

# Development of phoma canker (*Leptosphaeria maculans*) on stems of winter oilseed rape in England in relation to thermal time

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## ABSTRACT

In winter oilseed rape experiments at Rothamsted in 1997/98 (cvs Lipton and Capitol), 1998/99 (cv. Apex) and 1999/2000 (cvs Apex, Lipton and Capitol), development of crown canker and phoma stem lesions in spring was related to development of phoma leaf spot in the previous autumn/winter. Thermal time (degree-days) from first appearance of phoma leaf spot (autumn) to first appearance of crown canker (spring) differed between cultivars (cvs Lipton and Capitol, 1220-1240; cv. Apex, 1120-1140 degree-days) but not growing seasons. In 1998/99 and 1999/2000, fungicide (November) treatment delayed the start of crown canker development in spring but did not affect the rate of increase in severity. In 1997/98, fungicide treatments did not delay the appearance of crown canker but decreased the rate of increase in crown canker severity. The severity of both crown canker and phoma stem lesions increased linearly with accumulated degree-days in plots with or without fungicide treatment in 1997/98 (cv. Lipton), 1998/99 (cv. Apex) and 1999/2000 (cv. Apex). Regressions showed that severity of crown canker at harvest in July was related to severity in the spring in 1997/98 (early June, cv. Lipton), 1998/99 and 1999/2000 (April, cv. Apex).

**Key words:** Phoma stem canker, *Leptosphaeria maculans*, thermal time, phoma leaf spot

## INTRODUCTION

Phoma stem canker (*Leptosphaeria maculans*) is an oilseed rape disease of world-wide importance (West *et al.*, 2000). Ascospore initiated infections cause phoma leaf spots on leaves from which the pathogen spreads to stems by harvest (Hammond *et al.*, 1985; Sun *et al.*, 2000). Both crown cankers and phoma stem lesions can cause yield losses but in the UK greatest losses are usually from severe crown cankers (West *et al.*, 1999; 2002). Good relationships between severity (mean score on a 0-4 scale) and incidence (% plants affected) of canker on crops in England have been observed (Sun *et al.*, 2000). In England, control of severe canker epidemics relies on fungicide sprays in autumn, during the phoma leaf spot phase, some 6 months before symptoms appear on stems (Gladders *et al.*, 1998). There is thus a need to predict, in autumn, the risk that epidemics will become severe and cause yield loss. Attempts to relate the severity of phoma stem canker at harvest to the incidence of phoma leaf spotting in autumn have sometimes been successful (Sun *et al.*, 2000). One factor which affects the rate of progress of *L. maculans* down petioles to stems is temperature. This paper reports results of field experiments to study factors affecting development of crown canker and phoma stem lesions in spring, as a basis for predicting the severity of epidemics.

## MATERIALS AND METHODS

Data from four winter oilseed rape experiments at Rothamsted in the 1997/98, 1998/99 and 1999/2000 growing seasons were analysed. In all experiments, the fungicide used was difenoconazole plus carbendazim. Disease was assessed on plants sampled monthly throughout the growing season from all plots. Incidence (% plants affected) and severity of all diseases, were assessed. Severity of phoma leaf spot was assessed as % leaves affected in autumn/winter. Severities of crown canker and phoma stem lesions were assessed on a 0-4 scale. Five data sets from the four field experiments were analysed. Data sets 1, 2 and 3 provided detailed information for examining relationships between development of phoma leaf spot in autumn/winter and development of crown canker and phoma stem lesions from early spring to harvest, under different fungicide regimes. Data sets 4 and 5 were less detailed but provided additional information on the thermal time (accumulated degree-days >0°C) between

first appearance of phoma leaf spots (date when % plants affected reached 10%, estimated by interpolation between observed data points) and first appearance of crown cankers (10% plants affected). The relationship between development of phoma stem canker and accumulated degree-days was examined by linear regression:  $y = b_0 + b_1T$ , in which  $y$  is severity of crown cankers or phoma stem lesions,  $T$  is accumulated degree-days from date when crown cankers were first observed on stems, and  $b_0$  and  $b_1$  are the regression intercept and slope, respectively. Relationships between initial severities ( $x$ ) of crown canker (April/June) or phoma stem lesions (May/June) (first times when crown canker or phoma stem lesions were observed in most plots) and severities before harvest ( $S$ ) (July) were also examined by linear regression:  $S = a_0 + a_1x$ , in which  $a_0$  and  $a_1$  are the regression intercept and slope, respectively.

## RESULTS

In 1998/99 and 1999/2000, incidence and severity of phoma leaf spot in autumn/winter were greater than in 1997/98. Incidence of phoma leaf spot reached 50% by GS 1,7 (seven leaves) in 1999/2000 and 88% before GS 2,0 (start of stem extension) in 1998/99 but maximum observed incidence in 1997/98 was 35%. Differences between seasons in the start of crown canker development in spring reflected differences between them in development of phoma leaf spot in the previous autumn. Incidence of crown canker was less than 10% by 267 days after sowing in 1997/98 but was about 20% and 40% by 240 and 210 days after sowing in 1998/99 and 1999/2000, respectively. In 1999/2000, the crop reached GS 1,7 by 40 days after sowing, 2 weeks earlier than in 1997/98 and 1998/99. However, in 1997/98, stem extension (GS 2,0) and flowering (GS 4,0) started earlier than in other seasons. The first observation of phoma leaf spotting in 1999/2000 was earlier than in other seasons. In 1997/98 and 1998/99, % plants with phoma leaf spot reached 10% ( $t_p$ ) approximately 55 days after sowing, when plants were at GS 1,7. In 1999/2000, % plants affected reached 10% about 34 days after sowing (cvs Apex and Lipton) or 40 days after sowing (cv. Capitol). Incidence of plants with crown canker reached 10% ( $t_c$ ) about 243 days after sowing (c. 3 weeks after GS 4,0) in 1997/98; 238 days after sowing (at GS 4,0) in 1998/99; and 199, 217, or 229 days after sowing (before GS 4,0) in 1999/2000, for cvs Apex, Lipton and Capitol, respectively.

