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Epidemiology and Resistance to Sclerotinia Head Rot in Wild Sunflower Species

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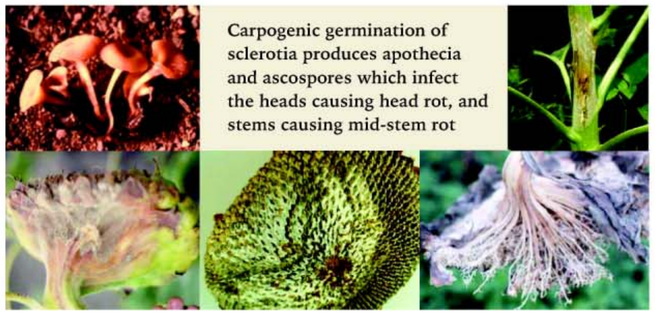
Genetic resistance to Sclerotinia infection in sunflower will save the growers, the processors, and the consumers millions of dollars by avoiding losses in seed yield and quality.

INTRODUCTION

Sunflower head rot results from infections by ascospores that are produced by carpogenic germination of sclerotia of *Sclerotinia sclerotiorum* under saturated soil moisture conditions (Figure 1). Lack of genetic resistance has led to a steady rise in prevalence (65%) and incidence of head rot (up to 50%) in the Red River Valley in recent years. This study aimed to understand the epidemiology of head rot in wild sunflower species, and identify sources of resistance in the wild population.

Figure 1. Sclerotinia head rot in sunflower



MATERIALS AND METHODS

Field experiments were conducted in 2002 and 2003 on 48 accessions each of the wild sunflower species *Helianthus maximiliani* and *H. Nuttallii* collected from southern Manitoba (Figure 2).

A split-split plot design was used with 4 replicates. Main plots were inoculum type and head covering, inoculation dates were secondary plots, and wild sunflower accessions were tertiary plots for a total of 384 plots. Plots consisted of a group of 10-15 plants grown in hills at 0.5 m apart.

Figure 2. Wild sunflower species

Helianthus maximiliani *Helianthus nuttallii*

Artificial inoculum consisted of: ascospores applied at $2x10^5$ per ml with 15 ml per plot; ground mycelia applied at 15 ml of suspension per plot, and ground infected millet seed dusted at the rate of 3 grams per plot. Plots were covered with a light brown paper bag, a thin transparent plastic bag, a perforated pollinating bag, or left open without any covering. Artificial inoculation was done at early flowering (week 1), mid flowering (week 3), and late flowering (week 5).

Plots were assessed weekly for head and stem infections starting one week after inoculation based on a scale of 0 to 5; 0 is no sign of infection and 5 is moderate to heavy infections on 50% or more of the heads or stems. The area under the disease progress curve was calculated for each treatment.

RESULTS AND DISCUSSION

Typical symptoms observed were shredded stems and peduncles below the heads, and shriveled infected heads with little seed setting (Figure 3). Wild sunflower heads are very small and mature rapidly. No typical head rot was observed.

Figure 3. Shredded stems and shriveled heads

The results from the various treatments were similar in both years in spite of the lower severity of infections in 2003. There were minor differences in the reactions of *H. maximiliani* and *H. nuttallii* to the various inoculation treatments. The natural infection in control plots was nil. The ground millet inoculum resulted in the highest disease indices with both wild sunflower species, followed by the ascospore inoculation and fresh ground mycelia as source of inoculum (Figure 4). The light brown paper bags provided the most favourable conditions for infection and disease development (Figure 5). Disease inoculations at mid-, and late flowering produced better results than at early flowering (Figure 6). Eight accessions of *H. maximiliani* and seven of *H. nuttallii* were identified with no sclerotinia infection in both years (Figure 7).

Figure 4. Disease index with type of inoculum

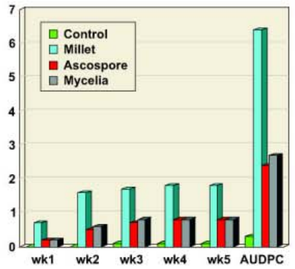


Figure 5. Disease Index with Head Covering

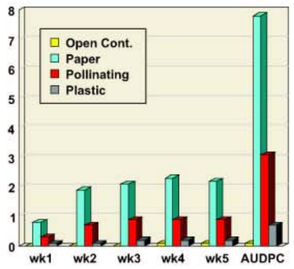


Figure 6. Disease index with inoculation time

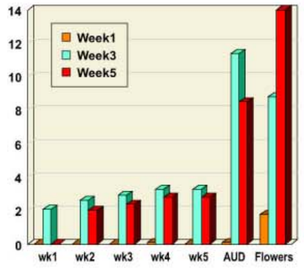
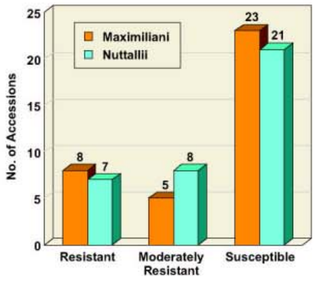


Figure 7. Selected Accessions with Resistance



CONCLUSIONS

Artificial epidemics can be created in wild sunflower:

- Ground infected millet seed or ascospores
- Applied at mid or late flowering stage
- Covered with light brown paper bags

Identified 15 accessions with resistance to *S. sclerotiorum* in wild sunflower species

ACKNOWLEDGMENT

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