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Originalarbeit

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SIMCOL – A Decision Support System for integrated control of anthracnose on blue lupin

SIMCOL – Ein Entscheidungshilfesystem zur integrierten Bekämpfung der Anthraknose an Blauer Lupine

Abstract

The weather-based decision support system (DSS) SIMCOL for the anthracnose – lupin pathosystem was developed. SIMCOL forecasts the lupin growth development on the basis of temperature (in form of BBCH growth stages) and recommends based on temperature, relative humidity, precipitation and field specific information a control strategy for anthracnose (*Colletotrichum lupini*) of blue lupin (*Lupinus angustifolius*).

The DSS consists of the following 3 models:
- SIMONTO-Lupin: simulation of the BBCH development of *L. angustifolius*,
- SIMCOL1: calculation of the first disease occurrence and the beginning of the treatment in the field,
- SIMCOL3: calculation of dangerous infectious periods and scheduling of treatments with the help of a fungicide module (factors for the influence of seed contamination and seed treatment were also integrated).

For the analysis of the pathosystem and the modelling the following data-sets were used:
- Data from climate chambers to study the relationship between temperature, leaf wetness duration, plant growth stage and disease development. These data were used as a basis for the modelling of the disease efficiency,
- Literature data for the development of the SIMONTO-Lupin model,
- Historical data of field disease development from monitoring activities for the models validation,
- Specific laboratory experiments to analyse the effect of fungicides on the disease development to obtain a fungicide efficacy factor.

The validation of SIMCOL1 and SIMONTO-Lupin was carried out with independent data from the Governmental Crop protection services of the federal states Brandenburg, Mecklenburg-Western Pomerania and Saxony-Anhalt, the seed breeder Saatzucht Steinach, the Julius Kühn-Institut (Federal Research Centre for Cultivated Plants) and the ZEPP and delivered good results.

The DSS is available since the season 2011 for advisory activities of the Governmental Crop protection services and for the seed producers.

Key words: *Colletotrichum lupini*, anthracnose, *Lupinus angustifolius*, blue lupin, Decision Support System, integrated control

Zusammenfassung

Für das Pathosystem Anthraknose an Lupine wurde das wetterbasierte Entscheidungshilfesystem (EHS) SIMCOL entwickelt. SIMCOL prognostiziert temperaturbasiert die Entwicklung des Lupinenbestandes als BBCH-Stadien und empfiehlt auf der Basis von Temperatur, relativer Luftfeuchte, Niederschlag und schlagspezifischen Informationen eine Bekämpfungsstrategie für die Anthraknose (*Colletotrichum lupini*) an Blauer Lupine (*Lupinus angustifolius*).

Das EHS besteht aus den 3 folgenden Modellen:
- SIMONTO-Lupine: Berechnung des BBCH-Verlaufs von *L. angustifolius*,
- SIMCOL1: Berechnung des Befallsbeginns,
- SIMCOL3: Berechnung gefährlicher Infektionsperioden und Planung der Fungizidstrategie mit einem Fungizid-

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modul (Faktoren für den Einfluss des Saatgutbefalls und der Beizung wurden zusätzlich integriert).

Für die Analyse des Pathosystems und die Modellerstellung wurden folgende Daten verwendet:
- Daten von Klimaschrankversuchen zur Untersuchung der Zusammenhänge zwischen Temperatur, Blattnässedauer, Pflanzenentwicklung und Krankheitsentwicklung als Basis zur Modellierung der Krankheitseffizienz,
- Literaturdaten zur Entwicklung des Modells SIMONTO-Lupine,
- Historische Monitoringdaten zur Krankheitsentwicklung im Feld für die Modellvalidierung,
- Spezielle Laboruntersuchungen zur Untersuchung der Fungizidwirkung auf die Krankheitsentwicklung zur Modellierung eines Faktors, der die Fungizidwirkung abbildet.

Die Validierung von SIMCOL1 und SIMONTO-Lupine wurde mit unabhängigen Daten der Pflanzenschutzdienste der Länder Brandenburg, Mecklenburg-Vorpommern und Sachsen-Anhalt, sowie der Saatzucht Steinach, dem Julius Kühn-Institut und der ZEPP durchgeführt und hat gute Ergebnisse geliefert. Das EHS steht ab der Saison 2011 der landwirtschaftlichen Praxis über die Beratung als auch im Bereich der Saatguterzeugung zur Verfügung.

Stichwörter: *Colletotrichum lupini*, Anthraknose, *Lupinus angustifolius*, blaue Lupine, Entscheidungshilfesysteme, Integrierte Bekämpfung

Introduction

Several species of the genus *Colletotrichum* cause serious anthracnose diseases of numerous important annual crop and ornamental plants (Agrios, 2005). Early and non-controlled infections can cause severe yield losses (Dick, 1994; Lindbeck et al., 1998; Read et al., 1996; Yang and Sweetingham, 1998). *Colletotrichum lupini* is the most widespread and damaging disease of narrow leafed lupin (*Lupinus angustifolius*) both in the major producing regions such as South America, New Zealand or Australia (Davidson et al., 2005) and in the lupin growers regions of Germany (Römer, 2000; Römer, 2007). It was first identified in Western Australian lupin crops in 1996. In 2002 the pathogen, previously been referred to as either *C. gloeosporioides* or *C. acutatum*, was renamed to *C. lupini* (Nirenberg et al., 2002). Recently, lupin anthracnose isolates have been classified in two variants, *C. lupini* var. *setosum* (widespread mostly in South Africa) and *C. lupini* var. *lupine* (widespread in the other lupin growing countries worldwide) (Lotter and Berger, 2005).

Biology of the lupin anthracnose

The pathogen survives mainly in form of mycelium with acervuli on the seeds (Feiler and Nirenberg, 2004) or on alternative hosts such as other legume crops, plant debris and rotten fruits in the field (Pring et al., 1995). *Colletotrichum* species can also naturally produce micro-sclerotia to allow dormancy in the soil during the winter or when exposed to stressful conditions (Pring et al., 1995). Anyway, in Germany, the primary mode of disease spread is through infected seed (Feiler and Nirenberg, 2004), only in growing areas in the southern hemisphere also the weed lupin (*L. nootkatensis*) can be considered a primary inoculum source (Thomas, 2007).

