Comparison of Three Methods to Assess Resistance in Sunflower to Basal Stem Rot Caused by *Sclerotinia sclerotiorum* and *S. minor*

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ABSTRACT

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Laboratory and field experiments assessed the relative resistance of sunflower lines to diseases caused by *Sclerotinia sclerotiorum* and *S. minor*. Resistance was measured in terms of the percentage of plants that developed disease symptoms, the time after inoculation when wilting and basal stem lesions became apparent, and the linear rate of lesion development. The linear rate of lesion development of basal stem lesions was considered to be a good measure of relative resistance to basal stem rots under laboratory and field conditions because lines with a high rate of lesion enlargement had a high incidence of basal stem lesions under field conditions and vice versa. The ranking of sunflower lines for disease resistance was similar for both *S. sclerotiorum* and *S. minor*.

In Australia, both *Sclerotinia sclerotiorum* (Lib.) de Bary and *S. minor* Jagger infect sunflowers (2,3,21,26). Australian isolates of these two fungi differ in the size of sclerotia (16,27) and in the predominant mode of sclerotial germination. Sclerotia of *S. minor* readily undergo mycelial germination and rarely produce apothecia under field conditions. Conversely, sclerotia of Australian isolates of *S. sclerotiorum* usually germinate carpogenically by producing apothecia and seldom undergo eruptive mycelial germination. Huang and Dueck (14) found eruptive mycelial germination commonly occurred with North American sunflower isolates of *S. sclerotiorum*.

The way sclerotia germinate influences the location of infection. Infection of sunflower receptacles, leaves, and leaf axils are caused by ascospores of *S. sclerotiorum* (5,11,15,19,28). Root and basal stem rots are caused by hyphae originating from sclerotia of either *S. sclerotiorum* or *S. minor*. Root, receptacle, and leaf lesions often spread to infect adjacent stem areas. Stem lesions cause reductions in potential yield by interrupting the flow of nutrients and water to the seed and increasing the amount of lodging. The earlier a stem becomes infected the greater the reduction in seed yield (8).

The incidence of Sclerotinia diseases of sunflower in New South Wales and Queensland ranged from 0 to as high as 60%, depending on the season and the time of year when the crops were examined (2,3). Both *S. sclerotiorum* and *S. minor* were common on crops in the areas surveyed. Over a 6-yr period from 1978 to 1984, 12% of the maturing sunflower crops in this area showed symptoms of infection by *S. sclerotiorum*, *S. minor*, or both pathogens (*authors' unpublished data*).

We are not aware of any attempts that have been made to screen sunflowers for resistance to *S. minor*. However, variations in susceptibility to *S. sclerotiorum* among experimental lines of sunflower have been reported (19,23–25). Putt (19) noted differences among sunflower lines in the mean percentage of plants that became infected by *S. sclerotiorum*. However, use of percentage of diseased plants to screen sunflowers for disease resistance is often impractical because large numbers of plants are

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required to obtain statistically meaningful results (12,17). Other methods of screening sunflowers for resistance to *S. sclerotiorum* have been developed to improve resolution and efficiency. These have involved placing mycelium in contact with the hypocotyl (1,10), inserting mycelia into stem wounds (17), placing sclerotia adjacent to the roots of field-grown sunflowers (23), and assessing the tolerance of seedlings to oxalic acid (4,13). Resistance to basal stem infection was not always associated with resistance to receptacle infection (6,23).

The objectives of this research were to investigate root and basal stem infection by *S. sclerotiorum* and *S. minor*, and to compare three methods of screening sunflower lines for their resistance to infection by these fungi.

MATERIALS AND METHODS

Selection of sunflower lines. The sunflower lines used in this study were the commercial hybrids Hysun 21R, Hysun 30, and Hysun 32 (Pacific Seeds, Toowoomba, Australia), Suncross 150 (Ag-Seed, Tamworth, Australia), Sunking (Cargill Oilseeds, Toowoomba, Australia), and the inbred line RHA 801 (20). Also included were several inbreds that were reported to have some resistance to *S. sclerotiorum*: Cm 361 (7), Cm 497, Cm 526 (12), HA 61 (9), and 77-5-67-8A. The selection from HA 61, 77-5-67-8A, has a moderate level of resistance to *S. sclerotiorum* (J. Dueck, Agriculture Canada, *personal communication*). Not all of these lines were used in all experiments.

