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# INTERSPECIFIC HYBRIDS AS SOURCE OF RESISTANCE TO *Sclerotinia* AND *Phomopsis* IN SUNFLOWER BREEDING

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SUMMARY

The fungal pathogens *Sclerotinia sclerotiorum* and *Phomopsis helianthi* are two major diseases in sunflower. While the cultivated sunflower has a narrow genetic base, wild species have been used as a source of disease resistance. In this study, 41 different lines derived from interspecific crosses and inbred line HA89 have been infected with mycelium of *Sclerotinia* and *Phomopsis* in 1996 and 1997 and evaluated for resistance. Lines derived from *H. argophyllus* and *H. tuberosus* had the lowest *Sclerotinia* stem lesion. Several lines showing significantly lower *Phomopsis* stem lesions than the sunflower inbred line HA89 were detected from crosses between HA89 and *H. argophyllus*, *H. tuberosus*, *H. deserticola* and *H. xlaetiflorus*. BE94-186-02, TUB-5-3235, TUB-5-326 and TUB-1705-327. These inbred lines, derived from interspecific crosses, were resistant to both fungi, hence they are recommended for use in resistance breeding programs.

**Key words:** **Artificial infection, *Helianthus* ssp., *Phomopsis helianthi*, resistance, *Sclerotinia sclerotiorum*, screening**

## INTRODUCTION

Fungal pathogens such as *Sclerotinia sclerotiorum* and *Phomopsis helianthi* are a major limiting factor to high productivity in sunflower. Cultivated sunflower lacks acceptable levels of resistance to these diseases. Interspecific hybridization of wild species of the genus *Helianthus* with the cultivated sunflower has become an important source of genes for disease resistance (Škorić, 1985) and was used for broadening the genetic variation in the cultivated sunflower (Seiler, 1992).

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*Sclerotinia sclerotiorum* (Lib.) de Bary is a common and widespread pathogen of sunflower, found in all sunflower-growing regions of the world (Gulya *et al.*, 1997). Mycelia from germinating sclerotia in the soil invade roots and basal stem area of the plant causing wilting. Furthermore, airborne ascospores cause leaf infection, midstalk rot, breaking and head rot. Losses can reach 100% under extreme circumstances (Sackston, 1992). *Sclerotinia* resistance appears to be under polygenic control (Robert *et al.*, 1987) with additive and dominance gene effects for root and head infections (Tourvieille and Vear, 1990). The reaction of a genotype may vary depending on the plant part infected (Thuault and Tourvieille, 1988). Several methods for inoculating sunflower with *Sclerotinia* mycelium or ascospores on different parts of the plant have been used to evaluate various kinds of resistance to attack (Castaño *et al.*, 1993). No sources of complete resistance to *Sclerotinia* are available in cultivated sunflower (Tourvieille *et al.*, 1996). However, wild species are promising sources of genes for *Sclerotinia* resistance (Hammann *et al.*, 1994; Köhler, 1997). Seiler *et al.* (1993) found promising genetic variation in interspecific germplasm that could be used for increasing tolerance to *Sclerotinia* infections.

*Phomopsis helianthi* was first identified on sunflower in Yugoslavia in 1981 (Mihaljčević *et al.*, 1982). The disease spread further east and is common now in Europe especially in Yugoslavia (Škorić, 1992), Romania (Illiescu *et al.*, 1985), Hungary (Virányi *et al.*, 1988) and France (Regnault, 1985), but also in the United States (Herr *et al.*, 1983). Ascospores infect leaves at a late vegetative stage. Mycelium spreads to the stem and causes lesions, which may lead to lodging, while toxins cause the whole plant to dry (Vear *et al.*, 1997). Average yield losses of 20 to 30% may occur (Besnard *et al.*, 1997). Genetic studies indicate that resistance to *Phomopsis* is polygenic (Tourvieille *et al.*, 1988) or oligogenic (Škorić, 1985) and that additive gene action prevails (Vear *et al.*, 1997). Tolerant sunflower genotypes can be obtained from crosses between sunflower and wild species (Škorić, 1985). It has been demonstrated that the wild parents were responsible for the introduction of resistance factors (Griveau *et al.*, 1992).

