CROP-ENVIRONMENT-DISEASE INTERACTION STUDY ON ASCOCHYTA BLIGHT OF CHICKPEA

(*Cicer arietinum* L.)

by

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(L-92-A-35-D)

Dissertation

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(Minor subject: Plant Pathology)

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1996

DEDICATED TO

my revered mother

Smt. Kasturi Devi

CERTIFICATE - I

This is to certify that this dissertation entitled, **"Crop-Environment-Disease Interaction study on Ascochyta Blight of Chickpea (*Cicer arietinun* L.)"**, submitted to the Punjab Agricultural University, Ludhiana, in partial fulfilment of the requirements for the degree of Doctor of Philosophy in the subject of Agricultural Meteorology (Minor subject: Plant Pathology), is a bonafide research work carried out by **Mr OM PARKASH JHORAR (L-92-A-35-D)** under the supervision of **Dr. H.S. Mavi**, Professor of Agrometeorology, upto September, 1995 and under my supervision thereafter and that no part of this thesis has been submitted for any other degree.

The assistance and help received during the course of investigation have been fully acknowledged.

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This is to certify that this dissertation entitled, **"Crop-Environment-Disease Interaction study on Ascochyta Blight of Chickpea (*Cicer arietinum* L.)"**, submitted by **Mr OM PARKASH JHORAR (L-92-A-35-D)** to the Punjab Agricultural University, Ludhiana, in partial fulfilment of the requirement for the degree of Doctor of Philosophy in the subject of Agricultural Meteorology (Minor subject: Plant Pathology) has been approved by the Student's Advisory Committee after an oral examination on the same, in collaboration with an External Examiner.

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ABSTRACT

A crop-environment-disease interaction on Ascochyta blight of Chickpea was studied to establish the empirical relationships between meteorological elements and the disease, effect of moisture conditions on various components of the disease cycle in controlled environment and to assess the disease progress and its influence on crop performance in the field. The empirical study classified the chickpea crop season into risky and non-risky periods for Ascochyta blight epidemics. The period between SMWs 9 to 14 was a risky period in which evening relative humidity (rh) and maximum temperature were good indicator of Ascochyta blight outbreaks. A ratio of these variables (HTR) was the best indicator (R^2=0.93) to depict quadratic relationship with disease development. This study revealed that the crop should be sprayed with protective fungicides in the 8th SMW to check the build-up of inoculum in the field if any. To confirm the empirical relationships, controlled environment experiments at 20°C were conducted. Wetness duration of 2h and 4h were the minimum requirements for germination and penetration of the pathogen and followed a linear increase with increasing wetness. Significant disease developed with 4h wetness and with increasing wetness, the disease increased linearly, reaching a plateau at 18h wetness for a single disease cycle. An increase in leaf dryness immediately after inoculation caused a linear (r =0.92) decrease in the disease development. *Ascochyta rabiei* did not cause infection on inoculated and air dried chickpea plants when exposed to rh $\leq$95 percent. The disease followed a steep linear increase between 98 and 100 percent rh.The disease developed significantly more in dark than light. Sporulation was

maximum with continuous wetness following non-linear trend (r=0.98). The empirical and laboratory studies were validated in field. Disease increased with increasing continuous wetness in the field, reaching a maximum of 86 per cent with seven days wetness. An exponential loss of seed yield was observed with increasing Ascochyta blight. When inoculated plots were supplied with additional wetness, the disease appeared significantly higher than un-inoculated, indicating that inoculum availability in nature needs further investigation. In the plots where inoculum was supplied without additional wetness, the disease did not appear significantly indicating that wetness is limiting for Ascochyta blight development. The study has provided sufficient information to issue weather based agro-advisories to manage Ascochyta blight with minimum use of fungitoxicants. A medium range weather forecasting can further add into this service by providing anticipated weather conditions favourable for the disease development well in advance for timely action by growers to sustain chickpea production.

SIGNATURE OF MAJOR ADVISOR **SIGNATURE OF STUDENT**

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CHAPTER-I

INTRODUCTION

Chickpea (*Cicer arietinum* L.) is a major source of protein for human consumption, provides high quality crop residues for animal feed and helps to maintain soil fertility through biological nitrogen fixation. This crop is grown in ecologically diverse environments in India, the Mediterranean region, eastern Africa, the Americas and Europe where it is subject to varying photoperiod and hydrothermal regimes during its life span. Under such variable environmental conditions, the crop yield ranges from 247 to 1979 kg/ha (Anonymous, 1994). Even under single production area of the world, large fluctuations in its productivity are observed. Interestingly, reduction in crop yield commonly coincides with catastrophic disease of chickpea *ie* Ascochyta blight caused by *Ascochyta rabiei* (Pass.) Lab. This is a major constraint to chickpea production with total failure of the crop being likely when the disease occurs in epidemic form.

Ascochyta blight is most prevalent in latitudes ranging from 26° N to 45° N. Below 26° N and above 45° N, tropical and temperate climatic conditions prevail which limit disease development. Conditions in the 26° N - 45° N latitudinal range often favour this disease which requires moderate temperature, high humidity and wet plant surface resulting from dew or intermittent rains received during the crop season (Nene, 1984). This disease is a major threat to chickpea production in several states of India such as Punjab, Haryana, parts of Rajasthan, Himachal Pradesh, Uttar Pradesh and Bihar where

such an environment prevails.

The large fluctuations in crop yield in relation to occurrence or non occurrence of this disease are well documented (Verma *et al.* 1981; Singh *et al.* 1982; Hawtin and Singh 1984; Malik and Tfail 1984; Singh *et al.* 1984; Trapero-Casas and Jimenez-Diaz 1986; Kaiser and Muehlbauer 1988) and emphasise the need to study the effects of variable temperature, humidity (amount and distribution, leaf wetness) and photoperiod etc., individually and in combination on the behaviour of the pathogen and in relation to crop performance. Quantification of interactions among the host-pathogen-environment will help to achieve consistently high yield by identification and exploitation of appropriate environmental windows favourable and unfavourable for the disease. Keeping this in view, the present study was planned with the following objectives.

1. To establish the empirical relationships between meteorological elements and Ascochyta blight.
2. To study the germination, penetration, sporulation and survival of conidia (pycnidiospores) and its impact on the disease progress under different environments.
3. To assess disease progress and its influence on crop performance under field conditions.

CHAPTER II

REVIEW OF LITERATURE

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The relevant literature covering interactive relationships among different components of the disease triangle *ie* the crop-disease-physical environment is reviewed under following heads:

2.1 EMPIRICAL RELATIONSHIPS BETWEEN DISEASE AND WEATHER

2.2 WEATHER AND CROP DISEASE

2.3 CROP GROWTH AND ENVIRONMENT

2.4 CROP PHENOLOGY

2.1 EMPIRICAL RELATIONSHIPS BETWEEN DISEASE AND WEATHER

The combination of a sufficiently large population of a susceptible host, a virulent pathogen and an adequate duration of favourable environment is required for plant disease epidemics. In an empirical study to quantify the interaction among the host-pathogen-environment, historical data on climatic variables which influence disease such as temperature, moisture, solar radiation and wind, as well as disease intensity are required for statistical analysis.

Diekmann (1992) studied the effect of climatic variables on Ascochyta blight of chickpea caused by *A. rabiei* for various agro-geographical zones of the world and developed a function using mean daily temperature in month 1 of the vegetative phase,mean precipitation in month 2, with the daily average precipitation and mean number of rainy days in months 1 and 2 to indicate blight epidemics globally.

Coakley (1988) developed a computer programme to identify most highly correlated climatic parameters with disease and used the computer programme called WINDOW to give forecasting models using two and three variables which explained 75 and 76 per cent variation respectively in stripe rust of wheat caused by *Puccenia striformis*.

Mavi *et al*(1992) studied the effect of climatic variables on karnal bunt disease of wheat and identified temperature and relative humidity as the weather elements which favour disease outbreaks. Jhorar *et al* (1992), developed a humid thermal index (HTI) by taking the ratio of humidity and temperature during contrasting periods (epidemic and non epidemic) for 20 years. A simple linear model using HTI as a single compound variable explained >90 per cent of the karnal bunt disease variation.

Empirical studies require long term historical data of not only climatic variables but also disease intensity, to establish meaningful relationship between disease and weather. Scanty information is available in case of Ascochyta blight, so there is ample scope for conducting empirical studies in different agroclimatic zones to identify the appropriate environmental windows both favourable and unfavourable to this disease.These could be exploited to achieve considerably higher chickpea yields.

2.2 WEATHER AND CROP DISEASE

Ascochyta blight is thought to be largely influenced by microclimate of the crop which in turn is influenced by prevailing weather conditions. The pathogen will be affected by the weather at all crop growth and development stages up to maturity. Therefore crop losses due to this could vary from 0 to 100 per cent, depending on the weather conditions, which emphasises the scope of using the degree of crop loss as an important tool to identify both the favourable as well as unfavourable weather conditions for the disease (Singh and Sharma, 1995). Quantitative information on crop microclimate and the response of the pathogen is a prerequisite to assess the importance of the microclimate on disease (Butler, 1993).

2.2.1 Effect of weather factors on primary inoculum

In the field, primary inoculum of *A. rabiei* multiplies under favourable weather conditions and spreads when it is wet and windy. Infected and broken plant parts can transport inoculum several hundred metres (Luthra *et al*, 1935). Choube and Mishra (1992), reported that the pathogen survives in crop debris left in the field after harvest and contaminated seed stored for the next sowing.

A study conducted by Ram Singh and Mohinder Pal (1993), revealed that survival of *Aschochyta rabiei* on plant debris declines rapidly with increasing temperature from 20 to 45°C. At 15, 20 and 45°C, the survival was 3.3, 10 and 0 per cent respectively. In field, the pathogen survived for 2.5 months at 5 cm depth and for shorter period at greater soil depths.

Kausar (1965), revealed that areas where chickpea crop follow hot summer and heavy rains, the fungus survive for very short duration because the crop debris gets decomposed and fungus can not withstand extreme weather.

Trapero-casas and Kaiser (1992) revealed that in the perfect stage of pathogen, ascospores are released during vegetative stage of chickpea indicating the possibility of serving as primary inoculum for Ascochyta blight under favourable weather conditions particularly when high moisture conditions prevail. However, the perfect stage of the pathogen has not yet been reported in India (Singh and Sharma, 1995).

The studies conducted in the field, under varied environments covering different ranges of temperature, rainfall and soil depth revealed that mild temperature($\sim$20°C) may be favourable for primary inoculum, whereas, hot summer ($\sim$45°C) has proved to be detrimental for survival of the pathogen. Soil depth up to 5 cm proved to be beneficial for pathogen, whereas greater depths reduced the survival period of the pathogen. Hot and humid conditions apart from their direct effect, through heat and moisture, has also shortened the survival period through indirect effect by decomposing the crop debris resulting in non availability of the food material for pathogen.

2.2.2 Effect of temperature on the disease

Colhun (1973) suggested that in general, disease development continues as long as healthy plant tissue is available under the favourable weather conditions. Temperature influences various fungal diseases and if it is too low or too high, the disease development stops. Ascochyta blight is favoured by moderate temperature (15 - 25°C) and

frequent rainfall (Zachos *et al*, 1963; Kauser, 1965; Askerov, 1968; Chauhan and Sinha, 1973; Weltzien and Kaack, 1984; Dey, 1990 and Trapero-Casas and Kaiser, 1992).

Weltzien and Kaack (1984) revealed that an epiphytotic onset of Ascochyta blight of chickpea, can be related to the weather. When the air temperature is below 6°C wet conditions prevailing for any length of time can not produce Ascochyta blight. Similarly, when wetness is < 6 h, Ascochyta blight does not develop even with most favourable temperature. Its development is fast when temperature is between 9 and 24° C and wetness is longer than 6 h. It can be concluded that cool, dry weather conditions are totally unfavourable, whereas, weather with mild temperatures, coupled with prolonged wet periods accelerate this disease. Reddy and Singh (1990) observed that cool temperatures (minimum temperature <5°C and maximum temperature <15°C) limited the disease development even with high humidity, whereas weekly mean temperatures between 10 and 20°C with high relative humidity promoted Ascochyta blight development.

The studies conducted by Singh (1984), revealed that pycnidiospore start germinating at 10°C and continue to germinate at an increasing rate with the increase in the temperature reaching to peak at 20°C. The spore germination stop at 30°C. With continuous wetness ,temperatures less than 10°C and greater than 25°C were found unfavourable for infection and 20°C was the optimum temperature for rapid establishment of pathogen (Trapero-casas and Kaiser 1992).

The effect of temperature in prolonging or shortening the incubation period has

been reported by several workers. The incubation period (the time taken from infection to the appearance of symptoms) of *A. rabiei* was minimum (about 4.5 days) at 20°C, and it increased with both lower and higher temperatures. A second degree polynomial equation with temperature as an independent variable explained 85 per cent of the variability in incubation period for temperatures below 30°C. The latent period (the time taken from infection to the beginning of sporulation) at temperatures of 5, 10, 15, 20 and 25°C was observed to be 23, 12, 9, 7 and 8 days respectively. A second degree polynomial equation with temperature as an independent variable explained 93 per cent variability in the latent period for temperatures below 30°C (Trapero-Casas and Kaiser, 1992).

Optimum temperature for fungal growth was observed to be 20°C, whereas, below and above this level, the growth was retarded. Minimum fungal growth was observed at 10 and 30°C (Bedi and Aujla,1970; Singh,1984 and Singh, 1987).

Kaiser (1973) reported that optimum temperature for spore production was 15 to 20°C. The pycnidia containing viable spores were formed on dried chickpea stem between 10 and 30°C but the formation was fast at 20°C. Pycnidial growth was observed to be maximum at 25°C, whereas the size of the pycnidia was maximum at 20°C followed by 15 and 25°C and the temperature at 10°C was least favourable for pycnidial size. The maximum size of pycnidiospore was observed at 25°C followed by 20, 15, 10 and 30°C (Singh, 1984 and Singh,1987).

Hassani (1981), on the basis of laboratory studies conducted in France, suggested

that temperature is not an inhibiting factor for spread of the disease but only a factor which modifies the rate at which the disease spreads. He advocated the importance of moisture more than the prevailing temperature.

The effect of temperature in regulating the duration of different stages of the disease cycle, *i.e.* from infection to symptom appearance, fungal growth, spore production and ultimately beginning of next disease cycle, has identified it as an important weather variable.

2.2.3 Effect of moisture on the disease

Moisture in general is an important weather element which in different forms, influences all fungal pathogens infecting aerial plant parts. When the moisture requirements of a pathogen are fully met under favourable temperature conditions, it multiplies on a susceptible host at a maximum rate to increase disease intensity and incidence. If such conditions prolong for sufficient time, epiphytotic outbreak is certain. This kind of potential role of moisture has been exploited frequently as a base to forecast disease (Royle and Butler, 1986; Butler, 1992).

2.2.3.1 Effect of rainfall on the disease

Movement of water in canopies is most commonly associated with rain. Of the diverse attributes of rain, time, frequency and duration are critical in determining plant surface wetness and pathogen dispersal in plant communities through trickling and splashing. **The intensity of rain as a function of the number, size and velocity of**

droplets affects disease in different ways. The cumulative effect of these factors may affect plant disease epidemic outbreaks (Ooka and Commedohl, 1977; Sutten and Yanes, 1979; Fitt *et al*, 1982; Sutten *et al*, 1984; Royle and Butler, 1986).

