR4" was located at the vertex near to A1 and G2 isolates and therefore, were resistant to these isolates of *S. minor* (Figure 2).

Clustering of the studied sunflower germplasm based on the disease severity scores of *S. sclerotiorum* isolates resulted in three main clusters (Figure 3). Group I included 35 genotypes such as “1059”, “110”, “8ASB2”, “803-1”, “H100A/83HR4”, and “H543R/H543R”. These genotypes showed relatively higher resistance to the isolates J2, A37, and J1. These genotypes fell in the same sector with the isolate J2 in the GGE biplot polygon view (Figure 1). Group II involved 33 genotypes with relative resistance to A37 or J1 isolates comprising such genotypes as “H049+fSB”, “LP-CSYB”, “BF1POPB”, “H100A/LC1064”, “AF1POPA”, and “1009370 3(100K)” (Figure 3). Regarding Figure 3, susceptible genotypes “38”, “SDR19”, and “SDB3” together with other identified

Table 1. Analysis variance of the area of necrotic stem tissue resulting from infection by *Sclerotinia sclerotiorum* and *S. minor* isolates on sunflower genotypes

Source of variation	df	*S. sclerotinia*		*S. minor*	
		Mean squares	The proportion of effects from total variation (%)	Mean squares	The proportion of effects from total variation (%)
Genotype	99	0.16**	24.83	0.10**	18.92
Isolate	2	1.06**	3.32	1.26**	4.82
Genotype × Isolate	198	0.08**	24.02	0.05	18.92
Error	600	0.05	-	0.05	-

df: degrees of freedom; **significant at $p \leq 0.01$

susceptible genotypes were located in group III.

Clustering of the studied sunflower germplasm based on the disease severity scores of *S. minor* isolates also resulted in three main clusters (Figure 4). Group I consisted of the genotypes such as "HA304", "Sf-023", "SDB1", "ENSAT-254", "RT948", "11×12", and "12ASB3" which were susceptible to all G2, A1, and M1 isolates. Group II consisted of the genotypes such as "ENSAT-699", "H205A/83HR4" "110", "RHA265", and "15031" which were identified as resistant to all three G2, A1, and M1 isolates. Group III was constructed by the genotypes such as "8A*/LC1064", "1009370 1(100K)", and "H209A/LC1064" that were resistant to the isolate M1 in the GGE biplot polygon view.

Discussion

In this study, the resistance of sunflower genotypes with various genetic backgrounds and origins were assessed simultaneously to *S. sclerotiorum* and *S. minor* isolates. As a result of partial resistance (Davar *et al.* 2010; Amouzadeh *et al.* 2015), disease severity data of the genotypes had vast range of fluctuation, and *Sclerotinia* spp. isolates varied in their pathogenesis. So, the current study emphasizes the importance of employing diverse representative pathogen isolates rather than a single isolate when screening for improved *Sclerotinia* spp. resistance. As well, differences in the isolate virulence may be one reason that host genotypes classified as resistant in one study may perform poorly in another study (Baergen *et al.* 1993).

The significant genotype by isolate interaction relies on the existence of vertical or isolate-specific resistance in the studied sunflower germplasm against *Sclerotinia* spp. These results are in agreement with the findings of Davar *et al.* (2011) who have reported highly significant genotype by isolate interaction in *S. Sclerotiorum*-sunflower pathosystem. Similar interaction has been observed between *Phomopsis helianthi* isolates and sunflower genotypes with partial resistance (Viguié *et al.* 1999) and it has been suggested that significant interaction in the pathosystems with polygenic host resistance is not unexpected (Flier *et al.* 2003). However, in contrast with our findings, Vear *et al.* (2004) using 16 sunflower lines bred by INRA (France), showed the presence of partial resistance with no significant interaction for sunflower against *S. sclerotiorum*. This difference could be due to the utilization of a small number of non-diverse, local sunflower germplasm by Vear *et al.* (2004).

