Evaluation of treatment strategies of the late blight *Phytophthora infestans* in Nepal by population dynamics modelling

H. Apel a,∗, M.S. Paudyal b, O. Richter a

a Institute of Geoecology, Technical University of Braunschweig, Langer Kamp 19c, 38104 Braunschweig, Germany
b Plant Protection Division, HMG Department of Agriculture, Haritar Bhawan, Lalitpur, Nepal

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Abstract

The late blight disease caused by the fungus *Phytophthora infestans* is a serious pest of solanaceous crops capable of destroying a complete harvest. Therefore it is heavily treated with fungicides, especially in areas where the knowledge among farmers about application and impacts of fungicides is generally low, as it is the case in Nepal. In order to derive efficient treatment strategies with a minimum of fungicide use, a model describing the population dynamics of the disease on potato was established. The model is based on a universal epidemiological theory with further specification for *P. infestans*. The parameter estimation for the model was accomplished with field data of an uncontrolled epidemic in the Mid-Hills of Nepal. Using this model, different treatment strategies were evaluated by introducing fungicide treatment effects of different fungicide types into the model. Based on both field trials and population dynamics simulation recommendations for an optimised conventional treatment strategy were elaborated. Additionally the modelling tool PhytMod was developed, which simulates population dynamics and treatments and can be used to evaluate treatment strategies for other regions or even for general diseases without the *P. infestans* specification.

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Keywords: *Phytophthora infestans*; Late blight; Population dynamics; Disease epidemiology; Modelling; Treatment strategies; Pest management; Fungicides; Nepal; PhytMod

Software availability

Name of software: PhytMod
Developer: Heiko Apel, Institute of Geoecology, TU Braunschweig, Langer Kamp 19c, 38106 Braunschweig, Germany
Purpose: PhytMod is a simulation tool for disease epidemics. It has a module for the simulation of general unspecified epidemics and a module for the specific simulation of *Phytophthora infestans* epidemics. The *P. infestans* module also contains options for the study of fungicide treatment strategies of the disease.
Comment: The copyright of PhytMod is held by Heiko Apel. PhytMod is freely available to all researchers for non-commercial use. The source code may be available upon request. The model is written in Java and runs on all platforms with a Java Runtime Environment (JRE) 1.3 or higher installed, which is freely available at [http://java.sun.com/j2se](http://java.sun.com/j2se). It works completely from a graphical user interface, has import and export options and textual as well as graphical data displays. All results of the article may be reproduced with PhytMod, except the parameter estimation. A help on how to use PhytMod is included. The program is available for download at [http://www.tu-bs.de/institute/igg/pub/PhytMod](http://www.tu-bs.de/institute/igg/pub/PhytMod)
Contact: h.apel@tu-bs.de

1. Introduction

The late blight, *Phytophthora infestans*, regularly causes serious crop losses in solanaceous crops worldwide. This is especially important in areas, where potatoes are the main staple crop or a prominent income...
source for the rural population. Due to the severity of the disease preventative chemical pest management measures are almost always the first option for the control of the disease, often resulting in over- and misuse of fungicides. This is especially true in the Mid-Hills of Nepal, where potatoes became a major source of income in the last decade. Along with the only recently developed market-oriented production of potatoes the regular use of fungicides was introduced in the agricultural system, often without proper training of farmers and pesticide retailers (Dahal, 1995; Kandel and Mainali, 1993; Kansakar et al., 2001b; Lowe Baker and Gyawali, 1994; Pujara and Khanal, 2001).

Motivated by these facts this work aims at the optimisation of fungicide application schemes in the Mid-Hills of Nepal, both providing sufficient crop protection and minimisation of fungicide input. The methodology applied is explicit modelling of the population dynamics of P. infestans as well as the effects of different fungicide treatments on the disease progression. The model is based on the epidemiological concept of van der Plank (1963), which was extended by Hau (1988) and further specified for P. infestans in the work by Apel (2002). This approach differs from the range of the late blight forecast models and decision support systems developed and applied in Europe and North-America. The presented model is not meant to predict outbreaks of a P. infestans epidemic nor is it a decision support tool as such, although it may be used for this purpose. It is meant to study the area specific population dynamics of the fungus and the influence of different fungicide treatment strategies on the disease progression.