During warm and wet periods, conidia from acervuli and micro-sclerotia are splashed by rain from diseased to healthy plants. The conidia germinate to form appressoria on plant surfaces, from which penetration hyphae develop into plant cells. Infection may occur through almost any plant surface. Under suitable conditions, the fungus can grow rapidly inside the plant and cause severe symptoms very quickly. Once the fungus has developed sufficiently inside the plant, dark fruit-bodies are produced, causing typical anthracnose symptoms. A pale pinkish or sometimes orange spore mass develops within the lesions. The spores are normally dispersed by water splash (Agrios, 2005; Davidson et al., 2005).

Warm and humid weather (Römer, 2007), rainfall and prolonged leaf wetness lead to a strong and rapid spread of the infestation (Thomas and Sweetingham, 2004). The disease affects all parts of the lupin plant but the inflorescences and pods are more susceptible than the young plants (Davidson et al., 2005; Klocke and Nirenberg, 2007).

Disease control

Several chemical and physical methods are used to acquire low or non-infested seed (Nirenberg, 1999; Römer, 2000; Thomas and Adcock, 2004; Thomas and Sweetingham, 2003) however, if favourable weather conditions exist, an epidemic development is always possible.

Some fungicide experiments demonstrate the impossibility to stop a *Colletotrichum* epidemic under way. As a consequence an early infection-oriented use of fungicides may be the correct strategy and suggests the development of a prognosis model (Klocke and Nirenberg, 2007; Nirenberg, 1999).

For this reason SIMCOL (**SIM**ulation model for **COL**letotrichum), a modular, weather-based decision support system (DSS) was developed in Germany by the Central Institution for Decision Support Systems in Crop Protection (acronym ZEPP). The DSS should be used by the Governmental Crop protection services as well as in seed production.

The DSS SIMCOL, according to the ZEPP nomenclature (Racca et al., 2010), consists of three prognosis models:
1. SIMONTO-Lupin to forecast the BBCH-stages of *L. angustifolius*;
2. SIMCOL1 to forecast the first disease appearance;
3. SIMCOL3 to forecast infectious periods and for the planning of the fungicide strategy.

Ontogenesis modelling (SIMONTO-Lupin)

The development of the infection of *C. lupini* is in addition to temperature and leaf wetness duration strongly dependent on the plant development stage (BBCH) (HACK et al., 1992). For the simulation of the crop stages a temperature development rate was modelled using literature data and historical monitoring data (RACCA et al., 2011; RACCA and TSCHÖPE, 2011; TSCHÖPE and RACCA, 2010).

Modelling Colletotrichum disease epidemics (SIMCOL1 and SIMCOL3)

Both models SIMCOL1 and SIMCOL3 were developed by correlating the monitoring field data (disease appearance and incidence) with the daily epidemic pressure index (EPI). The daily EPI was calculated as follows:

$$[1] \qquad EPI = \sum_{L=1}^{n} DE$$

Where:
EPI = daily epidemic pressure index
DE = disease efficiency
L = latency period (1 = start, n = end)

Modelling the disease efficiency
The epidemics of fungal diseases are highly correlated with weather conditions (particularly large impact parameters are the temperature and the leaf wetness). Disease development data (disease incidence) obtained from trials in which lupin plants in different BBCH growth stages were artificially inoculated with *C. lupini* and incubated under controlled conditions in a climate chamber (KLOCKE, 2007; KLOCKE and NIRENBERG, 2007) were used as basic data to model a disease efficiency rate (DE) (Tab. 1) (BERGER et al., 1995).

DE is dependent on temperature, leaf wetness and growth stages of lupin. During a leaf wetness period, the DE is calculated as follows:

$$[2] \qquad DE = f(Tw, LWD, BBCH\_S)$$

Where:
Tw = average temperature during the leaf wetness period (°C)
LWD = leaf wetness duration in hours
BBCH_S = BBCH-dependent susceptibility stage (from SIMONTO-Lupin)

The influence of temperature and leaf wetness on the DE was assessed by interpolating the values of the maximum disease incidence recorded in the trials using a combination of Richards function (RICHARDS, 1959) and modified beta- Hau function (HAU, 1988) [3] for each of the 4 different BBCH-susceptible stages (21–23 (2–4 leaves), 39 (12 leaves), 60–69 (flowering) and 70–87 (pods development)).

Tab. 1. Climate chamber trials on blue lupin cultivar Arabella and Bora – Disease development depending on temperature, leaf wetness and BBCH growth stages (KLOCKE, 2007; KLOCKE and NIRENBERG, 2007)

temperature	leaf wetness duration (hours)	BBCH – growth stage
5°C 10°C 15°C 20°C 25°C	4 to12	21–23 (2–4 leaves) 39 (12 leaves) 60–69 (flowering) 70–87 (pods)

$$[3] \qquad DE_{BBCH\_S} = [Y_{max} \times ((1 - EXP(-(a \times LWD))))^{b}] \times \left[Y_{max} \times \left(\frac{Tw - T_{min}}{T_{opt} - T_{min}} \right)^{\left(n \times \frac{T_{opt} - T_{min}}{T_{max} - T_{opt}} \right)} \times \left(\frac{T_{max} - Tw}{T_{max} - T_{opt}} \right)^{n} \right]$$

Where:
DE_{BBCH_S} = disease efficiency depending on Tw and LWD for each of the 4 different BBCH-susceptible stages
LWD = leaf wetness duration in hours
Tw = average temperature during the leaf wetness period (°C)
Y_{max} = maximum disease incidence
T_{min}, T_{max}, T_{opt} = estimated minimum, maximum and optimum temperature for the disease efficiency (°C)
a, b and n = equation parameters

The regression parameters are summarised in Tab. 2 and one example of the DE calculated for the BBCH 21–23 (2–4 leaves) is shown in Fig. 1.

