EFFECT OF PHYSICAL ENVIRONMENT ON *Cercospora carotae* AND DEVELOPMENT OF A MODEL TO PREDICT CERCOSPORA BLIGHT OF CARROT

by

Odile Carisse[c]

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Department of Plant Science
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McGill University

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SHORT TITLE

DEVELOPMENT OF MODEL TO PREDICT CERCOSPORA BLIGHT OF CARROT

Odile Carisse[c]

FOREWORD

This thesis is comprised of seven sections. Section one is a general introduction and a review of literature presenting the nature of the problem. Sections two to six are the body of the thesis. Section seven is a general discussion with a synthesis of the conclusions. Sections two and three are presented as complete manuscripts. The thesis format has been approved by the faculty of graduate Studies and Research of McGill University and follows the conditions outlined in the "guideline concerning theses preparation", section seven, "Manuscripts and authorship" which are as follows:

"The candidate has the option, subject to the approval of the Department, of including as part of the thesis the text, or duplicated published text (see below), of an original paper, or papers. In this case the thesis must still conform to all other requirements explained in Guideline concerning thesis preparation. Additionnal material (procedural and design data as well as description of equipment) must be provided in sufficient details (e.g. in appendices) to allow a clear and precise judgement to be made of the importance of the originality of the research reported. The thesis should be more then a mere collection collection of manuscripts published or to be published. It must include a general abstract, a full introduction and literature review and a final overall conclusion. Connecting text wich provide logical bridge between different manuscripts are usually desirable in the interest of cohesion.

It is acceptable for thesis to include as chapter authentic copies of papers already published, provided these are duplicated clearly on thesis stationary and bound as an integral part of the thesis. Photographs or other material wich do not duplicatewell must be included in their original form. In such instances connecting texts are mandatory and suplementary explanatory material is always almost necessary.

The inclusion of manuscripts co-authored by the candidate and others is acceptable but the candidate is required to make an explicit statement on who contributed to such work and to what extent, and supervisor must attest to the commitee. Since the task of the examiners is made more difficult in this cases, it is the candidate's interest to make the responsabilities of the author perfectly clear. Candidates following this option must inform the Department before it submits the thesis for review."

Although all the work presented here was the responsibility of the candidate, the project was supervised by Dr A. C. Kushalappa, Department of Plant Science, Macdonald College of McGill University, and Dr D.Cloutier, Agriculture Canada Experimental Farm, l'Assomption. The five manuscripts are co-authored by Dr. A. C. Kushalappa. For consistency and convenience, all manuscripts follow the same format. The copies that will be sent to the respective journals, however, will follow the specific requirements of each journal. The first manuscript was accepted for publication in July 1991 in the journal "PHYTOPATHOLOGY". The other manuscripts will be submitted for publication in suitable journals.

ABSTRACT

Ph. D. Odile Carisse Plant Science

EFFECT OF PHYSICAL ENVIRONMENT ON Cercospora carotae AND DEVELOPMENT OF A MODEL TO PREDICT CERCOSPORA BLIGHT OF CARROT

The effect of interrupted leaf wetness (IWP) and % RH on infection by Cercospora carotae (Pass.) Solh. was studied by inoculating carrot leaves (Daucus carota var sativa DC. L.) and subjecting the plants to different IWP treatment, continuous leaf wetness (CWP) and to different combinations of %RH and temperature with and without an initial wet period of 6 hr. IWP significantly reduced infection as compared to CWP. Infection was optimal under leaf wetness and decreased with decrease in percent RH. The effect of temperature and duration of moist period on sporulation of C. carotae was studied on carrot plants under leaf wetness, 96%RH, and 96%RH with an initial 12 hr of leaf wetness. For all types of moisture conditions, sporulation increased with the increase in temperature up to the optimum (28 °C) and then declined. Logistic and polynomial models were used to describe the effect of temperature and time on sporulation under these moisture conditions. The incubation period of Cercospora carotae was studied in the field. First lesions were observed 6 to 8 days after inoculation and new lesions appeared until the 10th to 14th day. The beginning, mean, and end of incubation period was modelled as a function of mean daily temperature and mean daily RH $\geq$ 90%. A model describing lesion appearance as a function of time was developed using a logistic function (R^2=0.84). A prediction model containing series of equations that described mathematically the interaction among predicted inoculum, infection and sporulation equivalents for the environment was developed and validated. In general, the model predicted adequately Cercospora blight progress. A weather-based forecasting system was developed to time the first fungicide spray to manage Cercospora blight of carrot based on the accumulation of critical number of disease severity units.

RESUME

Ph. D. Odile Carisse Phytologie

EFFET DE L'ENVIRONNEMENT PHYSIQUE SUR LE Cercospora carotae ET DEVELOPPEENT D'UN MODELE DE PREVISION DE LA BRULURE CERCOSPOREENNE DE LA CAROTTE

Les effets d'une mouillure interrompue des feuilles (IWP) et de l'humidité relative sur l'infection par le Cercospora carotae (Pass.) Solh. furent étudiés en soumettant des feuilles de carotte (Daucus sativa DC. L.) inoculées à différentes conditions de mouillure et % d'humidité relative (HR). La mouillure des feuilles interrompue a significativement réduit l'infection comparativement à la mouillure continue. L'infection fut optimale suite à une mouillure complète des feuilles et a diminué au fur et à mesure que l'humidité relative diminuait. L'effet de la température et de la durée de la période humide sur la sporulation du C. carotae fut étudié sur des feuilles de carottes exposées à une mouillure complète, à 96% HR et à 96% HR précédé de 12 heures de mouillure. Pour tous ces traitements, la sporulation a augmenté au fur et à mesure que la température augmentait jusqu'a 28 °C puis a diminué. Des modèles logistiques et un modèle polinômial ont été utilisés pour décrire l'effet de la température et du temps pour les conditions étudiées. La période d'incubation fut étudiée au champ sur 150 plantes en 1990. Les lésions furent observées de 6 à 8 jours jusqu'a 10 à 14 jours après l'inoculation. Le début, le milieu et la fin de la période d'incubation ont été décrits en fonction de la température journalière et du nombre moyen d'heures avec une humidité relative $\geq$90%. Une fonction logistique a également été utilisée pour décrire l'apparition des lésions en fonction du nombre de jour après l'inoculation. Un modèle de prévisions contenant plusieurs équations décrivant les interactions entre l'inoculum, l'infection et la sporulation exprimés en équivalents pour l'environnement fut développé et validé. En général, le modèle a prédit adéquatement la progression de la maladie.

Un système prévisionnel fut développé pour déterminer la date du premier traitement fongicide en fonction de l'accumulation d'unités d'infection.

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Finally, I can not exclude my daughters Emilie and Nora-Audrée. Part of this achievement is due to their collaboration. To my family, especially my grand-mother and my sister, I like to express my gratitude for their encouragement, support, and love.

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GENERAL INTRODUCTION

Carrot (*Daucus carota* L. var *sativa*) is an important vegetable crop in Canada with an annual production in 1987 of 7772 ha, of which 3651 were in Quebec (Statistics Canada, 1988). In Quebec, carrot is one of the most important vegetable crops with an annual value of 17 million Can$ in 1986 (Statistics Canada, 1990). In 1989, carrots alone represented 17.7% of the vegetable production value in Quebec (MAPAQ, 1989).

Cercospora leaf blight of carrots, induced by *Cercospora carotae* (Pass.) Solh. (Chupp,1953) is a major foliar disease of carrot in the organic soil region of Quebec and Ontario (Crête, 1978, Sutton and Gillespie, 1979). A survey conducted in 1988 and 1989, of commercial carrot fields in the southwestern part of Montreal, indicated the presence of blight in 91 and 99% of the carrot fields with 99 and 92% of the plants diseased in 1988 and 1989, respectively (Arcelin and Kushalappa, 1991). Cercospora blight is also present in the United States and Ontario (Thomas, 1943). The fungus may attack any aerial part of the plant and induces dark brown circular lesions. Blighted leaves weaken the petiole and reduce the grip required for mechanical harvesters to pull the carrots, which increases loss during harvesting.

Economic pressure to reduce cost of crop production, development of fungicide tolerance in pathogen populations and public awareness concerning pesticides have modified the traditional approach to disease control. The actual challenge of plant pathologists is to develop effective methods of disease management while reducing fungicide applications as much as possible. Appropriate disease management strategies should be based on knowledge of the disease development. Strategies to manage polycyclic diseases, such as Cercospora blight, must be based on factors that reduce the amount of initial inoculum and the rate of disease progress (Fry, 1982).

During the harvest, carrot leaves (infected or not) are left on the ground and provide an excellent source of initial inoculum. Reduction of initial inoculum through sanitation (removal of plant debris) is not at this time economically feasible. Spores of C. carotae are wind disseminated, making it difficult to prevent the influx of inoculum from adjacent fields. Rate of disease progress is influenced by host resistance and the environment. Information of carrot resistance to Cercospora blight is lacking. So far, no resistant or tolerant cultivars are available in Canada. Lebeda et al. (1988), tested 142 cultivars of carrots for their field resistance to C. carotae. Infection was observed in all the cultivars and field resistance was observed in only 30 % of the cultivars tested.

Cercospora blight is weather dependent (Carisse and Kushalappa, 1990, Thomas, 1943, Hooker, 1944). A good understanding of the influence of the physical environment on disease development could make it possible to develop a forecasting model. Such forecasting system could be used by growers to schedule fungicides only when needed rather than on a regular basis. This would provide substantial savings in production costs while reducing the impact of fungicides on the environment.

This research was prompted by the unnecessary use of fungicide to control Cercospora blight of carrot under weather conditions that may not promote disease development. Because little information about Cercospora blight was available, this research was initiated to study its epidemiology.

The objective was to predict the effect of the physical environment on blight progress. This was accomplished through the development of a model that simulates the progress of Cercospora blight. The impact of weather parameters on various phases of Cercospora carotae life cycle was considered. From this, a fundamental forecasting model was developed to time the first fungicide application to control Cercospora blight of carrot.

This research is comprised of three parts. Part one

consisted of monocyclic studies and included experiments on the effects of the physical environment (temperature, leaf wetness, and relative humidity) on infection, sporulation, and the incubation period. The second part was a study of the polycyclic process and included collection of field data on disease progression and weather conditions, used to develop and validate a simulation model. Part three involved the formulation of a forecasting system.

LITERATURE REVIEW

1. CERCOSPORA BLIGHT OF CARROT

1.1 Etiology and symptoms

Cercospora blight is caused by Cercospora carotae (Pass) Solh. The disease was reported first in Italy in 1889 (Barnett, 1960, Sherf and Macnab, 1986). Since that time, it has been reported to occur worldwide, but it is more prevalent in north temperate areas (Barnett, 1960, Chupp, 1953). Cercospora blight was not reported in Canada until 1978 and , at that time, was considered to be of little economic importance (Crete, 1978, Sutton and Gillespie, 1979). Symptoms of the disease are easily mistaken for those caused by Alternaria dauci which was considered responsible for the carrot leaf blight disease. In 1988 and 1989, however, a survey of carrot fields in the organic soil region of Quebec, indicated that Cercospora blight was the most prevalent disease with more than 90% of the fields infected. The same

study revealed that Alternaria blight like symptoms were present on only 5% of the fields. Similar surveys, in other provinces of Canada have not been recently reported and the actual importance of the disease throughout Canada remains unknown.

On carrot, symptoms of C. carotae resemble those of A. dauci, however, the lesions are generally more regular in shape. They are circular, greyish to tan on leaves, darker on petioles and can be seen on any aerial parts of the plant. Under warm and humid conditions, the lesions enlarge rapidly, coalesce and often an entire leaf can be killed. During humid weather, the fungus can sporulate abundantly, lesions will then appear grey or silvery due to the presence of hyaline conidia. The germinated conidia penetrate the leaves through stomata within 36 to 72 hr depending on infection conditions (Thomas, 1943). No direct penetration has been reported. Apparently, stomata do not exert any attraction on germ tubes which grow at random until they come in contact with stomata (Angell and Gabelman, 1968, Thomas, 1943). Penetrating hyphae obstruct the stomatal cavities and generally invade the mesophyll and epidermal cells rapidly. Later, the advancing hyphae will disrupt the stomatal opening and will produce a cluster of conidiophores measuring 60 to 120 μm in width. Each conidiophore produces several crops of conidia. The conidia range from 40 to 110 μm in length and 2.2 to 2.5 μm in

width (Ainsworth et al., 1973, Thomas, 1943). At maturity, they are easily detached and wind disseminated.

1.2 Disease development

The fungus invades the leaf tissue, develops through the leaf and the disease becomes visible as necrotic lesions, which in turn produce more conidia to infect other leaves. The infection of carrot leaves by C. carotae depends mainly on temperature and leaf wetness duration. Prolonged periods of leaf wetness ($\geq$ 24 hr) at temperatures between 18 and 30 C are very favorable for infection (Carisse and Kushalappa, 1990).

During harvest, large amount of carrot debris is left on the ground or plowed-in. This provides good conditions for the pathogen to survive which may explain the presence of blight in almost all carrot fields (Arcelin and Kushalappa, 1991). The fungus presumably overwinters as mycelium or stroma produced in the plant debris (Messian et al., 1991). Mycelium is capable of growing in soil with high organic matter in the absence of carrot leaves (Thomas, 1943). Conidia produced from overwintering structures are probably the source of initial inoculum for early carrots (sown in early May) which in turn, serve as inoculum source for late carrots (sown in June). In Quebec, Cercospora blight generally becomes evident toward the end of July when the distance between rows of early carrots is almost covered by

the leaves. The disease is generally more severe in fields where blight was severe in previous years (Brodeur, 1989, personal communication).

1.3 Cercospora blight management

In commercial carrot fields the disease is controlled by routine applications of protectant fungicides (CPVQ, 1988). The recommendations include a two-year crop rotation, 4 to 7 fungicide applications at 7 to 12 day intervals, depending on rain, starting when the carrots are 10 to 15 cm tall. Recommended fungicides and dosage are MENZATE-D, DITHANE M-22, 2.25 Kg/ha and BRAVO 50-WP 2.5 Kg/ha.

The above recommendation is not based on the epidemiology of the pathogen and may result in unnecessary fungicide treatments because of inadequate timing which reduces their efficiency. To address these problems, and in general, to reduce the cost of pest control of vegetables produced on organic soils, an integrated pest management (IPM) program was initiated in 1982. This IPM program proposed an alternative to conventional spray schedules by recommending to initiate fungicide applications only when Cercospora blight reached a critical disease level (CDL). Scouts survey the carrot fields and advise growers when to start fungicide applications. The carrot fields are monitored for Cercospora blight by visual inspection. The proportion of intermediate carrot leaves with

one or more lesions is recorded. In this IPM program, fungicide applications are initiated at a disease incidence threshold of 80 and 50 % for the early and late carrots, respectively, with the subsequent applications being made at 7 to 10 day intervals (Boivin et al., 1990). The use of a CDL to time the first fungicide application has decreased the number of fungicide treatment substantially (Brodeur, 1989, personal communication). However, sampling Cercospora blight is time-consuming and considerably increases the cost of sampling. A technique of sequential sampling was thus developed to facilitate the estimation of Cercospora blight incidence and reduce the number of samples needed (Boivin et al., 1990). A method of forecasting the incidence threshold of Cercospora blight based on growth stage, days since sowing, and degree days has been proposed (Kushalappa et al., 1989).

The presence of this IPM program has slowly encouraged changes in grower attitude concerning disease control toward more ecological methods and makes them more receptive to new management strategies, including disease forecasting.

1.4 Selected epidemiological components of Cercospora blight.

Infection. Infection of carrot leaves by conidia of C. carotae is mainly influenced by the leaf wetness duration and the temperature, like several other Cercospora spp. (Beckman and Payne, 1983, Rathaias, 1976). A model describing infection as

a function of temperature and continuous leaf wetness duration has been developed (Carisse and Kushalappa, 1990). Similar models have been used with success as a basis for disease forecasting (Dainello, 1984, Eisensmith and Jones, 1981a, Mackenzie, 1981, Pennypacker et al., 1983, Sutton et al., 1986). Depending on the prevailing weather patterns encountered in the area for which the forecasting model is developed, it is often necessary to adjust the infection model. Under field conditions in Quebec, prolonged leaf wetness periods rarely occurred, leaves are usually wet at night and dry during the day. Leaf wetness periods are generally preceded and followed by periods of high relative humidity. Information on the effects of interrupted leaf wetness and of relative humidity on infection can significantly improve disease forecasting (Alderman et al., 1985, Eisemsmith et al., 1981b).

Sporulation. The production of inoculum is a key element of epidemics development. Knowledge of conditions that influence inoculum production may be important in timing fungicide application (Grove et al., 1985, Vincelli and Lorbeer, 1989). For most fungi, sporulation is influenced by the leaf wetness duration, relative humidity, temperature, and photoperiod (Cooperman and Jenkins, 1986, Fresland and Schodter, 1987, Grove et al., 1985). Like infection, sporulation can be described mathematically and used to develop forecasting

models (Lalancette et al., 1988).

Incubation period. The incubation period, defined as the time from infection to symptom expression, determines the number of pathogen generations possible within a season, thus the rate of disease development (Campbell and Madden, 1990). A short latency period will lead to many pathogen cycles, thus to faster disease progression. Information on the incubation period is used in forecasting models to predict when disease will become visible after an infection. Knowledge about the characteristics of incubation period including the pattern and temporal scale of lesion appearance is a prerequisite for the development of simulation models (Hau, 1988).

When relationships between environmental factors and selected processes of the pathogen life cycle are quantified, it becomes possible to develop simulation and forecasting models that could be used as research and management tools, respectively.

2. DISEASE MODELLING

"Plant disease epidemiology is the study of temporal and spatial changes of plant pathogen populations on a population of host" (Kranz, 1974). These populations are characterized by a number of elements which are related and form a structure, the pathosystem. These elements obey to biological

principles which can be described mathematically (Teng and Zadoks, 1980). According to these statements, disease modelling simply means testing the validity and reliability of analytical results by means of models. However, behind this approach, there is the real complexity of natural epidemics which are not necessarily the results of simple stimulus-response relationships, but also the result of interactions between several elements that initiate and influence epidemics. In this regard, systems analysis has been used to structure prediction models and thus, facilitate mathematical disease modelling.

The systems approach is a philosophy that takes a holistic view of a pathosystem. In practice, systems analysis starts with a conceptual definition of the system, followed by analysis of the structural elements, the system environment (physical elements), and the interrelations between structural and environmental elements (rules governing system behaviour) (Kranz and Hau, 1980, Seem and Haith, 1986, Teng, 1985). The systems approach was used in this research to structure the simulation model including the definition of objective, design of submodels, and validation of the complete model.

2.1 Simulation models

Mathematical modelling and simulation are being used to help understand complex pathosystems. There is no perfect way of classifying modelling approaches. However, several

researchers classified models as either analytic or simulation (Berger, 1989, Teng, 1985). Analytic models usually consist of a single equation with few biological parameters that can be solved mathematically. Simulation models consist of several sub-models linked together, each one representing a part of the system. Simulation models usually cannot be solved mathematically (Kranz and Royle, 1978).

Traditionally, analysis of disease progress with growth functions, such as, the logistic, Gompterz, Richards, or Weibull, has been employed to describe epidemic development over time (Berger, 1981, Campbell and Madden, 1990, Pennypacker et al., 1980, Van der Plank, 1963). However, there are several limitations inherent to this analytical approach. Among them, the lack of biological realism and the presence of simplifying assumptions. The difficulties associated with the use of simple equations in the explanation of the complexity and stochasticity of epidemics has lead to the development of simulation models which integrate the elements of the pathogen life cycle.

To develop a simulation model, the epidemic is generally divided into sub-systems based on the pathogen life cycle where each one is modeled separately. Simple models may consider only a few sub-systems such as infection, lesion growth, incubation, etc... (Hau, 1985). More complex models

may include further sub-division of processes like infection which can be divided into germination, penetration, and colonisation. Even when models involve many sub and sub-sub-divisions, all processes of the pathogen life cycle are not necessarily included. For example, the dissemination process is often ignored in disease modelling.

The epidemic is simulated by all sub-models organized in a logical (usually mathematical) way. This approach was critized by Van der Plank (1982) who argued that pathosystems are so complex that some degree of synthesis (using a single model to explain many components) is needed. He also stated that the use of many sub-models, each one having its own experimental error, may lead to the accumulation of a very large error component. Other modellers (Hau et al., 1985, Hau, 1988, Jerger, 1986, Teng, 1985) argued that because epidemics are so complex, simulation is the only possible way of understanding them. The argument of Van der Plank should certainly not be ignored, however, it was demonstrated that simulators can be excellent research tools and indirectly very useful for disease management.

The first conceptual models, such as EPIDEM (Waggoner and Horsfall, 1969), EPIMAY (Waggoner et al., 1972) have contributed to a better understanding of the complex relationships between variables of an epidemic. One of the

most successful forecasters, FAST, used to time fungicide applications against Alternaria solani on tomato and potato was derived from analysis of disease using simulation (Fry and Fohner, 1985, Madden et al., 1978). These models have the potential to assist in decision-making and research guidance because in these models the relationships between variables are quantified and it becomes possible to determine where more research is needed. The very complex simulators are rarely used directly for prediction. However, when a simulator is available it is possible to study the effect of combined factors on disease development. The epidemiology of potato late blight (Phytophthora infestans) was studied using a simulator (Bruhn and Fry, 1981, Fry et al., 1983). The simulator included mathematical models that simulate the effects of the environment, fungicides, and cultivars on the disease development. The model includes sub-models for the major phases of the pathogen life cycle and operates on a daily basis. Using this model it was possible to compare different management strategies.

Often, only very complex, mostly computerized models are considered to be simulators, but in fact the distinction is not in the complexity but rather in the approach used to develop them. The model presented here is a simple simulation model including sub-models for infection, sporulation and the incubation period. The population of lesions of C. carotae

resulting from several infections were predicted (or projected in time) using projection matrices to determine the amount of inoculum that will be available for further infections. Projection matrices have been used mainly for animal and insect population dynamic studies (Caswell, 1989). In this work it was found to be a valuable alternative to dynamic simulation in describing the population of lesions of C. carotae. This model was developed so that it can be coupled with other models simulating fungicide effects and carrot growth. The resulting model can be used to study different management strategies.

2.2 Disease forecasting models

Programs for fungicide applications based on a calendar are straightforward to develop and easy to implement. However, programs in which fungicides are applied only when needed are more difficult to develop and implement because the growers must be able to predict disease with accuracy and confidence. A forecaster has to be reliable and practical for growers to adopt it (Fry, 1982).

Various approaches have been proposed and reviewed for disease prediction, disease warning, and development of forecasting models (Bourke, 1970, Fry and Fohner, 1985, Krause and Massie, 1975, Wagonner, 1960, Zadoks, 1984). Development of forecasters is not justified in all situations. It should

be considered as one approach to disease management which often gives better results when used with other methods (eg. resistant cultivars). Almost all forecasters are associated with the use of fungicides. Forecasts are useful for sporadic diseases or endemic diseases with varying severity levels. If a disease is always important or favorable conditions always present, control measures are always needed and forecasts will not improve disease management. Effective technology for disease control should also be available for forecasts to be effective, and finally forecasts must be accepted by the growers. There are two main approaches used for the development of forecasting models, the empirical and fundamental approaches.

Empirical models are developed from observations and analysis of historical data on disease and factors influencing disease development. Generally, these models required many years of observation in order to be reliable. Development of empirical forecasts consists essentially of establishing correlations between disease levels and some variables, mostly meteorological. Empirical models are either qualitative or quantitative. Qualitative models do not involve the use of statistics but rather rules or prediction criteria. On the other hand, quantitative models are developed based on statistical analysis (Campbell and Madden, 1990). These forecasting models may be very simple, including only one

equation that predicts disease severity based on few variables. Such a model was developed to predict stripe rust (Puccinia striiformis) on winter wheat (Coakley et al., 1982). It was based on data accumulated over several years, from many cultivars, and locations. Disease intensity was correlated to environmental factors and negative degree-day (accumulation of winter temperatures) was found to be the best predictor and a simple first order polynomial was developed.

Fundamental forecasting models are developed from experimentation. The experiments may be conducted in laboratories, growth chambers or in the field. The experiments are designed starting with hypotheses about disease development and use formal statistical methods (including experimental design). Experiments are conducted to establish relationships between environment, host and disease. Most of the fundamental forecasts are based on different components of the pathogen life cycle as influenced by the environment. For many pathogens, infection alone is a good predictor of disease progression (Eisensmith and Jones, 1981a). Whether or not a forecasting model for a given disease should include models for infection only or other components depends on the influence of each component on the overall disease progression in a given locality. For example, if the meteorological conditions influencing sporulation or if the amount of spores produced do not vary within a cropping

season then the sporulation would not be a good predictor. The same principle applies to the other components of epidemic. Fundamental forecasting models can be very simple, based on a single equation, or more complex, including several equations for sub-processes. Simple models are usually based on infection only whereas complex ones may include certain combinations of infection, sporulation, and dissemination processes, and infectious periods.

The classification of disease prediction models as either empirical or fundamental does not apply to all systems. Some fundamental models have their origins as empirical systems. Independent of the approach used in developing a forecaster, the appropriate type of forecasting model depends on the characteristics of the disease to be controlled (Fry, 1982). When one component of the epidemic contributes to most of the variation in disease occurrence, the forecasting model should be based on this component. Forecasting models may be classified based on the epidemiological characteristics of the disease (Campbell and Madden, 1990, Fry, 1982). Following this classification, forecasts can be grouped in three categories 1) forecasts based on initial inoculum or initial disease; 2) forecasts based on inoculum and environment; 3) forecasts based on both initial and secondary inoculum.

Forecasting systems can be used for two different

purposes. First, forecasting model may be used to time the initial spray and/or subsequent sprays (interval between sprays). A classical example is the forecasting of the seasonal maturation of Venturia inequalis ascospores (MacHardy and Gadoury, 1985). Other forecasts are used to time all fungicide applications. Examples of this type include systems used for potato late blight (Phytophthora infestans) and leaf spot of peanuts (Cercospora spp.) (Krause and Massie, 1975, Parvin et al., 1974)

To use a forecasting system efficiently it is important to understand how the system operates and to be aware of the limitations of the system and risks inherent to forecasts. Weather-based forecasting models based on infection usually predict infection periods that have already occurred, and rarely the future infections. These forecasters are called warning systems. When conditions known to be conducive to infection are monitored the infection has already started, unless the favorable conditions are based on forecasted weather rather than on the past events. These predictions are useful when fungicides with therapeutic activity are applied. Another approach has been to forecast the need for fungicide applications based on the accumulation of severity values. Examples of forecasters of this type are the models used to time fungicide applications to control cherry leaf spot, and Botrytis leaf blight of onion, (BOTECAST)(Eisensmith and

Jones, 1981b, Sutton et al., 1986). Although this approach has been successful, it was criticized because applications of protectant fungicides after infection may be ineffective, and for many diseases therapeutic fungicides are not available or too expensive. Despite these limitations, this type of forecasting model is very common and can be useful when adjusting the action threshold at which the fungicide treatments must be initiated. In regard to that, forecasts based on sporulation (inoculum production) and forecasted conditions favorable for infection were developed to anticipate infection, so that the fungicides can be applied before infection occurs. Such a system was developed for Botrytis leaf blight of onion (BLITE-ALERT) (Vincelli and Lorbeer, 1989).

Many predictive models have been developed during the past two decades but only a few are used in the field to manage disease. The reason for this may be that some models are not reliable when used in the field, others are too complex to be used by the growers, and some require sophisticated instrumentation. However, some forecasting models have been successfully used such as for cherry leaf spot (Eisensmith and Jones, 1981b), apple scab (Jones et al., 1980), potato late blight (Krause et al., 1975, Nutter and Machardy, 1980), tomato early blight (Madden et al., 1978, Pennypacker et al., 1983) and peanut leaf spot (Parvin et al.,

1974, Phipps and Powell, 1984). These models have increased the knowledge of the biology of these pathogens, have proven to be effective in lowering disease control costs and have reduced the impact of fungicides on the environment.

Cercospora blight of carrot can be managed with fewer fungicide applications when the first treatment is based on disease incidence thresholds. This method however, depends on field monitoring of disease. The forecasting model for Cercospora blight was developed to eliminate the need for disease monitoring. This forecasting model predicts the onset of epidemics and can be used to time the initial fungicide application only. The onset of Cercospora blight epidemics varied from late June to early August (unpublished data from the IPM program of southwestern Montreal). Forecasting the time of onset of the epidemic phase might save a considerable number of fungicide applications in some seasons. This forecasting system, developed fundamentally, uses monitored weather data to calculate daily infection index from which a cumulative blight severity value is computed. The decision to spray or not is made based on the cumulative blight severity value cumulated since crop emergence.

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STUDIES ON MONOCYCLIC PROCESS

PREFACE TO CHAPTER I

Most prediction models for foliar pathogens include a function for the infection process and several forecasting systems are entirely based on infection. A previous study of infection of C. carotae on carrot showed that a minimum of 24 hr of leaf wetness at temperatures ranging from 20 to 28 C is required to induce significant levels of infection under growth chamber conditions (Carisse and Kushalappa, 1990). However, under field conditions in Quebec such long periods of leaf wetness rarely occur, and in spite of this the disease often reachs epidemic levels. Carrot leaves are often wet at night and dry during the day, and almost all wet periods are preceded and followed by periods of high relative humidity. The presence of disease when only short periods of leaf wetness are available suggest that high relative humidity or interrupted leaf wetness may be sufficient to allow spore germination and penetration, as has been reported for other pathogens.

Information on the effect interrupted leaf wetness and relative humidity on infection can greatly improve disease prediction based on continuous leaf wetness.

Carisse, O., and Kushalappa, A. C. 1990. Development of an infection model based on temperature and duration of leaf wetness for Cercospora carotae on carrot. Phytopathology 80:1233-1238.

STUDY I

INFLUENCE OF INTERRUPTED WET PERIODS, RELATIVE HUMIDITY AND TEMPERATURE ON INFECTION OF CARROTS BY Cercospora carotae.

ABSTRACT

Carrot leaves (Daucus carotae L. sativa) were inoculated with a conidial suspension (10^4 conidia/ml) of Cercospora carotae and then subjected to interrupted and continuous wet periods of various durations, and to combinations of relative humidity (84, 88, 92, 96 and 100 % RH) and temperature (16,20 ,24, 28, and 32 °C) with and without an initial wet period of 6 hr. Number of lesions per leaf decreased with increasing length of dry period for dry periods greater than 3 hr. However, a dry period of 3 hr with initial and final wet periods of 24 and 12 hr, respectively, resulted in more lesions per plant than the corresponding continuous wet period (39 hr). The number of lesions increased with increase in initial wet period duration for a fixed dry interruption period of 6 hr. For all temperatures very few lesions developed at 84% RH. However, the number of lesions increased rapidly with increase in percent RH greater than 84%. In general, the plants exposed to an initial wet period of 6 hr developed more lesions than those exposed to RH only. The number of lesions per plant was transformed to proportion of those at continuous wet period, and to proportion of maximum number of lesions for the experiments on interrupted wet period and RH,

respectively. Polynomial models were used to describe the effects of dry period durations, initial wet period durations, and of RH and temperature on infection.

INTRODUCTION

The fungus Cercospora carotae (Pass.) Solh. is found in almost all carrot fields (organic soil) in Quebec and a very common in Ontario (Calpouzos and Stallknecth, 1965, Sutton and Gillespie, 1979). The fungus is also present in the United States (Thomas, 1943). The fungus attacks only the aerial parts of the plant. The economic loss due to this fungus occurs during mechanical harvesting when the diseased leaves and petioles break-off easily, making it difficult to pull the roots from the ground. In Quebec, Cercospora blight is controlled by weekly applications of protectant fungicides. However, not all of these fungicide applications are needed and the best time to initiate fungicide applications is not yet established. The combined effects of constant temperature and continuous leaf wetness on infection of carrots by C. carotae has been studied and a mathematical model to predict infection as a function of temperature and leaf wetness duration has been established (Carisse and Kushalappa, 1990). In carrot fields in Quebec, long periods of leaf wetness rarely occur, and in spite of this the disease often reaches epidemic levels. The presence of disease when only short periods of leaf wetness are available suggests that interrupted leaf wetness or periods of high humidity may be

sufficient for spore germination and penetration, as was reported for other pathogens (Alderman et al., 1985, Arauz aand Sutton, 1989, Bashi and Rotem, 1974, Esensmith et al., 1981, Elliot, 1998). The Cercospora spp. are known for their tolerance to drying (Good, Zathureczky, 1967). In addition, studies on C. beticola (Rathaias, 1977, Rathaias, 1978) indicated that nocturnal wetting and diurnal drying may be more favorable for spore germination and penetration than continuous wetting.

When infection models based on continuous wetness periods are used for disease prediction, it becomes difficult to interpret the results of cyclic wet-dry-wet periods. In such cases the two wet periods can be considered as one continuous wet period if the pathogen growth has momentarily stopped during the dry period and resumed with wet conditions. However, if the pathogen continues to grow or if the dry period has a detrimental effect on pathogen growth, the wet periods interrupted by a dry period should be considered as one continuous wet period corrected for the effect of the dry period.

In carrot fields, when the rows are almost covered by the carrot leaves, a microclimate with long periods of high relative humidity could occur in the absence of leaf wetness. In such situations an infection model based only on leaf wetness may underestimate infection. Studies on the influence of humidity and interrupted leaf wetness periods on infection could be helpful in refining the original model.

The objectives of this study were, first to examine the

influence of dry period and of initial wet period durations during interrupted wet period on infection. Second, to study the combined effects of humidity and temperature on infection with and without short initial wetness period. Third, to establish infection criteria and to develop mathematical models describing the effect of these factors on infection so that these models (and criteria) can be eventually used to correct the original infection model based on temperature and leaf wetness duration (Carisse and Kushalappa, 1990).

MATERIALS AND METHODS

Plant production. Carrot plants (cv. Dagger) were seeded in 13-cm diameter pots with 3:1 (v/v) mixture of organic soil (27-30% organic matter) and perlite. Fertilizers (200 ppm of 19-52-19 N-P-K) were applied every 2 days. For experiments on interrupted leaf wetness the plants were grown in a growth chamber maintained at 20 C and 12 hr of light per day (200 μE/m2/s). In experiments on temperature and relative humidity the carrot plants were grown in a greenhouse adjusted at 22 C $\pm$ 2 C and 12 hr of light per day (200-300 μE/m2/s). All experiments were conducted with 5-wk-old carrot plants of the cultivar Dagger (Calpouzos and Stallknecth, 1965).

Inoculum production and inoculation. A single-spore culture of C. carotae was maintained on carrot leaf infusion agar (CLA) at 26 C under 12 hr light per day until required for inoculation

(Beckman and Payne, 1983, Calpouzos and Stallknecth, 1965, Carisse and Kushalappa, 1989). Fresh cultures were obtained by successive inoculations of carrot leaves and reisolations from infected leaves (Carisse and Kushalappa, 1989). Conidia were harvested from 12-day-old cultures using a solution of 0.01% Tween 80. The concentration of the conidial suspensions was adjusted to 10^4 conidia/ml using an haemacytometer. Percent spore germination was estimated for all inoculations by spraying three water-agar plates with the conidial suspension used for the inoculations. One agar plate was sprayed at the beginning, one in the middle, and one at the end of each inoculation. The number of germinated spores was counted 3 hr after spraying the plates.

At the sixth leaf stage, the second and third true leaves from the bottom were tagged. The tagged leaves were inoculated on both surfaces until runoff using an artist air brush (Badger-350) operated at 100 kPa air pressure. Immediately after inoculation, the plants were placed in a mist chamber or a humidity controlled growth chamber (model PGW36 M10, with RH controlled by bypass dehumidification) kept at the required temperature. Because carrot leaves take less than 10 min to dry off in a growth chamber, drying time was not included in the wetness duration.

The effects of interrupted leaf wetness, and of temperature and relative humidity on infection were examined in four

experiments. The first two experiments were conducted to investigate the effects of dry period and duration of initial wet period on infection, respectively. The third and fourth experiments were conducted to examine the effects of temperature and relative humidity on infection, without and with an initial leaf wetness period, respectively. All four experiments were arranged as a completely randomized design, conducted twice, and each treatment included four experimental units (four plants, two leaves/plant). Infection was quantified by counting the number of lesions on each inoculated leaf at 2-day intervals starting 10 days after inoculation and continuing until two similar readings were obtained. The numbers of lesions on each of two inoculated leaves per plant were summed and the total number of lesions per plant was used in all analyses.

Influence of dry period and duration of initial wet period on infection. After inoculation with the conidial suspension of C. carotae, the plants were subjected to either continuous wet periods or interrupted wet periods. The interrupted wet period consisted of an initial wet period followed by a dry period and a final wet period. Dry period was defined as percent RH less than 65% $\pm$ 5%. Humidity during the dry period was monitored using a data logger (CR-10, Campbell Scientific Canada Corp.).
The interrupted wet period consisted of initial and final wet periods of 24 and 12 hr, respectively, separated by a dry period of 3, 6, 12, 18, 24, 30, and 36 hr. Durations of the continuous

wet periods were 36, 39, 42, 48, 54, 60, 66, and 72 hr. In a second experiment, the effects of various durations of initial leaf wetness period on infection were tested. After inoculation the plants were subjected to an initial wet period of 0, 3, 6, 12, 18, 24, 30, 36, and 42 hr, followed by a fixed dry period of 6 hr and a final wet period fixed so that the total length of the cycle was equal to 48 hr. Thus for each interrupted wet period treatment there was a corresponding continuous wet period treatment with the same total duration.

