https://doi.org/10.18406/2316-1817v17nunico20251913 e-ISSN 2316-1817

Pathogenic variability in monoascosporic strains of *Sclerotinia sclerotiorum* in soybean

Jéssica Gentil Lima[1], Adriano Teodoro Bruzi[2], Elaine Aparecida de Souza[3], Taine Teotônio Teixeira da Rocha[4], Pablo de Souza Arantes[5], Elaine Cristina Batista[6]

[1] Universidade Federal de Lavras - E-mail: gentiljessica@yahoo.com.br
[2] Universidade Federal de Lavras – E-mail: adrianobruzi@ufla.br
[3] Universidade Federal de Lavras – E-mail: easouza@ufla.br
[4] Universidade Federal de Lavras – E-mail: taine.rocha1@estudante.ufla.br
[5] Universidade Federal de Lavras – E-mail: pablosousaarantes95@gmail.com
[6] Universidade Federal de Lavras – E-mail: ebatista@aointl.com

Received in: 29/04/2024
Accepted in: 30/10/2024

Abstract

This work was developed with the objective of evaluating the pathogenic variability among monoascosporic strains of the same *S. sclerotiorum* isolate using the straw test in soybean cultivars. A total of 20 cultivars from the Soybean Germplasm Bank of the Federal University of Lavras (UFLA) and five monoascosporic *S. sclerotiorum* strains were included. A greenhouse experiment was carried out to assess the variability in aggressiveness among monoascosporic strains. Plants at the R1 stage were inoculated by the straw test. The experiment was implemented in a completely randomized design with three replicates. Evaluations were made with a graduated ruler at seven, 14, and 21 days after inoculation according to the proportion of damaged area. The area under the disease progress curve (AUDPC) was estimated. Monoascosporic strains 7.3 and 7.4 were the most aggressive, and the BRSMG 790A and BRSMG 850GRR cultivars had more stable resistance to the different monoascosporic strains. There was variability between the monoascosporic strains, and strains 7.3 and 7.4 are preferred for inoculation in breeding programs aimed at obtaining soybean cultivars resistant to *S. sclerotiorum.*

Key words: *Glycine max*, genetic resistance, straw test, white mold.

Introduction

White mold is a disease caused by the ascomycete fungus *Sclerotinia sclerotiorum* (Lib.) Bary, that can devastate several crops, including soybean. This pathogen produces resistant structures called sclerotia, which can survive in the soil for more than five years (STEADMAN; BOLAND, 2005). In soybean, white mold disease is also called Sclerotinia stem rot and causes significant damage to grain production and quality. Under favorable conditions, such as high humidity and mild temperatures, production losses of up to 70 % may occur. It is estimated that approximately 23 % of the Brazilian soybean production area is infested by this pathogen, comprising approximately 7.7 million hectares that require the adoption of integrated disease control measures (MEYER et al., 2016).

Infection of soybean plants by *S. sclerotiorum* occurs through fungal ascospores, which are produced in the apothecia, resulting from the carpogenic germination of sclerotia. Management of this disease involves the use of seed treatment, crop rotation and biological control, with fungi of the genus *Trichoderma* (HARMAN et al., 2004). Crop rotation with non-host species, especially grasses, can help reduce inoculum in an area and the presence of straw prevents plants from contacting the infested soil, hindering the formation of apothecia and dispersion of ascospores and favoring the action of microorganisms antagonists (PAULA JÚNIOR et al., 2010). However, the most economical and effective way to control the disease is the adoption of soybean cultivars resistant to the pathogen.

Studies were performed to develop soybean cultivars resistant to *S. sclerotiorum* through genetic diversity show that there is polymorphism among isolates from Brazil, although all the isolates are from the same mycelial compatibility group (MEINHARDT et al., 2002). In addition, different levels of aggressiveness have been reported among isolates from Spain (PASCUAL et al., 2010) and the United States (KULL et al., 2003). In Brazil, the diversity of *S. sclerotiorum* isolates was investigated in a study of resistance in common bean (LEHNER et al., 2016a). Although the general aggressiveness of the 20 isolates in susceptible or resistant cultivars was similar, subtle differences in the performance of the isolates were detected between cultivars. Thus, resistance evaluations should take into account the full extent of regional variation within the pathogen population to ensure the development and release of a cultivar with robust resistance. The diversity of pathogens should be thoroughly investigated to identify adequately representative isolates for use in resistance evaluations.

Finding new sources of resistance to white mold requires reliable inoculation and evaluation techniques as well as an understanding of the pathogenic variability in *S. sclerotiorum*. The inoculation method can influence the response of soybean genotypes to disease (KULL et al., 2003). One of the most commonly used techniques is the straw test, which was first described for bean plants by Petzoldt; Dickson (1996) and adapted for soybean by Auclair et al., (2004). In the straw test, a fungus is inoculated into the main stem tissue of a plant, and evaluations are made using a scale of scores or the length of the resulting lesion.

Several studies have been implemented targeting soybean cultivars resistant to white mold, utilizing the straw test (AUCLAIR et al., 2004; HULLER et al., 2016). However, pathogenic variability has not been considered in these studies, given the limited knowledge about the variability in *S. sclerotiorum* aggressiveness in soybean-producing areas in Brazil. Another point to be considered is the genetic unit used in studies of pathogenic variability. Isolates obtained from sclerotia have been the most commonly used genetic unit in previous studies for the characterization of individuals (HAMBLETON et al., 2002; MERT-TURK et al., 2007; CLARKSON et al., 2013). However, it has been shown that the use of sclerotia to identify individuals may not be appropriate and should be avoided because sclerotia may be formed by genetically distinct hyphae (LEHNER et al., 2015). Monoascosporic strains most closely represent individuals because each ascospore has two genetically identical nuclei (WEBSTER; WEBER, 2007). Thus, these strains enable the quantification of pathogenic variability as well as aggressiveness.

