

## Forecasting infections of the leaf curl disease on peaches caused by *Taphrina deformans*

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### Abstract

An Israeli model forecasting leaf curl disease on peaches caused by *Taphrina deformans* was validated in the Emilia-Romagna region of northern Italy, during a three-year period (1996–1998), in 13 cases (year × location × cultivar). When the peach trees are susceptible to infection, the model uses mathematical functions to calculate the risk of infection on the basis of weather conditions (daily rainfall greater than 10 mm, and maximum air temperature greater than 5 °C), and it forecasts periods of possible symptom appearance based on the length of incubation. Peach trees became susceptible to infection between the end of January and mid March, when the first leaf buds attained phenological stage C, i.e. appearance of leaf apex. The trees remained susceptible for at least 9 weeks: the last infection occurred in mid-May.

Since most of the leaf curl onsets observed in the orchards fell within the range of model forecasts, the model proved to be accurate in signalling both the first seasonal infection and repeated infections during the primary inoculum season. Few errors occurred, caused either by conditions of rainfall and temperature lower than the thresholds fixed in the model, or by discrepancies between forecast and actual length of incubation. Infection occurred also at 3.1–3.5 °C, and with 9.6 mm rainfall. Thus, thresholds should not be accepted too rigorously, and perhaps temperature should not be considered as a limiting factor for infection under the conditions of the present work. The length of incubation showed high variability: it was 23 days long on average, with a 95% confidence interval ranging from 20 to 27 days, and extreme values of 9 and 33 days.

### Introduction

The peach leaf curl disease, caused by *Taphrina deformans* (Berk.) Tul., is widespread in all peach-growing areas around the world (Sharma et al., 1987). Disease symptoms change significantly depending on the plant organ where the infection occurs. Yellow to reddish areas appear on young developing leaves in spring; these areas progressively thicken and pucker, causing the leaf to curl. Infected leaves abscise prematurely or sometimes remain attached, gradually turning dark brown on severely infected trees. Fruit set on these trees may be severely curtailed. Green shoots can also

be infected, becoming thickened and distorted. Fruit infections are less frequent and the resulting lesions are irregular raised, wrinkled, and reddish. Disease incidence depends on cultivar susceptibility, on the inoculum density present in the orchard, and on meteorological conditions influencing both overwintering and infection (Ponti and Spada, 1997).

Bud-conidia produced by ascospores during the peach-growing season can survive more than one year on peach twigs and bud surfaces, where they multiply saprophytically to constitute the overwintering inoculum (Mix, 1935). The environmental conditions that favour leaf curl development in spring are not

altogether known (Safran and Levy, 1995). Prolonged rainy periods favour severe outbreaks at orchard level (Mix, 1935), even though it has been demonstrated that the fungus can start to grow at 95% relative humidity (Lorenz, 1976). Cold temperatures favour leaf curl development, too; the temperature for fungal growth ranges from 6 to 26 °C, with an optimum at 18–20 °C (Jeay, 1986; Gautier, 1986). The risk of severe leaf curl outbreaks is high when favourable conditions are prolonged during bud development. Later in the season, general conditions become less favourable for disease development: host susceptibility decreases with the leaf age and higher temperatures limit the disease up until complete inhibition. When the leaves are fully expanded the risk becomes of no economic importance though it is not completely removed (Jeay, 1986).

Since sanitation and cultural practices are ineffective, peach leaf curl is primarily controlled by fungicide applications (Pscheidt, 1995). Spraying results in the destruction of overwintering conidia present on twig and bud surfaces (Mix, 1935). Fungicides are generally applied twice in North Italy: at the end of leaf fall and at the end of winter. A third treatment is recommended before blossoming in those peach orchards severely affected the year before, or when humid or rainy weather persists. The most effective fungicides used in Italy are Ziram and Dodine (Brunelli and Ponti, 1993). No peach cultivars are immune to leaf curl, but susceptibility varies among cultivars (Sharma and Badyiala, 1994), and only a few are resistant (Pscheidt, 1995).