During the chickpea growing season, Kauser (1965) studied the influence of winter rainfall on epiphytotic and revealed that high rainfall resulted in high chickpea blight incidence. Summer rains, however, had a negative effect on the disease. **Ascochyta blight is most serious, when the winter sown chickpea in north-west India and Pakistan and spring sown chickpea in Mediterranean region receive frequent rains during crop season** (Reddy *et al*, 1990).

2.2.3.2 Effect of surface wetness on the disease

Leaf surface wetness normally refers to dew or rain on aerial parts of the plant surface. The duration of wetness is of chief interest in epidemiology, though the amount and form of free water may also be important (Van der Wal, 1978). Wetness duration varies considerably not only with weather conditions but also with the type and developmental stages of crop, the position, angle and geometry of leaves and specific location of individual leaves. The role of wetness in infection by pathogens has been demonstrated by a number of researchers, indicating a global range of wetness duration requirements varying from 0.5 to more than 100 h (Huber and Gillespie, 1992). The effect of free water on sporulation has been little explored and remains a bottle-neck in the application of forecasting methods. Sporulation, however may require longer wetness periods than the infection process.

Host surface wetness is prerequisite for surface contamination, infection and rapid development of Ascochyta blight on chickpea. **Spores of *A. rabiei* are only released from wet pycnidia and at least 6 h of wetness is necessary for infection by this organism at the optimum temperature** (Weltzien and Kaack, 1984).

Trapero-Casas and Kaiser (1992) stated that longer wetness is required for the infection with sub optimal temperatures. They, however, could not establish the minimum threshold wetness to cause infection but observed the rapid increase in the disease from 3.5 to 81.1 per cent when wetness duration was increased from 6 to 48 h. When dryness of 6 h after inoculation was followed by 24 h wetness, the disease increased to 86 per cent.

Hassani (1981), revealed that rate of spore germination of *A. rabiei*. under free water conditions reached 100 per cent and also observed the germ tube formation on both ends of the spore.

2.2.3.3 Effect of humidity on the disease

Many research workers have reported the importance of humidity in the development of various diseases. It directly influences sporulation by many fungi and has implications for the persistence of wetness. It is often a good indicator of wetness or dryness of the plant surface, though it (humidity) may not always correlate with wetness (Royle and Butler, 1986).

Reddy and Singh (1990) reported that Ascochyta blight is influenced by high

humidity in association with favourable temperature. When weekly mean temperature was within optimum range, weekly mean relative humidity of >60 per cent resulted in epidemic outbreak in winter sown chickpeas. Singh *et al.*(1982), reported that relative humidity above 85 per cent is required for 48 h to cause the infection. The results of the study conducted by Chauhan and Singh (1973), revealed that relative humidity between 85 and 98 per cent is most favourable for disease development. Studies conducted by Hassani (1981), revealed that **humidity is a limiting factor for spore germination and for triggering epiphytotic. When relative humidity remained below 98 per cent, spores of *A. rabiei* did not germinate, whereas, between 98 and 100 per cent relative humidity, a delay in sporulation and germination compared to wetness, was observed.**

2.2.4 Effect of light and dark periods on the disease

The effect of light and dark periods on fungal sporulation has been studied by many researchers. Light may influence spore germination, penetration, infection type, spore release and viability (Colhoun, 1973).

A study conducted by Kaiser (1973) revealed that continuous light increased mycelial growth and sporulation of *A. rabiei* compared with continuous darkness on potato-dextrose agar (PDA) and chickpea seed meal (CSMA). However, the sporulation under alternate light and dark conditions was always greater. Nene (1984), revealed that increased light duration increased the sporulation of *A. rabiei.*

Raffray and Sequeira (1971) reported that light had an inhibitory effect on sporulation and suggested that at least 6 h darkness after 7 days of inoculation is required for *Bremia lactucae* sporulation.

2.3 CROP GROWTH AND ENVIRONMENT RELATIONSHIP

Chickpea is grown in ecologically diverse environments such as the post-rainy season in India, Pakistan and Nepal; during winter, spring and summer in the mediterranean regions (Saxena, 1984). Phenology, growth and yield of chickpea under such diverse environments differ considerably, being subjected to varying photoperiod, temperature and moisture regimes. In the tropical region of peninsular India, the southern limit of the crop is 17° N, where the crop is sown on stored soil moisture in the post-rainy season where 40 - 100 mm of rainfall is received during the crop season. The crop is sown when the minimum and maximum temperatures drop to 20 and 30°C respectively. Thereafter a slight decline in both minimum and maximum air temperature by 4 and 7°C favours the active growth of the crop. Later in the growing season, an increase in both minimum and maximum air temperatures by 5 and 6°C, respectively, coupled with depleting soil moisture forces the crop to complete its life span in only 100 days. In the semiarid subtropical climate of north India (~31°N) *ie* the central plain region of Punjab, the crop experiences a gradual but considerable decline in both maximum and minimum air temperatures over a longer period leading to prolonged active growth. Thereafter, a slight increase in air temperature favours pod setting A rapid rise in the minimum and maximum temperatures from 8 and 25°C to 20 and 37°C respectively, from February to

April hastens senescence and forces maturity even when the crop is raised with assured irrigation or receives well distributed rainfall of around 130 mm during the season. The growing season usually last between 150 and 170 days. In contrast to tropical climates, chickpea can be grown in cooler areas where minimum temperature falls below 0°C. However, crop growth duration in such areas is longer (Huda and Virmani, 1987; Virmani, 1991).

According to Summerfield (1980), the chickpea crop experiences markedly different photothermal regimes globally, and the rate of change of day-length during the crop season depends on where and when the crop is grown. However, for its growth and development, the crop requires warm days (20 - 25°C) and cool nights (5 - 10°C) coupled with 200 to 300 mm of well distributed rainfall during its life span (Sinha, 1977). Air temperatures of around 30°C and above were observed to be harmful to pod formation (Jagdish Kumar, 1990) but a slight rainfall coinciding with the reproductive phase was found to give best biomass and seed yield (Jodha and SubbaRao,1987).

A study conducted at New Delhi by Prabhakar and Saraf (1990), revealed that up to 60 days after sowing (DAS) dry matter accumulation is slow and thereafter, it increases rapidly reaching to a peak at around 120 DAS. The maximum dry matter accumulation is recorded at harvest. The increase in dry matter accumulation at 90 DAS is attributed to rise in day temperature. Under Punjab conditions, Gill *et al* (1988a), observed that dry matter accumulation in leaves is low up to 60 DAS which increase five fold after 60 days to reach a peak at 90 DAS. Stem dry matter continues to increase up to maturity. Pod dry matter accumulation is initiated 90 DAS and continues to increase up to maturity. The

maximum seed dry weight is also attained at maturity. The total dry matter increases as the crop growth stage advances (Gill *et al*, 1988b). Murinda and Saxena (1983), suggested that when chickpea is raised under moisture and temperature stress during the early phase of reproductive growth, it results in poor yield.

2.4 CROP PHENOLOGY

Phenology deals with periodic biological phenomena *ie* changes in the developmental state of plants in relation to climatic parameters. The rate at which these changes take place are sensitive to temperature, moisture and photoperiod (Edwards *et al*, 1986). An ability to predict phenology in different environmental circumstances enables the crop to be grown for maximum exploitation of environmental resources like moisture, temperature and solar radiation (Summerfield *et al*, 1991).

Chickpea passes through the pheno-phases of germination, emergence, vegetative growth, reproductive growth and maturity, which are strongly influenced by the prevailing weather elements. Sharma and Sonakiya (1990), used the Growing Degree Day (GDD) concept to compare different cultivars of chickpea and observed significant variability in GDD for various crop phenological stages. Summerfield *et al* (1981), observed that a group of cultivars suffered severely when exposed to hot days near maturity, reducing the average seed yield by one third while some cultivars escaped stress during the reproductive period by maturing early. In chickpea, crop duration is largely dependent on time between sowing and flowering (Pundir *et al*, 1988). An increase in the vegetative period results in a greater accumulation of the photosynthates which contribute to final

yield. When the length of the reproductive phase is increased, a prolonged change in the source-sink relationship contributes more towards seed yield. The time from sowing to flowering is determined by the mean photoperiod and temperature during the vegetative phase. The rate of progress towards flowering (f) is a linear function of temperature (t) and photoperiod (p) which can be expressed through an expression $^1/_f = a + bt + cp$ where a, b, and c are constants which vary with genotype (Roberts *et al*, 1985).

According to Roberts and Summerfield (1987), the rate of progress towards flowering rather than flowering time was best quantified by photoperiod and temperature. Eshal (1967), concluded that the effect of measured day length on shortening of the vegetative growth period was stronger than increased temperature. Studies conducted by Ellis *et al* (1994), revealed that the time from sowing to flower initiation varied from 30 to 160 days, as the mean pre-flowering temperature varied from 10.8 to 29.3°C and the mean photoperiod varied from 11.3 to 15.6 h d^{-1}.

A comparison of crop seasons under diverse climatic conditions, shows that under warm tropical conditions the combined effects of temperature, moisture, photoperiod not only shorten the total life span of the crop (the pre-flowering and post-flowering periods) but also result in poor yield. In cooler climates, the prevailing climatic conditions increase the total crop duration (the periods of vegetative growth as well as seed development) which results in greater crop production.

CHAPTER III

MATERIALS AND METHODS

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This investigation was primarily aimed to establish relationships between the chickpea crop, the environment and Ascochyta blight caused by *Ascochyta rabiei* (Pass.) Lab. The studies were divided in three parts *ie* (a) to establish an empirical relationship between climate and disease, (b) to investigate weather factors influencing the disease cycle under controlled environment, and (c) to substantiate the impact of weather elements on the crop and disease in field experiments.

3.1 ESTABLISHMENT OF EMPIRICAL RELATOINSHIPS BETWEEN CLIMATE AND DISEASE

3.2 LABORATORY EXPERIMENTS

3.3 FIELD EXPERIMENTS

3.1 ESTABLISHMENT OF EMPIRICAL RELATIONSHIPS BETWEEN CLIMATE AND DISEASE

In this study daily meteorological data from the meteorological observatory of department of Agricultural Meteorology situated at the experimental research farm of Punjab Agricultural University, Ludhiana (30° 54' N, 75° 48' E and at an altitude of 247 m above mean sea level) were collected for the last 25 years (1970 to 1994). The variables included were maximum, minimum and mean air temperature; maximum,

minimum and mean relative humidity (rh), sunshine duration, rainfall and rainy days. The data on Ascochyta blight for the corresponding period were obtained from a chickpea pathologist in the department of Plant Breeding, Punjab Agricultural University, Ludhiana. The disease data were collected on the experimental farm from naturally infected experimental plots. A scale of 0 to 9 rating was used uniformly, where 0 means no disease and 9 means more than 90 per cent disease (Appendix-I). The disease index (DI), calculated by taking ratio of the actual rating to the maximum rating for each year, was used for further analysis.

Weekly averages of temperature, rh and sunshine duration and weekly totals of rainfall and rainy days were calculated from the first standard meteorological week (SMW) (beginning from Ist January) to seventeenth SMW (ending on 29th April). This period covered the active vegetative growth, reproductive and maturity phases of the crop.

3.1.1 Coefficients of correlation

Correlations between meteorological elements and the terminal disease index of the corresponding years were obtained for each variable and each week to generate a window of disease-favourable weather. The disease favouring weather elements were segregated for further processing using the computer software STATISTIX (SX).

The weather elements *viz* maximum temperature and evening relative humidity falling in the disease favourable window (Table -1) were aggregated (with each year over 25 years period) for the period 9 to 14th SMWs in which regularly significant coefficients

of correlation were obtained. The aggregated values of these two weather variables were again correlated to find out any change in the coefficients. Evening relative humidity and maximum temperature falling in the window were converted in to a single variable for each year by taking ratio of evening relative humidity and corresponding maximum temperature. A correlation was carried out between this new variable referred to as the humid thermal ratio (HTR), and disease.

3.1.2 Fitting the curve

The most favourable weather factors *viz.* maximum temperature, minimum relative humidity and HTR were plotted against disease index (ratio of actual rating to maximum rating) to visualise the shape of the curves and the most appropriate equations were fitted *viz.* a straight line for temperature verses disease index, an asymptotic exponential function for evening relative humidity and a quadratic function for humid thermal ratio (HTR).

3.2 LABORATORY EXPERIMENTS

The laboratory study was divided into a number of short duration experiments in which wetness and rh requirements for the infection process were studied on 15 days old chickpea plants. These were raised in small pots (5 x 5 x 5 cm) filled with 50 per cent loam, 25 per cent sand and 25 per cent compost. The medium was steam sterilized and placed in a glass house where temperature ranged between 20 and 28°C (Plate 1). Four seeds were sown in each pot and two plants were retained for the experiments. Each

Plate 1. Chickpea plants growing in a glass house

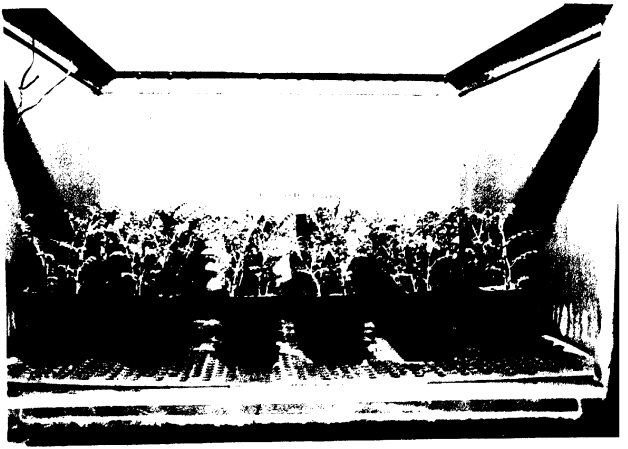


Plate 2. Chickpea plants provided with wetness in a dew chamber at 20°C

experiment was repeated for four times to confirm the results.

3.2.1 Inoculum standardisation

This experiment was conducted to standardise the inoculum requirement for subsequent experiments. The culture of *A. rabiei* race 11 was multiplied on water soaked and sterilised chickpea seeds (at 15 psi for 20 minutes in an autoclave). One week old pycnidia containing pycnidiospores were washed with distilled water by stirring for 10 minutes after adding a few drops of Tween 80 wetting agent (polyoxyethylene sorbitan mono-oleate) and filtering through muslin cloth. The spore concentration was checked with a haemocytometer and dilutions were made to obtain the desired concentration.

The 15 days old chickpea plants were inoculated with pycnidiospore suspensions of 1.25×10^5, 2.5×10^5, 5×10^5, 10×10^5 and 20×10^5 spores ml^{-1}. The spore suspension was sprayed with a plastic atomizer, ensuring that both sides of the leaves were completely wet. Inoculated plants were placed in dew chambers at 20°C for 24 h. The dew chambers were based on the design of Clifford (1973) with a cool outer cabinet and heated water bath in the inner chamber. The temperature of the inner chamber was controlled by switching on the water bath heater only when air temperature at plant height was below the set value. This resulted in the temperature always remaining within 0.5°C of that required. The temperature sensor was a thermocouple attached to a data logger (CR 10, Campbell Scientific Inc., Logan, UT, USA). A Randomised Block Design (RBD) was used to account for variation due to external factors by replicating the treatments four times (Plate 2). After 24 h the plants were moved to controlled environment growth

cabinet (Conviron Model CG 1011, Winnipeg, Canada), where the photosynthetic photon flux density (PPFD) was about 450 micro-mol $m^{-2}s^{-1}$ and the temperature and rh were constant at 20°C and 85 per cent respectively. During night, lights were switched off for 12h, keeping other conditions similar to that of day.

