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Originalarbeit

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Empirical-deterministic prediction of disease and losses caused by Cercospora leaf spots in sugar beets

Empirisch-deterministische Prognose von Befall und Verlust verursacht durch die Cercospora-Blattfleckenkrankheit in Zuckerrüben

Abstract

Besides negative-prognosis of epidemic onset, epidemic spraying thresholds (ET) and economic damage threshold (DT), loss prediction is a part of the Quaternary IPM (Integrated Pest Management)-concept to control Cercospora leaf spots (CLS). The practical need of loss prediction originates from the fact, that disease levels of ET and DT implicate an interval of 5–10 weeks. Disease severity (DS) of ET for an initial treatment is 0.01, whereas the beet plant may tolerate 5% DS without economic losses. Therefore, in order to assess the necessity of control measures, the model is focused on to predict whether DS will exceed DT at harvest time. The model is empiric, because loss prediction was derived from epidemic and yield data of 105 field trials conducted in Germany and Austria (1993-2000). The model is also deterministic, because the disease incidence at present date and cultivar susceptibility determine the prediction of future disease progress. In every field study, course of DS involved a period of slight followed by a more or less steep increase tending to a maximum of DS. The incidence prediction in submodel (i), therefore, was based on the sigmoidal function "$DS = DS_{max}/(1+\exp(-(CW-a)/b))$", where the calculation of DS is depending on the actual calendar week (CW) and the variables DS_{max}, a and b. These variables are estimated through curve fittings depending on the epidemic onset ($CW_{DIL5\%}$), respectively the calendar week when disease incidence per leaf (DIL) increases to ≥5%. Losses are dependent on the area under disease progress curve (AUDPC). Creation of AUDPC-values is based on the DS-values as calculated by submodel (i). The prediction of losses is performed through disease-loss-relationships (submodel ii, iii). The economic damage threshold is defined as AUDPC=1, equal to a loss of ≈1.5% sugar. Therefore fungicide sprays may be avoided, if the AUDPC remains beneath 1 till scheduled harvest time. All calculations for model development involved two grades of cultivar susceptibility, either highly or low susceptible. Moreover, prediction of yield loss needs indications of expected yield and scheduled harvest time. Proper diagnosis and disease scoring is a precondition for error free functioning of the model, since future progress is estimated by an assessment of the actual incidence situation.

Key words: *Beta vulgaris*, *Cercospora beticola*, Integrated pest management (IPM), disease prediction, epidemic threshold, economic damage threshold, loss prediction

Zusammenfassung

Neben einer Negativ-Prognose des Epidemiebeginns, epidemieorientierten Bekämpfungsschwellen (BK) und einer wirtschaftlichen Schadensschwelle (WS) beinhaltet das Quaternäre IPS (Integriertes Pflanzenschutz)-Konzept zur Kontrolle des Cercospora-Befalls eine Verlustprognose. Die Verlustprognose erhält ihren praktischen Sinn dadurch, dass die epidemischen Stadien von BK und WS ein Intervall von 5–10 Wochen beinhalten. Die Befallsstärke (BS) zum Zeitpunkt von BK beträgt 0,01%, hingegen toleriert die Zuckerrübe 5% BS ohne wirtschaftlichen Schaden. Die Verlustprognose trifft daher Vorhersagen, ob der künftige Befallsverlauf die WS zum Erntezeitpunkt überschreiten wird und insofern, ob Bekämpfungsmaßnahmen benötigt werden. Das Modell ist als empirisch zu charakterisieren, nachdem die Herlei-

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tung der Verlustprognose auf 105 Feldstudien (Deutschland und Österreich) einer Epidemie von *Cercospora beticola* und ihren ertraglichen Konsequenzen beruht. Des Weiteren ist das Modell deterministisch, weil die Krankheitssituation zum gegenwärtigen Zeitpunkt die Prognose der zukünftigen Befallsentwicklung determiniert. In jeglicher Feldstudie implizierte der Epidemieverlauf eine Phase geringer Progression der BS, gefolgt von einem steilen Anstieg mit Tendenz zu einem Maximum des Befalls. Die Prognose des Befallsverlaufes in Submodul (i) basiert daher auf der sigmoiden Funktion „BS = $BS_{max}/(1+\exp(-(CW-a)/b))$“. Demnach hängt die Kalkulation von BS von der Kalenderwoche (CW) und den Variablen BS_{max}, a und b ab. Letztere werden geschätzt mittels mathematischer Funktionen in Abhängigkeit vom Epidemiebeginn ($CW_{BH5\%}$), definiert als jene Kalenderwoche, zu der eine Befallshäufigkeit (BH) der Blätter von ≥5% eintritt. Die Verluste sind hierbei abhängig von der Fläche unter der Befallskurve (AUDPC). Für die Kalkulation der AUDPC-Werte finden die BS-Werte Verwendung, wie mit Submodul (i) geschätzt. Die Prognose von Verlusten an Rüben- und Bereinigtem Zuckerertrag geschieht auf Basis von Befalls-Verlust-Relationen (Submodul ii, iii). Die wirtschaftliche Schadensschwelle ist definiert als AUDPC=1, entsprechend einem Verlust an Bereinigtem Zuckerertrag von ≈1,5%. Folglich sind Fungizidapplikationen entbehrlich, sofern der Befall bis zur Ernte <AUDPC=1 verbleibt. Alle Berechnungen zur Modellentwicklung haben die Sorten-Anfälligkeiten „hoch“ und „gering“ berücksichtigt. Darüber hinaus benötigt die Verlustprognose Angaben über den zu erwartenden Ertrag und den voraussichtlichen Erntetermin. Diagnose und Erhebung des Befalls sind Voraussetzungen für die Anwendung des Modells, da die Einschätzung der zukünftigen Entwicklung auf einer Konkretisierung der gegenwärtigen Krankheitssituation gründet.