In order to improve disease management, a model integrating published data, laboratory experiments and field observations was developed in Israel (Safran and Levy, 1995). The model computes, for each day, the risk of infection as a function of air temperature, rainfall, susceptibility of peach cultivars, tree phenological stage, and inoculum level. The risk related to the maximum temperature (exceeding 5 °C) is computed by two linear equations, whereas the influence of rainfall is calculated using a Poisson distribution, which determines the probability of infection on the days with at least 10 mm rain. The trees are considered susceptible for a 42-day period after bud break. The influence of both cultivar susceptibility and inoculum level is accounted for by coefficients which are empirically determined. Symptom onset is expected after 14–21 days of incubation.

In this paper we have modified and validated the Israeli model with data collected at seven locations in the Emilia-Romagna region (districts of Ravenna

and Forlì) during a three-year period (1996–1998), with the aim of evaluating its use in our warning system for crop protection (Bugiani et al., 1996).

## Materials and methods

### The model

The infection risk (Risk) was calculated on each day  $i$ , as follows:

$$\text{Risk}_i = \text{Risk}(\text{Ph}_i) \cdot \text{Risk}(R_i) \cdot \text{Risk}(T_i)$$

where Risk ( $\text{Ph}_i$ ) = risk related to the phenological stage of plants; Risk ( $R_i$ ) = risk related to rainfall; Risk ( $T_i$ ) = risk related to temperature.

The risk related to the phenological stage of the peach trees was set at zero until plants reached a susceptible phenological stage, thereafter it was set at one.

To calculate Risk ( $R_i$ ) and Risk ( $T_i$ ), the Israeli model was modified in order to obtain equations more suitable for describing the effect of the meteorological conditions on different pathogen stages (Friesland and Schrödter, 1988).

The effect of daily rainfall ( $R$ ), measured in mm, was calculated by a two-parameter logistic model, which fitted the data of Safran and Levy (1995) well ( $R^2 = 0.99$ ):

$$\text{Risk}(R_i) = 0 \quad \text{when } R_i < 10$$

$$\text{Risk}(R_i) = \frac{1}{1 + e^{(10.7513 - 0.53997 \cdot R_i)}} \quad \text{when } R_i \geq 10$$

The influence of the maximum daily air temperature ( $T$ ), in °C, was calculated using the equation of Analytis (1980). Setting  $T_{\min} = -2$  °C and  $T_{\max} = 39$  °C, and calculating  $T_{\text{eq}} = (T_i + 2)/41$  this equation fitted the data of Safran and Levy (1995) well ( $R^2 = 0.97$ ):

$$\text{Risk}(T_i) = 0 \quad \text{when } T_i < 5$$

$$\text{Risk}(T_i) = [4.6313 \cdot T_{\text{eq}}^{1.3591} \cdot (1 - T_{\text{eq}})]^{1.5766} \quad \text{when } T_i \geq 5$$

Thresholds for both  $R$  and  $T$  were fixed by Safran and Levy (1995); unfortunately, they did not specify clearly whether these thresholds were integers or numbers with decimals. This difference becomes important in automatic model calculations: in the former case, 9.8 mm rainfall (for instance) is conducive to infection, whereas in the latter case it is not. In the present work, thresholds were considered to be 10.0 mm and 5.0 °C.

Table 1. Comparison between actual appearance of leaf curl symptoms on peach leaves in some commercial orchards, and forecasts made by the model