The forecasting models are generally based on temperature and humidity dependencies of P. infestans and predict the outbreak of late blight epidemics in advance in order to apply treatment measures in time. Most of the models like NegFry (Hansen et al., 1995), Guntz-Divoux (Rolot et al., 1998) or the forecast model of Johnson et al. (1996) work on a statistical or empirical basis without an underlying population dynamics model. This is achieved in the SIMPHYT models (Gutsche, 1998, 1999), which are based on the same assumption as the presented model. All of these models give recommendations for application times of fungicides, whereas SIMPHYT and Plant-Plus (Hadders, 1998) give additional informations on the type of fungicides to be used, because they distinguish different development stages. Prerequisite for all of the models are climatological data of the forecasting area usually in an hourly temporal resolution. Additionally all of the models are developed for Middle-European or North-American climatic conditions and genotypes of P. infestans.

Due to the fact that Nepali genotypes of P. infestans are most likely different from the European tribes and that the infrastructure for an extensive net of climatological stations is not present, we developed an independent model in order to study the population dynamics of the local tribes of P. infestans under the subtropical-monsoonal climate of Nepal. These climatic conditions offer a constantly perfect climate for the development of late blight epidemics\(^1\) during the potato growing season, which is further promoted by the prevailing ponding irrigation practice creating a very humid microclimate and ideal conditions for the oospore-movement within the crop. Hence the focus of this work was the study of local population dynamics of P. infestans and the development of a site specific, general treatment strategy rather than the establishment of a forecasting system. The local population dynamics parameters were estimated with the help of field infection data of an uncontrolled epidemic (Apel, 2002), whereas the treatment strategies were evaluated by scenario calculations. Based on these scenarios and the experiences in field trials recommendations for an improved fungicide treatment strategy under the given geographical situation could finally be derived.

In order to make the model available for a wide range of users, the model originally developed in Mathematica\(^\text{®}\) was rewritten in Java and provided with a graphical user-interface resulting in the PhytMod software.

2. Model development

The population dynamics model of P. infestans bases on the approaches of van der Plank (1963), who proposed that epidemics of plant diseases can be characterised by the three parameters latent period \(p\), infectious period \(i\) and infection rate \(r\). The latent period is the time between the infection of plant tissue and the outbreak of visible lesions. The infectious period is the timespan, in which the lesion sporulate, before the plant tissue finally dies. The progress of an epidemic was formulated in terms of normalised total infection \(Y(t)\) with the following delay differential equation (DDE):

\[
\frac{dY}{dt} = r (1 - Y(t)) \left[ Y(t-p) - Y(t-p) - Y(t) \right] \quad (1)
\]

This original model was extended by Hau (1988) to a model describing all relevant normalised stages (uninfected \(U\), latent \(L\), infected \(I\), dead \(A\)), as a set of DDEs:

\[
\frac{dU}{dt} = -r \ U(t) \ I(t) \quad (2)
\]

\[
\frac{dL}{dt} = r \ U(t) \ I(t) - r \ U(t-p) \ I(t-p) \quad (3)
\]

\(^1\) Mean daily temperatures measured during field trials: 10–20 °C; mean daily relative humidity: 80–100%; cf. optimum values for the development of P. infestans epidemics in the works of Schepers (1998).
This model concept describes the complete life-cycle of the fungus in terms of infected plant tissue. After the infection of uninfected, susceptible plant tissue \( U \) by the spores of the fungus, this plant material is denoted as latently infectious tissue \( L \), which becomes infectious plant material after passing through the latent period \( p \). The infectious plant material, the ‘lesions’, produce and release spores, which infect again susceptible tissue. After the infectious period \( i \) is passed the lesions die, thus comprising the dead plant tissue \( A \).