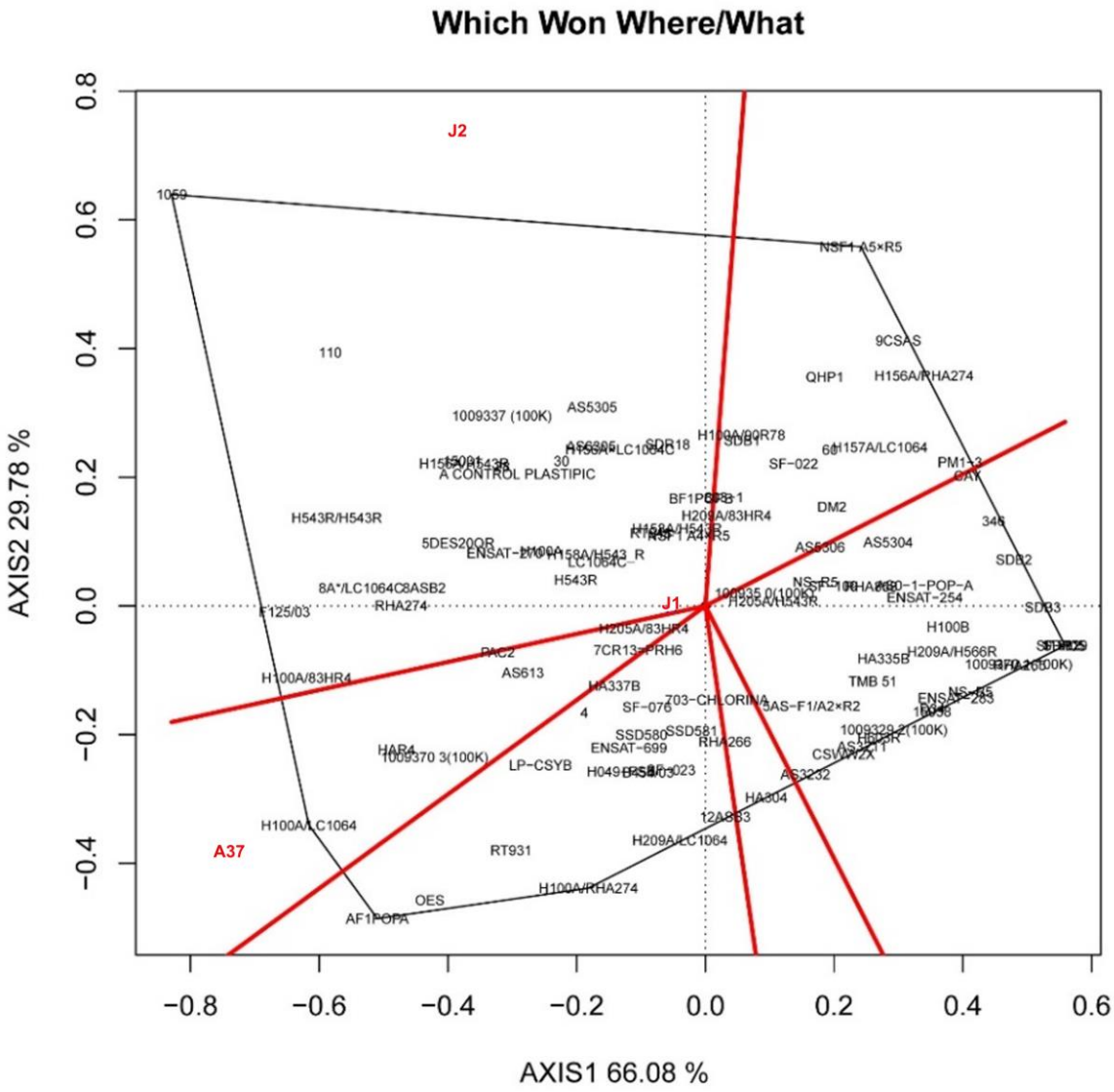

Figure 1. GGE biplot based on the genotype-focused model of the mean disease severity values showing the reaction of 100 sunflower genotypes to *Sclerotinia sclerotiorum* isolates. Sunflower genotypes and isolates are in black and red, respectively.