Stichwörter: Bekämpfungsschwelle, *Beta vulgaris*, *Cercospora beticola*, Integrierter Pflanzenschutz (IPS), Krankheitsprognose, Schadensschwelle, Verlustprognose

1 Introduction

Cercospora leaf spot is the most important leaf disease in sugar beets worldwide (Bleiholder and Weltzien, 1972; Cooke and Scott, 1993; Hoffmann and Schmutterer, 1999). Severe incidence may be followed by sugar yield losses up to 25–40%. Nevertheless, the occurrence of the disease is variable, for instance depending on site, weather and cropping conditions. Therefore, routine spray regimes are not opportune to optimize timing and frequency of treatments (Cooke and Scott, 1993; Hoffmann and Schmutterer, 1999; Rossi et al., 1988; Rossi and Battilani, 1990; Weis, 1998; Wolf et al., 1998; Wolf et al., 2000).

The IPM-(Integrated pest management) system presented here is aimed to get the most possible benefit of fungicide sprays in both, ecologically by reducing the chemical load on the environment and economically by saving costs and therefore optimizing the profit. Predictions of disease and losses are part of our IPM-system to control Cercospora leaf spots (Wolf et al., 2004; Wolf and Verreet, 2003; Wolf and Verreet, 2005b). This concept we called quaternary, because it consists of four elements, which complement themselves to one system. The need of IPM-tool combination resulted from the fact that single elements were not sufficient in view of keeping in the above mentioned principles of Integrated pest management.

The reasons were: (i) Prediction of disease onset is not accurate enough to decide on timings of fungicide sprays (Wolf and Verreet, 2005a). Only the risk of epidemic onset may be calculated by a so-called negative-prognosis where two periods are determined, one without risk and a following period with increasing risk. During the time of increasing risk the occurrence of first leaf spots has to be observed backed by a proper diagnosis. The latter is oriented to the typical asexual propagules, dark pigmented conidiophores and conidia appearing as black dots in the centre of the spots. (ii) Epidemic action thresholds may pinpoint fungicide sprays to optimize efficiency (Verreet et al., 1996; Wolf et al., 2001). But, on the other hand, epidemic action thresholds are defined as very early stages of the epidemic, and these stages are far away from direct yield loss. (iii) Therefore, in case of action threshold exceeding, an incidence resp. loss prediction is necessary to get insight, whether the economic damage threshold will be exceeded at harvest time (Wolf et al., 1998; Wolf et al., 2000). Only under this precondition fungicide sprays are justified, according to the principles of IPM.

This paper depicts the fourth element of the Quaternary IPM-concept by explaining the steps of developing an empirical-deterministic disease and loss prediction. The development is based on empirical field-data and is deterministic, because the actual disease situation determines the future disease development.

2 Materials and Methods

2.1 Sugar beet cultivation

Empirical data for model development and validation were gathered from field experiments (n=105) conducted in the period from 1993 to 2000 in Germany and Austria. Except the spraying of fungicides, sugar beets were grown according to the local practice, but in every case comprising a three to four years crop rotation, ploughing during the fall and weed control by chemical herbicides. Fertilisation was variable and done according to the advices of the sugar beet companies following the Electric-Ultra-Filtration (EUF)-method. Used Cultivars were classified according to the rating scheme of the Bundessortenamt (Anonymous, 2001) where 1 = lowest susceptibility and 9 = highest susceptibility; susceptibility includes the classes of 3–4 (n=52) for low respectively 5–6 (n=53) for highly susceptible.

2.2 Field experiments design

Field plots consisted of six rows; length was 7 m, row distance 50 cm. The plots were arranged in a completely randomized block design with four replicates per treatment. Fungicides used in the field trials were the commercial products of Cyproconazole (Alto 100 SL 0,8 l/ha, Syngenta), Difenoconazole (Bardos 1,0 l/ha; Syngenta), Difenoconazole&Fenpropidin (Spyrale 1,0 l/ha; Syngenta), Epoxiconazole (Opus 1,0 l/ha; BASF) and Flusilazole&Carbendazim (Harvesan 0,6 l/ha, DuPont). Each trial included at least a fungicide untreated control and, besides different epidemic oriented IPM-applications, a three times treated so-called "healthy control" to determine the disease free respectively site specific yield optimum.

2.3 Disease scoring and yield measurement

Disease severity on single leaves was assessed visually in weekly intervals using the rating scheme of Battilani et al. (1990). Disease observations began along with canopy closure and ended with the first decade of October (40th calendar week). Further detail about disease assessment and calculation of incidence parameters has already been published (Wolf, 2002; Wolf et al., 2004; Wolf and Verreet, 2005a). Yield effect of different treatments was measured by machine-harvest of three rows in the centre of the plot. The bags with sugar beets – filling weight was 80–100 kg / plot – were transported to the laboratory of South Sugar Company (Ochsenfurt) or Kleinwanzlebener Saatzucht AG (Einbeck) for analysis of root weights, sugar content and the non sugar ingredients Potassium, Sodium and αAmino-Nitrogen. For further detail of yield and quality analysis, please see former reports (Wolf et al., 1998; Wolf, 2002).

2.4 Conception of disease and loss prediction model

The future disease course is reproduced through models based on empirical data (Fig. 1). Submodel (i) is determining the future disease course by data input as follows: The calendar week (CW) where the disease incidence per leaf (DIL) respectively the percentage of leaves showing symptoms proceeds to ≥5% and cultivar susceptibility, which is either low or highly. The record of DIL is done by leaf picking (n=100) from the middle of the leaf mass (Wolf et al., 2000; Wolf, 2002). Submodel (ii) and (iii) enable the prediction of losses. Additionally, indication of scheduled harvest time and expected yield is necessary (Fig. 1).