The infectious plant material, the ‘lesions’, produce and release spores, which infect again susceptible tissue. After the infectious period \( i \) is passed the lesions die, thus comprising the dead plant tissue \( A \). Fig. 1 illustrates how the total infected plant tissue \( Y \) of Eq. (1) is divided into the four stages with the help of the delays \( (t-p) \) and \( (t-p-i) \). The stages \( U, L, I \) and \( A \) are dimensionless, because they are normalised with respect to the tissue of a whole host plant, which itself is variable during its development. This has to be taken into account in the interpretation of estimated parameter values (cf. Section 4).

Eqs. (2)–(5) are the ‘standard’ model used in this work, but for the description of the epidemic of \( P. infestans \) it has to be extended. According to Hau (1988) citing van der Plank (1963), the growth of infectious lesions is an important factor in Phytophthora epidemics. With linear lesion growth, Eq. (1) is written as (Hau, 1988):

\[
\frac{dY}{dt} = r (1-Y(t)) \left( Y(t-p) - Y(t-p-i) \right)
\]

\( + \ Y(0) \ w \ (1-Y(t)) \)  

with \( w \) as the lesion growth rate and the density control term for the growth \( (1-Y(t)) \).

The extended model (2)–(5) including lesion growth is formulated analogously to Eq. (6).

It is also known that only newly set lesions sporulate, but with a comparatively high intensity. Hau (1988) introduced sporulation functions into Eqs. (2)–(5) in order to describe an age dependent sporulation of the infectious plant material, but not specifically for \( P. infestans \) epidemics. This has been done in this work by defining a sporulation function, which allows only newly set lesions to sporulate, but with a sporulation intensity \( (sp) \) that is higher than the usual value of 1 used by Hau (1988). The sporulation function was formulated as \( g(s,t) \), describing the sporulation intensity of lesion, which were set at the time \( s \), at the model runtime \( t \) (Eq. (7)).

\[
g(s,t) = \begin{cases} 
0 & \text{if } 0 \leq t-s < p \\
sp & \text{if } p \leq t-s < p + 1 \\
0 & \text{if } p + 1 \leq t-s 
\end{cases}
\]

The sporulation function is introduced into the model by expressing the infectious part in Eq. (1) in integral form:

\[
Y(t-p) - Y(t-p-i) = \int_{t-p}^{t} \frac{dY(s)}{ds} \ ds
\]

\[
= \int_{0}^{t} g(s,t) \frac{dY(s)}{ds} \ ds
\]

Finally the complete epidemiological model for \( P. infestans \) is written in a set of difference equations (9)–(13), replacing the DDEs in Eqs. (2)–(5) with appropriate formulations in differences and writing the integrals in Eq. (8) as the equivalent sums (Hau, 1988):

\[
L(t + \Delta t) = L(t) + \Delta t \ FY(t) - \Delta t \ FY(t-p)
\]

\[
I(t + \Delta t) = I(t) + \Delta t \ FY(t-p)
\]

\[
+ \Delta t \ w \ U(t) \sum_{n=1}^{i} FY(t-n\Delta t-p) - \Delta t \ FY(t-i)
\]

\[
-p \left( 1 + \Delta t \ w \sum_{n=1}^{i} U(t-n\Delta t) \right)
\]

Fig. 1. Concept of the van der Plank model equation (1) (Gutsche, 1999).


\[ A(t + \Delta t) = A(t) + \Delta t \cdot FY(t-i-p) \]
\[ (1 + \Delta t \cdot \sum_{n=1}^{i\Delta t-1} U(t-n\Delta t)) \]
\[ U(t + \Delta t) = 1-L(t+\Delta t)-I(t+\Delta t)-A(t+\Delta t) \]  
with

\[ FY(t) = r \cdot U(t) \left( \sum_{n=1}^{i\Delta t-1} \left( r \cdot U(t-n\Delta t-p) \cdot I(t-n\Delta t) \right) \right) \]

\[ -p \left( 1 + \Delta t \cdot \sum_{m=1}^{n} U(t-m\Delta t) \cdot g(t-n\Delta t-p,t) \right) \]  