The search for wild sunflower species with resistance to *Sclerotinia* and *Phomopsis* in order to increase genetic variation is still ongoing. Breeders are searching for genotypes, especially with resistance to different fungi. A set of 90 inbred lines has been tested in the field using artificial infection by a *Sclerotinia* leaf test as well as by a *Phomopsis* leaf and petiole test to show the benefit of offspring from interspecific crosses as a source of resistance for practical sunflower breeding (Degener *et al.*, 1998). We conducted this study to (1) evaluate 41 lines derived from interspecific crosses for their reaction to *Sclerotinia*, (2) screen the same set for reaction to *Phomopsis*, and (3) detect lines less susceptible to both pathogens.

## MATERIALS AND METHODS

### Plant material

A total of 90 inbred lines of diverse origin and five $F_1$ hybrids used as control were evaluated for resistance to *Sclerotinia* and *Phomopsis* as described in the study of Degener *et al.* (1998). The inbred lines were chosen without prior information of their response to both pathogens. The study comprised a set of 41 lines derived from interspecific crosses with HA89. The latter line was included for the sake of comparison. The present publication only deals with this set of 41 lines and HA89.

### Field experiments

All experiments were realized at Eckartsweier in the Upper Rhine Valley in Southwest Germany. The total of 90 inbred lines were divided into two trials. The trials, each containing 45 inbred lines and five $F_1$ hybrids were conducted separately in a 10 x 5-alpha design (Patterson and Williams, 1976). Each replication contained 10 blocks of five plots each with 30 plants.

***Sclerotinia***: In 1996, each trial was sown on May 10 and August 1 with three replications and infected in June and September, respectively. In 1997, the trials were sown on April 11 with two replications and infected in June. Reaction of sunflower inbreds to *Sclerotinia* was assessed across these three environments, different in temperature and precipitation.

***Phomopsis***: In 1996, each trial was sown on May 10 with two replications and infected in June, while in 1997 the trials were sown on April 11 with two replications infected in June. Hence, reaction of sunflower lines to *Phomopsis* was tested in two environments.

### Fungal isolates

The *Sclerotinia* isolate used in the study originated from sclerotia collected in 1995 from naturally infected sunflowers at Eckartsweier. The *Phomopsis* isolate originated from infected stems in France, kindly provided from CETIOM. Mycelium was cultivated at 25°C on a 1.5% agar medium containing 2% malt and 0.2% peptone extract, respectively.

### Artificial infection and data recording

***Sclerotinia*:** The methods for infection and data recording were described in detail by Degener *et al.* (1998). Briefly, the leaf was infected with a *Sclerotinia* mycelium explant and fixed by a self-adhesive label. The whole infested leaf was covered with a transparent plastic bag containing water to prevent drying of the inoculum. We observed fungal symptoms on the leaf and on the stem by scoring development of disease in both tissues:

***Leaf lesion* (cm)**: The length of the brown rotted zone along the leaf vein beginning around the explant six days after infection.

***Petiole score (days)***: The number of days from leaf infection until the lesion of the fungus reached the base of the petiole. Genotypes requiring more days for the fungus to reach the petiole were considered more tolerant.

***Stem lesion* (cm)**: The lesion length on the stem one month after infection.

***Phomopsis:*** To infect the plants with *Phomopsis* in 1996 and 1997 we used the same leaf test as described for *Sclerotinia*. Petiole score and stem lesion were recorded as described above.

