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INFECTION COURTS AND LENGTH OF SUSCEPTIBLE PERIOD RELATED TO SUNFLOWER HEAD ROT (*Sclerotinia sclerotiorum*) RESISTANCE

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SUMMARY

Sunflower head rot (SHR) (*Sclerotinia sclerotiorum* (Lib.) de Bary) is one of the major diseases affecting this crop worldwide. In spite of reports of different levels of susceptibility among sunflower genotypes, there is no evidence of complete resistance in any commercial hybrid. The aim of this work was to evaluate the number of infection courts and the length of the susceptible period in two genotypes with contrasting but stable SHR susceptibility. Repeated experiments during three years in the field included the sunflower hybrids Paraíso-20, moderately resistant, and Rancul, susceptible. Plants were inoculated with ascospores by spray application. Only one or two infection courts were found in each infected sunflower head and this feature did not distinguish cultivar susceptibility. Differences between cultivars were detected considering the length of the susceptible period based on disease incidence higher than zero (LSP) or higher than 10% (LSP10) in two out of three years. LSP was longer in Rancul (28 days) than in Paraíso-20 (17 days). Also LSP10 was longer in Rancul (22 days) than in Paraíso-20 (9 days). The suitability of these two features as components of partial resistance is discussed

Key words: ***Helianthus annuus* L., infection courts, partial resistance, *Sclerotinia sclerotiorum*, sunflower head rot, susceptible period**

INTRODUCTION

Sunflower head rot (SHR), caused by *Sclerotinia sclerotiorum* (Lib.) de Bary, is one of the major diseases affecting this crop worldwide. Under favorable climatic conditions the fungus causes important yield reductions in many countries (e.g., Argentina, China, France, Spain, United States, etc.). In Argentina, the disease is endemic in the southeast of Buenos Aires province, the main sunflower growing area. In epiphytotic years, losses of 50% are commonly registered. Normally, it causes yield reductions by 10-20% (Pereyra and Escande, 1994) and increase in

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impurity and oil acidity, mainly when disease incidence is higher than 10% (Sala *et al.,* 1996; Agüero *et al.*, 1997).

An integrated SHR management is the best approach to decrease its effects. The use of genetic control is one of the main tools for this purpose. Immunity to the fungus has not yet been found in cultivated or wild sunflower plants, although a wide range of susceptibility to attacks under field conditions has been described among sunflower inbred lines, varieties and hybrids (Gulya, 1985; Pereyra *et al.*, 1995). This continuous variation of genotype susceptibility characterized a quantitative or partial resistance according to Parlevliet (1993). SHR resistance has been shown to be polygenic and largely under additive control (Robert *et al.*, 1985; Vear and Tourvieille, 1988). Selection to assemble a maximum number of resistance genes in one genotype appears to be the best method to improve resistance levels.

Many strategies have been used to evaluate the reaction of cultivars to SHR. Different methods to test genotype resistance has been explored with variable results (Vear and Guillaumin, 1977; Tourvieille and Vear, 1984; Bioley *et al.*, 1987; Hemery *et al.*, 1987; Sanlavillec *et al.*, 1987; Ivancia and Andrei, 1988; Tourvieille *et al.*, 1988; Castaño *et al.*, 1989 and 1993; Raducanu and Soare, 1994; Raducanu *et al.*, 1995; Rajender *et al.*, 1996; Blanco *et al.*, 1998; Vasić *et al.*, 1999 and 1999a). Nevertheless, trials under different field environments are the best way to evaluate the partial resistance/susceptibility of genotypes (Castaño *et al.*, 2001). Therefore, disease incidence becomes the component of partial resistance more suitable for this purpose (Sala *et al.*, 1996).

Since 1988, we tested in our lab the reaction of commercial hybrids to SHR. Plants in the field were inoculated with ascospores at the beginning of flowering (R5.2 stage of Schneiter and Miller, 1981) and disease incidence was registered at physiological maturity. These results were validated by comparison with those of natural inoculation in the field at two locations in the region (Quiroz *et al.*, 1998). Sunflower hybrids with stable performance through environments were detected. Two of them were Paraíso-20 and Rancul, the genotypes used in the present work.

Sunflower genotypes may provide different *anthoplane* environments leading to different number of infections courts and/or different length of the susceptible period. Moderately resistant genotypes could have less infections courts resulting in lower disease development. Moreover, these genotypes could have shorter length of susceptibility period. This feature would reduce the opportunity of a pathogen to land and penetrate a host and could explain the partial resistance of a genotype.

Flowering is the susceptible stage to SHR (Says-Lesage and Tourvieille de Labrouhe, 1988; Rajender *et al.*, 1996). The ascospores germinate and penetrate mainly through anthers (Says-Lesage and Tourvieille de Labrouhe, 1988) developing infection courts. In a field crop basis, flowering could last from 8 to 12 days. If integrated control strategies are considered to manage the disease, this period is too long and more precision is needed to decide, for example, when to target biocontrol agents (Escande *et al.*, 2002) or fungicides (Mantecón and Pereyra, 1997).

