

Population Dynamics and Treatment Strategies of *Phytophthora infestans* (late blight) in the Mid-Hills of Nepal

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Abstract: The fungus *Phytophthora infestans* is a major ubiquitous pest in solanaceous crops and causes great damages in the agricultural systems in Nepal. This is especially true in the cash crop orientated areas of the Mid-Hills and the Terai region, where potato and tomato cropping is a prominent income source of the rural population. This fact in combination with cheap and readily available pesticides is responsible for the frequent and perceived over-use of chemical plant protection agents. Based on this background a series of agronomic field trials in the Jhikhu-Khola watershed aiming at the optimisation of *P. infestans* treatment strategy was conducted. Within the field trials three different treatment strategies comprising both conventional and an alternative IPM approach were tested. Additionally a mathematical model for the simulation of the population dynamics of *P. infestans* was established, which is based on a universal epidemiological model with further specification for the late blight. The parameter estimation for the model was accomplished with uncontrolled field data. Using this model different treatment strategies were tested by introducing pesticide treatment strategies into the model. Based on both field trials and population dynamics simulation recommendations for an optimised conventional treatment strategy were developed.

Keywords: *Phytophthora infestans*; Population dynamics; Modelling; Treatment strategies; Nepal

1. INTRODUCTION

This paper deals with the most prominent pest of solanaceous crops world-wide, the late blight *Phytophthora infestans* and its population dynamics. This fungal disease is able to destroy a crop completely and consequently causes serious economic damages or even famines where potatoes are the main staple crop. This is historically documented as the infamous famine in Ireland in the mid-1840s, which resulted in the large immigration wave to America. Since then, the control of *P. infestans* has been a major issue in agricultural research and production.

Nepal is no exception to that. Since the introduction of potato and tomato as valuable cash crops, the control of late blight is the major concern of Nepali farmers. To a large extend the control bases on chemicals, as documented in national pesticide consumption and import statistics as well as in the surveys conducted in the investigation area, the Jhikhu-Khola watershed [Kansakar, Khanal et al. 2001; Pujara and Khanal

2001], being one of the largest production areas of potatoes and tomatoes for the Kathmandu valley.

Based on this background a series of agronomic field trials were conducted in co-operation with the Plant Protection Division (PPD) of the Department of Agriculture (DOA). These trials intended to investigate the reduction potential of and possible alternatives to the usual chemical control measures.

In the trials the visible disease development of *P. infestans* as well as the control measures were recorded. The results were statistically evaluated in order to compare the different applied treatment strategies.

Additionally a dynamic pest population model for *P. infestans* was developed for the simulation of the disease epidemic and the effect of fungicide treatment. This mathematical model was used to study the response of *P. infestans* epidemics under the given geographic and climatic circumstances to different treatment strategies. Based on these theoretical studies combined with the statistical evaluation of the field data recommendations for

fungicide application strategies were eventually derived.

2. EXPERIMENTAL DESIGN

For the field trials a representative plot on irrigated (khet) land was selected on the DOA Horticulture Farm in Tamaghat. Three different treatment strategies were applied, namely the “usual treatment”, “reduced treatment” and “IPM”. The usual treatment is an adoption of the mean treatment strategy of the local farmers, which means that the protective fungicide Mancozeb was non-discriminatory applied weekly and the systemic fungicide Metalaxyl when necessary [cf. CEAPRED 2000]. In the reduced treatment the same dose as in the usual treatment was applied but at half frequency only. The IPM treatment is an Integrated Pest Management strategy, using mainly biological and cultural pest management methods and chemical pesticides just as the last resort to save the crop. The biological alternatives to Mancozeb are the parasitic fungi *Trichoderma viridae* and *Trichoderma harzianum*, which thrive on the expenses of other fungi like *P. infestans*.

The selected site was separated into 12 sub-plot of approximately equal size (45-50 m²). The plots with the same treatment strategy were arranged in clusters, with 4 repetitions for each strategy. Figure 1 shows the layout of the experimental plots. On this plot potatoes were grown during the typical season for irrigated land, i.e. from November 1999 to February 2000. During this period a weekly pest assessment was done on 10 selected plants per plot. The disease pressure was recorded as the visibly infected foliage area per plant in steps of 10 %. Besides this the dates and concentrations of treatment measures within the three treatment strategies were also logged.