In 1996 we used additionally the petiole test (Bertrand and Tourvieille, 1987). The petiole was cut across 2 cm from the stem, mycelial explant was placed on the petiole and covered with wet cotton wool and a parafilm tape to prevent drying of the infection site. We measured:

***Stem lesion-p* (cm):** The length of necroses visible on the stem four weeks after petiole infection.

### Statistical analysis

Field data from individual plants of each plot were averaged to calculate plot means for each trait. Plants on which the inoculum was not successful in causing *Sclerotinia* infection were excluded from calculation. The set of 41 lines derived from interspecific crosses together with the public line HA89 was analyzed separately as randomized complete block design. Analyses of variance were based on field data from each environment. Adjusted entry means and corresponding error mean squares were used to compute combined analyses of variance across environments. Inbred lines and environments were considered as random effects in the ANOVA model. All computations were performed with the computer package PLABSTAT (Utz, 1991).

## RESULTS

### Reaction of germplasm to *Sclerotinia*

We found significant ($P<0.05$) genetic variation among the tested 42 inbred lines across three environments for all *Sclerotinia* resistance traits (Table 1). The means of individual lines showed a wide range, especially for stem lesion (2.2 – 14.6 cm). Lines listed in Table 1 were ranked according to increased susceptibility to stem lesion. Lines derived from crosses with HA89 and *H. argophyllus* (BE94-186-01, BE94-186-02) as well as *H. tuberosus* (TUB-5-3234, TUB-5-3235, TUB-5-326, TUB-1705-327) had the smallest stem lesion. Nevertheless, no line derived from interspecific crosses was detected showing significantly lower *Sclerotinia* damages than HA89.

Table 1: Mean scores of forty-one sunflower inbreds derived from interspecific crosses with wild species and inbred line HA89 studied in three environments for resistance to *Sclerotinia* and in one environment for resistance to *Phomopsis*