Furthermore, the knowledge of the duration of the susceptible period could help the decision.

The aim of this work was to evaluate the number of infection courts and the length of the susceptible period in two genotypes with contrasting but stable SHR susceptibility.

MATERIAL AND METHODS

Plant and fungal materials

The sunflower hybrids Paraíso-20, moderately resistant to SHR, and Rancul, susceptible, were used in all trials. These genotypes have a stable performance to SHR across different environments (Quiroz *et al.*, 1998). Repeated field experiments (in 1999 and 2000 to evaluate infection courts and in 1999, 2000 and 2001 to evaluate the length of the susceptible period) were run at Balcarce, Argentina (37° 45'S; 58° 18'W; altitude 130 m). Plants were 0.25 apart in the row. Rows were 0.7 m apart. Sprinkler irrigation was applied during growing season as required to get good plant development. Healthy plants were obtained at flowering stage. No infestation of other fungi than *S. sclerotiorum* occurred during the experiments.

Inoculum source was initially obtained from naturally infected plants at Balcarce production field in 1992. Each year this population was employed to inoculate sunflower field experiments by the ascospore method. Sclerotia obtained from approximately 4000 inoculated plants were induced to germinate carpogenically, as stated by Escande *et al.* (2002). Briefly, sclerotia were collected in the field and stored in paper bags at 13±10°C for three months. For apothecium production, sclerotia were exposed at -18±2°C for 7 days and buried 1 cm deep in humid pasteurized soil until stipe emergence. Then, cultures were incubated at 16±8°C and approximately 2500 lux of continuous daylight. Mature apothecia were harvested and positioned upside down in glass Petri dishes for 4 h to favor ascospore release. Ascospores were stored in Petri dishes at -18°C until use. The viability of ascospores after storage was close to 100%.

Inoculation and disease evaluation

Capitula were inoculated by spraying with 1.1±0.1 ml of a water suspension of ascospores (2500 ml^{-1}), with control plants receiving only a water treatment. In order to ensure high humidity conditions with no thermal stress, capitula were covered with glossy paper bags.

Infection courts. Plants were inoculated at R5.5 stage of Schneiter and Miller (1981) (50% of the disk flowers in anthesis). Capitula remain covered for four days. At 10, 17 and 25 days after inoculation (DAI), heads were removed and cut in 6-mm wide slices to detect infection courts. The number of infection courts per head was recorded. The maximum width and depth of the lesions at each infection court were

measured as indicated in Figure 1. Total number of infection courts per cultivar was registered. Disease incidence based on infection courts at the 25th DAI (DI-IC; ratio between heads with infection courts and total evaluated heads, expressed as percentage) was calculated.

The inoculated control plots remained undisturbed until harvest to assess SHR based on receptacle external symptoms on the 25th DAI. At physiological maturity (approximately 40 DAI), the number of heads with externally visible symptoms and the proportion of rotted area on receptacles of diseased plants were registered. Disease incidence (DI) was calculated as the ratio between the number of plants with externally visible rotted receptacles and the total number of evaluated plants expressed as percentage. Disease severity (DS) was calculated as the ratio between the proportion of rotted area on receptacles and the total number of diseased plants, expressed as percentage.

Length of the susceptible period. Plants were inoculated at the following phenological stages: R4 (the inflorescence begins to open; when viewed from directly above, small ray flowers are visible); R5.2 (the mature ray flowers are fully extended, all disk flowers are visible and 20% of the disk flowers are in anthesis); R5.5 (as previously described); R6 (anthesis is completed and the ray flowers have lost their turgidity and are wilting, the ray flowers may or may not wilt and abscise immediately); R7 (back of the inflorescence has started to turn a light yellow color, the yellowing may begin either at the center of the head near the base of the receptacle or at the periphery adjacent to the bracts); R8 (back of the head is yellow but the bracts remain green, some brown spotting may or may not be present on the back of the head); or R9 (physiological maturity, the bracts become yellow and brown, a large proportion of the back of the sunflower head may begin to turn brown) (Schneiter and Miller, 1981). Heads remained covered until first symptom appearance (approximately 20 DAI). From this time on until one week after inoculation at stage R9, the number of diseased plants was registered once a week. DI was calculated as previously described. The number of days between the first and the last phenological stage in which the inoculation caused disease was considered as the length of the susceptible period (LSP). Because yield and quality decrease significantly when incidence is 10% or higher (Sala *et al.*, 1996; Agüero *et al.*, 1997), the length of the susceptible period based on plots with at least 10% DI was calculated (LSP10).