The plants were observed daily to find out the time taken for first symptom appearance and the percent disease intensity was observed at seven day intervals. The concentration of the inoculum producing moderate disease was selected for subsequent experiments.

3.2.2 Effect of wetness on germination and penetration

In this experiment, six treatments of 0, 2, 3, 4, 6, 12, 18 and 24 h wetness at 20°C temperature were imposed in a dew chamber on 15 days old chickpea plants inoculated with a concentration of 10 X 10^5 spores ml^{-1}. The inoculum was prepared just before inoculations from a week-old culture using the techniques described above.

The plants were moved to the dew chamber to ensure wetness and to the growth chamber for periods of dryness. After providing the required treatments, the plants were finally moved to the growth chamber for symptom appearance.

Four leaflets from each of the 2nd, 3rd, 4th and 5th leaves were detached for microscopic observations of germination and penetration for each treatment. Samples were collected at 0, 3, 6, 9, 12, 18, 24, 36, 42, 48 and 54 h interval from the beginning of the experiment.

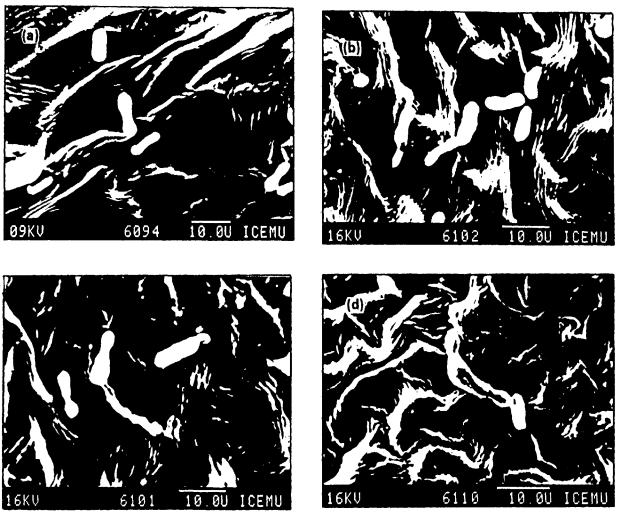


Plate 3. Scanning electron micrographs of (a) pycnidiospores, (b) germinating pycnidiospores, (c) pycnidiospores with long germ-tube and (d) pycnidiospores with typically swollen and elongated germ-tube

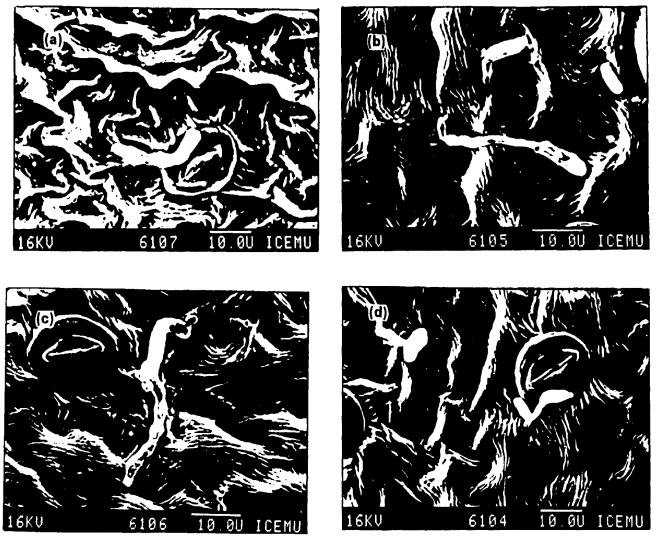


Plate 4. Scanning electron micrograph of (a) germinated pycnidiospore with germ-tube moving away from stomata, (b) elongated germ-tube in search of suitable place for penetration, (c) pycnidiospore producing germ-tubes on both ends, one end of which is about to penetrate in the host and (d) pycnidiospore lying on stomatal wall with germ-tube penetrated through the cuticle

Plate 5. Chickpea plants with a film of free water in a dew chamber

Plate 6. A close view of wetness on chickpea plants

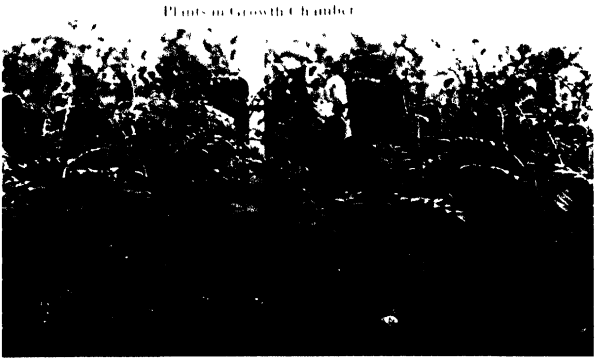
The detached leaflets were observed under scanning electron microscope (SEM) to confirm the identity of ungerminated, germinated and penetrated spores of *A. rabiei* (Plate 3 and 4) because the identity of these stages were not clearly explained in available literature. Samples were also taken to the laboratory and cleared by dipping in acetic acid and alcohol solution for 4 h. The cleared leaflets were washed in running water to remove acetic acid and alcohol solution. The leaves were then submersed in cotton blue dye to stain the fungus. The stained leaflets were examined under a light microscope with a 10 x 40X microscopic field. Five microscopic fields with one hundred spores in each field were observed and the number of germinated spores and host penetrations were recorded.

The plants were also observed regularly after one week to note the first appearance of symptoms and per cent disease intensity. They were then moved to the glass house.

3.2.3 Effect of wetness duration on the disease

In this experiment, six treatments of 2, 4, 6, 8, 10, 12, 18 and 24 h wetness at 20°C temperature were imposed in a dew chamber using RBD design on 15 day old (inoculated) chickpea plants (Plate 5,6). The plants were inoculated with a 10 X 10^5 spores ml^{-1} suspension from a seven day-old culture. The inoculum was prepared just before inoculations using the techniques described above.

The plants were moved to the growth chamber after completion of the treatment and were kept in the same design as in the dew chamber (Plate 7). The plants were observed regularly to note the first appearance of disease symptoms. On the sixth,



7 Chickpea plants under observation for symptom development in a growth chamber

Plate 8. Chickpea plants in a growth chamber with Ascochyta blight symptoms

seventh, eighth and ninth day after inoculation, each leaflet was observed carefully (except the top leaf which appeared after inoculation) and the percentage of necrotic area induced by the pathogen was assessed for each leaf (Plate 8). The disease intensities so assessed, were aggregated for each plant and recorded as per cent disease intensity for that day. In the first experiment, it was observed that the disease did not increase further seven days, so in subsequent experiments observations were recorded on the seventh day after the inoculations. The plants were removed to glass house after recording disease.

3.2.4 Effect of leaf dryness on Ascochyta blight

In this experiment, inoculated plants were exposed to six treatments of 0, 0.5, 2, 4, 6 and 8 h dryness at 20°C temperature and 85 per cent rh in plant growth chamber. The plants with 0 dryness were placed in dew chamber immediately after inoculation and those in other treatments were dried under a fan before exposure to respective treatments. The plants were moved to the dew chamber after the required period and then 24 h continuous wetness was applied to each treatment. The pots were kept in the same design as in the growth chamber. After 24 h wetness, plants were again moved back to the growth chamber for symptom appearance.

The plants were observed regularly to note the first appearance of symptoms and disease intensity (%) was recorded on each plant after seven days as described earlier. The plants were then moved to the glass house.

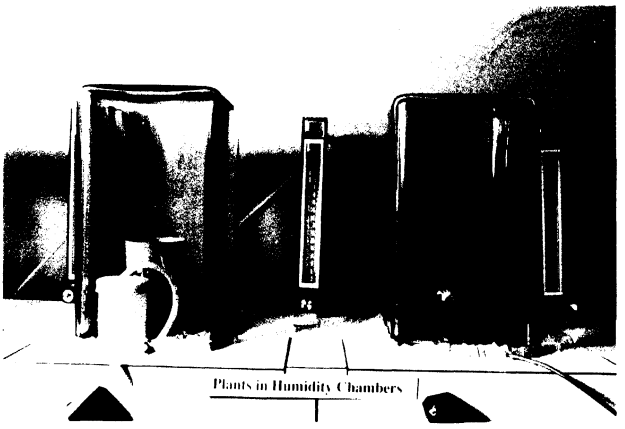


Plate 9. Chickpea plants in controlled humidity chambers at different humidity levels

3.2.5 Effect of relative humidity

The effect of relative humidity was studied in chambers (Plate 9), housed in a basement room. The air temperature of the room was maintained at 24°C with an evaporative cooling system. The chambers were 15(L) X 13 (W) X 24 (H) cm glass storage vessels inverted and sealed to a flat surface covered with laminated plastic. The base of each chamber was removable to insert a plant and the base of each plant stem was sealed with Blue-tac (Bostic Ltd, Leicester, UK).

Each chamber was supplied with moist and dry air mixed in an appropriate ratio to achieve the desired humidity. The plant chambers were used to study the effect of high rh on infection caused by *A. rabiei* resulting in chickpea blight. The inoculated plants were dried under a fan to remove wetness and then exposed to 90, 92, 95, 98, 99 and 100 per cent rh continuously for 48 h. The plants were then transferred to the controlled environment chamber for symptom appearance. As in earlier experiments, the first appearance of symptoms and the disease intensity were observed.

3.2.6 Effect of light and dark period

This experiment was conducted in a growth chamber in which the temperature was controlled at 20°C and rh at 85 per cent. The plants were given 3 treatments *viz.* (i) 12/12 h light and dark periods, (ii) 24 h dark followed by 12/12 h light and dark and (iii) 48 h dark followed by 12/12 h light and dark. The plants were kept in a 30 X 30 cm plastic tray containing free water and covered with polythene bags to maintain nearly saturated moisture conditions. Transparent bags were used to provide light to the plants and black

Plate 10 Chickpea plants in the dark and light

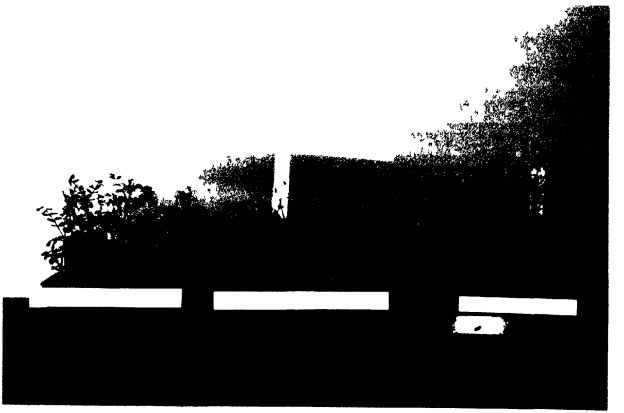


Plate 11 Chickpea plants with Ascochyta blight symptoms

coloured polythene bags were used to keep the plants in the dark (Plate 10).

The plants were kept continuously in the growth chamber after completion of the treatments and were observed regularly to note the first appearance of the disease symptoms and disease intensity (%) on each plant after seven days (Plate 11). The plants were then moved to a glass house.

3.2.7 Sporulation study

In this experiment, inoculated plants were placed in a dew chamber for 24 h and then moved to a growth cabinet set to 20°C and 85 per cent rh. Symptoms appeared after 5 days and pycnidia (black dots of pin head size) were observed on the leaves.

The diseased plants in one treatment were kept in the growth cabinet at 85 per cent rh. A second set of diseased plants was given the treatment of wetness and were moved to a dew chamber. A third set of diseased plants was provided high rh by covering with transparent polythene in a tray containing free water. A fourth set of diseased plants was covered with black polythene in tray containing free water.

The two leaves with the highest disease intensity were marked on each plant in all the treatments and number of pin head size pycnidia were counted daily on each leaflet using a stereo-microscope. Two other leaves from each treatment were detached and the spores were dispersed in a known volume of water using a stirrer. The spore suspension, so obtained, was observed under a light microscope and the number of spores were counted using a haemocytometer. The residue left after filtering the suspension was again stirred in known volume of water and observed under microscope. Interestingly no

spore was detected in the second suspension, confirming that no detachable spore was left in host tissue. This process was repeated each day for 8 days when number of pycnidia and pycnidiospores stopped increasing. This experiment was repeated twice to confirm the results.

3.3 FIELD EXPERIMENTS

3.3.1 Site

The field experiments were conducted, within the vicinity (radius of 100 m) of the meteorological observatory (situated at 30° 54' N and 75° 48' E at an altitude of 247 m above mean sea level) of department of Agricultural Meteorology, Punjab Agricultural University, Ludhiana, during *rabi* season of 1993-94 and 1994-95.

3.3.2 Climate

This area is characterised by subtropical, semiarid climate with very hot summers from April to June and cold winters from December to January. The average annual rainfall at Ludhiana is 660 mm about 75 per cent of which is received from July to September. The normal rainfall during the crop season (October to April) is 120 mm. The meteorological data for the chickpea crop seasons of 1993-94 and 1994-95 are presented in Figures 1 to 4.