Data analysis. The total number of lesions per plant was transformed to proportion of number of lesions obtained under continuous wet period (PCWP) calculated as follows:

$$PCWP = \frac{\text{number of lesions in interrupted wet period}}{\text{number of lesions in corresponding continuous wet period}} \quad (1.1)$$

The proportion of number of lesions (PCWP) for each interrupted wet period treatment was used to evaluate relationships between the continuous wet period and interrupted wet period of the same duration including dry period. The number of lesions per plant was used to compare the lesion production in interrupted wet period and in 36-hr continuous wet period. The number of lesions obtained for the seven interrupted wet period treatments were compared to the number of lesions resulting from 36 hr of continuous wetness to determine if the infection can be attributed only to the initial and final wet periods. F-test was

used to determine if pooling of the two experimental trials was allowed. The PCWP for all treatments (separately for each experiments) was subjected to analysis of variance and regression analysis to find equations that described best the PCWP as a function of dry period duration and as a function of initial wet period duration.

Influence of temperature and RH on infection. After inoculation with the conidial suspension of C. carotae, the plants were placed in four chambers all adjusted to a specific temperature and to relative humidities of 84, 88, 92, 96 % ± 1.5%. Humidity in the chamber was continuously monitored using a data logger (CR-10, Campbell Scientific Canada Corp.) and the growth chamber sensor. The wet treatment was created by enclosing the plants in plastic bags. After an infection period of 72 hr, all the plants were returned to the greenhouse until symptom development. This procedure was repeated for temperatures in the RH controlled chambers of 16, 20, 24, 28, and 32 C (in a random order).
A second experiment was designed to study the effects of temperature and relative humidity when the plants were exposed to a short leaf wetness period before the exposure to various levels of RH (84, 88, 92, 96% RH) and leaf wetness. Immediately after inoculation the plants were enclosed in plastic bags to maintain leaf wetness. After 6 hr the plastic bags were removed except for the leaf wetness treatment in which plants were retained in the plastic bags. After a total of 72 hr, all the plants were

returned to the greenhouse until symptom development. This procedure was repeated for temperatures in the RH controlled chambers of 16, 20, 24, 28, and 32 C (in a random order).

Data analysis and model development. The total number of lesions per plant was transformed to proportion of maximum number of lesions (PML) calculated as follows:

$$PML = \frac{\text{number of lesions observed}}{\text{maximum number of lesions observed}} \qquad (1.2)$$

The PML obtained for all humidity treatments were subjected to analysis of variance and linear regression analysis to find equations that described best the PML as a function of the percent RH and temperature, without and with an initial wet period of 6 hr.

RESULTS

Influence of duration of dry periods on infection. Interrupted wet periods resulted in significantly (P=0.05) fewer lesions per plant than continuous wet periods (Table 1.1). An increase in the duration of dry periods, between 24 hr of initial and 12 hr final wet periods, significantly reduced infection as compared to those at a corresponding duration of continuous wet period, except for the 3 hr interruption which resulted in more lesions than the corresponding continuous wet period treatment (39 hr continuous wet period) (Table 1.1). The number of lesions

produced under a 36 hr dry period was 29% of the number of lesions obtained at the corresponding continuous wet period treatment (72

Table 1.1 Influence of dry period duration during interrupted wet period and continuous wet period on infection of carrot leaves by conidia of Cercospora carotae.

Treatment				Lesions per plant[x]	
IWP[v]			CWP[w]		
Wet (h)	Dry (h)	Wet (h)	Wet (h)	IWP	CWP
24	0	12	36	54.6c[y]	54.6
24	3	12	39	92.1a	61.4**[z]
24	6	12	42	69.4b	91.2**
24	12	12	48	65.9bc	101.2**
24	18	12	54	71.7b	122.9**
24	24	12	60	72.7b	137.6**
24	30	12	66	53.0c	144.4**
24	36	12	72	55.5c	188.2**

[v] The interrupted wet period (IWP) consisted of an initial and final wet periods of 24 and 12 h, respectively, separated by a dry period of various lengths.

[w] The continuous wet period (CWP) duration consisted in continuous leaf wetness period equal to the total duration of the coresponding IWP treatment.

[x] Means of two trials with four plants per trial, two leaves per plants.

[y] Values followed by the same letter are not significantly different according to the Waller-Duncan K-ratio T test (K-ratio=100).

[z] Mean values between IWP and CWP colums differ significantly (P=0.01,**) according to the least significant difference test.

hr continuous wet period) for both trials. Mean number of lesions per plant from interrupted wet period treatments with dry periods ranging from 3 to 24 hr were significantly higher than the mean number of lesions obtained from 36 hr continuous wetness except for the treatment with 12 hr dry period (24-12-12). However for dry periods of 30 and 36 hr the number of lesions was not significantly different. Analysis of variance indicated that dry periods of 6 to 36 hr have a significant effect on lesion production (P=0.0001) and a first-order polynomial model explained the relationships between dry period durations and PCWP (R^2 = 0.77) (Fig. 1.1).

Influence of initial leaf wetness duration on infection. Plants exposed to an initial wet period of 0 to 24 hr had fewer lesions than the control (48 hr continuous wet period) (Fig. 1.2). The proportion of number of lesions at continuous wet period (PCWP) increased linearly with increase in initial wet period (Fig. 1.2). The interrupted wet period treatments with no initial wet period produced 8 and 18% of the number of lesions obtained at 48 hr continuous wet period for the first and second trial, respectively. Analysis of variance indicated that the initial wet period duration has a significant effect on infection (P=0.0001) and a first-order polynomial model explained the relationship between dry period durations and PCWP (R^2=0.74)(Fig. 1.2). For these two experiments the F-test indicated no significant difference (P > 0.05) between the two experimental

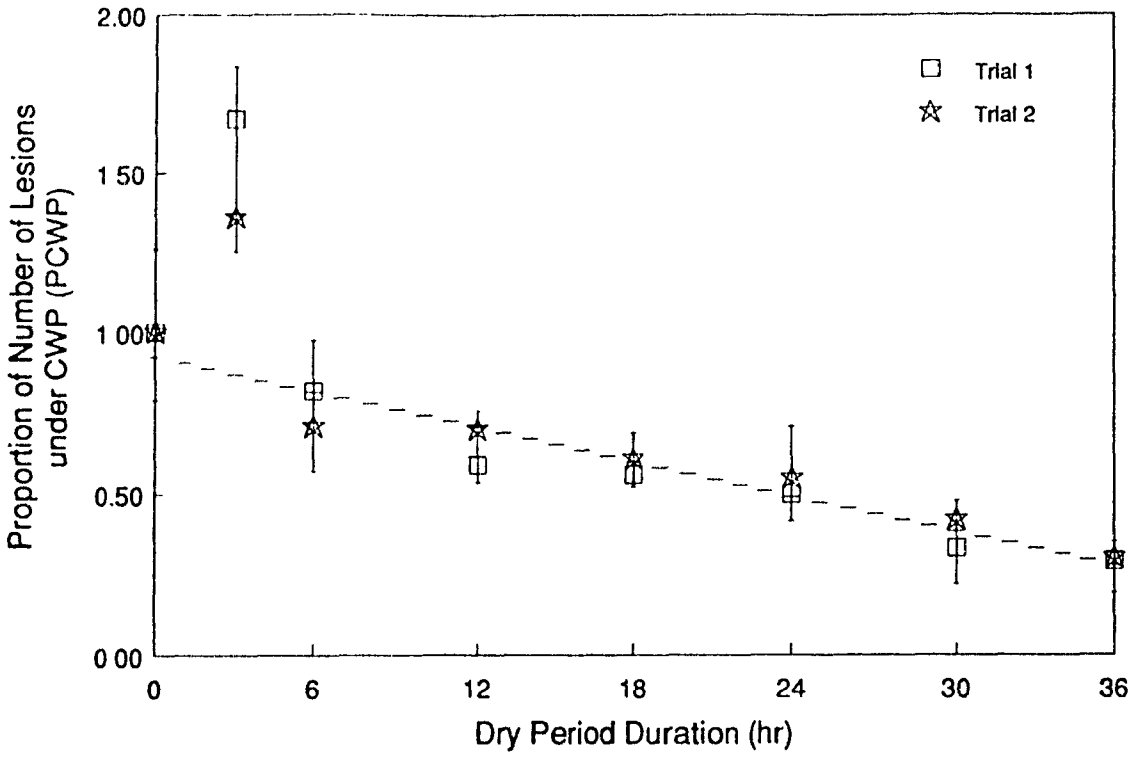


Fig. 1.1. Influence of the duration of dry periods on infection of carrot leaves by Cercospora carotae. The regression equation Y = 0.9243 - 0.018X, R^2 = 0.77, where, Y is proportion of number of lesions obtained under continuous wetness, X is the dry period duration between the 24 pre- and 12 hr post-dry wet periods. R^2 is the coefficient of determination. The dashed line represents the regression line and the error bars represent the range of observed values. Each point is an average of observations made on four plants, two leaves per plant. The 3 hr dry period treatment was not included in the regression analysis.

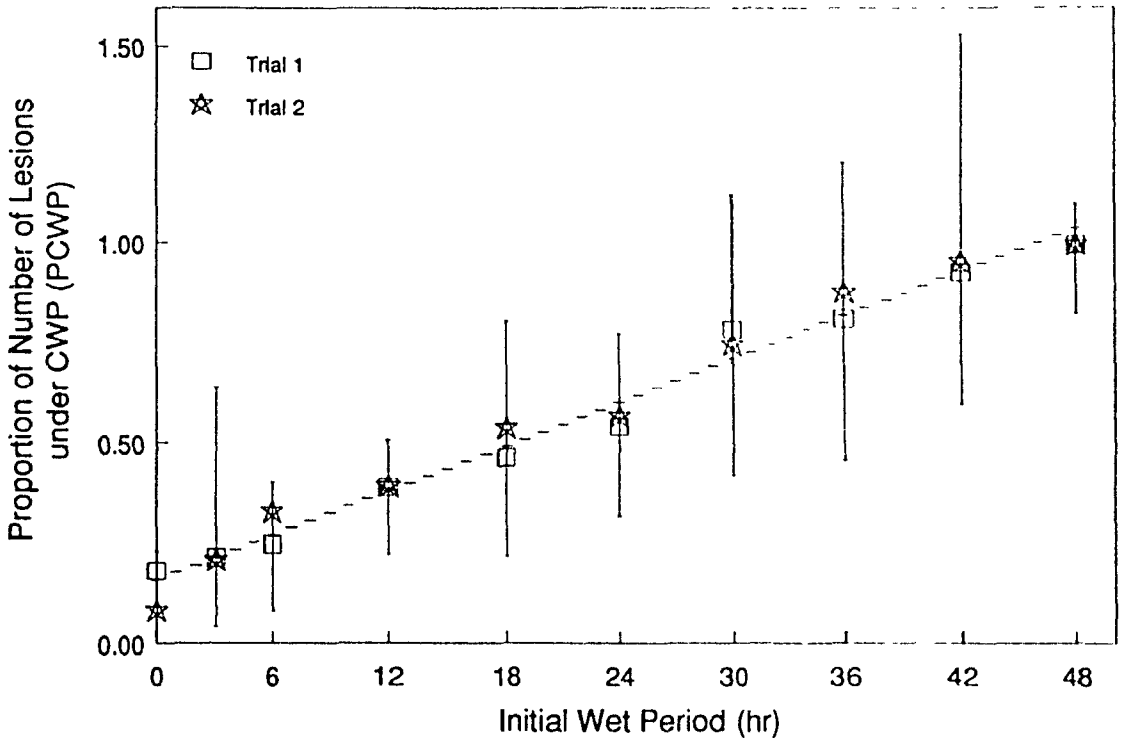


Fig. 1.2. Influence of the duration of initial wet periods on infection of carrot leaves by Cercospora carotae. The regression equation is $Y = 0.1593 + 0.0184X$, $R^2 = 0.74$, where, Y is proportion of number of lesions obtained under 48 hr continuous wetness, X is the duration of the initial wet period. All initial wet periods were followed by a 6 hr dry period and then by a wet period to make-up the total of 48 h. R^2 is the coefficient of determination. The dashed line represents the regression line and the error bars represent the range of observed values. Each point was an average of observations made on four plants, two leaves per plant.

trials.

Influence of relative humidity and temperature on infection. In general, the number of lesions per plant increased with increase in humidity levels for all temperatures (Fig. 1.3A-B). The relationship between relative humidity and PML was not linear for the temperatures studied. Maximum number of lesions was reached under leaf wetness for all temperatures. The number of lesions increased with increase in temperatures ranging from 16 to 28 °C and decreased at 32 °C. For the plants exposed to an initial wet period of 6 hr (Fig. 1.3B) the number of lesions increased rapidly between 84 and 96% RH but the increase was rather slow at 96 and 100% RH (leaf wetness). No lesions were observed at 84% RH (Fig. 1.3A-B) and only few lesions were observed at 88% RH (Fig. 1.3A) when plants were not exposed to an initial wet period. However, the number of lesions increased rapidly between RH of 88 and 100%. In general, number of lesions observed on the plants exposed to an initial wet period (Fig. 1.3B) was higher for all relative humidity levels and for all temperatures than those for the plants exposed to humidity only (Fig. 1.3A). Percentage of spore germination of the inoculum used in these experiments varied from 92 to 97% and the F-test indicated no significant difference ($P > 0.05$) between inocula. Therefore, the effect of inoculum associated with the temperature treatment (different inoculum was used for each temperature) was considered

negligible and data from all temperatures were pooled. Influence of relative humidity and temperature on infection was described by the following equations for experiments without and with initial wetness, respectively:

$$\text{Arcsin } \sqrt{PML} = 16.334-0.6844H+0.6569\times10^{-4}H^2+2.378T-0.3853T^2+ 0.0142T^3-0.1527\times10^{-4}T^4+0.0542HT-0.1111\times10^{-3}HT^2 \quad (1.3)$$

$$\text{Arcsin } \sqrt{PML} = -26.862+0.0575H-0.2443\times10^{-3}H^2+3.9208T-0.4144T^2+ 0.0141T^3-0.1537\times10^{-4}T^4+0.0384HT-0.7872\times10^{-4}HT^2 \quad (1.4)$$

where PML is the proportion of maximum number of lesions observed, H is percent relative humidity, and T is temperature (C) (Fig. 1.4A-B). The polynomial model accounted for 97 and 96 percent of the variation in proportion of maximum number of lesions for experiments without and with initial wetness, respectively. Both models indicated a quadratic relationship between H and PML, and a quartic relationship between T and PML. The interaction between H and T and between H and T^2 were found to be significant. Although the coefficient of determination was high for both models, the two models overestimated the proportion of maximum number of lesions at 20, 24, and 28 C and RH of 96 and 100% (Fig. 1.4A-B). The lack of a definite pattern of distribution of residuals indicated that the models are appropriate.

Fig. 1.3. Observed proportion of the maximum number of lesions of Cercospora carotae observed on carrot leaves at various temperatures and relative humidities. In A) the plants were exposed to continuous humidity. In B) the plants were exposed to 6 hr of leaf wetness before being subjected to various levels of relative humidity and the maximum number of lesions observed at 28 C under leaf wetness was 550 and 492 for the first and second trial, respectively. Each point was an average of observations made on 10 plants (two experimental replication, five plants/replication, two leaves/plant).

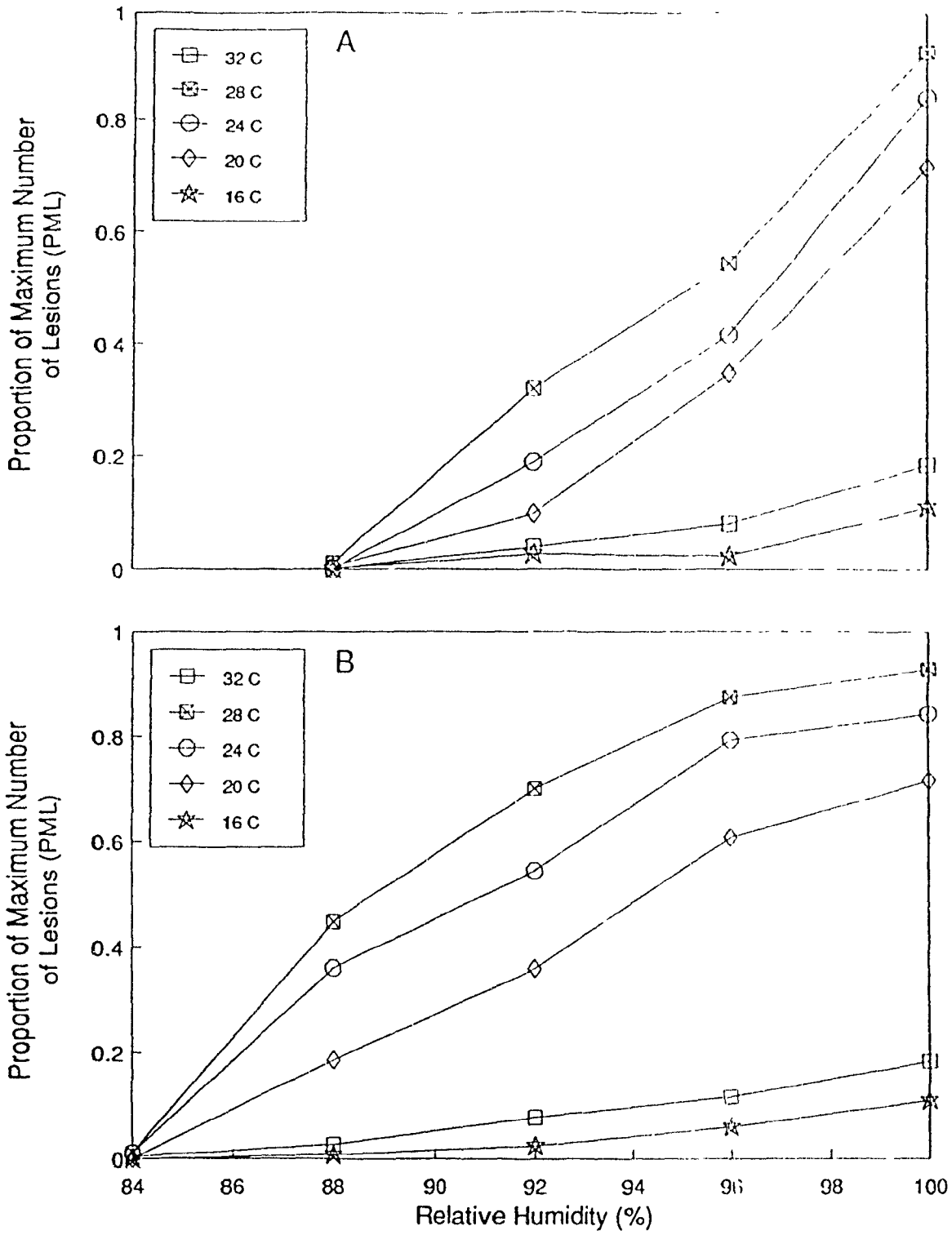
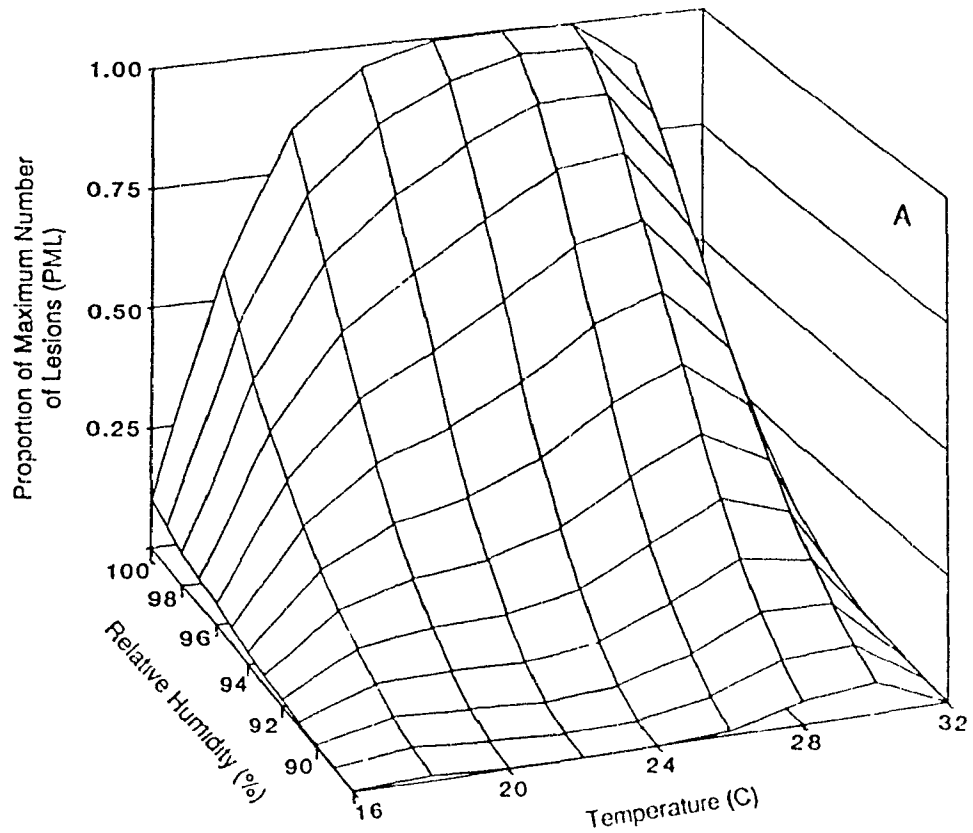
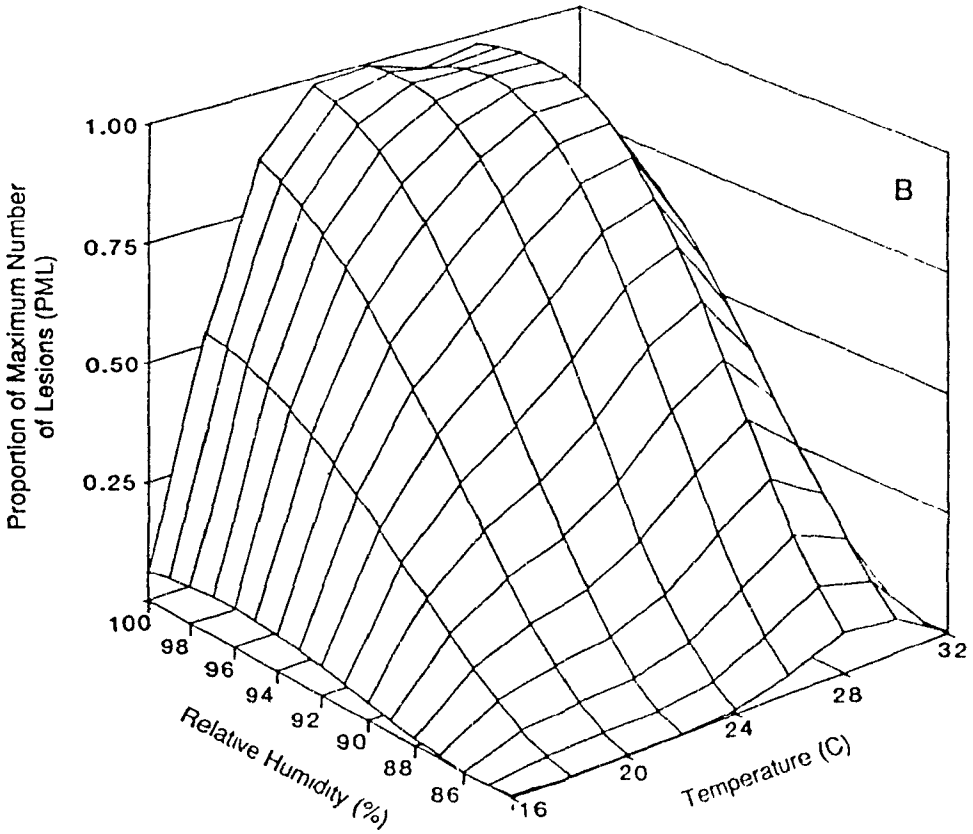

A
B
32 C
28 C
24 C
20 C
16 C
Proportion of Maximum Number of Lesions (PML)
Relative Humidity (%)
0
0.2
0.4
0.6
0.8
1
84
86
88
90
92
94
96
98
100

Fig. 1.4. Proportion of the maximum number of lesions of Cercospora carotae predicted by the polynomial model as a function of temperature and % relative humidity. In A) the predicted values were calculated using the equation 1.3 (see text). In B) the predicted values were calculated using the equation 1.4 (see text).


A
Proportion of Maximum Number of Lesions (PML)
1.00
0.75
0.50
0.25
100
98
96
94
92
90
Relative Humidity (%)
16
20
24
28
32
Temperature (C)


B
Proportion of Maximum Number of Lesions (PML)
1.00
0.75
0.50
0.25
100
98
96
94
92
90
88
86
Relative Humidity (%)
16
20
24
28
32
Temperature (C)

DISCUSSION

The interrupted wet period reduced infection significantly. This warrants incorporation of the effect of interrupted wet period into the infection model based on duration of continuous leaf wetness (Carisse and Kushalappa, 1990). Plants given a 24 hr initial wet period followed by 3 to 24 hr dry periods have more lesions per plant than those exposed to 36 hr continuous wet period except for the 12 hr dry period for which the number of lesions was not significantly different. However, for dry interruptions of 30 and 36 hr the number of lesions per plant was not significantly different than for the 36-hr continuous wet period. These data suggested that germinated spores can survive dry periods and resume growth when wetted again. These observations were similar to those reported for Cercospora species on other plants (Rathaias, 1977). Goods and Zathureczky (1967), demonstrated that spores of C. musae have a considerable ability to tolerate drying. Increased infection observed under 24-3-12 wet-dry-wet period compared to 36 hr continuous wet period remain unexplained and histopathological studies are needed to fully understand the mechanism of spore germination and penetration by C. carotae. This phenomenon can be partially explained by the presence of large droplets of water on the leaf surface which may have reduced germ tube contact with the leaf surface. It is not quite clear whether the 3-hr dry period has stimulated the infection process, and the effect gradually reduced with increase in dry period up to 24 hr, or whether the

continuous wet period is not the optimum. The 6 hr dry period was then used to examine the effects of initial wet period durations on infection. The results indicated that dry interruptions occurring after an initial wet period of 24 hr or less resulted in fewer lesions than after 24 hr. These results supported conclusions of a previous experiment on infection of carrot leaves by C. carotae (Carisse and Kushalappa, 1990) which indicated that a minimum of 24 hr of leaf wetness was required to induce infection in growth chamber experiments. Infection under interrupted wetness by a fungal pathogen is due either to rapid germination and penetration or to the capacity of the germinating spores to survive intermittent drying. Although no detailed studies of germination, penetration, and survival of spores were done, our results suggest that it is probably the ability of spores of C. carotae to survive drying rather than rapid germination and penetration that is responsible for successful infection under interrupted wet period. In practice, it means that two wet periods separated by a dry period of $\leq$ 12 hr should be considered as one infection period. Also, the cumulative effect must be calculated for wet-dry-wet periods, if the temperature is favorable.

The highest number of lesions was obtained under leaf wetness, indicating that leaf wetness is more favorable for infection than relative humidity greater than 84%. The results also indicated that high humidity reduced infection but, as for other pathogens, high relative humidity (84%<RH<100%) is

sufficient to allow infection (Rathaias, 1976, Rathaias, 1977, Reuveni and Rotem, 1974, Shew et al., 1988). A detailed study of the infection process of C. zea-maydis in corn leaves revealed that high relative humidity may be more favorable to spore penetration than free-water, which reduces tropistic response toward stomata, appressorium formation and subsequent penetration (Beckman and Payne, 1982). A similar trend was not observed for C. carotae. Decrease in relative humidity level caused rapid reduction in infection, even though the reduction was less rapid when the plants were exposed to a short initial wetness period (6 hr).

Under field conditions in Quebec, periods of leaf wetness are usually preceded and followed by periods of high relative humidity (> 90%). A short period of leaf wetness (< 6 hr) may be sufficient to trigger infection (spore germination and penetration) and subsequent high humidity probably supports the completion of remaining phases of the infection process (colonization).

An understanding of the influence of interrupted wet periods and relative humidity should improve prediction of infection of carrots by C. carotae and improve the forecasting. Two or more wet periods ($\leq$ 24 hr), for each of which no infection is predicted, can be added and the predicted infection can be corrected to account for the duration of the dry period and of the initial wet period. The wet period can also be extended when the relative humidity is high (>90%).

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PREFACE TO STUDY II

According to Zadoks and Schein (1979), the four factors that condition epidemic development are 1) the length of the latency period; 2) the number of spores produced per lesion; 3) the length of the infectious period; and 4) the effectiveness of inoculum (proportion of spores that initiate new infections). Sporulation is thus one of the major components of epidemics. Production of infection units and pathogen survival are critical and influence the rate of disease development in the field.

For most fungi, sporulation is influenced mainly by the leaf wetness and/or high relative humidity duration, temperature and light. Depending on the pathogen, leaf wetness may be necessary to trigger sporulation or for the early stages of the sporulation process (formation of mycelial mat and conidiophores). However for others sporulation can be initiated in the presence of high relative humidity. Since no information on sporulation of Cercospora carotae was available, this study was undertaken to examine the influence of leaf wetness, high relative humidity and temperature on spore production.

Zadoks, J. C., and Schein, R. D. 1979. Epidemiology and plant disease management. Oxford University press, New York. 427pp.

STUDY II

INFLUENCE OF TEMPERATURE, DURATION OF HIGH RH AND WET PERIOD ON SPORULATION OF Cercospora carotae ON CARROT LEAVES.

ABSTRACT

The influence of temperature (16 to 32 °C) and duration of moist period (6 to 96 hr) on sporulation of Cercospora carotae was quantified on carrot plants under three types of moisture conditions (leaf wetness, 96%RH, and 96%RH with an initial 12 hr of leaf wetness). Sporulation was quantified as the number of spores per lesion and then transformed to proportion of maximum number of spores (PMS). The highest PMS (1.78 X 10^6 spores/lesion) was obtained at 28 °C and 96 hr of leaf wetness. Similar temperature and time effects were observed under leaf wetness and 96%RH conditions, except for 96%RH where no sporulation was observed at any time at 16 and 32 °C. For all types of moisture conditions, PMS increased with the increase in temperature up to the optimum (28 °C) and then declined. The presence of an initial 12 hr of leaf wetness enhanced sporulation and accelerated the beginning of sporulation as compared to continuous 96%RH. The PMS was modeled as a nonlinear logistic function of time for the leaf wetness (R^2=0.98) and 96%RH moisture conditions (R^2=0.97). A polynomial model was

used to describe sporulation as a function of temperature and time under 96%RH with initial 12 hr leaf wetness (R^2=0.95).

INTRODUCTION

Cercospora blight of carrot, induced by Cercospora carotae (Pass.) Solh is a common disease of carrots grown on organic soil in Quebec. Routine fungicide applications, based on calendar, have been the main control measure. Recently, efforts have been made to reduce the number of fungicide applications required to manage Cercospora blight (Boivin et al., 1990, Kushalappa et al.,1989)

The short-range objective was to develop a weather-based forecasting system to manage Cercospora blight more efficiently. The long-range objective was to develop a simulation model. The first step in building simulation models is the construction of submodels that describe the influence of the environment on different phases of the pathogen development, such as sporulation (Kushslappa, 1989). Such a submodel has been developed for the infection process of C. carotae (Carisse and Kushalappa, 1990, Carisse and Kushalappa, 1991). However, no precise information on sporulation of C. carotae is available or suitable for building a submodel for sporulation.

Preliminary experiments were conducted to determine the

conditions favorable for sporulation that should be included in the sporulation submodels. The effect of temperature (16 to 32 °C) at various %RH levels (65 to 100%) were studied. In these experiments, no sporulation occurred at %RH $\leq$ 92%, while abundant spores were produced under leaf wetness conditions. However, in Quebec, periods of leaf wetness or are usually preceded and followed by periods of high RH.

This experiment was undertaken to establish the temperature and time requirements for sporulation under high relative humidity and leaf wetness conditions. This paper also presents three sporulation models that describe the influence of temperature and time on sporulation under three moisture conditions: leaf wetness, 96%RH, and 96%RH preceded by 12 hr of leaf wetness.

MATERIALS AND METHODS

Inoculum production. All inoculum used for these studies was obtained from a single spore culture of *C. carotae* isolated from naturally-infected carrot leaves collected in 1987 at the Agriculture Canada Experimental Farm in Sainte-Clotilde, Québec. The fungus was cultured on carrot leaf infusion agar (CLA) as previously described (Carisse and Kushalappa, 1989). Conidial suspensions were prepared from 12-day-old cultures incubated at 26 °C and 18 hr per day of

fluorescent light (100 $\mu Em^{-2}s^{-1}$). Conidia were suspended in a solution of 0.01% Tween 80 (v/v) in distilled water and the concentration was adjusted to 1 X 10^4 conidia per millilitre.

Plant production. Carrot plants cv. Dagger were grown in a greenhouse maintained at 22 $\pm$ 5.0 C with a photoperiod of 12 hr of light per day. The plants were produced in 13-cm diameter pots with a 5:1:1 (v/v) mixture of organic soil (27-30 % organic matter), perlite, and peatmoss. Fertilizers (15-15-17, 200 ppm) were applied twice a week. The plants were sprayed with insecticides (Trumpet 80W 0.75kg/1000L, and diatomaceous earth) every week to prevent thrips infestation. At the six-leaf stage (five weeks after sowing) the second and third leaves were tagged.

Inoculation procedure. Both surfaces of the tagged leaves were inoculated with a conidial suspension of C. carotae using an artist air brush (Badger-350) adjusted at 100 kPa. After inoculation the plants were placed in another greenhouse adjusted at 25 $\pm$ 5 C for 72 hr to promote infection (Carisse and Kushalappa, 1990). Free water on leaf surfaces was provided by a misting of 5 sec every 8 min. During the incubation period the plants were kept in a greenhouse set at 22 $\pm$ 5 C and RH $\leq$ 65% $\pm$ 5%. The percent

spore germination of the inoculum was estimated for each inoculation. Three water agar plates were sprayed, one at the beginning, one in the middle and one at the end of the inoculation procedure. After six hours, the percent spore germination was estimated for each plate.

Treatments. The whole experiment consisted of three types of moisture conditions: continuous leaf wetness (LW), continuous 96% RH (96RH), and 96% RH with an initial 12 hr of leaf wetness (96RHW). The moisture durations were 6 (except for the 96RHW treatment), 12, 24, 48, 72, and 96 hr. Thirteen days after inoculation (when the plants exhibited small chlorotic lesions), the number of lesions on each inoculated leaf was counted on all plants, which were transferred to a RH-controlled chamber (model PGW36 M10, with RH controlled by bypass dehumidification) maintained at specific temperature and 96 $\pm$2 % RH. In the RH-controlled chamber, the plants were exposed to a 12 hr photoperiod supplied by fluorescent and incandescent fixtures producing a light intensity of 250 $\mu E/m^2/s^{-1}$. The plants exposed to the leaf wetness conditions (18 plants) were misted with distilled water and enclosed in plastic bags. The plants exposed to an initial leaf wetness period (15 plants) were also misted with distilled water and enclosed in plastic bags that were removed after 12 hr. At the end of the

exposure time, three plants per moisture type were removed for observations (number of spores per lesion). Each treatment combination included three sampling units and the whole procedure was repeated five times for temperatures in the RH controlled chamber of 16, 20, 24, 28, and 32 C tested in a random order. The whole experiment was conducted twice.

Estimation of sporulation. The sporulation (SPO) was quantified as the number of spores produced per lesions per plant. Two inoculated leaves per plant were harvested, rolled in a wax paper, and then inserted into a test tube containing 10 mL of a solution of 1% formaldehyde and 0.01% Tween 80. The test tubes were agitated for two min and then the number of spores in the suspension was evaluated with an hemacytometer (four counts). The total number of spores per lesion per plant (average of the four counts) was calculated (Eq. 2.1-2.2).

spores = number of spores/ml X 10 mL of suspension (2.1)

SPO = spores / total number of lesions per plant (2.2)

Data analysis. The data (SPO) were transformed to proportion of maximum sporulation (PMS) by dividing the SPO (number of spores/lesion/plant) obtained for each treatment

by the maximum SPO obtained from any of the moisture conditions.

Tests for equality of variance (F-test) were carried out to determine whether the data from each experimental trial could be pooled. Because the temperatures were tested over time and using different inoculum suspensions (temperature and inoculum effects are confounded), an F-test was used to determine whether the percent spore germination of inoculum significantly varied among inoculations (Steel and Torrie, 1980). Regression analyses were performed separately for each moisture type (LW, 96RH, and 96RHW) using SAS., non-linear and linear models procedures (PROC NLIN, PROC GLM, and PROC REG) (SAS, 1987). Two types of models were used to describe the effects of temperature (T) and moist period duration (D) on PMS; a nonlinear logistic model for the LW and 96RH moisture types, and a polynomial model for the 96RHW. The fit of the nonlinear logistic models was evaluated by considering the coefficient of determination (R^2), the size of asymptotic standard error associated with the estimated parameters and by visual inspection and analysis of residuals plot (Draper and Smith, 1981). The polynomial model was evaluated based on the coefficient of determination (R^2 and R^2adj), by the significance of the estimated regression parameters, and by analysis of residuals distribution (Draper and Smith, 1981, Steel and

Torry, 1979).

