Monocropping to sunflower and decline of sclerotinia wilt

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Abstract. A 7-year study in two sunflower fields naturally infested with Sclerotinia sclerotiorum revealed a rapid decline in incidence of sclerotinia wilt associated with monocropping to sunflower. The wilt declined to very low levels in one (Wiebe) field by 1977, about 6 years after the disease reached its peak and in another (G) field in 1980, about 5 years after the peak. Results from testing in 1980 and 1981 in field G showed that the wilt decline phenomenon was persistent and even the addition of sclerotia of S. sclerotiorum to the soil at each seeding period was unable to significantly increase the level of the disease. Analyses of soil samples collected in 1980 indicated a high frequency of hyperparasites of S. sclerotiorum such as Coniothyrium minitans in field Wiebe and C. minitans and Trichoderma spp. in field G. Although C. minitans in field Wiebe originated from the increase of natural population, C. minitans and Trichoderma spp. in field G were probably due to the increase of both the natural population plus the inoculum that was artificially infested in this field to control sclerotinia wilt of sunflower during 1976-79. Because the hyperparasites affect survival of sclerotia of S. sclerotiorum, the significant increase of C. minitans and/or Trichoderma spp. in fields Wiebe and G, suggests that hyperparasitism may be important in the decline of sclerotinia wilt under sunflower monoculture.

Key words: Coniothyrium minitans; Helianthus annuus; Hyperparasitism; Monocropping; Sclerotinia sclerotiorum; Sclerotinia wilt; Sunflower.

Introduction

Wilt caused by Sclerotinia sclerotiorum (Lib.) de Bary is the most important disease of sunflower (Helianthus annuus L.) in Manitoba, Canada (Hoes and Huang, 1976). The disease is caused by infection of the below-ground tissues by hyphea originating from sclerotia of S. sclerotiorum that germinate myceliogenically in the soil (Huang and Dueck, 1980; Huang and Hoes, 1980). Sunflower head rot caused by ascospore infection following carpogenic germination of sclerotia also occurred in fields but it was generally less important than the wilt in Manitoba where sunflowers were grown under dryland conditions (Hoes and Huang, 1976). The plants are mostly infected and wilted during anthesis and seed development (Huang, 1980); thus, an outbreak of sclerotinia wilt can severely reduce yield and quality of sunflower seeds (Dorrell and Huang, 1978).

Numerous reports indicate that sclerotia, the survival structures of S. sclerotiorum, are subject to invasion by soil microorganisms including Coniothyrium minitans Campbell (Campbell, 1947; Jones and Watson, 1969; Schmidt, 1970; Ghaffar, 1972; Huang and Hoes, 1976; Turner and Tribe, 1976; Trutmann et al., 1980), Gliocladium catenulatum Gilman & Abbott (Huang, 1978, 1980), G. roseum (Link.) Bainier (Makkonen and Pohjakallio, 1960), G. virens Millar & Foster (Tu, 1980), and Trichoderma viride Pers. ex Fr. (Makkonen and Pohjakallio, 1960; Jones and Watson, 1969; Huang, 1980). Among these microorganisms, C. minitans was tested intensively under field conditions (Turner and Tribe, 1975; Huang, 1977, 1980; Trutmann et al., 1982) and found to be effective in controlling sclerotia of S.

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Application of C. minitans to the soil significantly reduced the incidence of sclerotinia wilt in sunflower (Huang, 1980; Bogdanova et al., 1986) and reduction of the disease was mainly due to effective destruction of S. sclerotiorum sclerotia by the hyperparasites (Huang, 1980).