Year	Location	Cultivar	Date of bud break <sup>(1)</sup>	Model outputs		Actual symptom appearance
				Estimated infection	Estimated symptom appearance	
1996	Cesena (FO)	Zincal 5	Jan. 24–31	Feb. 19 Feb. 21	Mar. 4–18 Mar. 6–20	Mar. 17
		Early Star	Mar. 1–8	Mar. 16 Mar. 27	Mar. 30–Apr. 12 Apr. 10–23	Apr. 10
		Big Top and Sweet Red	Mar. 12–19	Mar. 16	Mar. 30–Apr. 12	
				Mar. 27	Apr. 10–23	Apr. 10
1996	Tebano (RA)	Aurelio Grand	Mar. 11–18	Mar. 16	Mar. 30–Apr. 12	Apr. 11
1996	Conselice (RA)	Caldesi 2020	Mar. 11–18	Mar. 12	Mar. 26–Apr. 8	—
				Mar. 16	Mar. 30–Apr. 12	Apr. 11
1997	Tebano (RA)	Aurelio Grand	Feb. 19–26	Mar. 19 Mar. 24	Apr. 2–15 Apr. 7–20	Apr. 15
1997	Zattaglia (RA)	Julia	Feb. 8–15	—	—	Mar. 19
				Mar. 19	Apr. 2–16	Apr. 15
				Mar. 25	Apr. 8–22	
				Apr. 20–22	May 4–20	May 15
1998	Zattaglia (RA)	Venus	Feb. 12–19	Feb. 23	Mar. 9–22	Mar. 19
				Mar. 10	Mar. 24–Apr. 6	—
				Mar. 23	Apr. 6–19	Apr. 9
				Apr. 12	Apr. 26–May 9	May 6
				May 4	May 18–31	—
1998	Cesena (FO)	Zincal 5	Jan. 26–Feb. 2	—	—	Feb. 26
		Early Star	Feb. 9–16	Mar. 24	Apr. 8–21	Apr. 2
		Alessandra and	Feb. 19–26	Mar. 24	Apr. 8–21	Apr. 8
		Zaiger				

<sup>1</sup>When the first leaf buds reach the phenological stage C (appearance of leaf apex).

Cultivar susceptibility and inoculum level were not considered, because insufficient information about their role on the occurrence of infection was available. On the basis of our previous observations (unpublished), the incubation period (number of days elapsing between the date of infection and symptom appearance) was set at 14–28 days.

#### Model validation

Several observations were carried out to verify the following aspects of the model: (i) beginning and length of tree susceptibility to infection; (ii) infection occurrence; (iii) length of the incubation period.

The research was carried out in one experimental orchard at the 'Martorano V' Experimental Station (Cesena, Forlì), and in seven commercial orchards at different locations in the Ravenna and Forlì districts. At the Experimental Station, several observations were

performed in 1996 on an experimental site where 95 peach cultivars were grown under the same cultural conditions. In the commercial orchards observations were carried out during a three-year period (1996, 3 orchards; 1997, 2 orchards; 1998, 2 orchards); since at Cesena 1996 and 1998 four cultivars were observed in the same orchard, 13 cases (year × location × cultivar) were considered altogether (Table 1). To ensure the presence of the overwintering inoculum, orchards that had shown leaf curl symptoms in the previous season were selected, and no fungicides were applied either at leaf fall or at the end of winter. Plants were traditionally cultivated in the palmate or in the vase-shaped system. To obtain representative situations, the peach plants were grown according to common practice.

In the experimental orchard 12 trees × cultivar (3 contiguous trees × 4 replicates) were monitored, whereas in the commercial orchards 40 trees (10 contiguous trees × 4 contiguous rows) were tagged. Starting from early January, these trees were carefully

observed at weekly intervals, to assess the phenological stages of the plants, from dormancy to bud break. After bud break, the trees were inspected two or three times per week, to determine the time of symptom onset.

A simple assessment key for the development of leaf buds was developed to define tree phenology. As in the key proposed by EPPO (1984), three growth stages were considered: A, winter buds; B, bud swelling; C, appearance of leaf apex (or bud break). Assessments were made by observing all the buds of the tagged trees. The stage of the most developed buds was used to define the growth stage in an orchard, rather than the prevalent stage of all the buds. It was assumed that bud break could take place on any day between the day of observing bud break for the first time and the previous survey, when trees had not yet reached this phenological stage.

To detect symptom onset, all the young leaves of the tagged trees were carefully inspected to note the appearance of the first symptoms as reddish, thickened or puckered areas. When the symptoms were unclear, the leaves were marked and observed during the following surveys. In most cases, inspections were stopped after the appearance of the first seasonal disease symptoms, but in two cases (at Zattaglia in 1997 and 1998), inspections were continued to note the onset of subsequent symptoms.

