Plant Pathology Bulletin 11:15-18, 2002

Screening Sunflower for Resistance to Sclerotinia Wilt

Hung-Chang Huang

Agriculture and Agri-Food Canada, Lethbridge Research Centre, PO Box 3000, Lethbridge, Alberta T1J 4B1, Canada; E-mail: huangh@em.agr.ca; Fax: (403)382-3156
Accepted for publication: December 30, 2001

ABSTRACT

Huang, H. C. 2002, Screening Sunflower for Resistance to Sclerotinia Wilt. Plant Pathol. Bull. 11:15-18.

A 3-year field study was conducted to compare sunflower inbreds and hybrids for resistance to sclerotinia wilt caused by *Sclerotinia sclerotiorum*. Significant differences in resistance to sclerotinia wilt were found among the sunflower inbreds and hybrids tested in each year. The study reveals that the nature of resistance to sclerotinia wilt is inheritable as the hybrids derived from resistant inbreds were more resistant than those hybrids derived from susceptible parents. The study concludes that screening parental inbred lines for resistance to *S. sclerotiorum* is an important step in developing sunflower hybrids with improved resistance to sclerotinia wilt.

Key Words : Sunflower, *Helianthus annuus*, Wilt, Resistance, *Sclerotinia sclerotiorum*

INTRODUCTION

Sclerotinia sclerotiorum (Lib.) de Bary is a soilborne pathogen which can cause two distinct diseases, wilt and head rot, on sunflower (*Helianthus annuus* L.). Wilt is due to infection of sunflower roots by mycelia produced from myceliogenic germination of sclerotia [8] and head rot is due to infection of sunflower heads by ascospores produced from carpogenic germination of sclerotia [7]. Generally, sclerotinia wilt is more prevalent than sclerotinia head rot in Manitoba [6,15,16] and Alberta [13], Canada and North Dakota, South Dakota and Minnesota, USA [3]. Both sclerotinia wilt [2] and head rot [4] can cause heavy losses in seed yield of sunflower.

Several reports indicate that differences in resistance to sclerotinia wilt caused by *S. sclerotiorum* exist in sunflower inbreds [1,11,15] and hybrids [17]. Using an indoor screening technique, Huang and Dorrell [10] reported that wilt of sunflower was associated with the toxic metabolites, mainly oxalic acid, produced by *S. sclerotiorum*. They also observed a significant difference in tolerance to the wilt toxin of *S. sclerotiorum* among the sunflower hybrids and inbreds. The objective of this study was to assess the parental sunflower inbred lines and their hybrids for resistance to sclerotinia wilt in a field infested with *S. sclerotiorum*.

MATERIALS AND METHODS

Sunflower inbreds developed in North America were tested for resistance to sclerotinia wilt in a field near Morden, Manitoba during 1979-1981. All the inbred lines were developed at the Agriculture and Agri-Food Canada Research Centre, Morden, Manitoba, except for the inbreds, RHA 273, RHA 274, HA61, HA 61-1, HA89, HA 113, HA124, 953-88-3, and 8944-S3, which were developed in USA. For the experiments in 1979 and 1980, 21 inbreds were tested in a field with natural infestation of *S. sclerotiorum* and artificial infestation of the pathogen by burying sclerotia collected from diseased sunflower plants in the soil near the seeds at the time of seeding in May, 300 sclerotia per 6-m row. For the experiment in 1981, 25 inbreds were tested in a field naturally infested with *S. sclerotiorum*. Each sunflower inbred (treatment) was planted in two rows per plot (replicate), 6 m in row length and 75 cm in row spacing. After emergence, sunflower seedlings in each row were thinned to establish a within-row plant spacing of about 15 cm. The treatments for the experiment in each year were arranged in a randomized block design with four replicates per treatment. Plants killed by sclerotinia wilt were recorded weekly from late seedling stage to maturity.