Preparation of inocula and preliminary studies. Thirteen cultures of *S. sclerotiorum* and 14 of *S. minor* were isolated from infected sunflower plants collected from the Liverpool Plains area of New South Wales and from Queensland and were stored as air-dried sclerotia. Preliminary experiments demonstrated no significant differences among isolates of the same species in the percentage of plants that became infected or in the rate of development of basal stem lesions. Based on these results, one isolate derived from a single sclerotium of each species was used in all subsequent experiments. Inoculum was produced by growing each isolate separately on moist, autoclaved wheat grain (170 g of wheat and 150 ml of distilled water in a 500-ml conical flask) for 10 days at 25 C in darkness.

Preliminary studies on plants inoculated at different times after sowing had shown that stems of younger plants were colonized too quickly (up to 8 cm/day) to enable comparisons to be made among sunflower lines, and that older plants became too tall for the growth cabinets. Thus, in the controlled environment experiments all plants were inoculated 30–35 days after sowing, at the early bud stage of plant growth.

Another preliminary experiment investigated the location of *Sclerotinia* mycelia in sunflower stems with rapidly growing lesions arising from root, stem, and leaf inoculations. At various times after inoculation, infected plants were surface-sterilized (30 sec in 70% ethanol followed by 2 min in a 2% sodium hypochlorite solution), sectioned, placed onto V-8 agar, and incubated at 22 C.

Basal stem lesion development on sunflower lines under controlled conditions. A growth cabinet experiment was conducted to compare the susceptibility of six sunflower lines to *S. sclerotiorum* and *S. minor*. Based on previous work (2,3; Sedun and Brown, *unpublished data*), the sunflower lines Hysun 32 and Sunking were designated as susceptible, and Cm 497, Cm 526, and 77-5-67-8A as resistant. Preliminary experiments in our laboratory also had shown that RHA 801 had a moderate measure of resistance to both species of *Sclerotinia* and was, accordingly, designated as resistant.

Sunflower seedlings were grown in 10-cm-diameter plastic pots containing a mixture of peat, vermiculite, and sand (22) in a controlled environment cabinet with 16-hr days (450 $\mu E \cdot m^{-1} \cdot s^{-1}$) at 24 C and 8-hr nights at 18 C. Additional pots of each line served as uninoculated controls. Pots were watered twice daily and fertilized once every 7 days with a complete fertilizer (Aquasol, Hortico Limited, Revesby, NSW, Australia).

A randomized complete block design was used with 12 blocks. Each block contained 12 pots (six sunflower lines by two *Sclerotinia* species). At 30–35 days after sowing, 15 g of inoculum consisting of wheat grain and mycelium was inserted into the outer edge of the soil about 5 cm from the base of the hypocotyl, at a depth of 5 cm, in contact with the plant's roots. The development of lesions on the stem was measured daily, from the first appearance of the lesion above the soil line until the lesion front was no longer discernible. The rate of lesion expansion for each treatment was calculated as the slope resulting from the linear regression of lesion length on time (days). Daily observations were also taken on the development of wilt symptoms on leaves and stems.

Basal stem lesion development on sunflower lines under field conditions. During the 1982–1983 growing season, the sunflower lines Hysun 21R, Hysun 30, Hysun 32, Sunking, Suncross 150, Cm 361, and HA 61 were screened for their resistance to basal stem rot caused by *S. sclerotiorum*. A randomized block design was used with each treatment replicated three times. Plants were grown in four-row plots, 4 m in length, with an interrow spacing of 0.75 m. Shortly after emergence, seedlings were thinned to one plant every 0.25 m of each row. At the early bud stage of plant growth, the middle two rows of each plot were inoculated with 50 ml (25 g) of inoculum inserted into a 3-cm-diameter × 10-cm hole located 10 cm from the base of each stem. The number of plants that showed symptoms of wilt and basal stem lesions was recorded daily for 41 days after inoculation.

