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ScienceDirect
Crop Protection 26 (2007) 618–624
Crop Protection
www.elsevier.com/locate/cropro

Description of the elongation of fire blight canker, caused by *Erwinia amylovora*, in trunks of pear trees

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Received 10 February 2006; received in revised form 29 May 2006; accepted 29 May 2006

Abstract

Fire blight is the most destructive pathogen of pears and other pome fruit. The developing fire blight cankers can cause significant damage to a tree. The models described in this study are devoted to the quantitative evaluation of scale of canker and fitted to the situation of artificial inoculations in the beginning of the summer growth phase (season), in 1-year newly developed shoots of perennial trunks. We evaluated the length of the canker at the end of the summer phase based on data collected in the beginning and in the middle of the season. The models based on logistic, Gompertz and Richards functions were applied. The upper asymptotes of the models served for description of the end-of-season fire blight canker length. These asymptotes were estimated for incomplete data of the middle of the season using the suggested computational method. Results suggested that the elongation of the fire blight canker in trunks of pear trees could be described by logistic and Richards models with better precision than by the Gompertz one. The smallest AIC values were observed with the logistic model.

Keywords: Growth functions; Upper asymptote; Growth rate parameter; Artificial inoculation

1. Introduction

Fire blight, caused by the bacterium *Erwinia amylovora* (Burrill), is the most destructive pathogen of pears and other pome fruit in the Rosaceae worldwide (Beer and Norelli, 1977; Bonn and van der Zwet, 2000; Thomson, 2000). The major economic impact of the disease, reported in 40 countries, was reviewed by Bonn and van der Zwet (2000). The pathogen infects all plant parts, including blossoms, fruit, leaves, shoots, limbs, and trunks. The effects of the disease are devastating and severely infected trees may die (van der Zwet and Beer, 1995).

In Israel, the disease was first detected in 1985 and within 2 years the pathogen had spread to all pear production areas of the country (Zutra et al., 1986). In 1996, severe epidemics led to substantial damage and uprooting of many orchards (Shtienberg et al., 2002). Studies conducted at the Golan Research Institute in 1999 showed that, in the 1990s, in Northern Israel, fire blight caused annual losses ranging from 18% to 76% of income in packing house gate prices (G. Yom Din, unpublished data).

Managing fire blight is difficult, requiring a number of integrated disease management practices to eliminate pathogen sources, reduce bacterial inoculum, limit inoculum spread, prevent plant infection, and reduce plant susceptibility (Steiner, 2000a; van der Zwet and Beer, 1995). Shtienberg et al. (2002) suggested that economic losses could be decreased by making the correct decisions at each stage involving the potential dissemination of the pathogen, (i.e., multiple ''defence lines'' are needed in the management of fire blight).

In years, when blossom infection events do not occur or have been well controlled, active canker sites serve as the primary source of inoculum for a continuing epidemic of secondary shoot blight infections that can lead to major limb, fruit, and tree losses (Steiner, 2000b). The developing cankers that originate from diseased blossoms can cause

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0261-2194/$ - see front matter © 2006 Elsevier Ltd. All rights reserved.
doi:10.1016/j.cropro.2006.05.014

significant damage to a tree, and these cankers serve as the source of inoculum for the secondary phase of the disease (Johnson et al., 2004). Therefore, the quantitative evaluation of scale of canker at the end of its summer growth phase (season) based on data collected in the beginning and in the middle of the season, is important for management strategies and tactics on fire blight dynamics over time. The models for fire blight disease management, MARYBLYT system (Steiner, 1990), and COUGARBLIGHT model (Smith, 1996), are widely used. However, these models do not describe fire blight canker elongation, caused by *E. amylovora*, in trunks of pear trees.

Using nonlinear growth functions for statistical analysis of disease progress provides a useful tool for the analysis of growth. Models based on these functions have been used to describe biological processes in plant pathology, specifically, disease progress curves (Hildebrand et al., 2001; Jeger, 2004; Madden and Nutter, 1995; Madden et al., 2000; Nutter, 1997).