Therefore, this work was developed with the objective of assessing the existence of pathogenic variability among monoascosporic strains of the same *S. sclerotiorum* isolate using the straw test in soybean cultivars.

Material and methods

The experiments were carried out at the Plant Disease Resistance Laboratory and greenhouse of the Biology Department of the Federal University of Lavras (UFLA), state of Minas Gerais Brazil. Twenty cultivars from the Soy Germplasm Bank of the Federal University of Lavras (UFLA) were investigated, previously classified as resistant or susceptible by Garcia and Juliatti (2012), and the ten cultivars with the highest levels of resistance and susceptibility were used. (Table 1). The experiment was conducted in a completely randomized design, with three replicates in a factorial arrangement consisting of 20 cultivars and five monoascosporic strains. Each pot held one plant and was considered a plot.

Table 1. Soybean cultivars with the highest levels of resistance and susceptibility to *S. sclerotiorum*

Cultivar[1]	Source	Cultivar[2]	Source
1 Emgopa 316	Emater-GO	11 7166RSF IPRO	GDM
2 Emgopa 315	Agência Rural	12 BRS 213	Embrapa
3 BRS Milena	Embrapa	13 NS 7338 IPRO	Nidera
4 BRSMG 790A	Embrapa	14 BRSMG Garantia	Embrapa
5 BRSMG 850GRR	Embrapa	15 MG/BR 46 (Conquista)	Embrapa
6 BRS Baliza RR	Embrapa	16 BRS Silvânia RR	Embrapa
7 BRS Favorita RR	Embrapa	17 M-SOY 8001	D&PL Brazil
8 BRSGO Luziânia	Embrapa	18 M-SOY 6101	D&PL Brazil
9 M-SOY 8000RR	D&PL Brazil	19 M-SOY 8329	D&PL Brazil
10 BRSMG 68 (Vencedora)	Embrapa	20 TMG123RR	TMG

[1]Resistant cultivars (1 to 10); [2]Susceptible cultivars (11 to 20).
Source: authors (2024)

To obtain monoascosporic strains of *S. sclerotiorum*, the sclerotia of isolate UFLA 44 were placed in germination boxes containing an autoclaved mixture of sand, soil and substrate. Germination boxes were incubated at 19°C under a 12-hour photoperiod for 40 days to aid in the production of apothecium. Water was sprinkled over the mixture every day to maintain the high humidity required for carpogenic germination. A mature apothecium was collected from each sclerotium and placed in an Eppendorf tube containing distilled water. Ascospores were randomly collected using a microscope and transferred to Petri dishes containing potato dextrose agar (PDA) medium. Through this micromanipulation, five monoascosporic strains were obtained (7.1, 7.2, 7.3, 7.4 and 7.5).

In a greenhouse, the variability in severity between monoascosporic strains was evaluated. Plants were grown in plastic pots containing a mixture of clay and substrate until they reached the R1 stage, which marks the beginning of flowering. To obtain mycelium for inoculation, agar discs colonized with mycelium from each monoascosporic strain were transferred to Petri dishes containing PDA medium and kept at 22 ± 3°C under a photoperiod of 12 h for five days. The method used to inoculate the plants was the straw test, which consists of cutting the apex of the main stem of the plant and inoculating it with tips containing fungal mycelia. The tips are used to cut and remove the agar disc from the plate so that the mycelia are in contact with the cut stem (AUCLAIR et al., 2004).

Evaluations were performed seven days after inoculation using a graduated ruler to measure the proportion of damaged area. To monitor the progress of the disease, the plants were evaluated for two more weeks, totaling three evaluations, at seven, 14, and 21 days after inoculation. Subsequent evaluations were used to calculate the area under the disease progress curve (AUDPC), according to the methodology proposed by Shaner and Finney (1977), equation 1:

$$AUDPC = \sum_{i=1}^{n} \left[\frac{Y_{i+1} + Y_i}{2} \cdot \left(T_{i+1} - T_i \right) \right] \quad (1)$$

where:
Y_i is the disease severity at evaluation time i (i = 1..., n);
Y_{i+1} is the disease severity at evaluation time i +1;
T_i is evaluation time i, in number of days after plant emergence; and
T_{i+1} is evaluation time i+1.

The AUDPC estimates were subjected to analysis of variance (ANOVA), according to the following statistical model, equation 2:

$$Y_{ij} = \mu + c_i + m_j + cm_{ij} + e_{ij} \quad (2)$$

where:
Y_{ij} is the observation of cultivar and monoascosporic strain j;
μ is the overall mean;
c_i is the effect of cultivar i (i=1, 2, 3, ...20);
m_j is the effect of monoascosporic strain j (j=1, 2, 3, ...5);
cm_{ij} is the effect of the interaction between cultivar i and the monoascosporic strain j; and
e_{ij} is the experimental error associated with observation Y_{ij}.

The ANOVA was performed considering the AUDPC estimates. The means were compared using a Scott-Knott test (1974) ($p<0.05$). The classification of cultivars was based on patterns of resistance and susceptibility already described in the literature (GARCIA; JULIATTI, 2012; CASTRO et al., 2016). Cultivars with AUDPC values lower than those obtained for the Emgopa 316 cultivar were considered resistant, whereas cultivars with AUDPC values higher than those obtained for cultivar TMG123RR were considered susceptible. The cultivars with AUDPC values that fell between those of these two cultivars were considered moderately resistant. A GGE biplot analysis (genotype main effects + genotype environment interaction) was also performed to better compare the aggressiveness of monoascosporic strains and the behavior of cultivars.