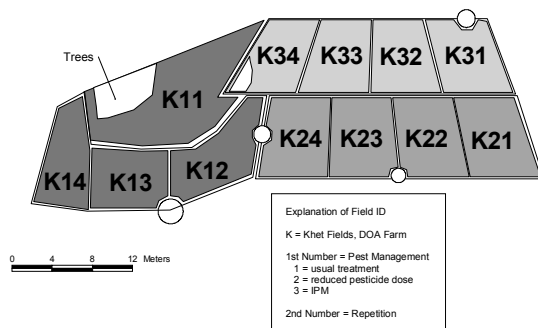


Figure 1. Experimental fields on khet land (DOA Farm at Tamaghat)

3. DYNAMIC POPULATION MODEL

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 factors 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 lesions. The infectious period is the timespan, in which the lesion sporulate, before the plant tissue finally dies. The original model of van der Plank [1963] formulating the epidemic in terms of total infected plant tissue was extended by Hau [1988] to a model describing all relevant stages (Uninfected U , Latent L , Infected I , Dead A) as a set of Delay Differential Equations (DDEs):

$$\frac{dU}{dt} = -R \cdot U(t) \cdot I(t) \quad (1)$$

$$\frac{dL}{dt} = R \cdot U(t) \cdot I(t) - R \cdot U(t-p) \cdot I(t-p) \quad (2)$$

$$\frac{dI}{dt} = R \cdot U(t-p) \cdot I(t-p) - R \cdot U(t-p-i) \cdot I(t-p-i) \quad (3)$$

$$\frac{dA}{dt} = R \cdot U(t-p-i) \cdot I(t-p-i) \quad (4)$$

(1) – (4) are the base 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. It is also known that only the newly set lesions sporulate, but with a comparatively high intensity. Hau [1988] introduced a linear lesion growth into the standard model, as well as sporulation functions in order to distinguish 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 and with a sporulation intensity (sp_int) 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 set at the time s at runtime t . (5) shows the rectangular step function.

$$g(s,t) = \begin{cases} 0 & \text{if } 0 \leq t-s < p \\ sp_int & \text{if } p \leq t-s < p+1 \\ 0 & \text{if } p+1 \leq t-s \end{cases} \quad (5)$$

The sporulation functions were introduced into the model by expressing the infectious part in integral form:

$$Y(t-p) - Y(t-p-i) = \int_{t-p-i}^{t-p} \frac{dY(s)}{ds} ds = \int_0^t g(s,t) \frac{dY(s)}{ds} ds \quad (6)$$

Finally the complete epidemiological model for *P. infestans* is written in a set of difference equations (7) – (11), replacing the DDEs in (1) – (4) with

$$L(t + \Delta t) = L(t) + \Delta t \cdot FY(t) - \Delta t \cdot FY(t - p) \quad (7)$$

$$I(t + \Delta t) = I(t) + \Delta t \cdot FY(t - p) + \Delta t \cdot W \cdot U(t) \cdot \sum_{x=1}^{i/\Delta t - 1} FY(t - x\Delta t - p) - \Delta t \cdot FY(t - i - p) \cdot \left(1 + \Delta t \cdot W \cdot \sum_{x=1}^{i/\Delta t - 1} U(t - x\Delta t) \right) \quad (8)$$

$$A(t + \Delta t) = A(t) + \Delta t \cdot FY(t - i - p) \cdot \left(1 + \Delta t \cdot W \cdot \sum_{x=1}^{i/\Delta t - 1} U(t - x\Delta t) \right) \quad (9)$$

$$U(t + \Delta t) = 1 - L(t + \Delta t) - I(t + \Delta t) - A(t + \Delta t) \quad (10)$$

with

$$FY(t) = R \cdot U(t) \cdot \left(\sum_{x=1}^{i/\Delta t - 1} \left(R \cdot U(t - x\Delta t - p) \cdot I(t - x\Delta t - p) \cdot \left(1 + \Delta t \cdot W \cdot \sum_{u=1}^x U(t - u\Delta t) \right) \cdot g(t - x\Delta t - p, t) \right) \right) \quad (11)$$

3.1 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 [Gutsche 1988, Schepers and Bouma 1999]:

- **protective action:** The spores are killed before germination/penetration.
- **curative action:** The fungicide is active during the post infection period (latent period), but before lesions become visible.
- **eradicant action:** *P. infestans* is killed within sporulating lesions and thus preventing further sporulation and lesion growth.