The response of studied sunflower genotypes varied based on the *Sclerotinia* species (*S. sclerotiorum* or *S. minor*). Albeit, there were some resistance resources among the Iranian sunflower genotypes (genotypes "110" and "1059") for *S. sclerotiorum* but there were no resistant genotypes for *S. minor* among domestic sunflower genotypes. This was predictable for *S. minor* because *S. sclerotiorum* has been the dominant causal agent of stem rot in North West of Iran especially West Azarbaijan province which was recently replaced with *S. minor* in most fields. Therefore, unlike *S. sclerotiorum*, domestic genotypes didn't evolve a broad-spectrum

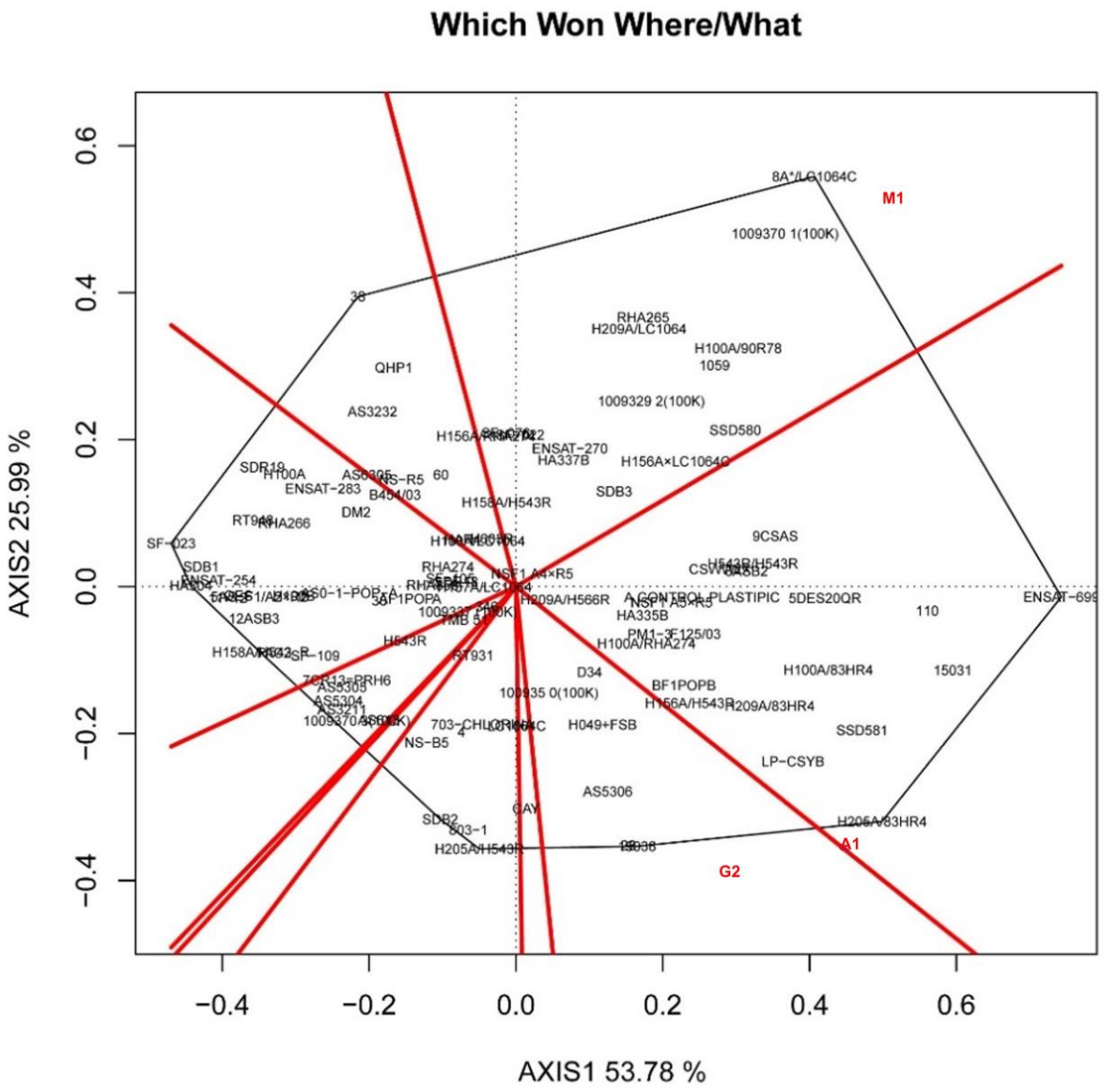

Figure 2. GGE biplot based on the genotype-focused model of the mean disease severity values showing the reaction of 100 sunflower genotypes to *Sclerotinia minor* isolates. Sunflower genotypes and isolates are in black and red, respectively.