| Nr.[1] | Line | Wild species | *Sclerotinia* (1996-1997) | | | *Phomopsis* (1996) | | |
|---|---|---|---|---|---|---|---|---|
| | | | Leaf lesion (cm) | Petiole score (days) | Stem lesion (cm) | Petiole score (days) | Stem lesion (cm) | Stem lesion-p (cm) |
| 26 | BE94-186-01 | *H. argophyllus* | 5.6 | 15.3 | 2.2 | 28.6 | 16.8 | 24.0 |
| 27 | TUB-5-3234 | *H. tuberosus-5* | 5.2 | 14.2 | 2.9 | 27.4 | 17.0 | 26.9 |
| 25 | BE94-186-02 | *H. argophyllus* | 5.6 | 16.0 | 2.9 | 34.3 | 0.1 | 11.9 |
| 35 | TUB-5-3235 | *H. tuberosus-5* | 4.9 | 14.2 | 3.0 | 23.8 | 0.2 | 8.2 |
| 29 | TUB-5-326 | *H. tuberosus-5* | 6.1 | 12.8 | 4.3 | 28.5 | 2.8 | 12.2 |
| 41 | TUB-1705-327 | *H. tuberosus-1705* | 5.6 | 14.6 | 4.6 | 26.7 | 1.1 | 6.7 |
| 42 | HIR34F | *H. hirsutus* | 6.7 | 14.1 | 4.8 | 24.3 | 9.5 | 15.5 |
| 13 | PAR-1673-2 | *H. paradoxus- 1673* | 6.3 | 13.6 | 4.9 | 36.8 | 20.2 | 29.2 |
| 6 | TUB-1709-3 | *H. tuberosus-1709-3* | 6.3 | 14.6 | 5.6 | 32.0 | 27.7 | 36.6 |
| 36 | TUB-1705-33704 | *H. tuberosus-1705* | 5.2 | 13.9 | 5.7 | 33.6 | 19.7 | 20.0 |
| 18 | ARG-283 | *H. argophyllus* | 4.9 | 16.6 | 5.8 | 31.5 | 7.1 | 21.2 |
| 9 | PRA-RUN-417-1 | *H. praecox subsp.runyonii-417* | 6.0 | 13.9 | 5.8 | 35.9 | 21.3 | 28.1 |
| 24 | HA337 | *H. praecox-417* | 6.3 | 15.3 | 5.9 | 34.6 | 27.3 | 38.3 |
| 16 | ARG-1575-1 | *H. argophyllus-1575* | 5.8 | 14.8 | 6.1 | 39.8 | 9.4 | 15.7 |
| 15 | DES-1474-3 | *H. deserticola-1474* | 6.6 | 12.5 | 6.1 | 29.8 | 0.5 | 22.6 |
| 39 | TUB-1705-347 | *H. tuberosus-1705* | 4.8 | 14.7 | 6.2 | 27.7 | 7.5 | 21.4 |
| 1 | HA89 | | 5.9 | 15.3 | 6.4 | 30.0 | 46.8 | 49.3 |
| 8 | PLH2 | *H. annuus* | 5.3 | 14.2 | 6.6 | 35.3 | 19.6 | 27.5 |
| 2 | TUB-346 | *H. tuberosus-346* | 5.5 | 14.6 | 6.7 | 34.1 | 29.7 | 41.2 |
| 33 | TUB-1705-33706 | *H. tuberosus-1705* | 6.7 | 13.8 | 7.0 | 30.8 | 15.0 | 10.0 |
| 38 | HA336 | *H. annuus* | 6.1 | 13.0 | 7.3 | 25.7 | 4.5 | 8.5 |
| 5 | TUB-1709-2 | *H. tuberosus-1709-2* | 6.6 | 13.3 | 7.7 | 35.5 | 29.3 | 39.5 |
| 19 | MAX-287 | *H. maximiliani* | 5.6 | 10.8 | 8.2 | 28.0 | 42.5 | 58.5 |
| 31 | TUB-1705-334 | *H. tuberosus-1705* | 5.7 | 14.2 | 8.5 | 32.5 | 31.5 | 20.6 |
| 4 | TUB-1709-1 | *H. tuberosus-1709-1* | 5.5 | 13.2 | 8.5 | 36.9 | 23.5 | 36.7 |
| 30 | TUB-1705-328 | *H. tuberosus-1705* | 6.7 | 12.3 | 8.5 | 28.9 | 19.7 | 12.1 |
| 20 | XLAE-288 | *H- xlaetiflorus* | 6.1 | 13.7 | 8.8 | 33.4 | 0.6 | 33.5 |
| 40 | TUB-1705-344 | *H. tuberosus-1705* | 6.4 | 13.8 | 8.8 | 30.9 | 9.4 | 12.2 |
| 34 | TUB-1705-338 | *H. tuberosus-1705* | 6.3 | 13.0 | 8.9 | 24.5 | 0.4 | 13.5 |
| 28 | TUB-5-324 | *H. tuberosus-5* | 6.9 | 11.9 | 9.1 | 22.8 | 10.5 | 12.4 |
| 3 | TUB-365 | *H. tuberosus-365* | 6.1 | 13.4 | 9.2 | 30.4 | 12.8 | 34.9 |
| 22 | XLAE-290 | *H. xlaetiflorus* | 6.1 | 13.3 | 9.6 | 31.7 | 19.8 | 30.2 |
| 23 | PLH1 | *H. annuus* | 7.3 | 15.2 | 9.6 | 38.4 | 36.7 | 42.4 |
| 12 | PAR-1673-1 | *H. paradoxus-1673* | 6.3 | 11.9 | 9.7 | 28.1 | 48.8 | 56.0 |
| 10 | PRA-RUN-417-3 | *H. praecox subsp.runyonii-417* | 5.9 | 13.2 | 9.9 | 31.2 | 45.8 | 53.0 |
| 11 | PRA-RUN-1329 | *H. praecox subsp.runyonii-1329* | 6.4 | 14.5 | 10.0 | 29.9 | 29.3 | 44.7 |