Modelling of the latency period
The function for the latency period was calculated by means of a beta-Hau function [4] with the same data-pool used to model the DE.

$$[4] \qquad L_{(1/d)} = \left[L_{max} \times \left(\frac{T - T_{min}}{T_{opt} - T_{min}} \right)^{\left(n \times \frac{T_{opt} - T_{min}}{T_{max} - T_{opt}} \right)} \times \left(\frac{T_{max} - T}{T_{max} - T_{opt}} \right)^{n} \right]$$

Where:
$L_{(1/d)}$ = latency (expressed as a daily rate 1/day)
L_{max} = maximum daily latency rate
T = daily mean temperature (°C)
T_{min}, T_{max}, T_{opt} = estimated minimum, maximum and opti-

Tab. 2. Estimated parameter values of the combined Richards and beta-Hau function for the DE of *C. lupini* as a function of temperature (°C) and leaf wetness duration (hours) for the BBCH stages 21–23, 39, 60–69 and 70–87

BBCH growth stage	parameter	value	S.E.	P	r^2
21–23 (2–4 leaves)	Y_{max}	1,00	0,04	<0,0001	0,89
	a	0,68	0,20	0,0011	
	b	66,39	80,96	0,4159	
	T_{min}	5,00	8,13	0,541	
	T_{max}	30,02	0,36	<0,0001	
	T_{opt}	23,05	0,54	<0,0001	
	n	1,20	0,70	0,0922	
39 (12 leaves)	Y_{max}	1,00	0,07	<0,0001	0,80
	a	0,66	0,25	0,0112	
	b	159,32	298,17	0,5956	
	T_{min}	5,00	11,08	0,6538	
	T_{max}	30,01	0,16	<0,0001	
	T_{opt}	20,64	1,07	<0,0001	
	n	0,85	0,70	0,2298	
60–69 (flowering)	Y_{max}	0,90	0,05	<0,0001	0,85
	a	0,78	0,29	0,0095	
	b	110,91	196,08	0,574	
	T_{min}	0,00	0,30	1	
	T_{max}	30,00	0,00	<0,0001	
	T_{opt}	26,05	2,07	<0,0001	
	n	0,42	0,39	0,2933	
70–87 (pods development)	Y_{max}	1,00	0,01	<0,0001	0,98
	a	1,51	0,65	0,0252	
	b	155,63	405,17	0,7025	
	T_{min}	5,00	5,26	0,3461	
	T_{max}	30,00	0,00	<0,0001	
	T_{opt}	21,62	0,56	<0,0001	
	n	0,25	0,13	0,0573	

mum temperature for the latency period (°C)
n = equation parameters

The parameter values are summarized in Tab. 3.

The EPI is calculated as the sum of the accumulated daily average values of DE during the latency period. An example of the EPI is shown in Fig. 2.

Development of SIMCOL1 model

For the prognosis of the disease onset (SIMCOL1) the calculated EPI was correlated to the monitored beginning of infestation in historical field data (n = 54). Particularly the number of days during the latency period (calculated retrospectively from the beginning of infestation) was classified as follows: days with EPI > 0.1, > 0.2, > 0.4, > 0.6 and > 0.8. The aim was to define a minimum combination of the EPI able to forecast a disease appearance. The classification criteria of the EPI were chosen by minimising the belated prognosis (simulated appearance later then recorded appearance).

After the data analysis the best result (77% of correct prognosis, 23% of belated prognosis) was obtained with the following criteria:

- at least 69% of the days during the latency period with EPI > 0.1;
- at least 54% with EPI > 0.2;
- at least 35% with EPI > 0.4;
- at least 12% with EPI > 0.6;
- at least 4% with EPI > 0.8.

The model simulation begins with the crop sowing date. When one of the above conditions is verified SIMCOL1 gives the disease first appearance date.

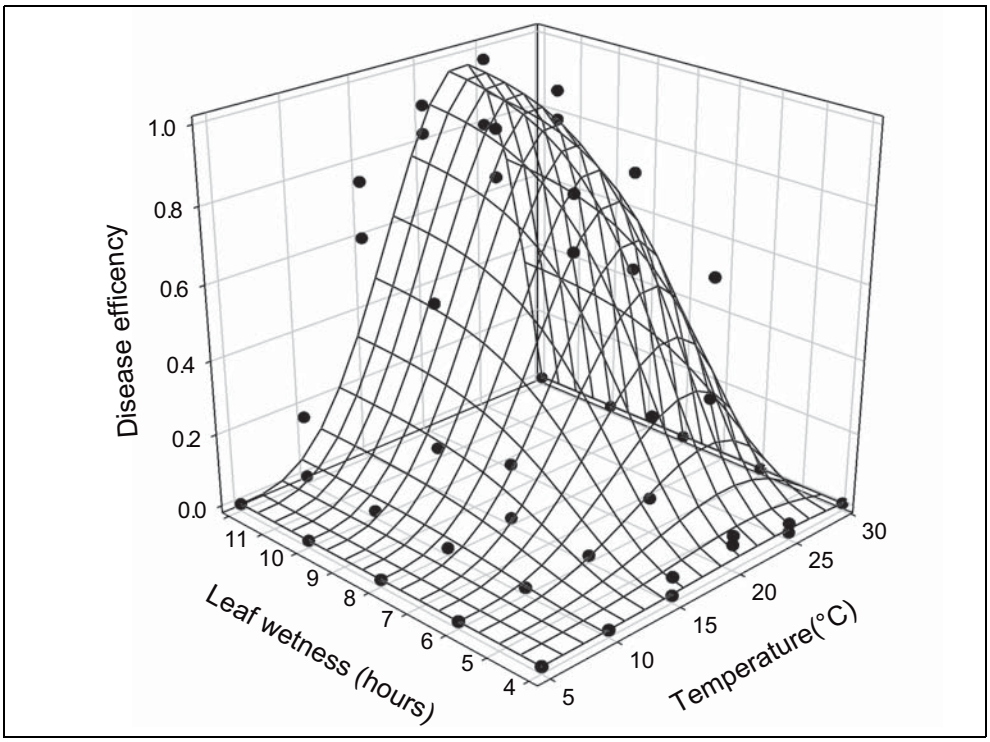

Fig. 1. Modeled Disease efficiency of *C. lupini*, depending on temperature (°C) and leaf wetness duration (hour) for the BBCH stages 21–23 (2–4 leaves)

Tab. 3. Estimated parameter values of the beta-Hau function for the latency rate of *C. lupini*, depending on the temperature (°C)

parameter	value	S.E.	p	r^2
Y_{max}	0,27	0,06	0,1486	
T_{min}	5,00	40,27	0,9214	
T_{opt}	24,37	9,03	0,226	0,53
T_{max}	30,00	0,00	< 0,0001	
n	0,50	2,69	0,8831	

Validation of SIMCOL1 model

The data for model validation have been obtained through field tests carried out from the Governmental Crop protection services of the states Brandenburg, Mecklenburg-Western Pomerania and Saxony-Anhalt, the Saatzucht Steinach, the Julius Kühn-Institut and the ZEPP. For the validation of SIMCOL1 the first appearance on the stem was rated. The validation was made by comparing the date of the first appearance forecasted by the model and the observed first appearance in the field with the following criteria (RACCA et al., 2010):

- correct – when the forecasted appearance date was earlier than the observed appearance date (0–7, 8–14, > 14 days);
- late – when the forecasted appearance date was later than the observed appearance date.