During the 1983–1984 and the 1984–1985 growing seasons, the six sunflower lines, Hysun 32, Sunking, Cm 497, Cm 526, 77-5-67-8A, and RHA 801 were planted in a split-plot design with four replicates. The main plot treatment was the fungal species that were used as inoculum, *S. sclerotiorum* and *S. minor*; sunflower genotypes were the subplot units. Each plot consisted of three 6-m rows of sunflowers during 1983–1984 and three 10-m rows during 1984–1985. The interrow spacing was 0.75 m and the intrarow spacing was 0.20 m. The center row of each plot was inoculated as described above. The remaining two rows were used to study the occurrence of "natural" head infections arising from airborne ascospores produced near the test area. The height of each basal stem lesion above the soil surface was measured daily for 37 days after inoculation or until the lesion front was no longer discernible. Plots established during 1984–1985 were located adjacent to the site of the 1983–1984 plots. During 1984–1985, the six sunflower lines were also sown on the same site as the 1983–1984 trial in a randomized complete block design to study the occurrence of "natural" root infection caused by sclerotia incorporated into the soil from the previous season's trial. Meteorological data was monitored at a weather station located 100 m from the trial area.

Lesion development on sunflower roots. To investigate the mechanism of the observed differences in the incidence of root infection between RHA 801 (resistant) and Hysun 32 (susceptible), a glasshouse experiment was conducted to compare lesion development on lateral roots. Plastic tubes (15 cm in diameter, 1 m long) were cut lengthwise and a glass panel was attached to the flat side so that lesion development on roots could be observed. A removable metal plate was slid over the glass to exclude sunlight. Seven plants of each sunflower line were grown singly in the tubes in soil collected from the field test area. At the early bud stage of growth, pregerminated sclerotia of *S. minor* were placed adjacent to a lateral root, about 50 cm from the base of the stem. *S. sclerotiorum* was not used in this study because it was difficult to induce sclerotia to germinate mycelially. The size of root lesions was recorded at daily intervals for 10 days after inoculation. Isolations were made from root lesions as described previously.

All growth cabinet, glasshouse, and field experiments were repeated at least once.

RESULTS

Location of hyphae in stem lesions. Microscopic examination of serial sections of basal stem lesions and the culture of surface-sterilized fresh sections of infected plant tissues showed that hyphae of *S. sclerotiorum* and *S. minor* were associated with the front of advancing lesions (±0.2 cm).

Basal stem lesion development of sunflower lines. During the 1982–1983 field trial, all of the lines developed high percentages of basal stem lesions after inoculation with *S. sclerotiorum*: Hysun 21R (99%), Hysun 30 (98%), Hysun 32 (100%), Sunking (96%), Suncross 150 (100%), Cm 361 (100%), and HA 61 (98%). No significant differences were found among the lines in this test.

During the 1983–1984 and 1984–1985 field trials, significant differences were found among the lines in the percentage of plants with basal stem lesions during midflowering (Table 1). The sunflower lines were divided into two groups based on their susceptibility to both *S. sclerotiorum* and *S. minor*. Hysun 32, Sunking, and Cm 497 had high incidences of infection, whereas Cm 526, RHA 801, and 77-5-67-8A had lower incidences of infection with both pathogens. Similar results were observed on inoculated plants and on plants grown in soil infested with sclerotia of *S. minor*. The two resistance class groupings, based on percent infection, were not evident in growth cabinet studies. With the exception of 77-5-67-8A, *S. minor* infected more plants than *S. sclerotiorum*.

The percentage of inoculated plants with basal stem infection under 1983–1984 and 1984–1985 field conditions was correlated ($r = 0.86$, $P = 0.028$) with the percentage of "natural" head infections observed in the 1983–1984 trial (Table 1). During both seasons, apothecia of *S. sclerotiorum* were found in areas adjacent to the test areas. No head infections were observed during the 1984–1985 season.

In both growth cabinet and field studies, no significant differences were detected among the different sunflower lines in the time after inoculation when wilt symptoms and basal stem lesions first appeared. Wilt symptoms only developed on plants whose tap roots were well colonized by either *Sclerotinia* species. Disease symptoms of *S. minor* developed more rapidly than those of *S. sclerotiorum*, regardless of whether the sunflower lines were grown in the field or in growth cabinets.