resistance gene or many resistance genes against *S. minor*, and introducing resistance resources from the foreign sunflower plant material is mandatory. In agreement with previous reports (Abrinbana *et al.* 2012; Ghaneie *et al.* 2012; Hatami Maleki and Darvishzadeh 2014), the GGE biplot analysis could concisely identify the true resistant genotypes for each of the *S. sclerotiorum* and *S. minor* isolates. Based on the GGE biplot analysis, "H100A/LC1064" and "1059" can be considered as promising resistant genotypes to the A37 and J2 isolates of *S. sclrotiorum*. The genotype "8A*/LC1064C" exhibited the best resistance to M1, and "H205A/83HR4" exhibited

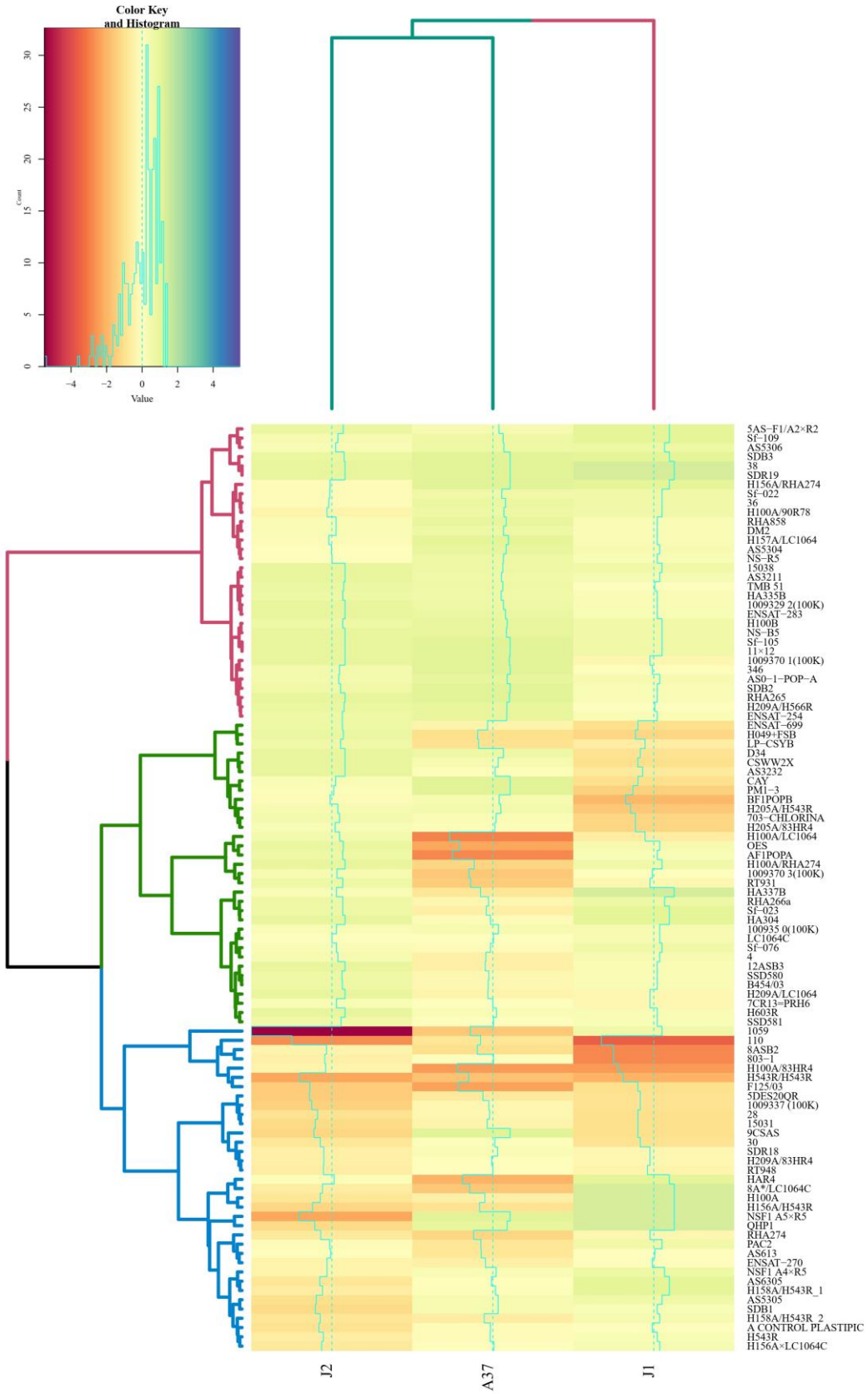

Figure 3. Dendrogram resulting from the hierarchical cluster analysis of 100 sunflower genotypes based on mean disease severities concerning *Sclerotinia sclerotiorum*.