A coincidence index, proposed by Hamblin and Zimmermann (1986), with a selection intensity of 20 %, was calculated to test the coincidence of the resistant cultivars according to each monoascosporic strain, according to the equation 3:

$$CI = \frac{A - C}{M - C} \times 100 \quad (3)$$

where:
A is the number of selected superior cultivars common to different monoascosporic strains;
C is the number of coincident cultivars due to chance; and
M is the total number of selected cultivars.

The selection accuracy (r_{gg}) was estimated according to expression 4 (RESENDE, 2007):

$$r_{gg'} = \sqrt{1 - \frac{1}{F}} \quad (4)$$

where:
F is the value of the Snedecor F-test for the effect of treatments on the ANOVA.

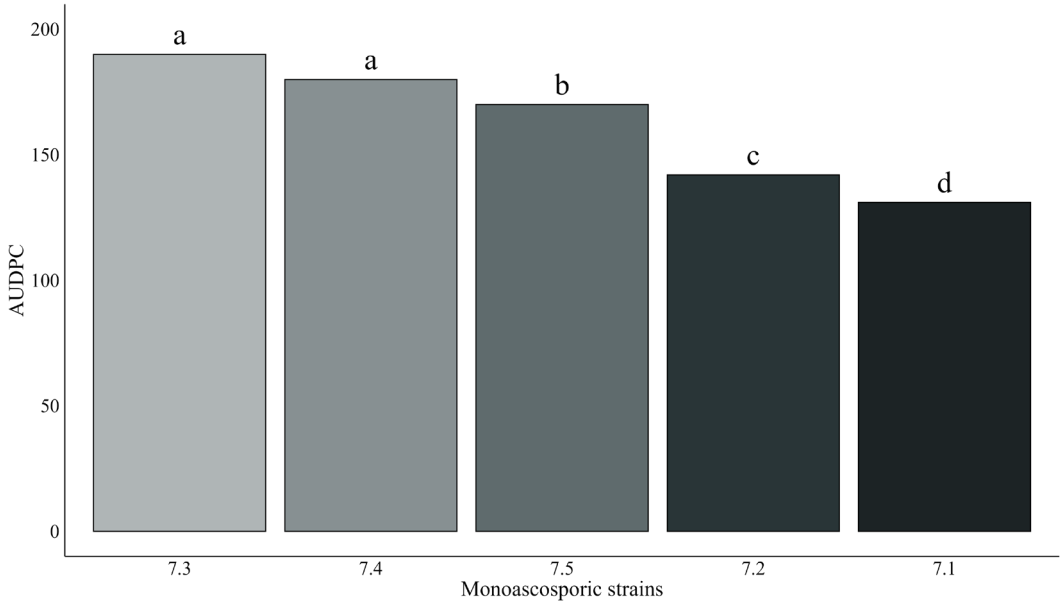
Spearman correlation was calculated to measure the degree of accuracy with which the different monoascosporic strains classified the cultivars. All analyses were performed using R software (R CORE TEAM, 2017).

Results and discussion

The experiment was concluded with good precision, indicated by the magnitude of the coefficient of variation (CV, 18.35 %) and high selection accuracy ($r_{gg'}$, 0.98). The cultivar vs monoascosporic strains interaction and the breakdown of this interaction were significant, enabling inferences about the pathogenic variability between the different monoascosporic strains.

The mean aggressiveness of each monoascosporic strain, considering all cultivars, was estimated. Greater aggressiveness was observed for monoascosporic strains 7.3 and 7.4. In turn, strain 7.1 was less efficient in causing symptoms in the cultivars (Figure 1). The behavior of the cultivars in response to the different monoascosporic strains was estimated in the breakdown of the ANOVA (Figure 2). Monoascosporic strain 7.5, on average, had higher AUDPC values, and together with strain 7.3, it better discriminated the cultivars, i.e., allowed the grouping of cultivars into a greater number of phenotypic classes. Noncoincident behavior of the cultivars was observed in the different inoculations (Figure 2). Note that the classification into the resistance classes was very divergent. This again indicates the variability among monoascosporic strains.

Figure 1. The mean aggressiveness of the monoascosporic strains by area under the disease progress curve (AUDPC) values.



The means followed by the same letter belong to the same group according to a Scott-Knott test at a 5 % probability level.