Twenty-five sunflower hybrids including experimental hybrids and commercial hybrids (CM101, Morden 15, IS8944, Cargill 205) were tested for resistance to sclerotinia wilt in the field in 1980 and 1981. An open-pollinated cultivar, Saturn, was also used in the test in 1981. The experiment in 1980 was conducted in a field with both natural and artificial infestations of *S. sclerotiorum* using the same seeding and inoculation methods described in the inbred experiments for 1979 and 1980. The experiment in 1981 was carried out in a field naturally infested with *S. sclerotiorum*. Most of the experimental hybrids used in the field trials were developed using the inbred lines that were tested for resistance to sclerotinia wilt in this study.

RESULTS

Resistance to sclerotinia wilt in sunflower inbreds

The incidence of sclerotinia wilt of sunflower varied with the inbred lines tested ranging from 6 to 50% in 1979, 12 to 73% in 1980 and 16 to 70% in 1981 (Table 1). For each year, there was a significant (P<0.05) difference in disease resistance between inbred lines (Table 1). Most of the inbreds showed a consistent reaction to the pathogen in the field trials between years. For instance, the test in each year showed that CM526, CM361, CM527 and HA61-1 were resistant to sclerotinia wilt and CM533, CM447, CM338 and CM379 were susceptible (Table 1). However, inconsistency between years was observed in some inbred lines. For example, CM392 was resistant (10% of wilted plants) in 1979 but was susceptible (49% of wilted plants) in 1980.

Resistance to sclerotinia wilt in sunflower hybrids

Differences in incidence of sclerotinia wilt were observed among hybrid sunflowers tested in the field in 1980 and 1981. Response of a hybrid to sclerotinia wilt was similar between the tests of 1980 and 1981. For example, the hydrid CM526 x HA61-1 was rated as resistant and the hybrids CM533 x CM447, CM338 x CM447, IS8944 and CM533 x CM338 were rated as susceptible in both years (Table 2). Moreover, the resistance in a sunflower hybrid was related to the disease reaction of its parental lines. For instance, the wilt resistant hybrids, CM526 x HA61-1 and CM575 x CM526, were derived from crosses between resistant inbreds, whereas the wilt susceptible hybrids, CM533 x CM447, CM338 x CM447 and CM533 x CM338, were derived from crosses between susceptible inbreds (Tables 1, 2). Also, the level of resistance in several experimental hybrids, CM526 x HA61-1, CM361 x CM526, CM526 x CM392T1, was significantly (P<0.05) higher than the commercial hybrids IS8944, CMH101, Morden 15 (CM400 x CM469) and the open pollinated cultivar Saturn (Table 2).

DISCUSSION

This study confirms previous reports [11,15] that differences in resistance to sclerotinia wilt exist in germplasms of sunflower. The resistant nature of the inbred lines CM526 and HA61-1 observed in present field trials

Table 1. Resistance of sunflower inbreds to sclerotinia wilt caused by *Sclerotinia sclerotiorum* (Field experiments 1979-1981)