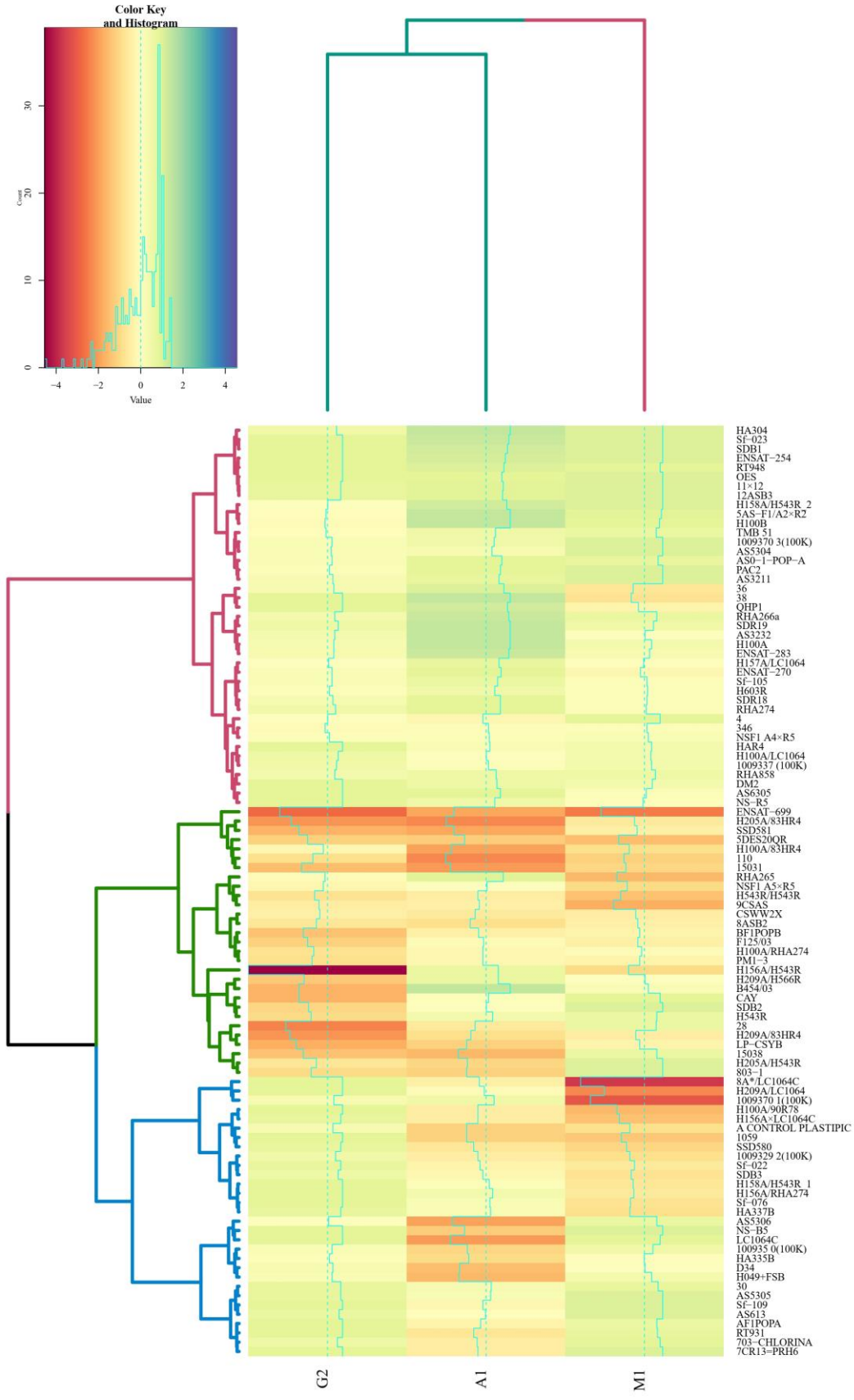

Figure 4. Dendrogram resulting from the hierarchical cluster analysis of 100 sunflower genotypes based on mean disease severities concerning *Sclerotinia minor*.

the best resistance to the A1 and G2 *S. minor* isolates, suggesting the presence of additional resistance genes and/or QTLs in these genotypes that are absent in other resistant genotypes.