Model development for the LW and 96RH conditions. The effect of time at the different temperatures tested produced a sigmoid curve for both LW and 96RH conditions. The nonlinear logistic model used for the LW and 96RH treatments was of the form:

$$PMS = \frac{PMS_m}{1 + [(PMS_m - PMS_0)/ PMS_0] \, EXP(-rD)} \qquad (2.3)$$

where PMS is the proportion of maximum observed sporulation at time D, PMS_m is the maximum PMS at any time for a given temperature (asymptote), PMS_0 is the initial level of sporulation (Y-intercept), r is the rate of sporulation, and D is the moist period duration (hr). The logistic model was fitted separately to the data of each experimental trial and to the pooled data using the nonlinear procedures with Marquardt iteration methods (SAS, 1987). For both LW and 96RH conditions the logistic model was fitted in four steps briefly described below (Carisse and Kushalappa, 1990, Lalancette et al., 1988). These steps are used to determine the values of some of the parameters which must be initially fixed in order to be able to run the NLIN procedure on data for all temperature together.

First, a separate equation for predicting the maximum

sporulation (PMS_m) was derived for each temperature. Because the effect of temperature on PMSm produced a curve that was skewed to the right the second-order polynomial model was not adequate thus two other types of models were tested. The first model was of the form:

$$PMSm = B_0 + b_1T + B_2T^2 + B_3T^3 + B_4T^4 \qquad (2.4)$$

The second model was a generalized form of the Analytis' Bete function (Analytis, 1977):

$$PMSm = pt^m(1-T)^n \qquad (2.5)$$

which can be transformed to:

$$\ln(PMSm) = \ln(p) + m\ln(t) + n\ln(1-t) \qquad (2.6)$$

where, p, n, m are parameters and $t=(T-T_{min})/(T_{max}-T_{min})$. The maximum temperature (T_{max}) and minimum temperature (T_{min}) were not known precisely, and values of 12 and 36 C were assigned for T_{min} and T_{max}, respectively. Other values did not result in good fit of the model.

In the second step, an equation predicting the PMSm (first step) was substituted in the nonlinear logistic function (Eq. 2.3) and the resulting function was fitted to the data for each temperature separately. The value of the rate parameter was estimated from the regression procedures.

The value of PMS_0 was arbitrarily assigned to 0.0001 since the intercept values for all temperatures were not significantly different from 0 when estimated by the regression procedures.

The third step consisted of deriving an equation for predicting the rate parameter, obtained from the second step, as a function of temperature. Second-order polynomial model and Bete function (Eq. 2.4 and 2.6) were tested. Regressions were performed for the pooled data only (r values obtained for each experiment were pooled).

The fourth and final step consisted of incorporating the equations predicting the PMSm and r parameters into the logistic model and fitting the resulting model to the data for all temperatures.

Model development for the 96RHW condition. The time effect on sporulation under 96RHw conditions did not followed a pattern that could be adequately explained by the logistic function. For this reason, two types of polynomial models were evaluated. The first was an extension of the Schodter (Schodter, 1965) sine-model of the form:

$$PMS = \sin^2(f[T,D]) \qquad (2.7)$$

which can be transformed to:

$$\arcsin(\sqrt{PMS}) = f(T,D) \qquad (2.8)$$

Where the $\sin^2$ is the trigonometric sine function and arcsin is the inverse sine function.

The second model was a general form of the polynomial function:

$$PMS = f(T,D) \qquad (2.9)$$

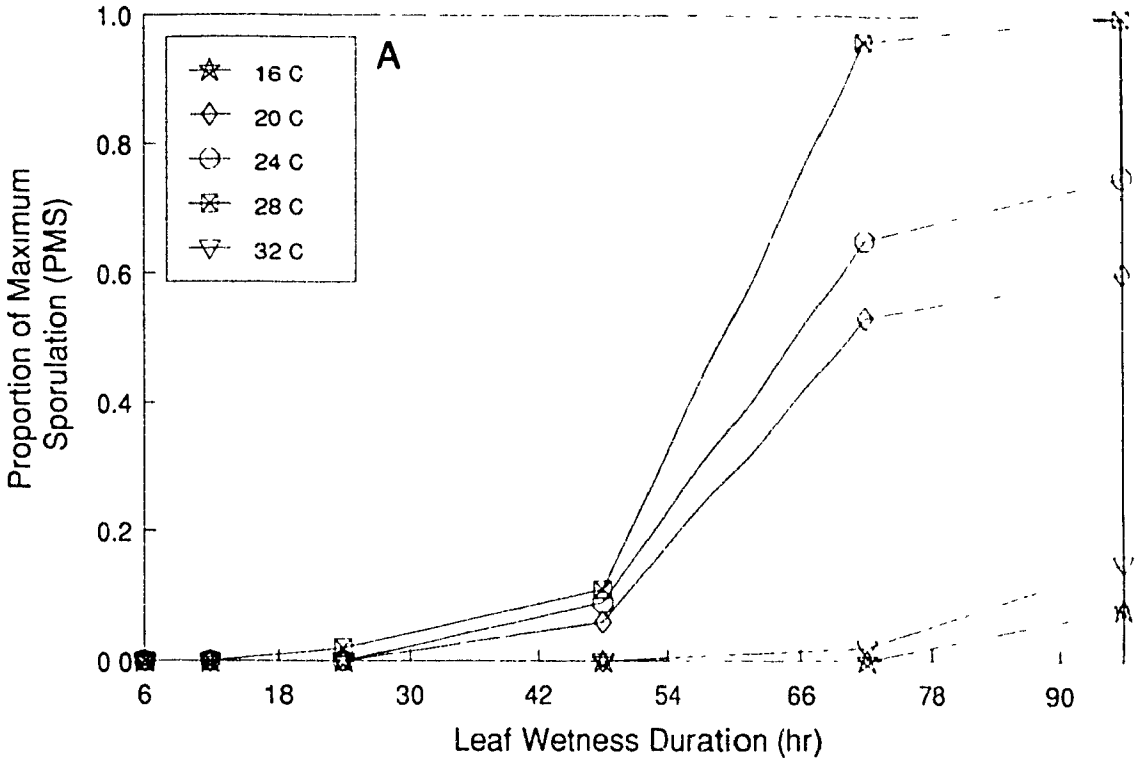
These two models (Eq. 2.8 and 2.9) were fitted to the data for each experiment and the pooled data and all possible combinations of temperature and moist period duration terms were tested for the significance of the estimated parameters (Freund and Littell, 1981).

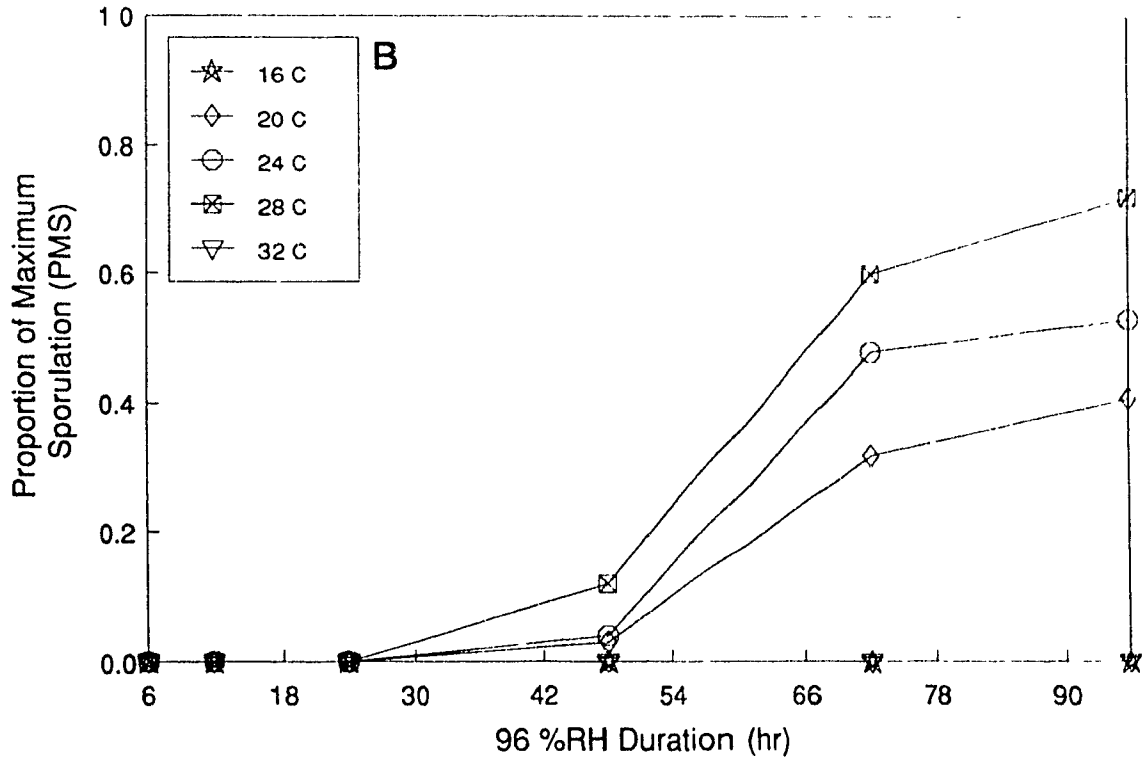
RESULTS

In general, sporulation occurred after 48 hr. Maximum number of spores per lesion was obtained after 96 hr under leaf wetness and 28 C (1 928 and 1 632 spores/lesion for the first and second experiment, respectively). For all temperatures, the sporulation increased with increases in wet or moist period duration and the number of spores per lesion was higher under leaf wetness than 96% RH with or without initial wetness period. For all moisture types, the sporulation increased with increase in temperature from 16 to 28 C then diminished at 32 C. The curves for proportion of maximum sporulation against time at 20, 24, and 28 C were

sigmoid for both leaf wetness and 96% RH. The sporulation increased slowly from 6 to 48 hr, and increased very rapidly between 48 to 72, then slowly again between 72 and 96 hr. (Figure 2.1A-B). Under leaf wetness, the number of spores per lesion obtained at 16 and 32 C was very low for the 6 to 72 hr durations and increased slowly from 72 to 96 hr, while no sporulation was obtained at 96% RH for these two temperatures. A different time effect was observed for the 96RHW moisture type, where the PMS increased gradually over time (Figure 2.2). The sporulation started after only 12 hr and increased until 72 hr.

Figure 2.1. Observed effect of temperature and time on the number of spores per lesion of C. carotae. Infected carrot plants were subjected to constant temperatures of 16, 20, 24, 28, and 32 °C. In A) the plants were misted and enclosed in plastic bags. In B) the plants were placed in a RH controlled chamber adjusted at 96 %RH. Data are presented as proportion of maximum number of spore/lesion (1928 and 1632, for the first and second trial, respectively) observed at 28 °C and 96 hr of leaf wetness. Each point is an average of six plants (3 plants/treatment/experiment).


A
16 C
20 C
24 C
28 C
32 C
Proportion of Maximum Sporulation (PMS)
1.0
0.8
0.6
0.4
0.2
0 0
6
18
30
42
54
66
78
90
Leaf Wetness Duration (hr)


B
16 C
20 C
24 C
28 C
32 C
Proportion of Maximum Sporulation (PMS)
1 0
0.8
0.6
0.4
0.2
0.0
6
18
30
42
54
66
78
90
96 %RH Duration (hr)

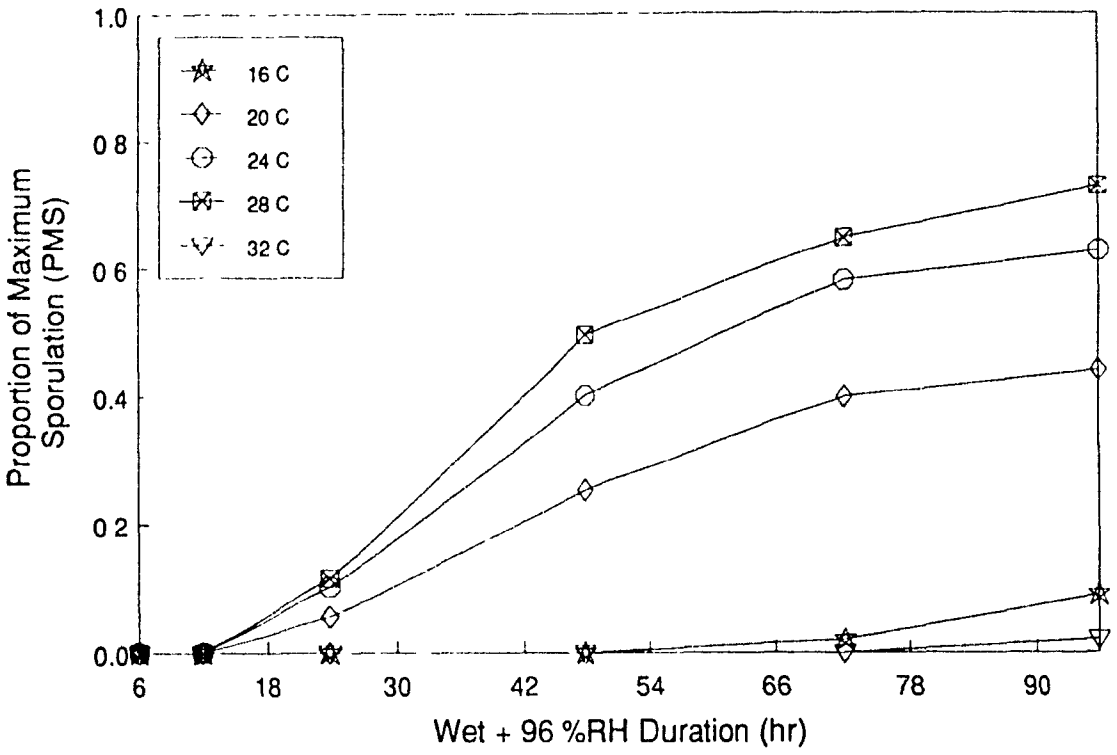
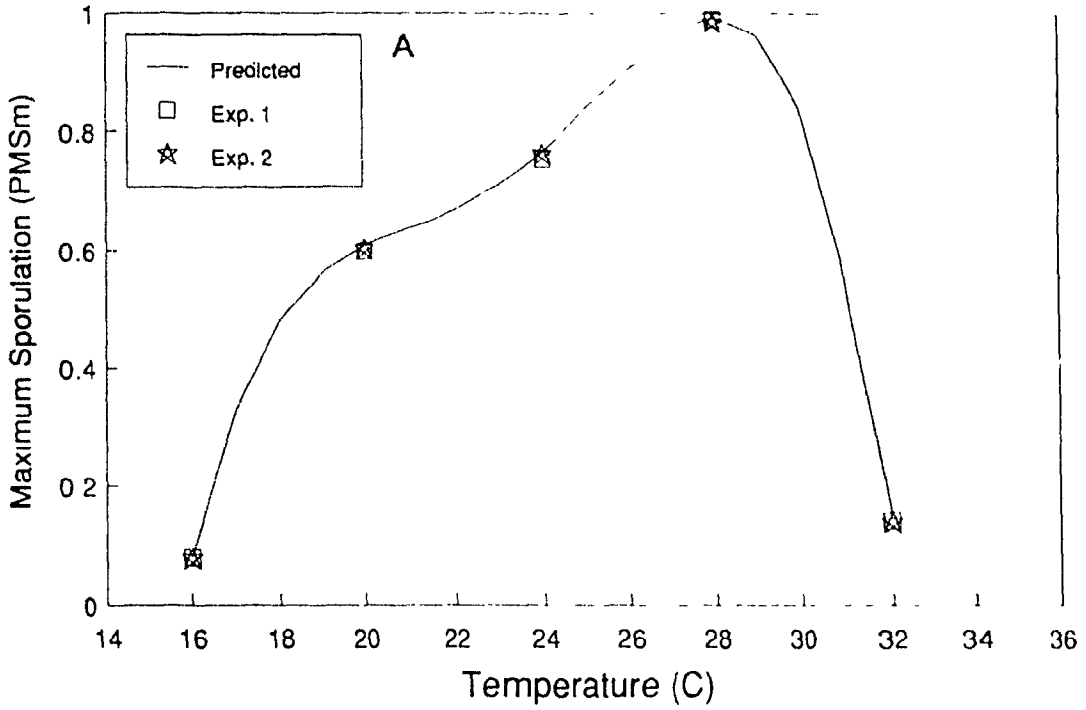


Figure 2.2. Observed effect of the duration of 96 % RH periods and temperature on the number of spores per lesion of C. carotae. Infected carrot plants were misted and enclosed in plastic bags for 12 hr and kept in a RH controlled chamber adjusted at 96 %RH and constant temperatures of 16, 20, 24, 28, and 32 °C. Data are presented as proportion of maximum number of spore/lesion (1928 and 1632,for the first and second trial, respectively) observed at 28 °C and 96 hr of leaf wetness. Each point is an average of six plants (3 plants/treatment/experiment).

Estimation of logistic model parameters for LW and 96RH conditions. Because the F-test showed no significant difference due to inoculation ($P>0.05$), parameters of the logistic function obtained for each temperature levels (PMS_m and r) were combined for the analysis. Maximum sporulation (PMS_m) was observed at 28 C for both LW and 96RH moisture types, but the temperature effect on PMS_m produced a curve skewed to the right that could be adequately explained only by a fourth-degree polynomial model (Figure 2.3A-B). Several other models were considered including lower levels of polynomial and Bete-function. These models resulted in unacceptable overestimation at 24 C and underestimation at 28 C of PMS_m. Considering the importance of the asymptote parameter (PMS_m) in the logistic model the fourth-degree polynomial model was retained. This model yielded high coefficients of determination with all parameter estimates significant ($P< 0.0001$) (Table 2.1). The distribution of residuals was normal and no patterns could be detected. This model predicted a maximum PMS of 0.99 and 0.73 for the LW and 96RH moisture type, respectively (observed were 1.00 and 0.73).

The rate parameter (r) for both moisture types was high at 20. 24, and 28 C while low at 16 and 32 C (Figure 2.4A-2.4B). The effect of temperature on the rate parameter produced a bell-shaped curve with small variation between

Figure 2.3. Relationship between maximum sporulation (PMS_m) parameter of the logistic function and temperature. In A) the plants were exposed to leaf wetness conditions. In B) the plants were exposed to 96 %RH conditions.


A
Predicted
Exp. 1
Exp. 2
Maximum Sporulation (PMSm)
0
0.2
0.4
0.6
0.8
1
14
16
18
20
22
24
26
28
30
32
34
36
Temperature (C)

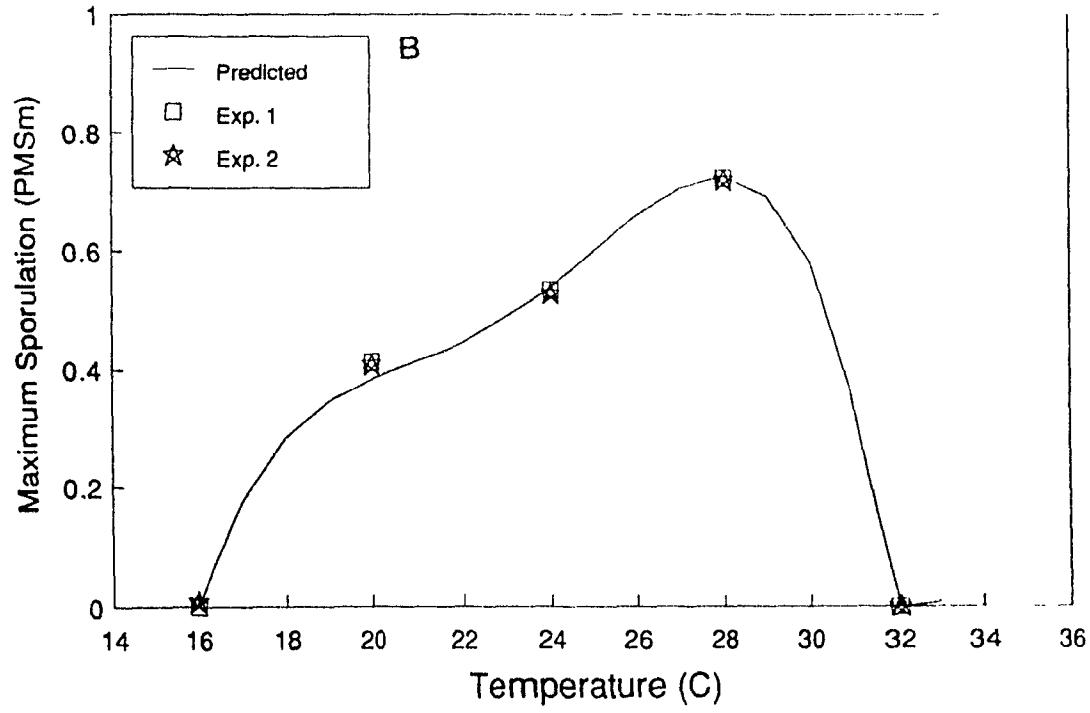

B
Predicted
Exp. 1
Exp. 2
Maximum Sporulation (PMSm)
0
0.2
0.4
0.6
0.8
1
14
16
18
20
22
24
26
28
30
32
34
36
Temperature (C)

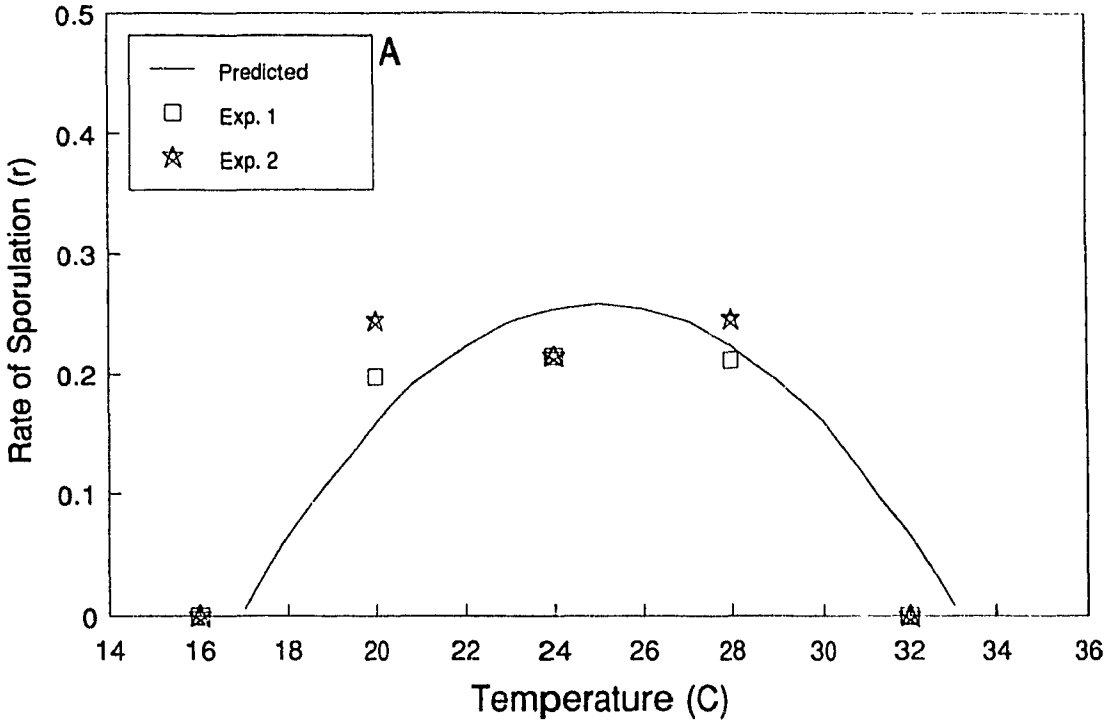
Table 2.1. Estimated regression parameters and associated statistics for the regression of maximum sporulation (PMS_m)[1] of C. carotae as a function of temperature for the leaf wetness and 96% RH conditions.

Model	DF Error	F P-value	R^2 adj.[2]	Estimate/P-value b0	b1	b2	b3	b4
Leaf wetness								
Exp.1	10	0.0001	0.99	-70.18	12.48	-0.823	0.024	-0.0002
				0.0001	0.0001	0.0001	0.0001	0.0001
Exp.2	10	0.0001	0.99	-70.82	12.56	-0.826	0.024	-0.0002
				0.0001	0.0001	0.0001	0.0001	0.0001
Pooled	25	0.0001	0.99	-70.51	12.53	-0.825	0.024	-0.0002
				0.0001	0.0001	0.0001	0.0001	0.0001
96 % relative humidity								
Exp.1	10	0.0001	0.99	-58.89	10.48	-0.692	0.020	-0.0002
				0.0001	0.0001	0.0001	0.0001	0.0001
Exp.2	10	0.0001	0.99	-43.81	7.894	-0.529	0.016	-0.0001
				0.0001	0.0001	0.0001	0.0001	0.0001
Pooled	25	0.0001	0.99	-51.35	9.190	-0.611	0.018	-0.0002
				0.0001	0.0001	0.0001	0.0001	0.0001

[1] The value of (PMS_m) was calculated using equation 2.4.

[2] Coefficient of determination adjusted for the number of independent variables.

Figure 2.4. Relationship between rate of sporulation (r) parameter of the logistic function and temperature. In A) the plants were exposed to leaf wetness conditions. In B) the plants were exposed to 96 %RH conditions.


A
Predicted
Exp. 1
Exp. 2
Rate of Sporulation (r)
0.5
0.4
0.3
0.2
0.1
0
14
16
18
20
22
24
26
28
30
32
34
36
Temperature (C)

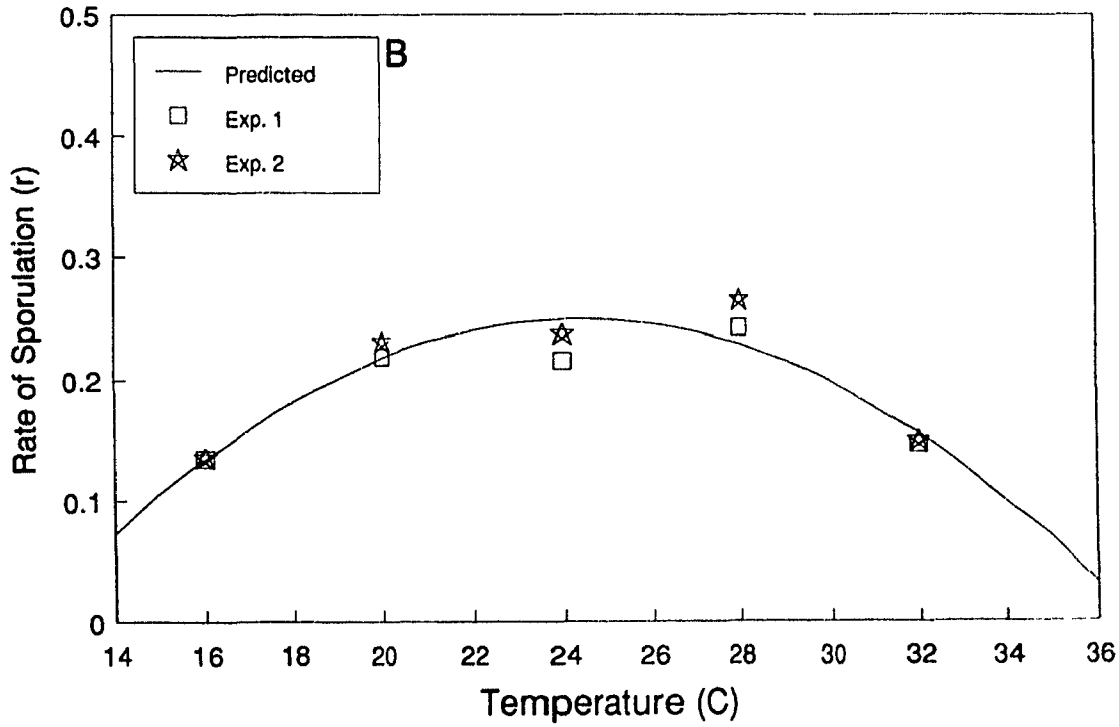

B
Predicted
Exp. 1
Exp. 2
Rate of Sporulation (r)
0.5
0.4
0.3
0.2
0.1
0
14
16
18
20
22
24
26
28
30
32
34
36
Temperature (C)

Table 2.2. Estimated regression parameters and associated statistics for the regression of rate of sporulation (r)[y] of C. carotae as a function of temperature for the leaf wetness and 96% RH conditions.

Model	DF Error	F P-value	R^2adj.[z]	Estimate/P-value b0	b1	b2
Leaf wetness[c]						
Pooled	5	0.0001	0.87	-0.7231	0.0797	-0.0016
				0.0001	0.0001	0.0001
96 % relative humidity						
Pooled	5	0.0001	0.92	-2.009	0.1888	-0.0039
				0.0001	0.0001	0.0001

[y] The value of (PMS_m) was calculated using a second-order polynomial of temperature model.

[z] Coefficient of determination adjusted for the number of independent variables.

20, 24 and 28 C (Figure 2.4A-2.4B). Several models were tested including two to four-degree polynomial, segmented polynomial and Bete-function. The second-order polynomial provided a good fit explaining 0.87 and 0.92 % of the variation in the rate parameter (r) with temperature for the LW and 96RH moisture type, respectively. All parameter estimates were significant ($P < 0.0001$) (Table 2.2) and no patterns were evident in the residuals. However, the rate of sporulation predicted by the model was slightly overestimated at 24 and underestimated at 28 °C (Figure 2.4A-B). The rate parameter was predicted to reach a maximum of 0.25 and 0.26 at 24 °C for the LW and 96RH moisture types, respectively.

Fitting the nonlinear logistic model to the pooled data resulted in high coefficient of determination, 0.98 and 0.97 % for the LW and 96RH moisture conditions, respectively. Furthermore, no patterns were observed in three-dimensional plots of residuals against temperature and time for both moisture conditions. The nonlinear relationship between PMS, T, and D for the LW moisture conditions:

$$PMS = \frac{(PMS_m)}{1 + [(PMS_m - PMS_0)/\ PMS_0]\ EXP(-rD)} \qquad (2.10)$$

where PMS is the proportion of maximum sporulation at time D, $PMS_m = -70.51+12.53T+0.8247T^2+0.0239T^3-0.0002T^4$, $PMS_0 = 0.00001$,

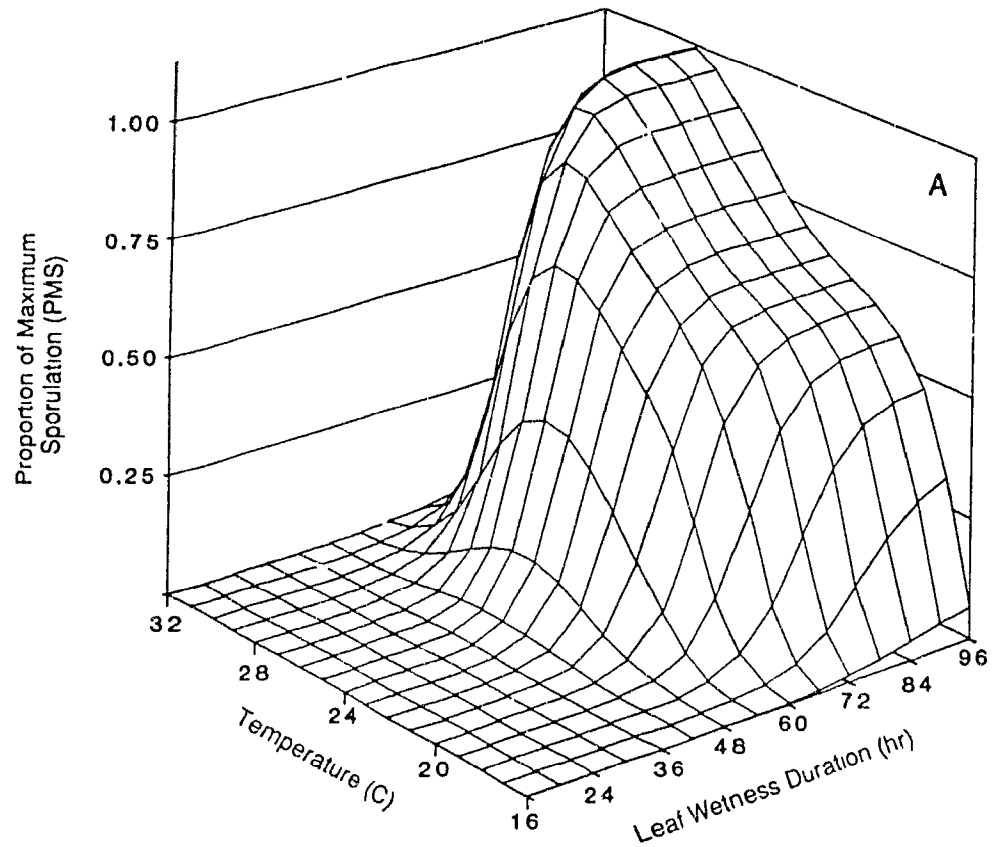
$r = -0.7231+0.0797T-0.0016T^2$, T is the temperature (°C), and D is the duration of wet period (hr) (Figure 2.5A).

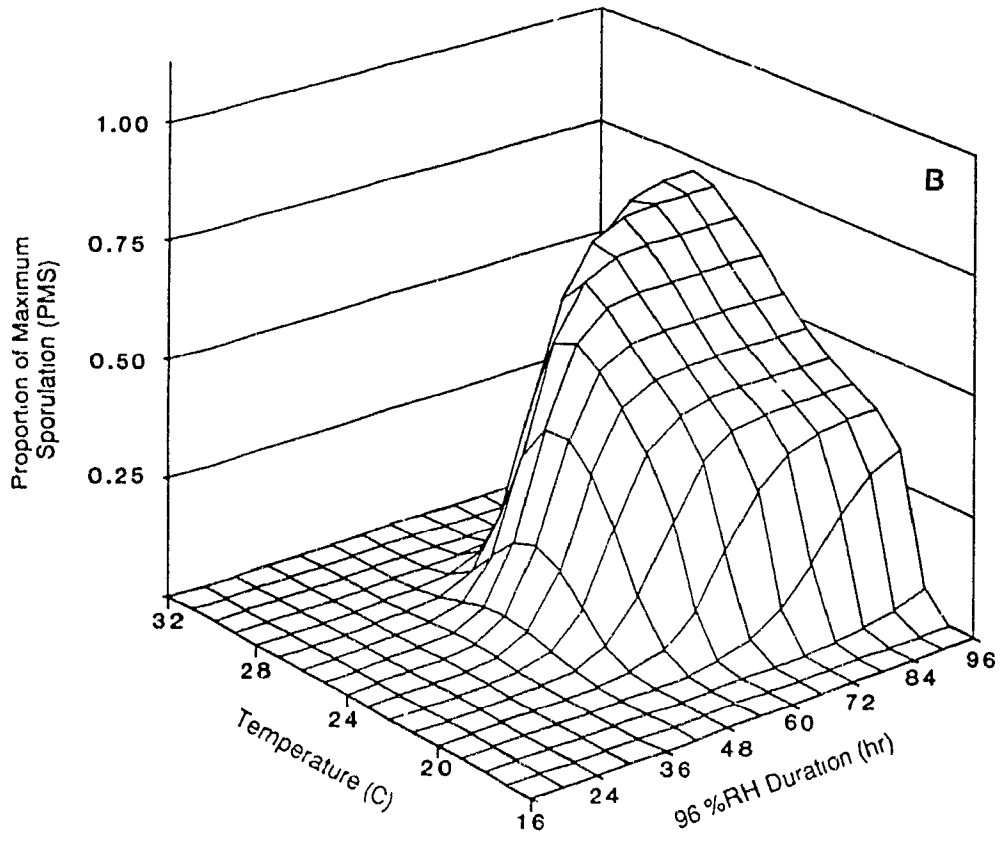
The equation 2.11 was used to describe the relationship of PMS, T, and D for the 96RH moisture conditions,

$$PMS = \frac{PMS_m}{1 + [(PMS_m - PMS_0) / PMS_0] \, EXP(-rD)} \qquad (2.11)$$

where PMS is the proportion of maximum sporulation at time D, $PMS_m = -51.35+9.190T+0.6112T^2+0.0179T^3-0.0002T^4$, $PMS_0 = 0.00001$, $r = -2.009+0.1888T-0.0039T^2$, T is the temperature (°C) and D is the duration of 96% RH period (hr) (Figure 2.5B).

Figure 2.5. Predicted values for proportion of maximum sporulation (PMS) as a function of time and temperature. In A) the response surface was generated using the equation 2.10. In B) the response surface was generated using the equation 2.11.