Experimental design and data analysis

To evaluate the infection courts, a randomized complete block design with four replications and two treatments (Paraíso-20 and Rancul) was used. The experimental unit had ten plant heads. To evaluate the length of the susceptible period, a randomized complete block design with split plots and four replications was used. Treatments were arranged in a factorial of two cultivars (main plots) and seven inoculation stages (subplots). The experimental unit had 15 plants.

Data were analyzed with the procedure GLM of SAS vs. 6.12 (SAS Institute, Cary, NC). Covariance analysis considering the data of the water control plots as covariant was run. Tests of homogeneity of variance (Steel and Torrie, 1992) and a combined analysis of the two or three experiments were performed for infection courts or length of susceptible period, respectively. Comparisons between treatments and their interactions were performed by *F* test or by *LSD* protected by the *F* test (alpha=0.05). Response variables defined as percentages were arcsine-transformed for statistical analysis. Non-transformed data were used in the figures and tables. Correlation analyses were performed between DI at physiological maturity (100±10 DAI, approximately) of plots inoculated at R5.2 or R5.5 stage and LSP or LSP10.

RESULTS

Infection courts

Data of both years had homogeneous variances for all response variables. No disease was observed in the water controls. Minimal DI was detected before 25 DAI in the inoculated-undisturbed control plots (data not shown). On the 25th DAI, non-difference between cultivars in the total number of infection courts was detected (P=0.82, data not shown). Both cultivars had one or two infection courts per head, but heads with only one lesion were more frequent (Table 1).

Table 1: Sunflower heads with one or two infection courts 25 days after inoculation. Plants of Paraíso-20 (moderately resistant) and Rancul (susceptible) were inoculated by the spraying of a water suspension of *Sclerotinia sclerotiorum* ascospores (1.1±0.1 ml head^{-1}, 2500 ascospores ml^{-1}) at R5.2 growing stage. Values are expressed in percentage and are the average of four replications and two years (N=8). The experimental unit had ten plant heads.

Cultivar	One infection court	Two infection courts
Paraíso-20	17	5
Rancul	25	3
	Analysis of Variance	
[a]Pr >F	0.44	0.71
[b]R^2	0.43	0.17
[c]CV%	99	258

[a]Pr > F: F test probability for the difference between cultivars.
[b]R^2: coefficient of determination.
[c]CV%: coefficient of variation.

The infection courts were always observed in flowers located in circles close to the middle of the head radius. At each infection court, the soft rot began in a disk flower ovary and reached the receptacle immediately below (Figure 1). Normally, the ovary of neighbor flowers became partially affected. Lesion size at the receptacle below the infected ovary was 0.8-2.0 cm wide and 0.3-1.0 cm deep. Depth and

width of the lesions did not distinguish cultivars (P=0.89 and P=0.57 for depth and width, respectively).

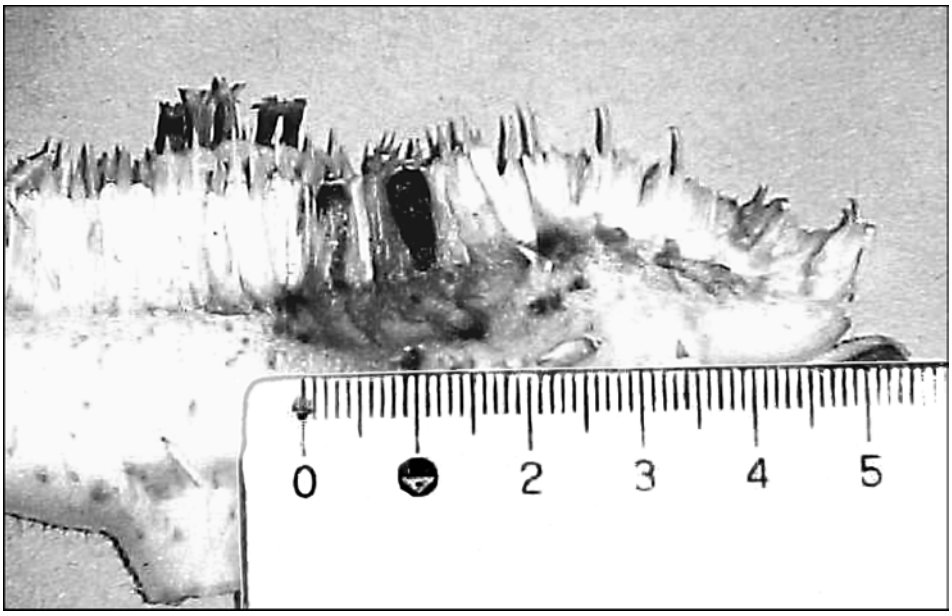

Figure 1: Slice of a sunflower head of the susceptible cultivar Rancul, 25 days after inoculation of Sclerotinia sclerotiorum at the R5.2 growing stage. Plants were inoculated by the spraying of a water suspension of ascospores (1.1±0.1-ml head^{-1}, 2500 ascospores ml^{-1}). The soft rot began in a disk flower ovary (arrow) and reached the receptacle immediately below.