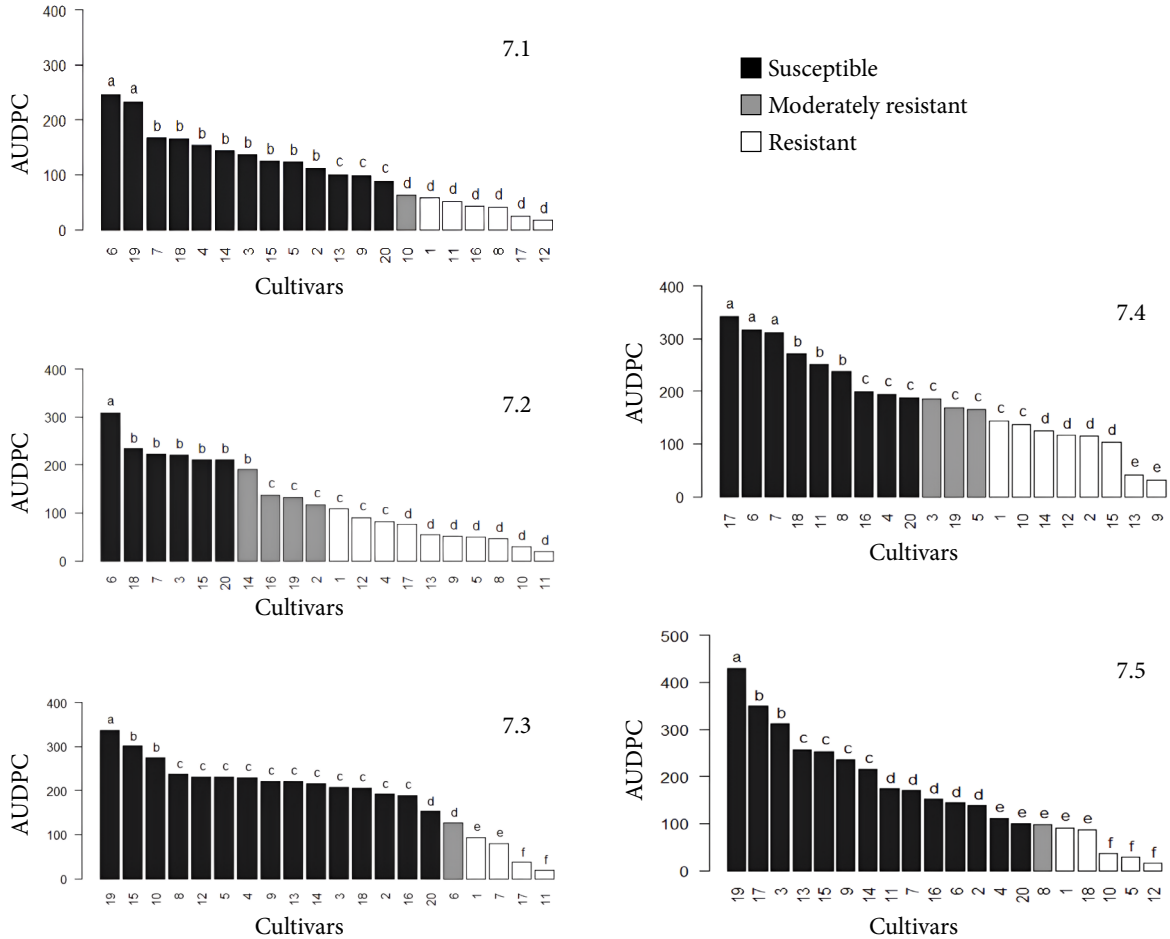
Source: authors (2024)

The correlation in the classification was generally moderate and was significant between only monoascosporic strains 7.1 and 7.2, which were classified as the least aggressive strains. In turn, among the monoascosporic strains that caused more severe symptoms, i.e., strains 7.3 and 7.4, the correlation was negative (Table 2). This way, that cultivar classification depends on the specific monoascosporic strains used for inoculation. The coincidence index also enables the inference of the variability in monoascosporic strains (Table 2). There was no coincidence between the cultivars classified as resistant when comparing monoascosporic strains 7.1, 7.2, and 7.3 with 7.4. Conversely, when the comparison was performed for the most susceptible cultivars, the coincidence index reached 75 %. Thus, it is difficult to make inferences about the level of resistance of soybean genotypes.

The graphical analysis of principal components via GGE biplot grouped the cultivars according to a resistance pattern for the monoascosporic strains. The cultivars of the same quadrant (6, 7, 17, and 18; 11 and 20; 1, 5, 8, and 16; 2, 4, 10, and 12; and 3, 13, 14, 15, and 19) probably have similar resistance alleles (Figure 3a). The GGE biplot also enabled an inference to be drawn about the interactions between the monoascosporic strains and each cultivar. Thus, the cultivars that contributed most to the interaction were 19, 6, 7, 13, and 9. Cultivars 5, 8, and 4 were the most stable (Figure 3 a). The graphical analysis also confirmed the greater aggressiveness of monoascosporic strains 7.3 and 7.4 as well as the lower aggressiveness of strain 7.1 (Figure 3b)

Studies on the aggressiveness of *S. sclerotiorum* isolates have been performed for a large number of genotypes (ABREU; SOUZA, 2015; LEHNER et al., 2016; LEHNER; PAULA JUNIOR, 2016; LIU et al., 2018; SILVA et al., 2014). However, these studies were performed, in most cases, for bean crops. To date, no studies have found pathogenic variability between monoascosporic strains of the same isolate in soybean crops.

Figure 2. Performance of the cultivars by mean area under the disease progress curve (AUDPC) value for each monoascosporic strain.



The means followed by same letter belong to the same group according to a Scott-Knott test at a 5 % probability level. 1. Emgopa 316; 2. Emgopa 315; 3. BRS Milena; 4. BRSMG 790A; 5. BRSMG 850GRR; 6. BRS Baliza RR; 7. BRS Favorita RR; 8. BRSGO Luziânia; 9. M-SOY 8000RR; 10. BRSMG 68 (Vencedora); 11. 7166RSF IPRO; 12. BRS 213; 13. NS 7338 IPRO; 14. BRSMG Garantia; 15. MG/BR 46 (Conquista); 16. BRS Silvânia RR; 17. M-SOY 800; 18. M-SOY 6101; 19. M-SOY 8329; 20. TMG123RR

7.1; 7.2; 7.3; 7.4; 7.5 - Monoascosporic strain *Sclerotinia sclerotiorum*.