Figure 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 for this classes on the market [Gutsche, Burth et al. 1994], the eradicant action is only qualitatively stated for Metalaxyl.

appropriate formulations in differences and writing the integrals in (6) as the equivalent sums [Hau 1988]. This step enables an explicit straight-forward solution of the model with negligible differences to the numerical solution of the DDEs and, more important, it also enables the incorporation of fungicide effects, which causes numerical problems in the solution of the DDEs.

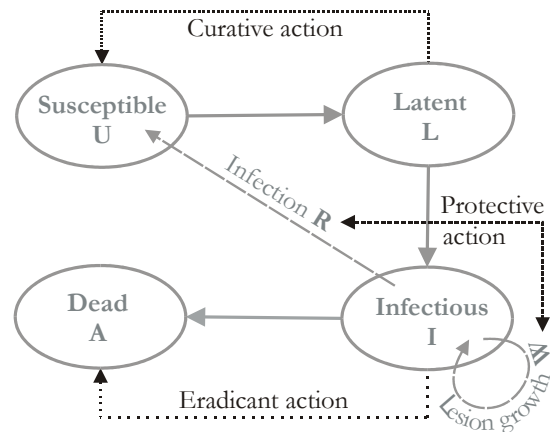


Figure 2. Different modes of fungicide action

The protective action is defined as a reduction of the infection rate R and the lesion growth rate W . The curative action is considered as a reduction of latently infected to susceptible, uninfected plant tissue. Finally the eradicant action is expressed as an immediate transition of infectious lesions to dead plant material.

The time dependent fungicide effects are formulated according to Gutsche, Burth, et al.

[1994] with a maximum efficacy of 1, i.e. 100 %. The incorporation of the fungicide effects into (7)–(11) was done according to the definitions above and are described in detail in Apel [2002].

4. RESULTS

4.1 Field experiments

The results of the field experiments are illustrated by Figure 3 in terms of mean relative infection levels, whereas an infection level of 1 is equivalent to a real infestation of > 50 % infected leaf area. It can be seen that the usual and reduced approach are equally effective in controlling the disease. However, the IPM approach apparently failed. But this was caused by the non-availability of the required biological agents and not by the efficacy of *Trichoderma*. The agents were only available after the infestation reached a level at which a control, even with chemicals was no longer possible. In consequence this means that the IPM data can be regarded as a untreated control series, which was further used for the parameter estimation of the population dynamics models (4.2).

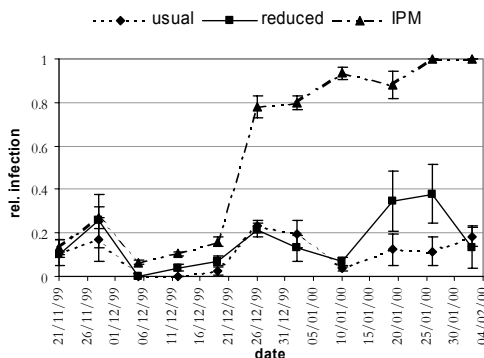


Figure 3. Normalised mean infection of *P. infestans* on potato at the different treatment strategies, Nov. 1999 – Feb. 2000; error bars indicate one standard deviation

The statistical significance of the influence of the treatments on the disease development were tested with a Repeated Measures Analysis [Winer 1971]. This showed that the treatments have a highly significant influence on both the trend and level of the disease development and hence corroborated the visual interpretation of the field results.

4.2 Population Dynamics

For the parameter estimation of the population dynamics model the “uncontrolled” IPM data

were used. In a first step the basic parameters p , i , and R were estimated with (1) – (4) on the basis of the Least Squares criterion. In a second step the *P. infestans* specific parameters sporulation intensity sp_int and lesion growth rate W were additionally estimated using the same technique using equations (7) – (11) with a stepsize of $\Delta t = 1$. This procedure resulted in the parameter set listed in Table 1 with a good fit to the experimental data ($R^2 = 0.908744$). Figure 4 shows the model fit and the trajectories of all stages.

p	3
i	11
R	0.333
W	0.326488
sp_int	3
y_0	0.0073

Table 1. Optimised parameter set for (7) – (11); initial infection y_0 distributed equally to L and I