Huang (1977), investigating the crop, pathogen, and hyperparasite relationships in a field naturally infested with S. sclerotiorum and C. minitans, found that in the presence of sunflower the population change of S. sclerotiorum was negatively correlated with that of C. minitans and the change was in a zig-zag pattern. Thus, when S. sclerotiorum grew, C. minitans would increase and cause it to decline. The C. minitans would then also decline, making possible renewed activity of S. sclerotiorum and a repetition of the cycle. This suggests that the population dynamics of S. sclerotiorum and its hyperparasites may be influenced by the types of crops, host or non-host, and the types of cropings, monoculture or crop rotation. This paper reports findings from a 7-year study to show the effect of continuous cropping with sunflowers on the development of sclerotinia wilt and to provide the evidence on wilt decline in a commercial field and in an experimental plot.

Materials and Methods

Field History
The study was carried out in three fields of known crop history including two experimental fields designated as G4 and G4 at the Research Station in Morden, and a commercial field designated as field Wiebe near Morden, Manitoba. In field Wiebe, sunflower was grown under monocropping from 1971 to 1977 (Table 1). This field had a heavy natural infestation of S. sclerotiorum and thus it was used as a disease nursery to screen sunflower cultivars for resistance to sclerotinia wilt since 1971. Over 90% of sunflower plants were infected and wilted due to S. sclerotiorum when the field was first used as a disease nursery (J. A. Hoes, pers. commun.). In 1977, the field had become unsuitable for this purpose because of a rapid decline in incidence of sclerotinia wilt; it was then cropped to barley (1978), sunflower (1979), wheat (1980), and pea (1981).

Field G4, about 30 m × 60 m, was used during 1976-1979 in studies on control of sclerotinia wilt by hyperparasites (Huang, 1980). Ninety percent of plants of the cv. Krasnodarets developed wilt in 1975 (Table 1), and the high level of S. sclerotiorum was further augmented by the addition of 44, 250 sclerotia during the 1978–1981 period. During 1976–1979, the field was amended with large amounts of C. minitans and with lesser amounts

<table>
<thead>
<tr>
<th>Year</th>
<th>Crop</th>
<th>Wilt (%)</th>
<th>N</th>
<th>Crop</th>
<th>Wilt (%)</th>
<th>N</th>
<th>Wilt (%)</th>
<th>N + A</th>
<th>Crop</th>
<th>Wilt (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1975</td>
<td>Sunflower</td>
<td>61</td>
<td></td>
<td>Sunflower</td>
<td>90</td>
<td></td>
<td>Barley</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1976</td>
<td>Sunflower</td>
<td>40</td>
<td></td>
<td>Sunflower</td>
<td>43</td>
<td></td>
<td>Alfalfa</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1977</td>
<td>Sunflower</td>
<td>6</td>
<td></td>
<td>Sunflower</td>
<td>40</td>
<td></td>
<td>Fallow</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1978</td>
<td>Sunflower</td>
<td>1</td>
<td></td>
<td>Sunflower</td>
<td>18</td>
<td>43</td>
<td>Sunflower</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1979</td>
<td>Sunflower</td>
<td>1</td>
<td></td>
<td>Sunflower</td>
<td>4</td>
<td>5</td>
<td>Oats</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1980</td>
<td>Wheat</td>
<td>-</td>
<td></td>
<td>Sunflower</td>
<td>5</td>
<td>6</td>
<td>Oats</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1981</td>
<td>Pea</td>
<td>-</td>
<td></td>
<td>Sunflower</td>
<td>1</td>
<td></td>
<td>barley</td>
<td>-</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Field Wiebe was under continuous monocropping of sunflower from 1971 to 1977. The sunflower cultivar was Peredovik for 1975–77 and Saturn for 1979.

*Sunflower cultivar was Krasnodarets for 1975 to 1978 and hybrid 894 for 1979 to 1981. Hyperparasites were applied to field G4 during 1976–78 (Huang, 1980) and 1979.

*N = fields were naturally infested with Sclerotinia sclerotiorum; N + A = fields naturally and artificially infested with sclerotia of S. sclerotiorum.
of *G. catenulatum* and of *T. viride* (Huang, 1980).

Field *G*, adjoining field *G*, was cropped to barley, alfalfa, sunflower and oats during 1975–1981 (Table 1). Field *F* represented a pile of top soil used as the greenhouse soil for the Research Station.