Source: authors (2024)

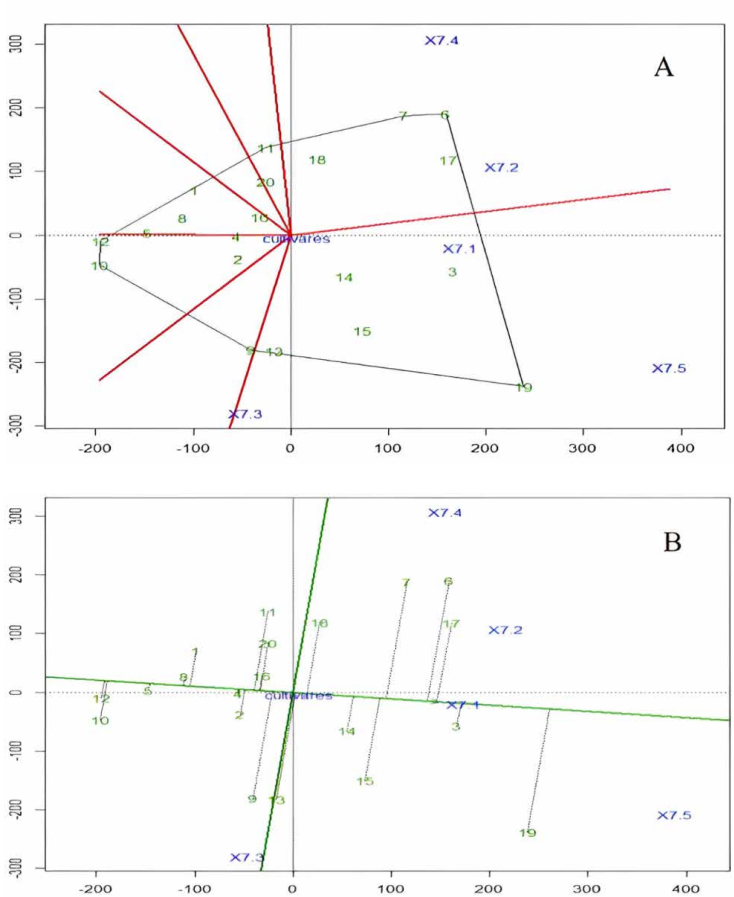
Table 2. Spearman correlation for the classification of cultivars according to inoculation with each monoascosporic strain (above the diagonal). The coincidence index between the four most resistant cultivars and the four most susceptible cultivars is shown in parentheses (below the diagonal).

	7.1	7.2	7.3	7.4	7.5
7.1	-	0.62**	0.10ns	0.10ns	0.24ns
7.2	0.25 (0.50)	-	-0.22ns	0.25ns	0.17ns
7.3	0.25 (0.25)	0.2 (0)	-	-0.55**	-0.11ns
7.4	0 (0.75)	0 (0.75)	0 (0)	-	-0.22ns
7.5	0.25 (0.25)	0 (0.25)	0 (0.25)	0 (0.25)	-

** Significant at 1 % probability; ns not significant.

Source: authors (2024)

Figure 3. (a) GGE biplot for the reactions of 20 soybean cultivars to five monoascosporic *S. sclerotiorum* strains. (b) Stability of the 20 soybean cultivars. Principal component 1 (AXIS 1) = 36.43 % and principal component 2 (AXIS 2) = 32.59 %.



The means followed by same letter belong to the same group according to a Scott-Knott test at a 5 % probability level. 1. Emgopa 316; 2. Emgopa 315; 3. BRS Milena; 4. BRSMG 790A; 5. BRSMG 850GRR; 6. BRS Baliza RR; 7. BRS Favorita RR; 8. BRSGO Luziânia; 9. M-SOY 8000RR; 10. BRSMG 68 (Vencedora); 11. 7166RSF IPRO; 12. BRS 213; 13. NS 7338 IPRO; 14. BRSMG Garantia; 15. MG/BR 46 (Conquista); 16. BRS Silvânia RR; 17. M-SOY 800; 18. M-SOY 6101; 19. M-SOY 8329; 20. TMG123RR

7.1; 7.2; 7.3; 7.4; 7.5 - Monoascosporic strain *Sclerotinia sclerotiorum*.

Source: authors (2024)

It is important to note that more aggressive isolates allow better discrimination of the resistance of cultivars. The UFLA 44 isolate, from which the monoascosporic strains were obtained, was identified as a relatively aggressive isolate in a study by Abreu and Souza (2015) in bean crops. The fact that the isolate used in this study originated in bean fields may have limited the ability to evaluate adaptive differences in the pathogen for each host. This means that when there is coevolution between pathogens and hosts, it is possible to identify plants with a higher level of resistance to the pathogen as well as pathogens that are more aggressive toward a particular host species. Such knowledge should be taken into account, once there is genetic differentiation of *S. sclerotiorum* populations from distantly related hosts, such as bean, canola, soybean, and sunflower (ALDRICH-WOLFE et al., 2015). Even so, monoascosporic strains 7.3, 7.4, and 7.5 were more efficient in causing symptoms and can be used in breeding programs for the selection of resistance to white mold.

The cultivars previously identified as resistant (GARCIA; JULIATTI, 2012) did not follow the same reaction pattern. For inoculations with all the monoascosporic strains, at least three cultivars considered resistant were classified as susceptible in the present study. This difference in resistance pattern may be due to the method used for the inoculation of the cultivars, as previous authors used the detached leaf method. In addition, the source of the inoculum was different. In the study cited, the isolate was obtained from sclerotia, while in the present study, monoascosporic strains were used. Thus, from the change in the classification of cultivars (from resistant to susceptible), it can be inferred that the monoascosporic strains were more aggressive than the isolates.