A
Proportion of Maximum Sporulation (PMS)
1.00
0.75
0.50
0.25
32
28
24
20
16
Temperature (C)
24
36
48
60
72
84
96
Leaf Wetness Duration (hr)


B
Proportion of Maximum Sporulation (PMS)
1.00
0.75
0.50
0.25
32
28
24
20
16
Temperature (C)
24
36
48
60
72
84
96
96 %RH Duration (hr)

Estimation of polynomial model parameters for 96RHW moisture conditions. The best polynomial model describing the relationship of PMS, T, and D was of the form:

$$PMS = 0.0439+0.0538D+0.3983X10^{-4}D^2-0.0018T-0.0003T^2+0.0829X10^{-4}T^3 -0.0088TD+0.0005DT^2-0.0850X10^{-4}TD^3 \quad (2.12)$$

where, PMS is the proportion of maximum sporulation, T is temperature and D is the moisture duration. This model explained 96 % of the variation in PMS (Table 2.3). Furthermore, no patterns were observed in a three-dimensional plot of residual against temperature and time. This model provided excellent prediction at temperatures of 20, 24, and 28 °C, but the model tended to overestimate sporulation at temperature of 16 and 32 °C for all durations except at 96 hr where the sporulation was slightly under estimated (Figure 2.6).

Table 2.3. Estimated regression parameters and associated statistics for the polynomial regression of proportion of maximum sporulation (PMS) of C. carotae as a function of temperature and duration of 96 %RH period preceded by 12 hr of leaf wetness.

Statistics	Model Exp.1	Exp.2	Pooled
DF Error	66	66	141
F P-value	0.0001	0.0001	0.0001
R^2	0.96	0.96	0.96
R^2adj	0.95	0.96	0.95
Estimate/P-value			
b0	0.0191/0.0001	-0.0191/0.0001	-0.0439/0.0001
b1	0.0517/0.0001	0.0558/0.0001	0.0538/0.0001
b2	-0.3633/0.0001 (x10-4)	-0.4333/0.0001 (x10-4)	-0.3983/0.0001 (x10-4)
b3	-0.0086/0.0001	0.0049/0.0002	-0.0018/0.0001
b4	-0.2592/0.0001 (x10-4)	-0.0005/0.0001	-0.0003/0.0001
b5	0.0533/0.0001 (x10-4)	0.1124/0.0001 (x10-4)	0.0821/0.0001 (x10-4)
b6	-0.0008/0.0068	-0.0091/0.0111	-0.0088/0.0002
b7	0.0005/0.0001	0.0005/0.0001	0.0005/0.0001
b8	-0.0836/0.0001 (x10-4)	-0.0838/0.0001 (x10-4)	-0.0850/0.0001 (x10-4)

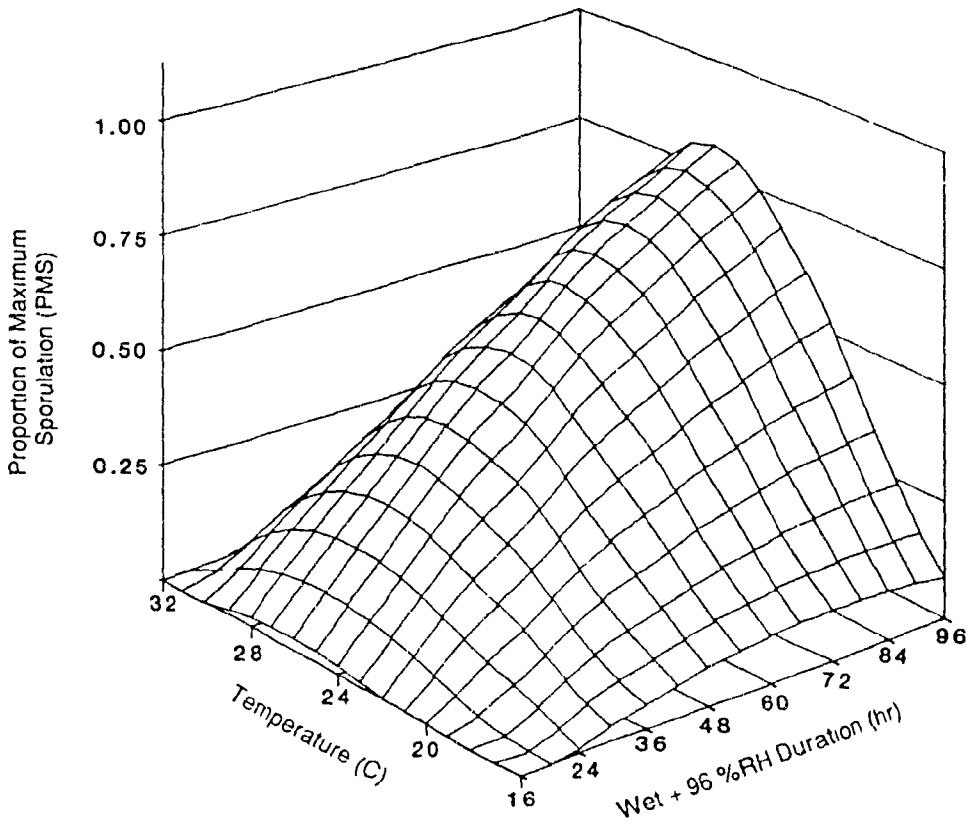


Figure 2.6. Predicted values for proportion of maximum sporulation (PMS) as a function of time and temperature. The response surface was generated using the polynomial regression equation 11.

DISCUSSION

The results showed that the optimum temperature for sporulation of C. carotae on carrot leaves was 28 °C and a minimum of 24 hr of leaf wetness or high relative humidity was required to induce sporulation at that temperature. Spores were numerous only when the wet period exceeded 48 hr at 20 to 28 °C. Although sporulation of C.carotae has not been previously quantified on carrot leaves, observations have been reported on sporulation at different temperatures on agar media. The optimum temperature of 28 °C observed for sporulation on carrot leaves was also found to be optimum for mycelial growth and sporulation in culture (Carisse and Kushalappa, 1989). In general, our results agree with those of Thomas (1943) who also observed maximum sporulation at 28 °C on carrot petioles. However, the latter study also reported abundant sporulation at 13 °C after only 24 hr. Unfortunately, no detailed information on the conditions under which the experiment was conducted is available to compare with. Nevertheless, our results stand in accordance with those from experiments on other species of Cercospora (Alderman and Beute, 1987, Cooperman and Jenkins, 1986).

Although this study was not extensive, it demonstrated that sporulation was favoured by high relative humidity (> 96 %) or leaf wetness and warm temperature (20 to 28 °C). Leaf wetness is more favorable than high relative humidity in

supporting abundant conidial production. A period of leaf wetness is not necessary to trigger sporulation but the presence of a short leaf wetness period prior to a prolonged period of high relative humidity accelerates the production of conidia.

The time effect on conidial production by C. carotae in controlled conditions produced a sigmoid response under leaf wetness or 96 % relative humidity for temperature between 20 and 28 °C. Although several models could be used to describe such response for time effect (Lalancette et al., 1988, Venus and Causton, 1979), the logistic model worked well in this case in describing the temperature and time effect on sporulation of C. carotae The effect of temperature on the maximum sporulation produced a curve skewed to the right because the highest sporulation was observed at 28 °C not at 24 C which would have produced a bell-shaped curve easily explained by a second-order polynomial. The Bete-function was proposed to address this problem (Analytis, 1977), however with our data this model was not suitable and did not work well, probably because the minimum and maximum temperatures for sporulation were not known. The use of a fourth-degree polynomial is not the ideal solution, but it was satisfactory in this work. Conidial production increased gradually over time for all temperatures tested when the plants were exposed to 96 % relative humidity preceded by 12 hr of leaf wetness.

The polynomial model was found to be appropriate even if nine terms were needed to explain the combined effect of temperature and time. Coefficients of determination adjusted for the degree of freedom (R^2_a) were high 0.95, 0.96, 0.95 for experiment one, two, and pooled data, respectively, indicating the importance of various terms in the model. The values of R^2_a were similar to the values of R^2 indicating that all terms were necessary.

There are limitations inherent to this type of study including instrumentation, sampling methods, integration of temperature-RH or temperature-leaf-wetness interactions, and effects of preconditioning of the lesions. In this experiment the effects of temperature and time on sporulation were examined during a single sporulation period. In the field however, sporulation may occur over few consecutive wet or humid periods. These results may not reflect the exact amount of conidia available in the field for infection because the harvesting techniques was probably too vigourous. Under field conditions only mature spores become detached. Nevertheless, from the results of this study the environmental requirements for sporulation of Cercospora carotae have been established and a basis for more detailed future investigations. These results should not be use to predict the exact amount of conidia available for infection but rather to estimate the

potential of the environment for sporulation.

The infection of C. carotae has been shown to occur at temperatures ranging from 20 to 28 °C (Carisse and Kushalappa, 1990). Maximum infection occurred after 24 hr at 28 °C. Consequently, the optimal range of temperature for infection is similar than for sporulation. Thus we can speculate that when conditions are favorable for infection (> 24 hr of leaf wetness at 20 to 28 °C) sporulation does not limit the epidemic development. In a forecasting system, periods of leaf wetness or relative humidity above 96 % for more than 24 hr at temperature greater than 16 °C and less than 32 °C could be considered favorable for sporulation of C. carotae.

These types of studies have limited value in predicting sporulation in the field where age of lesions vary. temperature fluctuates, and wetness is interrupted. Further research is needed to examine the effect of these factors on conidial production of C.carotae and to determine if the behaviour of the fungus in the controlled environment depicts the fungal behaviour in the field.

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PREFACE TO STUDY III

The latency and incubation periods are key factors in disease progression. The latency period is defined as "the time elapsed from arrival of a dispersal unit at a susceptible plant surface until the formation of the next generation of dispersal units." (Zadok and Shein, 1979). The incubation period (IP) is defined as the time from inoculation to symptom expression (Campbell and Madden, 1990) or more simply the time needed for symptoms to develop since inoculation. The latency period determines the number of pathogen generations possible within a season, thus the rate of disease development. Short latency period will lead to many pathogen cycles, thus to faster disease progression. In epidemiology the incubation period is important because, apart from the effect on rate of disease progression, disease severity is usually estimated based on visual symptoms. When weather conditions are favorable for infection the symptoms will be visible only one incubation period later. Knowledge on incubation period including temporal scale on which lesions appear as well as pattern of that appearance are a prerequisite for disease modelling.

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STUDY III

EFFECT OF TEMPERATURE AND MOISTURE ON INCUBATION PERIOD OF *Cercospora carotae* UNDER FIELD CONDITIONS.

ABSTRACT

The incubation period of *Cercospora carotae* on carrots was studied in the field on the cultivar Dagger. During the summer of 1990, ten plots of carrots were sown and inoculated at the sixth-leaf stage. First lesions were observed 6 to 8 days after inoculation and new lesions appeared until the 10th to 14th day. The plot of proportion of maximum number of lesions against days after inoculation for all plants produced a sigmoid curve, and this was adequately explained by a logistic equation (r=0.92 to 0.98). This equation was used to calculate the beginning (IP5), mean (IP50), and end (IP95) of incubation period defined as the time in days for 5, 50, and 95% of lesions appearance, respectively. Variation in IP5, IP50 and IP95 was explained by variation in temperature and mean daily RH $\geq$ 90%, with R^2 values of 0.56, 0.92, and 0.89, respectively. Mean daily temperature $\geq$ 21 C resulted in short incubation period (IP50 = 8 days) and mean daily temperature below 21 C resulted in longer IP (IP50=10 days). A general model describing lesion appearance as a function of time for all inoculations was developed using a logistic function. This model explained 84% of the variation in proportion of

maximum number of lesions.

INTRODUCTION

Cercospora blight, caused by Cercospora carotae (Pass.) Solh.,is one of the major disease of carrots grown on organic soil in Canada (Arcelin and Kushalappa, 1991). In most carrot fields, Cercospora blight is the only disease that require routine fungicide applications. Recently, several studies were reported on various components of Cercospora blight epidemic (Carisse and Kushalappa, 1990, Carisse and Kushalappa, 1991, study II) and on different methods of disease management that would allow reduction in number of fungicide applications (Boivin et al., 1990, Kushalappa et al.,1989).

Quantitative knowledge of the incubation period is essential for both understanding and managing the disease. Angell and Gabelman (1968) reported that symptoms of Cercospora blight on carrots can appear three to five days after inoculation. However, no detailed study on the influence of weather variables on incubation period is currently available. Incubation period greatly influences the rate of disease development of foliar pathogens since the possibility of rapid disease progress increases as the incubation period decreases. This in turn is influenced mainly by temperature and to some extent by % relative

humidity and leaf wetness duration (Shearer and Zadok, 1974).

The present study is part of a broader research program on Cercospora blight of carrots that aims to develop of a model to simulate blight progress as a function of the environment. While developing such model the incubation period can be incorporated in two ways. First, by including in the simulation model the mathematical equations predicting the incubation period as function of selected environmental variables (variable incubation period). Secondly, by considering a fixed incubation period.

The objectives of this study were thus first, to determine the influence of weather variables including temperature, leaf wetness and high relative humidity duration on the incubation period of C. carotae and to develop regression models that predict the incubation period as a function of these weather variables. Secondly, to develop a general model that describe the pattern of lesion appearance as a function of the number of days after inoculation independent of the weather conditions (fixed incubation period).

MATERIALS AND METHODS

Plot establishment. During the summer 1990, carrot (Daucus carota L sativa cv. Dagger) plots were established at the Agriculture Canada Experimental Farm at Sainte-Clotilde,

Quebec. Each of the ten plots consisted of three rows of 1.5 m long and the distance between rows was 0.5 m. Plots were mechanically prepared and fertilized according to the Quebec government recommendations (CPVQ) and seeded on different dates (Table 3.1) with a hand seeder at a rate of 80 to 100 seeds per meter. On ten occasions, from June to August, the incubation period was measured on carrot plants (see Table 3.1 for dates). At the six leaf stage the second and third leaves of 15 plants from the middle row of a plot were tagged and inoculated. A suspension of 10,000 conidia/ml of C. carotae and 0.1% Tween-80, prepared from fresh cultures (Carisse and Kushalappa, 1989), was sprayed with a hand sprayer on symptomless tagged leaves. To maintain leaf wetness, the inoculated plants were covered with a tunnel made of white plastic. The tunnel was installed three days before inoculation to prevent natural inoculation. Three days after inoculation the tunnel was removed. The number of lesions on each inoculated leaf was recorded three times per week, starting three days after inoculation until two identical readings were obtained (all lesions have appeared).

The weather data included hourly ambient temperature and relative humidity, and the presence of leaf wetness recorded with a datalogger (model CR-10, Campbell Scientific Canada Corp.) installed at proximity of the plots. The temperature and relative humidity were monitored at 1-min

intervals with a sensor (model Vaisala) located at 1.0 m from the ground installed in a Stevenson shelter. The leaf wetness was monitored at 1-min intervals with a sensor (model 237) installed within the plant canopy and moved up as the carrots grew. All weather data monitored with the datalogger were saved as 1-hr averages.

Experimental design. The dependent variables were the beginning (IP5), mean (IP50) and end (IP95) of incubation period defined as the period in days for the appearance of 5, 50 and 95 % of the lesions, respectively. The independent variables were the mean daily duration of leaf wetness (LW), the mean daily duration of relative humidity $\geq$ 90% (RH), the mean daily temperature (DT), the mean maximum daily temperature (MAX), the mean minimum daily temperature (MIN), and the mean daily difference between daily maximum and minimum temperature (DIF). It was assumed that during the first three days after inoculation the temperature under the tunnel was 3 C higher than outside. This was estimated based on a limited number of observations made under the tunnel during the course of the experiment using a minimum-maximum thermometer.

Data analysis and model development. The incubation period of Cercospora carotae was studied on 150 carrot plants during this experiment. The number of lesions on each of

the two inoculated leaves per plant was summed. The total number of lesions per plant was then transformed to proportion of maximum number of lesions (PML) by dividing the number of lesions observed on each reading by the number of lesions on the last reading (which was the maximum) so that the data ranged from 0 to 1.0.

Influence of weather variables on incubation period. The first part of the analysis consisted of developing simple models that predict the beginning, mean, and end of incubation from selected weather parameters. The influence of weather variables on incubation period was determined following two distinct steps.

First, the beginning (IP5), mean (IP50), and end (IP95) of incubation period were calculated for each plant. Secondly, the incubation period for each plant, separately for IP5, IP50, and IP95, was regressed against various combinations of weather variables to find the best equation to predict the incubation period.

Calculation of IP5, IP50, and IP95. Several models including Gompertz, Richard and logistic models (Berger, 1981, Madden, 1986), were evaluated for their ability to describe the pattern of lesion appearance as a function of time. The logistic model (Eq.3.1) was found to be the most appropriate. This model was fitted to the proportion of

maximum number of lesions (PML) separately for each plant and each inoculation using SAS non-linear procedure with Marquart iteration method (PROC NLIN, SAS, 1987). A total of 150 logistic equations (15 plants/inoculation X 10 inoculations) were thus obtained in this step, each of them predicting the proportion of maximum number of lesions for an individual plant as a function of days after inoculation.

$$PML = \left(1 + e^{-(\ln(Y_0/(1-Y_0)) \quad -rt} \right) \qquad (3.1)$$

where PML is the proportion of maximum number of lesions; Y_0 = $((1-B_0)/B_0)$ and B_0 is the initial proportion of maximum number of lesions (first reading); r is rate of increase in proportion of maximum number of lesions; t is time in days. The value of B_0 was fixed to 0.00001 since no lesion was observed on any of the plants on the first reading.

The beginning, mean, and end of incubation periods for PML= 0.05, 0.50, and 0.95, respectively, for each individual plant were then calculated by incorporating the value of the estimated rate parameter (r) obtained for each plant into a logistic equation.

Regression of IP5, IP50, and IP95 on weather variables. The dependent variables were either IP5, IP50, or IP95. The

independent variables (LW, RH, DT, MAX, MIN, and DIF) were calculated for each inoculation from monitored hourly weather data. The weather variables were calculated from the day of inoculation to the day corresponding to IP5, to IP50 and to IP95 as determined by the logistic equations (Eq. 3.1).

Regression analyses were performed separately for IP5, IP50, and IP95 using SAS general linear modelling procedure (PROC GLM, SAS, 1986). Calculated incubation values for all plants and all inoculations were pooled so that the prediction equation would include a range of different temperatures, leaf wetness, and high relative humidity durations. The form of the linear regression (Eq. 3.2) was chosen so that it would include linear and quadratic effects and a linear interaction effect of the two independent variables. This model was chosen because of it simplicity and it was adequate for other pathogens (Shearer and Zadoks, 1974).

$$IP = B_0 + B_1X_1 + B_2X_2 + B_3X_1^2 + B_4X_2^2 + B_5X_1X_2 \qquad (3.2)$$

where, IP is the incubation period , X_1 and X_2 are weather variables (LW, RH, DT, MAX, MIN or DIF), and B1 to B5 are partial regression coefficients. Eight different models were evaluated including X_1=LW or RH and X_2=DT, MAX, MIN, or DIF (Table 3.3). These models were evaluated first based on coefficients of determination. The models with higher R^2

values were then further analyzed based on regression of predicted on observed values (normal plot). Ideally the plot of predicted incubation period on observed incubation period should fall on a straight line with an intercept of zero and a slope of 1.0 (Draper and Smith, 1981, Thal et al., 1984).

General model for lesion appearance under field conditions.

The second part of the analysis consisted in developing a general model that predict the appearance of lesions as a function of the number of days after inoculation regardless of the environmental conditions. To do so, the proportion of maximum number of lesions (PML) for all plants and all inoculations (150 plants) were pooled and regressed over the number of days after inoculation. A logistic model (Eq. 3.3) was fitted to the data using SAS non-linear regression procedures with Marquart iteration method (SAS, 1987).

$$PML = \left(1 + e^{-(\ln(Y_0/(1-Y_0)) \; -rt} \right) \qquad (3.3)$$

where PML is the proportion of maximum number of lesions; $Y_0=((1-B_0)/B_0)$ and B_0 is initial proportion of maximum number of lesions (first reading); r is rate of increase in proportion of maximum number of lesions; t is time in days. The value of B_0 was fixed to 0.00001 since no lesion was

observed on any of the plants on the first reading. The logistic model was evaluated based on the correlation coefficient between observed and predicted proportion of maximum number of lesion, and size of asymptotic standard error associated with the estimated parameters. Where a small asymptotic standard error would indicate a good fit of the model (Draper and Smith, 1981, Thal et al., 1984).

RESULTS

In general, lesion appearance started and ended at 6-8 and 12-14 days after inoculation, respectively. The pattern of lesion appearance over time for individual plants was very similar for all inoculations, but the time scale varied according to prevailing weather conditions. The period over which the lesions appeared varied from 3 to 5 days. Mean daily temperature $\geq$ 21 C resulted in short incubation period (mean IP = 8 days) and mean daily temperature below 21 C resulted in longer IP (mean IP of 10-11 days) (Table 3.1 and 3.2).

Table 3.1. Predicted beginning, mean ,and end of incubation period as calculated from the logistic regression of proportion of maximum lesions against the number of days after field inoculation of carrots leaves by C. carotae.

INO.[w] #	Date of sowing	Date of inoculation	Rate[x]	Calculated incubation[z] r[y]	IP5	IP50	IP95
1	May 22	June 26	1.1899	0.997	9	12	14
2	May 25	June 29	1.3731	0.967	8	10	12
3	May 29	July 03	1.3965	0.998	8	10	12
4	June 6	July 11	1.7026	0.984	6	8	10
5	June 8	July 13	1.5668	0.998	7	9	11
6	June 11	July 16	1.7593	0.986	6	8	10
7	June 14	July 19	1.4613	0.990	7	9	11
8	June 22	July 27	1.3784	0.963	8	10	12
9	July 2	Aug. 06	1.3713	0.975	8	10	12
10	July 4	Aug. 08	1.2299	0.996	9	11	14
		Mean	1.4430	0.985	7.6	9.7	11.8

[w] Inoculation number.

[x] Rate of lesion production per inoculation estimated from the logistic equation, average of rate estimates of 15 plants per inoculations.

[y] Coefficients of correlation between observed and predicted proportion of maximum lesions, average of 15 regressions.

[z] Average incubation calculated by incorporating the rate estimate into the logistic equation (Eq. 3.1) for PML= 0.05, 0.50 and 0.95 to calculate IP5, IP50, IP95, respectively. Average of 15 plants per inoculation.

Table 3.2. Weather variables used to predict the beginning, mean, and end of incubation period of Cercospora carotae under field conditions.

	Weather during incubation[z]					
INO#	LW	RH	DT	MAX	MIN	DIF
IP5						
1	17.44	17.11	17.62	22.27	12.40	9.87
2	17.62	15.00	17.22	22.06	11.35	10.71
3	17.65	12.87	19.15	24.04	12.66	11.38
4	20.00	15.83	19.30	25.94	11.59	14.35
5	12.57	11.00	22.56	28.28	16.38	11.90
6	17.17	16.83	23.55	27.29	19.98	7.31
7	15.57	17.57	21.23	25.37	17.05	8.32
8	16.00	10.00	22.07	28.45	15.40	13.05
9	19.75	16.12	19.32	23.29	15.23	8.06
10	18.00	17.67	18.28	24.42	14.45	9.97
IP50						
1	16.75	14.67	17.43	22.29	11.84	10.45
2	17.00	13.30	17.41	22.45	11.29	11.16
3	17.00	11.50	18.69	23.83	11.97	11.86
4	15.62	11.87	20.69	26.93	13.84	13.09
5	11.89	11.78	22.18	27.46	16.69	10.77
6	16.75	16.50	22.57	26.35	19.02	7.33
7	14.22	15.11	21.57	25.53	16.56	8.97
8	14.50	10.50	22.24	29.61	15.82	12.79
9	19.20	15.40	19.12	23.36	15.07	8.29
10	17.27	17.00	19.75	25.25	14.21	11.04
IP95						
1	16.71	13.64	17.89	22.82	12.25	10.57
2	16.33	11.92	18.04	23.05	11.92	11.13
3	16.50	11.08	18.51	24.24	10.99	13.24
4	14.40	11.30	21.12	26.62	15.05	11.57
5	11.00	12.45	21.71	26.71	16.59	10.12
6	14.00	15.20	22.22	26.74	18.13	8.61
7	13.18	14.09	21.81	26.52	16.11	10.41
8	16.08	11.33	21.63	27.19	16.03	11.15
9	18.41	15.42	19.35	24.12	14.74	9.37
10	15.56	15.43	18.88	24.48	12.42	12.06

[z] The weather variables were calculated from the day of inoculation until 5, 50 or 95 of lesions appearance. LW is the mean daily duration of leaf wetness (hr), RH the mean daily duration of relative humidity $\geq$ 90% (hr), DT the mean daily temperature (°C), MAX the mean maximum daily temperature (°C), MIN the mean minimum daily temperature (°C), and DIF the mean daily difference between daily maximum and minimum temperature (°C)

Influence of weather variables on incubation period. The high coefficients of correlation for the logistic regression describing proportion of maximum number of lesions (PML) on the number of days after inoculation ranged from 0.96 to 0.99 (Table 3.1), indicated that this model was valid and useful in interpolating IP5, IP50, and IP95. However, this model tended to predict IP5 earlier than observed when lesions appeared over a very short period of time ($\leq$ 3 days). Nevertheless this phenomenon was observed on less than 14 % of the plants examined.

The coefficients of determination of the regression models describing the relationship between the incubation period and various combinations of weather variables are presented in Table 3.3. Although R^2 values are significant ($P<0.01$) for all models the proportion of the variation in incubation period explained by the different models varied from 0.92 for the model IP50=f(RH,DT) to 0.20 for the model IP95=f(RH,DIF).

Because of the low proportion of the variation explained by some models, only those with R^2 values $\geq$ 65 % were retained for further analysis. An exception to this was the model IP5=f(RH,DT) which has a R^2 value of 0.56, this model was considered because the relationship produced a very high R^2 values of 0.92 and 0.89 for IP50 and IP95. When R^2 values were equal, the model including the dependent variable RH was preferred over the variable LW because RH is

routinely available from most weather stations. The plot of predicted incubation period against observed incubation period for the four best models for each dependent variable are presented in Figures 3.1 to 3.3. For most of the regression lines fitted to predicted incubation period against observed incubation period, the intercept estimates were significantly higher than 0 as determined by a t-Test. This suggested that the models overestimated incubation period.

The best relations were IP5=f(LW,DT), IP50=f(RH,DT), and IP95=f(RH,DT) for the beginning, mean, and end of incubation period. For these models, the majority of the predicted incubation periods are clustered around the normal line (Fig. 3.1-3.3). However, for the relation IP5=f(LW,DT) a difference in DT of one degree (°C) or in LW of one hour delayed IP5 by 4 to 8 days making this model biologically unacceptable. For this reason the relation IP5=f(RH,DT) was preferred. This model explained only 56% of the variation in IP5, however, the predicted IP5 fall within the limits of IP5 observed in the field. The relationship between the beginning, mean, and end of incubation, mean daily hour of RH $\geq$ 90%, and temperature are presented in figures 3.4-3.6. For all these models, the relationship between the incubation period and one variable depended on the level of the other variable (Fig. 3.4-3.6).

Table 3.3. Regression models predicting the incubation period for Cercospora carotae from weather variables under field conditions.

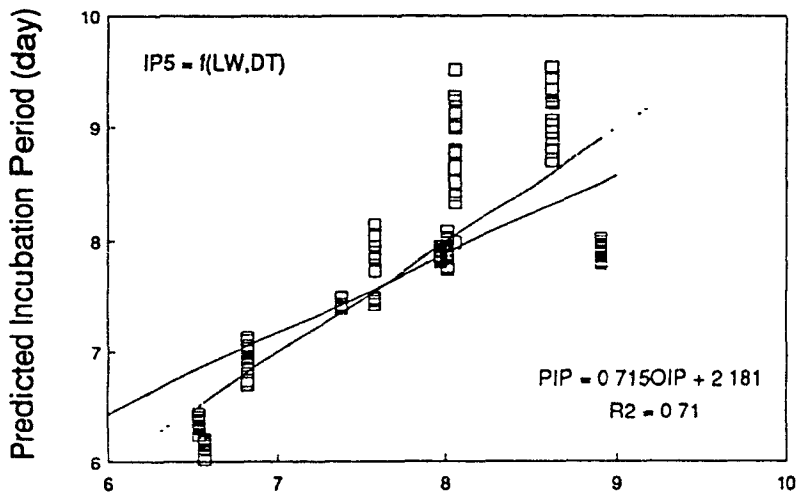
Y	Variables[x] X1	X2	R^{2y}	Range[z]	
IP5	LW	DT	0.72	13<LW<20	17.2<DT<23.5
IP5	LW	MAX	0.64	13<LW<20	23.3<MAX<28.3
IP5	LW	MIN	0.71	13<LW<20	11.3<MIN<20.0
IP5	LW	DIF	0.69	13<LW<20	8.00<DIF<14.3
IP5	RH	DT	0.56	11<RH<18	17.2<DT<23.5
IP5	RH	MAX	0.57	11<RH<18	23.3<MAX<28.3
IP5	RH	MIN	0.23	11<RH<18	11.3<MIN<20.0
IP5	RH	DIF	0.37	11<RH<18	8.00<DIF<14.3
IP50	LW	DT	0.49	12<LW<20	17.4<DT<22.6
IP50	LW	MAX	0.49	12<LW<20	22.3<MAX<28.6
IP50	LW	MIN	0.39	12<LW<20	11.3<MIN<19.0
IP50	LW	DIF	0.78	12<LW<20	8.30<DIF<13.1
IP50	RH	DT	0.92	11<RH<17	17.4<DT<22.6
IP50	RH	MAX	0.89	11<RH<17	22.3<MAX<28.6
IP50	RH	MIN	0.80	11<RH<17	11.3<MIN<19.0
IP50	RH	DIF	0.46	11<RH<17	8.60<DIF<13.1
IP95	LW	DT	0.77	11<LW<18	17.9<DT<22.2
IP95	LW	MAX	0.67	11<LW<18	22.8<MAX<27.2
IP95	LW	MIN	0.51	11<LW<18	11.0<MIN<18.1
IP95	LW	DIF	0.60	11<LW<18	8.60<DIF<13.2
IP95	RH	DT	0.88	11<RH<15	17.9<DT<22.2
IP95	RH	MAX	0.72	11<RH<15	22.8<MAX<27.2
IP95	RH	MIN	0.67	11<RH<15	11.0<MIN<18.1
IP95	RH	DIF	0.20	11<RH<15	8.60<DIF<13.2

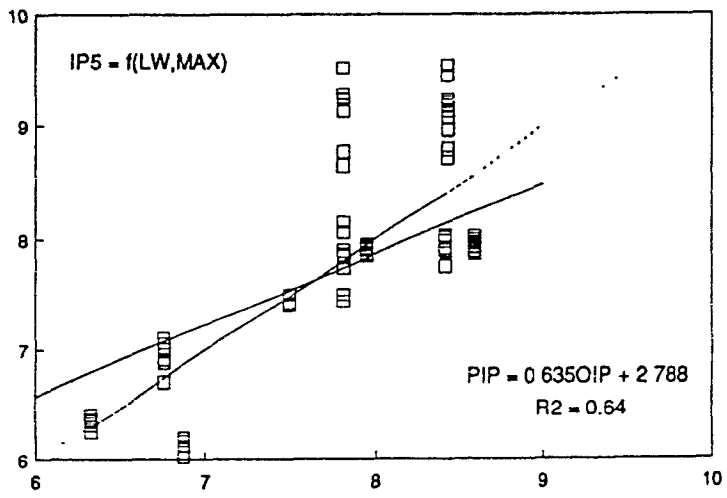
[x] The dependent variables were the beginning (IP5), mean (IP50) and end (IP95) of incubation period defined as the period in days for the appearance of 5, 50 and 95 % of the lesions, respectively. The independent variable X_1 was LW or RH, as mean daily duration of leaf wetness and relative humidity > 90%, respectively. The variable X_2 was DT, MAX, MIN, or DIF as mean daily temperature, maximum temperature, minimum temperature, difference between maximum and minimum temperature. (see text).

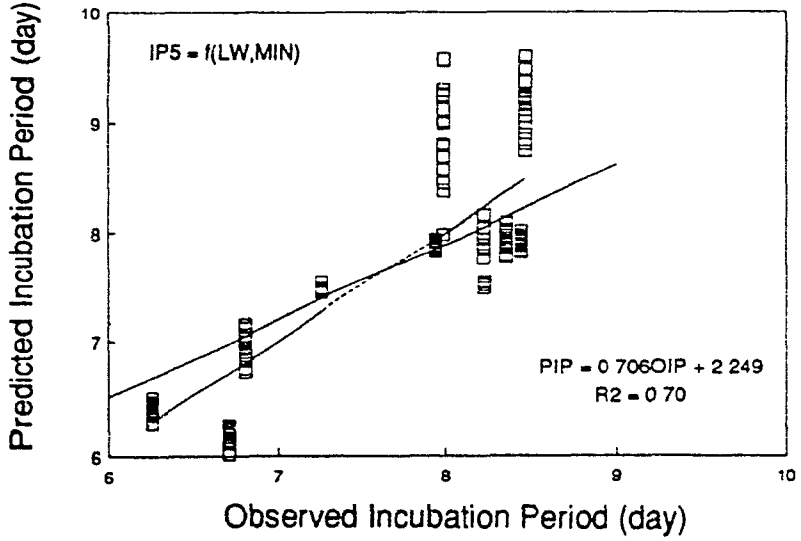
[y] Coefficient of determination for the various model tested (Eq. 3.2).

[z] Range of weather conditions observed during the course of the experiment.

Figure 3.1. Relationship between predicted (PIP) and observed (OIP) beginning of incubation period (IP5) of C. carotae. Predicted incubation periods were calculated using equation 3.2 and various combinations of two dependent variables as indicated on the graph (meaning of the symbols used for the dependents variables are given in the text). The dotted line represents the normal line and the solid line represents the linear regression of PIP against OIP.


IP5 = f(LW,DT)
PIP = 0 715OIP + 2 181
R2 = 0 71
Predicted Incubation Period (day)


IP5 = f(LW,MAX)
PIP = 0 635OIP + 2 788
R2 = 0.64


IP5 = f(LW,MIN)
PIP = 0 706OIP + 2 249
R2 = 0 70
Predicted Incubation Period (day)
Observed Incubation Period (day)

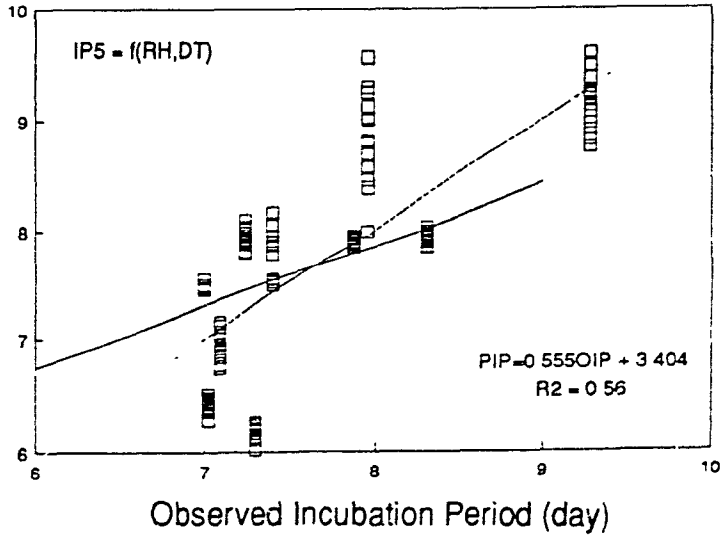
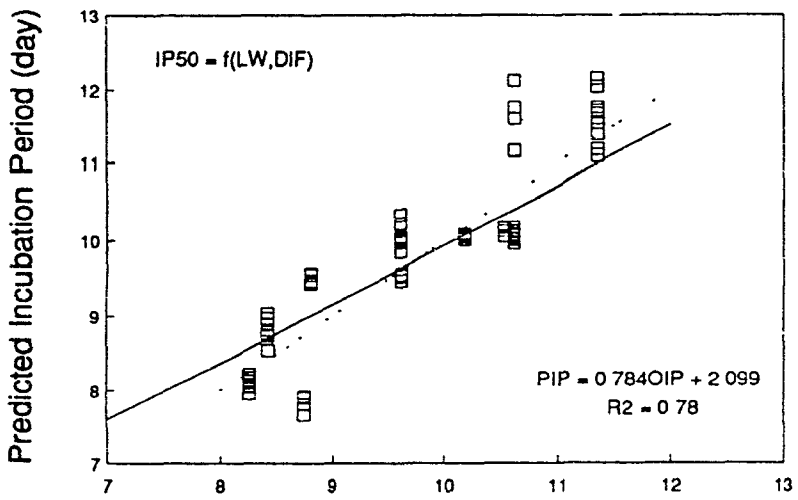
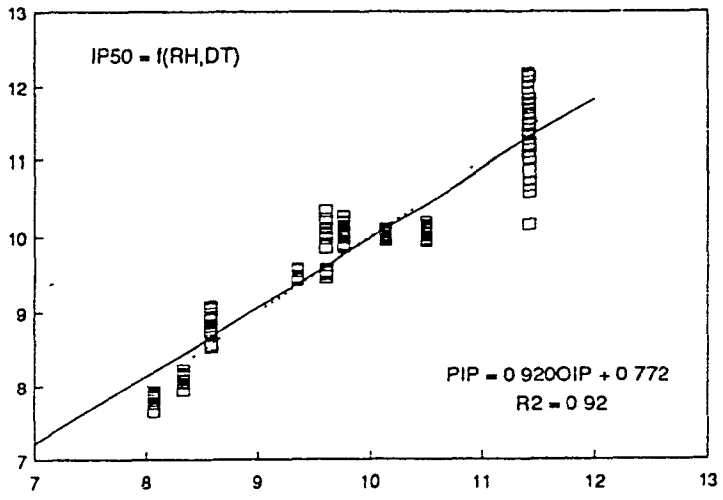
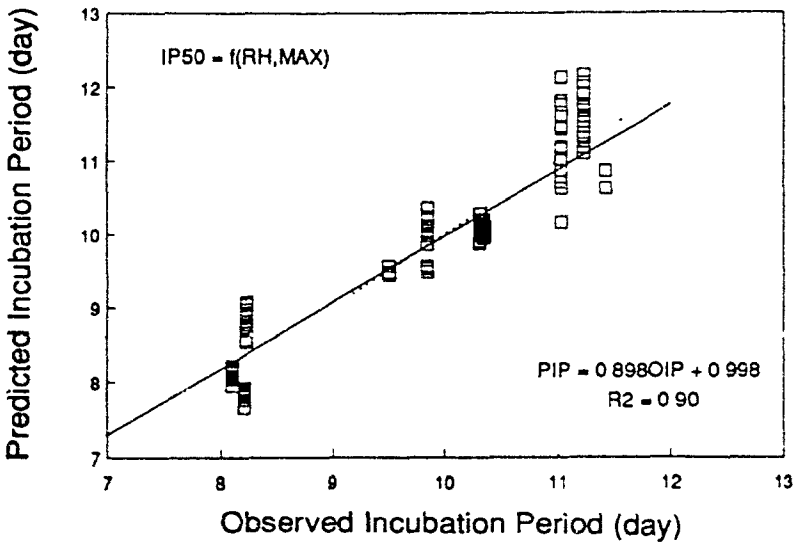

IP5 = f(RH,DT)
PIP=0 555OIP + 3 404
R2 = 0 56
Observed Incubation Period (day)

Figure 3.2. Relationship between predicted (PIP) and observed (OIP) mean incubation period (IP50) for C. carotae. Predicted incubation periods were calculated using equation 3.2 and various combinations of two dependent variables as indicated on the graph (meaning of the symbols used for the dependents variables are given in the text). The dotted line represents the normal line and the solid line represents the linear regression of PIP against OIP.