Table 1: Mean scores of forty-one sunflower inbreds derived from interspecific crosses with wild species and inbred line HA89 studied in three environments for resistance to *Sclerotinia* and in one environment for resistance to *Phomopsis*

| Nr.[1] | Line | Wild species | *Sclerotinia* (1996-1997) | | | *Phomopsis* (1996) | | |
|---|---|---|---|---|---|---|---|---|
| | | | Leaf lesion (cm) | Petiole score (days) | Stem lesion (cm) | Petiole score (days) | Stem lesion (cm) | Stem lesion-p (cm) |
| 17 | ARG-1575-3 | *H. argophyllus-1575* | 6.2 | 11.6 | 11.1 | 32.7 | 47.6 | 53.9 |
| 32 | TUB-1705-336 | *H. tuberosus-1705* | 6.4 | 13.1 | 11.1 | 21.1 | 8.8 | 24.9 |
| 7 | TUB-1789 | *H. tuberosus-1789* | 6.0 | 13.4 | 11.3 | 38.4 | 10.5 | 38.4 |
| 37 | HA335 | *H. annuus* | 7.0 | 13.0 | 12.6 | 30.8 | 43.5 | 47.4 |
| 14 | DES-1474-1 | *H. deserticola-1474* | 6.1 | 12.1 | 13.8 | 30.3 | 18.6 | 49.2 |
| 21 | GIG-289 | *H. giganteus* | 5.2 | 13.0 | 14.6 | 33.3 | 38.9 | 55.1 |
| | | Mean | 6.0 | 13.7 | 7.6 | 31.0 | 19.8 | 29.6 |
| | | Range | 4.8-7.3 | 10.8-15.3 | 2.2-14.6 | 21.1-39.8 | 0.1-48.8 | 6.7-58.5 |
| | | L.S.D. 5% | 1.7 | 2.3 | 5.2 | 6.5 | 24.4 | 18.8 |

[1] Number of 42 tested inbred lines

**Reaction of germplasm to *Phomopsis***

Due to unfavorable weather conditions leading to unsuccessful infections in 1997, only the *Phomopsis* infection tests of 1996 showed reliable ranking (Table 1). Several lines had significantly lower *Phomopsis* stem lesion after the leaf test than HA89. These lines originated from *H. argophyllus* (BE94-186-02), *H. tuberosus* (TUB-5-3235, TUB-1705-338, TUB-1705-327, TUB-5-326), *H. deserticola* (DES-1474-3), and *H. xlaetiflorus* (XLAE-288).

Although the environments of 1996 and 1997 were not comparable, the extremes were consistent. Some lines with low and high *Phomopsis* stem lesions in 1996 and 1997 were detected (Figure 1). In both years the lines TUB-5-3235, TUB-1705-338, TUB-1705-327 and BE94-186-02 (35, 34, 41, 25) showed low stem lesions after the leaf test. PAR-1673-1 derived from *H. paradoxus*, TUB-1705-336 from *H. tuberosus*, MAX-287 from *H. maximiliani*, PLH1 from *H. annuus* (12, 32, 19, 23) showed high stem lesions after 1996 and 1997 leaf infection just as HA89.

**Comparison of susceptibility to both fungi**

Correlation between *Phomopsis* stem lesion in 1996 and *Sclerotinia* stem lesion across three environments were highly significant ($r_p$ = 0.43**) (Figure 2). The four lines, BE94-186-02, TUB-5-3235, TUB-5-326, and TUB-1705-327 (25, 35, 29, 41), had low stem lesions after both fungal infections. Three lines (21, 37, 32) derived from *H. gigantheus* (GIG-289), *H. annuus* (HA335) and TUB-1705-336 showed large stem lesions after *Sclerotinia* as well as *Phomopsis* infection.