1979		1980		1981	
Inbred [1]	Disease (%)	Inbred	Disease (%)	Inbred	Disease (%)
CM 533 *	50 a [2]	CM 338 *	73 a '	CM 533 *	70 a '
CM 447 *	40 ab	CM 447 *	70 a	79-35-2 *	60 ab
CM 379 *	37 a-c	CM 533 *	57 ab	CM 379 *	49 bc
RHA 273 **	35 a-d	CM 558 *	53 a-c	953-88-3 **	48 bc
CM 338 *	34 a-e	CM 379 *	52 a-c	80-37-1 *	44 b-d
CM 303 *	32 a-f	CM 392 *	49 b-d	CM 447 *	39 c-e
CM 400 *	31 a-f	8944-S3 **	48 b-e	CM 338 *	38 c-e
RHA 274 **	31 a-f	CM 400 *	47 b-e	HA 113 **	38 c-e
CM 469 *	27 b-g	RHA 273 **	42 b-f	CM 575 *	37 c-f
CM 509 *	27 b-g	CM 469 *	41 b-f	RHA 273 **	36 c-g
CM 574 *	26 b-g	CM 566 *	40 b-g	79-87-1 *	35 c-h
953-88-3 **	25 b-g	CM 303 *	37 b-h	CM 384 *	30 c-I
CM 558 *	22 b-g	CM 557 *	36 b-h	CM 566 *	30 c-I
CM 361 *	18 c-h	CM 575 *	33 c-h	CM 392 *	29 c-I
CM 557 *	16 d-h	953-88-3 **	29 d-i	CM 382 *	27 d-I
CM 566 *	15 e-h	HA 113 **	27 e-i	CM 469 *	27 d-I
HA61-1 **	15 e-h	HA61-1 **	23 f-i	CM 497 *	25 d-I
CM 497 *	14 f-h	CM 497 *	22 f-i	HA 61 **	24 e-I
CM 575 *	13 f-h	CM 527 *	19 g-i	CM 10 *	23 e-I
CM 392 *	10 gh	CM 526 *	18 hi	CM 527 *	23 e-I
CM 526 *	6 h	CM 361 *	12 i	HA61-1 **	20 f-I
				HA 89 **	19 g-I
				CM 526 *	19 g-I
				CM 361 *	18 hi
				HA 124 **	16 I

[1] * = Canadian inbreds; ** = American inbreds.

[2] Means within columns followed by the same letter are not significantly different at the 0.05 level (Duncan=s multiple range test). Raw percentage disease data are converted to arc sine square root percent, prior to statistical analysis. Data presented using the original scale.

Table 2. Resistance of sunflower hybrids to sclerotinia wilt caused by *Sclerotinia sclerotiorum* (Field experiments 1980-1981)

1980		1981	
Hybrid [1]	Disease (%)	Hybrid	Disease (%)
CM 533 x CM 447 *	51 a [2]	CM 577 x CM 497 *	70 a '
CM 392 x CM 497 *	40 ab	CM (400 x 114) x CM 497 *	61 ab
CM 447 x RHA 273 *	39 a-c	CM 338 x CM 447 *	57 a-c
CM 338 x CM 447 *	37 a-d	CM 533 x RHA 273 *	55 a-c
IS 8944 **	36 a-e	CM 533 x CM 447 *	54 a-d
110-2 x 79-22 (CM 338 x CM 469) *	35 a-f	CM 566 x CM 361 *	52 a-e
CM 533 x CM 338 *	35 a-f	IS 8944 **	50 a-e
CMH 101 **	34 a-f	HA 301 x CM 497 *	50 a-e
CM 533 x RHA 273 *	32 a-g	CM 533 x CM 338 *	48 a-f
Morden 15 (CM400 x CM469) **	30 a-h	CM 526 x HA 113 *	46 a-f
Cargill 205 **	27 a-I	CM (566 x 526) x CM 497 *	45 a-f
CM 392T1 x CM 584T1 *	20 b-j	Saturn ***	44 b-f
CM 566 x CM 526 *	19 b-j	CM 392 x CM 361 *	40 b-g
CM 526 x CM 497 *	18 b-j	CM 392 x CM 497 *	39 b-g
CM 526 x HA 113 *	15 c-j	CM 566 x CM 497 *	39 b-g
HA 301 x CM 469 *	14 d-j	Morden 15 (CM 400 x CM 469) **	38 b-g
CM 497 x CM 361 *	14 d-j	CM (392 x 526) x CM 497 *	37 c-g
CM 526 x CM 361 *	13 e-j	CM 566 x HA 61-1 *	36 c-g
CM 566 x HA 61-1 *	13 e-j	CM 392 x CM 526 *	35 c-g
CM 392 x CM 526 *	13 e-j	CM 526 x CM 497 *	30 d-g
CM 575 x CM 526 *	12 f-j	CM 526 x CM 361 *	30 d-g
CM 392T1 x 8944-T1 *	10 g-j	CM 361 x CM 497 *	29 e-g
CM 392T1 x CM 361 *	9 h-j	Cargill 205 **	24 fg
CM 526 x CM 392T1 *	8 ij	CM 361 x CM 527 *	20 g
CM 526 x HA 61-1 *	5 j	CM 526 x HA 61-1 *	20 g
		CM 361 x CM 526 *	19 g