\( FY(t) \) in Eq. (13) denotes an auxiliary equation describing the infection process, where the term with in brackets is equivalent to \( I(t) \) considering lesion growth and sporulation function. All differences to the standard model are due to the extension of the \( I(t) \) term: in the main formulae (9)–(12) it has been extended with the lesion growth, while in \( FY(t) \) both lesion growth and sporulation function are introduced.

The formulations in Eqs. (9)–(13) are valid for a constant growth rate \( w \). If \( w \) is assumed time dependent, it has to be written within the sums. This is the case when fungicide actions are considered.

### 2.1. Fungicide effects

Prior to a mathematical formulation of fungicide effects, the different modes of action have to be described and categorised. The different fungicide effects can be divided into three main classes (Bradshaw, 1999; Gutsche, 1988).

- **Protective action:** The spores are killed before germination/penetration of the leaf surface.
- **Curative action:** The fungicide is active during the post-infection period (latent period), but before lesions become visible. Latently infectious plant material is ‘cured’ to uninfected.
- **Eradicant action:** *P. infestans* is killed within sporulating lesions and thus preventing further sporulation and lesion growth.

Fig. 2 depicts these modes of action and their influence on the different stages of a late blight epidemic. While the protective and curative action are quantitatively studied for Mancozeb and Metalaxyl, the most prominent compounds for this classes on the market (Gutsche et al., 1994), the eradicant action is only qualitatively stated for Metalaxyl (Bradshaw, 1999).

In the model the protective action is defined as a reduction of the infection rate \( r \) and the lesion growth rate \( w \), thus writing

\[ r \rightarrow r \cdot (1 - FE_{pro}(t)) \]
\[ w \rightarrow w \cdot (1 - FE_{pro}(t)) \]

\( FE_{pro}(t) \) is the time depending protective fungicide effect, which depends on the application time and calculates the consequent reduction of the effect due to degradation of the compound. Gutsche et al. (1994) studied the effect of Dithane Ultra Spiess-Urania, a Mancozeb formulation, on *P. infestans* epidemics. He gave a polynomial expression for the reduction of Mancozeb-efficacy and assumed that Mancozeb is ineffective 21 d after the foliar application. The resulting formula for the protective effect of Mancozeb reads as follows:

\[ FE_{pro,Mancozeb}(t) = \begin{cases} k_{pro} \cdot (1 - 0.18207 \cdot t^{2.278} \cdot 1.0398) & \text{if } t_{app} \leq t < t_{app} + 21 \\ 0 & \text{if } t < t_{app} \text{ or } t > t_{app} + 21 \end{cases} \]  

with \( k_{pro} \) as the maximum protective efficacy within the range (0, 1) and \( t_{app} \) as the application time.

The same author also studied the protective action of Ridomil MZ-Super, a Metalaxyl–Mancozeb combination, which is the mostly used fungicide combination with protective, curative and assumed eradicant action. Based on his findings the decline of the protective efficacy of this formulation is defined as follows:

\[ FE_{pro,Mancozeb}(t) = \begin{cases} k_{pro} \cdot (1 - 0.118061 t) & \text{if } t_{app} \leq t < t_{app} + 1 \\ k_{pro} \cdot (1 - 0.103826 \cdot t^{0.1505} \cdot 1.1371) & \text{if } t_{app} + 1 \leq t < t_{app} + 21 \\ 0 & \text{if } t < t_{app} \text{ or } t > t_{app} + 21 \end{cases} \]  

The original formula of Gutsche et al. (1994) has been corrected with a linear part for \( t < t_{app} + 1 \). This was necessary, because for \( t = 0 \) the original formula is not defined due to the negative exponent. Also, with the original formula the calculated efficacy rises for \( 0 \leq t \)
< 1 with increasing $t$, starting with a value smaller than $k_{pro}$.