For DI-IC, no interaction between year and cultivar was detected (P=0.27). Differences between Rancul (28%) and Paraíso-20 (22%) were not found (P=0.85). For DI of the inoculated-undisturbed controls, no interaction between year and cultivar was detected (P=0.29) and Rancul was more affected than Paraíso-20 (55% and 30%, respectively; P=0.0028). For DS, the interaction between year and cultivar was not significant (P=0.93) and differences between Rancul (78%) and Paraíso-20 (53%) were not detected (P=0.18).

Length of the susceptible period

There was no effect of the covariant (data of the water control plots) in any trial ($0.13<P<0.83$). A non-reversal interaction between cultivar and year was detected (P=0.06). The LSP was shorter for Paraíso-20 than for Rancul in two out of three years (Table 2). For Paraíso-20, this period included R5.2 and R5.5 in 1999 and only R5.2 in 2000 (Figures A and C). For Rancul, this period lasted from R5.2 to R7 in 1999 and from R4 to R8 in 2000 (Figures 2 B and D). In 2001, when levels of disease were higher, the susceptible period for both cultivars lasted from R4 to R8 (Figures 2 E and F).

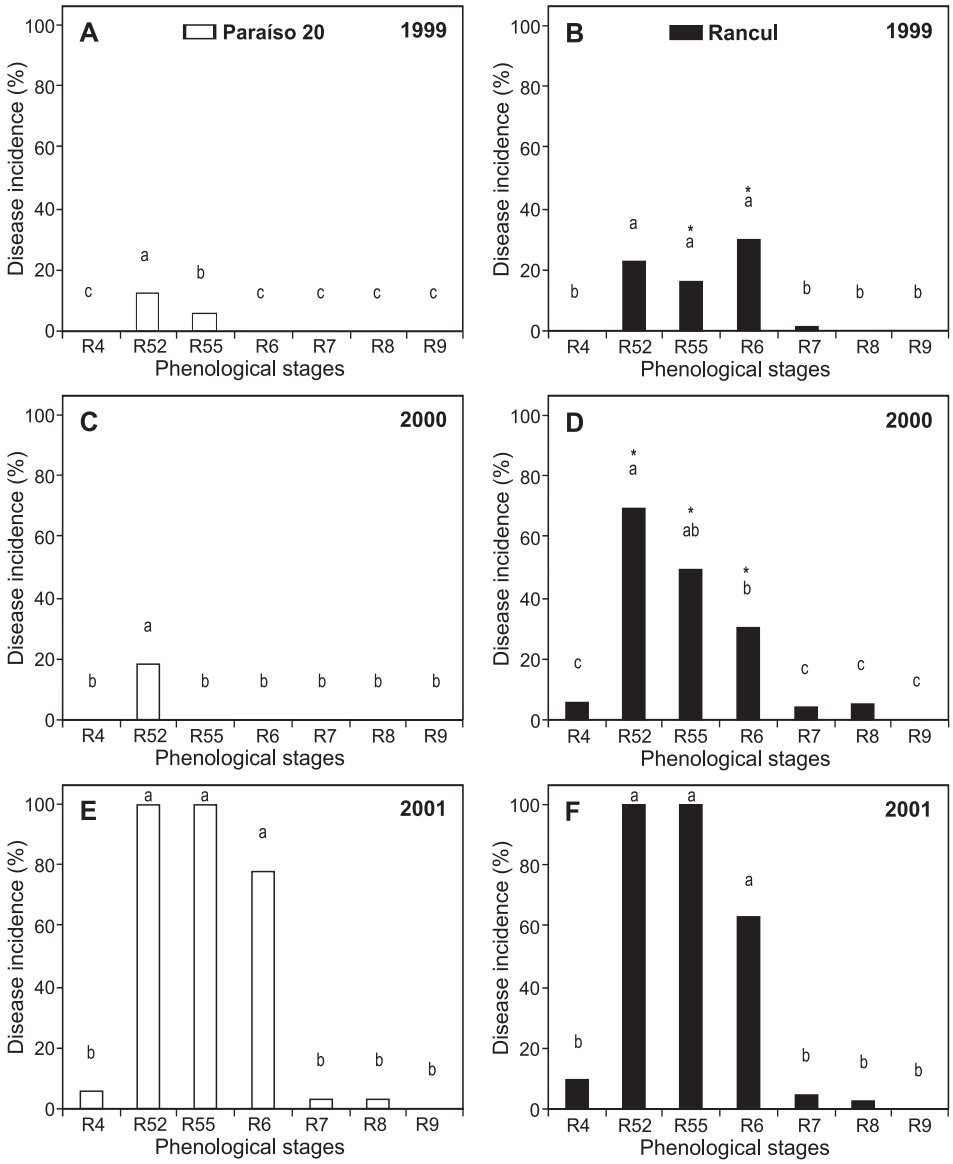

*Figure 2: Disease incidence (ratio between the number of plants with externally visible rotted receptacles and the total number of evaluated plants, expressed as percentage) at physiological maturity for cultivars Paraíso-20 (moderately resistant) (**A**, **C** and **E**) and Rancul (susceptible) (**B**, **D** and **F**), inoculated at R4, R5.2, R5.5, R6, R7, R8 or R9 stages (Schneiter and Miller, 1981), by the spraying of a water suspension of Sclerotinia sclerotiorum ascospores (1.1±0.1 ml head^{-1}, 2500 ascospores ml^{-1}). Values are the mean of four replications in each year. Means of each cultivar and year with the same letter are not significantly different (LSD, alpha=0.05). *Indicates higher disease incidence in cultivar Rancul than in Paraíso-20 inoculated at the same corresponding year and stage ($0.0036 < P < 0.03$; $0.76 < R^2 < 0.96$).*

For LSP10, no interaction between cultivar and year was detected (P=0.54) and the LSP10 was shorter for Paraíso-20 (Table 2). LSP10 for Rancul involved R5.2, R5.5 and R6 stages (Figures B, D and F). For Paraíso-20, LSP10 involved only the stage R5.2 in the first two years (Figures A and C); but when the environment was very favorable to disease development, it reached high disease levels at each flowering sub-stages (Figure 2 E). The correlation coefficients between DI of the plots inoculated at R5.2. and LSP or LSP were 0.59 (P=0.0037) and 0.56 (P=0.0069), respectively. For the plots inoculated at R5.5, these correlation coefficients were 0.49 for both variable relationships (P=0.02).