[1] * = experimental hybrid; ** = commercial hybrid; *** = open pollinated cultivar.

[2] Means within columns followed by the same letter are significantly different at the 0.05 level (Duncan=s multiple range test). Raw percentage disease data are converted to arc sine square root percent, prior to statistical analysis. Data presented using original scale.

confirmed the report by Bazzalo *et al.* [(1)] and the susceptible nature of the inbred line CM400 confirmed the reports by Bazzalo *et al.* [(1)] and Kolte [(11)]. The resistant inbred line, CM526, has been registered and released for use in sunflower breeding [(9)]. The present study showed some inconsistency in resistance to sclerotinia wilt in the inbred line CM392 (Table 1). Kolte *et al.* [(11)] reported that CM392 was susceptible to *S. sclerotiorum* but the progenies selected from two plants of this inbred line showed resistance to sclerotinia wilt. These findings suggest that it is possible to improve an existing inbred through further selection and screening of the progenies of a parental line.

Significant differences in resistance to sclerotinia wilt of sunflower were observed in the experimental hybrids of this study (Table 2) and the commercial hybrids of other studies [(17)]. The present field screening of sunflower inbreds and hybrids in this study indicates that resistance to sclerotinia wilt is inheritable as the resistant character is passable from the parental inbred lines to their hybrids. Thus, screening sunflower inbreds for resistance to sclerotinia wilt is of paramount importance and it should be part of the efforts in the development of new commercial hybrids with high yield potential and superior resistance to *S. sclerotiorum*.

Sunflower inbreds CM447 and CM400 and the hybrid CM338 x CM447 were rated as tolerant based on the tests against the cultural filtrate of *S. sclerotiorum*, containing wilt toxins (mainly oxalic acid) [(10)] but they were rated as susceptible to sclerotinia wilt based on the tests against the pathogen under field conditions (Tables 1, 2). The different results between the test against the wilt toxins of *S. sclerotiorum* and the test against the pathogen suggest that the severity of sclerotinia wilt of sunflower is a complex syndrome affected by the toxic substances such as oxalic acid [(10,12)], the blockage of vascular tissues by hyphal ramifications [(14)] and the degradation of tissues by enzymes [(5)] produced by *S. sclerotiorum*. A high level of resistance to sclerotinia wilt can only be achieved if a sunflower plant is tolerant to the wilt toxin, the cell wall degradation enzymes, and the hyphal proliferation of *S. sclerotiorum* in infected tissues. Therefore, screening sunflower genotypes for resistance to *S. sclerotiorum* by inoculation of the pathogen would be more effective than the tests against the wilt toxin (oxalic acid) produced by the pathogen.

ACKNOWLEDGMENTS

The author would like to express his sincere thanks to Dr. Walter Dedio, sunflower breeder, for providing sunflower inbreds and hybrids for this study and to Alison Rex for technical support.