Highly significant differences ($P < 0.01$) in the rates of lesion development were found among the sunflower lines (Table 2). Due to a very good fit of the regression of the height of the lesion above ground level and the time after inoculation, a very conservative error term was used, consisting of the sums of squares for slopes. In all tests, the lines could be divided into two groups on the basis of the rate of lesion development. Hysun 32, Sunking, and Cm 497 showed relatively high rates of lesion development compared with Cm 526, RHA 801, and 77-5-67-8A. The order of ranking was similar for both *S. sclerotiorum* and *S. minor*.

In all growth cabinet and field experiments, a range of stem lesion types were observed. The majority of lesions were tan colored, with rates of development greater than 0.4 cm/day. Only this type of lesion was used to compare the rate of expansion of lesions on different sunflower lines incited by either *S. sclerotiorum* or *S. minor*. About 5% of the inoculated plants developed dark brown lesions with very slow growth rates, often less than 0.2 cm/day. No relationship was found between the percentage of plants that developed dark brown lesions and the sunflower line-*Sclerotinia* spp. combination. *Alternaria alternata* (Fr.) Keissler and *Coniothyrium minitans* Campbell were commonly isolated from dark brown lesions. Occasionally, dark brown lesions started to expand rapidly and developed into the light brown lesion typical of *Sclerotinia*-incited lesions. Only the *Sclerotinia* species that was used as inoculum was isolated from the newly developed lesion front.

Lesion development on sunflower roots. Lateral roots of Hysun 32 were colonized by *S. minor* at a significantly ($P < 0.01$) faster rate (0.81 cm/day) than those of RHA 801 (0.51 cm/day). Fifty-six percent of the lateral roots of Hysun 32 developed rapidly expanding root lesions, whereas only 21% were observed on RHA 801. Lesions spread along the lateral roots in both directions from the site of infection. As with stem lesions, a range of lesion types was observed on

Table 1. Percentage of sunflower plants that became infected after inoculation with *Sclerotinia sclerotiorum* and *S. minor*

Sunflower line-pathogen combination	Incidence of basal stem lesions					Head rot
	Growth cabinet		Field trials			Field trials
	Test 1	Test 2	1983/1984	1984/1985	1984/1985	1983/1984
S. sclerotiorum						
Hysun 32	91 a[z]	69 a	88 a	48 c	0	3.9 ab
Sunking	92 a	62 a	82 a	49 c	0	5.9 ab
Cm 497	67 a	69 a	86 a	72 b	0	6.5 a
Cm 526	92 a	100 a	30 b	16 de	0	1.0 c
RHA 801	33 a	55 a	18 b	9 e	0	1.8 c
77-5-67-8A	67 a	33 a	18 b	31 d	0	2.4 bc
Mean	74	72	54	37	0	3.4
S. minor						
Hysun 32	92 a	77 a	97 a	88 ab	79 a	0
Sunking	42 a	62 a	93 a	81 ab	74 a	0
Cm 497	33 a	85 a	96 a	91 a	89 a	0
Cm 526	75 a	92 a	43 b	51 c	38 b	0
RHA 801	38 a	62 a	23 c	19 de	25 b	0
77-5-67-8A	71 a	92 a	40 b	25 de	34 b	0
Mean	59	78	65	59	57	0

[z] Means within each column followed by the same letter were not significantly different using a Duncan-Waller multiple range test at 5%.

Table 2. Rates of linear development of lesions on sunflower lines inoculated with *Sclerotinia sclerotiorum* and *S. minor*

Sunflower line-pathogen combination	Rate of lesion development (cm/day)			
	Growth cabinet		Field trials	
	Test 1	Test 2	1983/1984	1984/1985
S. sclerotiorum				
Hysun 32	1.47 a[z]	1.43 a	0.91 a	1.01 b
Sunking	1.46 a	1.66 a	0.82 a	1.04 b
Cm 497	1.51 a	1.40 a	0.97 a	1.16 a
Cm 526	0.90 b	1.05 b	0.65 b	0.74 de
RHA 801	0.88 b	0.82 b	0.51 b	0.76 d
77-5-67-8A	1.05 b	0.94 b	0.64 b	0.70 de
S. minor				
Hysun 32	1.25 a	1.33 a	0.82 a	0.91 c
Sunking	1.35 a	1.22 ab	0.83 a	0.92 c
Cm 497	1.23 a	1.13 ab	0.82 a	0.96 bc
Cm 526	0.93 b	0.94 b	0.55 b	0.64 e
RHA 801	0.94 b	0.90 b	0.66 b	0.63 e
77-5-67-8A	0.93 b	0.93 b	0.66 b	0.64 e