It is common to find reports on the noncoincident classification of cultivars, especially for bean crops. In a greenhouse study to evaluate the physiological resistance of some bean lines, cultivar G122 was used as a control resistant to white mold (LEHNER et al., 2016c). However, in another study, also conducted to identify genotypes with high levels of resistance to *S. sclerotiorum*, the authors observed a worse performance for the G122 cultivar than for other resistant controls (VITERI et al., 2015).

A possible explanation for the divergence of these results is the physiological resistance and escape mechanisms intrinsic to plants. Physiological resistance is conferred by plant defense mechanisms and escape mechanisms are conferred by characteristics related to plant architecture (MIKLAS et al., 2013). Under field conditions, both types of resistance contribute to the control of white mold; however, in a greenhouse or laboratory, only physiological resistance can be measured (VUONG et al., 2004). Thus, direct comparisons of experimental results should be made with caution because the cultivars used, the variability in the isolates, the inoculation method and the environmental conditions of the experiments can affect the results. Although the results of the present study suggest the existence of different levels of aggressiveness among monoascosporic strains, inoculations under field conditions should be performed to effectively demonstrate this pathogenic variability.

It is interesting to note the correlation in the classification of cultivars based on the different monoascosporic strains. There was a positive and significant correlation for only the less aggressive monoascosporic strains. Similar results were obtained in other studies (WILLBUR et al., 2017). In this study, three inbred lines of soybean were inoculated with nine *S. sclerotiorum* isolates (four more aggressive, four less aggressive and one control). For three of the less aggressive strains, the classification of the strains was similar. However, for the more aggressive isolates, the classification of the strains diverged. This finding suggests that for less aggressive isolates, plant requirements for resistance mechanisms are lower, and thus the reaction is similar. On the other hand, as the aggressiveness of the isolates increases, the cultivar-isolate interaction becomes more pronounced, i.e., the reactions that the plants may have to the isolates are increased.

The GGE biplot analysis characterized the cultivars according to the level of resistance they displayed and the monoascosporic strains in terms of aggressiveness (YAN; FALK, 2002). Thus, one of the advantages of using biplots is the ability to separate cultivars according to their resistance pattern and thus to infer the groups of alleles that each cultivar has. The BRSMG 790A and BRSMG 850GRR cultivars, considered resistant in previous studies, showed more stable resistance to the different monoascosporic strains. In addition, these two cultivars were grouped into distinct clusters, i.e., they have different groups of favorable alleles related to white mold resistance. Therefore, these cultivars could be intercrossed in a breeding program to obtain resistant strains.

The stability plot (Figure 3b) also corroborates the results of the mean data and reaffirms the finding that monoascosporic strains 7.3 and 7.4 had higher levels of aggression.

Conclusion

There is variability among the five monoascosporic strains of *S. sclerotiorum* evaluated. Monoascosporic lines are the most suitable genetic unit to be used in breeding programs that aim to develop soybean cultivars resistant to *S. sclerotiorum.*

Acknowledgements

The authors thank the National Council for Scientific and Technological Development (Conselho Nacional de Desenvolvimento Científico e Tecnológico - CNPq) and the Minas Gerais State Agency for Research and Development (Fundação de Amparo à Pesquisa do Estado de Minas Gerais - FAPEMIG) for their support, and the Brazilian Federal Agency for Support and Evaluation of Graduate Education (Coordenação de Aperfeiçoamento de Pessoal de Nível Superior - CAPES) for the scholarships granted and also for the Productivity scholarship. We would like to thanks the CNPq for the productivity fellowship for the author Dr. Adriano Teodoro Bruzi.

References

ABREU, M. J.; SOUZA, E. A. Investigation of *Sclerotinia sclerotiorum* strains variability in Brazil. **Genetics and molecular research (GMR),** v. 14, n. 2, p. 6879–6896, 2015. Disponivel em: http://dx.doi.org/10.4238/2015. June. 18.31. Acesso em: 10 out. 2023.

ALDRICH-WOLFE, L.; TRAVERS, S.; NELSON, B. D. Genetic variation of *Sclerotinia sclerotiorum* from multiple crops in the north central United States. **PloS one,** v. 10, n. 9, p. e0139188, 2015. Disponivel em: http://dx.doi.org/10.1371/journal.pone.0139188. Acesso em: 12 out. 2023.

AUCLAIR, J.; BOLAND, G.J.; KOHN, L.M.; RAICAN, I. Genetic interactions between *Glycine max* and *Sclerotinia sclerotiorum* using a straw inoculation method. **Plant Disease**, v. 88, n. 8, p. 891-895, 2004. Disponivel em: https://apsjournals.apsnet.org/doi/10.1094/PDIS.2004.88.8.891. Acesso em: 13 de mar. 2024.

CASTRO, L. H. S.; FIGUEIRÓ, A.A.; NOGUEIRA, A.P.O.; CLOUGH, S.J.; JULIATTI, F.C. Resistance of soybean genotypes to *Sclerotinia sclerotiorum* isolates in different incubation environments. **Genetics and molecular research (GMR**), v. 15, n. 4, gmr15049061, 2016. Diponível em: http://dx.doi.org/10.4238/gmr15049061. Acesso em: 29 out. 2023.

CLARKSON, J. P.; COVENTRY, E.; KITCHEN, J.; CARTER, H.E.; WHIPPS, J.M. Population structure of *Sclerotinia sclerotiorum* in crop and wild hosts in the UK. **Plant Pathology**, v. 62, n. 2, p. 309-324, 2013. Disponível em: https://doi.org/10.1111/j.1365-3059.2012.02635.x. Acesso em: 13 fev. 2024.