The results of the validation are summarised in Fig. 3.

In 89% of the cases the prognosis of SIMCOL1 was classified as correct, in 11% of the cases too late. In all cases, the prognosis was before the first appearance on the pods.

In Fig. 4 the observed periods of the disease occurrence on the leaves, stems and pods and the forecasted period of SIMCOL1 are shown in a box-plot. The average prognosis date of SIMCOL1 lies between the mean date of occurrence on the leaves and the appearance on the stems. The date of the forecasted first appearance is recommended to use as the date for the first treatment or as a starting date for the field control.

It was further analysed whether differences occur in the date of the first disease occurrence in trials with healthy and infected seed and in treated or untreated seed. However no clear connection could be determined. In the experiments, the first appearance in the untreated and in the treated variant sometimes was on the same day, in other cases there was a difference of up to 17 days or there was no infestation in the treated variant.

As a basis for the development of SIMCOL1 the data were obtained with artificially infected seed. In practice, however, the farmers mostly use certified healthy seed. To take into account a possible infection on the seed, a factor that changes the conditions for the beginning of disease for non-infected seed was introduced in the model. This factor changes the minimum criteria for a disease onset as follows:

- 100% of the days during the latency period must have an EPI > 0.1 or > 0.2;
- at least 71% with EPI > 0.4;
- at least 25% with EPI > 0.6;
- at least 7% of the days with EPI > 0.8.

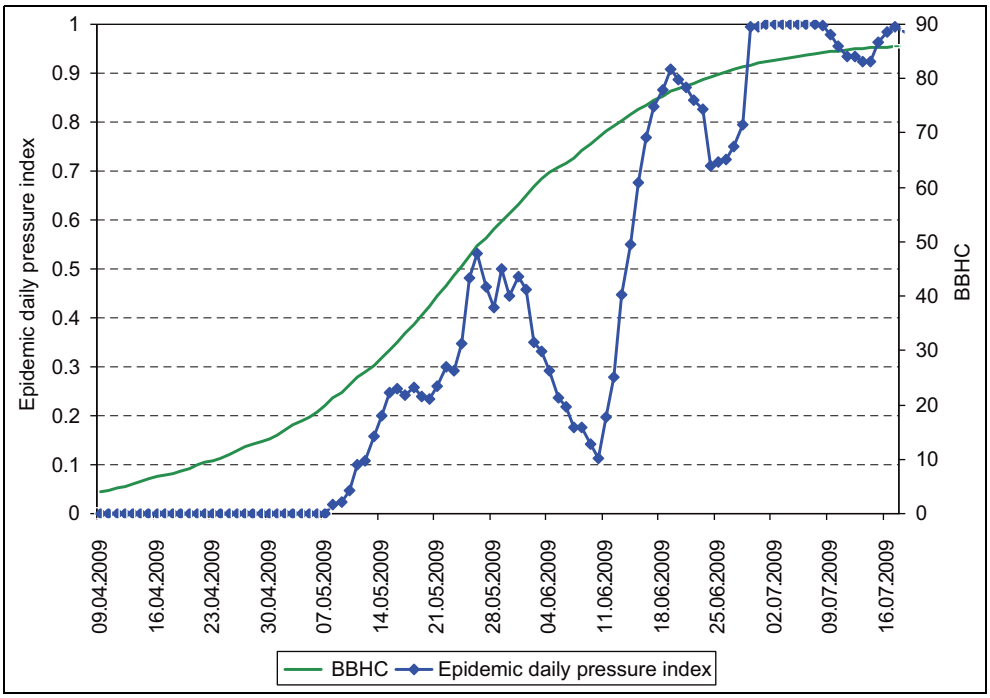

Fig. 2. Epidemic daily pressure index (EPI) of *C. lupini* and simulated BBCH-growth stages (with SIMONTO-Lupin model) for the met. station Rommersheim (Rhineland Palatinate, Germany), for the growing season 2009.

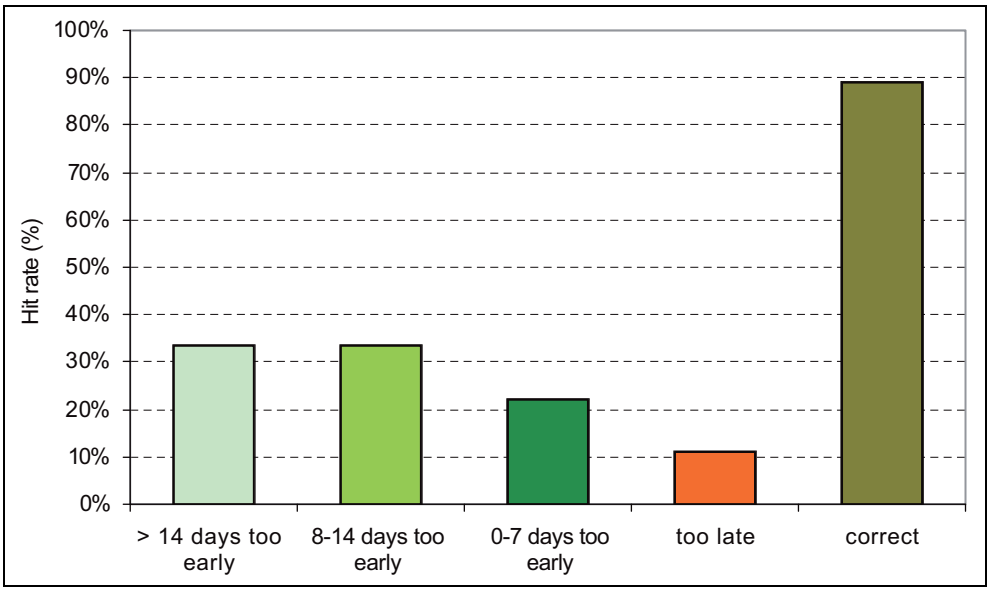

Fig. 3. Comparison of predicted and observed first appearance on the stems, SIMCOL trials in 2009 and 2010 (n = 9)

This factor must still be verified by future validation experiments.

Development of SIMCOL3 model

After the first treatment the model SIMCOL3 predicts, with the help of the calculated EPI, the increase of the disease and, with a fungicide efficacy factor, the dates of the following treatments.