IP50 = f(LW,DIF)
PIP = 0 784OIP + 2 099
R2 = 0 78
Predicted Incubation Period (day)


IP50 = f(RH,DT)
PIP = 0 920OIP + 0 772
R2 = 0 92


IP50 = f(RH,MAX)
PIP = 0 898OIP + 0 998
R2 = 0 90
Predicted Incubation Period (day)
Observed Incubation Period (day)

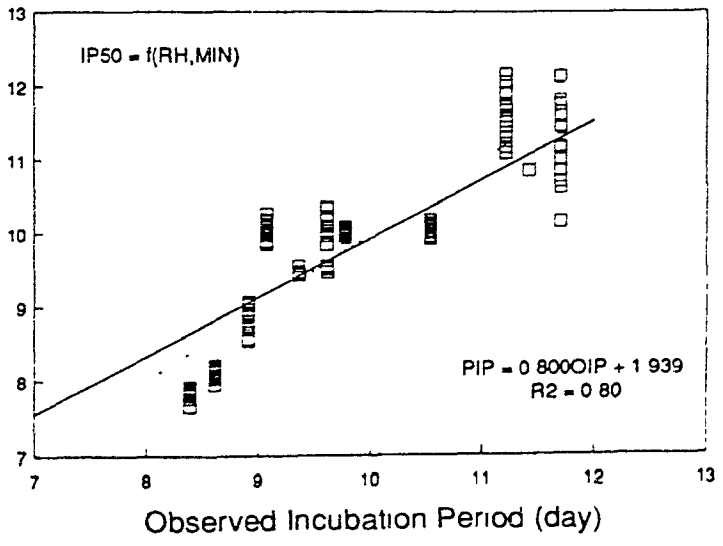
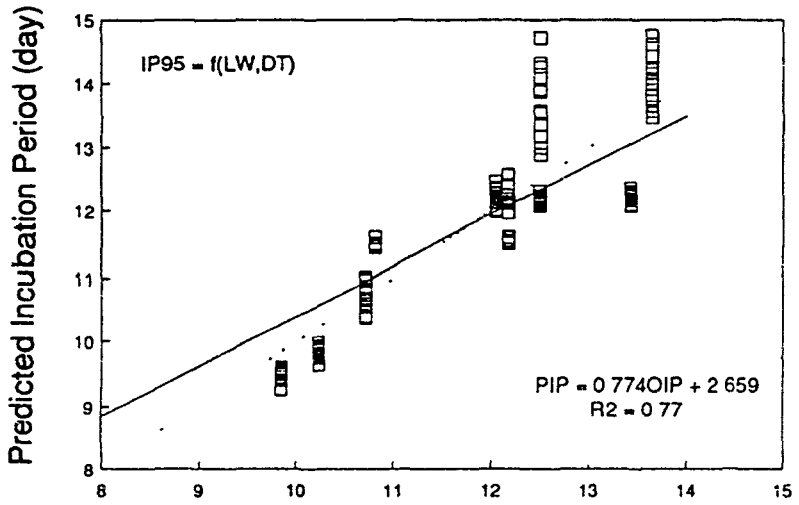
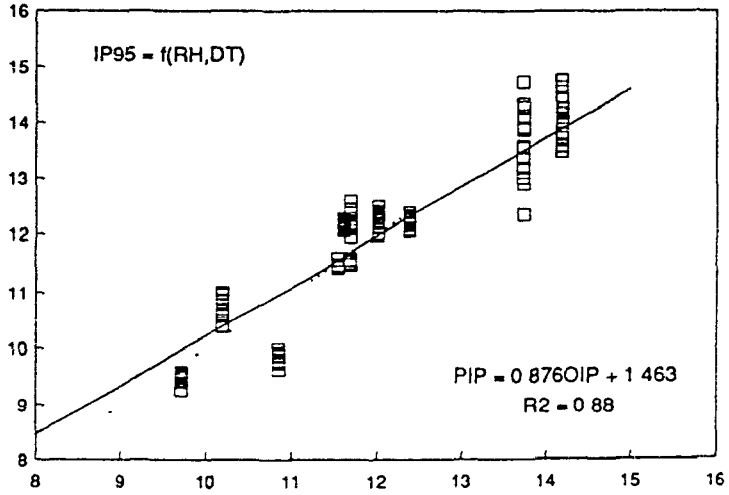
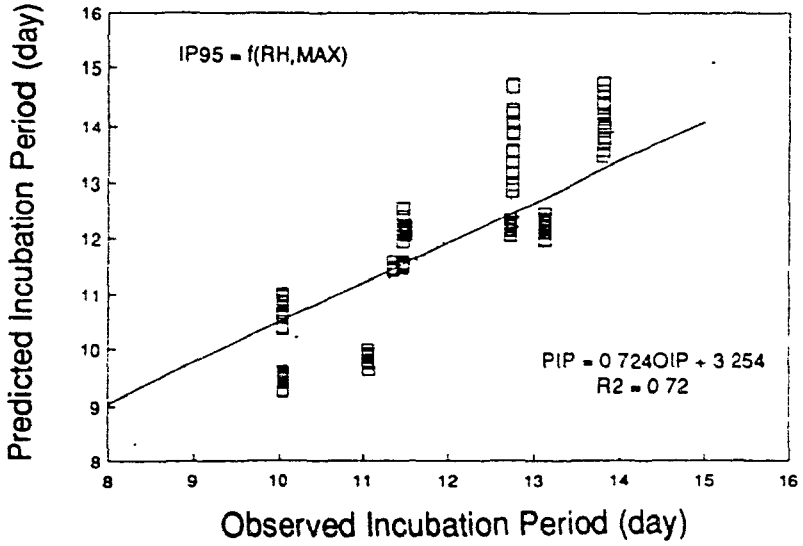
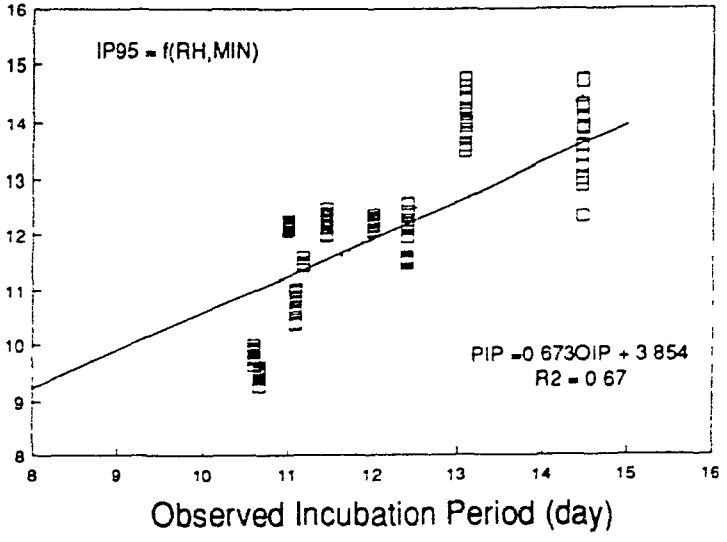

IP50 = f(RH,MIN)
PIP = 0 800OIP + 1 939
R2 = 0 80
Observed Incubation Period (day)

Figure 3.3. Relationship between predicted (PIP) and observed (OIP) end of incubation period (IP95) for C. carotae. Predicted incubation periods were calculated using equation 3.2 and various combinations of two dependent variables as indicated on the graph (meaning of the symbols used for the dependents variables are given in the text). The dotted line represents the normal line and the solid line represents the linear regression of PIP against OIP.


IP95 = f(LW,DT)
PIP = 0 774OIP + 2 659
R2 = 0 77
Predicted Incubation Period (day)


IP95 = f(RH,DT)
PIP = 0 876OIP + 1 463
R2 = 0 88


IP95 = f(RH,MAX)
PIP = 0 724OIP + 3 254
R2 = 0 72
Predicted Incubation Period (day)
Observed Incubation Period (day)


IP95 = f(RH,MIN)
PIP =0 673OIP + 3 854
R2 = 0 67
Observed Incubation Period (day)

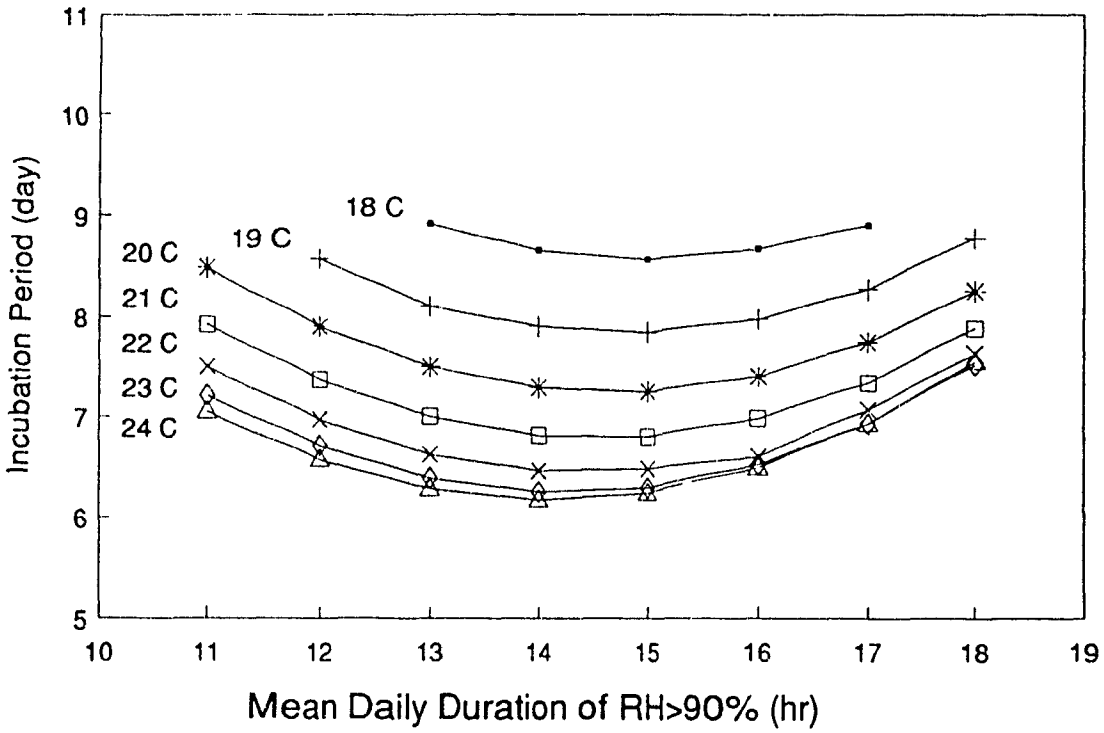


Figure 3.4. Relationships between beginning of incubation period (IP5), mean daily hour of relative humidity ≥90%, and temperature. The regression equation predicting the beginning of incubation period is $IP5 = 71.7739-3.1999X_1 - 3.5747X_2+0.0914X_1^2+0.0667X_2^2+0.0256X_1X_2$, where IP5 is the beginning of incubation period, X_1 is the mean daily hours of RH≥90% and X_2 is the mean daily temperature (see text).

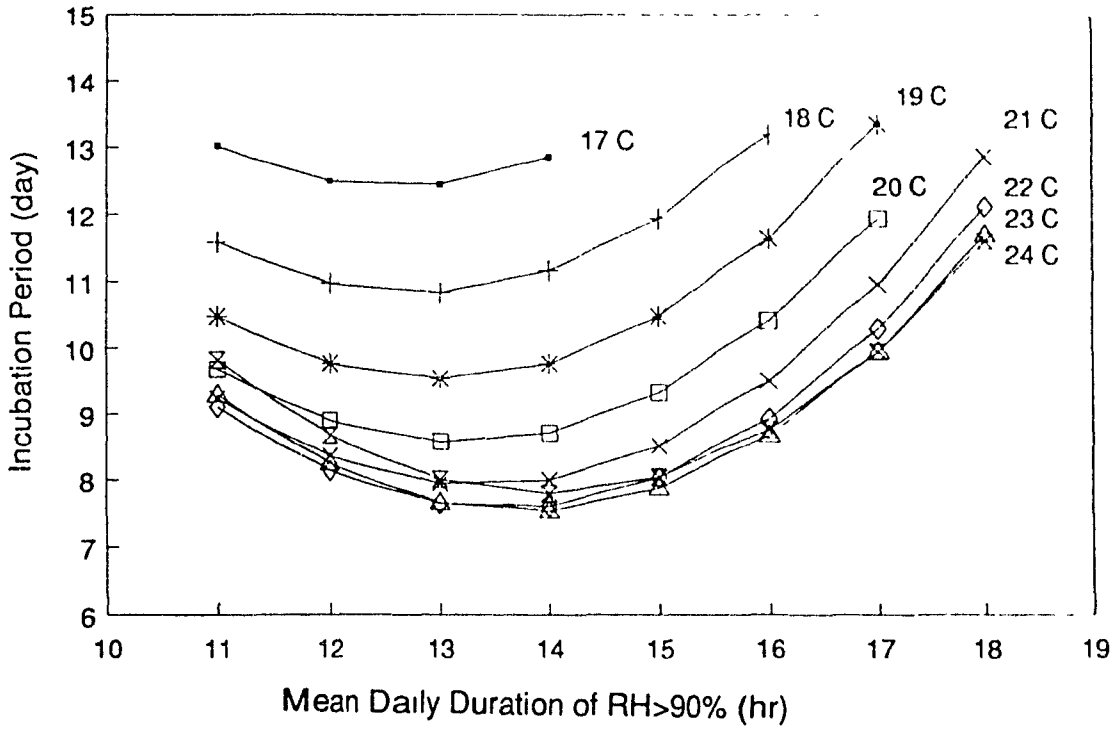


Figure 3.5. Relationships between mean of incubation period (IP50), mean daily hour of relative humidity $\geq$90%, and temperature. The regression equation predicting the mean incubation period is $IP50 = 107.1844-4.3537X_1-6.1736X_2+0.2320X_1^2+0.1632X_2^2-0.0882X_1X_2$, where IP50 is the mean incubation period, X_1 is the mean daily hours of RH$\geq$90% and X_2 is the mean daily temperature (see text).

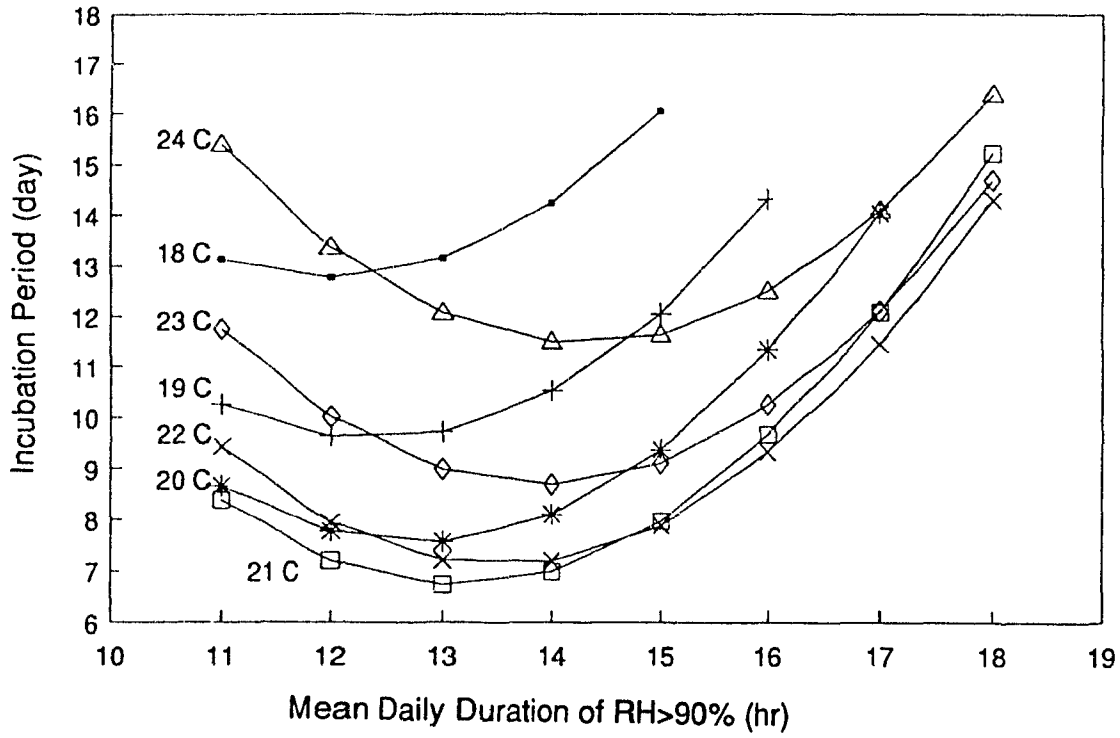


Figure 3.6. Relationships between end of incubation period (IP95), mean daily hour of relative humidity ≥90%, and temperature. The regression equation predicting the end of incubation period is IP95 = $284.6881-3.6273X_1-23.9811X_2+0.3604X_1^2+0.6529X_2^2-0.2785X_1X_2$, where IP95 is the end of incubation period, X_1 is the mean daily hours of RH≥90% and X_2 is the mean daily temperature (see text).

General model for lesion appearance under field conditions. In general, the first lesions were seen 6 to 8 days after inoculation (Fig. 3.7). The proportion of maximum number of lesions reached the maximum at around 11 to 14 days after inoculation (Fig. 3.7). However, some lesions appeared as early as 5 days after inoculation and some lesions were seen as late as 16 days after inoculation. The plot of proportion of maximum number of lesions against days after inoculation produced a sigmoid curve. The logistic model explained 84% ($r=0.917$) of the proportion of maximum number of lesions as a function of the number of days after inoculation. The estimated rate of lesion appearance was 1.41 with a small asymptotic standard error of 0.007 indicating the appropriateness of the model. However, the model was less accurate in predicting the end of incubation period (Fig.3.7).

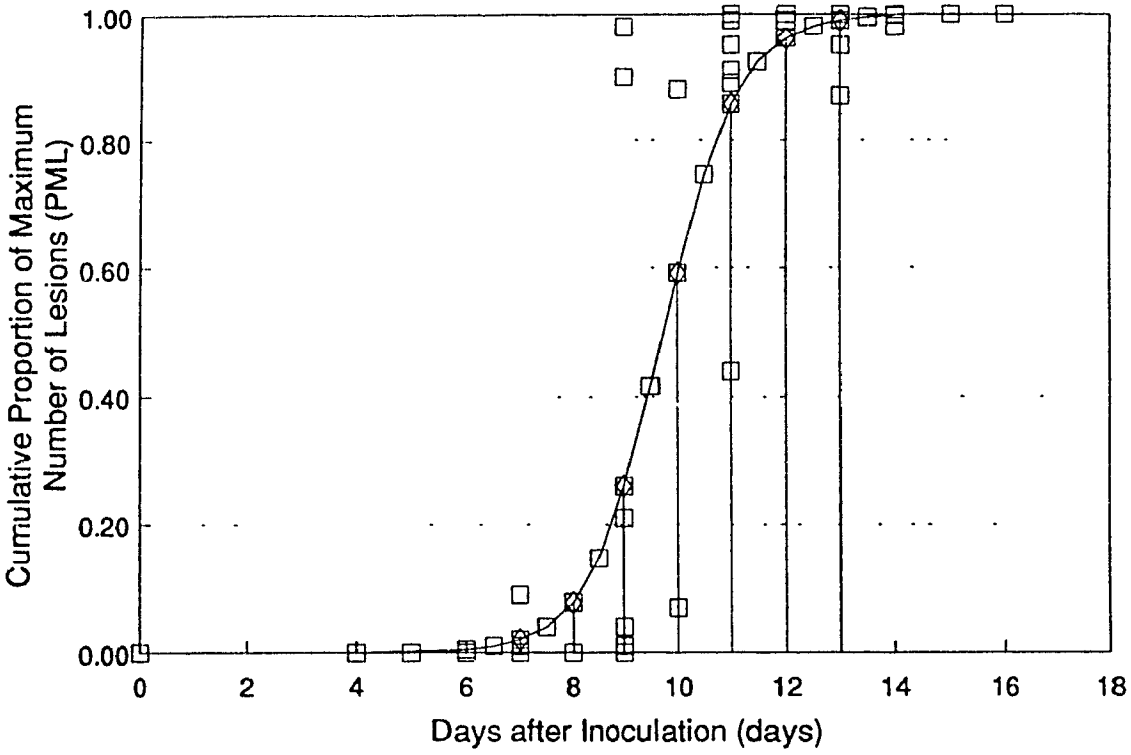


Figure 3.7. Relationship between predicted and observed proportion of maximum number of lesions (PML). The predicted values were calculated using equation 3.3, where $Y_0=(1-B_0)/B_0$ and B_0 is the initial PML and is equal to 0.0001; r is the rate of increase in PML and is equal to 1.41; t is the time in days from inoculation.

DISCUSSION

The mean incubation period of Cercospora carotae ranged from 8 to 12 days for different inoculations. Warm temperature ($\geq$ 21 °C) associated with prolonged high RH period ($\geq$ 12 hr/day) resulted in a short incubation period (IP5=8 days). This experiment demonstrated that incubation period of Cercospora carotae was influenced not only by the daily temperature but also by the duration of high relative humidity or leaf wetness. This phenomenon was observed for other fungi (Shaw, 1986, Shearer and Zadoks, 1972, Shearer and Zadoks, 1974). Because of the complex relationship between incubation period, temperature and duration of high moisture, and the difficulty in describing these relations mathematically, a regression model including linear, quadratic and a linear interaction effect was chosen. This model, even if empirical, was useful in identifying the weather parameters that have the greatest influence on incubation period. However, this model can be used only within the range of mean daily hours of RH $\geq$90% and temperature studied. Out of this range the predicted incubation period would not be biologically meaningful.

The period over which lesions appeared varied from 3 to 5 days, but always followed the same patterns of appearance, a sigmoid curve. The weather variables tested here influenced the length of incubation period but not the pattern of lesion appearance which was in all cases

adequately explained by a logistic function.

There are various definitions of latency period, the most common being by Shearer and Zadoks, 1972, who defined the latency period as the interval between inoculation and the first appearance of sporulating structures. The same definition can be used for the incubation period as the interval between inoculation and the first appearance of lesions. However, this definition causes problem when the intervals are not the same for all lesions which is expected while working with a population of fungal spores for which the appearance of lesions is expected to follow the normal distribution. Furthermore, this definition is not related to the intrinsic growth rate of the population of lesions (ie. does not describe lesion appearance over time).

An alternative definition was proposed by Johnson, 1980 and Shaw 1986, as the time of visible appearance of 50% of the final number of lesions (mean incubation period, MIP). However, this method of measuring the incubation period does not describe the range or the spread in incubation (ie. time of appearance of first lesions and time between beginning and end of incubation). The spread of incubation is an important parameter determining the shape (pattern) of population growth. If the spread is wide, the effect of perturbation on the population of spores will disappear, meaning that a wide spread in incubation can make an

epidemic resulting from a single event into an apparently continuous epidemic (Royle et al., 1986). Ideally a study of incubation period should thus describe both the temporal scale on which lesions appear following infection at a single time and the pattern of that appearance. To do so, the pattern of lesion appearance must be adequately described by a mathematical model which will allow interpolation between observations.

Shaner (1980), proposed the probit analysis to linearize sigmoid curves and to estimate T_{50} (mean IP). This method is appropriate when the T_{50} is used as a parameter to evaluate levels of resistance of different cultivars or in comparative epidemiology. However, when information on incubation is to be used for simulation or epidemiological modelling, it becomes important to describe with more details, the pattern of lesion appearance and the effect of weather on incubation period. In this work it was possible to describe the pattern of lesion appearance using a logistic function and to predict the beginning, mean, and end of incubation period from the mean daily hours of RH $\geq$90% and temperature.

Results of this study can be used to predict the development of Cercospora blight based on temperature and leaf wetness or high moisture duration since weather conditions that favor short incubation period will also favor epidemic development. The effect of variable

incubation period on epidemic development is complex. In Quebec, we can expect the incubation period to be long early and late in the season when the temperature is relatively low for C. carotae which can reduce the rate of disease progression during these periods. On the other hand, during the middle of the season, temperature is higher resulting in greater rate of disease progression. The models developed in this study can be utilised for simulation, where it is possible to integrate into a simulation model the effect of the environment on variable incubation period. However, the error associated with the type of models developed in this study is important. A small change in mean daily temperature or duration of high relative humidity may result in large change in predicted incubation period. This may not reflect on what happens in the field where the length of incubation period is limited by the genetic potential of the pathogen. According to our observations mean incubation period of Cercospora carotae of less than 6 days or greater than 14 days is not likely to occur in carrot field in Quebec independently of the weather conditions. The effect of varying incubation period on natural disease progress is complex and difficult to model (Berger and Jones, 1985), and thus several disease prediction models assumed a fixed incubation period. The general model predicting the lesion appearance as a function of day after inoculation, developed in this experiment, can be used to predict the lesion

production regardless of weather conditions.

In conclusion, this study demonstrated that the temperature and the duration of high relative humidity are important parameters affecting the incubation of Cercospora carotae. However, other factors that influence the length of incubation period have to be investigated. The proposed models were developed using the cultivar, Dagger, and one inoculum concentration so there is limitation on application for other cultivars and inoculation conditions.

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STUDIES ON POLYCYCLIC PROCESS

PREFACE TO STUDY IV

Because the different phases of the pathogen life cycle act together to create an epidemic, it is indispensable to understand the contribution of each on the overall result. The effect of each epidemic components on an epidemic can not be determined only by measuring its importance in a monocyclic experiment. However, these experiments are essential to quantify the components themselves, but the effect of all components on an epidemic must be defined with caution and may be difficult to interpret because these components act together and their effects are cumulative over the course of the epidemic.

Simulation modelling serves to organize available information about a pathosystem. The simulation model presented here contains series of simple equations that describe mathematically several biological processes such as infection, sporulation, and incubation.

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STUDY IV

DEVELOPMENT AND VALIDATION OF A MODEL TO PREDICT CERCOSPORA BLIGHT UNDER FIELD CONDITIONS

ABSTRACT

A mathematical model that simulates the effect of the physical environment on Cercospora blight of carrot was developed and validated. The model was derived from controlled condition studies on infection and sporulation and from a field study on incubation period. The simulation model uses projection matrix approach of population growth to predict disease severity from the interaction among predicted inoculum (PINOEE), infection (INFEE) and sporulation (SPOEE) equivalent for the environment. Inoculum was modeled as a function of daily INFEE and daily proportion of the maximum number of new lesions (PLES). The daily proportion of new lesions was a function of the incubation period treated as a distributed delay process. Infection equivalent was modeled as a function of leaf wetness or RH$\geq$90% duration and mean temperature during this period. Sporulation equivalent was modeled as a function of total duration of leaf wetness during the preceding five days and mean temperature during this period. The model was validated by comparing disease progress observed in the field for ten epidemics with disease progress generated by

the simulation model using weather data recorded in 1987, 1988, and 1990. The model was quite realistic when compared with field data. In general, the simulated epidemics were similar to those observed using the area under the disease progress curve and the regression of simulated disease severity on observed disease severity as criteria for comparison. However, the model was more accurate when weather data were collected in the plot for which simulation was run. When weather data were collected in other plots, the model generally predicted the pattern of disease progress but the onset was either too early or too late.

INTRODUCTION

Carrot (Daucus carota L. var sativa) is among the most important vegetable crops in Quebec, with an annual value of 17 million can$ in 1986 (Statistics Canada, 1988). In 1989, carrots alone represented 17.7% of the vegetable production value in Quebec (MAPAQ, 1989). Cercospora blight is a leaf spotting and blighting disease of carrots. The disease, caused by Cercospora carotae (Pass) Sohl., is important in Quebec, other provinces in Canada and also in the United States (Arcelin and Kushalappa, 1991, Hooker, 1944, Thomas, 1943). Leaves weakened by blight often break-off when gripped by mechanical harvesters, resulting in unharvested roots. Presently, it is a common practice to apply 3 to 7

fungicide sprays during each cropping season to manage Cercospora blight in the organic soil region of Quebec.

Recently, methods have been developed to help in reducing the number of fungicide applications required to control the disease. These methods are based on an observed or predicted critical disease level (CDL) at which the first fungicide must be applied. Kushalappa et al. (1989) proposed to use the number of days after emergence or the mean time after disease detection of 89 and 42 days, respectively, as action thresholds to initiate fungicide applications. A sequential sampling program has also been developed to facilitate the quantification of critical disease level (CDL) of 50 and 80% disease incidence to initiate fungicide applications (Boivin et al., 1990). These methods, however, depend on disease evaluation in the field, and thus require monitoring by scouts which can be expensive and time consuming. A model to predict Cercospora blight progress in the field would be a valuable research tool and can provide a framework for the development of a forecaster.

One important problem while developing simulators is to account for the incubation period. The effect of incubation on natural disease development may be very complex. When infection occurs on a given day (i) not all the lesions developing from that infection will appear on the same day

one incubation period later. The appearance of lesions will be distributed over several days or weeks depending on the pathogen, environmental conditions and host resistance. Because of this problem, Berger and Jones (1985) proposed a general model that include parameters for variable latency period. However, not all the information required to use this model was available for C. carotae. An alternative to Berger and Jones's model and to dynamic simulation for which knowledge of programming language is a prerequisite, was to use projection matrices to predict the growth of lesion population resulting from several infections (Bruhn and Fry, 1981, Caswell, 1989).

The objectives of this study were first to combine and synthesize the available information on Cercospora blight (Carisse and Kushalappa, 1990, 1991, study II, study III) and construct a simple simulation model describing the progress of Cercospora blight of carrot under field conditions. Secondly, to identify areas of future research that would be valuable in improving the understanding of this pathosystem.

MATERIALS AND METHODS

Theoretical basis of the model. For experimentation, a concept is needed for guidance in the interpretation of

epidemic structure, pattern and dynamic. Our concept was that Cercospora blight epidemic is governed mainly by the interaction among inoculum, infection, and sporulation as influenced by the environment (Table 1). In this model the effect of the environment on inoculum, infection and sporulation are quantified and expressed as equivalents for the environment. The environment equivalent values ranged from 0 to 1.0, where 1.0 represented the highest potential for these epidemiological processes (Kushalappa et al.,1983, Kushalappa, 1989). The three components of the model were, a) the predicted inoculum equivalent for the environment resulting from previous infections (PINOEE), b) the infection equivalent for the environment (INFEE), c) the sporulation equivalent for the environment (SPOEE).

In carrot blight, it is assumed that each effective infection by the fungus C. carotae produces a single lesion. There is no evidence that a single infection by C. carotae will give rise to more than one lesion. The disease becomes severe only after several successful infections that generate many lesions. Thus, Cercospora blight epidemic can be treated as an increase in a population of lesions.

Appearance of lesions is a function of the infection that occurred one incubation period earlier. If weather was favorable for infection it can be expected that many lesions will be produced and if weather was not favorable then only a few lesions will be produced. However, extension of

Table 4.1. Physical environment parameters affecting the development of Cercospora carotae used to simulate Cercospora blight of carrot epidemics.

Stage of pathogen development	Parameters affecting the process
Sporulation	Duration of leaf wetness or RH>95%, Temperature
Infection	Duration of leaf wetness or RH> 89%, Temperature
Incubation (Visible lesions)	Days after inoculation, magnitude of the infection

controlled conditions data from monocyclic infection experiments to predict the increase in lesion population in the field is complicated by the time-delay effect of the incubation period. When carrot plants were inoculated in the field with conidia of C. carotae, the first lesions appeared on the 6th day. More lesions appeared each day until the 13th or 14th day (study III). Thus, even when all infections took place during one day some infection units required more time than other to produce a lesion. In other words, the effect of a single time of infection can be seen over a period of time. To address this problem the incubation of C. carotae was treated as a delay function process while developing the simulation model (Berger and Jones, 1985).

Model description. The model was designed to predict the disease progress over the course of a growing season of naturally infected carrot, with a time step of one day. Like most simulation models (Knudsen et al., 1987) the state diagram of the model is similar to the pathogen life cycle diagram. A flow chart of the model illustrating the relationships between the model components is presented in Figure 4.1. However in this case, matrix notation provided a better representation of the model than standard state diagrams (Bruhn and Fry, 1981).

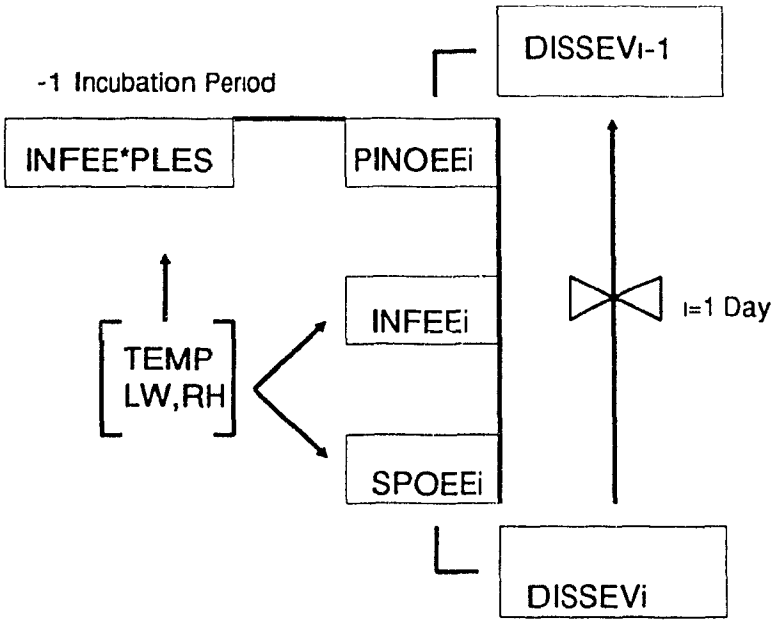


Figure 4.1. Diagram for a simulation model of Cercospora blight of carrot. The state variables, disease severity (DISSEV), is represented in rectangular boxes and other boxes represent the other variables: predicted inoculum (PINOEE), infection (INFEE), and sporulation (SPOEE) equivalents for the environment used to calculate DISSEV. Flow of influence of weather variables temperature (TEMP), duration of leaf wetness (LW) and relative humidity (RH) is indicated with arrows and d is the number of days after an infection and d=7,8...13.

Calculation of PINOEE. The increase in lesion population is dependent on the magnitude of infection as influenced by the environment and time between infection and appearance of lesions. To predict the increase in lesion population which can be seen as inoculum source for further infection cycle, and thus was denoted by PINOEE (predicted inoculum equivalent for the environment), the following equation was used:

$$PINOEE_i = \sum_{i=1}^{n} (INFEE_{i-d} * PLES_i) \qquad (4.1)$$

where, PINOEE is the daily predicted inoculum equivalent for the environment resulting from previous infections; n is the total number of days during the cropping season; d is the number of days after infection where, d=7,8,...13; $INFEE_{i-d}$ is the infection equivalent for the environment (infection potential) on the (i-d) th day; PLES is the proportion of the maximum number the lesions that will appear on the ith day (Fig.4.2).