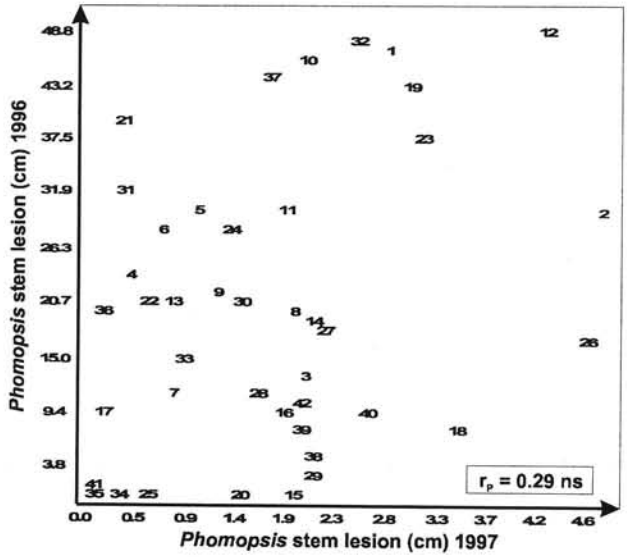


*Figure 1: Relation between Phomopsis stem lesion in 1996 and 1997 for 41 lines derived from interspecific crosses and HA89. Numbers of lines (1-42) are given in Table 1. ns: not significant*

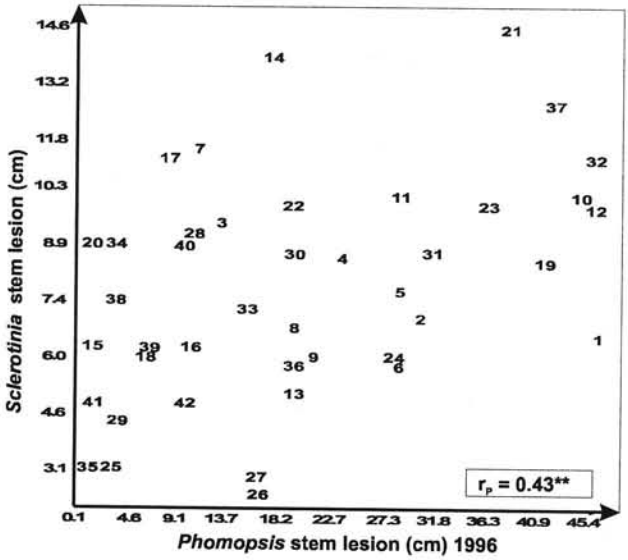


*Figure 2: Relation between Sclerotinia and Phomopsis stem lesion for 41 lines derived from interspecific crosses and HA89. Numbers of lines (1-42) are given in Table 1. ** : P < 0.05*

## DISCUSSION

Fungal diseases are the major limiting factor of sunflower production in most regions. Sources of genetic resistance for some diseases have been detected in wild sunflowers. However, there are still several pathogens such as *Sclerotinia* and *Phomopsis* for which resistance sources remain to be found. Diversity of the genus *Helianthus* offers possibilities of discovering them (Škorić, 1992).

### Susceptibility of interspecific hybrids to *Sclerotinia*

*Sclerotinia* appears to be complicated and using wild sunflowers for resistance to this disease has not proved to be successful (Seiler, 1992). Nevertheless, Maširević and Gulya (1992) stated that total immunity to *Sclerotinia* is an unrealistic objective and instead sources with minimal susceptibility or delayed infection should be searched for.