#### *Tree susceptibility to infection*

Data collected at the experimental station were used to define the phenological stage when peach trees become susceptible to *T. deformans* infection. The 95 peach cultivars were observed weekly to assess the bud phenological stage, as described in the previous paragraph. On each day when weather conditions were favourable to infection ( $R_i \geq 10.0$  mm;  $T_i \geq 5.0$  °C), the number of cultivars in each phenological stage was counted. All these potential infections were divided into real infections (infections that resulted in disease symptoms) and not real infections (infections that did not lead to any symptoms); two or more close infections which resulted in only one symptom onset were considered as a successful clustered infection. Then, for each phenological stage, the relative frequencies of successful and non-successful infections were calculated.

To define the length of the period when trees remain susceptible to infection, the number of days that elapsed between the date when plants became susceptible (i.e. bud break) and the date of the last successful infection

were calculated, separately in each of the 13 cases considered in the commercial orchards.

#### *Infection*

Validation of the model accuracy in estimating the day when infection occurred was performed by comparing actual symptom appearance with the model forecasts, in all the 13 cases considered (year  $\times$  location  $\times$  cultivar).

Daily values of temperature and rainfall were recorded at each site by mechanical or electronic meteorological equipment set at the orchard level. The model was operated daily starting from February 1, using  $R_i$  and  $T_i$  as driving variables, and the risk of infection based on weather conditions was calculated, Risk ( $R_i$ ,  $T_i$ ). When trees became susceptible to infection, the period of possible appearance of the first symptoms was calculated on each day when Risk ( $R_i$ ,  $T_i$ ) was greater than zero, considering an incubation period of 14–28 days.

It was assumed that the model provided a satisfactory prediction when the observed symptom onset coincided with the interval forecast by the model.

#### *Length of the incubation period*

The length of the incubation period was calculated by computing both the number of days and the degree-day accumulation (base 0 °C) between infection and symptom appearance.

### **Results**

#### *Tree susceptibility to infection*

In the experimental orchard, no symptoms were observed on plants exposed to weather conditions favourable for infection at phenological stage A. At stage B, symptoms were observed in 3 cases out of 25, and at stage C, symptoms appeared in 70 out of 72 cases (97%). Therefore, in the following validation, it was assumed that trees became susceptible to *T. deformans* infection when some leaf buds reached phenological stage C (bud break). In the 13 cases considered in commercial orchards, the number of days between bud break and the last infection varied from a few to 65 days at Zattaglia in 1997 (Table 1).

## Infection

Leaf curl always appeared in all the peach orchards considered. In 11 of them, only the first seasonal symptom onset was observed, while at Zattaglia in 1997 and in 1998 symptoms appeared on three separate occasions. Therefore, 17 cases of symptom appearance were considered altogether (Table 1). Trees reached phenological stage C between the beginning of February and the middle of March, and the first seasonal disease symptoms were observed between the end of February and the first half of April (Table 1). When observations were repeated during the season to note the appearance of new symptoms after the first onset, symptoms continued to appear until mid-May (Table 1).

At Cesena in 1996, bud break was first observed on January 31 on 'Zincal 5', on March 8 on 'Early Star', and on March 19 on 'Big Top' and 'Sweet Red'; the model predicted infections on February 19 and 21, March 16 and 27, with risks of 0.52, 0.01, 0.44, and 0.07, respectively. Disease appearance was forecast to occur between March 4 and 20 on 'Zincal 5', between March 30 and April 12 or between April 10 and 23 on the other two cultivars. The first disease symptoms were actually observed on March 17 on 'Zincal 5'; on April 10, leaf curl symptoms were present on all the other cultivars. Clustered infections that occurred on 'Zincal 5' on February 19 and 21 were correctly estimated by the model. Disease onset on the other cultivars was also correctly estimated, considering infection occurred on March 16. No more disease symptoms appeared following the infection signalled on March 27, or they overlapped with the ones caused by the previous infection.

At Tebano in 1996, bud break occurred between March 11 and 18. Afterwards, favourable weather conditions for infection were recorded on March 16, with a risk of 0.35. Symptoms were forecast to appear between March 30 and April 12, and actual disease appearance was observed on April 11.