Model development commenced with classification of field studies according to the criteria epidemic onset ($CW_{DIL5\%}$) respectively the calendar week when ≥5% of beet leaves were showing symptoms and cultivar susceptibility which was either low or highly. In a next step, means were calculated for each class and curve fittings were performed using submodel (i) (Fig. 1). After receipt of all curve fitting data, equations (1a, 1b, 1c) for parameter estimation of the variables DS_{max}, a and b were derived from regressions in dependency of $CW_{DIL5\%}$. The variable "a" is corresponding with the inflection point of the sigmoidal curve, respectively the calendar week where the increase of disease is the steepest. The variable "b" is controlling the conductance of the curve fit. Through the regressions, at $CW_{DIL5\%}$ all variables can be calculated and therefore, under use of submodel (i), the future disease course till end of the season. Moreover, this prediction reproduces weekly DS-values, which may be used for calculation of the AUDPC (area under disease progress curve)-value (Fig. 1). The AUDPC is calculated with equation (3) and expresses the entire disease curve comprising the beginning, progression and severity of incidence through one value (Kranz and Holz, 1993). Now, the AUDPC-value serves to indicate the significance of losses by comparing it with the economic damage threshold. Otherwise, disease loss relationships were created after regression of AUDPC-values and corresponding losses of beet yield (LBY, submodel (ii)) respectively recoverable sugar yield (LSY, submodel (iii)). Losses were calculated as the percentage of yield reduction in relation to the dis-

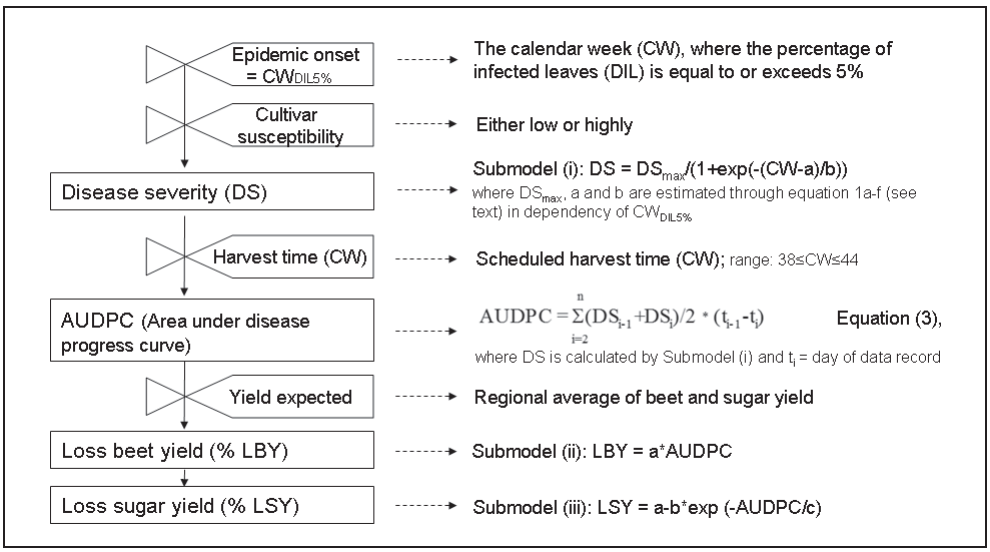

Fig. 1. Relational flow diagram of the disease and loss prediction model with calculated values in boxes and rate regulating influence parameters in the arrow boxes. Arrows with dotted lines indicate data input, used models or functions for parameter estimation.

Tab. 1. Means of disease severity from calendar week 26 to 40 depending on $CW_{DIL5\%}$ [a] and cultivar susceptibility[b]; A: Highly susceptible cultivars, B: Low susceptible cultivars

A	$CW_{DIL5\%}$, highly susceptible cultivars										
	26	27	28	29	30	31	32	33	34	35	36
Calendar week	Disease severity (% infected leaf area)										
26	0.01	0	0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
27	0.10	0.02	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
28	0.23	0.06	0.02	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
29	0.35	0.06	0.08	0.02	0.00	0.00	0.00	0.00	0.00	0.00	0.00
30	0.40	0.15	0.30	0.12	0.03	0.00	0.00	0.00	0.00	0.00	0.00
31	2.57	1.02	1.71	0.37	0.16	0.02	0.00	0.00	0.00	0.00	0.00
32	8.58	3.05	4.20	0.90	0.36	0.09	0.03	0.00	0.00	0.00	0.00
33	17.83	9.74	10.05	3.05	1.49	0.23	0.08	0.02	0.00	0.00	0.00
34	31.36	17.65	19.88	8.09	4.27	0.51	0.23	0.21	0.04	0.00	0.00
35	44.44	33.19	29.77	15.79	11.98	2.23	0.86	0.46	0.13	0.02	0.00
36	49.80	42.01	37.99	25.04	18.61	5.64	2.36	0.69	0.28	0.04	0.01
37	55.80	52.33	44.36	34.23	27.44	9.55	5.63	0.88	0.48	0.08	0.01
38	58.36	59.89	49.08	44.47	34.06	13.96	8.91	1.18	0.62	0.12	0.03
39	62.46	63.41	53.41	48.12	38.54	19.92	12.53	1.90	1.07	0.17	0.04
40	65.87	65.31	58.80	57.87	42.84	25.99	15.62	2.77	1.63	0.19	0.05
n[c]	1	2	5	3	5	14	9	4	7	2	1