LITERATURE CITED

1. Bazzalo, M.E., Dimarco, F., Martinez, F., and Daleo, G.R. 1991. Indicators of resistance of sunflower plant to basal stalk rot (*Sclerotinia sclerotiorum*): Symptomatological, biochemical, anatomical, and morphological characters of the host. Euphytica 57:195-205.
2. Dorrell, D.G., and Huang, H.C. 1978. Influence of sclerotinia wilt on seed yield and quality of sunflower wilted at different stages of development. Crop Sci. 18:974-976.
3. Gulya, T.J. and MacArthur, R.A. 1984. Incidence and severity of sunflower diseases in the Dakotas and Minnesota during the 1984 growing season. Page 6 *in*: Proc. Sunflower Res. Workshop. Dec.10, 1984. Fargo, North Dakota.
4. Gulya, T.J., Vick, B.A., and Nelson, B.D. 1989. Sclerotinia head rot of sunflower in North Dakota: 1986 incidence, effect on yield and oil content, and sources of resistance. Plant Dis. 73:504-507.
5. Hancock, J.G. 1966. Degradation of pectic substances associated with pathogenesis by *Sclerotinia sclerotiorum* in sunflower and tomato stems. Phytopathology 56:975-979.
6. Hoes, J.A. and Huang, H.C. 1976. Importance of diseases to sunflower in Manitoba in 1975. Can. Plant Dis. Surv. 56:75-76.
7. Huang, H.C. 1983. Sclerotinia wilt and head rot of sunflower. Canadex 632.145. Agric. Canada.
8. Huang, H.C., and Dueck, J. 1980. Wilt of sunflower from infection by mycelial-germinating sclerotia of *Sclerotinia sclerotiorum*. Can. J. Plant Pathol. 2:47-52.
9. Huang, H.C., and Dedio, W. 1982. Registration of CM497 and CM526 sunflower parental lines. Crop Sci. 22:166.
10. Huang, H.C., and Dorrell, D.G. 1978. Screening sunflower seedlings for resistance to toxic metabolites produced by *Sclerotinia sclerotiorum*. Can. J. Plant Sci. 58:1107-1110.
11. Kolte, S. J., Singh, B., and Tewari, A.N. 1976. Evaluation of sunflower genotypes for resistance to *Sclerotinia sclerotiorum*. Indian J. Mycol. Plant Pathol. 6:65-67.
12. Maxwell, D.P., and Lumsden, R.D. 1970. Oxalic acid production by *Sclerotinia sclerotiorum* in infected bean and in culture. Phytopathology 60:1395-1398.
13. McLaren, D.L., Rimmer, S.R. and Huang, H.C. 1988. Survey of sclerotinia wilt and head rot of sunflower in southern Alberta, 1987. Can. Plant Dis. Surv. 68:126-127.
14. Pawlowski, S.H., and Hawn, E.J. 1964. Host-parasite relationships in sunflower wilt incited by *Sclerotinia sclerotiorum* as determined by the twin technique. Phytopathology 54:33-35.
15. Putt, E.D. 1958. Note on differences in susceptibility to sclerotinia wilt in sunflower. Can. J. Plant Sci. 38:380-381.
16. Rashid, K.Y. and Platford, R.G. 1990. Survey of sunflower diseases in Manitoba in 1989. Can. Plant Dis. Surv. 70:85-86.
17. Rashid, K.Y. and Dedio, W. 1992. Differences in the reaction of sunflower hybrids to sclerotinia wilt. Can. J. Plant Sci. 72:925-930.

摘 要

黃鴻章. 2002. 向日葵菌核病之抗病篩選. 植病會刊11:15-18. (加拿大農部Lethbridge 研究中心；聯絡作者，電子郵件：huangh@em.agr.ca；傳眞：403-382-3156)

由三年的田間試驗結果顯示，於向日葵的自交品系 (inbreds) 或雜交品種(hybrids) 之間對菌核病 (*Sclerotinia sclerotiorum*) 的抗病性均有顯著的差異。此等試驗也進一步表明屬於抗病性的雜交品種，大多源自於抗病性的親本，而屬於感病性的雜交品種，大多源自於感病性的親本。此一研究証明，測定向葵日葵的親本(即自交品系) 對菌核病的抗病性是雜交育種上的一項重要策略。

關鍵詞：向日葵菌核病、抗病性、菌核病菌