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Genotype-isolate interaction for resistance to *Sclerotinia sclerotiorum* in sunflower

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Summary. The sunflower (*Helianthus annuus* L.) is one of the most important crops grown for edible oil. *Sclerotinia sclerotiorum* (Lib.) de Bary is a common and widespread pathogen of sunflower. In the present study the reaction of 35 genotypes, including recombinant inbred lines and their parents, M7 mutant lines developed by gamma irradiation, and some genotypes from different geographical origins, were evaluated against eight *S. sclerotiorum* isolates in controlled conditions. The proportion of the subsequent basal stem lesions was measured 3 days after inoculation. Highly significant differences were observed among sunflower genotypes and *S. sclerotiorum* isolates, with the isolates interacting differentially with sunflower genotypes. Two genotypes had high partial resistance to all *S. sclerotiorum* isolates, whereas others were susceptible to all isolates. Isolates of *S. sclerotiorum* differed in virulence to host genotypes. Some genotypes showed specific interactions with *S. sclerotiorum* isolates, being resistant to some isolates but susceptible to others. Recombinant inbred lines used in this study showed different reactions to eight isolates of *S. sclerotiorum* when compared with their parental lines. The isolate-specific and isolate-nonspecific partial resistant genotypes identified in present experiments should be used in crossing programmes for breeding of durable resistance to *Sclerotinia* basal stem disease.

Key words: basal stem rot, *Helianthus annuus* L., isolate specific and non-specific partial resistance, interaction effect slicing.

Introduction

Sclerotinia sclerotiorum (Lib.) de Bary, which causes white rot and wilt, is a widespread pathogen infecting over 400 species of plants, including many important crop species. The majority of these hosts are dicotyledons, although a number of agriculturally significant monocotyledon plants are also host of this pathogen (Boland and Hall, 1994). Among hosts, sunflower is an important plant that is susceptible to *S. sclerotiorum* infections during almost its entire life cycle. Sunflower cotyledons, apical buds, stem bases, leaves and head are susceptible to infection by fungi and show white rot (Gulya *et al*., 1997).

Sclerotinia sclerotiorum is a homothallic fungus producing sexual ascospores, but no conidia. The fungus produces sclerotia that are the asexual resting propagules that germinate to produce either hyphae or apothecia (Mitchell and Wheeler, 1990). White rot, caused by *S. sclerotiorum*, is a major yield-limiting factor in sunflower in temperate regions of the world. Rapid drying of the leaves and development of lesions on the tap roots and basal portions of stems cause plants to die within a few days after the onset of wilting (Dorrell and Huang, 1978). Yield losses can reach 100% when the climatic conditions are favorable for the fungus (Sackston, 1992). In most cases, fungus penetration of host plants is directly through the cuticle

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and not through stomata (Boyle, 1921), and enzymatic digestion of the cuticle plays a role in the penetration process (Tariq and Jeffries, 1986). Infection of healthy tissue by myceliogenic infection depends on the formation of appressoria (Tariq and Jeffries, 1984). Soil and climatic conditions in production areas influence the plant tissues most attacked, but economic losses following more than one form of attack may occur in the same region (Tourvieille de Labrouhe *et al*., 1992). In Iran, attacks by the pathogen on basal stems are considered a potential danger for the sunflower crop.

Chemical control of Sclerotinia disease on sunflower either does not exist or it is difficult to apply on a large scale (Peres and Regnault, 1985). Control using host resistance is therefore of considerable importance, and the aim must be to select genotypes with high levels of resistance to all forms of *S. sclerotiorum* attack found in the regions in which sunflower crops may be cultivated. Utilization of sunflower cultivars with improved partial resistance to *S. sclerotiorum* in combination with appropriate crop management practices is an effective way to control the disease. Genetic variability for susceptibility to white rot in sunflower has been reported both in field and controlled conditions, but no complete resistance has been identified in the cultivated sunflower (Tourvieille *et al*., 1996; Degener *et al*., 1998, 1999; Rönicke *et al*., 2004). Quantitative trait loci (QTL) associated with partial resistance to *S. sclerotiorum* in sunflowers have been identified in several studies (Mestries *et al*., 1998; Bert *et al*., 2002; Hahn, 2002; Micic *et al*., 2004; Davar *et al*., 2010). Using parental genotypes and their recombinant inbred lines, Davar *et al*. (2010) identified several QTLs for partial resistance to *S. sclerotiorum* under controlled conditions.