At Conselice in 1996, both tree phenology and meteorological conditions favoured the infection on March 12 and 16 (with risks equal to 0.007 and 0.45, respectively). Symptoms of these infections were forecast to appear between March 26 and April 8, and March 30 and April 12, respectively. Actual disease appearance was observed on April 11, in agreement with the second infection signalled by the model, while, the first infection did not result in the appearance of symptoms during the forecast period.

At Tebano in 1997, bud break was observed on February 26, but weather conditions were unfavourable to infection establishment until March 19 and 24, when risk was equal to 0.07 and 0.4, respectively. Disease onset was predicted between April 2 and 20 and was observed on April 15.

At Zattaglia in 1997 (Figure 1), the model forecast 5 infections following bud break (first observed on February 15): March 19 and 25, and April 20–22; the corresponding risks were 0.71, 0.49, 0.4, 0.02, and 0.003. Disease appearance was forecast in the periods: April 2–22 and May 4–20. Sporadic leaf curl symptoms appeared on March 19; very severe symptoms were observed on April 15 and, to a lesser extent, on May 15. The last two disease onsets were correctly predicted by the model, whereas the first disease appearance was not. The analysis of meteorological data showed

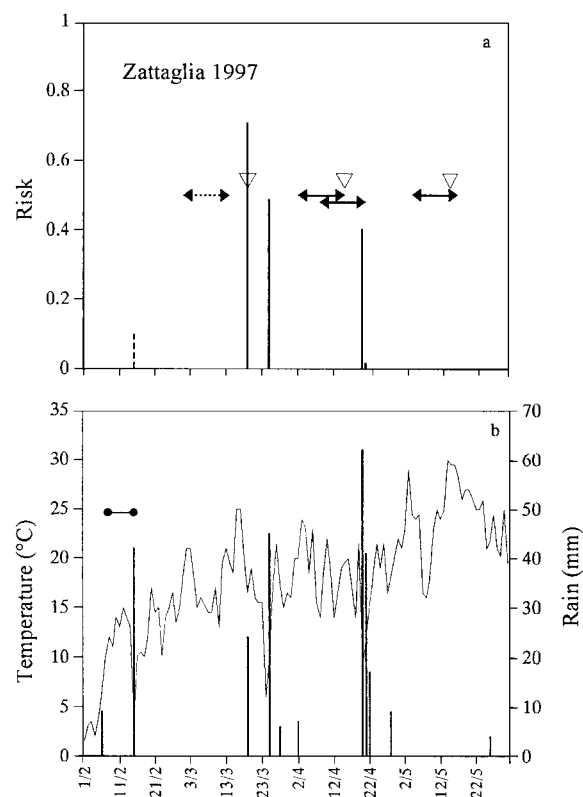


Figure 1. Validation of model outputs against *T. deformans* infections occurring at Zattaglia in 1997: (a) risk of infection (|), period of forecast disease appearance (↔), actual disease onset (▽) (dotted lines represent infections not signalled by the model); (b) maximum air temperature (—), rainfall (|), period when leaf buds break (●—●).