**Assessment of Wilt Incidence**

Symptoms of sclerotinia wilt were recognized by the formation of a brown lesion at stem base and wilting of sunflower leaves. Sunflower cultivars such as open pollinated cvs. Peredovik, Krasnodarrets and Saturn and hybrid 894 are susceptible to sclerotinia wilt. Reported examples of high incidence of the disease on these cultivars in Manitoba include 66% on Peredovik in 1972 and 36% on Krasnodarrets in 1977 (Huang and Hoes, 1980), 60% on Saturn in 1977 (Dorrell and Huang, 1978) and 77% on hybrid 894 in 1984 (McLaren, 1989).

In field Wiebe, the estimated percentage of sunflower plants with sclerotinia wilt in 1975 was based on the yield trial of the sunflower cv. Peredovik grown at 90 cm row spacing and 25 cm within-row spacing (Hoes and Huang, 1985). The percentage wilted plants for 1976 and 1977 was based on the average of four replicates, eight rows/replicate, of the cv. Peredovik used as the check in disease-screening trials; for 1979, it was based on 60 plants of the cv. Saturn at each of 10 random locations, assessed at the late seed development stage.

In field *G* and *F*, the incidence of sclerotinia wilt for 1975 was based on 60 plants of cv. Krasnodarrets at each of 10 random locations, assessed near maturity. For 1976 to 1978, the disease estimates were based on replicated control plots where no hyperparasites were applied in the biological control experiments on the same cultivar as well as on plots artificially infested with *S. sclerotiorum* in 1978 (Huang, 1980). From 1979 to 1981, the percentage wilt was estimated on hybrid 894. In 1979, sunflower was planted in three blocks (replicates) with 90 cm row spacing and 13 cm within-row plant spacing. Sclerotia of *S. sclerotiorum* were applied to each row at the rate of 250 sclerotia per 9 m row. In 1980 and 1981, there were sclerotia treated (300 sclerotia per 12 m row) and untreated control treatments in a randomized block design with five replicates. Each replicate contained six rows of sunflower in 1980 and 4 rows in 1981.

Sunflowers were planted in field *G* in 1978 and 1981. In 1978, the disease was rated on cv. Krasnodarrets from four replicates at four rows/replicate. In 1981, the disease was rated on hybrid 894 in four plots with four rows/plot. The row spacing in this field in 1981 was the same as that in field *G*.

**Testing Soils for Hyperparasites of *S. sclerotiorum***

Three experiments were carried out to test the field soils for presence of hyperparasites of *S. sclerotiorum*. Soil samples taken at 10–15 cm depth and collected during September–October 1980 from 20 random locations in each of the four fields, were pooled by field, and stored at room temperature until processed during December 1980 to April 1981. Actual assessment of hyperparasitic activity was achieved by enclosing sclerotia of the pathogen in fiberglass mesh bags and burying the bags in soil. Sclerotia of tan and black strains of *S. sclerotiorum* (Huang, 1981b) harvested from cultures grown on potato dextrose agar (PDA) at 16°C for 3 weeks were used. Each bag contained 10 black and 10 tan sclerotia in experiment 1, 20 tan sclerotia in experiment 2, and 50 black sclerotia in experiment 3. Bags containing sclerotia were buried at 3 cm depth in the soil samples contained in 20 cm clay pots, one bag/pot, four pots per field soil treatment in each experiment. Pots were placed on a greenhouse bench and irrigated to maintain a moisture of 15 to 25%. Sclerotia were processed following retrieval after 4 weeks of burial in experiments 1 and 3, and after 2 weeks in experiment 2. The retrieved sclerotia were surface-sterilized for 60 seconds in a solution of 95% ethanol and 6% sodium hypochlorite (1:1, v/v), rinsed in sterile distilled water, and dried on paper towels. They were aseptically plated on PDA plates, five sclerotia/plate, incubated at room temperature for 1 week, and checked for viability and contamination by microorganisms.