Fig.1 Air temperature and relative humidity in rabi 1993-94

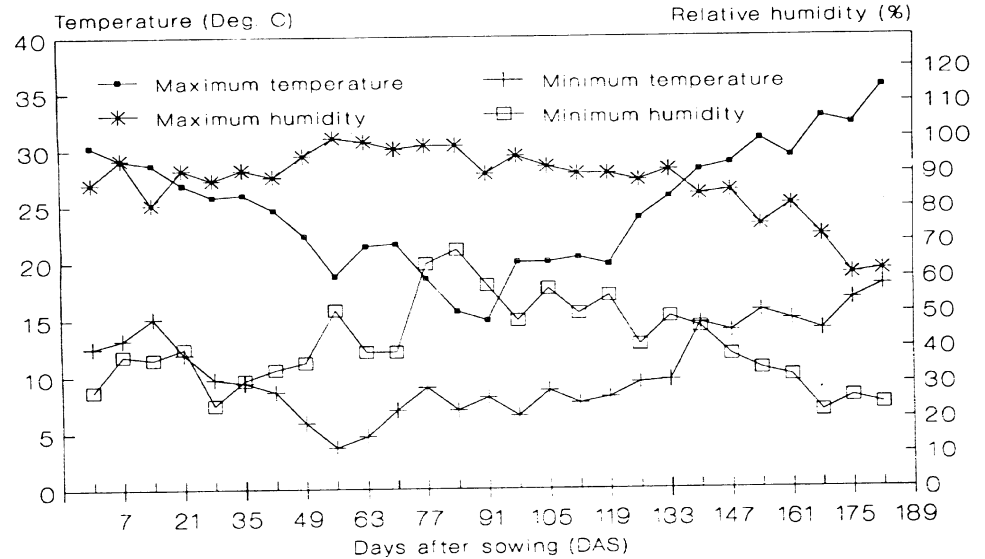


Fig.2 Insolation, sunshine, day length, rainfall and fog in rabi 1993-94

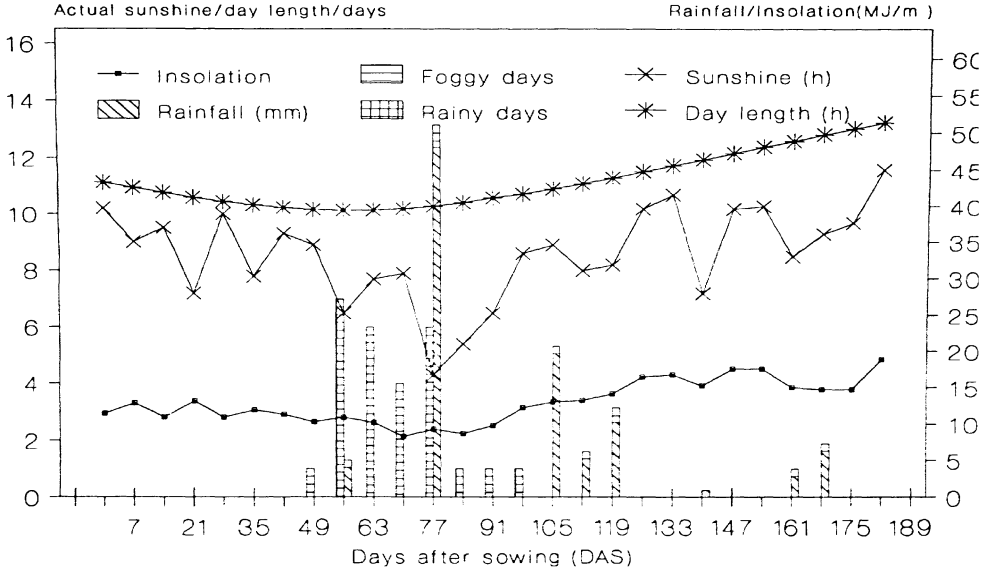


Fig.3 Air temperature and relative humidity in rabi 1994-95

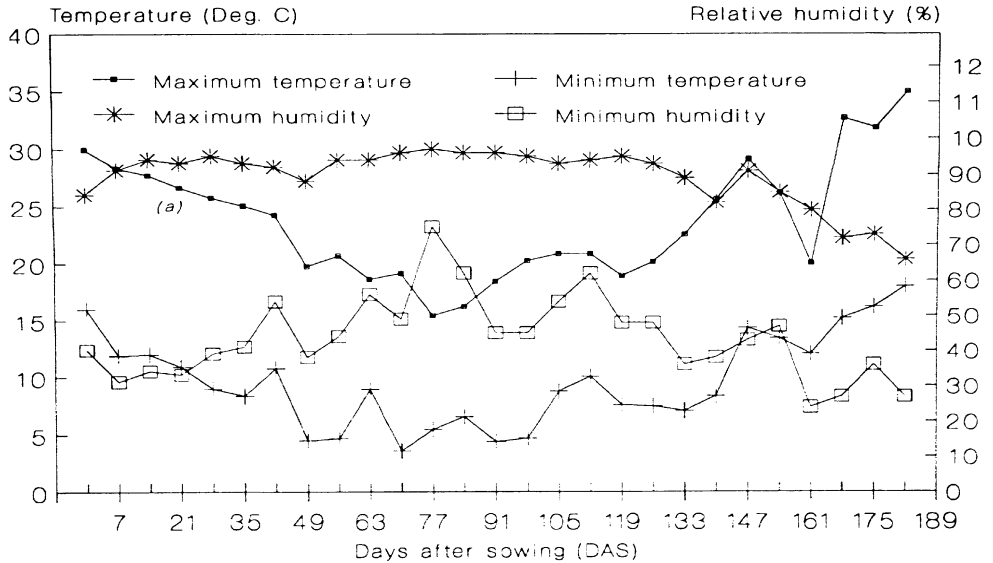
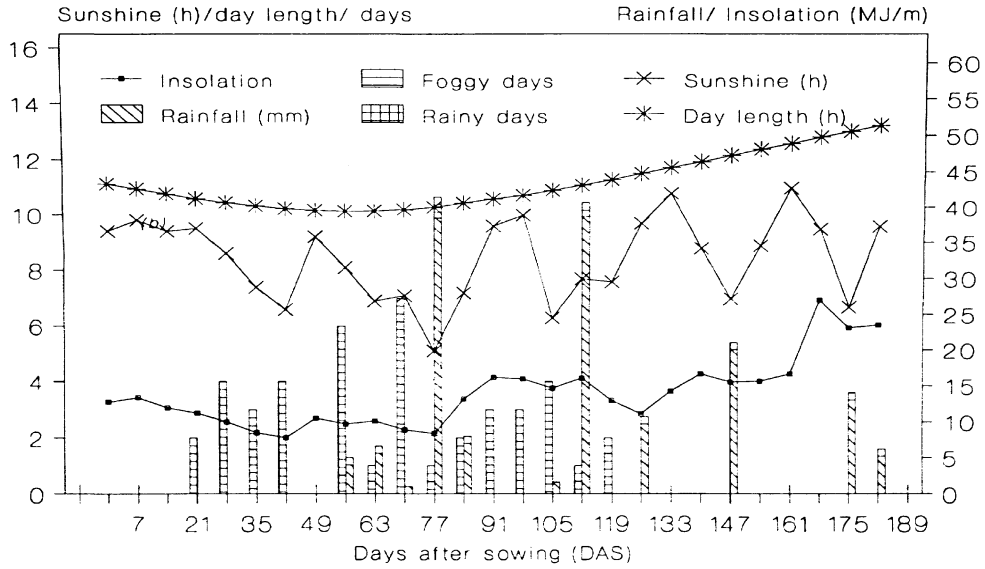


Fig.4 Insolation, sunshine, day length, rainfall and fog in rabi 1994-95



3.3.3 Soil

The experiments were conducted on a loamy sand with 88, 1.4 and 10.6 per cent sand, silt and clay respectively. The bulk density of the soil was 1.44 g cm^{-1} and the field capacity was 22.8 cm m^{-1} in the top 15 cm. The pH of the soil was 8.2, the EC was 0.12 ds m^{-1} and the CEC was 4.18 me $(100g)^{-1}$.

3.3.4 Cropping history of the field

The cropping history of the field is given below:

Year	Crops grown	
	Kharif	*Rabi*
1991 - 92	Fallow	Fallow
1992 - 93	Fallow	Fallow
1993 - 94	Moongbean	Chickpea
1994 - 95	Peanut	Chickpea

NW: Natural infection with additional wetness

In the case of the second treatment, the plots were neither subjected to inoculum spray nor to the chemical spray. The plants were exposed to natural infection and were observed regularly to assess disease, if any.

CW: Chemical control with additional wetness

In this treatment, the plots were sprayed to control the disease. The contact fungicide Kavach (Cholorothelonil) was sprayed @ 500g ha^{-1} in 250 l of water at weekly intervals to prevent the pathogen from causing infection.

All the plots under the treatment IW, NW and CW were provided with continuous wetness for one week from the date of inoculation using rotating sprinklers and disease observations pertaining to the primary infection (cycle) were recorded. After the first wetness cycle of seven days, the plants were exposed to natural wetness conditions for two weeks and thereafter continuous wetness was provided for seven more days. Then the secondary infection (cycle) of the disease was recorded.

I: Artificial inoculation

Another set of four randomly selected plots were inoculated using 1×10^5 pycnidiospores ml^{-1}. The inoculum was sprayed in the evening at around 1800 hrs when dew began to wet the foliage. These inoculations were also carried out on 1st March, 1994 and 21st February, 1995 using a knapsack sprayer and taking care to prevent inoculum drift from the treatment plot to the adjoining plots.

N: Natural infection

In the case of this treatment, the plots were neither subjected to the inoculum spray nor to the chemical spray. The plants were exposed to natural infection and were observed regularly for disease assessment.

C: Chemical control

In this treatment the plots were sprayed to control the disease. A contact fungicide Kavach (Cholorothelonil) was sprayed @ 500 g ha^{-1} in 250 l of water at weekly intervals to prevent infection by *A. rabiei*

The plots under I, N and C treatments were only exposed to natural wetness.

3.3.6 Cultural operations

3.3.6.1 Preparation of the field

The experimental field (dry) was ploughed twice with a disc harrow and then a pre-sowing flood irrigation was applied. When the moisture reached field capacity, the plot was cultivated and planked to sow the crop.

3.3.6.2 Fertilizer application

Diammonium phosphate (DAP) and Urea were applied to provide 15 kg N /ha and 20 kg P_2O_5/ha at the time of sowing as per PAU recommendations.

3.3.5 Treatments and experimental details

Design: Randomised Block Design (RBD)

Treatments: 6 (Six)

IW: Artificial inoculation with additional wetness

NW: Natural infection with additional wetness

CW: Chemical control with additional wetness

I: Artificial inoculation

N: Natural infection

C: Chemical control

Replications: 4 (Four)

Gross plot size: 8 X 6 m

Net plot size: 7 X 5 m

IW: Artificial inoculation with additional wetness

Four randomly selected plots were inoculated using 1 X 10^5 pycnidiospores ml^{-1}. The inoculum was sprayed in the evening at around 1800 hrs when dew began to wet the foliage. The inoculations were done on 1st March, 1994 and 21st February, 1995 using a knapsack sprayer taking care to prevent the inoculum drift from the treatment plot to the neighbouring one.

3.3.6.3 Method of sowing

The crops were sown on October 22, 1993 and October 26, 1994 by using a seed rate of 92 kg/ha. The seed (*cv* L-550) was drilled at 5 cm depth keeping row to row spacing 30 cm and plant to plant spacing 5 cm.

3.3.6.4 Irrigation

During both the crop seasons, a post-sowing irrigation of 75 mm depth was applied during the first week of January.

3.3.6.5 Hoeing

Two hoeing were done at 30 and 60 days after sowing (DAS) to check the weeds during crop seasons of 1993-94 and 1994-95.

3.3.6.6 Harvesting

The crop was harvested manually on same date *ie* April 24, in both 1994 and 1995.

3.3.7 Meteorological observations

The observations were recorded daily as well as diurnally. Air temperature, rh and leaf wetness duration were recorded within the crop diurnally from mid-February to mid-April. Daily observations *viz.* maximum, minimum air temperature, morning and evening rh, solar radiation, sunshine duration, rainfall and foggy conditions were recorded on the

3.3.7.5 Rainfall

Rainfall was recorded in the meteorological observatory with the a manual rain gauge.

3.3.7.6 Foggy conditions

The presence of fog was recorded daily at 0730 h during the crop season on the basis of visibility.

3.3.8 Observations on disease progress

Five randomly selected areas of 1 m^2 in each plot were observed daily for disease incidence and intensity using a 0 - 9 disease scale . These data recorded in mid-March were considered to be disease data of first infection cycle. The second and final disease data were recorded in mid-April in both the crop seasons.

3.3.9 Crop characteristics

3.3.9.1 Phenological studies

3.3.9.1.1 Date of emergence

Complete emergence was recorded by counting daily, the number of emerged plants from two randomly selected sites of one metre length in each plot.

3.3.9.1.2 Date of flowering

The date of first flower appearance was recorded in the experimental plots to

identify the beginning of the reproductive stage.

3.3.9.1.3 Date of pod formation

The date of first pod appearance was recorded in each plot when the first pod was visible.

3.3.9.1.4 Date of seed setting

After pod formation, five pods from each plot were collected daily at random and the date of seed setting was recorded.

3.3.9.1.5 Date of full seed formation

Pods from each plot were picked at random to record the date of full seed formation.

3.3.9.1.6 Date of leaf drying

The date when 90 per cent of the leaves became dry was recorded.

3.3.9.1.7 Physiological maturity

The date of physiological maturity was recorded when 95 per cent of the pods became dry and seeds became hard (they could be crushed with teeth but not with nails).

meteorological observatory of the department of Agricultural Meteorology.

3.3.7.1 Air temperature and relative humidity

The maximum and minimum air temperature, dry and wet bulb temperatures were recorded using thermometers with India Meteorology Department (IMD) specifications. These thermometers were installed in a Stevenson's screen at 1.2 m height above the ground. These variables were recorded at 0730 h and 1430 h.

3.3.7.2 Leaf wetness duration, air temperature and relative humidity in the crop

Three microclimatic variables *viz.* leaf wetness, air temperature and rh were recorded by installing two portable leaf wetness recorders (Belfort Inst. Co.) within the crop canopy at half its height. One leaf wetness recorder was installed in the plot with the IW treatment and another with the N treatment.

3.3.7.3 Solar radiation

Solar radiation was measured with a tube solarimeter (Delta-T Devices, U.K.) installed at 3 m height. The measurements integrated over 42 h were recorded daily at 0830 h.

3.3.7.4 Sunshine duration

Sunshine duration was recorded with a sunshine recorder (Campbell Inst. U.K.)

3.3.9.2 Growth Characters

3.3.9.2.1 Plant population

The final number of plants in four randomly selected one meter lengths of row were recorded in each plot at harvest.

3.3.9.2.2 Plant height

The height of six plants tagged at random in each plot was recorded at fortnightly intervals. Plant height was measured from the ground surface to the tip of the topmost leaf of the tallest branch of each plant.

3.3.9.2.3 Leaf area

Leaf area was measured with a leaf area meter (LI COR 300 DA) and the leaf area index was calculated by dividing the leaf area by the ground area.

3.3.9.2.4 Dry matter partitioning

Three randomly selected plants were taken from a one meter row length of the second row in each plot. For each subsequent sampling at fortnightly intervals, the next one meter row length was considered to take 3 plants at random. Dust free above ground parts of these harvested plants were separated into leaves, stem + branches, pods and seeds and their dry weight was recorded after drying at 70°C for 48 h.

3.3.10 Pot experiments

Pot experiments were conducted during the 1994-95 crop season. Chickpea plants were raised in 6 X 14 cm polythene bags filled with a mixture of sand, silt and clay in the ratio of 30, 50, and 20 per cent respectively. In each pot, 5 seeds were sown and finally four plants were retained.

One set of 10 pots with one month old plants were inoculated with a 1 X 10^5 spore suspension of *A. rabiei*. prepared with the method described earlier. Another set of 10 pots was kept un-inoculated. These two sets were placed in the chickpea plots for one week under the field conditions. After one week the inoculated plants were removed to dry conditions with a favourable temperature. The un-inoculated plants were kept in wet and high humidity conditions for a one week duration. The first appearance of disease symptoms was recorded and one week later the disease intensity on one plant in each pot was recorded. This experiment was started in the second week of January and continued until the second week of April.

3.3.11 Statistical analysis

Analysis of variance for reaction of *A. rabiei* to different environments and its impact on various growth characters of the chickpea crop were presented in following manner:

Source of variance	Degree of Freedom	Mean Square	F ratio
Replications	R - 1	M_1	M_1/M_3
Treatments	T - 1	M_2	M_2/M_3
Error	(R - 1)*(T - 1)	M_3	
Total	R*T - 1		

3.11.1 Coefficient of correlation

The coefficients of correlation among different variables were calculated following the method suggested by Snedecor and Cochran (1967) using the formula:

$$\text{Coefficient of correlation} = \frac{\text{Sum XY}}{[(\text{Sum } X^2)(\text{Sum } Y^2)]^{0.5}}$$

Where

X = one variable
Y = another variable

3.11.2 Regression analysis

Simple regression analysis was carried out as suggested by Panse and Sukhatme (1978). The formula used to calculate the regression coefficient (b) was:

$$b = \frac{\text{Sum}\{(Y - \bar{Y})*(X - \bar{X})\}}{\text{Sum}(X-X)^2}$$

Where

X = Independent variable

Y = Dependent variable

The quantity Sum{(Y -$\overline{Y}$)*(X -$\overline{X}$)} denotes the sum of product of deviations of X and Y from the respective means. Sum(X -$\overline{X}$)^2 denotes the sum of squares of deviation of X from the mean of the independent variable.