Similarly, cluster analysis partially could depict the existence of genetic variability among the studied germplasm and classify the sunflower genotypes in three separate classes based on the disease severity score of both *S. sclerotiorum* and *S. minor*. It was obvious from the cluster analysis that the structure of genetic variability in relation to resistance to *S. sclerotiorum* and *S. minor* does not pursue the genotypes' geographical origins. In the state of infection by *S. sclerotiorum* as well as by *S. minor*, the susceptible genotypes were located in a distinct group.

Conclusion

A total of 100 sunflower genotypes from different worldwide agricultural research institutions were evaluated for their responses to three isolates of each of the *S. sclerotiorum* and *S. minor* at the seedling stage in controlled conditions. The GGE biplot related to *S. sclerotiorum* isolates depicted that out of the studied genotypes, "H100A/LC1064" was resistant against the A37 isolate of *S. sclerotiorum*. Genotype "1059" was identified as the resistant genotype against the J2 isolate of *S. sclerotiorum*. None of the genotypes were resistant to the J1 isolate of *S. sclerotiorum*. Genotype "8A*/LC1064C" was the most resistant sunflower genotype against the M1 isolate of *S. minor*. Genotype "H205A/83HR4" which was located in the vertex near to the A1 and G2 isolates was resistant to these isolates of *S. minor*. Classification of this sunflower collection could provide a vision for future breeding programs like the selection of parental line for the construction of mapping population for QTL analysis of resistance to *S. sclerotiorum* and *S. minor*.

Acknowledgement

We appreciate the financial support of this work by the Tarbiat Modares University, Iran.

Conflict of Interest

The authors declare that they have no conflict of interest with any people or organization concerning the subject of the manuscript.

References

Abawi GS and Grogan RG, 1979. Epidemiology of diseases caused by *Sclerotinia* species. Phytopathology 69: 899-904.

Abrinbana M, Mozafari J, Shams-Bakhsh M, and Mehrabi R, 2012. Resistance spectra of wheat genotypes and virulence patterns of *Mycosphaerella graminicola* isolates in Iran. Euphytica 186: 75-90.

Amouzadeh M, Darvishzadeh R, Davar R, Aabdollahi Mandoulakani B, Haddadi P, and Basirnia A, 2015. Quantitative trait loci associated with isolate specific and isolate non-specific partial resistance to *Sclerotinia sclerotiorum* in sunflower. Journal of Agricultural Science and Technology 17(1): 213-226.

Anonymous 2010. Sunflower- Production guideline. Department of Agriculture, Forestry, and Fisheries, Pretoria, South Africa, pp. 28.

Baergen KD, Hewitt JD, and St. Clair DS, 1993. Resistance of tomato genotypes to four isolates of *Verticillium dahliae* race 2. HortScience 28(8): 833-836.

Boland GJ and Hall R, 1994. Index of plant hosts of *Sclerotinia sclerotiorum*. Canadian Journal of Plant Pathology 16(2): 93-108.

Clarke RG, Porter IJ, and Woodroofe M, 1990. Potential strategies for control of sclerotinia stem rot in sunflowers. Proceedings of the Australian Sunflower Association 7th Workshop, Moama, NSW, Australia. Australian Sunflower Association, Queensland, Australia.

Davar R, Darvishzadeh R, and Majd A, 2011. Genotype-isolate interaction for resistance to *Sclerotinia sclerotiorum* in sunflower. Phytopathologia Mediterranea 50(3): 442-449.

Davar R, Darvishzadeh R, Majd A, Ghosta Y, and Sarrafi A, 2010. QTL mapping of partial resistance to basal stem rot in sunflower using recombinant inbred lines. Phytopathologia Mediterranea 49(3): 330-341.

Dorrell GD and Vick BA, 1997. Properties and processing of oilseed sunflower. In: Schneiter AA (ed.) Sunflower Technology and Production. Agronomy Monograph No. 35. ASA, CSSA, SSSA, Madison, WI, USA, pp. 709-745.