For experiment 1, an analysis of variance was carried out for percentage of viable sclerotia using a split-plot model that included field soils as whole plots in a completely randomized design and sclerotial type as subplots (Snedecor and Cochran, 1980). For experiments 2 and 3, analyses of variance were carried out to examine effects of field soil on viability of sclerotia and contamination by various microorganisms. An arcsine transformation was applied to the percentage data where necessary to stabilize the variances, and contrasts were calculated to compare control (*G*, *F*) soils.
with hypothesized suppressive (Wiebe, G₄) soils. Regression analyses of percent viable sclerotia on percent sclerotia colonized by microorganisms were also carried out using data from experiment 3.

**Results**

**Decline of Sclerotinia Wilt of Sunflower in Fields Wiebe and G₄**

In field Wiebe the incidence of sclerotinia wilt of sunflower declined sharply between 1975 and 1977 from an initial level of 61% in 1975 and was only 1% in 1979 (Table 1). In field G₄, the percentage of wilt was 90% in 1975 and declined rapidly in the following years, leveling off at about 5% by 1981. A similar response was evident from 1978 to 1981 in plots containing both natural and artificial inoculum of *S. sclerotiorum*. Thus, adding sclerotia to this field failed to cause a significant increase in wilt incidence after four years of treatments with hyperparasites. The sharp decline of sclerotinia wilt between 1975 and 1979 in fields Wiebe and G₄ was not due to climatic factors, as examples of high incidence of sclerotinia wilt were found in those years in other research plots at the Morden Research Station and in some commercial fields near Morden, Manitoba.

In field G₄, the incidence of sclerotinia wilt was very low (1%) in 1978 and 1981 when sunflower was grown (Table 1).

**Viability of Sclerotia of *S. sclerotiorum* in Different Field Soils**

Results of the greenhouse tests showed a significant effect on survival of sclerotia of *S. sclerotiorum* in soil samples from fields F₂, G₂, G₄, and Wiebe (Tables 2-4). Analyses of variance on the viability of tan and black sclerotia retrieved from a 4-week burial in the field soils in experiment 1 revealed significant effects of soil (P<0.05) and sclerotial type (P<0.01) (Table 2). Of the total number of black and tan sclerotia retrieved, the viability of those from fields Wiebe and G₄ was much lower (mean of 46.6%) than those from fields G₂ and F₂ (mean of 82.7%) (P<0.01) (Table 2). Tan sclerotia lost viability more readily than black sclerotia (Table 2). In experiment 2, in which only tan sclerotia had been used, there were fewer (P<0.01) buried sclerotia retrieved from fields Wiebe and G₄ (mean of 43.8%) than from fields F₂ and G₂ (mean of 77.5%) (Table 3). Of the former two fields, 63.9 and 78.5% of the recovered sclerotia were colonized by *C. minitans*, whereas those from fields F₂ and G₂ were all free of this hyperparasite (Table 3).

In experiment 3, where black sclerotia were buried in the soils for 4 weeks, the percentage of retrievable

**Table 2. Viability of tan and black sclerotia of *Sclerotinia sclerotiorum* retrieved from soils after burial for 4 weeks (experiment 1)**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Viable sclerotia (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean±SE*</td>
</tr>
<tr>
<td><strong>Field soil</strong></td>
<td></td>
</tr>
<tr>
<td>Wiebe</td>
<td>41.3±6.1 (43.6)</td>
</tr>
<tr>
<td>G₄</td>
<td>44.7±5.3 (49.5)</td>
</tr>
<tr>
<td>G₂</td>
<td>64.6±5.3 (81.6)</td>
</tr>
<tr>
<td>F₂</td>
<td>66.3±5.3 (83.8)</td>
</tr>
<tr>
<td><strong>Contrast of Wiebe,</strong></td>
<td></td>
</tr>
<tr>
<td>G₄ vs. G₂, F₂</td>
<td>P&lt;0.05</td>
</tr>
<tr>
<td><strong>Sclerotial type</strong></td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td>66.2±3.7 (83.7)</td>
</tr>
<tr>
<td>Tan</td>
<td>42.2±4.0 (45.1)</td>
</tr>
<tr>
<td></td>
<td>P&lt;0.01</td>
</tr>
</tbody>
</table>

*Given in transformed scale (arc sine) with back-transformed means in parentheses; df=23 for the standard error of a mean (SE) following pooling of whole plot and subplot error in the analysis of variance.