Recently, in separate experiments, Ekins *et al*. (2007) in Australia and Davar *et al*. (2010) in Iran evaluated the aggressiveness of several *S. sclerotiorum* isolates on sunflower in controlled conditions. They showed that *S. sclerotiorum* isolates differed in their aggressiveness on sunflower plants. Screening of the aggressiveness of *S. sclerotiorum* isolates would be best conducted before their use in resistance screening, to ensure that hypovirulent isolates are not used in resistance screening. The use of multiple isolates would also be beneficial for resistance screening programs, not only to ensure aggressive isolates are tested, but also to screen against other traits of the pathogen (Ekins *et al*., 2007).

Differences in aggressiveness of *S. sclerotiorum* isolates suggests the existence of genotype ´ isolate interactions in the sunflower/*S. sclerotiorum* pathosystem. Existence of any interactions between sunflower genotypes and fungal isolates can influence the efficiency of breeding programs. Understanding differential responses of sunflower genotypes to different *S. sclerotiorum* isolates is useful for development of durable resistance and for plant breeding, provided that breeders know which types of *S. sclerotiorum* exist in the geographic areas they are breeding for. The high genetic variability for pathogenicity in *S. sclerotiorum* requires simultaneous incorporation of several genes for resistance into host cultivars if they are to remain effective for use over a large area. The lack of information on the interactions between resistance genes and pathogen populations places limitations on the effective deployment of resistance. The present study was aimed to determine the amount of variation in *S. sclerotiorum*, and whether any interactions occur in the response of sunflower genotypes to a range of isolates. The information presented here will assist sunflower breeders to choose parents of crosses for breeding of durable resistance to basal stem rot of sunflower.

Materials and methods

Sunflower genotypes and fungal isolates

Thirty five sunflower genotypes were selected on the basis of their agricultural characteristics and levels of susceptibility to *S. sclerotiorum*. Recombinant inbred lines (RILs) derived from a cross between PAC2 and RHA266 were selected for their partial resistance to isolate SSU107 of *S. sclerotiorum* (Davar *et al*., 2010). Several mutants were identified that consistently showed altered resistance to black stem disease caused by *Phoma macdonaldii* (Darvishzadeh *et al*., 2010), and four of them were selected for this investigation. Mutant lines were developed by irradiation of the AS613 genotype with gamma rays and advanced by modified single-seed descent (SSD) with no prior selection for resistance to the disease (Sarrafi *et al*., 2000). Other genotypes used in this study were Iranian inbred lines (provided by Mehdi Ghaffari,

Table 1. Analysis of variance for disease severity in sunflower genotypes infected by eight *Sclerotinia sclerotiorum* isolates in controlled conditions. Coefficient of variation = 16.72.

Source of variation	df[a]	Sum of squares	Mean square	F value[b]
Genotype	34	16.02	0.472	37.37**
Isolate	7	5.99	0.861	68.14**
Genotype × isolate	238	14.42	0.061	4.79**
Genotype × isolate effect sliced by isolate				
Isolate[c]	df[a]	Sum of squares	Mean square	F value[b]
SSKH2	34	2.85	0.083	6.63**
SSKH26	34	2.85	0.084	6.62**
SSS45	34	6.04	0.178	14.06**
SSU35	34	3.88	0.114	9.02**
SSU53	34	4.12	0.121	9.59**
SSU55	34	2.62	0.077	6.11**
SSU73	34	6.10	0.078	6.19**
SSU87	34	5.44	0.160	12.66**

[a]df = degrees of freedom.
[b] ** = Significant at P=0.01.
[c] For each isolate the first two letters refer to *Sclerotinia sclerotiorum* Lib. de Bary. The third and fourth letters show the abbreviated name of the locations where the isolates were collected. S, Salmas; KH, Khoy; U, Urmia. The locations were ~200 km apart.

The Seed and Plant Improvement Institute, Khoy, Iran) and lines introduced from the United States Department of Agriculture (USDA), Yugoslavian, Hungarian, and French seed companies.

These isolates (SSU53, SSU55, SSU73, SSU87, SSKH2, SSKH26, SSU35, SSS45)are intermediate to the most aggressive and were derived from samples collected from northwest regions of Iran where sunflower is cultivated. The selected isolates were SSU53, SSU55, SSU73, SSU87, SSKH2, SSKH26, SSU35, SSS45.