Typically, a fungicide treatment is necessary if the disease (incidence or severity) increases in an exponential phase. *C. lupini* is a very dangerous disease; some small lesions can produce in a short time many spores and, under favourable conditions, the epidemics grow up rapidly. Due to this high aggressiveness and the fact that there is no disease threshold for anthracnose available, infection must be avoided (0-tolerance).

In a further step, disease incidence (DI) data from 66 trials (historical field data) were analysed and divided into two classes of disease increase (class 0, no disease increase, DI = 0; and class 1, positive disease increase, DI > 0). For this data at first the latency period was computed and then the EPI was accumulated during the latency period (EPIsum). The data are presented as a box plot in Fig. 5.

The disease increase (class 1, DI > 0) is in the range of 0.1 to about 5 of the EPIsum. 50% of the data range from 1.8 to approximately 4. Class 0 (no disease increase, DI = 0) has a low EPIsum of 0.1 to 0.8. To establish a limit

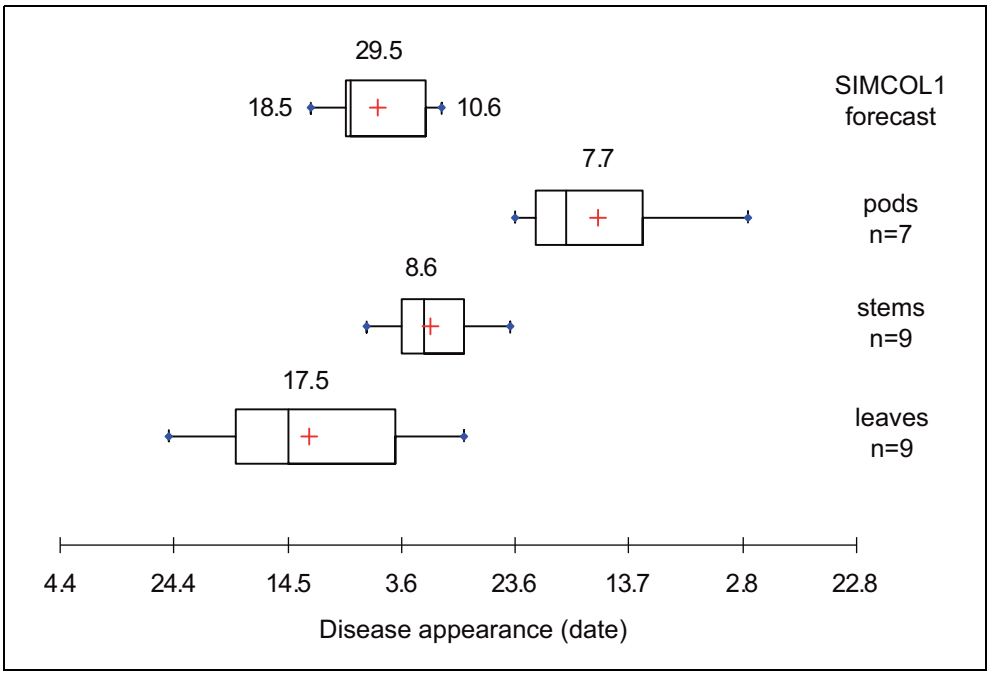

Fig. 4. Comparison of predicted and observed first appearance on leaf, stem and pods. SIMCOL trials in 2009 and 2010 (n = 9)

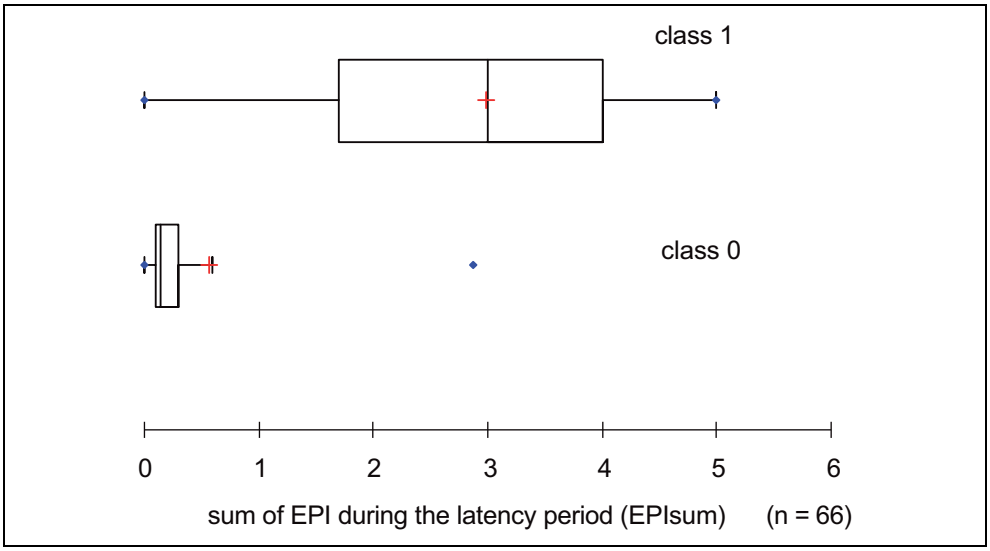

Fig. 5. Classification of the EPIsum in the two classes of disease increase (0 and 1)

of the EPIsum, which discriminates the two classes, the 66 cases were analysed. A good agreement was achieved using a cutoff of EPIsum = 2. With this threshold in 85% of the cases a disease increase was correctly forecasted. In 12% of the cases the disease increase was underestimated (the EPIsum predicts no increase in infection, but an increase in infection was recorded) and in 3% of the cases overestimation (disease increase was forecasted but not recorded) occurred.

This limit of EPIsum = 2 was used as an internal model threshold. This means that if the EPIsum (after a treatment) reaches this threshold, a new treatment recommendation is issued.

Fungicide activity in SIMCOL3

For recommending additional fungicide treatments, it is necessary to evaluate the effectiveness of the previous treatment. Through the integration of a module for the evaluation of fungicide effects, a simulation model can accurately calculate whether and when a new treatment is necessary (JÖRG and RACCA, 2007; RACCA and JÖRG, 2007). A follow-up treatment can be recommended when a control threshold is exceeded.

The fungicide effect is taken into account in the model as a reduction factor of the epidemic, depending on the effectiveness of the fungicide.