The underlying relation between Y and X in a bivariate population was expressed in the form of a mathematical equation which presents the regression of the variate Y on the variate X. For a linear relationship, the equation takes the form:

$$Y = a + bX$$

Where

Y = Expected value of the dependent variable (Y) corresponding to any given value of the independent variable (X).

The significance of the regression coefficient (b) was estimated by calculating the t value using the formula:

$$t = \frac{b}{\text{S.E. of } b}$$

The significance of b was tested from t values at (n-2) degrees of freedom.

Multiple regression equations were fitted with the help of partial regression coefficients of disease reaction on the plant characters. The following simultaneous equations were used in the solution.

$$b_1t_{11} + b_2t_{12} + b_3t_{13} + \ldots\ldots\ldots\ldots b_pt_{1p} = t_{1y}$$

$$b_1t_{12} + b_2t_{22} + b_3t_{23} + \ldots\ldots\ldots\ldots b_pt_{2p} = t_{2y}$$

$b_1t_{1p} + b_2t_{p2} + b_3t_{p3} + \ldots\ldots\ldots\ldots b_pt_{pp} = t_{py}$

Where

p = Number of independent variables accounted for in calculation

$t_{11}\ t_{22} \ldots\ t_{pp}$ = Variances of the independent variables.

$t_{12}\ t_{13} \ldots\ t_{1p}$ = The covariance of the independent variables among themselves

$t_{1y}\ t_{2y} \ldots\ldots t_{py}$ = The covariance of the independent variables (X) with disease reaction (Y)

$b_1\ b_2\ \ldots\ldots b_p$ = Partial regression coefficient of the dependent variable (Y) on the independent variable (X) when the effects of another variable are kept constant

The standard error (S.E.) of partial regression coefficient was worked out by the formula:

$$\text{S.E. of } b_i = (S^2 * C_{ii})^{0.5}$$

Where

S^2 = Residual variance

C_{ii} = The ith diagonal vector of the inverse matrix

The significances of the partial regression coefficients were tested using t test as in case of simple regression coefficients.

Multi-regression functions with more than two variables can be expressed as dependent variables in terms of independent variable by means of a linear equation.

$Y_e = a + b_1Y_1 + b_2Y_2 + ...b_pX_p$

Where

$a = Y - (b_1X_1 + b_2X_2 + ...b_pX_p)$

Ye= Expected disease reaction

Y = Mean disease reaction of population

The efficiency of the multiple regression equation was calculated by the formula:

Coefficient of determination (R^2) = $\frac{\text{S.S. due to regression}}{\text{S.S due to disease reaction}}$

XY12...p was tested for its significance using Fisher's "r" table at (n-p-1) degrees of freedom at the 5 per cent level of significance.

The exponential functions were fitted by converting the dependent variable in natural logarithmic values and then a simple regression equation [$Y = \exp(a + bX)$] were fitted.

Second degree polynomials [$Y = a + bX + cX^2$] were fitted by taking the square of the independent variable and using it as a second variable in a multiple regression equation [$Y = a + bX_1 + cX_2$].

An asymptotic regression ($Y = a - b*e^{cx}$) function was fitted by putting maximum limit in a function and then simplifying in the terms of the logarithmic values and fitting a simple regression equation.

CHAPTER-IV

EXPERIMENTAL RESULTS

Results of the empirical study and experiments conducted in the laboratory at ICRISAT in 1994 and in the field during *rabi* seasons of 1993-94 and 1994-95 and are presented in this chapter.

4.1 EMPIRICAL RELATIONSHIPS BETWEEN METEOROLOGICAL ELEMENTS AND ASCOCHYTA BLIGHT

4.2 LABORATORY EXPERIMENTS

4.3 FIELD EXPERIMENTS

4.1 EMPIRICAL RELATIONSHIPS BETWEEN METEOROLOGICAL ELEMENTS AND ASCOCHYTA BLIGHT

4.1.1 Coefficient of Correlation

Correlation coefficients between disease index (DI) and each of the meteorological variables are presented in Table 1.

4.1.1.1 Temperature

A significant correlation was found between the disease index (DI) and minimum air temperature only in the 7th SMW. With maximum air temperature, however, there was a negative and significant correlation for each week between the 9th and 14th SMW. This shows that with an increase in maximum temperature, there will be a decrease in the disease index.

Table 1: Coefficients of correlation between meteorological elements and Ascochyta blight of chickpea using 25 years (1970-1994) data

Met. Weeks	Temperature(°C) Minimum	Maximum	Relative Humidity(%) Minimum	Maximum	Sunshine Duration(h)	Rainy Days	Total Rain(mm)
1	0.17	-0.05	0.35	0.31	-0.07	***0.51***	***0.54***
2	0.23	-0.12	0.32	***0.42***	-0.05	-0.04	0.24
3	-0.08	0.18	0.13	***0.55***	0.20	-0.27	-0.07
4	0.31	0.23	0.04	***0.39***	-0.10	0.20	0.38
5	0.23	-0.05	0.34	***0.40***	0.06	-0.21	0.09
6	0.34	-0.10	***0.56***	0.29	***-0.44***	0.04	0.24
7	*0.61*	-0.23	***0.80***	***0.49***	***-0.57***	0.04	0.28
8	0.07	-0.34	***0.58***	0.35	-0.32	0.04	0.17
9	0.31	***-0.52***	***0.76***	***0.55***	***-0.48***	***0.42***	0.35
10	-0.20	***-0.54***	***0.59***	***0.47***	-0.27	0.34	*0.48*
11	0.12	***-0.59***	***0.81***	***0.60***	***-0.42***	0.30	0.38
12	-0.20	***-0.78***	***0.56***	***0.51***	-0.19	***0.43***	***0.53***
13	-0.29	***-0.55***	***0.41***	0.23	0.12	0.23	0.23
14	-0.22	***-0.51***	***0.52***	0.30	0.05	0.06	-0.19
15	0.02	0.01	0.23	0.25	-0.08	-0.23	0.23
16	0.08	-0.16	0.30	0.27	0.04	0.00	0.50
17	-0.06	-0.24	0.22	0.29	-0.03	-0.01	0.14
Average of 9-14 SMW	**-0.15**	**-0.85**	**0.89**	**0.63**	--	--	--

Humid Thermal Ratio (HTR): **0.90**

Figures in italics are significant at 95 per cent confidence interval
Bold figures indicate the continuity of significance

4.1.1.2 Relative humidity

There was a positive and significant correlation between disease index and minimum rh for each week from 6th to 14th SMWs. Correlations were non significant from the 1st to 5th and 15th to 17th SMW. Then were positive and significant correlations between disease index and maximum rh for each week from the 2nd to 12th SMWs except for 6th and 8th SMWs. It shows that increase in rh will have a favourable effect on disease.

4.1.1.3 Sunshine duration

There was a significant and negative correlation between disease index and sunshine duration only for the 6th, 7th, 9th and 11 th SMWs.

4.1.1.4 Rainfall

Significant and positive correlation between disease index and total rainfall was observed for the 1st, 10th 12th and 16th SMWs. A significant and positive correlation was obtained between disease index and rainy days only for the 1st, 9th and 12th SMWs.

The reasonably high correlations between disease and both maximum temperature and minimum rh were consistent from the 9th to 14th SMWs, so these two variables were selected for further analysis.

The coefficient of correlation between disease index and maximum temperature (averaged from 9th to 14th SMW) was -0.85 and between disease index and minimum

Fig.5 Effect of maximum air temperature on Ascochyta blight at Ludhiana

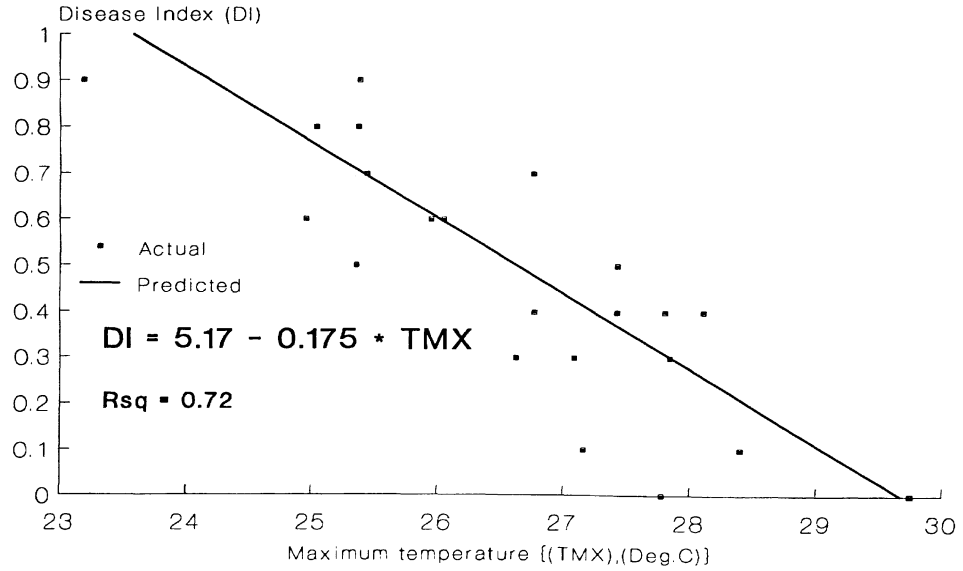


Fig.5 Effect of maximum air temperature on Ascochyta blight at Ludhiana

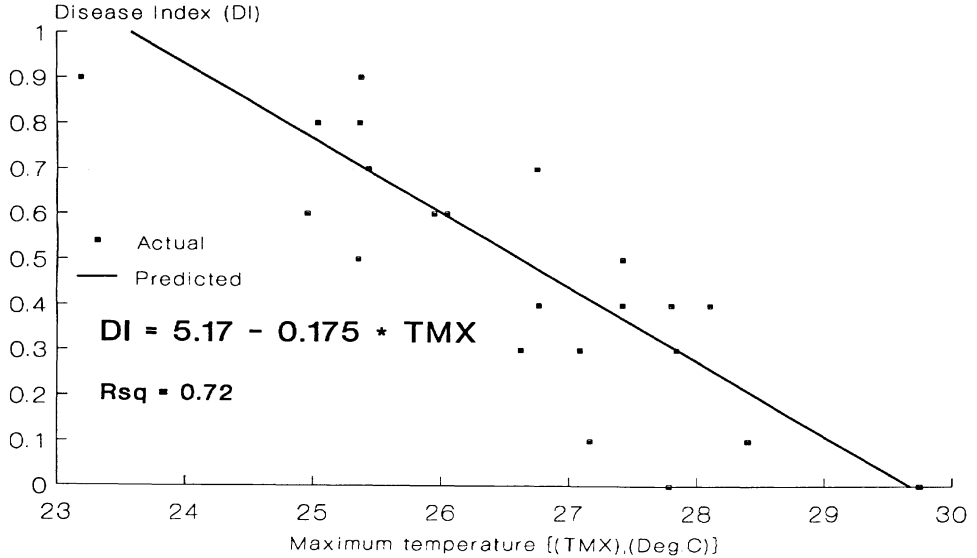


Fig.6 Effect of relative humidity on Ascochyta blight at Ludhiana

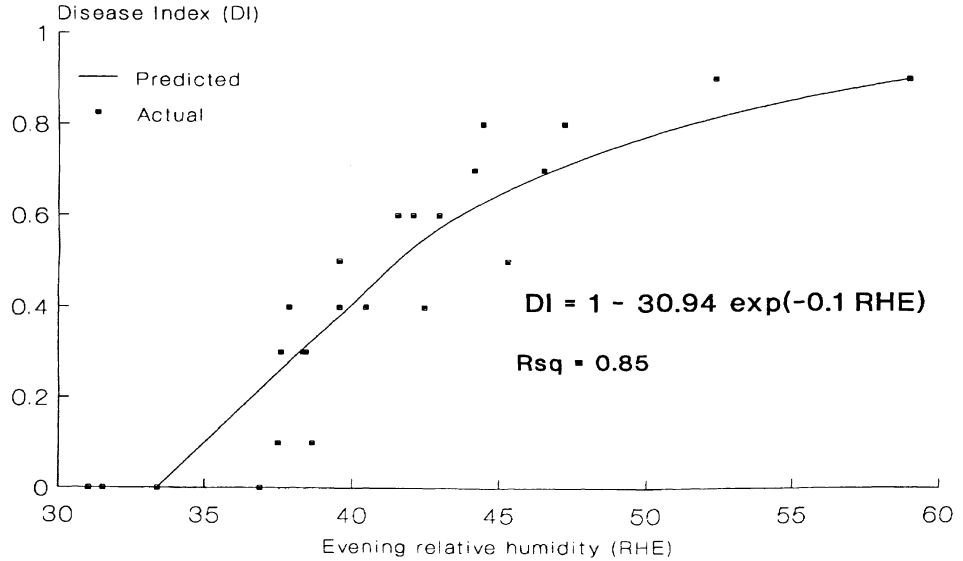
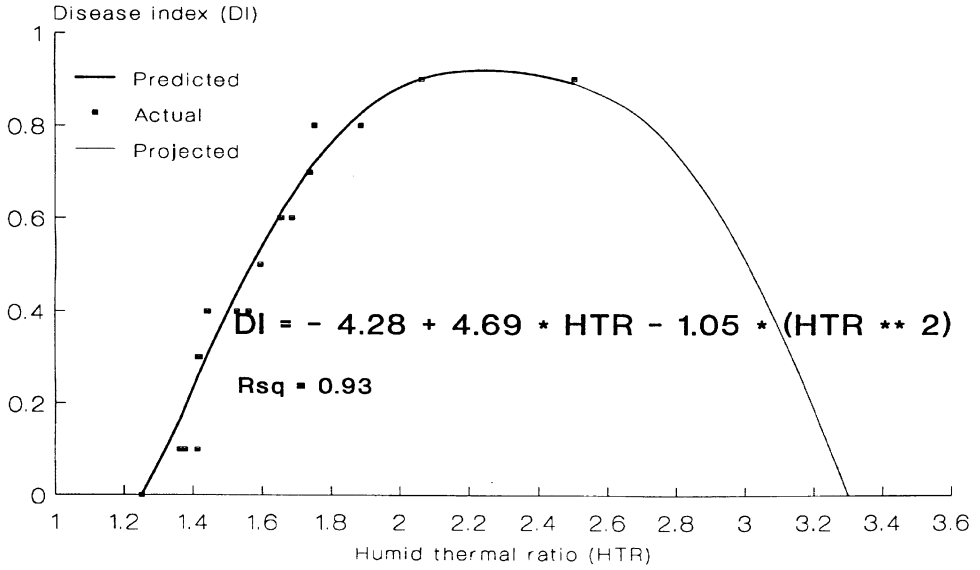


Fig.7 Combined effect of humidity and temperature on Ascochyta blight



rh (averaged from 9th to 14th SMWs) was 0.89.

Two important variables *viz* maximum temperature and minimum rh (day time) from 25th February to 8th April were identified to be most crucial for disease development. The two variables were significantly correlated with each other, so could not be combined in a bivariate regression model because the effect of multicollinearity. To overcome this problem, a ratio of the evening rh and corresponding maximum temperature was worked out to obtain a humid thermal ratio (HTR) which combines these two important variables. The coefficient of correlation between the disease and HTR was 0.90.

4.1.2 Fitting the Curve

Values of disease index (DI) were plotted against maximum temperature, minimum rh and the humid thermal ratio (Fig.5, 6, 7) and appropriate functions were selected to fit the data.

The relationship between the disease index and maximum air temperature was linear and a **straight line** *i.e.* **DI = 5.17 - 0.175*TMX** explained 72 per cent of the variation in the disease (Fig 5).