[z] Means within each column followed by the same letter were not significantly different using a Duncan-Waller multiple range test at 5%.

inoculated lateral roots. These included: 1) no visible lesions (rarely observed); 2) small, dark brown lesions (0.1–1.0 cm in length) that did not develop further; 3) rapidly expanding lesions that stopped suddenly and became dark brown; and 4) rapidly expanding lesions that spread from the inoculated root to the basal stem region of the plant. Lesions on narrow roots (less than 1.5 mm in diameter) stopped suddenly and became dark brown more often than those on wider roots. On very narrow roots (less than 0.5 mm in diameter), fast-growing lesions were not observed. Several fungi, including *S. minor*, *Trichoderma viride* Pers. ex Fr., *Penicillium* spp., and *Fusarium* spp., and several unidentified bacteria, were isolated from dark brown lesions. These fungi were not isolated from rapidly expanding lesions. The spread of hyphae of *S. minor* between roots was only observed when diseased and healthy roots were less than 1 cm apart. Sclerotia were only observed to develop on roots that were more than 1.5 mm in diameter.

DISCUSSION

Various methods have been used to screen sunflowers for resistance to Sclerotinia diseases. The percentage of plants that became infected has been widely used to rank sunflower lines for their resistance to *S. sclerotiorum* (1,5,9,10,12,18,19). The major disadvantages of this method are that large numbers of plants are required to enable valid statistical comparisons to be made and that favorable environmental conditions for disease development do not always occur in the field. Moreover, our results showed that the percentage of plants that developed basal stem lesions under growth cabinet conditions was not a reliable method for ranking sunflower lines for resistance to Sclerotinia diseases.

The time after inoculation, when basal stem lesions and wilt symptoms appeared, varied among the sunflower line-*Sclerotinia* spp. combinations tested. However, these differences were not consistent between the growth cabinet experiments, nor between the growth cabinet and field studies. It was concluded, therefore, that the use of these parameters was also an unsatisfactory method of screening sunflower lines for resistance to basal stem rot.

The use of the rate of basal stem lesion expansion to rank sunflower lines for resistance to *S. sclerotiorum* and *S. minor* provided very consistent results in both field and laboratory studies. Moreover, the ranking obtained using this method was similar to that obtained using disease incidence under field conditions and was in general agreement with reports in the literature. Thus, the rate of lesion development appears to be a simple and effective method of screening sunflower lines for resistance to basal stem rot caused by either *S. sclerotiorum* or *S. minor* under both field and controlled conditions.

Ranking for disease resistance was similar for both *S. sclerotiorum* and *S. minor* among the sunflower lines tested. If this is a general phenomenon, it has obvious implications to sunflower breeding programs and would indicate that the mechanisms of resistance to *S. sclerotiorum* are similar to those for *S. minor*.

Our results showed that the lines Cm 497, HA 61, and Cm 361 were very susceptible to both *S. sclerotiorum* and *S. minor*. This observation differs from previous reports in the literature (7,9,12) that had indicated these lines possessed some resistance to *S. sclerotiorum*. The reasons for these discrepancies require further investigation. The disease reactions of the other lines and commercial hybrids tested were as expected.

Factors affecting differences in the incidence of basal stem lesions between Hysun 32 and RHA 801 (Table 1) were investigated in the experiment studying the colonization of lateral roots by *S. minor*. The lower number of basal stem lesions that were observed on RHA 801 compared with Hysun 32 appeared to have been due to the failure of a larger proportion of root infections to develop into rapidly enlarging lesions. It is possible that the slower rates of lesion enlargement of lateral roots of RHA 801 made the lesions more susceptible to colonization by secondary invaders (15) and that they inhibited the pathogenicity of *S. minor*.

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