Experimental design

The responses of the 35 sunflower genotypes were evaluated with eight *S. sclerotiorum* isolates under controlled conditions. A factorial experiment was arranged in completely randomized design with six replications. Seeds were sterilized for 5 min in sodium hypochlorite solution (6 chlorometric degrees) and then sown in 10×12 cm pots filled with sterilized soil. The soil was silty clay with a pH of 7.6 and an EC of 0.6 dSm^{-1}. Plants were grown in a controlled environment with a 12 h day, 65% relative humidity and a day/night temperature of 24±1/18±1°C with a daylight intensity of 200 $mEm^{-2}s^{-1}$ for 4 weeks, until they reached growth stage V6–V8 (Schneiter and Miller, 1981). *Sclerotinia sclerioriorum* isolates were separately grown on PDA medium in the dark at room temperature (25±2°C). Mycelial plugs (3 mm diam.) of each isolate were cut from the growing edges of colonies (3 days old on PDA) and were placed against the basal stems of the sunflower plants at V6–V8 growth stage. The stem of each inoculated plant and mycelial plug were wrapped with parafilm for 48 h to preserve humidity, following the method of Price and Colhoun (1975). For each plant, the percentage of necrotic area on 1 cm of the stem base and all around it was assessed visually 3 days after inoculation.

Statistical analysis

The normality of disease severity data were assessed with the Shapiro-Wilks test (Proc Univari-

ate; SAS Institute Inc., Cary, NC, USA). Analysis of variance was performed using the general linear model (GLM) procedure in the SAS software. The main effects of genotypes and isolates as well as their interactions were determined. Host line × isolate interaction effects were sliced by isolate in the SAS software in order to identify the specificity of sunflower lines to particular isolates. When significant treatment effects were found in the analysis of variance, mean comparisons were performed with the Student-Newman-Keuls (SNK) test.

Results

The infection tests on basal stems showed that there were statistically significant differences ($P \leq 0.01$) in isolate aggressivities on the 35 sunflower genotypes tested (Table 1). The variation in the mean responses of host genotypes to infection by *S. sclerotiorum* was also identified. On the other hand, the genotype × isolate interaction was significant (Table 1), indicating that the sunflower genotypes differentially responded to *S. sclerotiorum* isolates. Isolate SSKH26 was the most aggressive on the genotypes tested, whereas isolate SSU35 was the least aggressive (Table 2). Host genotype LR67 and our mutant line 'M7-575-1' were the most resistant genotypes across all isolates tested, while the lines M7-2861, HA337B and SDB3 were susceptible to the all isolates tested. Some of genotypes, such as LR57 and C94, showed intermediate responses across *S. sclerotiorum* isolates, while others had different responses against different isolates. Specific interactions between host lines and isolates were identified using 'interaction effect slicing'. Line × isolate effects sliced by isolate identified specifically resistant or susceptible lines to particular isolates.

Line × isolate slicing revealed that line RHA265 had specific interactions with isolate SSU73, whereas it was more susceptible to most other isolates. Line PAC2 had specific resistance to isolate SSU35 but was susceptible to other studied isolates. Line H229 showed resistance to isolates SSU35 and SSU53. Genotype NSATB5 showed moderate resistance to isolate SSU35, but was susceptible to all other isolates. Line ENSAT-B4 was especially susceptible to isolates SSKH2 and SSKH26. Line × isolate slicing revealed that line HC59 had specific susceptibility to isolates SSKH2 and SSKH26, but had specific resistance to isolate SSS45. The RILs showed different reactions to the *S. sclerotiorum* isolates when compared with their parental lines (PAC2 and RHA266) (Table 2). For instance, line LR19 was more resistant to isolate SSU55 in comparison to its parents PAC2 and RHA266.

Discussion

Scoring the damage caused by a pathogen in naturally infected plants under field conditions can be reliable, but it is not always possible to evenly expose plants to the pathogen and so achieve uniform infection. Homogeneous infection of each genotype is essential for the precise identification of the level of susceptibility in each inbred line (Hahn, 2000). For this reason, the artificial inoculation method developed by Price and Colhoun (1975) was used in the present study to apply standardized infection. The basal stem test allowed the inoculum load per plant to be controlled, minimizing plants escaping infection and consequently reducing potential for false negatives in responses to the pathogen. No fully resistant genotypes have been identified in sunflower for resistance to *Sclerotinia* (Hahn, 2002); all plantlets with no visible lesions on the basal stems were defined as not infected, and excluded from the present experiment.