Table 2: Length of the susceptible period to the infection by *Sclerotinia sclerotiorum* (LSP) for cultivars Paraíso-20 (moderately resistant) and Rancul (susceptible). LSP was considered as the number of days between the first and the last phenological stage in which the inoculation caused disease. LSP10 was based on plots with at least 10% disease incidence. Disease incidence was based on plants with externally visible rotted receptacles at physiological maturity. Plants were inoculated by the spraying of a water suspension of *S. sclerotiorum* ascospores (1.1±0.1 ml head^{-1}, 2500 ascospores ml^{-1}). Inoculation was performed at R4, R5.2, R5.5, R6, R7, R8 or R9 stages (Schneiter and Miller, 1981). LSP values are the mean of four replications and LSP10 values are the mean of four replications and three years. LSP and LSP10 are expressed in days.

Cultivar	LSP [a]			LSP10[b]
	1999	2000	2001	
Paraíso-20	11	15	24	9
Rancul	19	37	29	22
LSD	7	19	22	8
		Analysis of Variance		
[c]Pr > F	0.036	0.032	0.416	0.009
[d]R^2	0.95	0.88	0.34	0.77
[e]CV%	21	31	24	54

[a]Interaction between year and cultivar for LSP (P=0.06)
[b]Interaction between year and cultivar for LSP10 (P=0.54)
[c]Pr > F: F test probability.
[d]R^2: coefficient of determination.
[e]CV%: coefficient of variation.

DISCUSSION

On the 25th DAI, each infected sunflower head had only one or two infection courts. Why so few infection courts if we inoculated approximately 1700 flowers on the flowering disc with 2500 ascospores? Says-Lessage and Tourvieille de Labrouhe (1988) stated that anthers are the organs where ascospores germinate and penetrate. If we consider that the inoculation was done at the R5.5 stage and that the infected flowers were always located in the middle zone of the head radius, only two or three flower circles were susceptible to infection (equivalent to 100 or 200 flowers per head). A possible reason for the few infection sites could be an activation of

the host resistant mechanisms by the penetration of germinative tubes (Agrios, 1997, Prats-Pérez et al., 2000).

Another fact is that at the end of the crop season, the inoculated-undisturbed control plots had higher DI than the DI-IC detected on the 25th DAI (55 vs. 28% for Rancul and 30 vs. 22% for Paraíso-20, respectively). Probably, the early evaluation did not allow detection of some infection courts. However, if we had delayed the observation, the spread of the soft rot in the receptacles would not have allowed them to be distinguished. Differences in number of infection courts did not explain differences in SHR susceptibility between Rancul and Paraíso-20 (Table 1). Therefore, the number of infection courts would not account for the partial resistance of Paraíso-20 to SHR.

