Resistance of canola cultivars to *Fusarium oxysporum* f. sp *conglutinans*

Jeremy Klassen, W. G. Dilantha Fernando

Department of Plant Science, University of Manitoba, Winnipeg, MB, Canada R3T 2N2
Email: D_Fernando@Umanitoba.ca

Abstract

Fusarium wilt of canola (*Brassica napus* L.), caused by *Fusarium oxysporum* f. sp *conglutinans* (Wollenweber) Snyder & Hansen, is a disease that has emerged in Western Canada recently. Since it appears that varieties differ in their susceptibility to this disease, one of the first priorities is to determine the availability of genetically resistant germplasm in *B. napus* varieties and advanced breeding lines. One of the current methods to evaluate resistance is a greenhouse root dip inoculation procedure. To validate this method, the greenhouse results for 30 lines were compared to level of infection in a field trial with natural inoculum. Results indicate a positive correlation between greenhouse and field results, substantiating the use of this procedure. In addition, progeny from three crosses between susceptible and resistant lines were evaluated in the greenhouse. The F1 generation (60 plants) appeared resistant, while the segregation of the F2 generation (120 plants) fit a 3:1 ratio of resistant to susceptible. This supports the hypothesis that resistance in *B. napus* is conditioned by a single dominant gene, which would make it easy to exploit in a breeding program.

Key words: Canola, Brassica napus, Fusarium oxysporum

Introduction

Fusarium wilt is a soil borne disease that affects many crops (Nelson et al 1981). Fusarium wilt was not identified in canola however until recently, and was only reported for the first time in Western Canada in 1999 (Alberta Research Council). As such, there is very limited information on the host-pathogen relationship of this particular disease. While occurrence of this disease is relatively rare, when a field is infected yield losses can be high (Alberta Research Council). As a result it is important to understand the host-pathogen interaction to prevent occurrence of the disease as much as possible.

As there is currently no published method for pathogenicity testing of fusarium wilt of canola, it was desirable to determine if greenhouse evaluation of plant susceptibly correlated with field evaluation. To accomplish this, a cross section of lines tested in the greenhouse was evaluated in a field trial where fusarium wilt had been observed the previous year.

It is also desirable to determine the heritability of plant resistance, and so the greenhouse testing procedure was used to evaluate the progeny of crosses between resistant and susceptible lines. Both F1 and F2 generations were tested and compared to expected Mendelian segregation ratios. Since single gene resistance was assumed, the F1 generation was expected to be resistant, with the F2 generation expected to segregate into a 3:1 ratio of resistant to susceptible.

Materials and methods

Greenhouse pathogenicity testing was performed using a root-dip method. In brief, plants are grown in trays in a large heated water bath to provide optimum soil conditions for pathogen development. At the two-four leaf stage plants were uprooted and dipped in an inoculum solution for one hour, and then replanted. After 10-14 days the seedlings were rated for symptoms of fusarium wilt on a scale of 0-9, with 0 representing no symptoms, and 9 representing plant death. For each line tested, two reps of 10 plants were evaluated. An average score greater than 3.5 was considered and indication of susceptibility. From all of the lines tested, a sub sample of 30 was chosen for field evaluation. These were selected to include the full range of greenhouse results, with average scores of 0 through 9. Lines in the field trial were rated at maturity with four reps of 50 plants rated for incidence and severity. The relationship of greenhouse to field results was correlated using linear regression.

To examine the genetic heritability of plant resistance to fusarium wilt a single susceptible line was crossed with three resistant lines, including reciprocals. A sub sample of each cross was evaluated in the greenhouse at the F1 generation to determine the resistance of a heterozygous population. The remaining seed was selfed to produce F2 populations, which were then also evaluated in the greenhouse. The segregation ratios observed in the F2 generation were compared to expected Mendelian ratios using chi square analysis.

Results

Disease levels in the field trial were not very strong, with no lines having disease incidence levels higher than about 60%. Despite being low, they did however correspond well to greenhouse results, with incidence and severity both resulting in linear regression R^2 values of about 84% (Fig. 1).

The F1 progenies of the resistant by susceptible crosses tested in the greenhouse did not differ significantly from the resistant check, with no difference between reciprocal crosses. This indicates that resistance is dominant to susceptibility. In the F2 generation there was no significant difference between crosses. Of the 120 plants evaluated 86 were observed to be resistant with the remaining 34 susceptible. These results were not statistically different from the expected 3:1 ratio of 90

resistant to 30 susceptible. The next closest possible ratio would be the 13:3 ratio expected if there was dominant and recessive epistasis. Chi-square analysis of this ratio results in a rejection of the hypothesis as the probability of our observed ratio would be less than 1% in this scenario.

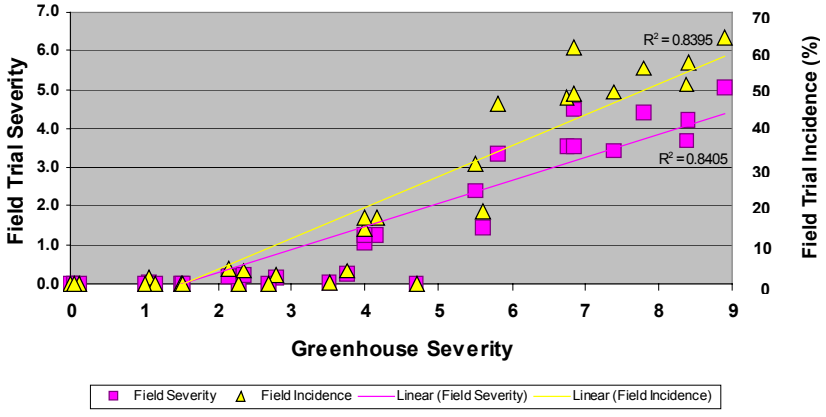

Fig. 1. Field severity and incidence of fusarium wilt vs. greenhouse results in 30 *Brassica napus* lines

Discussion

The positive correlation between field and greenhouse pathogenicity testing indicate that the greenhouse may be a reliable method of determining the susceptibility of *Brassica napus* lines to *Fusarium oxysporum*. This is important as reliable field sites may be difficult to find, and greenhouse testing can be done year round.

The testing of the progeny of susceptible by resistant *Brassica napus* crosses support the hypothesis that resistance to *Fusarium oxysporum* is conditioned by a single dominant gene. It should be reminded though that this type of testing cannot prove a single gene is responsible, and the population may have met this ratio by chance for another reason entirely. It does however indicate that it should be possible to control this disease by selecting against susceptible germplasm and eliminating it from breeding programs.

References

Alberta Research Council. 12 April, 2004. <http://www.arc.ab.ca/extranet/fusarium/>.
Nelson, P. E., Toussoun, T. A. and Cook, R. J. (1981). *Fusarium*: Disease, Biology and Taxonomy. Pennsylvania State University Press, University Park, PA.