The curative effect of a fungicide application is considered by the ‘healing’ of the latent infected plant tissue with an effectiveness of $k_{cur}$ at application day and thus reducing $L(t)$ and adding the reduced amount to the uninfected compartment $U(t)$:

$$L(t_{app} + \Delta t) = L(t_{app}) (1-k_{cur}) U(t_{app} + \Delta t)$$

$$= U(t_{app}) + L(t_{app}) k_{cur}$$

This reduction works immediately, i.e. only in one time step. By applying Eq. (16), it is implied that the curative effect works indiscriminately well on the different age stages of the latent plant tissue. This is acceptable taking the relatively short latent periods of $P$. infestans and the findings of Gutschke et al. (1994) stating that Metalaxyl effectively reduces latent infections of all age stages.

The eradicant action is defined similarly to the curative action, only that the infected plant tissue is reduced and assigned to the dead compartment. Also the same assumptions hold true, i.e. that all age stages of the lesions are equally affected. Eq. (17) give the definitions:

$$I(t_{app} + \Delta t) = I(t_{app}) (1-k_{era}) A(t_{app} + \Delta t)$$

$$= A(t_{app}) + I(t_{app}) k_{era}$$

When introducing fungicide action into the model, one has to take care about the appropriate delay definition of the fungicide action. In case of the protective action this is easily done by using delayed $FE_{pro}$-functions: $FE_{pro}(t-p)$ and $FE_{pro}(t-p-i)$. In case of the curative effect this is more complicated, because $L$ does not appear at the right-hand sides of equation systems (9)–(13) and thus any changes in $L$ do not influence the progress of the infection in the desired way. Additional problems arise due to the delays in the model. In the delays ‘old’ values of the compartments are used, which are no longer valid due to the reduction of all age stages. Therefore the fungicide effect has to be traced through the development of the epidemic after the application. This is achieved by reducing the transition of the latent infections to open lesions by the factor $k_{cur}$ for the time $p$ after the application. The dying of these infectious parts must be retarded analogously, starting at $t_{app} + i$, again for the period $p$. Technically the retardation of the transition to lesions is done by the function $FE_{cur}(t)$, which is introduced at the appropriate positions in Eqs. (9)–(13):

$$FE_{cur}(t) = \begin{cases} k_{cur} & \text{if } t_{app} < t \leq t_{app} + i \\ 0 & \text{if } t \leq t_{app} \text{ or } t > t_{app} + i \end{cases}$$

The dying of plant tissue is analogously reduced with $FE_{cur}(t-i)$.

The eradicant action has to be treated analogously, with a function for the delayed effect on the dying of infections.

$$FE_{era}(t) = \begin{cases} k_{era} & \text{if } t_{app} < t \leq t_{app} + i \\ 0 & \text{if } t \leq t_{app} \text{ or } t > t_{app} + i \end{cases}$$

$$L(t + \Delta t) = (L(t) + \Delta t FY(t) - \Delta t FY(t-p)) (1 - FE_{cur}(t)) (1-k_{cur})$$

$$I(t + \Delta t) = (I(t) + \Delta t FY(t-p)) (1 - FE_{cur}(t))$$

$$A(t + \Delta t) = A(t) + \Delta t FY(t-i-p) \left(1 - FE_{cur}(t) - i\right)$$

$$- i + \Delta t \sum_{n=1}^{\Delta t - 1} w (1-FE_{pro}(t-n\Delta t)) U(t-n\Delta t)$$

$$- n\Delta t) \right) (1-FE_{era}(t)) + I(t) k_{era}$$

$$U(t + \Delta t) = 1 - L(t + \Delta t) - I(t + \Delta t) - A(t + \Delta t)$$

with

$$FY(t) = r \left(1 - FE_{pro}(t)\right) U(t) \left(\sum_{n=1}^{\Delta t - 1} \left( r \left(1 - FE_{pro}(t - n\Delta t - p) U(t-n\Delta t - p) - n\Delta t - p\right) \right) \sum_{m=1}^{n} U(t-m\Delta t) g(t-n\Delta t-p) \right)$$
nition along with the units used in this study. With this model definition a thorough investigation of pesticide application strategies and their effects can be undertaken, as outlined in Section 3.