The incubation period was treated as a delay function process, where all the lesions resulting from a given time of infection are appearing over a period of 6 days (Fig.4.2). To solve equation 4.1 for any ith day it is necessary to look back 7 to 13 days to see what was the infection potential ($INFEE_{i-d}$) on that day. PLES was

calculated as a function of the number of days after infection using equation 4.2:

$$PLES = \left(1 + e^{-(\ln(Y_0/(1-Y_0)) - rt} \right) \qquad (4.2)$$

where PLES is the proportion of maximum number of lesions; $Y_0=((1-B_0)/B_0)$ and B_0 is the initial proportion of maximum number of lesions and is equal to 0.0001; r is the rate of increase in cumulative proportion of the maximum number of lesions and is equal to 1.41; t is time in days after infection (study III). When equation 4.2 was solved the following values of PLES were obtained: 0.02, 0.08, 0.26, 0.60, 0.86, 0.96, and 0.99 for the 7th to the 13th day following infection, respectively. Matrices calculations were used to determine the daily INO values resulting from previous infections (Fig. 4.2)

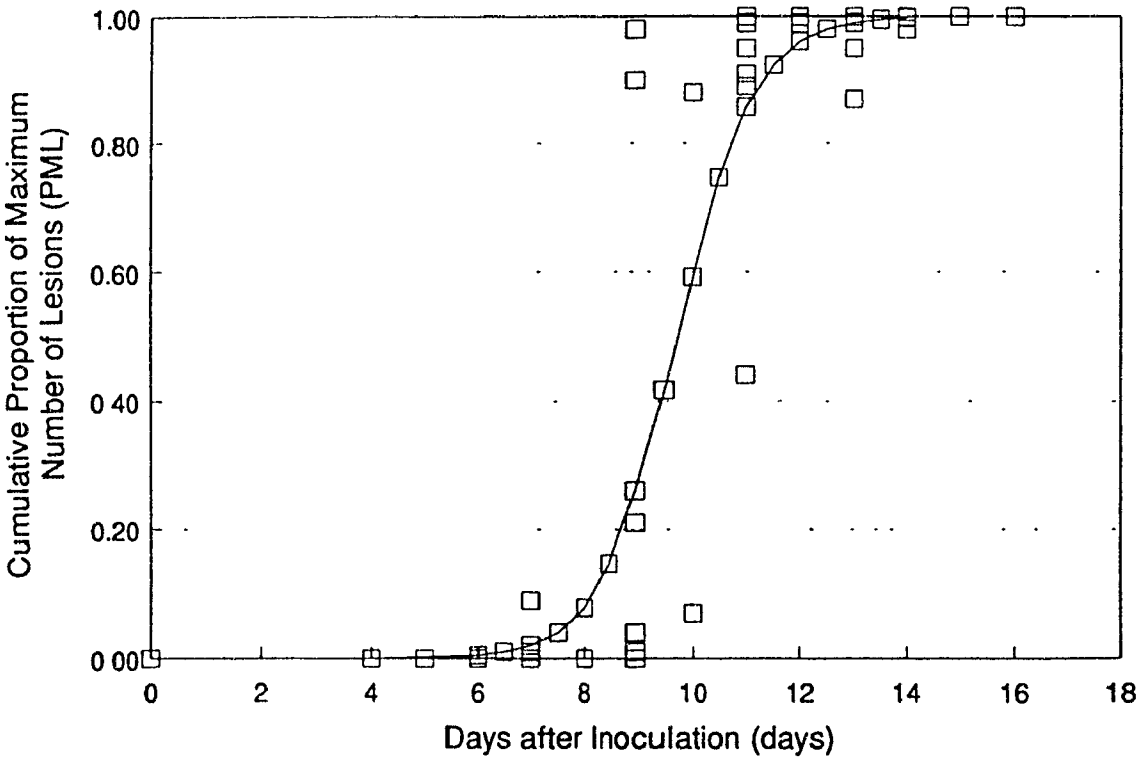


Figure 4.2. Course of appearance of lesions of Cercospora carotae on cultivar Dagger, following inoculation at day 0. Data from study III.

Construction of projection matrices. Projection matrices have been used mainly for animal and insect population dynamic studies (Caswell, 1989). These demographic analyses are often based on a system of tabulating age-specific survival and reproduction known as life table. A population of lesions can be grouped into age-specific groups corresponding to the infection from which the lesions arose and a cohort life table can be constructed (Zadoks and Schein, 1979). From this table it is possible to project the growth of the population of lesions using simple matrices calculation.

The first step in constructing a projection matrix is to put the unit to be projected into groups. In this case the proportion of lesions (PLES) multiplied by the infection equivalent ($INFEE_{i-d}$) on day i is the unit of projection. In other words, all the lesions resulting from infection occurring on one day are considered to be in one group.

The second step consists of choosing a projection interval defining the time step of the projection matrix. Here, seven projection intervals corresponding to 7 to 13 days after an infection, have been selected for any given group. The predicted inoculum equivalent for the environment on day i in each of the seven age groups are represented by the vector PINOEE described by the following:

$$PINOEE = INFEE_{i-d} * PLES_{i} \qquad (4.3)$$

Where, PLES describes the proportion of lesions appearing on day i, and INFEE describes the magnitude of infection that had generated these lesions and depends on weather factors (Table 4.1).

For instance, the PINOEE value on a given day could be

$$\text{PINOEE} \quad \begin{vmatrix} (0.64*0.86) \\ (0.32*0.60) \\ (0.27*0.26) \\ (0.81*0.08) \\ (0.94*0.02) \end{vmatrix} = \begin{vmatrix} (0.550) \\ (0.192) \\ (0.070) \\ (0.065) \\ (0.018) \end{vmatrix} = \text{PINOEE}=0.895$$

Where, the predicted inoculum equivalent for the environment on that day is equal to 0.895 resulting from five infections. On that day 86, 60, 26, 8, and 2% of the lesions from the first to the fifth infection, respectively, are expected to appear. An example of these calculation for several days is given in Table 3.2. This matrix represents the day-to-day change in inoculum potential associated with each age group.

Table 4.2 Example of a projection matrix used to calculate the daily predicted inoculum equivalent for the environment (PINOEE) values used to simulate Cercospora blight of carrot development.

DAY	1	2	3	4	n
INFEE	0.2562	0.0002	0.6514	0.9875	INOEE[y]
1					
2					
3					
4					
5					
6					
7	(0.2562*0.02)[z]				0.005
8	(0.2562*0.08)+(0.0002*0.02)				0.021
9	(0.2562*0.26)+(0.0002*0.08)+(0.6514*0.02)				0.080
10	(0.2562*0.60)+(0.0002*0.26)+(0.6514*0.08)+(0.9875*0.02)				0.226
11	(0.2562*0.86)+(0.0002*0.60)+(0.6514*0.26)+(0.9875*0.08)				0.469
12	(0.2562*0.96)+(0.0002*0.86)+(0.6514*0.60)+(0.9875*0.26)				0.894
13	(0.2562*0.99)+(0.0002*0.96)+(0.6514*0.86)+(0.9875*0.60)				1.407
.					
.					
n					

[y] PINOEEi is calculate using equation 4.1 (see text).

[z] PLES values are 0.02, 0.08, 0.26, 0.60, 0.86, 0.96, 0.99, for 7, 8, 9, 10, 11, 12, and 13 days after infection as calculated using equation 4.2 (see text).

Calculation of INFEE. A previously developed infection function relating the INFEE to temperature and leaf wetness duration was used to calculate the INFEE values (Eq. 4.4-4.5) (Carisse and Kushalappa, 1990):

$$INFEE = K\ (1 + e^{-(0.642W+0.063TW-0.0013T^2)})^{1/(1-1.02)} \quad (4.4)$$

$$K = -6.1633+0.5941T-0.0124T^2 \quad (4.5)$$

where INFEE is the infection equivalent for the environment, K is the asymptote (highest infection potential for any given temperature), T is temperature (°C), and W is the duration of leaf wetness (hr). This function is based on temperature and continuous leaf wetness. However, for more realistic predictions and simplicity, the following assumptions were made (Carisse and Kushalappa, 1991): a) relative humidity $\geq$ 90% is equivalent to leaf wetness in promoting infection; b) two leaf wetness or RH $\geq$90% periods separated by a dry period greater than 6 hr and less or equal to 12 hr were added together (including the dry period); c) any dry period $\leq$ 6 hr were treated as a continuous leaf wetness period d) the presence of a dry period greater than 12 hr ended an infection period. The cumulative duration of leaf wetness (or equivalent conditions) and mean temperature during this period were used as input in the model.

Calculation of SPOEE. The daily sporulation equivalert for the environment on a given day (SPOEE) was derived from temperature and duration of leaf wetness (study II). Total number of hours of leaf wetness and mean daily temperature during the five preceding days (i-5) were used as input in the equation (Eq. 4.6-4.8). It was assumed that intermittent wetting does not reduce or enhance sporulation. The relationship between SPOEE, temperature and leaf wetness duration was calculated using a non-linear logistic function of the form:

$$SPOEE = \frac{PMS_m}{1 + [(PMS_m - PMS_0)/\ PMS_0]\ EXP(-rW)} \qquad (4.6)$$

$$PMS_m = -70.51 + 12.53T + 0.8247T^2 + 0.0239T^3 - 0.0002T^4 \qquad (4.7)$$

$$r = -0.7231 + 0.0797T - 0.0016T^2 \qquad (4.8)$$

where SPOEE is the sporulation equivalent for the environment on the (i-5)th day, PMS_m is the asymptote (highest sporulation potential for a given temperature), PMS_0 is the initial sporulation and is equal to 0.00001, r is the rate of sporulation for any given temperature, T is the temperature (°C) and W is the duration of wet period.

Calculation of DISSEV. The daily disease severity (DISSEV) was calculated by multiplying the daily predicted inoculum by the infection and by the sporulation equivalents for the

environment as follows:

$$DISSEV_i = PINOEE_i * INFEE_i * SPOEE_{i-5} \quad (4.9)$$

where $DISSEV_i$ is the predicted daily disease severity; $PINOEE_i$ is the daily predicted inoculum, $INFEE_i$, and $SPOEE_{i-5}$, are the infection and sporulation equivalents for the environment on the ith day, respectively.

Model performance. Collection of field data for model validation was conducted during the summers of 1987, 1988, and 1990. The carrot cultivar Dagger was used, and plots received no fungicide applications. Data used for validation were independent of those used for model development.

Data for validation : Plot establishment. Plots were established at the Agriculture Canada Experimental Farm at Sainte-Clotilde, Quebec. Each plot was 10 X 10 m, the distance between rows was 0.5 m and each plot consisted of 20 rows of 10 m long. Plots were mechanically prepared and fertilized according to Quebec government recommendations (CPVQ, 1988) and seeded with a mechanical seeder at a rate of 80 to 100 seeds per meter. There were four sowing dates each year and plots were seeded at two week intervals starting on May 12, May 17, and May 10 in 1987, 1988, and 1990, respectively. However, the plot of the first sowing

date and the plot of the last sowing date in 1988 and 1990, respectively, were not used due to adverse condition that affected either the emergence or the growth of the carrots. Data from a total of ten epidemics were thus used for validation.

Data for validation : Disease assessment. The plots were not inoculated with C. carotae and the disease was allowed to develop with just the inoculum surviving naturally. Cercospora blight assessment was started when carrots were at the three-leaf stage and was performed once a week until the disease started its exponential phase, and after that twice a week until the end of the cropping season. At each assessment, the number of lesions per leaf on 30 plants (15 marked and 15 at random) per plot was recorded. The marked plants were initially selected at random and identified at the first date of sampling. For both marked (same plants for all sampling dates) and randomly selected plants, the leaves were numbered (from bottom) and the number of lesions was recorded on each leaf separately. Only the fully expanded leaves were sampled and leaf fall was also recorded. Disease severity on individual leaves was expressed as infected area per leaf (INFAREA) calculated as:

$$\text{INFAREA} = \frac{\text{total \# of lesions/plant X ALA}}{\text{total \# of leaves present}} \qquad (4.10)$$

where INFAREA is the infected area per leaf (cm^2/leaf); ALA is the average lesion area. The ALA (0.06 cm 2) was estimated from a sample of five lesions per leaf on ten plants (total of 50 lesions) and the lesion area was estimated with a digital area meter (Decagon).

Data for validation : Weather monitoring. Weather data were monitored with different instruments depending on years; a mechanical (Belfort) and electronic (Agriscribe) hygrothermographs in 1987; and a datalogger (model CR-10, Campbell Scientific Instruments) in 1988 and 1990. In 1987, all measurements were done at 1 m from the ground except for the leaf wetness sensor which was installed within the plant canopy in the second plot (sown on May 25). In 1988, the RH and temperature were monitored at 1-min intervals with a sensor (P-207) located in a Stevenson shelter at 1 m from the ground. Leaf wetness was monitored with one sensor installed in the third plot (sown on June 16). In 1990, the RH and temperature were monitored at 1-min intervals with a sensor (model Vaisala) located in a Stevenson shelter at 1.0 m from the ground. The leaf wetness was monitored at 1-min intervals with sensors (model 237) installed within the plant canopy and moved up as the carrots grew. One sensor was placed in each of the different planting dates except for the last planting date. All weather data monitored with the datalogger were saved as 15-min averages. The wetness

sensor were calibrated by spraying both carrot leaves and the sensor with distilled water; the dry point was determined by visual observation of the carrots leaves. The calibration procedures were repeated twice in the laboratory and five times in the field at the time of instrument installation.

Validation. Simulation runs were conducted for each epidemic separately. The simulation runs were initiated 15 days after sowing. The model predicted disease severity for each remaining day of the cropping season for any particular plot. Observed infected area per leaf was transformed to percent disease severity by dividing values for each sampling date by the value observed on the last sampling date. Predicted disease severity was transformed to percent disease severity by dividing the values calculated for each day by the value calculated for the last day of simulation.

The reliability of the simulation model was evaluated based on the regression between simulated and observed disease severity and the area under the observed and simulated disease progress curve (AUDPC). The simulated values for each plot and each year were regressed against the observed values. A slope for the regression line of 1.0 would indicate a perfect relationship between simulated and observed values. The area under the disease progress curve

for each of the observed and predicted epidemics was estimated by the trapezoidal integration method (Eq.4.11):

$$AUDPC = \sum_{i=1}^{n-i} (((Y_i + Y_{i+1})/2)(X_{i+1} - X_i) \qquad (4.11)$$

where, Y is the infected area or the predicted disease severity on the i^{th} day; X is number of days since beginning (i=0).

RESULTS

Observed and predicted disease progress curves resulting from the validation are shown in Figures 4.3 to 4.5. Predicted disease progress curves generally followed the observed pattern of disease increase, although predicted disease did not always fall within the range of observed values. In general, the model overestimated disease severity and tended to predict disease too early as compared to field observations, with the exception of

Figure 4.3. Simulated (solid line) and observed (■) percent disease severity for Cercospora blight of carrot cultivar Dagger during the summer of 1987. A) represents data for an epidemic on carrots sown on May 12. B) represents data for an epidemic on carrots sown on May 25. C) represents data for an epidemic on carrots sown on June 8. D) represents data for an epidemic on carrots sown on June 22.

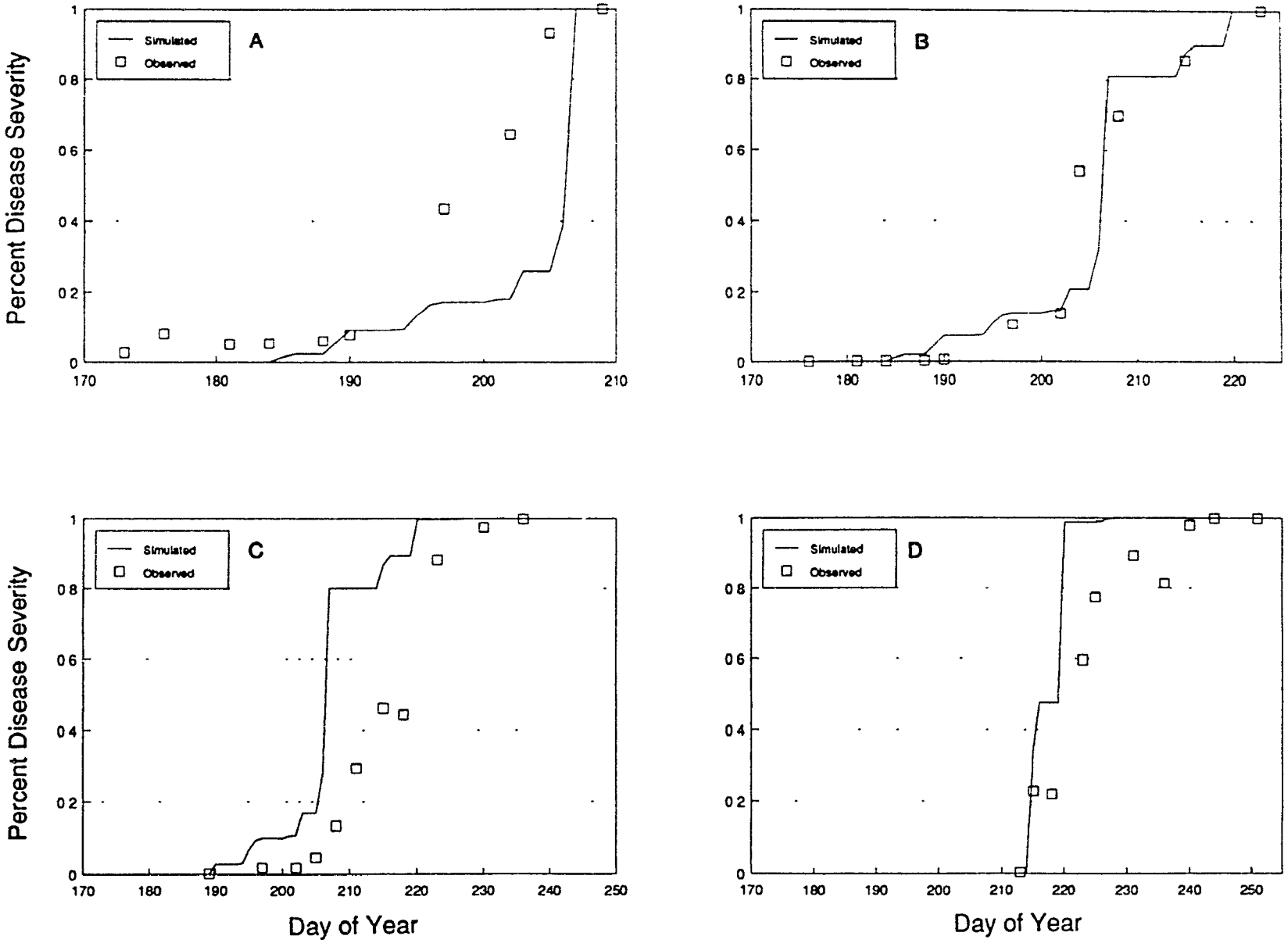
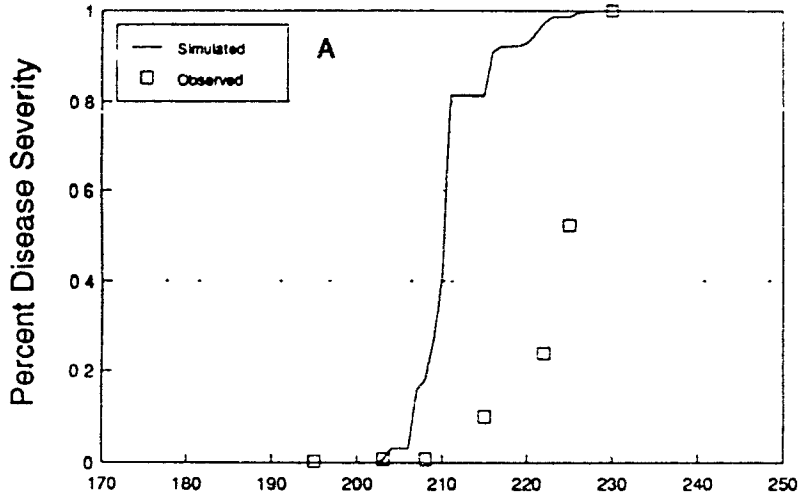

Percent Disease Severity
Simulated
Observed
A
B
C
D
0
0.2
0.4
0.6
0.8
1
170
180
190
200
210
220
230
240
250
Day of Year

Figure 4.4. Simulated (solid line) and observed (■) percent disease severity for Cercospora blight of carrot cultivar Dagger during the summer of 1988. A) represents data for an epidemic on carrots sown on June 1. B) represents data for an epidemic on carrots sown on June 16. C) represents data for an epidemic on carrots sown on July 1.


Simulated
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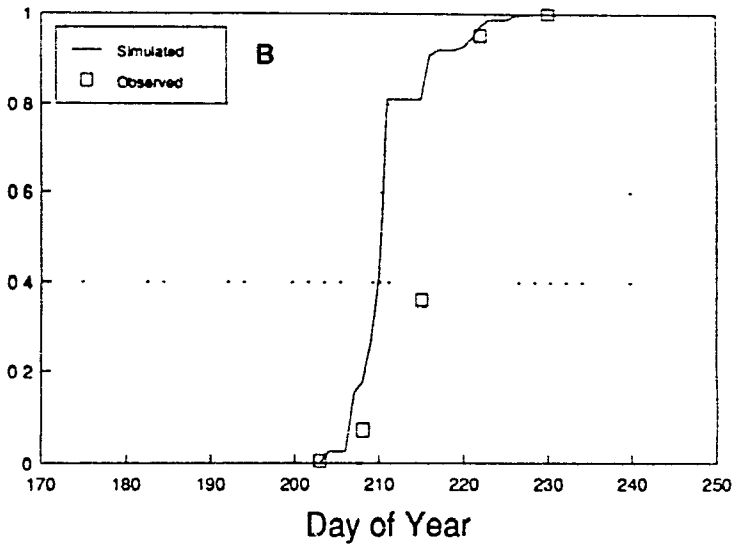
240
250


Simulated
Observed
B
0
0.2
0.4
0.6
0.8
1
170
180
190
200
210
220
230
240
250
Day of Year

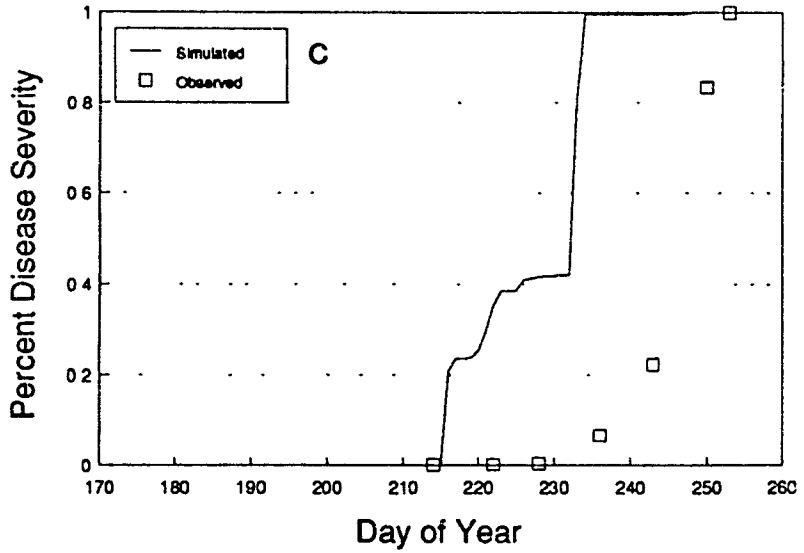
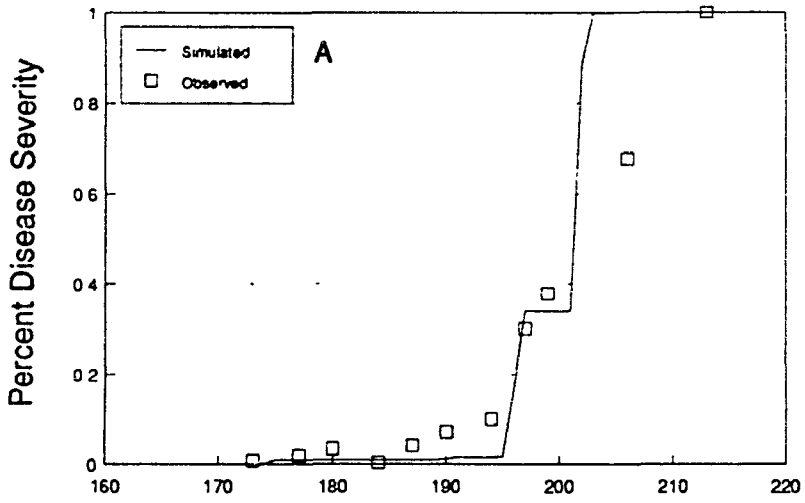
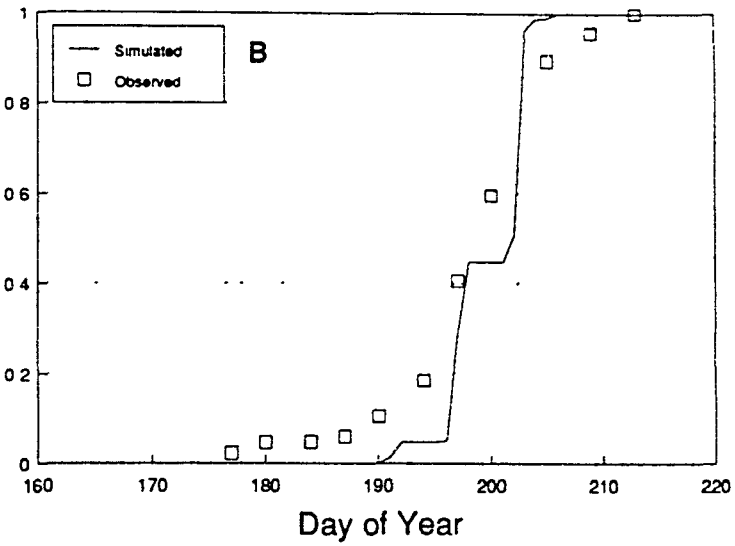
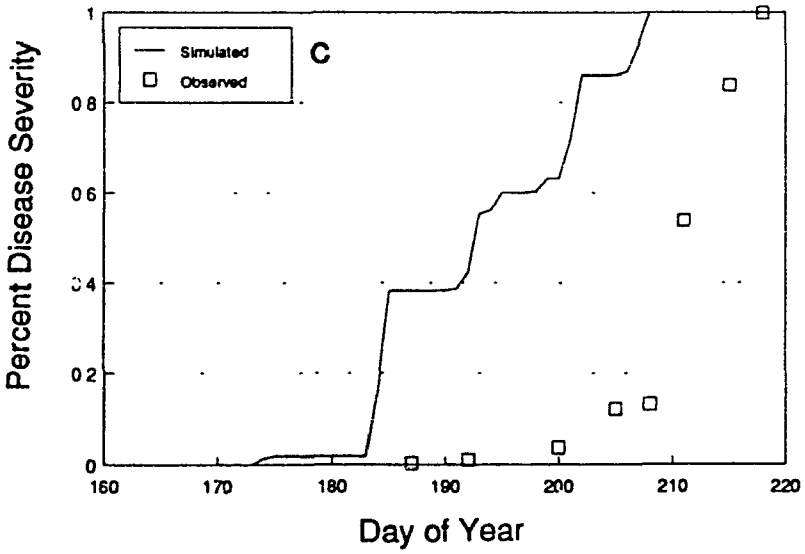

Simulated
Observed
C
Percent Disease Severity
0
0.2
0.4
0.6
0.8
1
170
180
190
200
210
220
230
240
250
260
Day of Year

Figure 4.5. Simulated (solid line) and observed (■) percent disease severity for Cercospora blight of carrot cultivar Dagger during the summer of 1990. A) represents data for an epidemic on carrots sown on May 10. B) represents data for an epidemic on carrots sown on May 25. C) represents data for an epidemic on carrots sown on June 9.


Simulated
Observed
A
Percent Disease Severity
0
0.2
0.4
0.6
0.8
1
160
170
180
190
200
210
220


Simulated
Observed
B
0
0.2
0.4
0.6
0.8
1
160
170
180
190
200
210
220
Day of Year


Simulated
Observed
C
Percent Disease Severity
0
0.2
0.4
0.6
0.8
1
160
170
180
190
200
210
220
Day of Year

the first epidemic of 1987 (Fig. 4.3A). It was observed that predictions were more reliable when weather data used as input in the model were collected in the plot for which the simulation was run.

The R^2 values for the linear regression of simulated against observed values for each plot and each year are presented in Table 4.3. The accuracy of the simulation model varied according to the plot and year. In general, higher R^2 values were obtained in 1987 (0.65, 0.92, 0.69, 0.88) and for plots in which the leaf wetness was recorded (R_2= 0.92, 0.82, 0.58, 0.98). Area under the simulated and observed disease progress curve (SAUDPC and OAUDPC, respectively) were similar for the following epidemics: 2 and 4 in 1987, 1 in 1988, and 1 and 2 in 1990, but large differences were obtained for the remaining epidemics (Table 4.3).

Table 4.3. Area under the disease progress curve and linear regression of simulated on observed percent disease severity of Cercospora blight of carrot during 1987, 1988, 1990.

	AUDPC		Regression	
Year	Simulated[1]	Observed	R^2	Slope[2]
1987				
Plot 1	5.144	11.757	0.65	1.0534 (0.2563)
Plot 2	17.404	17.064	0.92	0.9531 (0.0954)
Plot 3	28.898	19.810	0.69	0.7902 (0.1759)
Plot 4	26.190	21.148	0.88	0.9115 (0.1272)
1988				
Plot 2	18.098	6.566	0.51	0.5608 (0.2440)
Plot 3	17.598	14.089	0.82	0.9378 (0.2489)
Plot 4	26.332	7.744	0.40	0.6484 (0.3575)
1990				
Plot 1	13.096	11.584	0.58	0.5615 (0.1599)
Plot 2	13.248	14.965	0.98	0.8913 (0.0445)
Plot 3	25.438	7.516	0.45	0.7239 (0.3021)

[1] Simulation using weather data recorded during the epidemic development.

[2] Standard error on slope coefficient are shown in parentheses.

DISCUSSION

A model that simulates the progress of Cercospora blight of carrot from weather variables (duration of leaf wetness or high RH and temperature) was developed in this study. This model provided satisfactory simulations and explained about 0.69% of the variation in observed disease severity for the different epidemics and years. Although this is a relatively high value for this type of study, the model explained less than 55% of the variation for three of the ten epidemics observed, indicating that some factors still need to be considered.

During the three years of field data used for validation, the weather conditions varied substantially. In 1987, the weather conditions were moderately favorable for disease. The summer 1988 was very hot and dry, and thus less favorable to blight development. Finally, the summer 1990 was warm and humid and therefore extremely favorable. Nevertheless, the model adequately predicted the pattern of disease development under these three different weather conditions.

Given the limitation on the development of the model, the results of this study showed that Cercospora blight is very weather-dependent, and that conditions within the plant canopy greatly influence the disease development. This supports conclusion from other experiments showing that leaf wetness was an important weather parameter influencing

infection of carrot leaves by C. carotae (Carisse and Kushalappa, 1990, 1991). When leaf wetness data were collected in another plot the model failed to adequately predict disease. In these cases the microclimate created by the carrot leaves, prolonged leaf wetness periods, was not properly measured and inputed in the model. When leaf wetness data were correctly measured, the SAUDPC and OAUDPC were similar and the pattern of disease progress was accurately predicted.

The time difference between observed and predicted disease severity can also be explained by the use of a fixed incubation period even though the production of new lesions was considered to occur over a period of a few days. In the field, it is expected that the incubation period is longer early in the season while shorter later (study III). A varying rather than fixed incubation period in such simulation model would probably improve prediction of epidemics of early sown carrots (sown in May).

Generally, simulation models are very complex and require knowledge of computer programming. Projection of lesion population growth can involve many computations requiring the use of complex models. In this work the use of matrices calculation was considered to be a good alternative to complex models since all computations could be done using a standard spreadsheet program. Although

projection matrices are not widely used in plant disease epidemiology, they have been was used for the development of simulation models for potato late blight (Phyphthora infestans)(Bruhn and Fry, 1981). In this work it was found to be a valuable alternative to dynamic simulation in describing the population of lesions of C. carotae. However, there are several limitations inherent to the use of projection matrices, among them, the choice of a set of age class and the projection interval. A projection is an attempt to describe what will happen, given certain hypothesis, and thus assuming that the environment is constant and the density effect unimportant.

The model presented here is a simple simulation model and does not account for various components of epidemic such as increase in infected area, varying incubation period, infectious period, influx of inoculum, initial inoculum and leaf fall. The genetic potential and growth of the host also need to be considered. The model simulates the progress of Cercospora blight on the cultivar Dagger which is susceptible, however it can be used for other cultivars with variable level of resistance by adding a genetic coefficient to the model (Matyac and Bailey, 1988, Shaner and Hess, 1978). Nevertheless, this model could serve as a basis for forecasting and dynamic simulation. This proposed model has at least two advantages; Cercospora blight can be predicted without any disease monitoring in the field; the

calculations procedures are simple and can be done with standard spreadsheet computer programs.

One of the objectives of this work was to identify the need for further research. The following areas have been outlined as needing more data to confirm and strengthen the present model and as directions for future expansion of the model: 1) data on the influence of the environment on carrot growth; 2) data on the effect of the disease on yield and establishment of an economic thresholds; 3) data describing the effectiveness of fungicide applications on reduction of number of lesions or blighted tissues in the field; 4) more data concerning the interrelations between environment and spore production, availability (release and dissemination), and spore viability; 5) data on the influence of the environment on lesion expansion and relationships between lesion expansion and sporulation; 6) eventual application of the model to field situations so that management of Cercospora blight on carrot can be made both more efficient and economical.

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PREFACE TO STUDY V

Forecasting systems to time fungicide applications have been developed for several diseases such as potato late blight, early blight of tomato and cercospora blight of peanut. The success of these systems indicate that fungicides timed by monitoring the environment often control disease with fewer sprays and as are effective as fixed time interval schedules. Timing of fungicide application based on the accumulation of critical number of disease severity units has been employed with success to manage other polycyclic diseases (Krause et al., 1975, Madden et al., 1978). Forecasters can be used to time the initial fungicide application and the intervals between subsequent applications or only the initial application. The forecasting system for Cercospora blight predicts critical disease levels of 50 and 80% disease incidence and can be used to time the initial fungicide application only. The onset varies from late June to early August. Forecasting the time of onset of the explosive phase might save a considerable number of fungicide applications in some seasons.

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STUDY V

DEVELOPMENT OF A FORECASTING SYSTEM TO TIME THE INITIAL FUNGICIDE APPLICATION TO MANAGE CERCOSPORA BLIGHT OF CARROT.

ABSTRACT

A weather-based predictive system was developed to time the initial fungicide spray to manage Cercospora blight of carrot, induced by Cercospora carotae. The system used quantitative relationships of environmental variables and infection and sporulation of C. caroate established from controlled environment studies to predict a critical disease level. Daily weather variables were used to calculate daily infection (INFV) and sporulation (SPOV) values from which a cumulative blight severity values (CBSV) was computed. Forecasting systems based on INFV only (BSV1=INFV) and on INFV and SPOV (BSV2=INFV+SPOV) were tested for their reliability in predicting critical disease incidence levels of 50 and 80%. Field data collected during summers of 1987, 1988 and 1990 were used to evaluate the two systems. Predictions of critical disease level were more reliable using the system based on INFV only, the addition of SPOV resulted in unreliable predictions. For the ten epidemics used to test the system, disease incidence of 50% and 80% were observed when CBSV1 based on INFV ranged from 14 to 16 (mean of 14.9) and 18 to 20 (mean of 19.2), respectively.

Based on that, two thresholds were proposed. First, a warning threshold of CBSV1=14-16 at which fungicides may be applied only if conditions favorable to infection are probable. Secondly, a spray threshold of CBSV1=18-21 at which fungicide must be applied as soon as possible.

INTRODUCTION

Cercospora blight of carrot, caused by Cercospora carotae (Pass) Solh., is the principal foliar disease of cultivated carrots in Quebec (Arcelin, Kushalappa, 1991). The disease is characterized by dark lesions on leaves and petioles, first evident on the lower leaves. Severe infections weaken the foliage, resulting in increased loss during mechanical harvesting. Precise information on economic impact of the disease is not available, however, according to Gillespie and Sutton (1979), harvesting loss occurs when more than 10-20% of the leaf area is blighted.

The recommendation to manage Cercospora blight of carrot includes crop rotation on a three years schedule and application of protectant fungicides at 7 to 10-day intervals beginning when the carrot plants are 15 cm tall (Crête, 1981). These recommendations provide adequate control of Cercospora blight on carrots grown commercially in Quebec, but, result in sprays that are not necessarily properly timed and may be wasted if the weather is not

favorable to disease development. Depending on the years the onset of the Cercospora epidemic may occur as early as the end of June or as late as end of July. Consequently, it would be desirable to delay spraying as long as possible and still obtain effective control. In Quebec, some growers participate in the Integrated Pest Management program for southwestern Montreal and are advised to begin fungicide applications when Cercospora blight reaches a critical level of 80 and 50% disease incidence for the carrots sown in May and June, respectively. This provides growers with a warning that C. carotae is present at a potentially damaging level. Fungicide sprays initiated at these critical disease levels provide control equal to that obtaind from a standard timing spray program initiated at the beginning of the season (Boivin et al., 1990). Using this method it is possible to reduce the number of sprays required to control Cercospora blight. However, this method depends on disease monitoring in the field which can be expensive and is available only to those growers participating to the IPM program.