Genotypes used in this study differed considerably in resistance to *Sclerotinia* wilt (Table 2, and b). Among the 35 sunflower genotypes used, three lines, M7-2861, HA337B and SDB3, were susceptible to all isolates of *S. sclerotiorum*, whereas LR67 and our mutant line M7-575-1 showed partial resistance to all *S. sclerotiorum* isolates. Lines HC133, H229, NSATB5, ENSAT-54 and HC59 showed different susceptibility levels to studied isolates (Table 2). Results confirm the genetic variability for partial resistance to *S. sclerotiorum* on the basal stems of sunflower observed in previous studies under field and controlled conditions (Micic *et al*., 2005a, 2005b; Davar *et al*., 2010). These results indicate the potential for enhancing resistance to mycelial extension of the pathogen in sunflower. Variation in susceptibility of genotypes can be attributed predominantly to genetic causes, because the infection of plants at the same developmental stage and grown under similar

Table 2. Mean percent necrotic area on 1 cm of the stem bases of 35 sunflower genotypes inoculated with eight *Sclerotinia sclerotiorum* isolates (SSU53, SSU55, SSU73, SSU87; SSKH2, SSKH26, SSU35, SSS45) under controlled conditions. Percentage of necrotic area was measured visually 3 days after inoculation.

Genotype	[1]Type	Origin	[2]Isolate								$\overline{X}$ genotype[4]
			SSU53	SSU55	SSU73	SSU87	SSKH2	SSKH26	SSU35	SSS45	
AS5304	BL	France	[3]63 bcdefghi	50 def	73 abcde	63 cdefghij	70 abcde	73 abcdefg	65 abcdef	49 ijkl	63.25
AS613	BL	France	78 abcdefg	66 bcdef	85 abc	83 abcdef	100 a	68 bcdefg	60 abcdefg	88 abcdef	78.50
C94	RIL	France	46 hij	55 cdef	64 cdef	47 hij	56 de	75 abcdefg	43 fghi	45 ijkl	53.88
ENSAT-B4	BL	France	51 fghij	58 bcdef	61 cdef	54 fghij	80 abcde	90 abcd	53 cdefghi	46 ijkl	61.63
LC1064-C	BL	France	36 ij	78 abcd	83 abc	51 ghij	60 bcde	84 abcdef	51 cdefghi	56 ghijkl	62.38
LR19	RIL	France	85 abcd	42 f	45 ef	99 a	54 e	54 fg	75 abcde	53 hijkl	63.38
LR57	RIL	France	48 ghij	51 def	56 cdef	54 fghij	55 de	50 g	43 fghi	33 l	48.75
LR67	RIL	France	29 j	45 ef	70 abcdef	35 j	71 abcde	66 bcdefg	26 i	38 jkl	47.50
M7-2861	M	France	92 ab	71 abcdef	68 bcdef	90 abcd	88 abc	100 a	85 ab	99 a	86.63
M7-381-1-1	M	France	68 abcdefgh	56 cdef	73 abcde	66 bcdefghij	74 abcde	58 efg	48 defghi	64 defghijkl	63.38