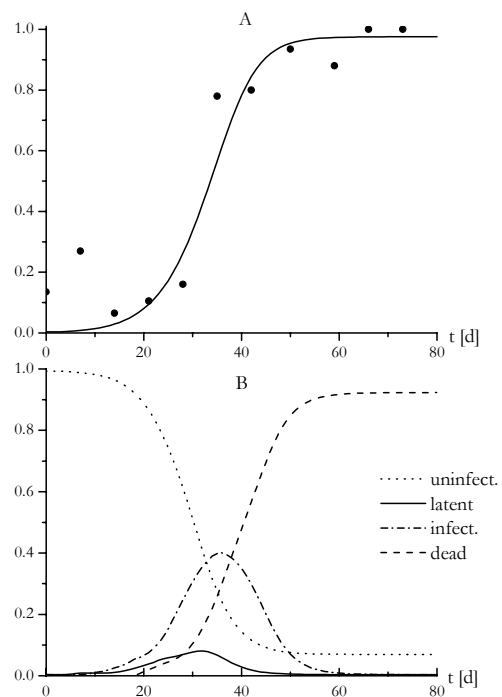


Figure 4. Phytophthora model (7)–(11) with optimised parameters: A: visible infection ($L + I$) and data, B: all stages

4.3 Fungicide application scenarios

According to the experimental design and the different modes of fungicide action different treatment scenarios were calculated with the Phytophthora model (7) – (11) and the optimised parameter set. For all the three modes of fungicide action associated to frequently used fungicides on the market, scenarios were defined

with two different application intervals, 7 and 14 days, and different application thresholds, depending on the infestation level. Figures 5 – 7 show the resulting calculated disease developments. t_a in the legend gives the application start in days equivalent to the defined thresholds of infestation levels of 0, 0.05, 0.1, 0.15 and 0.2 respectively.

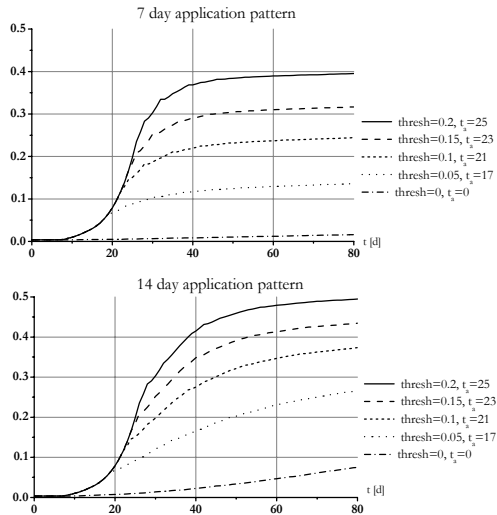


Figure 5. Model response (visible infection) to the control of *P. infestans* with a protective fungicide (Mancozeb)

5. DISCUSSION AND RECOMMENDATION

The experimental comparison of different treatment strategies of *P. infestans* showed that a reduced approach applying fungicides only half as often as it is usually practised in the Jhikhu-Khola watershed is sufficient for the control of the disease. An assessment of the efficacy of the IPM approach cannot be given with the present data, because the biological control agents were not available on the Nepali market in time. However, the consequently uncontrolled disease development served as a valuable data 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].

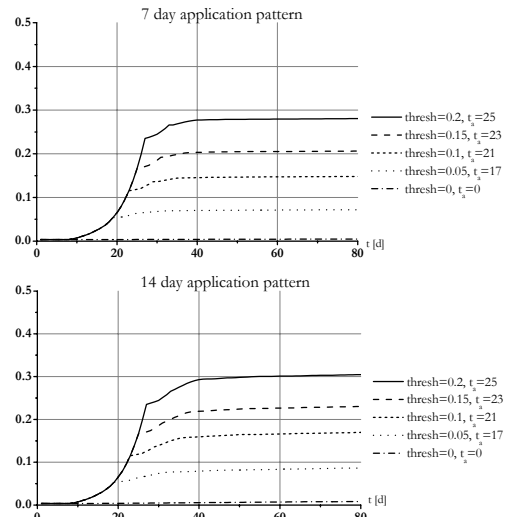


Figure 6. Model response (visible infection) to the control of *P. infestans* with a fungicide with curative and protective action (Metalaxyl + Mancozeb)