The relationship between DI and the evening rh appeared to be exponential so an **asymptotic exponential function** *i.e.* **DI = 1-30.94*exp$^{(-0.1*RHE)}$** was fitted. This explained 85 per cent of the variation in DI (Fig.6).

Similarly the relationship between DI and HTR appeared to be **quadratic** *i.e.* **DI = -4.28 + 4.69*HTR - 1.05*HTR^2** and this function explained 93 per cent of the

variation in DI (Fig.7).

The strength of the relationships between DI and each of the above mentioned meteorological variables indicated by the **coefficient of determination (R^2) were 0.72, 0.85 and 0.93 respectively**. These functions can be used to identify appropriate ranges of each of these variables which favour the disease.

The favourable range of the maximum temperature for the disease in the central plain region of Punjab was observed between 25 to 28°C. The range of evening rh favouring the disease in this region was > 40 per cent. These two variables when combined as HTR favoured the disease in the range of 1.3 to 3.3 (projected from 2.5 to 3.2) with a maxima at 2.3 (Fig. 5 to 7).

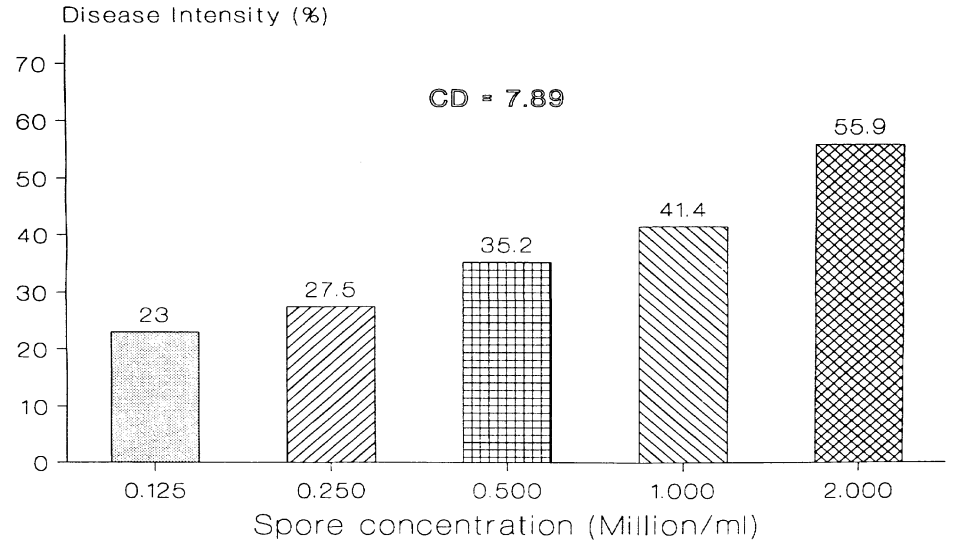
4.2 LABORATORY EXPERIMENTS

Laboratory experiments were conducted to study germination, penetration, sporulation and survival of pycnidiospores and its impact on disease progress under different environments. The host and pathogen were exposed to varied duration of leaf wetness, dryness, light, darkness and different levels of rh. A constant temperature of 20°C was maintained in all the experiments except that for high rh where the temperature remained constant at 24°C.

4.2.1 Standardization of inoculum concentration

Disease intensity increased with increase in spore concentration from 1.25×10^5

Fig.8 Effect of inoculum concentration on Ascochyta blight with 24 h wetness



CD = Critical Difference

Fig.9 Effect of leaf wetness duration on spore germination and penetration

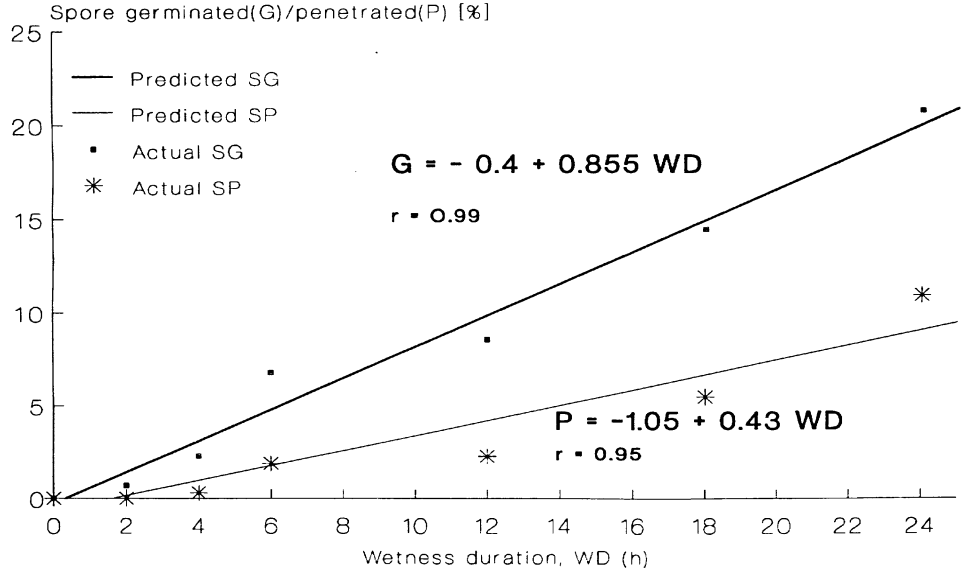
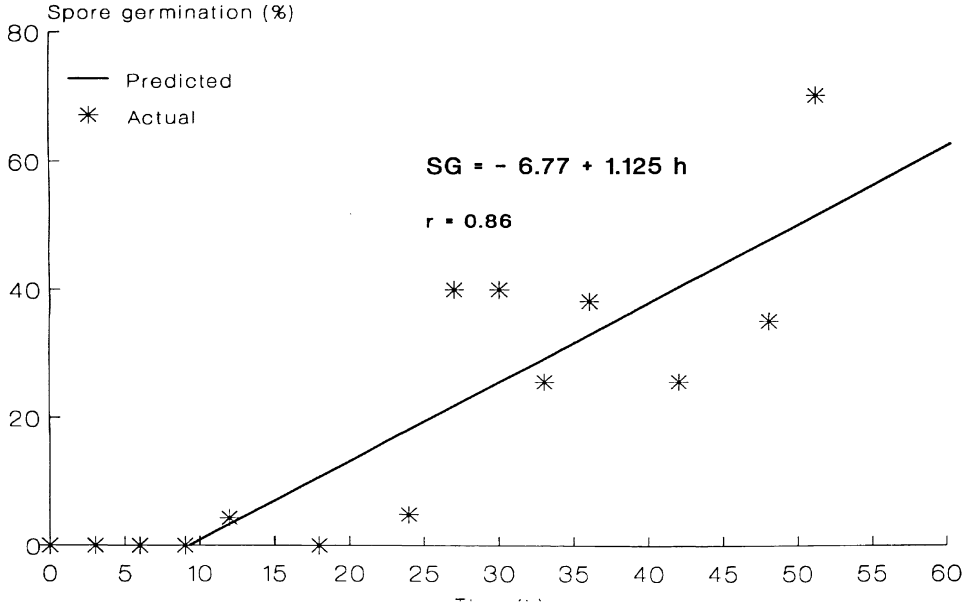


Fig.10 Spore germination with time



spores ml^{-1} to 2 X 10^6 spores ml^{-1}. Considerable increase was observed at each of the spore concentration, whereas, 0.125 X 10^6 spores concentration differed significantly from 0.5, 1.0 and 2.0 X 10^6; 0.25 X 10^6 differed significantly from 1.0 and 2.0 X 10^6 and 0.5 X 10^6 and 1.0 X 10^6 differed significantly from 2 X 10^6(Fig.8). On the basis of this study, a moderate concentration of 1 X 10^6 spores ml^{-1} was used for all the laboratory experiments.

4.2.2 Effect of leaf wetness duration

The spore germination recorded after 42 h of inoculation with different wetness periods (Table 2) was only 0.7 per cent with 2 h of wetness but it increased with increasing wetness duration. A significant and linear increase in the percentage of spore germination was observed at each leaf wetness duration from 2 to 24 h (Fig.9). Spore germination with 24 h wetness was observed 12 h after inoculation and thereafter spore germination increased linearly reaching a maximum 52 h after inoculation (Fig.10).

The effect of leaf wetness on spore penetration is shown in Table 2. Spore penetration did not take place with 2 h of wetness and it was negligible with 4 h of wetness. Thereafter a significant increase in spore penetration was observed at each wetness interval from 6 to 24 h except between 6 and 12 h of wetness. Spore penetration ranged from 1.9 with 6 h of wetness to 11.0 per cent with 24 h of wetness. Spore penetration followed similar trend to spore germination but the magnitude of penetration was about half that of germination (Fig.9).

The effect of leaf wetness duration was studied at 20°C. The results showed that

Table 2: Effect of leaf wetness duration on spore germination and penetration on live chickpea plants

Wetness Duration (h)	Spore Germination (%)	Spore Penetration (%)
2	0.7	0.0
4	2.3	0.3
6	6.8	1.9
12	8.6	2.3
18	14.5	5.5
24	20.8	11.0
CD (p=0.05)	0.69	0.66

CD is critical difference

Table 3: Effect of leaf wetness duration on Ascochyta blight disease intensity.

Exp-eriment	Wetness duration (h) 2	3	4	6	8	10	12	18	24
1	0.00	0.30	4.40	22.4	35.7	36.8	42.7	42.4	52.5
	(0.78)	(0.90)	(2.22)	(4.78)	(6.02)	(6.11)	(6.57)	(6.47)	(7.28)
2	0.00	0.00	2.20	16.6	18.8	24.3	28.0	41.1	45.1
	(0.71)	(0.80)	(1.65)	(4.14)	(4.93)	(4.98)	(5.34)	(6.45)	(6.75)
3	0.00	0.38	2.10	17.5	32.4	43.5	44.9	48.5	48.5
	(0.78)	(0.94)	(1.61)	(4.24)	(5.65)	(6.56)	(6.74)	(7.00)	(7.00)
4	0.00	0.20	5.20	13.6	26.2	26.1	32.7	29.5	36.6
	(0.85)	(0.88)	(2.38)	(3.75)	(5.17)	(5.16)	(6.76)	(5.48)	(6.09)
Mean	(0.78)	(0.88)	(1.96)	(4.22)	(5.44)	(5.70)	(6.10)	(6.35)	(6.78)

CD (p=0.05) = 0.58,

Figures in parenthesis are$(X+0.5)^{0.5}$ transformed values of per cent disease intensity

Fig.11 Effect of leaf wetness duration on Ascochyta blight

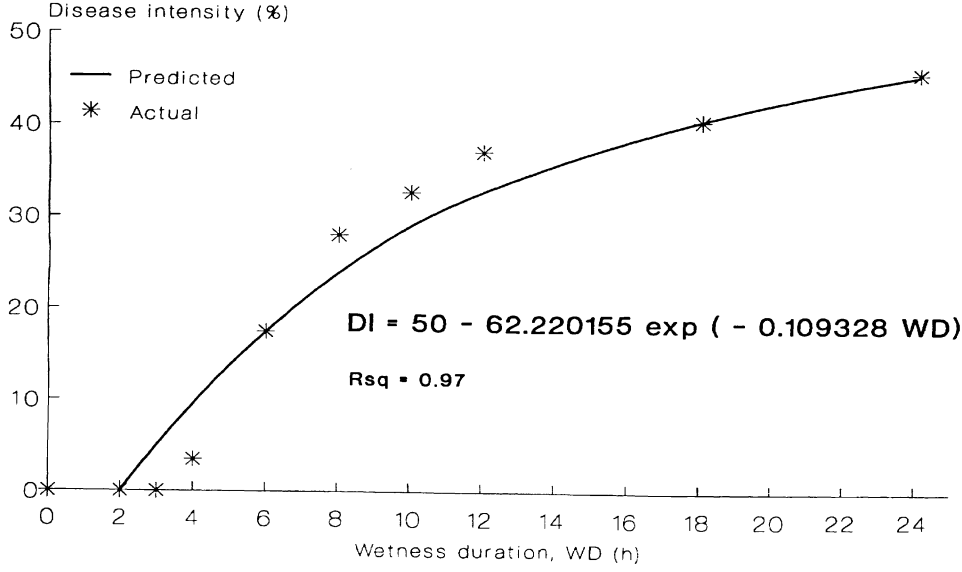
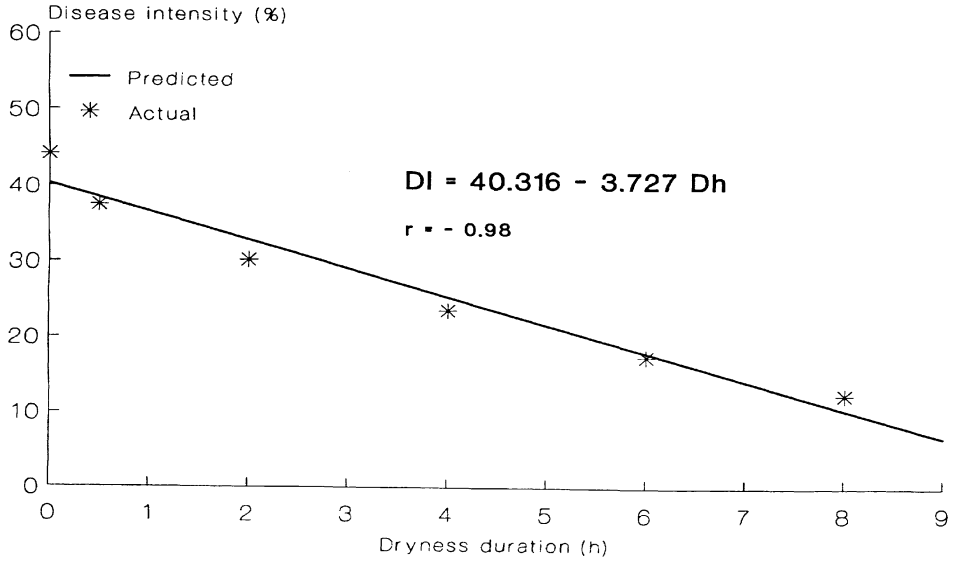


Fig.12 Effect of leaf dryness duration followed by 24 h wetness on the disease



with 2 h wetness, infection by *A. rabiei* did not take place (Fig. 11). With 3 h of wetness, infection was slight and the disease appeared in traces. The figure also indicates that with less than 4 h wetness duration the disease development was negligible. When the wetness duration increased beyond 4 h the disease increased rapidly and approached a plateau by about 18 h. The difference of mean of the disease observed under 2 and 3 h, 8 and 10 h, 10 and 12 h, 12 and 18 h and 18 and 24 h of wetness was non significant (Table 3).

4.2.3 Effect of dry period

The effect of leaf dryness immediately after inoculation with wet inoculum was studied and the results are presented in Table 4. Maximum disease intensity (44.2 %) was observed with no dryness (24 h wetness) and thereafter there was a linear decline up to 8 h dry period. The decline in the disease was significant at each dryness level from 0.5 to 8 h (Fig.12).