M7-54-1	M	France	55 defghij	54 cdef	59 cdef	78 abcdefgh	70 abcde	64 bcdefg	45 efghi	34 kl	57.38
M7-575-1	M	France	35 ij	45 ef	55 cdef	48 hij	58 cde	64 bcdefg	31 ghi	36 jkl	46.50
NSATB5	BL	France	62 bcdefghi	78 abcd	75 abcde	80 abcdefg	99 a	100 a	45 defghi	75 abcdefghi	76.75
NSATR5	BL	France	71 abcdefgh	59 bcdef	98 ab	90 abcd	80 abcde	84 abcdef	80 abc	65 cdefghijk	78.38
PAC2	BL	France	50 fghij	61 bcdef	61 cdef	63 cdefghij	71 abcde	74 abcdefg	40 fghi	52 hijkl	59.00
RT931	BL	France	73 abcdefgh	79 abcd	61 cdef	100 a	85 abcde	68 bcdefg	87 a	67 bcdefghij	77.50
B454/03	BL	Hungary	66 abcdefghi	75 abcde	78 abcd	73 abcdefghi	84 abcde	85 abcdef	51 cdefghi	96 abc	76.00
F1250/03	BL	Hungary	65 abcdefghi	66 bcdef	78 abcd	96 ab	73 abcde	91 abc	77 abcd	100 a	80.75
H227	BL	Iran	65 abcdefghi	76 abcde	83 abc	93 abc	100 a	100 a	76 abcde	95 abcd	86.00
H228	BL	Iran	48 ghij	49 def	55 cdef	78 abcdefgh	100 a	81 abcdefg	58 abcdefgh	39 jkl	63.50
H229	BL	Iran	36 ij	64 bcdef	83 abc	50 ij	74 abcde	60 defg	28 hi	63 efghijkl	57.25
HC113	BL	Iran	45 hij	64 bcdef	75 abcde	55 efghij	78 abcde	73 abcdefg	54 bcdefghi	50 ijkl	61.75
HC133	BL	Iran	85 abcd	88 ab	77 abcd	99 a	100 a	93 ab	75 abcde	53 hijkl	83.75
HC59	BL	Iran	52 efghij	63 bcdef	51 def	61 defghij	90 ab	76 abcdefg	61 abcdefg	35 jkl	61.13
RHA265	BL	USA	74 abcdefgh	85 abc	35 f	95 ab	78 abcde	93 ab	60 abcdefgh	98 ab	77.25
RHA266	BL	USA	51 fghij	100 a	50 def	94 abc	100 a	76 abcdefg	69 abcdef	94 abcde	79.25
RHA340	BL	USA	49 fghij	48 def	63 cdef	55 efghij	64 bcde	84 abcdef	54 bcdefghi	48 ijkl	58.13
SB1	BL	USA	60 cdefghij	53 def	79 abcd	98 a	65 bcde	100 a	75 abcde	82 abcdefgh	76.50
SDB3	BL	USA	95 a	73 abcdef	100 a	93 abc	79 abcde	70 bcdefg	70 abcdef	86 abcdefg	83.25
SDR19	BL	USA	88 abc	75 abcde	98 ab	84 abcdef	83 abcde	94 ab	86 a	58 fghijkl	83.25
803-1	BL	Yugoslav	80 abcdef	53 def	64 cdef	92 abcd	85 abcde	100 a	71 abcdef	58 fghijkl	75.38
HA337B	BL	Yugoslav	83 abcde	85 abc	75 abcde	100 a	100 a	88 abcde	78 abcd	70 cdefghij	84.88
HAR4	BL	Yugoslav	52 efghij	54 cdef	68 bcdef	86 abcde	86 abcd	79 abcdefg	71 abcdef	48 ijkl	68.00
PM1-3	BL	Yugoslav	63 bcdefghi	68 bcdef	75 abcde	99 a	78 abcde	92 ab	75 abcde	62 fghijkl	76.50
QHP-1	BL	Yugoslav	43 hij	52 def	65 cdef	48 hij	57 cde	60 cdefg	41 fghi	38 jkl	50.50
$\overline{X}$ isolate[5]			60.20	62.09	69.77	63.97	78.43	79.06	60.20	62.09	