2.2. Implementation details

Initially all the models described in the previous sections were implemented and solved in Mathematica®, both the model definitions with DDEs or the difference equations. For the solution of the DDE model (Eqs. (2)–(5)) the AddOn-Package NDelayDSolve was used. This package is based on the standard numerical ODE-solvers implemented in Mathematica® with added algorithms for the consideration of the delays in the solution process. While this tool could be used to obtain accurate solutions of the standard model, it cannot handle fungicide effects, because any fungicide impact changes the history of the solution. This feature is not supported by NDelayDSolve.

For this reason the DDE model was transformed into a set of difference equations, which enabled the consideration of fungicide effects, as shown in Eqs. (20)–(24). The original code of the solution was also developed in Mathematica® and later ported to Java as the computational kernel of PhytMod.

2.2.1. Accuracy

The accuracy of the difference equations model was assessed by a comparison of solution of the standard DDE model (Eqs. (2)–(5)) with solutions of the equivalent difference equations at different stepsizes. As Fig. 3(A) shows, the difference equations model is sensitive to changes in the stepsize, with solutions approximating the reference solution of the DDE model with smaller stepsizes. The main differences occur typically when the system state is changing fast, i.e. when the epidemic gains momentum before it reaches the climax. However, in this phase the absolute differences shown in Fig. 3(B) are negligibly small in terms of disease progression and management strategies, since the delay of the climax is only marginal.

2.2.2. Technical details

The algorithms currently implemented in PhytMod restrict the length of $p$ and $i$ to integer values. This implies a restriction of the stepsize selection in a way that the upper limits of the sums in Eqs. (9)–(13) and (20)–(24) always evaluate to integers. Hence, in PhytMod the stepsize selection is restricted to $\Delta t = 0.1, 0.2, 0.5$ or 1.

The initial infection $y_0$ is always equally distributed to $L$ and $I$ on day 0, while the initial history is set to 0, i.e. no infection before the starting inoculation.

2.3. Parameter estimation

The parameter estimation was performed with data collected in field trials in the Jhikhu-Khola watershed in the Mid-Hills in Nepal. From these field trials an untreated disease development (Apel, 2002) was taken for the estimation of the model parameters. The data were derived by a weekly pest assessment on 10 sample plants per plot, with four plot repetitions, each approximately 50 m² large. From these records the mean percentage of visible infestation (lesions plus dead plant material) per plot was calculated. Finally, for the parameter estimation the mean infestation of the four repetition plots calculated from the mean plot infestation was used.

The estimation itself was divided into two steps: first the basic parameters $p$, $i$, $r$ and initial infection $y_0$ were estimated with the standard model (Eqs. (2)–(5)) by minimisation of the residual sum of squares (least squares criterion). For this estimation a constraint optimisation procedure had to be applied, which restricts the values of $p$, $i$ and $r$. This was necessary, because the field data do not distinguish between infected and dead plant material resulting in an ambiguous minimisation problem.² In the second step the $P. infestans$ specific parameters $w$ and $sp$ were estimated using Eqs. (9)–(13)

² A mathematically possible solution of the problem is to set $i$ to very large values, thus disabling the dying of lesions. This means that the field data comprising infectious and dead plant tissue would be modelled with the infectious plant tissue $I$ only. Of course, this is an unrealistic solution and not acceptable.
and the prior estimated basic parameters using the same optimisation criterion. The optimisation was undertaken using the FindMinimum-algorithm implemented in Mathematica®, which itself uses a gradient, Newton or quasi-Newton algorithm, depending on the problem.