Considering LSP or LSP10, the interaction between genotype and environment (different years) was significant. In the first two years, susceptible periods were shorter for Paraíso-20 than for Rancul. In 2001, in which disease incidence reached 100%, differences between cultivars were not detected (Figures 2 E and F, Table 2). It is well known that the environment has a large effect on partial resistance (Castaño et al., 2001). An environment highly conducive to disease could mask the partial resistance of a genotype and the poor expression of resistance in environments highly favorable for SHR was also noted by Pereyra et al. (1995). However, this high incidence in moderately resistant cultivars growing in the field has not been reported before (Quiroz *et al*., 1998). Then, disease levels registered in 2001 are not frequent under field natural infections.

The period of susceptibility included different phenological stages according to cultivar susceptibility. Pierre and Regnault (1985) studied the influence of the sunflower growth stage on head infection but only one susceptible cultivar was considered. The stages involved in LSP10 for Rancul agree with those of these authors. Paraíso-20 was susceptible for a shorter period than Rancul and this could be a reason for its lower disease incidence under natural infections in field trials (Quiroz *et al*., 1998).

Sunflower breeding programs normally inoculate at R5.2. For the genotypes included in our work, final disease incidence by inoculating at this stage and duration of susceptibility were correlated. Whether these two characters are under the control of different genes/groups of genes or not require more studies. According to our results, LSP and LSP10 could account for the better performance of Paraíso-20 to SHR. This trait could be a new source to increase the level of partial resistance to SHR. The length of the susceptible period has not been studied as a component of partial resistance previously. To our knowledge, this is the first report of the relationship between susceptible periods and cultivar susceptibility to SHR.

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Abbreviations

DAI - days after inoculation; DI - disease incidence: ratio between the number of plants with externally visible rotted receptacles and the total number of evaluated plants, expressed as percentage; DI-IC - disease incidence based on infection courts: ratio between heads with infection courts and total evaluated heads, expressed as percentage; DS - disease severity: ratio between the proportion of rotted area on receptacles and the total number of diseased plants, expressed as percentage; LSP - length of susceptible period when disease incidence is higher than zero percent, expressed in days; LSP10 - length of susceptible period when disease incidence is higher than ten percent, expressed in days; SHR - sunflower head rot.

REFERENCES

Agrios, G.N., 1997. Plant Pathology. San Diego, California, USA: Academic Press.

Agüero, M.E., V.R. Pereyra, F.S. Laich and A.R. Escande, 1997. Efecto de la incidencia de podredumbre húmeda del capítulo de girasol (*Sclerotinia sclerotiorum*) sobre el rendimiento en grano y calidad comercial. *In*: Asoc. Latinoamericana de Fitopatología, (Eds.) *IX Congreso Latinoamericano de Fitopatología.* Montevideo, Uruguay 217.

Bioley, J.P., M. Jay and J.L. Fiasson, 1987. *Sclerotinia sclerotiorum* in sunflower: study of a biochemical way for looking for resistance. 1: Biochemical mechanisms of the secondary mechanism in the reaction of cultivated plants to fungi pathogens. Infs. Tech. C.E.T.I.O.M. 101: 7-19.

Blanco, F.A., M.E. Bazzalo and L. de la Canal, 1998. A growth room inoculation method to detect different levels of sunflower susceptibility to *Sclerotinia sclerotiorum*. A seedling test for sunflower susceptibility to *S. sclerotiorum*. Oleagineux Corps Gras Lipides 5 (1): 61-64.

Castaño, F., F. Vear and D. Tourvieille de Labrouhe, 2001. Horizontal resistances in sunflower: a review of a workshop at the 15th Int. Sunflower Conf. OCL 8 (3): 211-215.

Castaño, F., F. Vear and D. Tourvieille de Labrouhe, 1989. Simultaneous utilisation of several tests in breeding for *Sclerotinia sclerotiorum* resistance in sunflower. Infs. Tech. C.E.T.I.O.M. 107: 14-20.

Castaño, F., F. Vear and D. Tourvieille de Labrouhe, 1993. Resistance of sunflower inbred lines to various forms of attack by *Sclerotinia sclerotiorum* and relations with some morphological characters. Euphytica 68 (1-2): 85-98.

Escande, A.R., F.S. Laich and M.V. Pedraza, 2002. Field Testing of *Trichoderma* spp. to manage sunflower head rot (*Sclerotinia sclerotiorum*). Pl. Path. 51 (3): 346-350.

Gulya, T.J., 1985. Registration of five disease-resistance sunflower germoplasms. Crop Sci 25: 719-720.

Hemery, M.C., D. Tourvieille de Labrouhe, M. Jay and F. Vear, 1987. *Sclerotinia sclerotiorum* in sunflower: the study of a biochemical way for looking for resistance. 2: Looking for phenolic markers involved in sunflower-resistance to Sclerotinia. Infs. Tech. C.E.T.I.O.M. 101: 20-29.