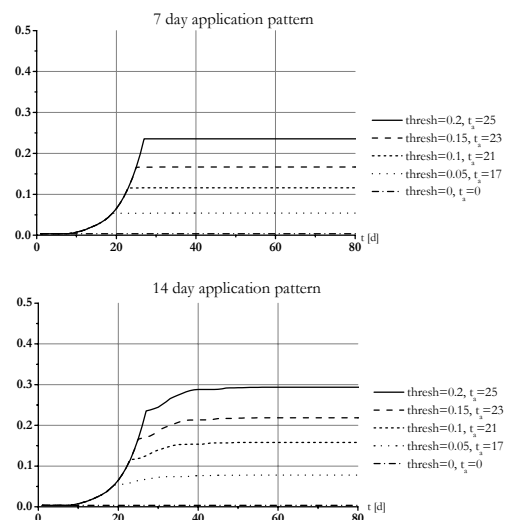


Figure 7. Model response (visible infection) to the control of *P. infestans* with a fungicide with eradicant, curative and protective action (Metalaxyl + Mancozeb)

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. This is also the reason why a 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 stage and the climatic influence have to be modelled explicitly.

However, the model can of course be used for the assessment of fungicide treatment strategies under site specific conditions. The fungicide application scenarios corroborated the findings of the field trials: A 14-day application interval of a protective fungicide (Mancozeb) can be sufficient for the control of the late blight (cf. Figure 5).

But the model calculations revealed another important fact: the start of the applications. As shown in Figures 5 – 7, an early application start is necessary to control the disease effectively, even with a combined protective, curative and eradicator effect. Due to the aggressiveness of the disease an effective control is hardly possible if the disease exceeds an infestation levels above 0.2, equivalent to 10 % real infected leaf area, especially in the reproductive stage of the host plant. In other words this means that the economic threshold level for *P. infestans* treatments would be close to zero, especially under the low cost conditions of pesticides in Nepal in comparison to the high market value of the crops.

From these findings 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 regularly application of Mancozeb at the recommended dose in 14 days 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.

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.

6. REFERENCES

Apel, H., Risk Assessment of Pesticides in the Mid-Hills of Nepal - Environmental Fate and Population Dynamics Modelling. PhD Thesis, *Institute of Geoecology, TU Braunschweig*: 127 p., 2002.

CEAPRED, Socioeconomic survey of the Jhikhu-Khola watershed, *Center for Environmental and Agricultural Policy Research, Extension and Development (CEAPRED)*, unpublished project report, 47 p., 2000.

Gutsche, V., Die Entwicklung und Nutzung von Schaderregermodellen in Forschung und Praxis des Pflanzenschutzes, Dissertation, *Institut für Pflanzenschutzforschung Kleinmachnow, Akademie der Landwirtschaftswissenschaften der DDR*: 147 p., 1988.

Gutsche, V., Das Modell SIMPHYT 3 zur Berechnung des witterungsbedingten Epidemiedruckes der Krautfäule der Kartoffel (*Phytophthora infestans* (Mont.) de Bary), *Nachrichtenblatt des Deutschen Pflanzenschutzdienstes* 51(7): 169-175, 1999.

Gutsche, V., U. Burth, et al., Abbildung der Wirkung von Phytophthora-Fungiziden im Simulationsmodell, *Nachrichtenblatt des Deutschen Pflanzenschutzdienstes* 46(10): 224-230, 1994.

Hau, B., Ein erweitertes analytisches Modell für Epidemien von Pflanzenkrankheiten. Habilitationsschrift, *Fakultät für Agrarwissenschaften, Justus-Liebig-Universität Gießen*, 168 p., 1988.

Kansakar, V. B. S., N. R. Khanal, et al., Use of insecticides in Nepal. International Workshop on Environmental Risk Assessment of Pesticides and Integrated Pesticide Management in Developing Countries (Abstracts), Kathmandu, Nepal, *Institute of Geoecology, TU Braunschweig, Germany*, 35, 2001.

Pujara, D. S. and N. R. Khanal, Use of pesticides in Jaishidi subcatchment, Khikhu Khola watershed, Middle Mountains of Nepal. International Workshop on Environmental Risk Assessment of Pesticides and Integrated Pesticide Management in Developing Countries (Abstracts), Kathmandu, Nepal, *Institute of Geoecology, TU Braunschweig, Germany*, 42, 2001.

Schepers, H. and E. Bouma, (Eds.), PAV-Special Report no. 5. Lelystad, Applied Research for Arable Farming and Field Production of Vegetables, 289 p., 1999.

van der Plank, J. E., Plant Diseases: Epidemics and Control. *New York - London, Academic Press*, 349 p., 1963.

Winer, B. J., Statistical principles in experimental design, *New York, McGraw-Hill*,. 907 p., 1971.