B	$CW_{DIL5\%}$, low susceptible cultivars										
	26	27	28	29	30	31	32	33	34	35	36
Calendar week	Disease severity (% infected leaf area)										
26		0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
27		0.03	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
28		0.06	0.02	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
29		0.11	0.05	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.00
30		0.35	0.38	0.02	0.02	0.00	0.00	0.00	0.00	0.00	0.00
31		1.43	1.12	0.20	0.05	0.02	0.00	0.00	0.00	0.00	0.00
32		3.61	2.51	0.58	0.13	0.05	0.02	0.00	0.00	0.00	0.00
33		6.87	6.21	1.32	0.84	0.12	0.04	0.02	0.00	0.00	0.00
34		19.00	12.35	3.53	1.73	0.33	0.12	0.06	0.03	0.00	0.00
35		31.28	24.50	7.04	2.20	0.88	0.29	0.14	0.08	0.02	0.00
36		42.91	33.81	13.35	3.90	1.91	0.66	0.30	0.18	0.06	0.01
37		49.76	43.44	18.85	7.02	3.81	1.59	0.46	0.30	0.13	0.04
38		55.94	52.17	25.23	10.28	7.38	3.02	0.67	0.45	0.22	0.03
39		58.36	56.86	31.22	15.26	10.81	5.94	1.28	0.84	0.40	0.04
40		62.73		36.06		15.00	9.00	1.50	1.50	0,84	
n[c]		1	3	1	3	9	5	10	9	9	2

[a] $CW_{DIL5\%}$ = the calendar week when a leaf infection frequency of 5% is reached respectively a disease severity of 0.01% is exceeded.
[b] Classification of cultivar susceptibility, see text.
[c] Number of field studies within the particular category of $CW_{DIL5\%}$.

ease free "healthy control" as described above. For calculation of total loss amounts and economical loss assessments, indications of expected beet yield and sugar content on a regional basis is required (Fig. 1).

2.5 Data analysis

Electronic data processing was performed by using specific excel-data sheets. Prepared data selections were further processed by curve fittings using the program "Slide-

Write Plus" (Advanced Graphics Software, Inc. Encinitas, CA). The probability level was 95%. The criterion for selection of mathematical models was the determination coefficient of regression (r^2, α=0.05).

3 Results

3.1 Empirical disease progress

The prediction of disease progress is based on the present incidence situation in order to forecast the future progress until harvest. The period of forecast spans 5–12 weeks, in practice of sugar beet growing, from the time when an acting threshold is exceeded until harvest. Therefore, effects during this period, mainly originating from the weather, cannot be considered. The empirical investigation of 105 field studies revealed a substantial variance of disease progress curves (Tab. 1-A,B). Besides the weather, the epidemic is mainly affected through the cultivar susceptibility and the time of epidemic onset. The latter here is defined as $CW_{DIL5\%}$, respectively the week when a leaf infection frequency of 5% or a disease severity of 0.01% was exceeded. In order to elaborate the effects of the above mentioned criteria, each field study was assigned accordingly to $CW_{DIL5\%}$ and category of cultivar susceptibility. Subsequently, means of disease severity were calculated (Tab. 1).

The earlier the epidemic onset the more likely a severe disease progression is following. If $CW_{DIL5\%}$ <28, disease development may induce 65% loss of the leaf mass, but the disease curves slow down as later as the epidemic commences (Tab. 1-A). If the epidemic onset occurs after calendar week 32, the final DS remains below 5%. Generally, low susceptible cultivars reduce disease severity, but mostly the epidemic onset is delayed significantly, which is evident by a higher frequency of epidemic onset in late season (Tab. 1-B).

Now, the target of disease prediction is to reproduce the disease behaviour as empirical found.

3.2 Prediction of future disease course

In a first step, the development of prediction is performed through curve fittings according to submodel (i). The curve fits were applied to the mean values of disease severity as presented in Tab. 1. The procedure in detail is shown by two depicted examples (Fig. 2). For instance, if the epidemic onset occurred during the 27th calendar week and cultivar susceptibility is highly, a severe disease progress is following (Fig. 2-A). However, the disease course is rather moderate when the epidemic stage of DIL 5% is indicated during the 30th calendar week and cultivar susceptibility is low (Fig. 2-B). For both examples, curve fit statistics show coefficients of determination >0.99 (Tab. 2).

The procedure as shown by the above examples (Fig. 2) was applied to each class of cultivar susceptibility x $CW_{DIL5\%}$ (Tab. 1). Curve fit statistics of all assigned classes were listed in Tab. 2, in particular the values of the equation variables DS_{max}, a and b; in addition, the coefficients of determination, which are ≥0.99 in the period $CW_{DIL5\%}$ <32 respectively <30 in case of low susceptible cultivars. Hence, the aptitude of the selected submodel (i) concerning the reproduction of the disease course becomes evident. Moreover, it became apparent, that there were relationships between $CW_{DIL5\%}$ and the equation variables of submodel (i). DS_{max} is decreasing with delay of $CW_{DIL5\%}$. The variable "a" is corresponding with the inflection point of the sigmoidal curve, respectively the calendar week where the increase of disease is the steepest. As logical, the value of variable "a" is increasing as later as the epidemic is initiated. The variable "b" is controlling the conductance of the curve fit. The b-values are also increasing along the epidemic onset is delayed. Thus, the curves are tending to get more flat (Tab. 2).

The overall conclusion is, in order to summarize Tab. 2 that the variables of submodel (i) depend on $CW_{DIL5\%}$ and cultivar susceptibility. This fact, therefore, offers the possibility to estimate the variables of submodel (i) through regressions. The relationship of $CW_{DIL5\%}$ and DS_{max} is negative sigmoidal (Fig. 3-A). Hence, the maximum DS is determined by 68%; the curves are slowing down along the epidemic onset is delayed and are tending asymptotic towards 0. Cultivar susceptibility is of substantial impact. Differences between highly and low susceptible cultivars account 10-15% DS, in particular during periods where the curves are the steepest. Estimation of parameters is quite accurate as coefficients of determination =0.99 (Tab. 3).