The objective of this study is to make a statistical description of the elongation of fire blight canker during the summer growth phase (May–August) on trunks of pear trees. In this study, we employ logistic, Gompertz and Richards functions for describing growth processes, in relation to fire blight canker elongation in trunks of pear trees. Particularly, the models are compared by their performance on incomplete data records.

2. Materials and methods

2.1. Plants and inoculation

Pear trees, *Pyrus communis* L. cultivars Spadona and Spadocina, which are highly susceptible to *E. amylovora*, were planted in 1996 at the Avney-Eithan experimental station in the Golan region of northern Israel. Fertilization, irrigation, and other cultural practices were as recommended to commercial growers by the Extension Service of the Ministry of Agriculture, Israel (Blachinsky et al., 2003). The annual rainfall in the specific studied region during the winter (October–April) is 500–600 mm. During the spring, moderate temperatures (10–25 °C) and favorable humidity conditions exist (National Weather Forecast Service). The sky is cloudless during most of the summer. The average mid-day relative humidity and temperature in summer are 35–40% and 30 °C, respectively, though night temperatures occasionally fall to 14–20 °C and dew sometimes accumulates on leaf surfaces.

An isolate of *E. amylovora* strain Ea238 was used for all artificial inoculations. This strain is sensitive to streptomycin sulfate, a bactericide that is commonly used for fire blight management by pear growers in Israel. Inoculation was made by injecting a drop (0.1 ml) of bacteria suspension (containing about 10^8 *E. amylovora* cells per ml) to the fresh tissue of newly developed shoots growing from the middle of the trunk of the tree. Two new shoots were inoculated on each tree. To maintain high humidity following inoculation and improve the likelihood of infection, inoculated shoots were covered with wet plastic bags. The bags were removed the following morning. Six trees of each cv. were inoculated in 1999 and six trees of Spadocina and 26 of Spadona were inoculated in 2000. Inoculations were made on 29 April 1999, and on 10 and 23 April 2000. Inoculations in 2000 were repeated on the same trees (on different shoots) to ensure a higher percentage of infection. Since some of the inoculated shoots dried up in the middle of the summer, fire blight canker lengths (cm) were measured only on those trees in which the infection progressed to the supporting branch or trunk. Canker lengths (cm) were measured every 2 weeks in 1999 and every week in 2000 on a total of 32 trees. Measurements made at the end of the summer growth phase served to estimate accuracy of the suggested method.

2.2. The model

Logistic, Gompertz, and Richards functions were used for mathematical description of the change in length of fire blight canker. These functions can be written as the following:

$$y_t = y_0 + (y_\infty - y_0)/(1 + \exp(\alpha + \beta t)) \quad \text{(logistic function)}, \tag{1a}$$

$$y_t = y_0 + (y_\infty - y_0)/\exp(\exp(\alpha + \beta t)) \quad \text{(Gompertz function)}, \tag{1b}$$

$$y_t = y_0 + (y_\infty - y_0)\Big/(1 + v\exp(\alpha + \beta t))^{1/v} \quad \text{(Richards function)} \tag{1c}$$

Evaluation of the suitability of different mathematical functions for describing growth curves in biological applications has been previously demonstrated (Lopez et al., 2004). The symbols used in the models (1a)–(1c) are explained in Table 1. The Richards function has a shape parameter v that can make it equivalent to the logistic ($v = 1$) or Gompertz ($v \to 0$) functions. In this study, we used the shape parameter $v = 2$ of the Richards function (studying different microorganisms, Dalgaard and Koutsoumanis (2001) received stable parameters estimates of Richards model using values of $v = 0.5, 1, 2$).

In models (1a)–(1c), the lower asymptote, y_0, is the minimum canker length, equal to the initial measured canker length, y_t, for each tree. For trees where the sigmoid process developed immediately after inoculation, the value of the lower asymptote y_0 was zero (Fig. 1A). For other trees, there was a delay before the sigmoid process began, and the canker length, not necessarily zero, did not change for several measurements (Fig. 1B). For such trees, the value of asymptote y_0 was the canker length prior to the start of the sigmoid process. The end-of-season