The fungicide effect was calculated as follows:

$$[5] \qquad FE = FE_T\lfloor f(T,D_{ppm}) \rfloor \times FE_D[f(sumT)]$$

Where:

FE = fungicide effect (as a reducing factor for the disease efficiency)

FE_T = fungicide efficacy depending on the temperature and the relative dose
FE_D = fungicide efficacy duration depending on the temperature sum
D_{ppm} = relative dose
sumT = sum of the temperature starting from the day of the last treatment (°C)

For the development of the fungicide module in SIMCOL laboratory tests were performed, in which the temperature-dependent fungicidal activity against *C. lupini* was investigated for the active ingredient azoxystrobin (product Amistar) and tebuconazole (product Folicur).

With the data collected by the laboratory tests the FE_T [6] and the FE_D [7] functions were obtained:

$$[6] \qquad FE_T = [((1 - EXP(-(a \times pD))))^b] \times \left[\left(\frac{Tw - T_{min}}{T_{opt} - T_{min}} \right)^{\left(n \times \frac{T_{opt} - T_{min}}{T_{max} - T_{opt}} \right)} \times \left(\frac{T_{max} - Tw}{T_{max} - T_{opt}} \right)^n \right]$$

Where:
FE_T = fungicide efficacy depending on the temperature and the relative dose
T = temperature (°C)
T_{min}, T_{max}, T_{opt} = estimated minimum, maximum and optimum temperature for the fungicide efficacy (°C)
a, b, pD and n = function parameters

$$[7] \qquad FE_D = \frac{1}{1 + EXP(-(a + b \times sumT))}$$

Where:
FE_D = fungicide efficacy duration depending on the temperature sum
sumT = temperature sum from the date of the last treatment (°C)
a, b = shape parameters

The parameters and the functions obtained with [6] and [7] are summarised in Tab. 4, Fig. 6 and Tab. 5, Fig. 7, respectively.

The efficacy of fungicides reduces the disease efficiency (DE) from the day of treatment, depending on the daily mean temperature and the sum of the daily mean temperature from the date of the treatment and is calculated as follows:

$$[8] \qquad DE_R = DE \times [FE_T \times FE_D]$$

Where:
DE_R = reduced disease efficiency
DE = disease efficiency
FE_T = fungicide efficacy depending on the temperature and the relative dose
FE_D = fungicide efficacy duration depending on the temperature sum

With this modified DE_R in a further step, a new EPI can be calculated. If the sum of the EPI reaches the internal model threshold (EPIsum = 2) a new treatment is recommended (Fig. 8).

Concerning the influence of the seed treatment, it could be demonstrated with historical trial data (17 trials with 124 surveys) that with fungicide treatment on seed further disease development was reduced significantly. The statistical analysis of these results using ANOVA (not published) showed that the reduction of the disease development can be described by a factor of about 0.5. Since there are no other data to assess the effectiveness of seed treatment a reduction factor was introduced in the model, which reduces the DE by 0.5.

Deployment of SIMCOL1 and SIMCOL3
The DSS SIMCOL was programmed and included in the software package PASO (prognosis of agricultural pests) (KLEINHENZ and JÖRG, 1998). An example of the simulation results is shown in Fig. 9. The system can be used both for the agricultural practice through the advisory and for the seed production.

Discussion and conclusions

The DSS SIMCOL was developed to manage the pathosystem *C. lupini* – *L. angustifolius*. By using laboratory and literature data the main epidemic processes disease efficiency and latency period of *C. lupini* were modelled.

Since the susceptibility of the lupin plants was related to their growing stage, the simple model SIMONTO-lupin

Tab. 4. Estimated parameter values of the combined Richards- and beta-Hau function for the fungicide effects depending on the temperature (°C) (* sign. p < 0.05)

Active ingredient	Y_{max}	a	b	pD	T_{min}	T_{max}	T_{opt}	n
azoxystrobin	1,074	0,560	1,194	2	10,000	30,000	19,859	0,708
tebuconazole	1,000	1,6166*	0,8153*	2	9,999	30,052	17,347	1,634*
azoxystrobin (0.5) + tebuconazole (0.5)	1,000	0,8594*	0,8774*	2	10,000	30,145	18,188	1,1694*

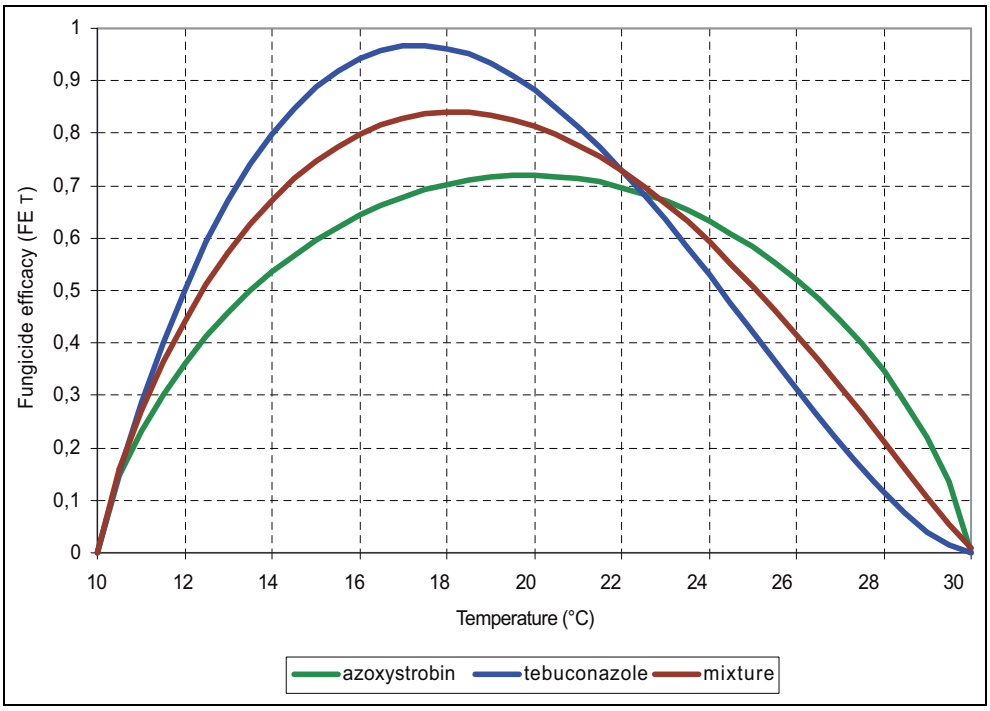

Fig. 6. Simulated fungicide efficacy depending on the temperature (°C) for the active ingredient azoxystrobin, tebuconazole and a fungicide mixture.