GARCIA, R. A.; JULIATTI, F. C. Avaliação da resistência da soja a *Sclerotinia sclerotiorum* em diferentes estádios fenológicos e períodos de exposição ao inóculo. **Tropical Plant Pathology**, v. 37, n. 3, p. 196–203, 2012. Diponível em: http://dx.doi.org/10.1590/s1982-56762012000300006. Acesso em: 23 out. 2023.

HAMBLIN, J.; ZIMMERMANN, M.J.D.O. Breeding common bean for yield in mixtures. In: JANICK, J. **Plant Breeding Reviews**. Nova York: John Wiley, 1986. p. 245-272. Disponivel em: https://doi.org/10.1002/9781118061015.ch8. Acesso em: 29 out. 2024

HAMBLETON, S.; WALKER, C.; KOHN, L. M. Clonal lineages of *Sclerotinia sclerotiorum*

previously known from other crops predominate in 1999-2000 samples from Ontario and Quebec soybean. **Canadian Journal of Plant Pathology**, v. 24, n. 3, p. 309-315, 2002.

HARMAN, G. E.; HOWELL, C.R.; VITERBO, A.; CHET, I.; LORITO, M. Trichoderma species-opportunistic, avirulent plant symbionts**. Nature Reviews Microbiology**, v. 2, n. 1, p. 43-56, 2004. Disponível em: https://doi.org/10.1038/nrmicro797. Acesso em: 15 nov. 2023.

HULLER, G.; FILHO, D.S.J.; PIERRE, M.L.C.; TULIO, H.E.; GRABICOSKI, E.M.G.; JULIATTI, F.C. Different methods of assessing susceptibility of soybean genotypes to white mold. **Bioscience Journal**, v. 32, n. 2, p. 389–402, 2016. Disponível em: https://seer.ufu.br/index.php/biosciencejournal/article/view/31365. Acesso em: 17 out. 2023

KULL, L. S.; VUONG, T.D.; PODERES, K.S.; ESKRIDGE, K. M.; STEADMAN, J. R.; HARTMAN, G.L. Evaluation of resistance screening methods for Sclerotinia stem rot of soybean and dry bean. **Plant Disease,** v. 87, n. 12, p. 1471–1476, 2003. Disponível em: http://dx.doi.org/10.1094/PDIS.2003.87.12.1471. Acesso em: 26 dez. 2023.

LEHNER, M.S.; PAULA JUNIOR, T.J.; JUNIOR, B.T.; TEIXEIRA, H.; VIEIRA, R.F.; CARNEIRO, J.E.S.; MIZUBUTI, E.S.G. Low genetic variability in *Sclerotinia sclerotiorum* populations from common bean fields in Minas Gerais State, Brazil, at regional, local and micro-scales. **Plant Pathology**, v. 8, n. 64, p. 4–21, 2015.

LEHNER, M. S.; LIMA, R. C.; PAULA JUNIOR, T.J.; VIEIRA, R.F.; MIZUBUTI, E.S.G. Similar aggressiveness of phenotypically and genotypically distinct isolates of *Sclerotinia sclerotiorum*. **Plant Disease**, v. 100, n. 2, p. 360–366, 2016. Disponível em: http://dx.doi.org/10.1094/PDIS-04-15-0400-RE, 2016a. Acesso em: 15 nov. 2023.

LEHNER, M. S.; PAULA JUNIOR, T.J. does hyphal-tip ensure the same allelic composition at SSR loci as monosporic isolates of *Sclerotinia sclerotiorum*? **Journal of Phytopathology**, v. 164, n. 6, p. 417-420, 2016b. Disponível em: https://doi.org/10.1111/jph.12429. Acesso em: 17 nov. 2023.

LEHNER, M.S.; PAULA JUNIOR, T.J.; VIEIRA, R.; LIMA, R.C. Reaction of sources of resistance to white mold to microsatellite haplotypes of *Sclerotinia sclerotiorum*. **Scientia Agricola,** v. 73, n. 2, p. 184–188, 2016c. Disponível em: http://dx.doi.org/10.1590/0103-9016-2015-0085. Acesso em: 21 dez. 2023.

LIU, J.; MENG, Q.; ZHANGB, Y.; XIANG, H.; LI, Y.; SHI, F. M.; MA, L.; LIUB, C.; LIUBI-YU; SU, B.; LI, Z. Mycelial compatibility group and genetic variation of sunflower *Sclerotinia sclerotiorum* in Northeast China. **Physiological and Molecular Plant Pathology**, v. 102, p. 185–192, 2018. Disponível em: http://dx.doi.org/10.1016/j.pmpp.2018.03.006. Acesso em: 22 dez. 2023.

MEINHARDT, L. W.; WULFF, E.A.; BELLATO, C.; TSAI, S. M. Telomere and microsatellite primers reveal diversity among *Sclerotinia sclerotiorum* isolates from Brazil. **Fitopatologia Brasileira**, v. 27, n. 2, p. 211–215, 2002. Disponível em: http://dx.doi.org/10.1590/s0100-41582002000200015. Acesso em: 13 dez. 2023.

MERT-TURK, F.; IPEK, M.; MERMER, D.; NICHOLSON, P. Microsatellite and morphological markers reveal genetic variation within a population of *Sclerotinia sclerotiorum* from oilseed rape in the Canakkale Province of Turkey. **Journal of Phytopathology**, v. 155, n. 3, p. 182-187, 2007. Disponivel em: https://doi.org/10.1111/j.1439-0434.2007.01223.x. Acesso em: 13 dez 2023.