Because of the difficult optimisation problem and the specialised software used in the optimisation process the PhytMod program does not contain a parameter estimation feature in the current version.

2.4. Scenario definition

Based on the model with the optimised parameter set the effectivity of different fungicide treatment strategies were evaluated using the fungicide definitions and equations of Section 2.1, with \( k_{\text{pro}}, k_{\text{cur}} \) and \( k_{\text{era}} \) set to 1. According to the different modes of fungicide action different treatment scenarios were calculated with the Phytophthora model including fungicide activity (Eqs. (20)–(24)). For all the three modes of fungicide action associated to frequently used fungicides on the market (Mancozeb, Metalaxyl), scenarios were defined with two different application intervals, 7 and 14 d, and different starting days of the applications, depending on the infestation level. The infestation thresholds were set to 0, 0.05, 0.1, 0.15 and 0.2 visible infections, respectively. Among these scenarios the 7-d application interval starting from an infestation level of 0 is equivalent to the locally adopted treatment strategy of the farmers in the Mid-Hills of Nepal. The other scenarios served as possible alternative management option in order to reduce the amount of fungicides applied.

3. Results

The parameter estimation procedure described in Section 2.3 produced the estimated parameter set listed in

Table 1. The length of the latent and infectious periods \( p \) and \( i \) were estimated at 3 and 11 d, respectively, with an infection rate \( r \) of 0.333 d\(^{-1}\). The lesion growth rate \( w \) evaluated into 0.326 d\(^{-1}\) with a sporulation intensity \( \text{sp} \) of 3 d\(^{-1}\). However, it has to be noted that \( w \) and \( \text{sp} \) proved to be highly correlated and could consequently not be statistically distinguished in the estimation procedure with the present data set. But due to the fact that lesion growth and the specific sporulation function are typical for an \( P. \text{infestans} \) epidemic as well as important for the assessment of fungicide treatments, these parameters were kept in the model and the biologically most plausible pair of estimated values for \( w \) and \( \text{sp} \) were selected (Apel, 2002). The model fit was satisfyingly high as expressed by the \( R^2 \)-value (quotient of model sum of squares to total sum of squares) of 0.908 with a \( p < 0.001 \). Fig. 4 shows the resulting model trajectories along with the optimisation data.

The results of the scenario calculations with the protective fungicide (Fig. 5) showed that the infection is fairly controlled if the applications start right after plant establishment, even in the case of a 14-d application interval. In the cases of a later application start the disease proved to be hard to control. The general trend is the later the application starts, the harder the disease is to control, as expected.

The same holds true for the results obtained with a combined curative and protective effect as shown in Fig. 6. However, it can be seen that the control is significantly more efficient in comparison to the sole protective action. The disease progression is stopped quickly, while the protective action requires some time to bring the epidemic to halt. Finally, the introduction of an additional eradicant fungicide effect (Fig. 7) produced only slightly better results than the combined curative and protective action scenarios in case of a 14-d application scheme. However, in case of a 7-d application scheme the disease progress is stopped immediately.
Fig. 4. Phytophthora model (9)–(13) trajectories with optimised parameters: (A) visible infection \((L + I)\) and data with standard deviation; (B) all stages.

Fig. 5. Model response (visible infection) to the control of \(P.\) 
infestans with a fungicide with curative and protective action (Metalaxyl + Mancozeb) in different management scenarios; thresh, infestation threshold for start of fungicide application scheme; \(t_a\), equivalent day of application start (d).

Fig. 6. Model response (visible infection) to the control of \(P.\) 
infestans with a fungicide with curative and protective action (Metalaxyl + Mancozeb) in different management scenarios; thresh, infestation threshold for start of fungicide application scheme; \(t_a\), equivalent day of application start (d).