Timing the initial spray application for Cercospora blight control has been of concern recently. A method to forecast inoculum threshold based on plant growth stage, days since planting and degree days have been proposed (Kushalappa et al.,1989). A recently developed simulation model was accurate in modelling Cercospora blight in

experimental carrot plots in Quebec (study IV). However, this simulation model was not designed for timing of fungicide applications and can not be used directly in the field to manage Cercospora blight. Nevertheless, it can serve as a frame-work for the development of a forecasting system.

The objective of this study was to develop a weather-based forecasting system to time the initial fungicide application to manage Cercospora blight of carrots grown in the organic soil region of southwestern Montreal.

MATERIALS AND METHODS

Disease dynamic. Information on Cercospora carotae epidemiology is limited. The precise means of overwintering is still unknown, the fungus probably overwinters on crop debris and conidia produced on overwintering mycelium are the source of initial inoculum (Thomas, 1943). Environmental conditions favorable to initial inoculum production are unidentified. Secondary inoculum is produced on necrotic lesions of carrot leaves independently of leaf age (Carisse, unpublished). Conidia are mainly disseminated by wind and can be blown over long distance (Sherf and Macnab, 1986).

Forecasting system. The forecasting system was developed

based on two mathematical models that predict the infection and sporulation of Cercospora carotae from hours of leaf wetness and temperature. To determine the influence of weather on infection and sporulation, the hours of leaf wetness (or equivalent conditions) and temperature during this period were combined to derive daily infection (INFV) and sporulation (SPOV) values. Two different forecasting models were evaluated. First, a model based on infection only and secondly, a model based on both infection and sporulation.

Infection model. Infection of carrot leaves by C. carotae conidia is favoured by the presence of leaf wetness and temperature between 18 and 28°C (Carisse and Kushalappa, 1990). Infection may occurs after 12 hr of leaf wetness, but, optimal infection occur when the leaves remain wet for more than 24 hr and increased with increasing duration of leaf wetness. Maximum infection develop under free moisture and decrease with decreasing relative humidity from 100% (leaf wetness) to 84%. The infection under high relative humidity conditions is enhanced by a short leaf wetness period preceding a high RH period (Carisse and Kushalappa, 1991). Infection is affected by dry periods occurring within an infection cycle. The infection decreases linearly with increase in length of the dry period. However, spores can survive long dry periods ($\leq$ 24 hr) and resume growth

when rewet. The effect of a dry interruption on infection also depends on the duration of the initial wet period, the effect being less important as the duration of the initial wet period increase (Carisse and Kushalappa, 1991).

A mathematical model was previously developed to relate the infection to leaf wetness duration and temperature (Carisse and Kushalappa, 1990):

$$PML = K\ (1 + e^{-(0.642W+0.063TW-0.0013T^2)})^{1/(1-1.02)} \quad (5.1)$$

$$K = -6.1633+0.5941T-0.0124T^2 \quad (5.2)$$

where PML is the proportion of maximum number of lesions, K is the asymptote (highest PML value for any given temperature), T is temperature (°C), and W is the duration of leaf wetness (hr).

Sporulation model. A previous study on sporulation indicated that the fungus sporulates when the leaves are wet or exposed to high relative humidity conditions for more than 48 hr at temperature between 20 and 30°C (study II). For all temperatures, production of conidia increased as wetness or high RH duration increased from 48 to 96 hr. The sporulation was higher under leaf wetness than under high RH (96%), however, the sporulation started only after 12 hr when the high RH period was preceded by a 12 hr leaf wetness period. From these experiments a mathematical model was

developed to relate the sporulation to leaf wetness duration and temperature (study II):

$$PMS = \frac{PMS_m}{1 + [(PMS_m - PMS_0)/ PMS_0] \, EXP(-rW)} \quad (5.3)$$

$$PMS_m = -70.51 + 12.53T + 0.8247T^2 + 0.0239T^3 - 0.0002T^4 \quad (5.4)$$

$$r = -0.7231 + 0.0797T - 0.0016T^2 \quad (5.5)$$

where PMS is the proportion of maximum number of spores per lesion, PMS_m is the asymptote (highest PMS value for a given temperature), PMS_0 is the initial PMS value and is equal to 0.00001, r is the rate of sporulation for any given temperature, T is the temperature (°C) and W is the duration of wet period (hr).

Determination of infection values (INFV). Infection values were determined from quantitative relationships of temperature and leaf wetness duration. The predicted infection (PML) was calculated for temperatures of 16, 17, 18, ..., 32 °C and leaf wetness durations of 12, 18, 24, ..., 96 hr (17 temperatures X 15 durations) using equation 5.1 and 5.2. The predicted infection values (PML) were analyzed with cluster analysis to classify each combination of temperature and leaf wetness duration (total of 255) into different groups corresponding to different infection levels

(Romesburg, 1984, Sutton et al. ,1986). Three clusters were found to be significantly different (R^2=0.93). Each one of the cluster including the following range of PML values; $0.00 < PML \leq 0.15$, $0.15 < PML \leq 0.57$, and $0.57 < PML \leq 1.00$ for the first, second and third clusters, respectively. Infection values (INFV) of 0, 1, and 2 were assigned to these three categories corresponding to low, moderate, and high infection, respectively (Table 5.1).

Determination of sporulation values (SPOV). The sporulation values were fixed based on the effect of temperature and leaf wetness duration on sporulation C. carotae. The predicted sporulation (SPO) was calculated for temperature of 16, 17, 18, ..., 32 °C and leaf wetness durations of 6, 12, 18, ..., 96 hr (17 temperatures X 16 durations) using equations 5.3 and 5.4. The predicted sporulation (PMS) values were analyzed with cluster analysis to classify each combination of temperature and leaf wetness duration (total of 272) into different groups corresponding to different sporulation levels. Three clusters were established (R^2 =0.89) including sporulation (PMS) of $0.00 < PMS \leq 0.15$, $0.15 < PMS \leq 0.50$, and $0.50 < PMS \leq 1.0$ for the first, second and third clusters, respectively. Sporulation values (SPOV) of 0, 1, and 2 were assigned to these three categories corresponding to low, moderate, and high sporulation, respectively (Table 5.2).

Table 5.1. Infection values (INFV) based on temperature and leaf wetness duration.

	Duration of wet period (hr)											
Temp	D≤24	24 to 30	30 to 36	36 to 43	42 to 48	48 to 54	54 to 60	60 to 66	66 to 70	72 to 78	78 to 84	84 to 90	D≥90
16	0	0	0	0	0	0	0	0	0	0	0	1	1
17	0	0	0	0	0	0	0	1	1	1	1	1	1
18	0	0	0	0	1	1	1	1	2	2	2	2	2
19	0	0	0	1	1	1	2	2	2	2	2	2	2
20	0	0	1	1	2	2	2	2	2	2	2	2	2
21	0	1	1	2	2	2	2	2	2	2	2	2	2
22	0	1	1	2	2	2	2	2	2	2	2	2	2
23	0	1	1	2	2	2	2	2	2	2	2	2	2
24	0	1	2	2	2	2	2	2	2	2	2	2	2
25	0	1	2	2	2	2	2	2	2	2	2	2	2
26	0	1	1	2	2	2	2	2	2	2	2	2	2
27	0	1	1	2	2	2	2	2	2	2	2	2	2
28	0	0	1	2	2	2	2	2	2	2	2	2	2
29	0	0	1	1	2	2	2	2	2	2	2	2	2
30	0	0	0	1	1	1	2	2	2	2	2	2	2
31	0	0	0	0	0	1	1	1	1	1	2	2	2
32	0	0	0	0	0	0	0	0	0	0	1	1	1

INFV = 0 correspond to low infection; INFV = 1 correspond to moderate infection; INFV = 2 correspond to high infection (see text).

Table 5.2. Sporulation values (SPOV) based on temperature and leaf wetness duration.

Temp	Duration of wet period (hr)											
	D≤24	24 to 30	30 to 36	36 to 43	42 to 48	48 to 54	54 to 60	60 to 66	66 to 70	72 to 78	78 to 84	84 to 90	D≥90
16	0	0	0	0	0	0	0	0	0	0	0	0	0
17	0	0	0	0	0	0	0	0	0	0	0	0	0
18	0	0	0	0	0	0	0	0	0	0	0	0	0
19	0	0	0	0	0	0	0	0	0	1	1	1	1
20	0	0	0	0	0	0	0	1	1	1	1	1	1
21	0	0	0	0	0	0	1	1	1	1	1	1	1
22	0	0	0	0	0	1	1	1	1	1	1	1	1
23	0	0	0	0	0	1	1	1	1	1	1	1	1
24	0	0	0	0	1	1	1	2	2	2	2	2	2
25	0	0	0	0	1	1	2	2	2	2	2	2	2
26	0	0	0	0	0	1	2	2	2	2	2	2	2
27	0	0	0	0	0	0	1	2	2	2	2	2	2
28	0	0	0	0	0	0	0	1	1	2	2	2	2
29	0	0	0	0	0	0	0	0	0	1	1	2	2
30	0	0	0	0	0	0	0	0	0	0	0	0	0
31	0	0	0	0	0	0	0	0	0	0	0	0	0
32	0	0	0	0	0	0	0	0	0	0	0	0	0

SPOV = 0 correspond to low sporulation; SPOV = 1 correspond to moderate sporulation; SPOV = 2 correspond to high sporulation.

Infection and sporulation rules. Because the infection and sporulation values were determined based on temperature and continuous leaf wetness only, it was necessary to formulate rules to allow prediction under the different weather conditions prevailing in carrots field of Quebec. These rules were derived from the synthesis of previous works (Carisse and Kushalappa, 1991, study II, study IV).

The infection rules were: a) relative humidity $\geq$ 90% is equivalent to leaf wetness in promoting infection; b) any dry periods $\leq$ 6 hr are treated like a leaf wetness period; c) two leaf wetness or %RH $\geq$90% periods separated by a dry period greater than 6 hr and less or equal to 12 hr are added together (including the dry period) if the first wet period is at least 12 hr, in this case no more than two wet periods can be added; d) the presence of a dry period greater than 12 hr ended an infection period. The duration of leaf wetness (or equivalent conditions) and mean temperature during this period are used to predict infection.

The sporulation rule was: relative humidity $\geq$ 95% is equivalent to leaf wetness in promoting sporulation.

Assumptions. By definition a forecasting model is a simplified prediction model and thus assumptions have to be made for elements that are not included in the forecasting model. Assumptions are: 1) inoculum from overwintering fungal structures is always available in sufficient amount to

initiate epidemic; 2) conditions for initial inoculum production are the same as those for secondary inoculum production; 3) Interrupted wetting does not reduce or enhance sporulation; 4) If the mean hourly temperature is $\geq$ 32 °C for 4 hr or more on at least one of the 4 preceding days there would be no infection or sporulation and the INFV or SPOV are equal to 0 for that day; 5) conditions are always favorable for spore dispersal.

Calculation of cumulative blight severity value. The input variables were hourly ambient temperature, % relative humidity, and presence of leaf wetness monitored in carrot fields. The calculation of cumulative blight severity values was accomplished following four distinct steps.

Step one consisted of determining the duration of leaf wetness (or equivalent conditions) and mean temperature during this period for the four preceding days. This was done considering the infection and sporulation rules and assumptions discussed above.

Step two consisted in finding the infection (INFV) and sporulation (SPOV) values from table 5.1 and 5.2, respectively.

The third step consisted in calculating the daily blight severity value (BSV) as:

$$BSV1 = INFV \qquad (5.6)$$

$$BSV2 = INFV + SPOV \quad (5.7)$$

The final step consisted of computing the cumulative blight severity value (CBSV1 and CBSV2) from the daily blight severity values (BSV1 or BSV2) obtained from the third step starting 15 days after sowing as:

$$CBSV1 = \sum_{i=1}^{i=n} BSV1 \quad (5.8)$$

$$CBSV2 = \sum_{i=1}^{i=n} BSV2 \quad (5.9)$$

where, i=1 15 days after sowing and n is the date of prediction.

Determination of an action threshold based on CBSV values. The action threshold at which the first fungicide should be applied was determined by comparing the cumulative blight severity value (CBSV1 and CBSV2) with disease incidence of 50 and 80% observed in 10 plots representing different sowing dates and years. Instrumentation and methods used to collect weather and disease data are described elsewhere (study IV).

Field plots. Ten sets of weather and disease progress data collected at the Agriculture Canada Experimental Farm of Saint-Clotilde, Quebec were analyzed. These plots of carrot cv. Dagger were established on organic soil and included: four sets of data for 1987 corresponding to carrots plots sown on May 12, May 25, June 8, and June 22; three sets of data for

1988 corresponding to carrot plots sown on June 1, June 16, July 1; three sets of data for 1990 corresponding to carrot plots sown on May 10, May 25, and June 9. Data for a total of ten epidemics were thus used for analysis.

Disease incidence and severity assessment. The plots were not inoculated with C. carotae and the disease was allowed to develop with just the inoculum surviving naturally. The ten carrot plots were monitored every week for Cercospora blight by visual inspection of carrot plants. The proportion of intermediate leaves with one or more lesions was recorded on ten plants per plot selected at random. The day at which 50 and 80% disease incidence were reached was recorded.

Cercospora blight severity was also assessed 7 to 11 times, at 4 to 7-day intervals, during the cropping season depending on the plots. For each assessment, the number of lesions per leaf on 30 plants was recorded. Disease severity on individual plants was expressed as infected area per plant (INFAREA in cm^2/leaf) calculated as:

$$\text{INFAREA} = \frac{\text{total \# of lesions/plant X 0.06}}{\text{total \# of leaves present}} \qquad (5.10)$$

Environmental monitoring. Each weather data set consisted in weather variables monitored during carrot growing seasons and

included hourly ambient temperature and relative humidity, and leaf wetness (study IV). Environmental data were monitored with different instrument depending on year, in 1987 data were collected with an electronic (Agriscribe) hygrothermographs while in 1988 and 1990 with a datalogger (model CR-10, Campbell Scientific Instruments).

RESULTS

The disease incidence of 50% was observed when CBSV1 values reached 14 to 16 (mean=14.9) except for the plot 1 and 3 in 1988 and 1990 where CBSV1 were 13 and 19, respectively (Table 5.3). For all plots, the 80% disease incidence was observed when a CBSV1 ranged from 18 to 20 (Table 5.3). The CBSV2 reached values ranging from 11 to 48 on the day at which 50% disease incidence was observed and values ranging from 16 to 58 on the day at which 80% disease incidence was observed (Table 5.3).

Cumulative blight severity values were more reliable in predicting the critical levels of 50 and 80% incidence when calculated based on infection only (BSV1=INFV) than based on both infection and sporulation (BSV2=INFV+SPOV). The later model resulted in unreliable predictions (Table 5.3). Using this model the CBSV2 values did not seem to be related to disease incidence since similar CBSV2 were reached for both critical levels. Furthermore, this model resulted in very high CBSV2 (48-58) in 1988, while low disease progress was

observed in the field.

However, for almost all plots, cumulative blight severity values calculated based on infection only (BSV1) was very stable in predicting both disease incidence of 50 and 80%. For this reason only the model BSV1=INFV was compared with disease progress observed in the field. Accumulation of CBSV1 and progress of infected area/leaf over time for all the epidemics studied is presented in Figures 5.1-5.3. In general, the progress of CBSV1 followed the pattern of disease progress curve. For almost all epidemics, CBSV=14-16 was reached when the infected area per leaf ranged from about 0.5 to 1.0 cm^2/leaf and 2 to 10 days before the beginning of the exponential phase of disease increase (Fig. 5.1-5.3).

The 1987 and 1990 seasons were characterized by warm and humid weather favorable to Cercospora blight development. In 1988, the weather was hot and generally dry, resulting in low blight development. Because of that, the accumulation of CBSV1 was more rapid in 1987 and 1990 than in 1988 which corresponded to the disease progress observed in the field (Fig. 5.1-5.3).

The structure of the proposed forecasting system is diagrammed in table 5.4.

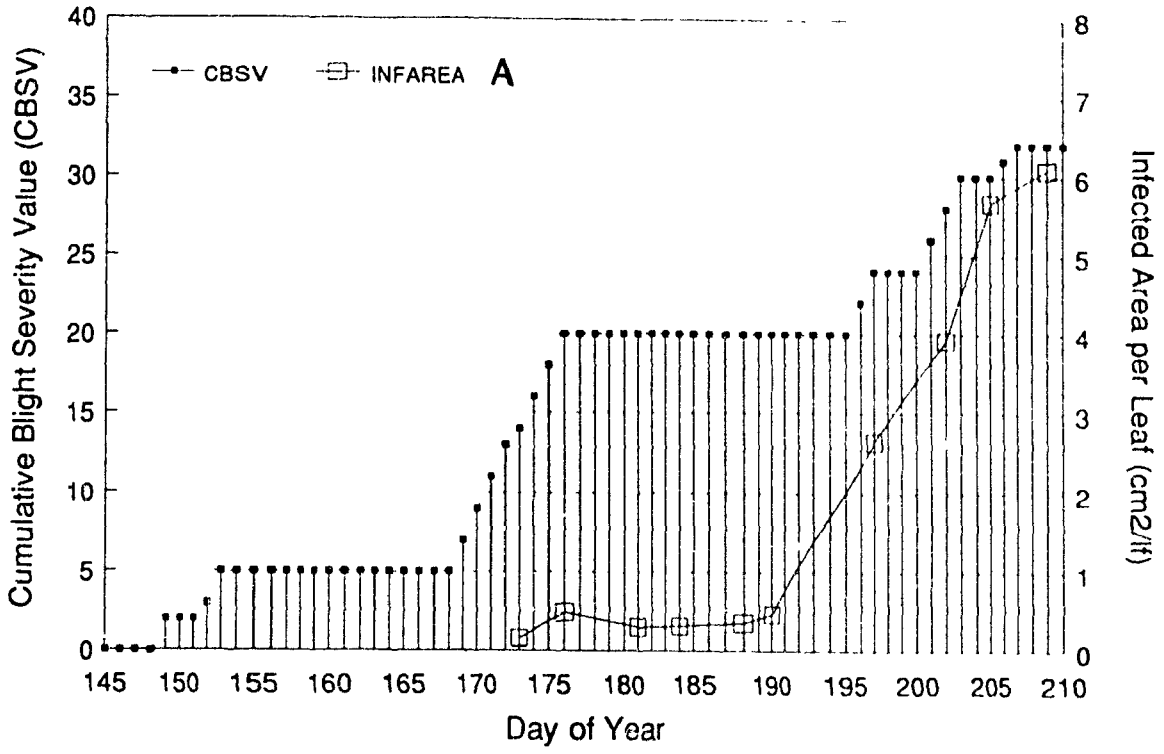
Table 5.3. Day of year, CBSV1, and CBSV2 on which 50 and 80% disease incidence were observed at Sainte-Clotilde during 1987, 1988 and 1990.

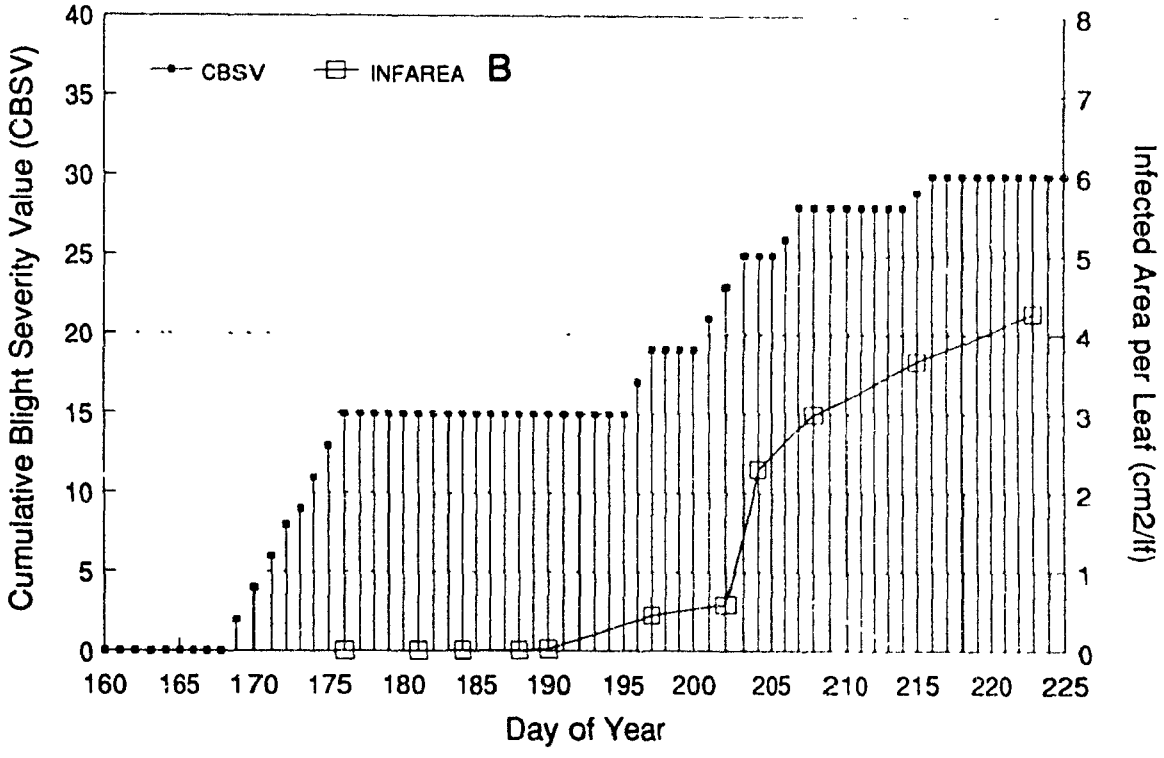
	Disease incidence of 50%			Disease incidence of 80%		
Plot	Day	CBSV1[y]	CBSV2[z]	Day	CBSV1	CBSV2
1987						
Plot 1	174	16	21	177	20	27
Plot 2	177	15	20	197	19	31
Plot 3	202	14	24	207	19	29
Plot 4	208	14	17	-	-	-
1988						
Plot 1	203	13	48	208	20	58
Plot 2	208	17	43	210	21	49
Plot 3	209	15	25	-	-	-
1990						
Plot 1	174	14	13	190	18	17
Plot 2	191	14	11	197	18	16
Plot 3	198	19	15	-	-	-

[y] CBSV1 is the cumulative blight severity values calculated using equation 5.8.

[z] CBSV2 is the cumulative blight severity values calculated using equation 5.9.

Figure 5.1. Development of Cercospora blight of carrots (infected area/leaf in cm^2/leaf) during 1987 and cumulative blight severity values (CBSV1). CBSV1 was calculated using equation 5.8. A, B, C, and D, represent data for carrot plots sown on May 12, May 25, June 8, and June 22, respectively.


CBSV
INFAREA
A
Cumulative Blight Severity Value (CBSV)
Infected Area per Leaf (cm2/lf)
Day of Year
0
5
10
15
20
25
30
35
40
0
1
2
3
4
5
6
7
8
145
150
155
160
165
170
175
180
185
190
195
200
205
210


CBSV
INFAREA
B
Cumulative Blight Severity Value (CBSV)
Infected Area per Leaf (cm2/lf)
Day of Year
0
5
10
15
20
25
30
35
40
0
1
2
3
4
5
6
7
8
160
165
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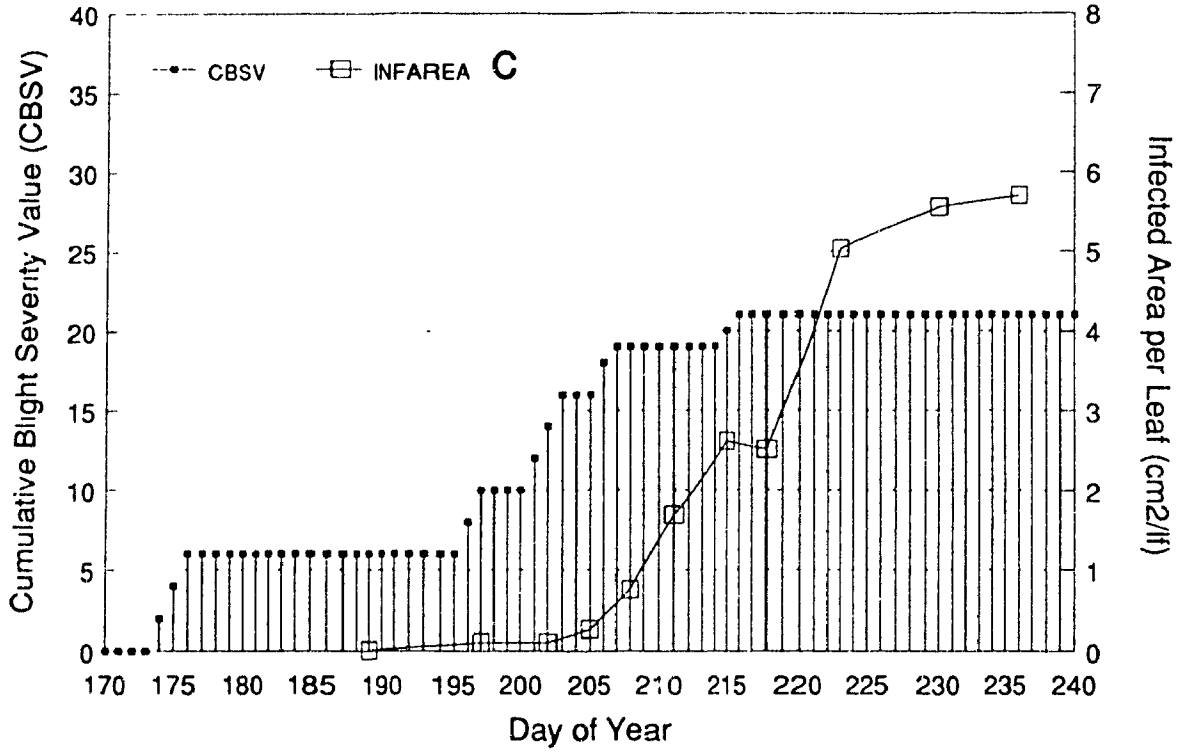
175
180
185
190
195
200
205
210
215
220
225


CBSV
INFAREA
C
Cumulative Blight Severity Value (CBSV)
Infected Area per Leaf (cm2/lf)
Day of Year
0
5
10
15
20
25
30
35
40
0
1
2
3
4
5
6
7
8
170
175
180
185
190
195
200
205
210
215
220
225
230
235
240

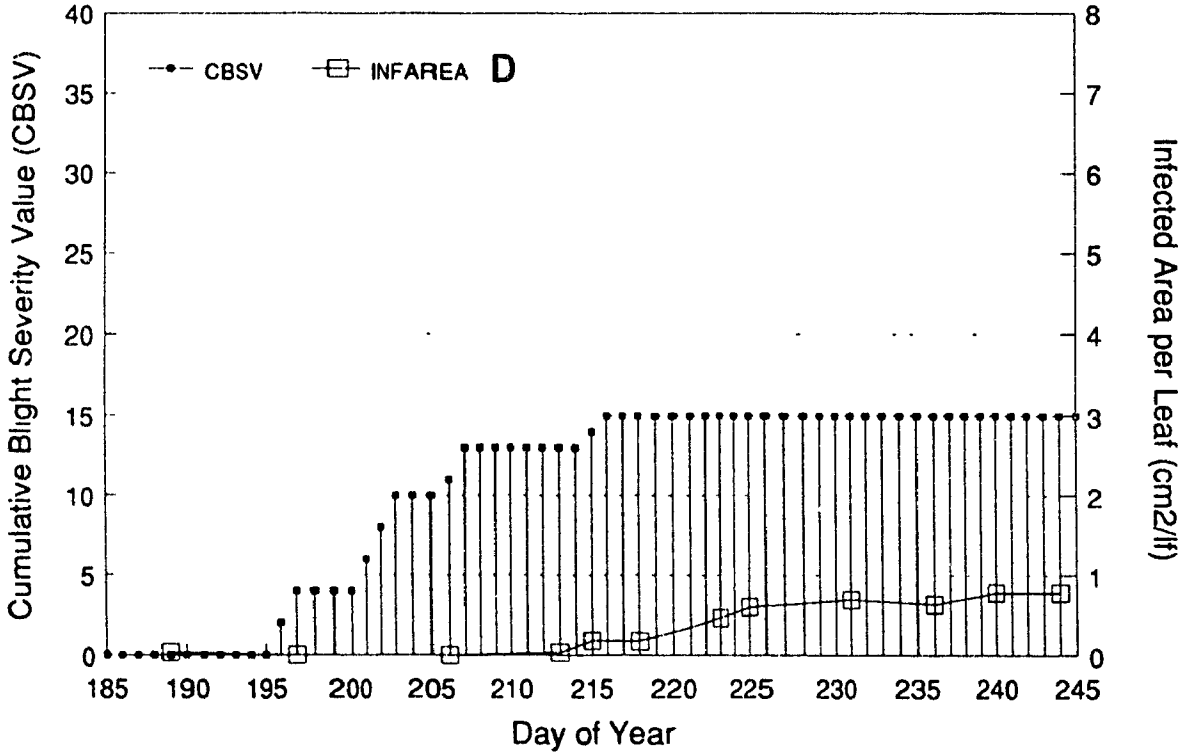
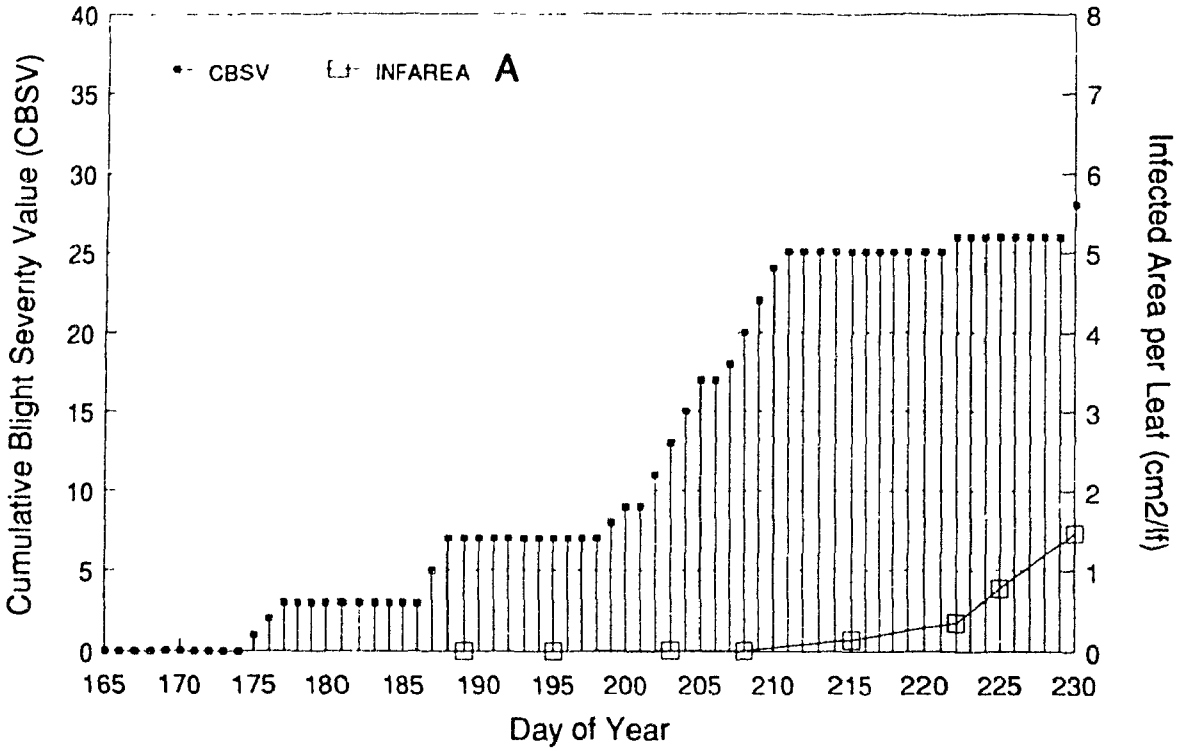
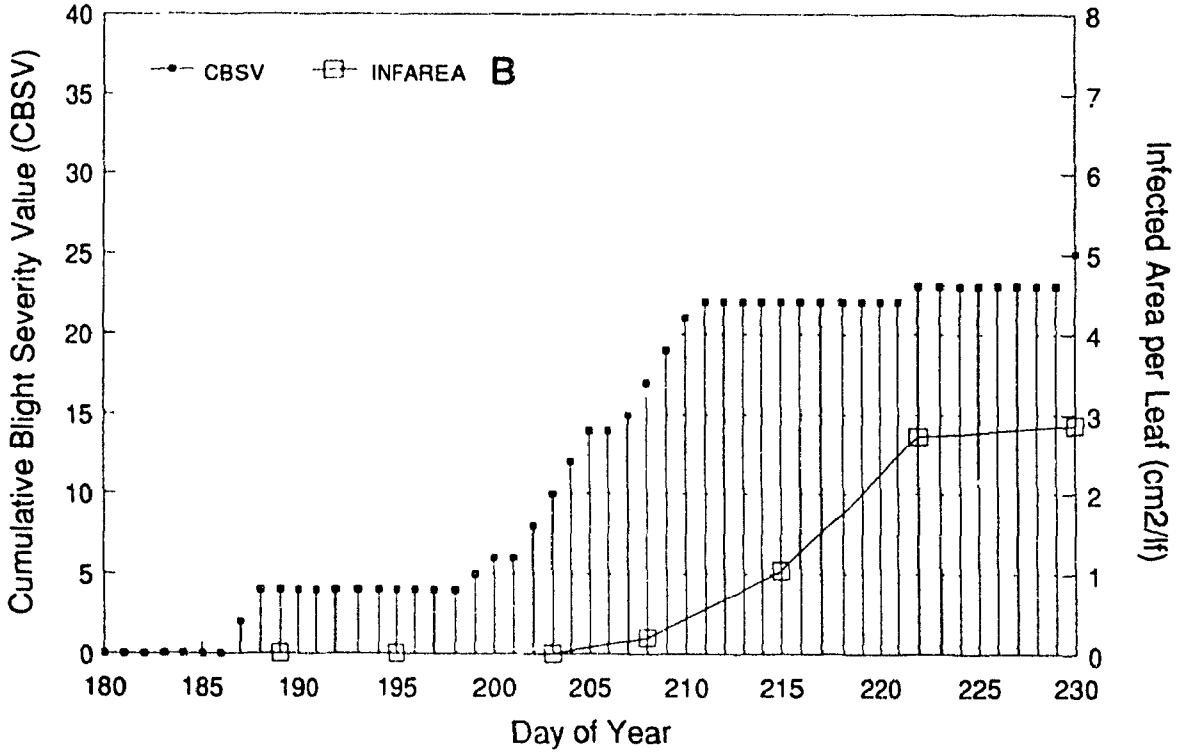

CBSV
INFAREA
D
Cumulative Blight Severity Value (CBSV)
Infected Area per Leaf (cm2/lf)
Day of Year
0
5
10
15
20
25
30
35
40
0
1
2
3
4
5
6
7
8
185
190
195
200
205
210
215
220
225
230
235
240
245

Figure 5.2. Development of Cercospora blight of carrots (infected area/leaf in cm^2/leaf) during 1988 and cumulative blight severity values (CBSV1). CBSV1 was calculated using equation 5.8. A, B, and C, represent data for carrot plots sown on June 1, June 16, July 1, respectively.