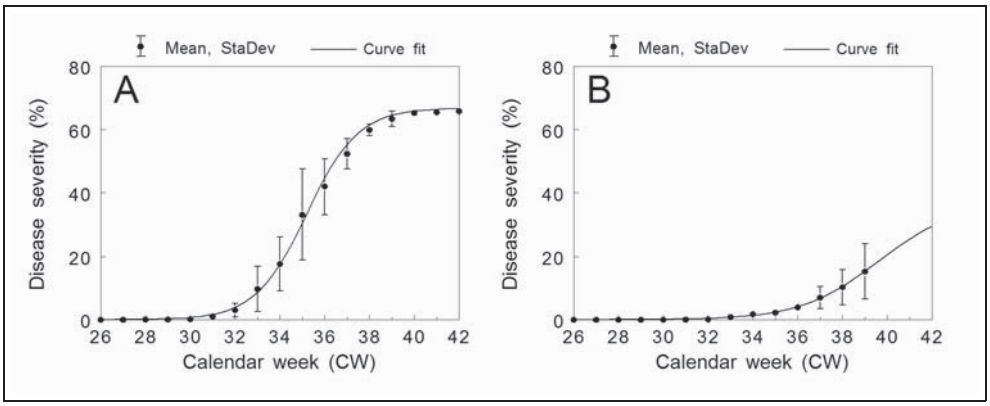

Fig. 2. Two examples of field data assignment and calculation of DS-averages are shown; the criterion of $DIL_{5\%}$ was exceeded during the 27th (Fig. 1-A, n=2 field studies, $CW_{DIL5\%}$=27, highly susceptible cultivar) respectively the 30th calendar week (Fig. 1-B, n=3 field studies, $CW_{DIL5\%}$=30, low susceptible cultivar). Dots are means calculated from empirical field data (Tab. 1), the curves are fitted according to submodel (i) DS = DSmax/(1+exp(-(CW-a)/b)); error bars = standard deviation.

Tab. 2. Curve fit statistics after application of submodel (i)[a] to the means of disease severity recorded in the field (Tab. 1-A, 1-B). Field studies were assigned according to $CW_{DIL5\%}$[b] and cultivar susceptibility[c]

$CW_{DIL5\%}$ [b]	highly susceptible[c]				low susceptible[c]			
	DS_{max}	a	b	r^2	DS_{max}	a	b	r^2
26	66.6	34.2	1.10	0.99	-	-	-	-
27	66.9	35.2	1.15	1.00	65.0	35.5	1.30	1.00
28	64.0	35.5	1.35	0.99	61.0	35.9	1.35	1.00
29	60.0	36.6	1.40	1.00	45.0	37.5	1.50	1.00
30	51.0	37.2	1.47	1.00	37.0	39.6	1.74	1.00
31	40.0	38.9	1.50	1.00	29.0	40.5	1.85	0.95
32	28.0	39.4	1.55	0.99	20.0	41.0	1.90	0.94
33	9.0	41.0	1.80	0.97	7.0	42.5	2.00	0.98
34	5.0	41.6	1.80	0.98	4.5	43.0	2.30	0.94
35	0.7	42.0	2.00	0.98	3.0	43.9	2.40	0.92
36	0.2	43.0	2.30	0.88	0.2	44.0	3.00	0.62

[a] Submodel (i): $DS = DS_{max}/(1+\exp(-(CW-a)/b))$, where DS_{max}, a and b are estimated through equation 1a-f in dependency of $CW_{DIL5\%}$.
[b] $CW_{DIL5\%}$ = the calendar week when a leaf infection frequency of 5% is reached respectively a disease severity of 0.01% is exceeded.
[c] Classification of cultivar susceptibility, see text.

Tab. 3. Curve fit statistics after regression of $CW_{DIL5\%}$[a] to the fitted variables of submodel (i) as indicated in table 2; equations (Eq. 1a-f) enable estimation of variables in submodel (i) in dependency of $CW_{DIL5\%}$ and cultivar susceptibility[b]

Estimation of variable	highly susceptible		low susceptible	
	Equation	r^2	Equation	r^2
DS_{max}	Equation 1a: $68/(1+\exp(-(CW_{DIL5\%}-31.5)/-1.1))$	0.99	Equation 1d: $67/(1+\exp(-(CW_{DIL5\%}-30.5)/-1.2))$	0.99
a	Equation 1b: $10.277+0.914* CW_{DIL5\%}$	0.99	Equation 1e: $7.738+1.035* CW_{DIL5\%}$	0.97
b	Equation 1c: $4.25815-0.28231* CW_{DIL5\%} +0.00626* CW_{DIL5\%}^2$	0.96	Equation 1f: $7.25778-0.51303* CW_{DIL5\%} +0.01083* CW_{DIL5\%}^2$	0.96

[a] $CW_{DIL5\%}$ = the calendar week when a leaf infection frequency of 5% is reached respectively a disease severity of 0.01% is exceeded.
[b] Classification of cultivar susceptibility, see text.

If the "a"-values are regressed to $CW_{DIL5\%}$ the relationship is linear (Fig. 3-B, Tab. 3). Along the epidemic onset is delayed, the value of "a" is increasing. Differences between highly and low susceptible cultivars are slight in case of early epidemic onset and are increasing to 1 CW when $CW_{DIL5\%}$ is occurring in late season.

The relationship between $CW_{DIL5\%}$ and the variable "b" is the best to be reproduced through a polynomial function (Fig. 3-C, Tab. 3). The coefficient of determination is 0.96. There is a tendency of the values increasing slight over proportional when the epidemic onset is delayed. As a consequence, the calculated DS-curves are becoming more flat in case of late disease occurrence, according to the empirical records of disease development. Congruently to the behaviour of the "a"-variable, the differences between highly and low susceptible cultivars are increasing when the epidemic is delayed. This means that cultivar resistance is more effective under slight-moderate disease pressure.