Stem lesion is the best predictor of *Sclerotinia* yield losses (Degener *et al.*, 1998), hence we concentrated our investigations on this trait. In our study we did not find any line derived from interspecific crosses to be significantly less susceptible to *Sclerotinia* than HA89 parental line. However, we detected significant differences in susceptibility among the 42 inbreds. Based on our data, lines derived from crosses with HA89 and *H. argophyllus* as well as *H. tuberosus* showed the highest level of resistance to *Sclerotinia*. Consistent with our results, Thompson *et al.* (1981) identified *H. tuberosus* as a possible source of resistance to *Sclerotinia*. Furthermore, Köhler (1997) found crosses with *H. tuberosus* to be less susceptible to *Sclerotinia* stem infection than the $F_1$ hybrids used as controls ('Sunking-256', 'Alphasol,' and 'Frankasol'). Seiler *et al.* (1993) discovered lines from crosses with *H. paradoxus* and *H. praecox* ssp. *runyonii* as potential sources for tolerance to natural infection by *Sclerotinia*. In our study, PAR-1673-2 and PRA-RUN-417-1, derived from these mentioned wild species, were among the twelve lines exhibiting the lowest average stem lesion. As the reaction to *Sclerotinia* is varying between lines deriving from the same wild species (Köhler, 1997; Hammann *et al.*, 1994), there were also lines from crosses with *H. argophyllus* (ARG-1575-3) *and H. tuberosus* (TUB-1705-336, TUB-1789) showing significantly higher stem lesion than lines derived from the same interspecific crosses (Table 1). A general statement about susceptibility of interspecific hybrids to *Sclerotinia* cannot be made, every line derived from interspecific hybridization has to be tested seperately. BE94-186-01, BE94-186-02, TUB-5-3234 and TUB-5-3235 showed the lowest values for *Sclerotinia*, but further research is needed to investigate whether these lines have resistance genes different from those in line HA89.

### Susceptiblity of interspecific hybrids to *Phomopsis*

Optimum temperature for *Phomopsis* mycelial growth is 23 to 25°C (Maširević and Gulya, 1992). One week after the leaf infection in 1997, temperatures were

about 18°C and high precipitation (94 mm) occurred within the following 14 days. The mycelial growth seemed to stagnate and only the petiole score could be measured clearly. Because of high genotype x environment interactions in the combined analysis across both environments we were forced to restrict our analysis to data from 1996.

The classification obtained for *Phomopsis* symptoms on the leaf and on the stem (Table 1) was not identical, indicating that several resistance mechanisms are involved in the different phases of the infection process (petiole score, stem lesion). Similar results have been reported by Tourvieille *et al.* (1988) and Dozet (1990). The main resistance to *Phomopsis* seems to be located in the passage petiole-stem (Bertrand and Tourvieille, 1987) and the length of the stem lesion stands for the degree of susceptibility of a genotype (Gulya *et al.*, 1997). Consequentely, we decided to concentrate our study on the trait stem lesion. Although there is the danger of premature drying of the leaf after the infection, the leaf test is more likely comparable with natural infection (Tourvieille *et al.*, 1988). The petiole test seemed to be a very drastic and severe test, although a significant correlation of 0.83 ($P< 0.01$) between stem lesion and stem lesion-p was found (data not presented).

The best potential lines were obtained from crosses with *H. argophyllus*, *H. tuberosus*, *H. deserticola* and *H. xlaetiflorus* (Table 1), exhibiting stem lesions from 0.1 to 1.1 cm. According to Gulya *et al.* (1997) lesions on stems of resistant plants remain small, while on susceptible genotypes stem lesions may eventually reach 15 to 20 cm. *H. tuberosus* as well as *H. argophyllus* have already been described as sources of resistance to *Phomopsis* (Seiler, 1992; Dozet, 1990). Moreover, hybrids based on these two wild *Helianthus* species have been developed to keep a high field-tolerance to *Phomopsis* stem canker (Škorić, 1985). Especially the lines BE94-186-02, Tub-5-3235, TUB-1705-338 and TUB-1705-327 seem to possess a reliable level of resistance to *Phomopsis*. We concluded that the wild parents carried the resistance factors, because they were exhibiting stem lesions significantly lower than HA89. Although 1997 was not a suitable environment for reliable screening, these four lines again showed the lowest stem lesion (Figure 1). These lines should be used as new sources of resistance in breeding programs.