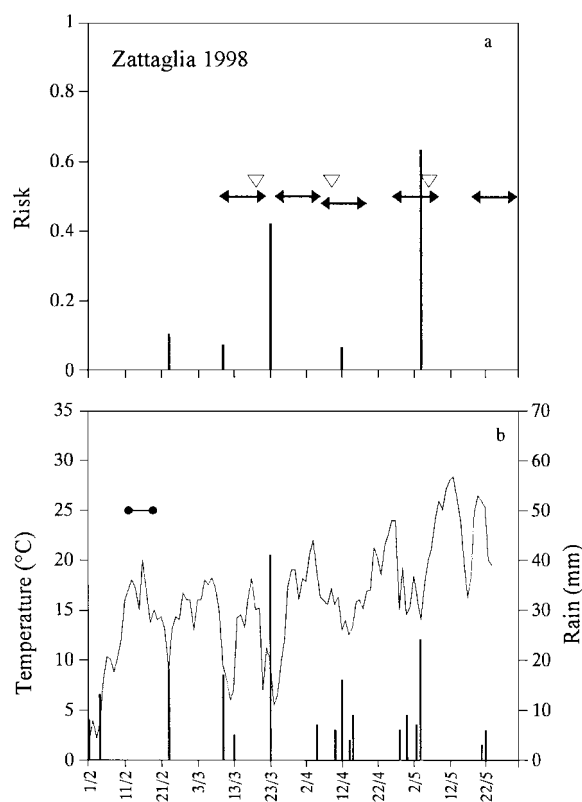


Figure 2. Validation of model outputs against *T. deformans* infections occurring at Zattaglia in 1998: (a) risk of infection (|), period of forecast disease appearance ( $\leftrightarrow$ ), actual disease onset ( $\nabla$ ); (b) maximum air temperature (—), rainfall (|), period when leaf buds break (●—●).

that the infection provoking the symptoms observed on March 19 probably occurred on February 15, because it was the only rainy day between bud break and symptom onset (Figure 1). The model did not signal this infection because  $T$  was less than  $5.0^{\circ}\text{C}$  (precisely  $3.5^{\circ}\text{C}$ ); if this infection actually occurred, the following incubation period was 33 days.

At Zattaglia in 1998 (Figure 2), bud break was first observed on February 19. The model signalled five possible infections: on February 23, March 10 and 23, April 12, and May 4, with risks equal to 0.1, 0.07, 0.42, 0.06, and 0.63, respectively. Disease appearance was considered to be possible in the periods: March 9–22, March 24–April 6, April 6–19, April 26–May 9, May 18–31. The disease actually appeared on March 19, April 9 and May 6, as a consequence of the infections on February 23, March 23 and April 12, respectively. No symptoms were observed following the infection

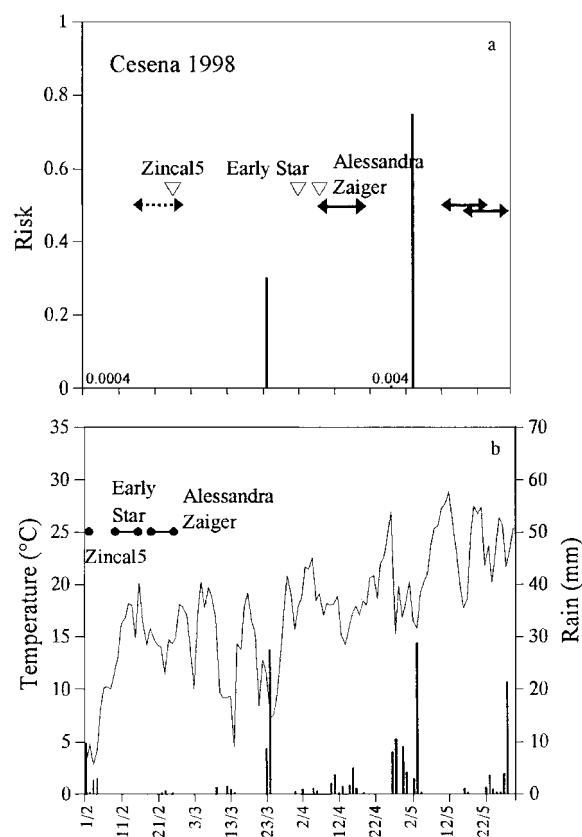


Figure 3. Validation of model outputs against *T. deformans* infections occurring at Cesena in 1998: (a) risk of infection (|), period of forecast disease appearance ( $\leftrightarrow$ ), actual disease onset ( $\nabla$ ) (dotted lines represent infections not signalled by the model); (b) maximum air temperature (—), rainfall (|), period when leaf buds break (●—●).

signalled on March 10, though they were forecast to appear between March 26 and April 6. The appearance of new symptoms following the infection signalled on May 4 was not detected.