Flier WG, Grunwald NJ, Kroon LPNM, Sturbaum AK, Van Den Bosch TBM, Garay-Serrano E, Lozoya-Saldana H, Fry WE, and Turkensteen LJ, 2003. The population structure of *Phytophthora infestans* from the Toluca Valley of central Mexico suggests genetic differentiation between populations from cultivated potato and wild *Solanum* spp. Phytopathology 93(4): 382-390.

Ghaneie A, Mehrabi R, Safaie N, Abrinbana M, Saidi A, and Aghaee M, 2012. Genetic variation for resistance to *Septoria tritici* blotch in Iranian tetraploid wheat landraces. European Journal of Plant Pathology 132: 191-202.

Godoy M, Castaño F, Ré J, and Rodríguez R, 2005. Sclerotinia resistance in sunflower: I. Genotypic variations of hybrids in three environments of Argentina. Euphytica 145: 147-154.

Hatami Maleki H and Darvishzadeh R, 2014. Study of interactions between sunflower genotypes and black stem (*Phoma macdonaldii*) isolates using GGE biplot approach. Journal of Crop Protection 3(1): 51-57.

Melzer MS, Smith EA, and Boland GJ, 1997. Index of plant hosts of *Sclerotinia minor*. Canadian Journal of Plant Pathology 19(3): 272-280.

Mousa Khalifani Kh, Darvishzadeh R, and Abrinbana M, 2018. Aggressiveness diversity of *Sclerotinia sclerotiorum* and *S. minor* isolates in west Azarbaijan province and specific interaction of sunflower lines with the isolates of these pathogens. Journal of Applied Researches in Plant Protection 7(1): 135-150.

Price K and Colhoun J, 1975. A study of variability of isolates of *Sclerotinia sclerotiorum* (Lib.) de Bary from different hosts. Journal of Phytopathology 83(2): 159-166.

Putt ED, 1997. Early history of sunflower. In: Schneiter AA (ed.) Sunflower Technology and Production. Agronomy Monograph No. 35. ASA, CSSA, SSSA, Madison, WI, USA, pp. 1-19.

Satyabrata M, Hedge MR, and Chattopadhay SB, 1988. Handbook of Annual Oilseed Crops. Oxford IBH Pub. Co. (Pvt.) Ltd. New Delhi, pp. 176.

Schneiter AA and Miller JF, 1981. Description of sunflower growth stages. Crop Science 21(6): 901-903.

Skoric D and Marinkovic R, 1986. Most recent results in sunflower breeding. International Symposium on Sunflower, Budapest, Hungary, pp. 118-119.

Smolińska U and Kowalska B, 2018. Biological control of the soil-borne fungal pathogen *Sclerotinia sclerotiorum* — a review. Journal of Plant Pathology 100(1): 1-12.

Sosulski F, 1979. Food uses of sunflower proteins. Journal of the American Oil Chemists' Society 56(3): 438- 442.

Tabrizi M, Hassanzadeh F, Moghaddam M, Alavikia S, Aharizad S, and Ghaffari M, 2012. Combining ability and gene action in sunflower using line × tester method. Journal of Plant Physiology and Breeding 2(2): 35-44.

Talukder ZI, Hulke BH, Qi L, Scheffler BE, Pegadaraju V, McPhee K, and Gulya TJ, 2014. Candidate gene association mapping of Sclerotinia stalk rot resistance in sunflower (*Helianthus annuus* L.) uncovers the importance of COI1 homologs. Theoretical and Applied Genetics 127(1): 193-209.

Vear F, Willefert D, Walser P, Serre F, and Tourvieille de Labrouhe D, 2004. Reaction of sunflower lines to a series of *Sclerotinia sclerotiorum* isolates. Proceedings of the 16th International Sunflower Conference, Fargo, ND, USA, pp. 135-140.

Viguié A, Vear F, and Tourvieille de Labrouhe D, 1999. Interactions between French isolates of *Phomopsis / Diaporthe helianthi* Munt.-Cvet. et al. and sunflower (*Helianthus annuus* L.) genotypes. European Journal of Plant Pathology 105: 693-702.

Yan W and Falk DE, 2002. Biplot analysis of host- by-pathogen data. Plant Disease 86(12): 1396-1401.