**Table 3. Retrieval of sclerotia and colonization of *Coniothyrium minitans* on tan sclerotia of *Sclerotinia sclerotiorum* buried in the soils for 2 weeks (experiment 2)**

<table>
<thead>
<tr>
<th>Field soil</th>
<th>Retrieved (%)</th>
<th>Colonized by <em>C. minitans</em> (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wiebe</td>
<td>27.5±8.6*</td>
<td>63.9±9.5</td>
</tr>
<tr>
<td>G₄</td>
<td>60.0±8.6</td>
<td>78.5±8.2</td>
</tr>
<tr>
<td>G₂</td>
<td>70.0±8.6</td>
<td>0</td>
</tr>
<tr>
<td>F₂</td>
<td>85.0±8.6</td>
<td>0</td>
</tr>
<tr>
<td>Wiebe vs. G₄</td>
<td>P&lt;0.01</td>
<td>P&gt;0.05</td>
</tr>
<tr>
<td>G₄ vs. G₂, F₂</td>
<td>P&lt;0.01</td>
<td></td>
</tr>
</tbody>
</table>

*Mean±standard error of a mean (SE) with 12 df for percentage retrieved and 5 df for percentage colonized by *C. minitans*; data from G₂, F₂ excluded from statistical analysis for *C. minitans*.
Table 4. Colonization of microorganisms on black sclerotia of Sclerotinia sclerotiorum buried in the soils for 4 weeks (experiment 3)

<table>
<thead>
<tr>
<th>Field soil</th>
<th>Sclerotia retrieved (%)</th>
<th>CM</th>
<th>TRI</th>
<th>AC</th>
<th>FUS</th>
<th>AT</th>
<th>RHIZP</th>
<th>BACT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wiebe</td>
<td>77.1(^c)</td>
<td>44.6</td>
<td>12.6</td>
<td>27.7</td>
<td>26.6</td>
<td>7.5</td>
<td>13.9</td>
<td>12.6</td>
</tr>
<tr>
<td></td>
<td>(95.0)</td>
<td>(49.3)</td>
<td>(4.8)</td>
<td>(21.6)</td>
<td>(20.0)</td>
<td>(1.7)</td>
<td>(5.8)</td>
<td>(4.8)</td>
</tr>
<tr>
<td>G(_4)</td>
<td>85.0</td>
<td>55.4</td>
<td>34.8</td>
<td>24.4</td>
<td>13.0</td>
<td>12.6</td>
<td>14.8</td>
<td>12.6</td>
</tr>
<tr>
<td></td>
<td>(99.5)</td>
<td>(67.8)</td>
<td>(32.6)</td>
<td>(17.1)</td>
<td>(5.0)</td>
<td>(4.8)</td>
<td>(6.5)</td>
<td>(2.8)</td>
</tr>
<tr>
<td>G(_2)</td>
<td>79.6</td>
<td>10.0</td>
<td>15.2</td>
<td>30.6</td>
<td>15.2</td>
<td>6.0</td>
<td>7.9</td>
<td>9.8</td>
</tr>
<tr>
<td></td>
<td>(96.7)</td>
<td>(3.0)</td>
<td>(6.9)</td>
<td>(26.0)</td>
<td>(6.9)</td>
<td>(1.1)</td>
<td>(1.9)</td>
<td>(2.9)</td>
</tr>
<tr>
<td>F(_2)</td>
<td>82.9</td>
<td>6.6</td>
<td>11.7</td>
<td>15.8</td>
<td>23.7</td>
<td>14.4</td>
<td>7.8</td>
<td>0.0</td>
</tr>
<tr>
<td></td>
<td>(98.5)</td>
<td>(1.3)</td>
<td>(4.1)</td>
<td>(7.5)</td>
<td>(16.2)</td>
<td>(6.2)</td>
<td>(1.8)</td>
<td></td>
</tr>
</tbody>
</table>