At Cesena in 1998 (Figure 3), four cultivars were considered; bud break was recorded on February 2 on 'Zincal 5', on February 16 on 'Early Star', whereas on 'Alessandra' and 'Zaiger' this stage was reached on February 26. Weather conditions were favourable to infection on February 24, with a risk value of 0.3; disease onset was forecast between April 8 and 21. Leaf curl symptoms appeared on February 26 on 'Zincal 5', on April 2 on 'Early Star', and on April 8 on the other cultivars. Model outputs agreed with actual leaf curl onset for 'Alessandra' and 'Zaiger', whereas symptom appearance on 'Zincal 5' and 'Early Star' was not

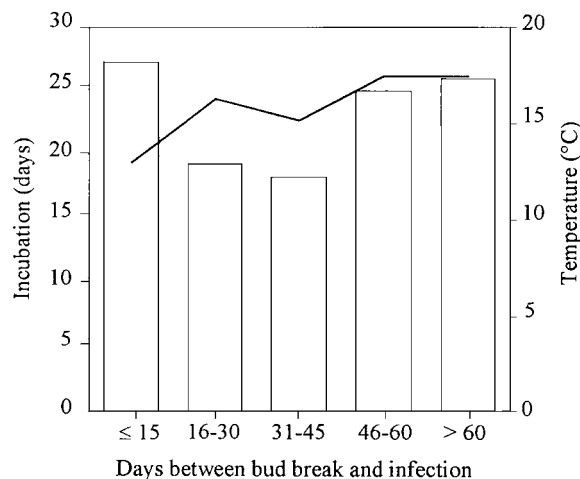


Figure 4. Relationship between the length of the incubation period of the *T. deformans* infections and the time elapsed between bud break and infection establishment (divided into 15-day intervals). The line shows the mean air temperature measured in the different time intervals.

forecast by the model. For the former cultivar, leaf curl onset was probably caused by an infection that occurred on February 1, because no other significant rain fell either before or after this date (Figure 3); the model failed to signal this infection because  $R$  was 9.6 mm and  $T$  was 3.1 °C (both lower than the thresholds fixed in the model, 10.0 mm and 5.0 °C, respectively). For 'Early Star', disease onset was probably caused by the infection established on March 24, because no other significant rainfall occurred after bud break (Figure 3). Therefore, incubation was very short (9 days). The model signalled two more infections, on April 28 and May 4 (Figure 4), but no observations were made to check the appearance of new symptoms on peach leaves.

No relationship was found between the magnitude of the risk values and the occurrence of infection. In fact, infections occurred at about the same frequency over the whole range of the risk values.

#### *Length of the incubation period*

Incubation length was very variable, ranging between a minimum of 9 days (registered at Cesena in 1998, on 'Early Star'), and a maximum of 33 days (the first appearance at Zattaglia in 1997). The average number of days elapsed between the date of infection and the date of disease appearance was 23, with a

coefficient of variation (CV) equal to 25%. The degree-day accumulation did not reduce variability in incubation length. Degree-days were 127.5 and 493.0 °C for the two previously cited extremes of the range of variation, respectively, and the average was 342 °C, with CV equal to 28%.

Incubation length changed according to the time that elapsed between the time of bud break and of infection; it was longer for the infections that occurred within 15 days of bud break (28 days incubation on average), or in the 4th and 5th 15-day period after bud break (25–26 days incubation), than for those established between the 2nd and 3rd 15-day period after bud break (19 and 18 days incubation on average, respectively) (Figure 4). During the above-mentioned 15-day periods, the average air temperature increased from 12.9–17.4 °C (Figure 4).

#### **Discussion**

Results showed that peach trees became susceptible to infection when the first leaf buds broke. In fact, plants always became infected when the first leaf buds reached phenological stage C (appearance of leaf apex) and the weather was favourable to infection establishment. In only three cases were plants infected at stage B. In these cases the phenological stage was probably underestimated because of differences between cultivars in both bud morphology and development after dormancy, and the high variability between and within plants of the same cultivar throughout the orchard.

After bud break, trees remained susceptible for at least 9 weeks. In fact, infections occurred up to 65 days after bud break, until mid-May. This result agrees with Jeay (1986), who found that the tree may remain susceptible till June, but is in contrast with Safran and Levy (1995), who fixed the limit of susceptibility at 42 days after bud break. Probably, the meteorological conditions that influence the rate of plant phenological development affect the length of plant susceptibility. In fact, peach leaves are susceptible to *T. deformans* when emerging from the bud and for a short time thereafter, until they develop undifferentiated tissue, and are no longer receptive (Mix, 1935; Lorenz, 1976). The findings from the present work are not exhaustive and do not exclude later infections. Therefore, it will be necessary to perform specific studies to define the length of peach susceptibility to infection better.