Yan W and Tinker NA, 2006. Biplot analysis of multi-environment trial data: principles and applications. Canadian Journal of Plant Science 86(3): 623-645.

تجزیه GGE بای‌پلات اثر متقابل جدایه-ژنوتیپ در پاتوسیستم آفتابگردان-اسکلروتینیا

خدیجه موسی-خلیفانی[1]، رضا درویش زاده[1*]، مسعود ابرین بنا[2] و هادی علیپور[1]

۱- گروه تولید و ژنتیک گیاهی، دانشکده کشاورزی و منابع طبیعی، دانشگاه ارومیه، ارومیه
۲- گروه گیاهپزشکی، دانشکده کشاورزی و منابع طبیعی، دانشگاه ارومیه، ارومیه
*مسئول مکاتبه؛ Email: r.darvishzadeh@urmia.ac.ir

چکیده

آفتابگردان (*Helianthus annuus* L.) یکی از مهمترین محصولات دانه روغنی در جهان است. پوسیدگی پایه ساقه، ناشی از *Sclerotinia* spp.، یکی از بیماری‌های مهم آفتابگردان است که باعث از بین رفتن قابل توجه عملکرد در سراسر جهان می‌شود. بهبود مؤثر برای مقاومت در برابر بیماری متکی به درک متقابل بین عامل بیماری‌زا و میزبان است. در این مطالعه، واکنش ۱۰۰ ژنوتیپ آفتابگردان از مؤسسات تحقیقاتی مختلف کشاورزی در جهان به سه جدایه از هر یک از گونه‌های *S. sclerotiorum* و *S. minor* در مرحله گیاهچه در شرایط کنترل شده بررسی شد. وجود اثر متقابل معنی‌دار حاکی از مقاومت جدایه اختصاصی یا عمودی در برابر گونه‌های اسکلروتینیا در ژرم‌پلاسم آفتابگردان مورد مطالعه است. تجزیه و تحلیل بای‌پلات ژنوتیپ - پاتوژن برای تشریح بیماری‌زایی دو قارچ روی ژنوتیپ‌های میزبان انجام شد که تجسم همزمان رابطه بین عامل بیماری‌زا و ژنوتیپ را تسهیل می‌کند. دو مؤلفه اصلی اول در تجزیه بای‌پلات به ترتیب ۹۵/۸۶ و ۷۹/۷۷ از تغییرات کل در تعاملات جدایه-ژنوتیپ گونه‌های *S. sclerotrium* و *S. minor* را توجیه کردند. GGE بای‌پلات مربوط به جدایه-های *S. sclerotiorum* نشان داد که ژنوتیپ "H100A/LC1064" از ژرم‌پلاسم مورد مطالعه در برابر جدایه A37 از *S. sclerotiorum* مقاوم است. از بین ژنوتیپ‌های مورد مطالعه، ژنوتیپ "۱۰۵۹" به عنوان ژنوتیپ مقاوم در برابر جدایه J2 از *S. sclerotiorum* شناخته شد. ژنوتیپ مقاومی برای جدایه J1 گونه *S. sclerotiorum* شناسایی نشد. در ارتباط با تجزیه GGE بای‌پلات انجام گرفته برای گونه *S. minor*، ژنوتیپ "8A*/LC1064C" مقاوم‌ترین ژنوتیپ آفتابگردان در برابر جدایه M1 از گونه *S. minor* بود. ژنوتیپ "H205A/83HR4" واقع در رئوس نزدیک به جدایه‌های A1 و G2 گونه *S. minor* به این جدایه‌ها مقاوم بود. تنوع ژنتیکی شناسایی شده در مجموعه ژرم‌پلاسم آفتابگردان مورد مطالعه می‌تواند در انتخاب والدین متنوع برای برنامه‌های اصلاح برای مقاومت و همچنین برای توسعه جمعیت‌های مکان‌یابی برای شناسایی و تجزیه و تحلیل QTLهای دخیل در مقاومت در برابر گونه‌های *S. sclerotiorum* و *S. minor* استفاده شود.

واژه‌های کلیدی: آفتابگردان؛ اثر متقابل عامل بیماری‌زا-میزبان، پوسیدگی پایه ساقه اسکلروتینیایی؛ تجزیه بای‌پلات؛ مقاومت به بیماری؛ مقاومت جدایه اختصاصی