4.2.4 Effect of relative humidity

The effect of high relative humidity (rh) on infection was studied in humidity chambers (Butler *et al.* 1995) which where adjusted to 90, 92, 95 98, 99 and 100 per cent rh. The leaf surface were dry upto 98 per cent rh, however, some free moisture might have condensed on plant at 99 and 100 per cent rh. The results (Fig. 13) indicated that no infection occurred with less then 95 per cent rh. There was a large change between the disease intensity at 95 per cent rh (0.8 % disease) and that at 98 per cent rh (24.8 %

Table 4: Effect of different dry periods after inoculation followed by 24 hours of wetness on Ascochyta blight of chickpea

Experiment Number	Dry period (h) 0	0.5	2	4	6	8
1	45.75	33.75	28.00	24.00	19.00	13.25
2	41.75	37.00	30.25	16.75	11.50	7.25
3	42.39	42.38	34.25	28.5	22.75	16.63
4	46.88	36.75	28.25	25.38	16.50	12.88
Mean	**44.19**	**37.47**	**30.19**	**23.66**	**17.44**	**12.50**

CD (p=0.05) = **4.23**

Fig.13 Effect of high humidity on Ascochyta blight

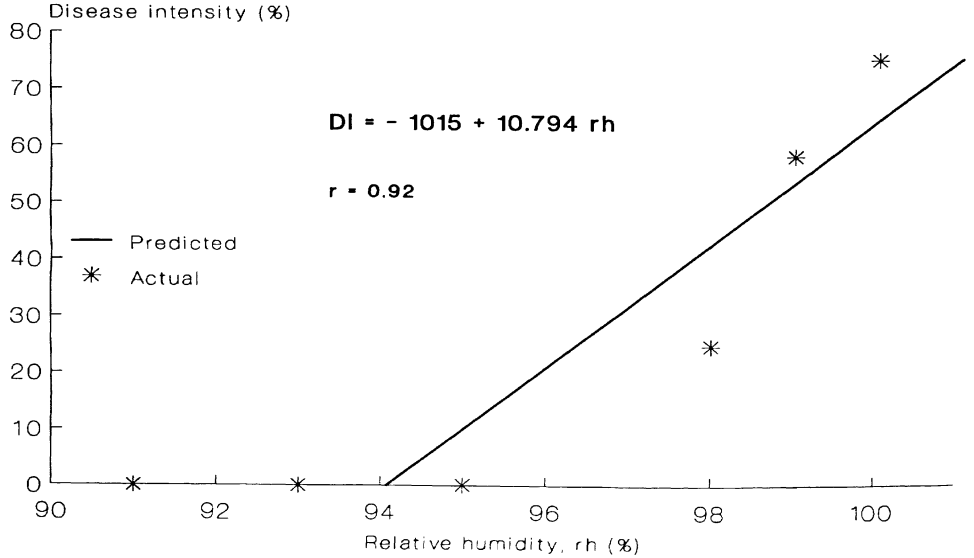
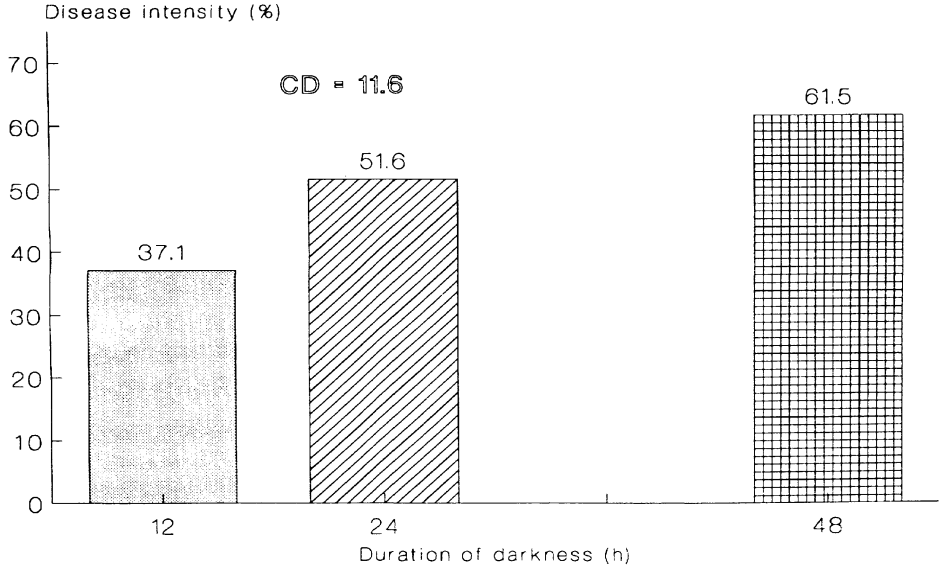


Fig.14 Effect of light and darkness on Ascochyta blight



disease). Between 98 and 100 per cent rh, there was a steep linear increase in the disease.

4.2.5 Effect of light and dark conditions

The effect of light and dark conditions indicated that with an increase in darkness, there was increase in the disease intensity (Fig. 14). With a 12 h photoperiod, there was 37.1 per cent disease which differed significantly from the other treatments. With 24 h darkness followed by 12 h photoperiod the disease intensity was 51.6 per cent which was not significantly different to that with 48 h darkness, where 61.5 per cent disease intensity was observed.

4.2.6 Effect of moisture and darkness on spore production

4.2.6.1 Density of pycnidia

There were considerably more pycnidia with wetness than all other treatments. The wetness treatment showed a steep increase in pycnidial density for the first 4 days, after which there was little increase in pycnidial density indicating a non-linear trend [Py = 1200 - 1636.146 exp(-0.5468*WD), r = 0.98] trend (Fig.15). A slight increase in pycnidial density was observed up to seven days (from the beginning of treatments) in the other treatments *viz*. 95 per cent rh and darkness, 95 per cent rh and 90 per cent rh. There were no significant differences between these treatments. The average number of pycnidia per leaf with the same treatments showed a similar trend (Table 5).

Table 5: Effects of wetness, high relative humidity and darkness on sporulation of *A. rabiei* 7 days after application of treatments

Treatments	Number of Pycnidia/Leaf			Number of spores/Leaf (10^4)		
	Exp1	Exp2	Average	Exp1	Exp2	Average
Wetness	1115	1342	1229	5618	6074	5846
> 95 % RH + darkness	665	561	613	1004	1520	1262
> 95 % RH	602	436	519	894	942	918
90 per cent RH	544	475	510	910	562	736

Fig.15 Effects of humidity, darkness and wetness on production on pycnidia

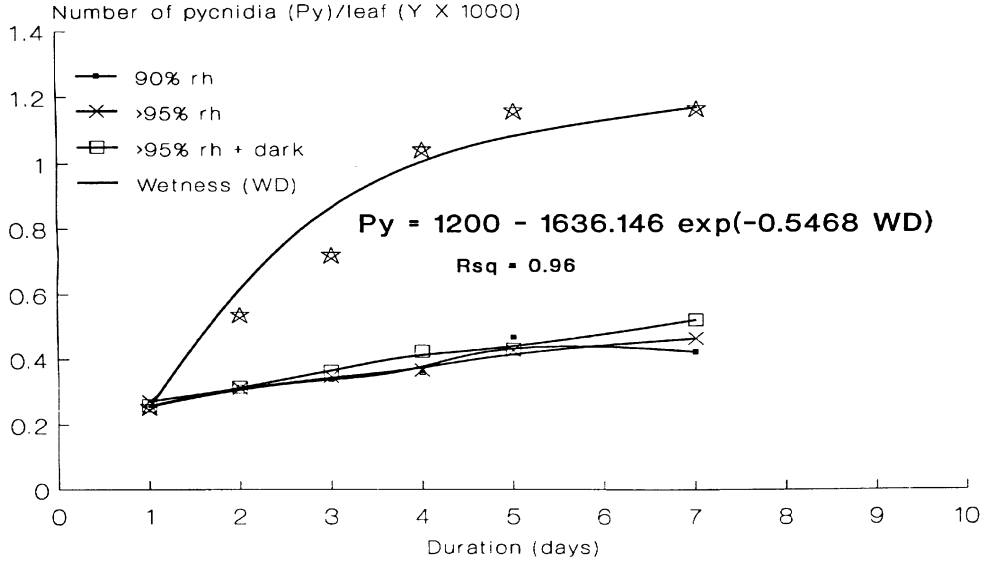
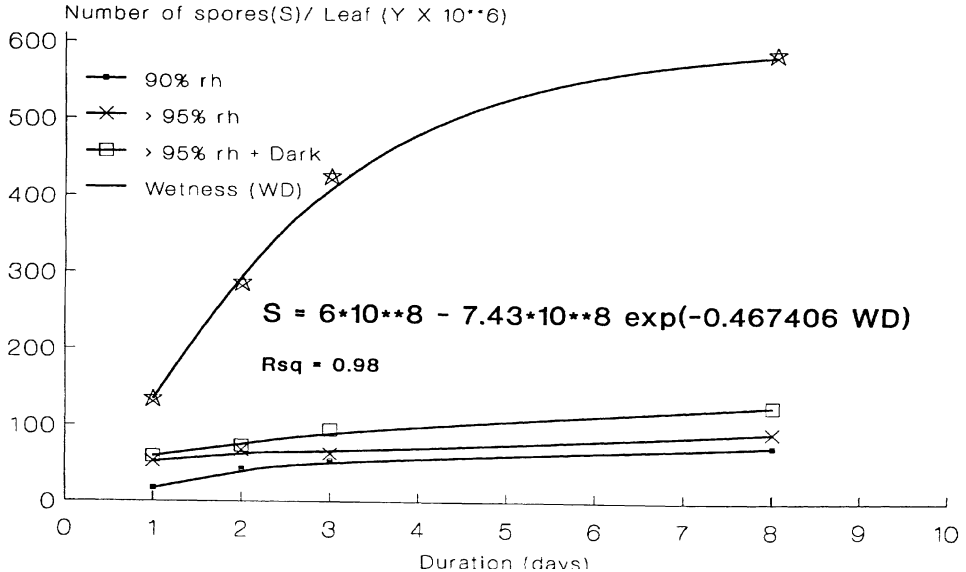


Fig.16 Effects of humidity, darkness and wetness on production of pycnidiospores



4.2.6.1 Density of pycnidiospores

The results on the effect of different treatments on the number of pycnidiospores per leaf are presented in Figure 16. With wetness there was a steep increase in the number of pycnidiospores for the first three days of the treatment. Subsequently up to eight days, the rate of increase was slow. This indicated a non-linear increase [$S = 6*10^8 - 7.43*10^8 \exp(-0.467406*WD)$, $r = 0.99$]. With the other treatments there was little or no increase in the number of pycnidiospores with time. However, there were slightly more pycnidiospores with >95% rh + darkness than with 95 per cent rh and less with 90 per cent rh.

Similar trends were observed for the average number of pycnidiospores per leaf with these treatments (Table 5).

4.3 FIELD EXPERIMENTS

4.3.1 Disease progress under different treatments

In 1993-94, the disease was observed at 140 DAS for the first infection cycle and at 160 DAS for the second infection cycle (Table 6).

In 1993-94, no disease was observed at the time of the first infection cycle without inoculation *viz.* NW, CW, N and C. A significant and high infection in the first cycle was observed with inoculation *viz.* I and IW, where disease intensities were 8.0 and 30. 2 per cent respectively.

In 1994-95, the disease was monitored at 137 DAS for the first cycle of infection and at 160 DAS for the second cycle of infection (Table 6).

Table 6: Ascochyta blight intensity after first and second infection cycles

Treatment	First infection cycle(%)		Second infection cycle(%)	
	1993-94	1994-95	1993-94	1994-95
IW	30.2	16.9	81.75	89.0
	(33.01)	*(24.03)*	*(64.83)*	*(70.72)*
NW	0.00	0.75	1.15	7.50
	(1.28)	*(4.90)*	*(5.93)*	*(15.85)*
CW	0.00	0.50	0.48	4.30
	(1.28)	*(4.05)*	*(3.25)*	*(12.01)*
I	8.50	0.97	14.2	5.05
	(16.88)	*(4.50)*	*(21.97)*	*(11.53)*
N	0.00	0.46	0.88	6.55
	(1.28)	*(3.65)*	*(4.11)*	*(15.53)*
C	0.00	0.00	0.34	1.65
	(1.28)	*(1.28)*	*(2.07)*	*(7.21)*
CD (p= 0.05)	*(3.55)*	*(3.30)*	*(3.27)*	*(5.04)*

IW = Inoculated plots with additional wetness
NW = Naturally infected plots with additional wetness
CW = Chemically controlled plots with additional wetness
I = Inoculated plots under natural conditions
N = naturally infected plots under natural conditions
C = Chemically controlled plots under natural conditions

Figures in italics are ArcSine transformed values of per cent disease intensity

Table 6: Ascochyta blight intensity after first and second infection cycles

Treatment	First infection cycle(%)		Second infection cycle(%)	
	1993-94	1994-95	1993-94	1994-95
IW	30.2	16.9	81.75	89.0
	(33.01)	*(24.03)*	*(64.83)*	*(70.72)*
NW	0.00	0.75	1.15	7.50
	(1.28)	*(4.90)*	*(5.93)*	*(15.85)*
CW	0.00	0.50	0.48	4.30
	(1.28)	*(4.05)*	*(3.25)*	*(12.01)*
I	8.50	0.97	14.2	5.05
	(16.88)	*(4.50)*	*(21.97)*	*(11.53)*
N	0.00	0.46	0.88	6.55
	(1.28)	*(3.65)*	*(4.11)*	*(15.53)*
C	0.00	0.00	0.34	1.65
	(1.28)	*(1.28)*	*(2.07)*	*(7.21)*
CD (p= 0.05)	*(3.55)*	*(3.30)*	*(3.27)*	*(5.04)*

IW = Inoculated plots with additional wetness
NW = Naturally infected plots with additional wetness
CW = Chemically controlled plots with additional wetness
I = Inoculated plots under natural conditions
N = naturally infected plots under natural conditions
C = Chemically controlled plots under natural conditions

Figures in italics are ArcSine transformed values of per cent disease intensity

In 1994-95, the disease appeared in traces in all treatments except C and IW in the first infection cycle. With IW, there was significantly more disease (16.9 %) than the other treatments and there was no disease in treatment C.

In 1993-94, the disease appeared in traces without inoculation (treatments NW, CW, N and C) in the second infection cycle. Significantly more disease of 14.2 and 81.8 per cent was observed with the inoculated treatments I and IW respectively.

In 1994-95, there was significantly more (89 %) disease in the second infection cycle with treatment IW, than all the remaining treatments. In treatment C the disease appeared in traces.

A comparison of disease intensities, both at the first infection cycle and the second infection cycle during the years 1993-94, and 1994-95 showed that in the first year the disease in the first cycle for IW and I remained significantly higher than the second year. The final observations of the disease during the second year of the experiment showed high disease intensity in all treatments except C.

4.3.2 Disease progress under variable wetness durations

The disease intensity at the first and second infection cycles with different duration of wetness is presented in Table 7.

As the period of wetness increased from 0 to 7 days, there was a clear increase in disease for both the first and second infection cycles. Five percent disease appeared with two days of wetness and with 3 to 5 days an increase (8 to 13 %) in disease for

Table 7: Ascochyta blight intensity at the primary and secondary infection cycle under varied periods of wetness in field

Spell-1 Wetness period (day)	First cycle of infection*(%)	Spell-2 Wetness period (day)	Second cycle of infection*(%)
0	0.0	0	4.0
1	5.5	1	26.0
2	5.5	2	33.0
3	12.0	3	42.0
4	13.0	4	48.0
5	13.0	5	57.0
7	20.0	7	86.0

*Average of five points in a plot

primary infection was observed. With seven days duration of leaf wetness, maximum disease (20 %) was observed.

For the second infection cycle, the disease was less than 5% under natural conditions, while, leaf wetness for one day increased the disease to 26 per cent. Afterward there was a consistent increase in the disease with the number of days of wetness up to 6 days, and with 7 days of wetness it increased to 86 per cent.