3.3 Loss prediction through submodel (ii) and (iii)

The target here is to predict the losses of beet mass and recoverable sugar yield. The calculation of losses is based on the disease progress as predicted by submodel (i). In order to relate the disease progress to losses caused by *Cercospora beticola*, the AUDPC-value was introduced. Subsequently, for each field study, the AUDPC-value is correlated to the yield reduction (Fig. 4-A,B). The reduction here is calculated as the percentage of losses based on the relation of diseased and disease free sugar beet

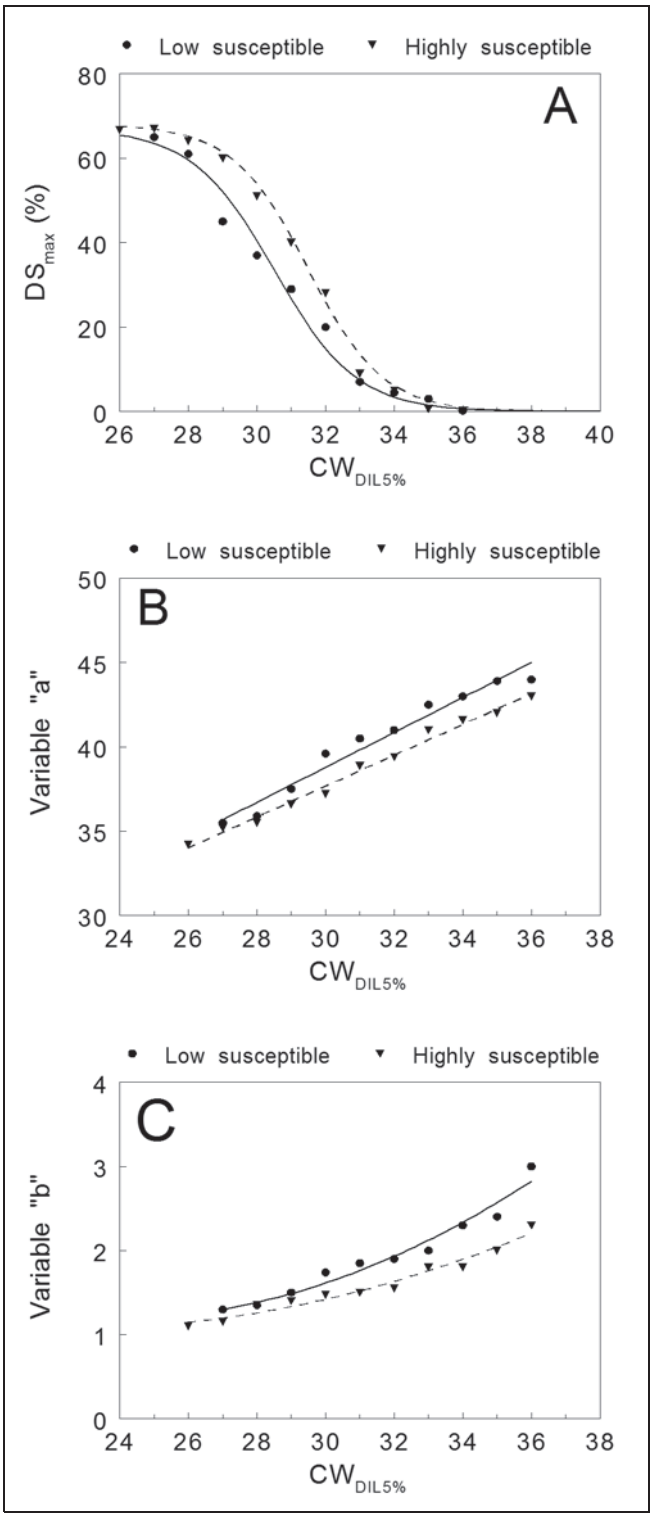

Fig. 3. Estimation of variables in dependency of $CW_{DIL5\%}$[a] and cultivar susceptibility for prediction of DS through submodel (i). A: DS_{max}, determining the maximum of the DS-curve; B: Variable "a", corresponding with the inflection point of the curve; C: Variable "b", controlling the conductance of the DS-curve. [a] $CW_{DIL5\%}$ = the calendar week when a leaf infection frequency of 5% is reached respectively a disease severity of 0.01% is exceeded.

plots, in order to eliminate the effects of different years and sites. Maximum losses of 20-25% beet respectively 30-35% sugar yield occurred at AUDPC 30-35. If cultivars are affected by equal disease level, there is no indication of different yield loss respond. For prediction of beet yield loss, a linear regression was used according to submodel (ii) (Fig. 4-A). Thus, AUDPC 1 is equal to a beet yield loss of 0.75%. Prediction of sugar yield loss is based on an exponential function according to submodel (iii) (Fig. 4-B). Exponential function instead of linear was chosen due to a higher trend of losses slowing down at higher AUDPC-values. Losses of sugar are ≈1.5% if AUDPC=1. Hence, this value is established as a damage threshold, where the losses equalize the costs of disease control through fungicide application. Nevertheless, a relatively wide scattering of the single values could be seen which is mainly caused by different environmental conditions depending mainly on the factors of year, site, weather, and fungicide.

The goodness of loss prediction, on one hand, may be assessed by the relationship of epidemic onset ($CW_{DIL5\%}$) and AUDPC (Fig. 5). The crucial criterion here is the economic damage threshold of AUDPC 1 and, therefore, whether the time of threshold exceeding is predicted with high accuracy. The AUDPC-values marked as dots in Fig. 5 were calculated from DS-values recorded in the field till CW 40. Maximum AUDPC-values in the range of 20-30 result from early epidemic onset in CW 26-28 according to the first half of July; the values are declining rapidly along the epidemic onset is delayed. They tend asymptotic to 0 when $CW_{DIL5\%} \geq 32$. If the epidemic onset ≥CW 33 the AUDPC is <1 in all cases. This relationship is confirmed by the predicted values as indicated by the curves (Fig. 5), even though the regression is affected by a broad scattering of the single values, in particular at early epidemic onset. In general, low cultivar susceptibility is affecting a one week delay of disease development (Fig. 5, see dotted line).