Ivancia, V. and E. Andrei, 1988. Contributions to the study of testing methods of sunflower resistance to the attack of *Sclerotinia sclerotiorum* (Hb.) de By. Cercetari agricole in Moldova (Romania) 3 (83): 57-59.

Mantecón, J.D. and V.R. Pereyra, 1997. Integrated control methods for managing sunflower head rot in Argentina. Int J Pest Mgmt 43: 143-144.

Parlevliet, J.E., 1993. Selecting components of partial resistance. *In*: Stalkes, H.I. and J.P. Murphy (Eds.), Plant Breeding in the 1990's. CAB International.

Pereyra, V.R. and A.R. Escande, 1994. Enfermedades Del Girasol. Guía Para Productores Del Sudeste Bonaerense. Balcarce, Argentina: Unidad Integrada Instituto Nacional de Tecnología Agropecuaria-Universidad Nacional de Mar del Plata.

Pereyra, V.R., S.M. Prioletta and A.R. Escande, 1995. Influencia del ambiente sobre el comportamiento de cultivares de girasol ante la podredumbre del capítulo causada por ascosporas de *Sclerotinia sclerotiorum*. In: Asociación Argentina de Oleaginosos, (Ed.), *Segunda Reunión Nacional de Oleaginosos, 1995*. Pergamino, Argentina, 2: 28-34.

Pierre, J.G. and Y. Regnault, 1985. Determination au champ du stade vegetatif sensible du capitule de tournesol aux attaques de *Sclerotinia sclerotiorum*. *In*: Proc. 11th Int. Sunflower Conf. 1985. Mar del Plata, Argentina 2, 341-346.

Prats-Pérez, E.; M.E. Bazzalo, A. León and J.V. Jorrín-Novo, 2000. Accumulation of soluble phenolic compounds in sunflower capitula correlates with tolerance to *Sclerotinia sclerotiorum*. *In*: Proc. 15th Int. Sunflower Conf. 2000. Toulouse, France 2: K35-41.

Quiroz, F.; V. Pereyra and A. Escande, 1998. Comportamiento de cultivares de girasol frente a infecciones de *Sclerotinia sclerotiorum* inducidas por pulverización de ascosporas o por inóculo natural. *In*: Asociación Argentina de Oleaginosos, (Ed.), Tercera Reunión Nacional de Oleaginosos 1998, Bahía Blanca, Argentina, 1: 35-36.

Raducanu, F. and G. Soare, 1994. An approach of in vitro testing sunflower for resistance to *Sclerotinia sclerotiorum* (Lib.) de Bary. Rom. Agric. Res. 2: 35-39.

Raducanu, F., G. Soare and D.S. Craiciu, 1995. Screening the reaction of sunflower genotypes to *Sclerotinia sclerotiorum* (Lib.) de Bary by anther culture. Rom. Agric. Res. 3: 1-5.

Rajender, S.; N.N. Tripathi and R. Singh, 1996. Vulnerability of sunflower (*Hellianthus annus* L.) head to *Sclerotinia sclerotiorum* (Lib.) de Bary infection. Indian J. Agric. Res. 30 (1): 22-26.

Robert, N.; F. Vear and D. Tourvieille de Labrouhe, 1985. L'heredité de la résistance au *Sclerotinia sclerotiorum* chez le tournesol. I. Etude des réactions à deux tests mycéliens. Agronomie 7: 423-429.

Sala C.A., A.N. Vázquez, A.B. Romano and S. Piubello, 1996. Yield losses in sunflower (*Helianthus annus*) due to head rot caused by *Sclerotinia sclerotiorum*. (Lib.) de Bary. Helia 19: 95-104.

Sanlavillec, C., J. Guiard and M. Jay, 1987. *Sclerotinia sclerotiorum* in sunflower: the study of a biochemical way for looking for resistance. 3: Utilization of the linear model of variance analysis with two controlled factors to study variability of phenolic metabolism in sunflower lines. Infs. Tech. C.E.T.I.O.M. 101: 30-47.

Says-Lesage, V. and D. Tourvieille de Labrouhe, 1988. Recherche des sites de pollution et d' infection des fleurons de tournesol, in situ, par les spores de *Sclerotinia sclerotiorum*. Infs. Tech. C.E.T.I.O.M. 102: 3-13.

Schneiter, A.A. and J.F. Miller, 1981. Description of sunflower growth stages. Crop Sci 21: 901-903.

Steel, R.G.D. and J.H. Torrie, 1992. Bioestadística: Principios y Procedimientos. Segunda Edición. McGraw-Hill/Interamericana de México, S. A. de C. V.