**Interspecific hybrids with resistance to multiple fungal diseases**

Most interesting for sunflower breeding programs are lines showing resistance to *Sclerotinia* as well as to *Phomopsis* (Figure 2). BE94-186-02, TUB-5-3235 and TUB-1705-327 which already showed resistance to *Phomopsis* across both environments represent together with TUB-5-326 a source of resistance to both *Phomopsis* and *Sclerotinia*. In contrast, the lines TUB-1705-336 and GIG-289 seem to be unsuitable for further breeding because of high fungal susceptibility. HA335, resistant to all known races of *Plasmopara halstedii* (Garcia and Gulya, 1991), is highly susceptible to *Phomopsis* and *Sclerotinia*. Hence it cannot be considered in breeding programs for resistance to multiple diseases.

## CONCLUSIONS

This study confirmed that lines derived from interspecific crosses can be used as a source of resistance to *Sclerotinia* and *Phomopsis*. Above all, *H. argophyllus and H. tuberosus* may increase the genetic variation of cultivated sunflower in their reaction to both fungi.

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## HÍBRIDOS INTERESPECIFICOS COMO FUENTE DE RESISTENCIA A LA *Sclerotinia* Y AL *Phomopsis* EN EL CULTIVO DEL GIRASOL

RESUMEN

Los agentes patógenos de los hongos *Sclerotinia sclerotiorum* y *Phomopsis helianthi* son dos enfermedades importantes del girasol. Mientras que el girasol cultivado tiene una base genética limitada, las especies silvestres son utilizadas como fuente de resistencia a las enfermedades. En este estudio, 41 líneas diferentes que provienen de cruzamientos interespecíficos, así como el HA89, fueron infectadas por el micelio de *Sclerotinia* y *Phomopsis* en los a□os 1996 y 1997, y despues evaluadas en cuanto a su resistencia. Algunas líneas proveniendo del *H. argophyllus* y del *H. tuberosus* han presentado al nivel del tallo las lesiones de *Phomopsis* las más mínimas inducidas por la *Sclerotinia*. Algunas otras líneas, que han presentado lesiones de *Phomopsis* bastante menores al nivel del tallo que le girasol HA 89, fueron observadas en cruzamiento con el *H. argophyllus*, *H. tuberosus*, *H. deserticola* y *H. xlaetiflorus*. Puesto que BE-94-186-02, TUB-5-3235, TUB-5-326 y TUB-1705-327 presentaron resistencia hacia ambos hongos, su uso en los programas de selección de plantas resistentes se puede aconsejar.

## HYBRIDES INTERSPECIFIQUES EN SOURCE DE RESISTANCE AU *Sclerotinia* ET *Phomopsis* A LA CULTURE DES TOURNESOLS

RÉSUMÉ

*Sclerotinia sclerotiorum* et *Phomopsis helianthi* sont deux agents de maladie importants du tournesol. Parce que le tournesol cultivé a une base génétique restreinte, des espèces sauvages sont utilisées comme source de résistance aux maladies. Dans cette étude, 41 lignées différentes provenant de croisements interspecifiques et la lignée HA89 ont été contaminées infectés par *Sclerotinia* et *Phomopsis* en 1996 et 1997. Les lignées provenant de *H. argophyllus* et *H. tuberosus* ont présenté les lésions les plus restreintes induites par *Scleotinia* au niveau des tiges. Quelques lignées, provenant de croisements avec *H. argophyllus*, *H. tuberosus*, H. *deserticola* et *H. xlaetiflorus* ont montré des lésions de *Phomopsis* au niveau de la tige moins importantes que sur la lignée HA89 du tournesol cultivé. Les lignées BE94-186-02, TUB-5-3235, TUB-5-326 et TUB-1705-327 étaient résistantes aux deux champignons, c'est pourquoi elles sont recommandées pour des programmes de sélection pour la résistance à ces deux pathogènes.