Tab. 5. Estimated parameter values of the logistic function for the fungicide efficacy duration depending on the temperature sum (* sign. with p < 0.05) (for the active ingredient mixture the mean values of both fungicides were calculated)

active ingredient	parameter	value	S.E.
azoxystrobin	a	5.512	1.863
	b	−0.015	0.005
tebuconazole	a	3.520	1.186
	b	−0.008	0.003
azoxystrobin (0.5) + tebuconazole (0.5)	a	4.516	
	b	−0.012	

that simulates the appearance of the BBCH growth stages, calculated with a temperature growth rate, was developed and integrated in SIMCOL.

Field data were correlated with an epidemic pressure index in order to obtain the prognosis of the disease occurrence (with model SIMCOL1) and a threshold for the treatment scheduling (SIMCOL3).

The basis for both models SIMCOL1 and 3 is the calculated disease efficiency depending on temperature, leaf wetness and BBCH-growth stages. The estimated regression parameters for the calculation of the DE are described in Tab. 2: the estimated minimum temperature for the development of the disease is 5°C for all tested BBCH-stages. Exceptions are the stages 60–69, in which even at 0°C a development of the disease is possible. This estimated parameter shows no statistical significance ($p > 0.05$). This means that the experimental data at low temperature used for the estimation of the DE are not satisfying and need further improvement. The estimated maximum temperature for development is approximately 30°C for all stages. The optimum temperatures are around 23, 21, 26 and 22°C for BBCH 21–23, 39, 60–69 and 70–87, respectively. The statistical significance of these parameters ($p < 0.05$) shows that the maximum and optimum temperature were estimated correctly.

To get a minimum DE for stages 21–23, 39 and 60–69, a minimum of 4 hours of leaf wetness duration is required. For stages 70–87 only two hours are sufficient. After 7–8 hours of leaf wetness already high values of the DE for the BBCH stages 21–23, 39 and 60–69 are reached. At BBCH 70–87 4 hours of leaf wetness are sufficient to obtain the maximum DE. This confirms the pathogen's aggressiveness and the higher susceptibility of the plants during the pods development stage (70–87).

The aggressiveness of the pathogen can also be confirmed by the very short latency period which is only about 4 days at the optimum temperature of 25°C.

The excellent quality of the calculated DE, the latency period and the EPI (combination of the two former parameters) was confirmed by showing correct prognosis of the disease appearance (89% of correct cases) in SIMCOL1 and the disease increase (85% of correct cases) in SIMCOL3.

Laboratory experiments were carried out to obtain data for the modelling of the fungicide efficacy. Fungicide efficacy functions depending on temperature where calculated for two active ingredients and introduced in the model SIMCOL3 to obtain the possibility to forecast the dates of the subsequent treatments. The results of the laboratory experiments demonstrate an increased efficacy of the a.i. tebuconazole compared to the a.i. azoxystrobin (Fig. 6). The optimum temperature for the higher efficacy was about 19.8 for azoxystrobin and 17.3°C for tebuconazole (Tab. 4).

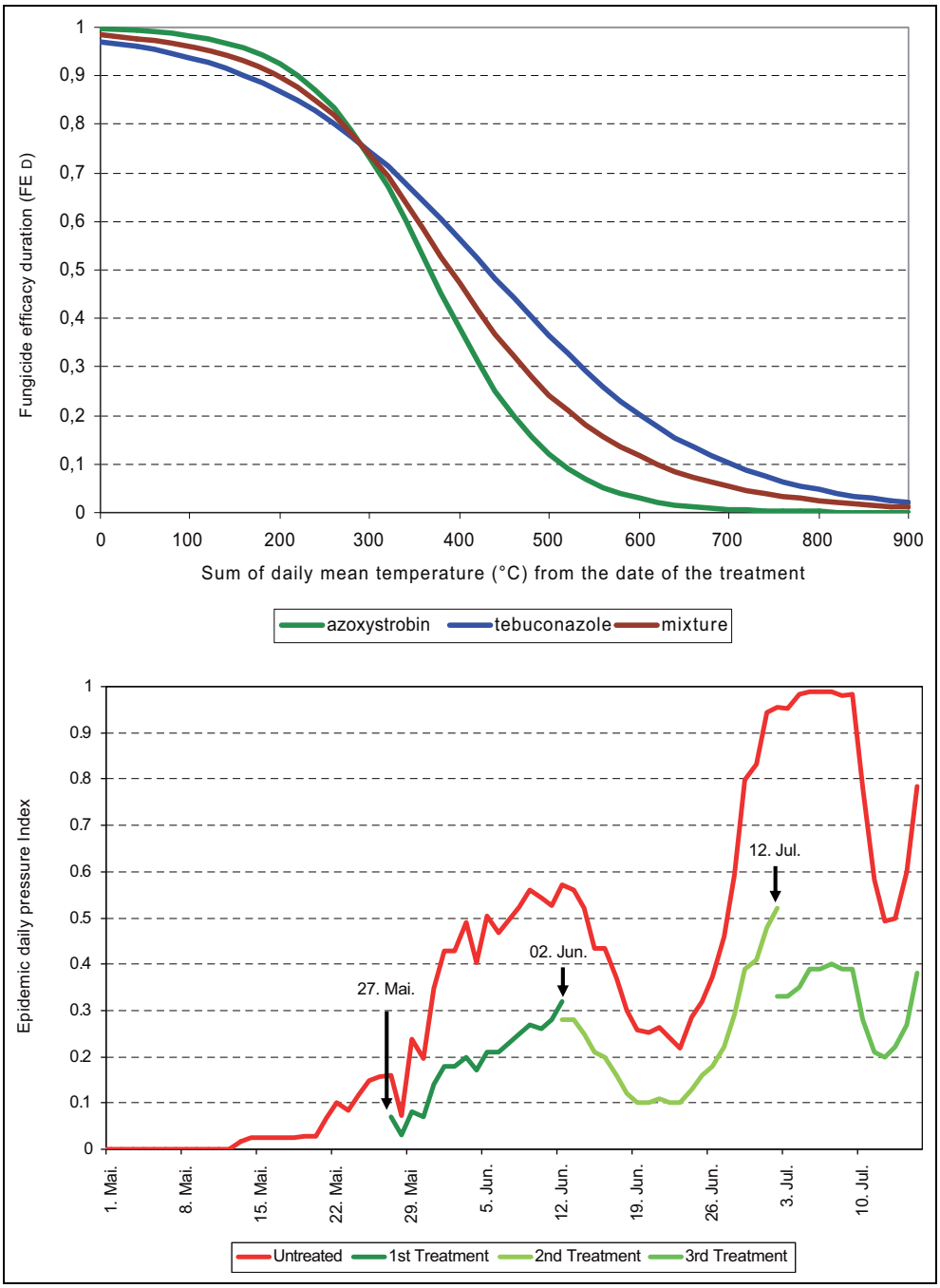

Fig. 7. Simulated fungicide efficacy duration depending on the temperature sum from the date of treatment (for the fungicide mixture the mean values of both fungicides were calculated).