Tourvieille de Labrouhe, D.; M.H. Pham-Delegue, F. Vear, R. Marilleau, Y. Loublier, P. Douault and J.P. Philippon, 1988. Comparison between the susceptibility of sunflower to *Sclerotinia* infection of the capitulum and nectar and pollen production. Helia 11: 59-63.

Tourvieille de Labrouhe, D. and F. Vear, 1984. Comparison de méthodes d'estimation de la résistance au *Sclerotinia sclerotiorum*. Agronomie 4: 517-525.

Vasić, D., G. Alibert and D. Škorić, 1999. In vitro screening of sunflower for resistance to *Sclerotinia sclerotiorum* (Lib.) de Bary. Helia 22(31): 95-103.

Vasić, D., D. Škorić and A. Gilbert, 1999a. The use of protoplast culture and oxalic acid for testing sunflower for resistance to white rot [*Sclerotinia sclerotiorum*]. Proc. 40th Conf. Production and Processing of Oilseeds, 147-150.

Vear, F. and J. Guillaumin, 1977. Etude de méthodes d'inoculation de torunesol par *Sclerotinia sclerotiorum* et application à la sélection. Ann. Amelior. Plantes 28: 523-537.

Vear, F. and D. Tourvieille de Labrouhe, 1988. Heredity of resistance to *Sclerotinia sclerotiorum* in sunflowers II. Study of capitulum resistance to natural and artificial ascospore infections. Agronomie 8: 503-508.

LUGARES DE INFECCIÓN Y DURACIÓN DEL PERÍODO DE SENSIBILIDAD, RELACIONADO CON LA RESISTENCIA DE LA PODREDUMBRE HÚMEDA DEL CAPÍTULO DE GIRASOL (*Sclerotinia sclerotiorum*)

RESUMEN

La podredumbre húmeda del capítulo de girasol (PHC) (*Sclerotinia sclerotiorum* (Lib.) de Bary) es una de las enfermedades más importantes que atacan a esta especie vegetal en el mundo. A pesar de los informes sobre diferentes niveles de sensibilidad en los genotipos de girasol, no hay pruebas de que ni en un solo híbrido comercial exista una resistencia total a esta enfermedad. El objetivo de este trabajo fue determinar el número de sitios de infección y la duración del período de sensibilidad en dos genotipos con una sensibilidad opuesta pero estable, a PHC. En los experimentos que se llevaban a cabo durante tres anos, estábamos investigando el híbrido de girasol moderamente resistente, Paraiso-20 y el híbrido sensible Rancul. Las plantas fueron inoculadas con ascosporas, mediante spray. En los dos híbridos, en cada cabeza de girasol infectada, se encontró sólo uno o dos sitios de infección. Entre los híbridos se establecieron diferencias en cuanto a la duración del período de sensibilidad, a base de la intensidad de la enfermedad mayor de cero (LSP) y mayor de 10% (LSP10) en dos a tres anos de investigaciones. El LSP era más largo en el híbrido Rancul (28 días) que en el híbrido Paraíso-20 (17 días). El LSP10 también resultó más largo en el híbrido Rancul (22 días) que en el híbrido Paraíso-20 (9 días). Se está considerando la posibilidad de utilizar estas dos características como componentes de una resistencia parcial.

LIEUX D'INFECTION ET LONGUEUR DE PÉRIODE DE SENSIBILITÉ CONCERNANT LA RÉSISTANCE DE POURRITURE DE CAPITULE DE TOURNESOL (*Sclerotinia sclerotiorum*)

RESUME

La pourriture de capitule de tournesol (*Sclerotinia sclerotiorum* (Lib.) de Bary) est la maladie la plus importante qui attaque cette espèce végétale dans le monde. Malgré les rapports sur les niveaux différents de sensibilité parmi les génotypes de tournesol, il n'y a pas de preuves de résistance complète dans aucun hybride commercial. Le but de cette étude était de mettre au point le nombre de lieux d'infection et la période de sensibilité de deux génotypes d'une sensibilité contrastée mais stable concernant la pourriture de capitule. Dans les tests pendant la période de trois ans, l'hybride de tournesol de résistance tempérée Paraiso-20 et l'hybride sensible Rancul étaient examinés. Les plantes étaient inoculées des ascospores par application de spray. Chez les deux génotypes, sur chaque capitule infecté, il n'y avait qu'un ou deux lieux d'infection. Entre les hybrides, les différences concernant la période de sensibilité basée sur l'incidence de maladie plus élevée de zéro (LSP) ou plus élevée de 10% (LSP10) en deux ans de la période de recherche durant trois ans. LPS était plus longue pour l'hybride Rancul (28 jours) que pour l'hybride Paraiso-20 (17 jours). LSP10 était plus longue pour l'hybride Rancul (22 jours) que pour l'hybride Paraiso-20 (9 jours). La possibilité d'utiliser ces deux traits comme components de résistance partielle est un sujet de discussion.