CBSV
INFAREA
A
Cumulative Blight Severity Value (CBSV)
Infected Area per Leaf (cm2/lf)
Day of Year
0
5
10
15
20
25
30
35
40
0
1
2
3
4
5
6
7
8
165
170
175
180
185
190
195
200
205
210
215
220
225
230


CBSV
INFAREA
B
Cumulative Blight Severity Value (CBSV)
Infected Area per Leaf (cm2/lf)
Day of Year
0
5
10
15
20
25
30
35
40
0
1
2
3
4
5
6
7
8
180
185
190
195
200
205
210
215
220
225
230

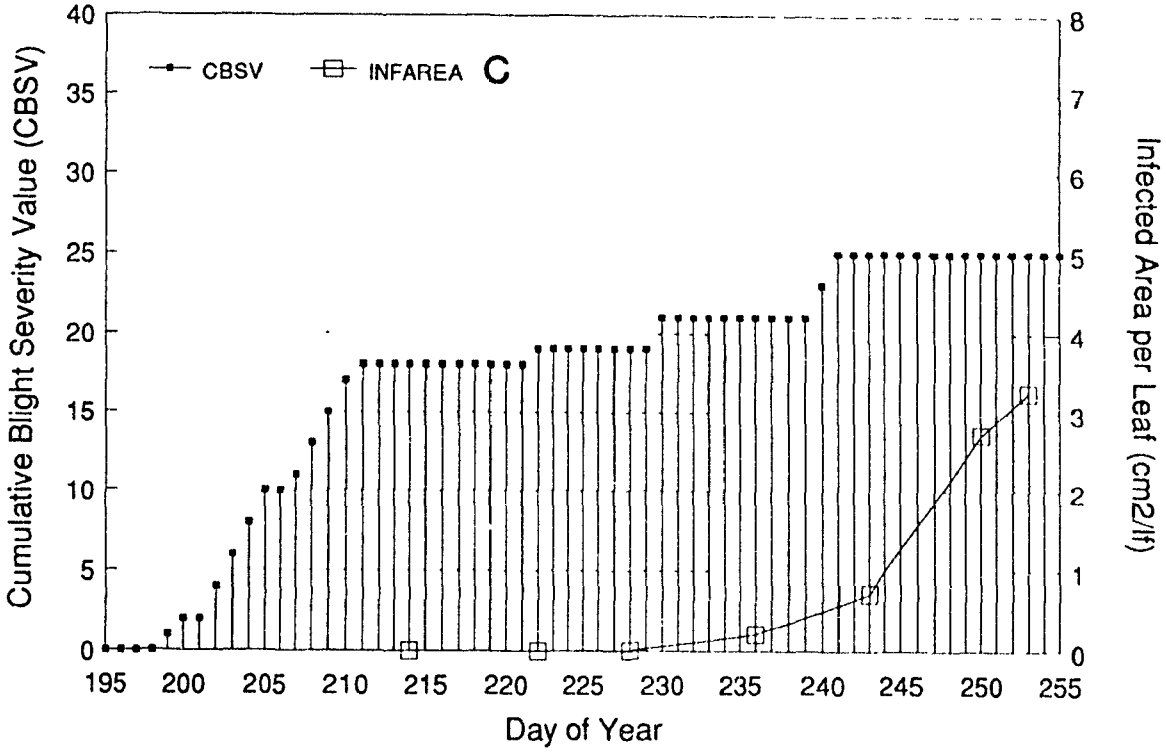
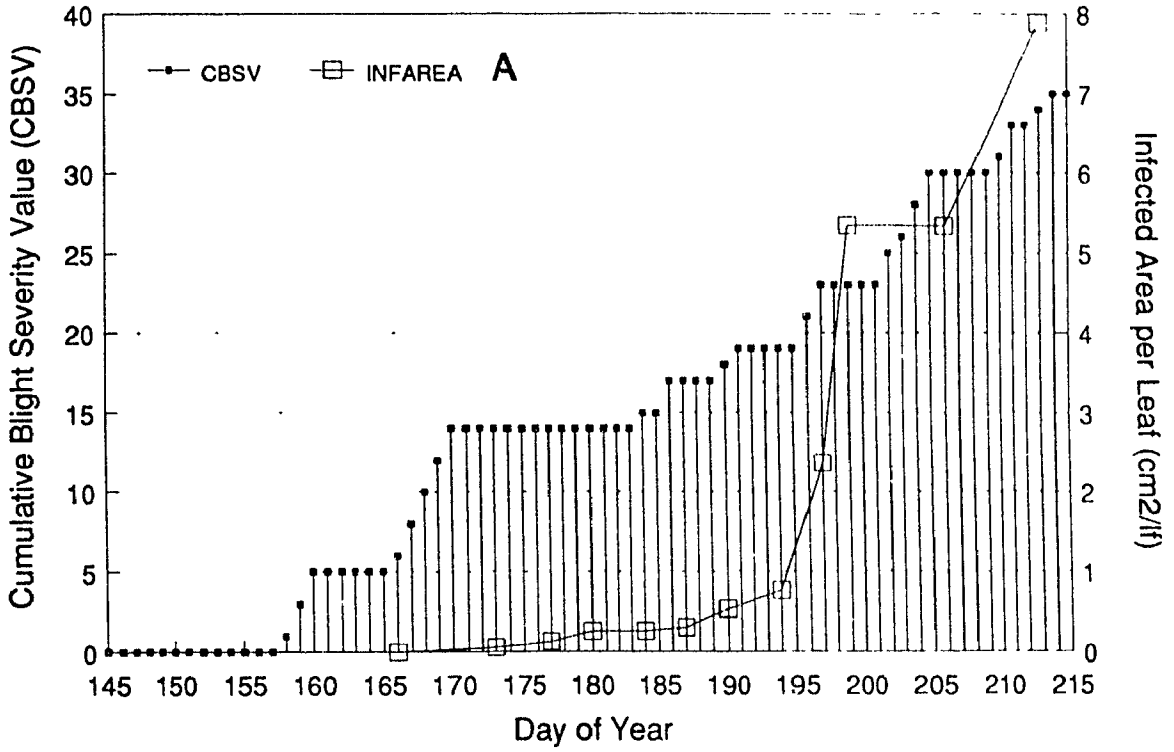

CBSV
INFAREA
C
Cumulative Blight Severity Value (CBSV)
Infected Area per Leaf (cm2/lf)
Day of Year
0
5
10
15
20
25
30
35
40
0
1
2
3
4
5
6
7
8
195
200
205
210
215
220
225
230
235
240
245
250
255

Figure 5.3. Development of Cercospora blight of carrots (infected area/leaf in cm^2.leaf) during 1990 and cumulative blight severity values (CBSV1). CBSV1 was calculated using equation 5.8. A, B, and C, represent data for carrot plots sown on May 10, May 25, and June 9, respectively.


CBSV
INFAREA
A
Cumulative Blight Severity Value (CBSV)
Infected Area per Leaf (cm2/lf)
Day of Year
0
5
10
15
20
25
30
35
40
0
1
2
3
4
5
6
7
8
145
150
155
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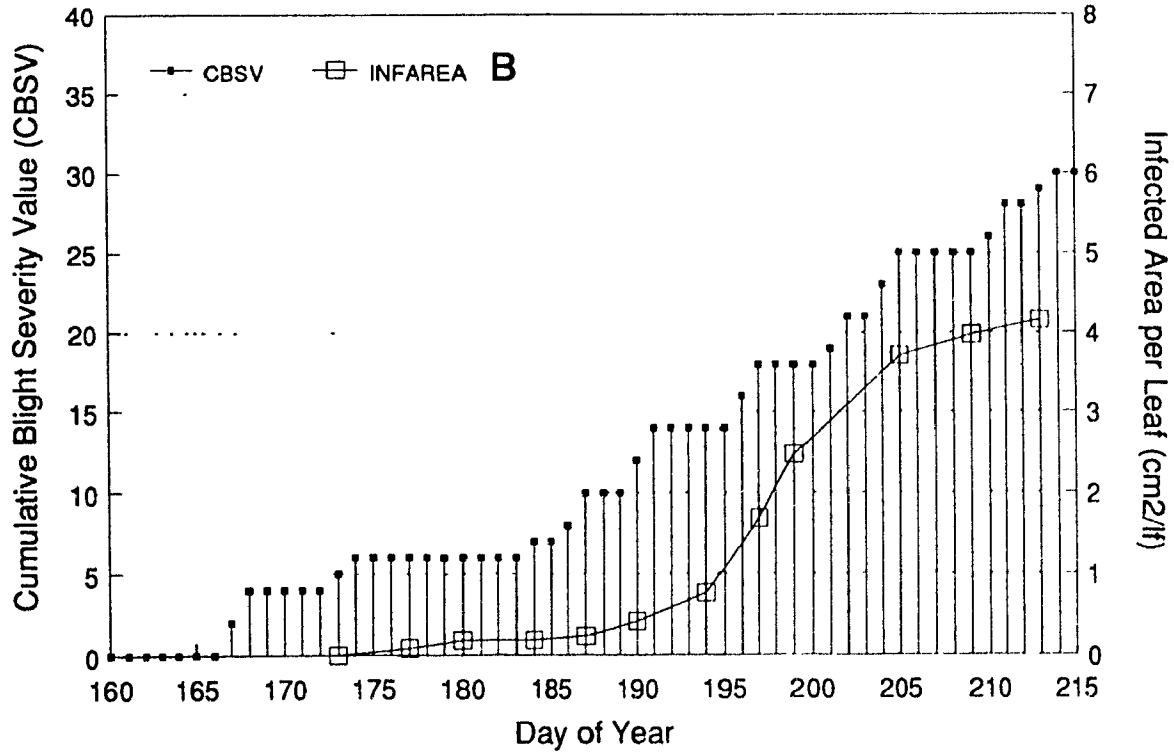
170
175
180
185
190
195
200
205
210
215


CBSV
INFAREA
B
Cumulative Blight Severity Value (CBSV)
Infected Area per Leaf (cm2/lf)
Day of Year
0
5
10
15
20
25
30
35
40
0
1
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160
165
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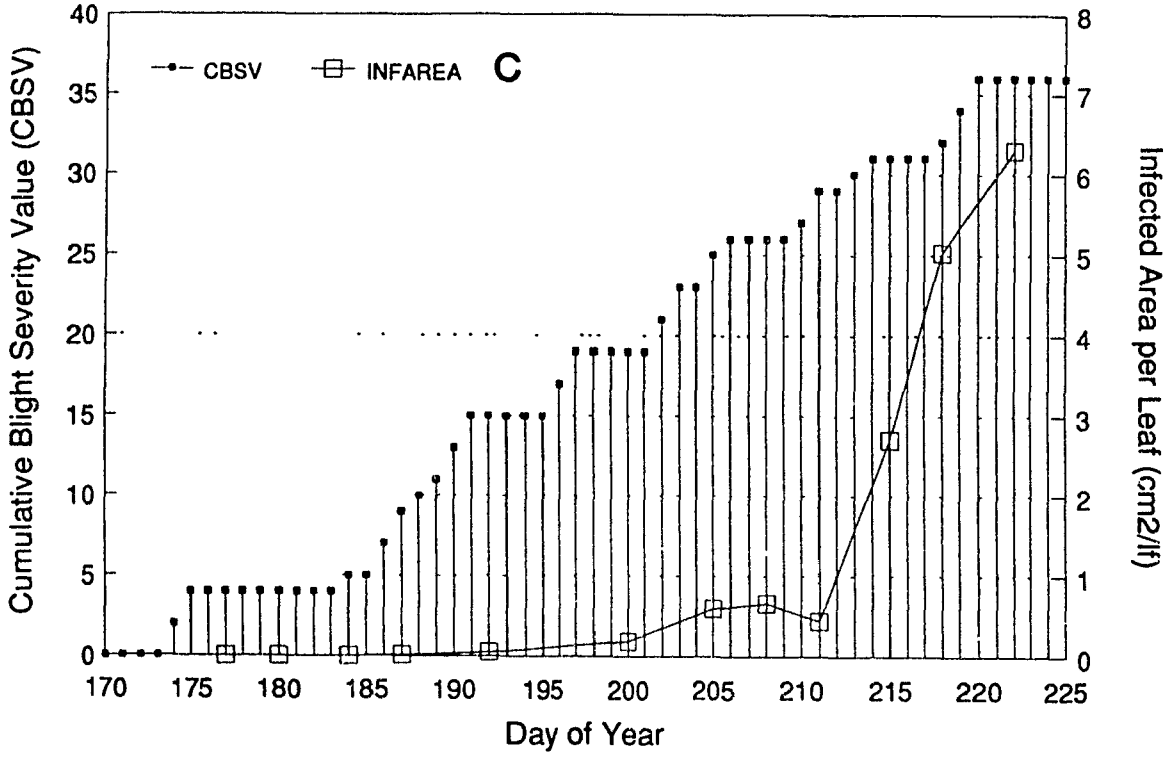
CBSV
INFAREA
C
Cumulative Blight Severity Value (CBSV)
Infected Area per Leaf (cm2/lf)
Day of Year
0
5
10
15
20
25
30
35
40
0
1
2
3
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175
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225

Table 5.4. Proposed forecasting system for the timing of the initial fungicide spray to manage cercospora blight of carrot.

STEP I

Calculate the duration of leaf wetness (or equivalent conditions) and temperature during this period from field data for the four preceding days from the date of prediction.

STEP II

Estimate INFV from the table 5.1.

STEP III

Compute cumulative blight severity value (CBSV1): add the daily INFV since 15 days after sowing until the date of prediction.

STEP IV

A. Warning threshold:

When CBSV1 reaches 14 to 16 apply an initial spray only if rain or periods of high humidity are probable.

B. Spray threshold:

When CBSV1 reaches 18 to 20 start spraying because the risk of rapid increase in disease is high.

DISCUSSION

Timing of the initial spray for the control of Cercospora blight of carrot is essential to minimize the number of fungicide applications required to manage this disease. The weather-based forecasting system was developed to provide a method of dealing with spray schedule initiation for carrots grown on organic soil in Quebec. Systems based on infection and on both infection and sporulation were tested for their reliability in predicting disease incidence thresholds of 50 and 80%. Only the system based on infection alone was stable in predicting these thresholds. This is probably because the infection is more directly related to disease severity in the field than the sporulation. The relationship between the amount of spores produced and disease severity is not linear, meaning that an increase in sporulation will not necessarily result in a proportional increase in disease severity.

The proposed system forecasts the likelihood of infection and thus provides a warning for the need for a fungicide application. This approach was investigated because it was used for several other pathosystems (Couture and Sutton, 1978, Dainello and Jones, 1984, Danneberger et al., 1984, Eisensmith and Jones, 1981, Machardy, 1979). Timing of fungicide application based on the accumulation of critical number of disease severity units has been employed with success to manage other polycyclic diseases (Jensen and Boyle, 1966,

Krause et al., 1975, Madden et al., 1978).

The forecasting system proposed here has at least three advantages 1) it is relatively simple and would be easy to implement, the daily INFV can be obtained from a table without any calculations; 2) it does not require disease monitoring in the field ; 3) it requires monitoring of only a few weather parameters.

The CBSV1 of 14 to 16 was reached before the onset of epidemics was observed which mean that this threshold may be conservative and higher CBSV limit could be considered. The proposed limits (CBSV1=14-16 and CBSV1=18-20) of cumulative blight severity values (CBSV) need to be tested with independent field data to determine if they result in consistent levels of disease control. Also it must be evaluated for different cultivars and for commercial field conditions to determine if the spray schedule produced by the forecasting system provide efficient and economical control of Cercospora blight (Jesperson and Sutton, 1987, Pennypacker,1983, Phipps and Powell, 1984). These tests are necessary before offering the system to the carrot growers in the organic soils of Quebec. Implementation of the forecasting system would probably be best accomplished using a preprogrammed microcomputer that monitored the required weather data and issued the appropriate forecasts.

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GENERAL CONCLUSION

Cercospora blight of carrot, induced by Cercospora carotae, is an endemic foliar disease of carrots grown in organic soils of Quebec. During most years, this is the only disease requiring field applications of fungicides. Other carrots diseases such as Alternaria leaf blight (Alternaria dauci) and Cottony soft rot (Sclerotinia sclerotiorum) are sporadic or controlled during storage. Management of Cercospora blight represents a significant cost of production. However, relationships between blight severity and yield loss are not well established. This is part because until recently Cercospora blight of carrot was considered to be of minor importance. However, a survey conducted recently demonstrated that it is the most important foliar disease of carrots in the southwestern region of Quebec.

The recommendations to manage cercospora blight of carrot include crop rotation on a three year schedule and application of protectant fungicides. The crop rotation is recommended to reduced the initial inoculum and the fungicide application to reduce the apparent infection rate. Fungicides are the most effective measures of disease control when conditions are favorable to disease development, provided that initial inoculum is available. With the availability of effective agricultural chemicals

for controlling foliar disease, such as Cercospora blight, growers can successfully protect their crop, but at a cost for the growers and the environment.

When information on the epidemiology of a given disease is not available the only possible control strategy is to apply fungicides on a calendar basis starting early in the season so that risks for the growers are reduced to the minimum. This approach was considered workable as long as the problems and environmental risks associated with the use of pesticides were not known. These days, growers want to reduce their production costs and consumers wish to have vegetables free of pesticides. This is a great challenge for plant pathologists who now have to develop disease management programs that are as efficient as the traditional ones but that reduce the use of pesticides to the minimum. To achieve that it is essential to understand how the disease progress and which factors influence this progression.

The first part of this research was designed to gain information on the effect of the environment on the major processes of the pathogen life cycle infection, sporulation and other aspect such as incubation period).

The study on influence of environmental factors on infection revealed that the interrupted leaf wetness significantly reduced infection as compare to continuous

leaf wetness but also that Cercospora carotae conidia can survive long drying (up to 24 hr) at optimal temperatures. The results suggested that it is presumably the ability of conidia of Cercospora carotae to survive drying rather than rapid germination and penetration that is responsible of effective infection under interrupted wet period. The same experiment revealed that high humidity (84%<RH<100%) reduced infection as compared to leaf wetness, but was sufficient to allow infection. Decrease in percent relative humidity resulted in rapid reduction in infection, even though the reduction was less rapid when the plants were exposed to a short initial wetness period (6 hr). This information is essential to interpret the weather pattern prevailing in carrot field, where carrot leaves are often wet at night and dry during the day and wet periods generally preceded and followed by periods of high relative humidity. From this study several mathematical models were developed and infection criteria were established. It was concluded that two wet periods separated by a dry period of $\leq$ 12 hr should be considered as one infection period (including the dry period) and wet periods can be extended when relative humidity is 90% or more.

The study on the influence of temperature and moisture on sporulation of Cercospora carotae demonstrated that conditions favorable for sporulation were quite similar to

those for infection with the same optimal temperature range of 20 to 28 °C but longer time was required to induce sporulation (>48 hr) than for infection (>24hr) at these temperatures. Leaf wetness was not necessary to trigger sporulation, even though more sporulation was observed under leaf wetness than high relative humidity. From this study the sporulation requirements were well established, however, the results should not be used to predict the exact amount of spores available in the field but rather the potential of the environment for sporulation.

The length of incubation period in the field was determined and the pattern of lesion appearance established. The mean incubation period of Cercospora carotae ranged from 8 to 12 days depending on the weather conditions. The incubation period of Cercospora carotae was influenced by both the daily temperature and the duration of high relative humidity or leaf wetness. Mean daily temperature of $\geq$ 21 °C and prolonged high RH period ($\geq$ 12 hr/day) resulted in a short incubation period (mean IP=8 days). The pattern of lesion appearance was similar for all inoculations regardless of the weather conditions, even though the time scale was different. The effect of weather on incubation period was studied and models were developed to predict the incubation period as a function of temperature and hours of high humidity. These models can be used for simulation or

forecasting where it is possible to integrate a variable incubation period. For the situation where only a fixed incubation period is to be used a general model predicting the appearance of lesions as a function of time only was developed.

These studies on monocyclic process are essential to understand the influence of the environment on each phase of the pathogen life cycle. However, in reality the different processes of the pathogen life cycle operate together to generate an epidemic. The contribution of each component and their interactions on epidemic development must be studied to understand and predict the disease progress.

Simulation models are an interesting alternative to study the interaction between the environment and each processes of the pathogen life cycle. The simulation model presented here contains series of simple equations that describe mathematically several biological processes such as infection, sporulation, and incubation. The simulation model was evaluated for three years with data representing different sowing dates. The percent disease severity (infected area/leaf) was adequately predicted for most of the epidemics studied. The adequacy of the model in simulating Cercospora blight progression means that models, criteria for monocyclic process, and the relationships among

them considered for the model development were quite realistic. This model was developed mainly as a research tool to develop more ecological management programs for Cercospora blight of carrots.

A simple forecasting system was developed using information available on Cercospora blight. Timing of fungicide applications based on visual estimation of disease incidence is currently used by carrot growers and allow a reduction in the number of fungicide applications required. The forecasting system predicts critical disease levels of 50 and 80% disease incidence from the accumulation of critical number of disease severity units and can be used to time the initial fungicide application only. The forecasting system has to be evaluated for commercial conditions and different cultivars to determine if the spray schedule produced by the forecasting system provide efficient and economical control of Cercospora blight.

Research on Cercospora blight epidemiology is not completed and more work needs to be done to understand better Cercospora blight development. Additional information on sporulation including the effect of lesion age, preconditioning of the lesions, interrupted leaf wetness on spore production and field data on spore viability and dispersal is needed to adequately understand

the sporulation process of Cercospora carotae. Other aspects of this pathosystem also need to be investigated including carrot growth, influence of leaf age on infection, effect of Cercospora blight on yield, and the effectiveness of fungicide applications on the reduction of blighted tissues in the field so that the most appropriate economic threshold could be established.

This research represents a significant contribution to the advancement toward a better understanding of Cercospora blight of carrot. The simulation model and the forecasting system may aid to make Cercospora blight management both more efficient and economical.

APPENDIX

Table A.1 Weather data during summer of 1987 at Sainte-Clotilde, Quebec.

Mth	Day	Day of Year	Duration of LW	HRH	DT	TMAX	TMIN
5	14	134	0	0	16.13	24.00	4.00
5	15	135	9	7	9.79	18.00	0.00
5	16	136	0	3	9.08	17.00	-2.00
5	17	137	4	0	13.83	16.00	12.00
5	18	138	2	5	9.88	15.00	0.00
5	19	139	0	4	9.38	19.00	-3.00
5	20	140	0	3	11.79	20.00	1.00
5	21	141	5	0	14.29	21.00	3.00
5	22	142	3	3	18.08	23.00	10.00
5	23	143	16	16	9.33	11.00	8.00
5	24	144	10	8	10.54	14.00	8.00
5	25	145	0	11	12.88	18.00	6.00
5	26	146	0	5	15.13	23.00	4.00
5	27	147	10	9	15.96	20.00	14.00
5	28	148	14	13	19.79	29.00	14.00
5	29	149	14	8	23.25	29.00	19.00
5	30	150	6	0	25.96	31.00	22.00
5	31	151	11	7	22.75	27.00	19.00
6	01	152	7	10	22.08	28.00	17.00
6	02	153	11	4	21.67	28.00	17.00
6	03	154	10	0	19.79	24.00	18.00
6	04	155	14	6	18.38	22.00	14.00
6	05	156	12	3	15.63	22.00	11.00
6	06	157	8	2	12.67	18.00	7.00
6	07	158	0	1	14.54	21.00	3.00
6	08	159	18	12	16.21	22.00	13.00
6	09	160	19	18	14.08	17.00	12.00
6	10	161	9	2	14.75	20.00	8.00
6	11	162	0	0	17.63	23.00	13.00
6	12	163	20	23	14.79	18.00	11.00
6	13	164	10	11	17.04	24.00	12.00
6	14	165	0	3	21.54	27.00	13.00
6	15	166	0	5	22.29	27.00	16.00
6	16	167	11	6	16.08	24.00	6.00
6	17	168	8	4	16.63	23.00	10.00
6	18	169	18	10	20.42	28.00	11.00
6	19	170	17	12	21.71	30.00	11.00
6	20	171	13	9	17.79	25.00	9.00
6	21	172	20	13	20.38	31.00	7.00
6	22	173	12	10	20.54	25.00	13.00
6	23	174	18	9	24.17	32.00	17.00

Table A.1 Continued.

Mth	Day	Day of Year	Duration of LW	HRH	DT	TMAX	TMIN
6	24	175	13	7	25.58	32.00	15.00
6	25	176	12	8	22.54	28.00	18.00
6	26	177	2	0	20.50	27.00	17.00
6	27	178	7	11	18.79	23.00	16.00
6	28	179	11	9	16.75	22.00	13.00
6	29	180	12	4	19.33	24.00	14.00
6	30	181	10	1	21.63	25.00	18.00
7	01	182	0	4	18.25	24.00	10.00
7	02	183	0	5	18.29	25.00	6.00
7	03	184	0	21	17.83	21.00	16.00
7	04	185	10	7	20.13	24.00	17.00
7	05	186	0	9	21.33	30.00	13.00
7	06	187	0	5	22.88	31.00	11.00
7	07	188	0	1	21.71	28.00	18.00
7	08	189	0	12	22.54	30.00	18.00
7	09	190	0	9	25.46	32.00	17.00
7	10	191	0	8	26.88	35.00	18.00
7	11	192	0	9	26.54	33.00	18.00
7	12	193	0	7	28.33	34.00	22.00
7	13	194	10	12	25.83	34.00	20.00
7	14	195	18	16	22.54	31.00	15.00
7	15	196	8	5	15.88	22.00	9.00
7	16	197	0	9	15.96	24.00	5.00
7	17	198	0	3	20.04	27.00	7.00
7	18	199	8	7	22.96	30.00	16.00
7	19	200	13	12	19.33	26.00	15.00
7	20	201	4	14	19.33	24.00	16.00
7	21	202	12	13	20.17	28.00	16.00
7	22	203	0	10	23.04	30.00	16.00
7	23	204	0	6	25.38	32.00	15.00
7	24	205	10	10	24.71	33.00	19.00
7	25	206	11	12	23.79	29.00	19.00
7	26	207	10	8	20.50	26.00	14.00
7	27	208	0	0	18.17	23.00	11.00
7	28	209	0	6	12.79	18.00	6.00
7	29	210	0	5	16.17	25.00	6.00
7	30	211	8	5	18.38	25.00	12.00
7	31	212	8	8	15.58	24.00	8.00
8	01	213	8	8	15.71	24.00	5.00
8	02	214	13	11	18.33	27.00	6.00
8	03	215	12	14	21.17	25.00	15.00
8	04	216	7	9	21.33	29.00	13.00
8	05	217	1	4	15.71	20.00	10.00
8	06	218	6	10	16.75	26.00	5.00

Table A.1 Continued.

Mth	Day	Day of Year	Duration of LW	HRH	DT	TMAX	TMIN
8	07	219	7	7	20.50	27.00	10.00
8	08	220	13	8	20.00	25.00	11.00
8	09	221	10	9	17.63	25.00	7.00
8	10	222	13	14	17.75	24.00	12.00
8	11	223	0	4	17.33	24.00	9.00
8	12	224	6	10	16.04	26.00	5.00
8	13	225	3	8	19.46	29.00	7.00
8	14	226	7	10	19.75	27.00	13.00
8	15	227	9	11	22.50	28.00	19.00
8	16	228	0	9	25.50	34.00	15.00
8	17	229	0	0	28.88	35.00	24.00
8	18	230	0	4	22.50	28.00	13.00
8	19	231	10	10	18.92	27.00	12.00
8	20	232	13	7	17.42	22.00	13.00
8	21	233	10	9	16.88	25.00	6.00
8	22	234	9	1	18.83	26.00	13.00
8	23	235	4	1	13.75	19.00	6.00
8	24	236	0	5	10.50	17.00	3.00
8	25	237	0	4	14.25	21.00	4.00
8	26	238	0	6	10.83	19.00	1.00
8	27	239	0	7	12.58	22.00	1.00
8	28	240	0	8	15.83	23.00	10.00
8	29	241	18	17	14.21	21.00	7.00
8	30	242	14	22	15.63	25.00	5.00
8	31	243	6	24	18.00	28.00	10.00
9	01	244	10	16	14.58	16.00	12.00
9	02	245	9	12	12.75	19.00	5.00
9	03	246	12	12	11.29	19.00	3.00
9	04	247	11	13	12.92	23.00	1.00
9	05	248	7	12	17.33	29.00	4.00
9	06	249	0	11	18.46	28.00	11.00
9	07	250	11	14	19.38	27.00	12.00
9	08	251	16	17	20.13	25.00	14.00

Table B.1 Weather data during summer of 1988 at Sainte-Clotilde, Quebec.

Mth	Day	Day of Year	Duration of LW	HRH	DT	TMAX	TMIN
6	09	160	0	0	19.12	23.14	10.04
6	10	161	0	0	21.08	24.31	10.97
6	11	162	0	0	19.27	24.89	11.89
6	12	163	0	0	18.47	25.20	11.53
6	13	164	0	0	23.68	28.88	17.80
6	14	165	0	0	25.43	31.61	19.72
6	15	166	0	0	26.49	32.98	16.20
6	16	167	0	0	28.24	34.45	20.38
6	17	168	0	0	22.14	25.93	9.70
6	18	169	0	7	18.71	27.97	7.41
6	19	170	0	3	22.95	31.07	9.38
6	20	171	0	0	26.42	31.54	18.16
6	21	172	9	0	27.19	32.88	18.72
6	22	173	11	0	21.98	26.58	14.08
6	23	174	18	9	20.19	26.32	11.63
6	24	175	24	6	14.72	18.83	8.70
6	25	176	18	0	14.36	20.01	6.98
6	26	177	23	14	15.63	22.25	12.45
6	27	178	24	7	17.49	22.24	15.37
6	28	179	8	10	17.34	22.66	13.27
6	29	180	24	4	16.21	24.00	12.00
6	30	181	24	0	11.50	15.00	10.00
7	01	182	24	24	11.67	14.00	10.00
7	02	183	24	12	15.83	22.00	10.00
7	03	184	5	1	20.00	26.00	14.00
7	04	185	0	8	20.33	30.00	9.00
7	05	186	15	5	21.20	28.98	12.35
7	06	187	24	9	21.58	30.75	11.70
7	07	188	11	8	22.64	28.98	14.28
7	08	189	7	7	24.83	31.45	15.54
7	09	190	2	0	28.75	34.39	22.27
7	10	191	9	0	29.26	35.05	23.53
7	11	192	24	5	27.17	33.51	20.16
7	12	193	24	3	24.83	27.96	20.39
7	13	194	24	8	22.32	27.02	16.06
7	14	195	9	6	20.28	27.91	12.72
7	15	196	10	5	21.12	27.65	15.11
7	16	197	11	3	17.82	23.00	8.58
7	17	198	8	0	22.09	29.25	16.32
7	18	199	24	24	19.38	22.31	17.23
7	19	200	7	9	24.23	30.61	18.72
7	20	201	18	14	21.09	24.46	18.34

Table B.1 Continued.

Mth	Day	Day of Year	Duration of LW	HRH	DT	TMAX	TMIN
7	21	202	13	11	20.20	25.09	15.56
7	22	203	17	19	19.09	21.23	16.03
7	23	204	12	12	20.65	28.58	13.62
7	24	205	12	10	21.40	29.02	11.33
7	25	206	10	7	23.55	29.52	18.97
7	26	207	18	11	21.58	27.22	15.70
7	27	208	24	19	19.47	27.29	14.80
7	28	209	20	13	22.20	27.97	17.33
7	29	210	15	10	22.28	29.03	13.77
7	30	211	13	9	23.85	32.27	12.93
7	31	212	8	2	27.07	34.08	20.19
8	01	213	14	7	24.09	29.55	15.01
8	02	214	11	7	22.87	31.27	11.81
8	03	215	10	2	25.30	33.20	17.14
8	04	216	16	14	25.15	35.74	18.71
8	05	217	11	7	27.73	33.77	21.92
8	06	218	14	10	25.47	32.26	20.81
8	07	219	22	10	24.70	28.67	20.31
8	08	220	14	7	22.78	27.92	18.82
8	09	221	14	9	23.86	29.13	19.31
8	10	222	13	4	24.72	29.35	19.25
8	11	223	8	0	25.71	30.30	19.62
8	12	224	11	9	22.38	30.00	13.83
8	13	225	11	4	23.86	31.03	15.80
8	14	226	17	11	24.39	33.08	17.56
8	15	227	21	21	21.91	26.72	18.38
8	16	228	17	16	22.63	25.92	18.63
8	17	229	21	14	18.74	23.02	13.66
8	18	230	18	16	19.11	24.36	13.64
8	19	231	14	1	16.22	20.86	9.70
8	20	232	11	8	15.95	23.21	7.57
8	21	233	13	6	17.79	23.16	12.89
8	22	234	19	9	15.04	18.07	11.14
8	23	235	23	5	12.74	19.58	7.28
8	24	236	18	10	8.59	15.20	2.82
8	25	237	24	20	9.50	16.05	3.03
8	26	238	24	7	13.69	21.30	6.98
8	27	239	19	9	18.84	24.82	14.90
8	28	240	16	8	18.47	25.66	9.09
8	29	241	24	17	19.95	22.42	14.71
8	30	242	24	24	16.02	19.20	13.43
8	31	243	16	15	15.30	21.74	9.34
9	01	244	16	13	14.79	22.68	6.87
9	02	245	14	10	17.02	25.20	7.07
9	03	246	8	3	20.98	27.38	13.70

Table B.1 Continued.

Mth	Day	Day of Year	Duration of LW	HRH	DT	TMAX	TMIN
9	04	247	15	12	18.80	27.08	11.10
9	05	248	19	18	17.18	20.99	14.94
9	06	249	14	15	12.71	16.89	7.57
9	07	250	6	9	10.31	15.50	5.41
9	08	251	13	12	13.15	19.92	6.02
9	09	252	2	8	15.16	23.58	4.56
9	10	253	8	8	17.35	25.77	7.37

Table C.1 Weather data during summer of 1990 at Sainte-Clotilde, Quebec.

Mth	Day	Day of Year	Duration of LW	HRH	DT	TMAX	TMIN
6	04	155	0	0	16.22	19.24	14.59
6	05	156	4	3	17.17	19.88	14.96
6	06	157	7	5	18.61	23.21	14.69
6	07	158	3	4	19.08	23.97	15.28
6	08	159	0	0	17.15	22.76	15.64
6	09	160	11	12	18.26	22.97	19.47
6	10	161	9	11	19.35	23.04	19.92
6	11	162	0	0	22.12	22.92	21.58
6	12	163	0	0	23.38	34.39	13.19
6	13	164	3	1	17.45	22.93	9.45
6	14	165	6	0	22.51	27.45	18.33
6	15	166	20	7	20.28	23.28	18.19
6	16	167	12	10	21.84	27.25	15.55
6	17	168	12	8	23.60	29.97	17.86
6	18	169	16	2	24.09	28.10	19.15
6	19	170	19	2	19.63	23.17	14.35
6	20	171	3	5	14.25	17.41	11.26
6	21	172	10	15	16.58	18.57	14.92
6	22	173	9	11	20.36	27.12	14.68
6	23	174	14	14	19.75	24.13	16.80
6	24	175	8	8	18.35	21.75	15.62
6	25	176	7	3	17.58	23.01	14.85
6	26	177	5	0	21.70	27.19	15.96
6	27	178	9	4	18.20	21.44	15.58
6	28	179	9	5	14.52	18.70	9.89
6	29	180	22	18	11.62	15.80	5.72
6	30	181	9	12	16.72	21.25	12.74
7	01	182	15	18	16.90	21.61	14.47
7	02	183	10	13	18.01	25.61	8.89
7	03	184	10	12	18.31	24.80	8.87
7	04	185	19	9	22.58	24.04	19.50
7	05	186	15	6	18.49	22.47	12.08
7	06	187	15	8	15.17	20.88	8.57
7	07	188	14	8	16.89	23.71	9.81
7	08	189	15	5	19.42	24.34	12.25
7	09	190	18	10	21.88	27.62	17.18
7	10	191	8	0	20.48	24.43	13.00
7	11	192	11	3	17.74	24.03	11.16
7	12	193	17	9	15.91	21.99	7.36
7	13	194	16	11	16.32	25.55	5.12
7	14	195	16	7	18.90	27.03	7.05

Table C.1. Continued.

Mth	Day	Day of Year	Duration of LW	HRH	DT	TMAX	TMIN
7	15	196	21	11	23.09	29.77	18.09
7	16	197	11	5	23.82	27.26	20.77
7	17	198	5	0	23.43	28.79	18.75
7	18	199	0	0	26.36	31.05	22.41
7	19	200	0	0	25.99	28.54	22.46
7	20	201	19	18	19.67	22.21	17.31
7	21	202	12	11	22.08	26.94	18.22
7	22	203	13	10	21.20	26.26	15.80
7	23	204	18	21	17.99	20.41	16.51
7	24	205	6	10	21.06	25.74	16.67
7	25	206	0	10	20.65	27.50	12.40
7	26	207	10	3	22.87	29.45	15.68
7	27	208	9	10	22.60	29.32	14.04
7	28	209	10	10	22.70	30.50	14.33
7	29	210	7	9	23.20	31.42	13.79
7	30	211	8	8	25.24	31.58	18.00
7	31	212	19	14	18.00	22.85	15.26
8	01	213	17	9	19.18	25.27	13.77
8	02	214	8	0	21.75	28.15	15.36
8	03	215	4	0	23.94	28.48	18.66
8	04	216	1	1	24.58	31.16	18.39
8	05	217	16	5	21.29	27.33	16.64
8	06	218	24	20	19.12	20.87	17.32
8	07	219	24	22	18.00	19.39	16.88
8	08	220	10	9	19.27	25.92	11.03
8	09	221	2	3	20.29	27.22	10.73
8	10	222	16	7	20.09	22.81	17.56
8	11	222	24	7	20.09	22.81	17.56
8	12	223	21	14	21.47	27.58	17.54
8	13	224	15	9	20.60	25.67	15.86
8	14	225	24	24	15.72	16.89	14.92
8	15	226	16	12	17.94	23.00	14.20
8	16	227	18	13	18.72	24.24	14.65
8	17	228	16	15	19.44	26.45	13.54
8	18	229	13	10	21.54	29.48	12.70
8	19	230	15	8	22.18	28.53	13.56
8	20	231	9	6	14.55	19.34	8.32
8	21	232	2	12	13.65	22.25	4.18
8	22	233	5	11	14.89	23.30	5.11
8	23	234	4	11	16.92	26.23	6.81
8	24	235	17	12	18.78	27.14	9.84
8	25	236	16	10	20.82	26.93	15.01
8	26	237	14	13	22.21	28.89	16.61
8	27	238	15	8	23.44	30.07	15.86
8	28	239	12	4	23.14	27.23	17.46