The model also includes a factor which takes into account the influence of the seed treatment (DE reduced mean a 0.5 factor) and the seed infestation on the disease development (changes of the minimum criteria for a disease onset in SIMCOL1). Both factors needed further evaluation.

The validation of SIMONTO-Lupin (Racca and Tschöpe, 2011; Tschöpe and Racca, 2010) and SIMCOL1 with independent data obtained from the Governmental Crop protection services of Brandenburg, Mecklenburg-Western Pomerania and Saxony-Anhalt, the Saatzucht Steinach, the Julius-Kühn-Institut and the ZEPP has delivered good results. A further and appropriate validation of the model SIMCOL3 must still be done. Some parameters like the efficacy of the seed treatment and the influence of the infestation level on the seeds need some further improvement.

The DSS SIMCOL was programmed and included in the software package PASO and can be used to optimise the control strategy against the anthracnose in blue lupin both for the agricultural practice through the advisory and for the seed production. Moreover SIMCOL helps in planning field assessments and fungicide treatments thus saving costs for crop protection products.

Acknowledgements

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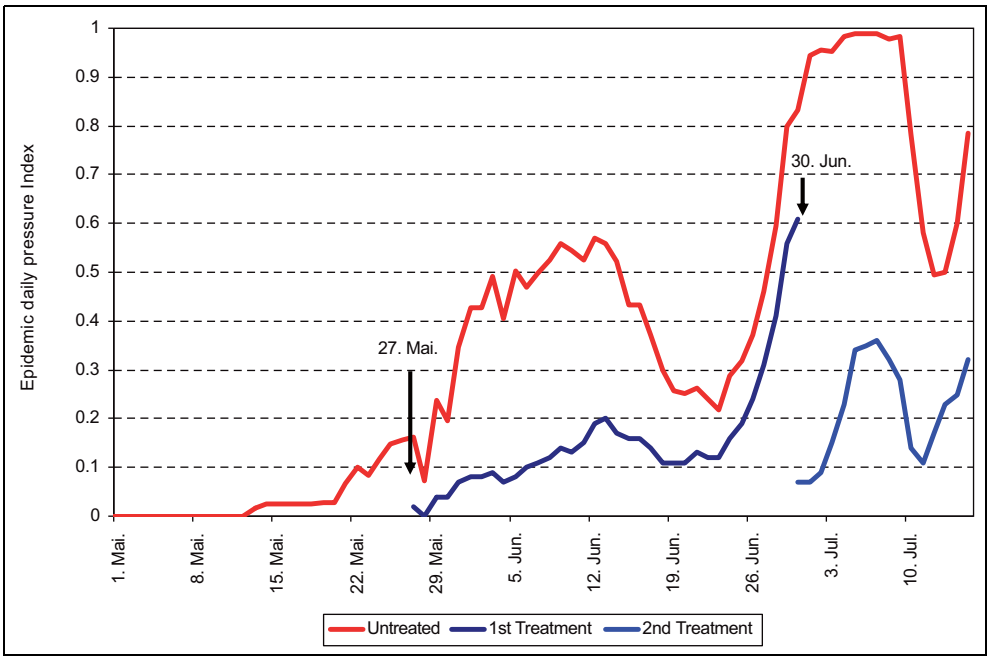

Fig. 8. SIMCOL3 results for met. station Potsdam in 1999, timing of fungicide treatments and the effect of different treatments with the active ingredient azoxystrobin (A) and tebuconazole (B).

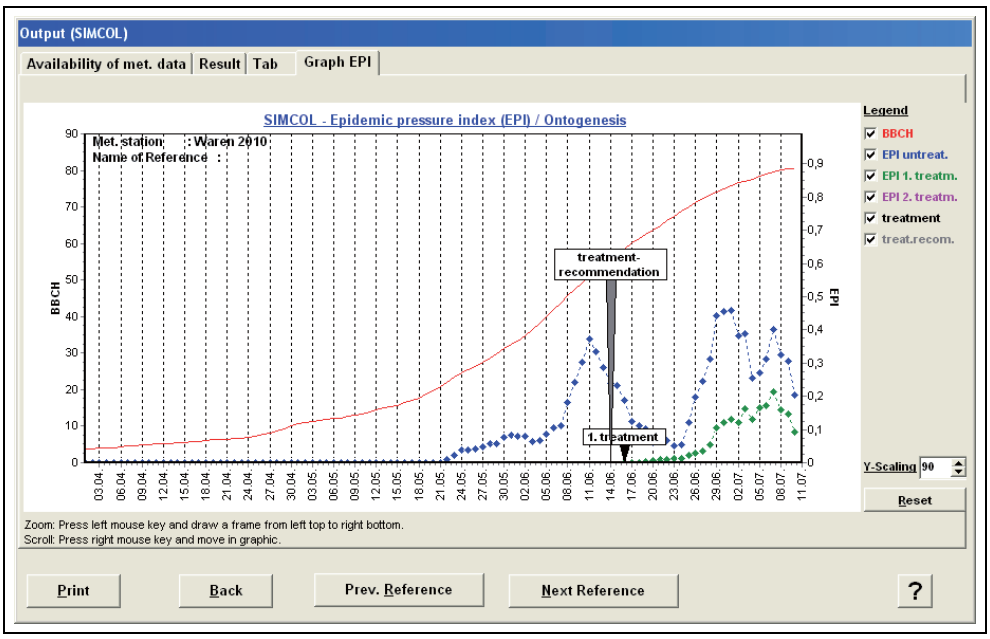

Fig. 9. SIMCOL model results in PASO software.

Claire-Waldoff-Str. 7, 10117 Berlin). Further thanks go to the Governmental Crop protection services of the federal states of Brandenburg, Mecklenburg-Western Pomerania and Saxony-Anhalt, the Saatzucht Steinach and the Julius Kühn-Institut Braunschweig for providing data, conducting the validation tests and for the intensive exchange of expertise in project meetings.

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