A mechanistic model for stem canker (*Leptosphaeria maculans*) on winter oilseed rape (*Brassica napus*)

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ABSTRACT

Stem canker (*Leptosphaeria maculans*) is an economically important disease affecting winter oilseed rape in the UK. The dynamics of the disease are driven by weather variables, as temperature and leaf wetness are important for infection to occur (West et al., 1999). The initial phase of infection (caused by air-borne ascospores) happens in autumn and it is followed by four more phases: period to lesion appearance, systemic growth through the petiole, asymptomatic infection of the stem and stem canker development (Hammond and Lewis, 1986). The autumn infections are important because the development of phoma lesions during autumn/winter and severe/damaging stem cankers in spring are related (Sun et al., 2000). Therefore, the rosette stage is critical in understanding the mechanism of the initial infection process. A mechanistic model is developed to describe infection of leaves in autumn and subsequent growth to reach the stem. The model estimates healthy leaf area (new and existing healthy leaf area after dead healthy leaf area has been removed), infected leaf area (symptomless), visible phoma lesion area and probability of the stem getting infected. Therefore, a probability profile can be calculated that associates times of initial infection in autumn with times of stem canker appearance in spring for different weather scenarios and ascospore patterns.

Key words: Stem canker-Weather-Mechanistic model-Rosette stage-Stem infection

INTRODUCTION

Stem canker on winter oilseed rape is a disease of economic importance in Europe, Canada and Australia. The different climatic conditions, growing seasons and oilseed rape cultivars of these areas are some proof of how complex this disease really is. It has already been recognised that *L. maculans* consists of two groups named as A and B group. Furthermore the A group has been divided into more groups of varying pathogenicity and it has been speculated that even more may exist based on the plant species they have been isolated from (West et al., 2001). The presence of the two main A and B groups varies between areas and in the U.K. the A group is predominant. It has also been shown that the A group is mainly responsible for the destructive crown cankers, while lesions caused by spores of the B group have been mainly found higher up on the stem. For any spraying regime to be effective, it is essential to be able to predict if and when severe stem cankers will occur in the spring. As stem canker occurrence has been shown to be linked to the appearance of phoma lesions earlier in the autumn season (Sun et al., 2000), predictions of stem canker risk based on phoma incidence is of considerable practical importance. In a modelling scheme aiming at predicting stem canker risk and more specifically, probability of stem infection occurrence, weather conditions, namely wetness and temperature (West et al., 1999), have to be taken into consideration as these determine the infection conditions. In addition, A group ascospores should provide the inoculum input as these are the ones linked to the majority of crown cankers. Furthermore, since stem canker grows systemically down the leaf petiole (Hammond et al., 1985), the infection load of oilseed rape leaves should be important for how quickly the pathogen can reach the stem. Therefore, we have considered leaf area to be the most appropriate representation of a host-plant during the rosette stage and here we present

a mechanistic model which describes healthy (susceptible), dead (removed), infected and with visible phoma leaf area and calculates the probability of stem infection (Papastamati et al., 2002; Papastamati et al., 2001). The model refers to single-plant level.

MATERIALS AND METHODS

A combination of field data and glasshouse data were used to develop relationships that estimate the area of the (true) leaf layers up to position 21 along thermal time calculated from sowing day of the plant. The field data used are from 30 tagged plants (cv. Apex) sown at Rothamsted during the 2000/01 and 2001/02 seasons. The plants were sown on 23 August 2000 and 14 August 2001, respectively. Every true leaf was marked on appearance so that it could be followed along its life span and recorded as healthy, infected (with visible phoma lesions) or dead. The leaf assessments started on 6 October 2000 and ended on 1 February 2001 during the first season and started on 16 October 2001 and ended on 23 January 2002 during the second season. A glasshouse experiment on an average of 10 ^{o}C and under normal day/night conditions was set up during 2001/02 so that the growth of the petioles of all true leaf layers present during the rosette stage could be observed. Two functions were developed and used to estimate total plant leaf area. The first set of functions estimate the petiole length along thermal time (from sowing) for every true leaf separately. Then, a second function relates the estimated petiole length to a value of leaf area. The estimated total plant leaf areas of all 30 plants from each of the 2000/01 and 2001/02 seasons were used to define a function representing production and death of leaf area, in absence of infection. Then the stem canker model can be defined in terms of leaf area. The model consists of differential equations describing the rate of change of the leaf area in each of the following compartments: susceptible (healthy), S, infected (no visible phoma), I, latent, L, and (visible) phoma, F. There are two delays in the model for the incubation period, τ_1, and the time between phoma appearance and stem infection, τ_2. These are also estimated with differential equations and are temperature-dependent. Three more differential equations estimate the survival probability of leaf tissue during the incubation period, the survival probability of leaf tissue with newly appeared phoma until the pathogen reaches the stem and finally the probability of the stem not getting infected. When the latter is subtracted from unity, the probability of stem infection is derived.

RESULTS

The model was run using daily mean temperature recorded in the field during each season. The leaf wetness duration was estimated by an environmental physics based model predicting leaf wetness duration on winter oilseed rape leaves during the rosette stage. Some of the model parameters were estimated by a fitting procedure (parameters relating to the net healthy leaf area), while the rest were estimated by consulting previous studies on the subject. The model predicts the start of phoma appearance at a time closely agreeing to that recorded in the data. After the phoma starts appearing, a period of time (temperature dependent) elapses before the probability of stem infection starts increasing.

DISCUSSION

Even though stem canker is a monocyclic disease, at least in the U.K., it is quite an involved pathogen to model in a mechanistic format that will provide estimates of stem canker risk. The risk of stem infection must clearly depend not only on the infection load of a leaf during the autumn period, (Hammond and Lewis, 1986; Hammond et al., 1985), but also on the position of each initial leaf infection point on the leaf and its distance from the petiole, as the petiole is the vessel the pathogen uses to travel along the petiole and reach the stem. However, such detailed information is unavailable and would be very difficult to introduce into any model. The model presented here takes into account only the infection load to predict the probability pattern of stem infection, but gives a good agreement between the time of phoma appearance as indicated by the data and as predicted by the model. There is more scope for investigation

relating to looking into stem canker scores recorded much later in each season and seeing how these relate to the probability of stem infection. Such an investigation might give at least a better estimate for the initial value of the time period between phoma appearance and stem iinfection. The model parameter of ascospore infectivity is another variable which is very difficult to measure, but certainly is of interest and also provides scope for further investigation as information relating to inoculum presence and infectivity is important in plant disease modelling.

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