12 df, SE\(^d\)  3.8  3.8 < 0.01 < 0.05 > 0.05 > 0.05 > 0.05 > 0.05

P > 0.05

Contrast of Wiebe, G\(_4\) vs. G\(_2\), F\(_2\)

P > 0.05

< 0.01 < 0.10 > 0.05 > 0.05 > 0.05 > 0.05

\(^a\) Based on 200 sclerotia in each treatment, 50 sclerotia/replicate.
\(^b\) CM = Coniothyrium minitans, TRI = Trichoderma spp., AC = Actinomycetes, FUS = Fusarium spp., AT = Alternaria spp., RHIZP = Rhizopus spp., and BACT = bacteria.
\(^c\) Means given in transformed scale (arc sine) with backtransformed means in parentheses.
\(^d\) Standard error of a mean based on 12 df except for BACT which had 4 df; data from F\(_2\) excluded from the statistical analysis for BACT.

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Fig. 1. Regression of percentage viable sclerotia of Sclerotinia sclerotiorum on percentage sclerotia colonized by Coniothyrium minitans in the soils from fields Wiebe, G\(_4\), G\(_2\), and F\(_2\).

Fig. 2. Regression of percentage viable sclerotia of Sclerotinia sclerotiorum on percentage sclerotia colonized by Trichoderma spp. in the soils from fields Wiebe, G\(_4\), G\(_2\), and F\(_2\).
sclerotia was 95% or higher and there was no significant difference among soil samples from the four fields (Table 4). Microorganisms isolated from the retrieved sclerotia included *C. minitans*, *Trichoderma* spp., *Fusarium* spp., *Alternaria* spp., *Rhizopus* spp., Actinomycetes, and bacteria. No bacteria were isolated, however, from sclerotia buried in the F2 soil. Analyses of variance indicated significant differences among the four field soils in frequencies of sclerotia colonized by *C. minitans* and *Trichoderma* spp. (Table 4). *C. minitans* was more frequent (P < 0.01) on the sclerotia retrieved from soils from fields Wiebe and G4 (mean of 58.6%) than from fields F2 and G2 (mean of 2.2%). *Trichoderma* spp. occurred on 32.6% of the sclerotia buried in soil of field G4, an increase that was significantly higher than those occurring on sclerotia buried in the other three soils (Table 4).

Regression analyses of percentage viable sclerotia (Y) on percentage sclerotia colonized by *C. minitans* (X1) and *Trichoderma* spp. (X2) resulted in significant (Y = 78.82 − 0.2825X1, R² = 0.21, P < 0.10, and Y = 82.71 − 0.893X2, R² = 0.48, P < 0.01) regressions for *C. minitans* (Fig. 1) and *Trichoderma* spp. (Fig. 2), respectively. The regressions of percentage viable sclerotia on the other microorganisms (Table 4) were not significant (P > 0.05).

**Discussion**

The drastic decline in incidence of sclerotinia wilt of sunflower observed in field Wiebe in 1975–79 and field G4 in 1975–81 (Table 1) suggests that as a result of monocropping with sunflower, soil in these fields may have become suppressive rather than conducive to *S. sclerotiorum*. The suppression appeared to be stable for at least 2 years as the disease incidence remained very low in these fields following the initial detection of the dramatic wilt decline.