The results presented showed a reasonable agreement between the infections forecast by the model and

the actual occurrence of infection, as inferred from the appearance of leaf curl symptoms on peach trees. In only four cases were the prediction of the model and reality far apart. This occurred in two cases (at Zattaglia in 1997 and at Cesena in 1998 on 'Zincal 5') where the model failed to signal an actual infection, and in two cases (first infection signalled at Conselice in 1996, and second infection signalled at Zattaglia in 1998) where the model predicted infections but symptoms did not actually appear within the subsequent forecast period. The first kind of error occurred when  $R$  and  $T$  were lower than the thresholds fixed in the model (10.0 mm and 5.0 °C, respectively): at Zattaglia in 1997 infection probably occurred with  $T$  of 3.5 °C, whereas at Cesena in 1998 infection on 'Zincal 5' probably occurred with 9.6 mm of rain and 3.1 °C of maximum temperature. While the differences in rainfall were small and possibly due to the use of decimals in fixing the threshold (10.0 instead of the integer 10), the differences in temperature were certainly significant. Perhaps temperature should not be considered a limiting factor for infection. In fact, Agarwala et al. (1966) showed that leaves became affected when maximum temperatures ranged between 1.5 and 20 °C. The second kind of error made by the model resulted from a discrepancy between forecast and actual length of incubation. Actually, at Conselice in 1996 and at Zattaglia in 1998 the leaf curl symptoms appeared 3 days after the period predicted by the model. Generally, the length of incubation showed high variability, even greater than the range of 14–21 days fixed by Safran and Levy (1995) and of 14–28 days assumed in the present work. The incubation period was 23 days long on average, with a 95% confidence interval ranging from 20 to 27 days, but minimum and maximum values were 9 and 33 days. Considering the variability in incubation length of different peach cultivars (Sharma and Badiyala, 1994), these results are in substantial agreement with Jeay (1986), who observed that incubation lasts 2 or 3 weeks depending on weather, and with Gautier (1986), who indicated that the incubation is 20–25 days for early infections and 15 days for late infections, occurring at the end of March to beginning of April. However, data of the present work showed that later infections, occurring after the first ten days of April, are connected with longer incubation periods of 25–26 days. Increasing air temperature could account for the shortening of incubation during the first 45-day period after bud break (from 13 to 15–16 °C), but it did not explain the lengthening of incubation in the following period, when temperature (around 17.5 °C) was close to the optimum levels

for the fungus (Agarwala et al., 1966). Probably, when such late infections occur, the longer incubation period is caused by a reduced susceptibility of the host tissues to fungal invasion, as demonstrated in other pathosystems (Yang et al., 1998; Van Delden et al., 1993). The shortest incubation period observed in the present work was 9 days and is clearly below the previously cited ranges; therefore it must be verified.

The magnitude of the response variable Risk ( $T_i$ ,  $R_i$ ) calculated by the model does not seem to play a role in the occurrence of infection. In fact, no relationship between the risk values and infection was found, the former ranging from little more than zero to more than 0.6 when infections occurred. Certainly, the understanding of Risk (which gives a measure of weather conductivity to infection) in forecasting disease outbreaks cannot leave the inoculum dose out of consideration.

During the period of tree susceptibility, the model signals new infections whenever favourable weather conditions occur, even after the first disease appearance. The results from Zattaglia in 1997 and 1998 showed that the model made accurate forecasts of the infections occurring at any time during the primary inoculum season. However, these results are insufficient to draw general conclusions about the accuracy of the model in forecasting the appearance of leaf curl symptoms caused by repeated infections during the season. Additional studies on the dynamic of overwintering of *T. deformans* inoculum during the peach-growing season will be necessary for the practical application of the model. In fact, the repeated occurrence of infections depends on the contemporary presence of susceptible plant tissue, a sufficient inoculum dose on plants and favourable weather conditions.

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