On the other hand, in order to check the validity of loss prediction, the loss of recoverable sugar yield may be used as dependent variable (Fig. 6). The relationship is similar to Fig. 5 with high sugar losses of 25–35% occurring in case of early epidemic onset. Sugar losses are slowing down during the period of $CW_{DIL5\%}$ =31-34, congruently as the AUDPC-values have done, and approach to 0 beginning with $CW_{DIL5\%}$ 33 respectively 32 if cultivar susceptibility is low. Accordingly, losses are predicted to drop below 1% during this period. Also here we find a wide variation when considering single values. But crucial is here, in view of prediction, to distinguish periods of risk from such without any risk of losses.

4 Discussion

The loss prediction is embedded in the Quaternary IPM-concept for the control of Cercospora leaf spots (Wolf et al., 2004; Wolf and Verreet, 2003; Wolf and Verreet, 2005b). It is not only a scientific model but rather adapted to match the practical purpose of confining fungicide use to economic risk situations. In view of control measures, during a current growing season, fungi-

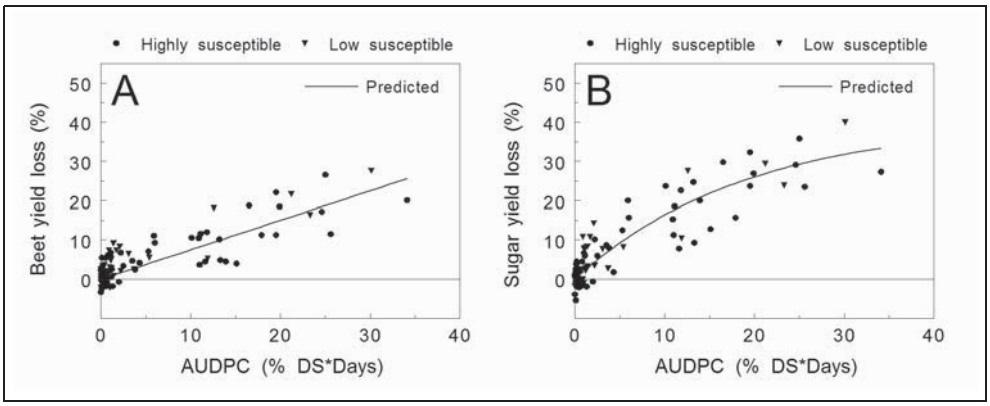

Fig. 4. Disease loss relationship for prediction of beet yield loss[a] (Fig. 4-A) and sugar yield loss[b] (Fig. 4-B) depending on AUDPC[c]. Dots represent the results of n=105 field studies. [a] Loss prediction according to submodel (ii): LBY = 0.75*AUDPC, r^2 = 0.73. [b] Loss prediction according to submodel (iii): LSY = 40-40*exp (-AUDPC/19), r^2 = 0.83. [c] Calculation of AUDPC according to equation (3), see fig. 1.

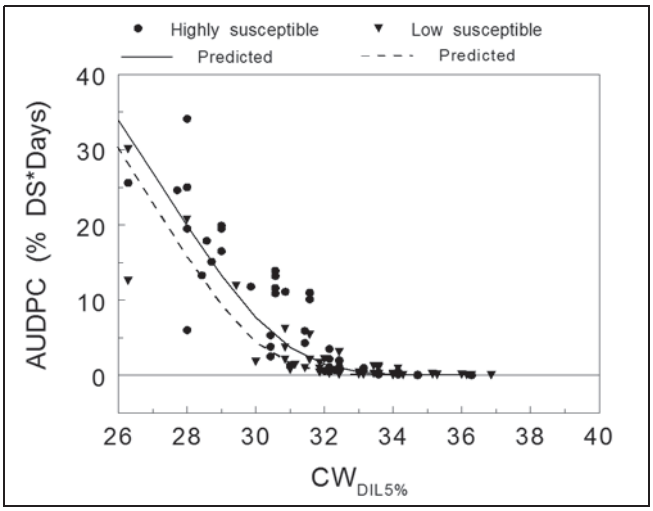

Fig. 5. Relationship of epidemic onset=$CW_{DIL5\%}$[a], and AUDPC[b] at calendar week 40 (n=105 fieldstudies), Prediction of AUDPC is indicated through curves, full line representing highly susceptible respectively dotted line low susceptible cultivars. [a] $CW_{DIL5\%}$ = the calendar week when a leaf infection frequency of 5% is reached respectively a disease severity of 0.01% is exceeded. [b] Calculation of AUDPC according to equation (3), see fig 1.

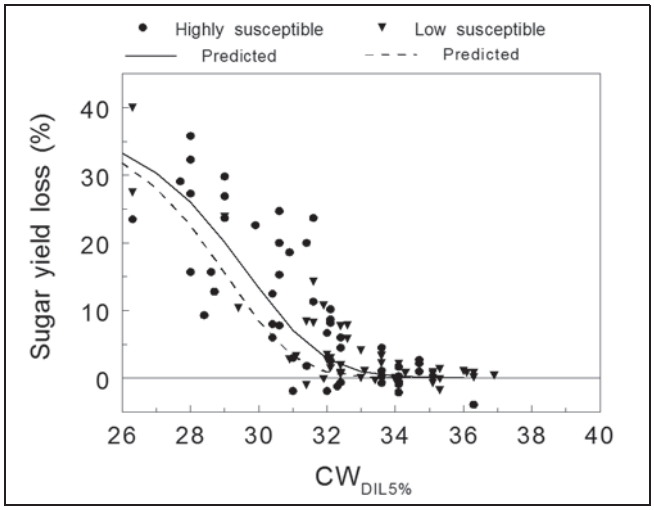

Fig. 6. Relationship of epidemic onset ($CW_{DIL5\%}$[a]), and sugar yield loss[b], harvest time = CW 40-41. Prediction of sugar loss is indicated through curves, full line representing highly susceptible respectively dotted line low susceptible cultivars. [a] $CW_{DIL5\%}$ = the calendar week when a leaf infection frequency of 5% is reached respectively a disease severity of 0.01% is exceeded. [b] Sugar yield loss calculated by submodel (iii), see text.