Soil analyses on the kind and frequency of microorganisms colonizing sclerotia indicated a high population of the hyperparasite *C. minitans* in fields Wiebe and G4. Previous reports show that application of *C. minitans* to *S. sclerotiorum*–infested fields is effective in controlling sclerotinia wilt of sunflowers in Canada (Huang, 1980) and USSR (Bogdanova *et al.*, 1986). Therefore, wilt decline observed in fields Wiebe and G4 may be associated with natural increase (field Wiebe) (Hoes and Huang, 1975) and/or artificial infestation (field G4) (Huang, 1980) of *C. minitans* in the soils. The high frequency of *Trichoderma* spp. detected in field G4 in 1980 may have resulted from the application of *T. viride* to this field in 1976 and 1977 (Huang, 1980). *T. viride* (Huang, 1980) and *T. roseum* (Bogdanova *et al.*, 1986) are ineffective in controlling sclerotinia wilt of sunflower. The fact that *Trichoderma* spp. was high in field G4 but low in field Wiebe suggests that this organism may be of only secondary importance in the decline of sclerotinia wilt in sunflower.

The extremely low levels of sclerotinia wilt of sunflower in field G4 in 1980 and 1981 suggest that preparation of the seedbed each year by ploughing and harrowing may have contributed to the uniform distribution of hyperparasites *C. minitans* and *T. viride*, which were applied to this field in previous years (Huang, 1980). A suppressive effect to natural inoculum of *S. sclerotiorum* appears to have occurred 2 years after the biological control trials commenced in field G4, as the disease declined from over 40% in 1976–77 to 18% in 1978 (Table 1). Another experiment in field G4 in 1978 showed that the addition of sclerotia to the soil at a rate of 300 sclerotia/12 m row increased the disease to 43% (Table 1). However, this level of disease was not maintained and it declined to about 5% by 1980. Thus, adding sclerotia to the field appears to delay the change of soil from conducive to suppressive but does not reverse the process. Once the soil has become suppressive the low level of disease can be maintained for at least 2 years.

Hyakumachi *et al.* (1990) reported rhizoctonia root rot decline in sugarbeet monoculture and they found that the decrease in viable sclerotia of the pathogen and increase in antagonistic microorganisms were involved in the disease decline phenomenon. The present study of sunflower reveals that *C. minitans* may play an important role in inducing soil suppression to *S. sclerotiorum*. This finding is further confirmed in another field trial conducted in Alberta (McLaren, 1989). Since *C. minitans* is widely distributed in sunflower fields in Manitoba (Huang, 1981a) and is highly destructive to *S. sclerotiorum* during the sunflower growing season (Huang, 1977, 1983), any method that will encourage a rapid buildup in the population of the fungus may accelerate the development of a soil suppressive to *S. sclerotiorum*. Thus, further studies on factors affecting population dynamics of *C. minitans* are warranted to determine the importance and signifi-
cance of this hyperparasite and perhaps other microorganisms in soil suppressive to *S. sclerotiorum*.

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向日葵連作與萎凋病之衰褪

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在加拿大 Manitoba 省一塊農田 (Field Wiebe) 及一塊試驗田 (Field G4) 的研究結果發現長期連作可導致向日葵萎凋病 (Sclerotinia wilt of sunflower) 之衰褪，此衰褪現象在第一塊田 (Wiebe) 發生於羅病高峯期後第 6 年 (1977)，而第二塊田 (G4) 於羅病高峯期後第 5 年 (1980)，在第二塊田進一步試驗結果顯示這種萎凋衰褪現象至少可以維持兩年，即使將萎凋病之菌核 (Scelestia of Sclerotinia sclerotiorum) 加入土中亦無法使該田之罹病率再度增高。1980 年秋自由間土壤採樣分析結果顯示菌核病菌之重寄生菌 (hyperparasites) 諸如 Cnitothyrium minitans 有顯著增加的現象，Trichoderma spp. 在第二塊田亦顯著增加，由此推測重寄生菌之劇增可能是導致向日葵萎凋病衰褪的重要原因之一。