[a] BL, breeder's line; RIL, recombinant inbred line; M, gamma-irradiation induced mutant line.
[b] For each isolate the first two letters refer to *Sclerotinia sclerotiorum* Lib. de Bary. The third and fourth letters show the abbreviated name of the locations where the isolates were collected. S, Salmas; KH, Khoy; U, Urmia. The locations were ~200 km apart.
[c] Mean percent necrotic area of each genotype challenged by each *Sclerotinia sclerotiorum* isolate, 3 days after basal stem inoculation. Means followed by the same letters are not significantly different (P=0.05) according to the Student-Newman-Keuls (SNK) test.
[d] Main effect of genotype.
[e] Main effect of isolate.

conditions helped to minimize the influence of environmental factors.

This study showed significant differences in aggressiveness among *S. sclerotiorum* isolates obtained from basal stem lesions in sunflower in the main regions of Iran where this crop is grown. The least and most aggressive isolates were SSU35 and SSKH26, respectively (Table 2). Similar variation in aggressiveness was demonstrated among isolates of *S. sclerotiorum* originating from different geographical locations based on pathogenicity tests on basal stem (Davar *et al*., 2010; Ekins *et al*., 2007).

The results provide strong indications of the existence of specificity between *S. sclerotiorum* isolates and sunflower genotypes for partial resistance. The line × isolate interaction effect sliced by isolate allowed individual interactions to be clearly identified. Genotype RHA265 had specific resistance to isolate SSU73 (Table 2). Also lines HC133, H229, NSATB5, ENSAT-54 and HC59 had specific resistance or susceptibility to some isolates. These results are in agreement with those of Darvishzadeh *et al*. (2007), who also found large differences between French *P. macdonaldii* isolates and those of other countries in aggressiveness on sunflower genotypes. In their study, the two host genotypes AS613 and PAC2 showed specific resistance to isolate MP8 of *P. macdonaldii*. In the present study, specific resistance was also detected in genotype PAC2 against isolate SSU35 of *S. sclerotiorum*. If a line was resistant to all isolates no interaction could be detected, and, likewise, if one isolate had low aggressiveness on all lines tested no interaction could be detected. Host lines LR67 and M7-575-1, the most resistant lines, had high partial resistance to all isolates (Table 2).

Genotype × isolate interactions for partial resistance have been observed in other pathosystems, such as sunflower/*Phomopsis* (Viguié *et al*., 1999), maize/*Fusarium* (Reid *et al*. (1993) and rice/*Magnaporthe* (Zenbayashi-Sawata *et al*., 2002). All reports on genotype × isolate interactions draw the same conclusion concerning inoculum to be used when breeding for durable resistance. In studies of strawberry resistance to *Colletotrichum acutatum*, Denoyes-Rothan and Guerin (1996) observed fluctuations in disease response and warned breeders of the necessity of using strains representative of the population to screen for stable resistant cultivars. Baergen *et al*. (1993), working on *Verticillium dahliae* on tomato, suggested that several isolates should be used to improve resistance to race 2 of the pathogen.

Specific interactions can be used to postulate the presence of resistance genes that operate partially in compatible interactions, as the percentage of basal stem area exhibiting disease symptoms in partially resistant genotypes corresponds to very localized necrosis and not to a hypersensitive response, as basal stem necrosis spreads slightly with time. Gene-for-gene relationships were reported between a partial resistance gene in rice and a gene for aggressiveness in *Magnaporthe grisea* (Zenbayashi- Sawata *et al*., 2005). Zenbayashi- Sawata *et al*. (2005) concluded that the gene-for-gene relationship between host and pathogen might operate both for complete resistance, as expressed in incompatible combinations, and also for partial resistance in compatible interactions.

In the present study, mutant host line M7-575-1 showed strongly enhanced partial resistance towards all *S. sclerotiorum* isolates compared to the original line AS613 (Table 2). This might have been caused by the lack of some susceptibility factors in the mutant line. Plant resistance towards a pathogen is often correlated to receptor-mediated perception of the pathogen, which triggers fast and efficient defense responses in the host (Montesano *et al*., 2003). A possible hypothesis to explain the phenotype of mutant M7-575-1 is that a mutation modified a putative receptor involved in resistance towards *S. sclerotiorum* isolates and changed ligand specificity. Enhanced partial resistance has previously been reported by Darvishzadeh *et al*. (2007, 2008) in the sunflower mutant line M6-54-1 for partial resistance towards three *P. macdonaldii* isolates (MA6, MP10 and MP6) compared to the original line AS613.

Recombinant inbred lines used in the present study showed different reactions to eight isolates of *S. sclerotiorum* when compared with their parental lines (PAC2 and RHA266) (Table 2). This is in agreement with the work of Darvishzadeh *et al*. (2007), who observed that the susceptibility of sunflower RILs that were infected by seven isolates of *P. macdonaldii* varied in comparison to their parents. Bert *et al*. (2004) reported that susceptibility of sunflower genotypes in F3 families infected by

an isolate of *P. macdonaldii* and *S. sclerotiorum* varied in both directions when compared with their parents. Davar *et al*. (2010) observed that susceptibility of sunflower genotypes in F9 lines infected by an isolate of *S. sclerotiorum* varied significantly. Some RILs produced lower disease severity than their parents, but others produced greater severity than the parents. This phenomenon, considered as transgressive segregation, is the result of accumulation of alleles with positive or negative additive effects in the offspring (Zhang *et al.*, 2001). This was supported by QTL mapping in our previous study, since the sign of the gene effects showed that both parental lines contributed to positive alleles for resistance to basal stem rot (Davar *et al*., 2010).

In conclusion, new sources of resistance to *Sclerotinia* basal stem disease were identified in the genotypes selected from the mutant sunflower population. The isolate-specific and isolate-nonspecific partial resistant genotypes identified in this experiment could be used in crossing programmes for breeding of durable resistance to *Sclerotinia* basal stem disease.

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