Fig. 7. Model response (visible infection) to the control of \(P.\) 
infestans with a fungicide with curative and protective action (Metalaxyl + Mancozeb) in different management scenarios; thresh, infestation threshold for start of fungicide application scheme; \(t_a\), equivalent day of application start (d).
4. Discussion and recommendations

The uncontrolled disease development data served as a valuable source for the parameter estimation of a population dynamics model for *P. infestans*. With these data site-specific epidemiological parameters could be identified. The values stated in Table 1 indicate that the latent and infectious period of Nepali tribes of *P. infestans* are of comparable length to European tribes (Gutsche, 1999).

The infection rate \(r\), however, is almost three times higher under Nepali conditions indicating either a higher aggressiveness of the local tribes or favourable climatic conditions or both. However, while interpreting the parameter values it has to be kept in mind that these are effective parameters, summarising not only disease specific effects, but also external influences like climatic conditions and growth of the host plant. This has a significant consequence on the validity of the model, viz. the estimated parameters: it is only valid for *P. infestans* epidemics on potato under post-monsoon climate in the Mid-Hills of Nepal on irrigated land. This validity assessment is based on the following facts: The climatic conditions during the field trials (cf. Section 1) is representative for the area (Kansak et al., 2001a) and the climatic conditions of the investigation area are again representative for the Mid-Hill region of Nepal, which shows a rather uniform climate (DHM, 1999). Additionally the ponding irrigation practice widely used in potato cropping supports this validity range due to its regulating effect on the microclimate.

The presented model structure is also the reason why a realistic reduction of the disease level cannot be simulated with this model. In order to achieve this, the growth of the host plant, the recovery potential of the host plant in the different phenological stages and the climatic influence have to be modelled explicitly.

Nevertheless, the model can of course be used for the assessment of fungicide treatment strategies under site specific conditions. The fungicide application scenarios indicated that a 14-d application interval of a protective fungicide (Mancozeb) can sufficiently control the late blight disease (cf. Fig. 5). These theoretical findings are corroborated by the experimental variations of application strategies in the field trials equivalent to the model application scenarios (Apel, 2002).

Additionally the model calculations revealed another important fact: the starting day of the applications. As shown in Figs. 5–7, an early application start is necessary to control the disease effectively, even with a combined protective, curative and eradicant effect. Due to the aggressiveness of the disease as expressed in the high infection rate, an effective control is hardly possible if the disease exceeds an infestation levels above approximately 0.1. Above this level the spread of the disease accelerates quickly: within 25 d the disease is able to increase from 10 to over 90% visibly infected plant tissue (cf. Fig. 4). Although the fungicide treatments, especially the curative and eradicant actions are able to stop the epidemic quickly, in case of a 7-d application scheme of eradicant fungicides even immediately, the infestation level may be already that high, that the crop is severely impaired or has to be regarded as economically lost. This result is in line with practical studies of fungicide control schemes, e.g. as recently published by Tsakiris et al. (2002).

From these findings and the general assumption that an infestation level of less than 0.1 is acceptable without impairing yield, two basic recommendations for the conventional treatment of *P. infestans* in the Mid-Hills of Nepal can be derived:

1. For the control of *P. infestans* on potato in the winter season in the Mid-Hills of Nepal a regular application of Mancozeb at the recommended dose in 14 d intervals is sufficient. The application should start within the first week after crop establishment.
2. In severe cases a Metalaxyl + Mancozeb fungicide formulation may be used to stop the epidemic. In this case a weekly application frequency is recommended for at least 2–3 weeks in order to stabilise the infection level. Thereafter a 14-d interval is sufficient.

With this recommendation it is most likely that *P. infestans* is controlled sufficiently (i.e. not completely eradicated) while keeping investments in chemical control low, improving the economic benefit and reducing the environmental contamination risk. It also keeps the risks of Metalaxyl resistance of *P. infestans* low, a phenomenon often observed in cases of frequent Metalaxyl applications (Bradshaw, 1999; Griffin et al., 1998).

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References


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3 This figure is deduced from the practical experiences of the farmers in the investigation area as well as from the extension officers of the Department of Agriculture.