

# Importance of disease to sunflower in Manitoba in 1975<sup>1</sup>

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Sclerotinia basal stem rot or wilt, verticillium wilt, and premature ripening ascribed to an unidentified fungal root rot complex appeared to be the most prevalent and the most severe diseases of sunflower in Manitoba in 1975. Downy mildew, head rots, and rust were of lesser importance. Seed contamination, dense plant spacing, improper crop sequence, and host weeds are suggested to be epidemiological factors of sclerotinia wilt. Basal stem rot also occurred in wild *Helianthus annuus*, annual sow thistle, burdock, wild mustard, and volunteer rapeseed. Verticillium wilt often caused uncharacteristic symptoms of leaf yellowing and wilting rather than typical leaf mottle symptoms of chlorosis and necrosis, but masses of microsclerotia in tap roots were diagnostic.

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La sclerotiniose [*Sclerotinia sclerotiorum*], la verticilliose [*Verticillium dahliae*], et une maladie provoquant maturité hâtive, attribuée aux fongues non-identifiées, étaient les maladies les plus communes et graves de tournesols en Manitoba en 1975. Le mildiou [*Plasmopara halstedii*], des pourritures de capitules et la rouille [*Puccinia helianthi*] étaient moins importantes. Graines contaminées avec des scléroties, des espacements petits des plantes trop serrées, des successions de récoltes inappropriées et de mauvaises herbes sont des facteurs épidémiologiques de la sclerotiniose. Les tournesols naturels [*Helianthus annuus*], le laiteron rude, la petite bardane, le moutarde des champs et le moutarde des oiseaux ont été atteints aussi par la sclerotiniose. La verticilliose a causé des symptômes non-caractéristiques de jaunissement et flétrissement de feuilles, plutôt que causant de chlorose et de nécrose, symptômes caractéristiques; des masses de microscléroties dans des racines pivotantes étaient diagnostiques.

Surveys on sunflower (*Helianthus annuus* L.) in 1975 were carried out throughout Manitoba in the September 10-17 period, a time when most sunflower diseases have reached a climax. Fifty-seven randomly selected fields were inspected; their total acreage was equivalent to about 5% of the 1975 acreage, estimated at 28,000 hectares. Oilseed varieties were encountered in all but one field. The distribution of disease within fields was commonly uniform; 300 or more plants in random areas in each field were examined.

## Observations and discussion

The most important sunflower diseases in descending order of prevalence and severity were basal stem rot or wilt caused by *Sclerotinia sclerotiorum* (Lib.) de Bary, wilt caused by *Verticillium dahliae* Kleb., and premature ripening ascribed to an unidentified fungal root rot complex (Table 1).

Downy mildew [*Plasmopara halstedii* (Farl.) Berl. et de Toni], head rots and rust [*Puccinia helianthi* Schw.] were of lesser importance (Table 1). Head rots were caused by *S. sclerotiorum*, *Botrytis cinerea* Pers. and *Rhizopus* sp., among which *S. sclerotiorum* was the most serious pathogen. Rust was widespread but was rather severe in only one field where it caused defoliation.

While no field was completely free of disease, it was common to find two or more diseases in the same field.

For instance, there were 3 fields in which five diseases occurred, and 10 fields in which four diseases were prominent. As a consequence, the importance of disease to sunflower is greater than appears from Table 1. The number of fields with light, moderate, severe, or very severe disease (Table 1) was, respectively, 17, 14, 12 and 3. Therefore, about half of the fields were at least moderately diseased, and there is no doubt that yields in 1975 were seriously depressed. Estimates are that the yield of fields with light or traces of disease ranged from 900 to 2,000 kg/ha, averaging 1,360 kg/ha, while fields with moderate to very severe disease ranged from 100 to 1,350 kg/ha, averaging 800 kg/ha.

Sclerotinia wilt being the most important sunflower disease, the history of fields with a high disease incidence was traced in several instances. Seed contamination, dense plant spacing, improper crop sequence, and weediness are suggested to be epidemiological factors of sclerotinia wilt. The disease spreads by root contact (H. C. Huang and J. A. Hoes, unpublished data; Young and Morris 1927), and, as in solid seeded fields, the closer the spacing, the faster the spread of wilt (J. A. Hoes and H. C. Huang, unpublished data). Contaminated seed stock appeared to be the prime source of inoculum in a field in which 95% of the plants were wilted. No host crop was known to have been grown in that field during the past 10 years, and weeds were no problem. Inspection of the seed stock showed the presence of sclerotia at the rate of 1 per 100 seeds. The seed had obviously been produced by plants with sclerotinia head rot. The sclerotia were free of *Coniothyrium minitans* Campbell and other hyperparasites, and sclerotial viability was high in contrast to that of sclerotia

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Table 1. Incidence of diseases in 57 sunflower fields in Manitoba in 1975

| Disease severity and incidence | Sclerotinia wilt | Verticillium wilt | Premature ripening | Downy mildew | Head rots | Rust |
|--------------------------------|------------------|-------------------|--------------------|--------------|-----------|------|
| None or trace                  | 28               | 37                | 46                 | 51           | 53        | 56   |
| Light (1–10%)                  | 13               | 12                | 9                  | 4            | 3         |      |
| Moderate (11–40%)              | 5                | 7                 | 2                  | 2            | 1         | 1    |
| Severe (41–80%)                | 8                | 1                 |                    |              |           |      |
| Very severe ( 80%)             | 3                |                   |                    |              |           |      |

from plants with basal stem rot (Hoes and Huang 1975). Sclerotia and seed were deposited close together thus facilitating early infection, while disease spread was favored by dense seeding, which resulted in a within-row spacing of only 10 cm between plants. Another field with 95% wilt was solid seeded, and had been grown to rapeseed in 1970, sunflowers in 1971, and peas in 1973. A third instance of a field with 60% sclerotinia wilt and 20% sclerotinia head rot had been partially grown to sunflowers in 1971 and 1972 and had abundant wild mustard. In a field with 70% wilt, sunflowers were grown in rotation with rapeseed. In another instance of a field with 70% wilt, rapeseed had been grown in 1962, sunflowers in 1968, a year in which both sclerotinia head rot and wilt were rampant (Hoes 1969), and oilseed radish (*Raphanus sativus* L.) in 1972. Infested sunflower refuse dragged from an adjoining field was the undoubted source of inoculum in a solid-seeded field with 95% wilt. The adjoining field had been grown to sunflowers in 1972 and again in 1974 and sclerotinia wilt occurred there both years. Sclerotinia wilt also occurred in wild *Helianthus annuus* along a roadside, in annual sow thistle (*Sonchus oleracea* L.), burdock (*Arctium minus* (Hill) Bernh.), and in wild mustard [*Brassica kaber* (DC.) L.C. Wheeler] and volunteer rapeseed (*Brassica campestris* L.).

*Verticillium dahliae* in oilseed sunflowers often caused uncharacteristic symptoms of leaf yellowing and wilting rather than typical "leaf mottle" symptoms of chlorosis and necrosis (Sackston et al. 1957). However, the masses of black microsclerotia discoloring the inside of tap roots were diagnostic.

#### Literature cited

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