The time interval in days between appearance of phoma leaf spot  $t_p$  (10% plants affected) and appearance of crown canker  $t_c$  (10% plants affected) differed between cultivars and seasons. For cv. Apex, the time interval  $t_c - t_p$  was 183 days in 1998/99 and 166 days in 1999/2000. For cv. Lipton, it was 188 days in 1997/98 and 182 days in 1999/2000. When time intervals were expressed in thermal time (accumulated degree-days  $>0^{\circ}\text{C}$ ) differences between seasons were much smaller. For cv. Apex, the thermal time interval was 1139 and 1124 degree-days in 1998/99 and 1999/2000, respectively, a difference of 15 degree-days. For cv. Lipton, it was 1223 and 1231 days in 1997/98 and 1999/2000, respectively, a difference of 8 degree-days. For cv. Capitol, it was 1223 and 1236 in 1997/98 and 1999/2000, respectively, a difference of 13 degree-days.

Regressions of severity of crown canker or phoma stem lesions on thermal time in degree-days for controls and fungicide treatments were all significant ( $P < 0.001$ ), all accounting for  $> 80\%$  of the variance. Analyses of parallelism showed no significant differences in the slope  $b_1$  between the eight regression equations for 1998/99 and 1999/2000 (cv. Apex). Thus, the rate of increase in disease severity with thermal time on cv. Apex was similar in both seasons in plots with or without fungicide treatment, although there were different intercepts ( $b_0$ ) for plots with or without fungicide, since fungicide delayed the start of crown canker development. Analyses also showed no significant differences in the value of the intercept ( $b_0$ ) between regressions for untreated and fungicide-treated plots in 1997/98 (cv. Lipton) but there were differences in slopes  $b_1$ , suggesting that fungicide changed the rate of development of the disease in this season. Regressions of crown canker severity at harvest on crown canker severity in spring (on the first date when crown canker was observed in most plots) accounted for 91% of the variance in 1998, 85% in 1999 and 46% in 2000. Analysis of parallelism showed that there was no significant difference in the values of the regression slope  $b_1$  between the 1998/99 and 1999/2000 with cv. Apex. Regressions of phoma lesion severity at harvest on phoma lesion severity in spring accounted for 85% of the variance in 1998 but were not significant in 1999 or 2000. Regressions of incidence of crown canker or phoma stem lesions at harvest on incidence in spring were not significant.

## DISCUSSION

These experiments indicate how the development of crown canker and phoma stem lesions on winter oilseed rape in England in the spring is influenced by the timing and incidence of phoma leaf spot in autumn/winter in relation to cultivar, seasonal temperature and fungicide use. The detailed study of relationships between phoma leaf spot and phoma stem canker in these three seasons confirms that severity of crown cankers in spring is related to the timing and incidence of phoma leaf spot in autumn/winter before stem extension (GS 2,0) (Gladders *et al.*, 1998; West *et al.*, 1999; 2002; Sun *et al.*, 2000), and that severity of phoma stem lesions is related to incidence of phoma leaf spot at or shortly after GS 2,0. The observation that the thermal time interval (degree-days) between 10% incidence of phoma leaf spot and 10% incidence of crown canker may differ between cultivars but not between seasons suggests that the rate of spread of the pathogen *L. maculans* down the leaf petiole may be a function of accumulated temperature, for a given cultivar.

These results confirm the need to accurately time fungicide sprays in early autumn to effectively control severe phoma stem canker epidemics (Gladders *et al.*, 1998; West *et al.*, 1999). The differences in severity of crown canker epidemics on winter oilseed rape between the three seasons studied in the experiments (with similar sowing dates) were associated with large differences in the start of crown canker development. These differences in the start of crown canker development were, in turn, associated with differences between the seasons in the start of leaf phoma spotting in early autumn. Furthermore, it is early in the autumn when plants are at greatest risk of developing leaf spot infections which develop into severe crown cankers, since the leaves have shorter petioles and temperatures are greater than later in autumn/winter (West *et al.*, 1999; West *et al.*, 2000).

The linear relationships between the mean severity scores of phoma stem canker at harvest and accumulated degree-days from canker appearance for different cultivars, different seasons and plots with or without fungicides suggest that increase in severity ( $y$ ) of crown canker and phoma stem lesions is related to accumulated temperature ( $T$ ). Evidence for this relationship is provided by regression analyses showing that crown canker severity at harvest was closely related to crown canker severity in spring, suggesting that the date when crown canker development starts in spring can indicate the likely severity of crown canker at harvest.

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