cide sprays are the only mean to take action against the disease. The strategy here is to pinpoint fungicide sprays most precisely through epidemic oriented action thresholds (ET) in order to get optimum disease control and economic benefit. The problem here is, that the action threshold for an initial treatment is corresponding with a very early stage of the epidemic: 5% of the leaves are showing symptoms (DIL) respectively disease severity (DS) = 0.01% infected leaf area (Wolf et al., 2001; Wolf and Verreet, 2002). On the other hand, definition of the economic damage threshold (DT) is 5% DS and is exceeded 5–10 weeks after ET (Wolf et al., 1998; Wolf et al., 2000; Wolf and Verreet, 2002). Therefore, in order to assess the necessity of a fungicide spray, in case of ET-exceeding information is required whether the future disease progress will lead to a DT-exceeding before harvest time. These practical aspects were the background of development and in this context the sense of the quaternary IPM-concept becomes obvious. The four elements complement each other and only the linking gives insight into the timing and necessity of a fungicide spray. As a consequence of the above mentioned reasons, loss prediction must be empirical because it has to perform long time forecasts. From a practical point of view, multivariate simulations, which incorporate in particular the weather and other influence factors, are not suitable to match the advised object. The reason is, logically, because they work step- or daywise so far as weather events and/or disease courses are known (Kelber, 1977; Rossi et al., 1994). Thus, for long term forecasts, just the experience of past years may be used for risk assessment at the time of epidemic onset. In conclusion, the experience is based on empirical field data of former years, which are transferred through regressions and adapted models to a current growing season. Therefore we called the model empirical. But it is deterministic, too. Deterministic models use variables, parameters, constants and mathematical relationships to obtain a single outcome (Zadoks, 1971). In our model, the single outcome is a disease severity curve enabling the calculation of the "area under disease progress curve" (AUDPC) which, in turn, is correlated to yield losses, either of the beet mass or sugar. For

use in practise, the model just needs data input of date, DIL and cultivar susceptibility. Or in simple words, the present incidence situation determines the prediction of future disease course. Hence, this model conception requires the user, sugar beet growers or advisory services, to perform weekly records about the incidence situation, at least when a risk of disease occurrence is issued by the negative-prognosis (Wolf and Verreet, 2005a). Also skills in diagnosing the sugar beet diseases are necessary, because, definitely the model doesn't work in case of false disease records.

The principles of our loss prediction model may be applied also for some other host parasite systems. Precondition is a data set spanning several years to record the disease variation depending on the climate and growing conditions. These empirical experiences can only be gained by field studies, which, in turn, can only reflect the situation of a specific environment. From this point of view, the results and therefore the specific definitions of prediction cannot be easily transferred to other growing regions, in particular, when the basic conditions of sugar beet cropping are different. And, moreover, there are an appropriate number of field studies necessary to reproduce the variation of disease. We gathered a dataset comprising more than 120 field studies – a big effort, but sometimes not enough to cover the range of disease variability sufficient. Thus, due to some lack of data, extrapolations had to be used, for instance concerning the effect of differing harvest times where only a few data were available. Some field studies were not appropriate due to high infection rates of other diseases, especially powdery mildew. That's another impediment that diseases may interact what's not wishful, and the losses must be able to relate them exceptionally to the main object. Therefore, suitable field studies must be selected following definite criteria, for example the epidemic of powdery mildew remaining under the DT of AUDPC=2 (Wolf, 2002).

In view of loss prediction, model development commenced with classification of field studies, depending on the time of epidemic onset respectively calendar week when the acting threshold of DIL 5% was exceeded and cultivar susceptibility. These factors substantially had impact on the severity of the epidemic (Wolf et al., 1998; Wolf et al., 2000; Wolf and Verreet, 1997) and are the only factors which determine the outcome of the disease course prediction. Thus, actual incidence situation is incorporated which, on the other hand, is the consequence of all past influences. Next to the actual incidence situation, future influences on disease progress consist preferably on cultivar susceptibility and weather. But weather forecasts are not reliable over long periods and, therefore, must be neglected. It can only be regressed to an annual average of disease progress which indirectly involves the typical climate of the growing region on an average base. Next step was the calculation of DS-means according to the classifications and curve fittings by application of a sigmoidal model. Determination coefficients mostly >0.95 confirmed the high aptitude of the chosen model. But more important was the finding of correlations between the model-variables and the classification criteria. So, besides prediction of disease severity curves through curve fittings, submodel (i) enabled to estimate the model variables through regressions. This could be seen as a big advantage because it makes the prediction much easier to handle.

Loss prediction based on the above mentioned principles has not been reported before. Our intention was directed to fill the gap in the development of the IPM sugar beet model, namely to get information about the likelihood whether the future disease course will exceed DT. A multivariate model for the estimation of yield losses in sugar beet was presented by Kelber (Kelber, 1977). Despite estimated losses did account the observed losses by 89%, the model cannot give indications about the yield risk during a current growing season. The estimation is retrospective because data of each quarter of the disease course are required. Therefore, important influence variables are only known at the end of the growing season. Rossi et al. (Rossi et al., 1994) presented the model CERCODEP. This model simulates the progress of disease severity over the course of a growing season with a time step of one day. By means of system analysis, the model considers the relationships between disease stages as infection, incubation and sporulation, etc., weather factors and host characteristics. Despite the model is reported to simulate the disease course with high accuracy, the authors concede that it's rather scientific and less practical important (Racca and Jörg, 2003).

The vision of our model conception, on the contrary, was to meet the practical aspect of risk managing during a current growing season in order to back decisions on plant protection measures.

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