MEYER, M. C.; MAZARRO, S.M.; SILVA, J.C. **Eficiência de fungicidas para controle de**

mofo-branco (*Sclerotinia sclerotiorum*) em soja, na safra 2021/2022: resultados sumarizados dos experimentos cooperativos Londrina: Embrapa Soja, 2022. 15p

MIKLAS, P. N.; PORTER, L.; KELLY, J. D.; MYERS, J. R. Characterization of white mold disease avoidance in common bean. **European Journal of Plant Pathology**, v. 135, n. 3, p. 525–543, 2013. Disponível: http://dx.doi.org/10.1007/s10658-012-0153-8. Acesso em: 2 jan. 2024.

PASCUAL, A.; CAMPA, A.; VEGA, E. P.; GIRALDEZ, R.; MIKLAS.; FERREIRA, J. J. Screening common bean for resistance to four *Sclerotinia sclerotiorum* isolates collected in northern Spain. **Plant Disease**, v. 94, n. 7, p. 885–890, 2010. Disponível em: http://dx.doi.org/10.1094/PDIS-94-7-0885. Acesso em: 9 jan. 2024.

PAULA JÚNIOR, T. J. de.; CARNEIRO, J.E. de S.; VIEIRA, R.F.; ABREU, A. de F.B.; RAMALHO, M.A.P.; DEL PELOSO, M.J.; TEIXEIRA, H. **Cultivares de feijão-comum para Minas Gerais**. Belo Horizonte: EPAMIG, 2010. 40p.

PETZOLDT, R.; DICKSON, M. H. Straw test for resistance to white mold in beans. **Annual Report Bean Improvement Cooperative,** v. 39, n. 39, p. 142–143, 1996.

R CORE TEAM. R: A **Language and environment for statistical computing**, 2020. Disponivel em: https://www.r-project.org/. Acesso em: 9 set. 2023.

RESENDE, M. **Matemática e estatística na análise de experimentos e no melhoramento genético**. Colombo: Embrapa Florestas, 2007. 561 p. Disponivel em: https://www.alice.cnptia.embrapa.br/alice/handle/doc/314759. Acesso em: 23 out. 2024.

SCOTT, A. J.; KNOTT, M. A cluster analysis method for grouping means in the analysis of variance. **Biometrics**, v. 30, n. 3, p. 507-512, 1974. Disponível em: http://dx.doi.org/10.2307/2529204. Acesso em: 05 jan. 2024.

SHANER, G.; FINNEY, R. E. The effect of nitrogen fertilization on the expression of slow-mildewing resistance in Knox wheat. **Phytopathology**, v. 77, n. 8, p. 1051-1056, 1977. Disponível em: http://dx.doi.org/10.1094/phyto-67-1051. Acesso em: 05 jan. 2024.

SILVA, P. H.; SANTOS, J.B.; LIMA, L.A.; LARA, L.A.C.; ALVES, F.C. Reaction of common bean lines and aggressiveness of *Sclerotinia sclerotiorum* isolates. **Genetics and Molecular Research** (GMR), v. 13, n. 4, p. 9138–9151, 2014. Disponível em: http://dx.doi.org/10.4238/2014.November.7.11. Acesso em: 05 jan. 2024.

STEADMAN, J.R.; BOLAND, G**. White mold in compendium of bean diseases**. In: SCHWARTZ, H.F.; STEADMAN, J.R.; HALL, R.; FORSTER, R.L. St Paul: American Phytopathological Society, 2005. p. 44-46

VITERI, D. M.; OTTO, K.; TERÁN, H.; SCHWARTZ, H.F.; SINGH, S.P. Use of four *Sclerotinia sclerotiorum* isolates of different aggressiveness, three inoculations per plant, and delayed multiple evaluations to select common beans with high levels of white mold resistance. **Euphytica**, v. 204, n. 2, p. 457–472, 2015. Disponível em: https://link.springer.com/article/10.1007/s10681-015-1366-7. Acesso em: 13 fev. 2024.

VUONG, T.D.; HOFFMAN, D.D.; DIERS, B.W.; MILLER, J.F.; STEADMAN, J.R.; HARTMAN, G.L. Evaluation of soybean, dry bean, and sunflower for resistance to *Sclerotinia sclerotiorum*. **Crop Science**, v. 44, n. 3, p. 777-783, 2004. Disponível em: http://soydiseases.illinois.edu/publications/CutStemMethod.pdf. Acesso em: 23 out. 2024

WEBSTER, J; WEBER, R. **Introduction to fungi**. 3 ed. New York: Cambridge University Press, 2007. 840 p. Disponivel em: http://deskuenvis.nic.in/pdf/WEBSTER30521807395.pdf. Acesso em: 21 out. 2024

WILLBUR, J. F.; DING, S.; MARKS.; LUCAS, H.; GRAU, C.R.; GROVES, C.L.; KABBAGE, M.; SMITH, D. L. Comprehensive Sclerotinia stem rot screening of soybean germplasm requires multiple isolates of *Sclerotinia sclerotiorum*. **Plant Disease**, v. 101, n. 2, p. 344–353, 2017. Disponivel em: http://dx.doi.org/10.1094/PDIS-07-16-1055-RE. Acesso em: 13 fev. 2024.

YAN, W.; FALK, D. E. Biplot analysis of host-by-pathogen data. **Plant Disease**, v. 86, n. 12, p. 1396–1401, 2002. Disponível em: https://doi.org/10.1094/PDIS.2002.86.12.1396. Acesso em: 15 mar. 2024.