4.3.3 Disease favouring period under field conditions

In a potted plant experiment, chickpea plants were raised in polythene bags and 13 different sets of 10 plants each at the age of one month were inoculated and exposed to natural weather conditions for one week each, beginning from 12.01.1995 to 17.04.1995. The incubation period (Table 8) showed a continuous decrease from 12 days to 4 days from the second week of January (12/1/1995) to the second week of April (10/4/1995).

The coefficients of correlation between disease intensity and maximum, minimum and mean temperatures indicated that these temperatures at Ludhiana are inversely related to disease during the chickpea growing season. The maximum temperature during 24 h immediately after inoculation gave the highest coefficient of correlation followed by subsequent 48 h and 168 h. These were -0.70, -0.65 and -0.60 respectively. The effect of minimum and mean temperatures on disease, however, remained non-significant (Table 9).

The coefficients of correlation between disease and maximum, minimum and mean

Table 8: Disease intensity and incubation period of Ascochyta blight of chickpea plants exposed to different weather conditions in the field during crop season (1995)

Exposure period			Incubation Period (Days)	Disease* Intensity (%)
From	To	Days		
12/01	19/01	7	12	10.5
20/01	27/01	7	11	10.5
28/01	06/02	9	11	5.5
06/02	13/02	7	12	3.0
13/02	20/02	7	7	16.0
20/02	27/02	7	9	8.0
27/02	06/03	7	8	3.0
06/03	13/03	7	7	3.0
13/03	20/03	7	7	5.5
20/03	27/03	7	5	5.5
27/03	03/04	7	5	7.0
03/04	10/04	7	4	1.0
10/04	17/04	7	NIL	0.0

*Disease intensity was recorded three weeks after inoculation

Table 9: Coefficients of correlation between meteorological variables (relative humidity and temperature) and Ascochyta blight under field conditions

Duration(h) of weather considered	Average Temperature(°C)			Average Relative humidity(%)		
	Maximum	Minimum	Mean	Maximum	Minimum	Mean
Correlation coefficient with disease intensity						
24	**-0.70**	-0.20	-0.50	**0.68**	**0.84**	**0.86**
48	**-0.65**	-0.27	-0.50	**0.61**	**0.84**	**0.85**
168	**-0.60**	-0.37	-0.52	**0.70**	**0.74**	**0.76**
Correlation coefficient with incubation period						
24	**-0.86**	**-0.89**	**-0.90**	**0.82**	0.33	0.52
48	**-0.85**	**-0.86**	**-0.89**	**0.86**	0.36	**0.61**
168	**-0.9**	**-0.79**	**-0.87**	**0.87**	**0.75**	**0.83**

Bold figures are significant at 5% levels of significance

rh indicated that rh at Ludhiana is strongly related to disease during the chickpea growing season. The maximum rh during the first 24 h, 48 h and 168 h after inoculation gave coefficients of correlation of 0.68, 0.61 and 0.70 respectively. The effect of minimum rh was greater than the maximum rh. The coefficients of correlation between the disease and minimum rh in the first 24, 48 and 168 h after inoculation were 0.84, 0.84 and 0.74 respectively. The mean rh gave coefficients of correlation of 0.86, 0.85 and 0.76 respectively (Table 9).

The coefficients of correlation between the incubation period and maximum, minimum and mean temperatures showed an inverse relationship. The maximum temperature in the first 24 h, 48 h and 168 h after inoculation gave coefficients of correlation of -0.86, -0.85 and -0.90 respectively. The minimum temperature during the first 24 h, 48 h and 168 h gave the coefficients of correlation of -0.89, -0.86 and -0.79 respectively. The mean temperature during the first 24 h, 48 h and 168 h gave coefficients of correlation of -0.90, -0.89 and -0.87 respectively (Table 9). Thus it showed that an increase in air temperature from January to April has shortened the incubation period considerably.

The coefficients of correlation between incubation period of *A. rabiei* and maximum, minimum and mean rh indicated a strong relationship between rh. The maximum rh in the first 24 h, 48 h and 168 h after inoculation gave significantly high coefficients of correlation of 0.82, 0.86 and 0.87 respectively. The effect of minimum rh was less than the maximum rh. The coefficients of correlation were non-significant between the incubation period and the minimum rh considered for the first 24 and 48 h

Fig.17 Phenological stages of chickpea in 1993-94

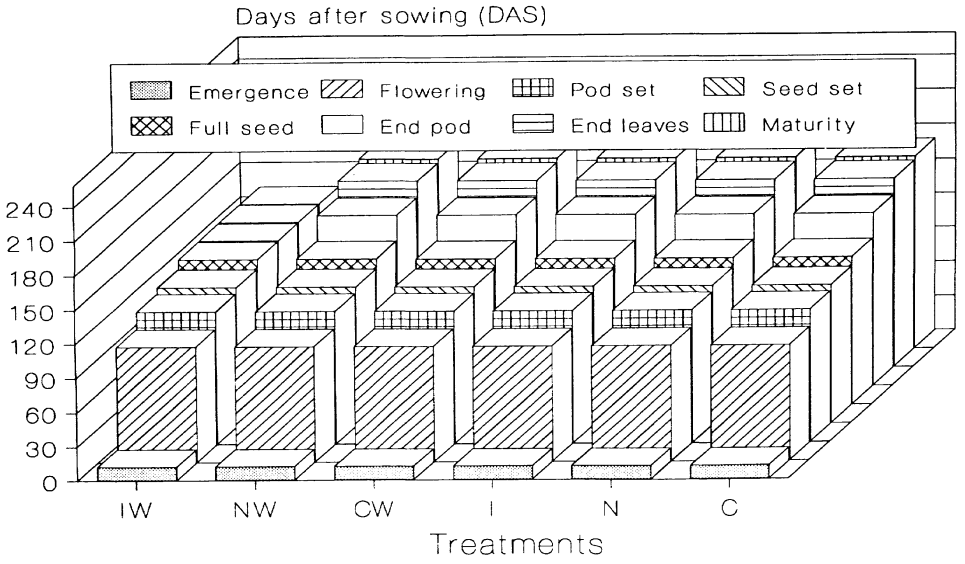
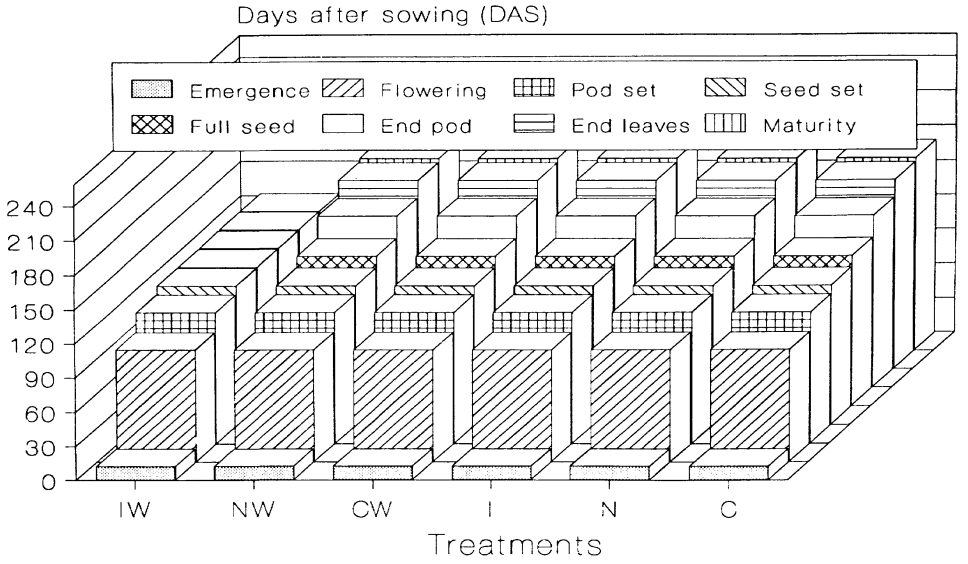


Fig.18 Phenological stages of chickpea in 1994-95



respectively after inoculation respectively but it was significantly high with rh considered for next 168 h. Mean rh gave the coefficients of correlation to the extent of 0.52 (non significant), 0.61 and 0.83 respectively with incubation period of *A. rabiei*.

4.4 CROP PHENOLOGY

Phenological stages of the chickpea crop *viz.* emergence, flowering, pod setting, seed setting, full seed formation, end of pod formation, end of the leaves and maturity stage of the crop in 1993-94 were recorded at 12, 101, 116, 121, 129, 151, 165 and 168 days after sowing (DAS) respectively (Fig.17). In 1994-95, these stages were recorded at 12, 98, 115, 122, 131, 150, 165 and 168 DAS respectively (Fig. 18), to find out the effect of the disease on the phenology of the crop.

Phenological stages of the crop progressed normally during both the years under all the treatments *viz.* IW, NW, CW, I,N and C. up to the observation of first full seed formation *ie.* 129 DAS in 1993-94 and 131 DAS in 1994-95. The crop with IW treatment did not show further progress in the developmental stages, plant growth stagnated and the developed plant parts started senescence. In rest of the treatments, the crop development was normal.

4.5 CROP CHARACTERISTICS

4.5.1 Plant population

During both years the plant populations were similar in all the treatments. However, the plant population in 1993-94 was considerably more (247139 to 294137

Fig.19 Plant height in 1993-94

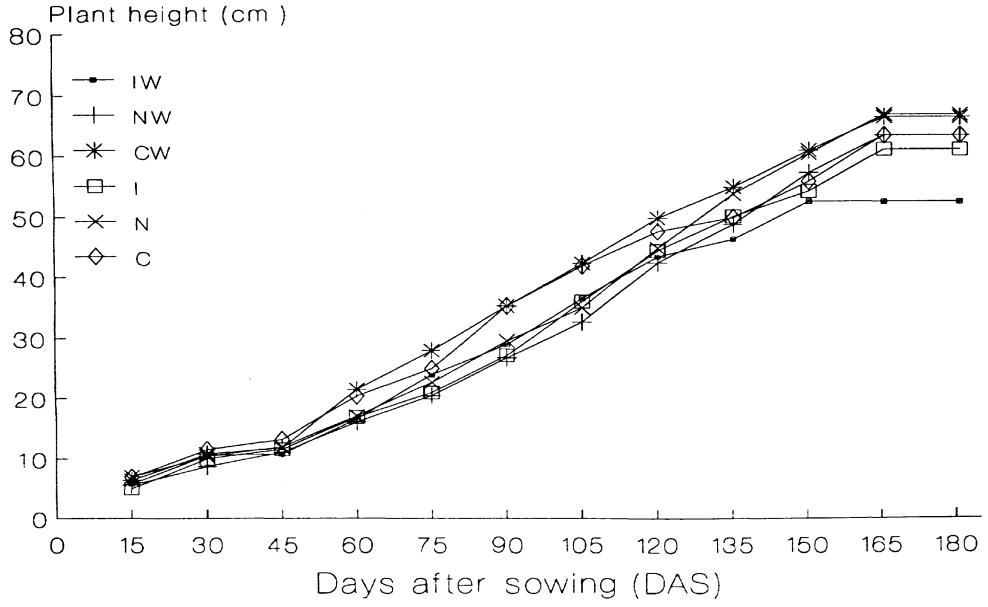
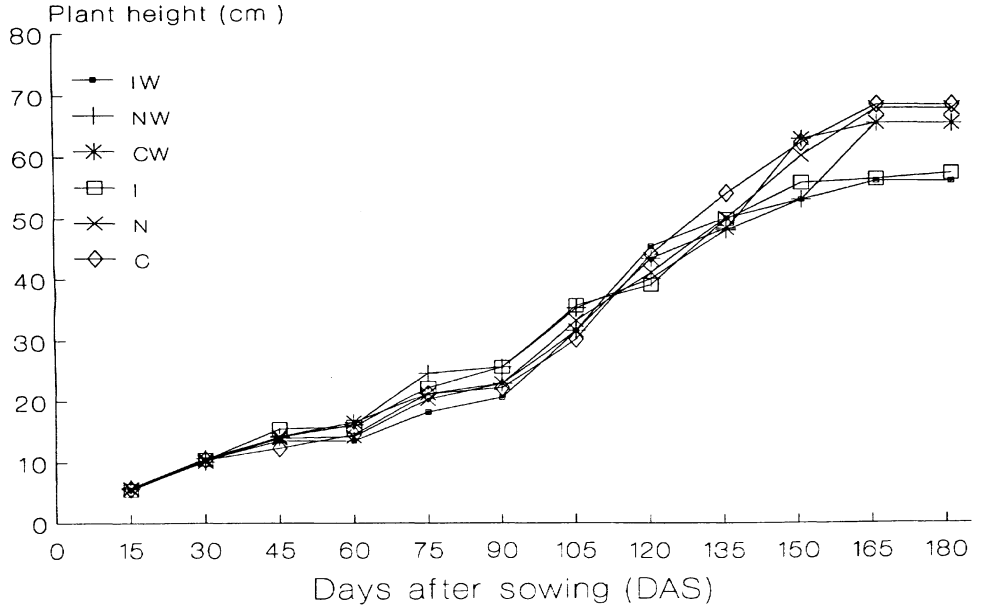


Fig.20 Plant height in 1994-95



plants/ha) than in 1994-95 (157484 to 193314 plants/ha) as indicated in Table 10. The plant population was less during second year due to an attack of wilt disease.

4.5.2 Plant height

Plant height was monitored at fortnightly intervals in both years to assess the comparative progress of chickpea under various treatments (Fig.19, 20).

In 1993-94, up to 45 DAS the plants showed a very slow rate of growth. From 45 to 165 DAS a linear trend of growth was observed in all the treatments except for IW, where the increase in plant height continued only up to 150 DAS (Fig.19). The final plant height was similar in all treatments in 1993-94 (Table 10), except for reduced blight in IW.

In 1994-95, a linear increase in plant height was observed in all the treatments up to 45 DAS in a slow rate. From 45 to 60 DAS, the increase was slightly fast and thereafter, the height was picked up at faster rate up to 120 DAS. In IW and I treatments crop showed a decline in increase of height, whereas in other treatments, the increase was observed up 165 DAS (Fig.20).

In the year 1994-95, the plant height was similar in all un-inoculated treatments (Table 10) and the height was less in the IW and I treatments.

4.6.1 Leaf area index

In 1993-94 the leaf area index (LAI) was recorded at fortnightly intervals. It increased gradually up to 120 DAS and rapidly from 120 days to 150 days. The

Table 10: Final plant population, plant height and leaf area index in different treatments

.

Plant character	IW	NW	CW	I	N	C	CD
			1993-94				
Plant population (ha^{-1})	259144	274472	263307	286638	281638	294133	NS
Plant height (cm)	52.5	63.3	66.3	61.0	66.7	63.3	NS
LAI (Maximum)	2.1	2.3	2.4	2.3	2.4	2.5	NS
			1994-95				
Plant population (ha^{-1})	178333	156667	173333	183333	183333	193333	NS
Plant height (cm)	56	65.3	65.7	57.3	67.7	68.3	NS
LAI (Maximum)	1.7	2.3	2.3	2.4	2.0	2.1	NS

IW = Inoculated plots with additional wetness
NW = Naturally infected plots with additional wetness
CW = Chemically controlled plots with additional wetness
I = Inoculated plots under natural conditions
N = Naturally infected plots under natural conditions C = Chemically controlled plots under natural conditions

Fig.21 Leaf area index in 1993-94

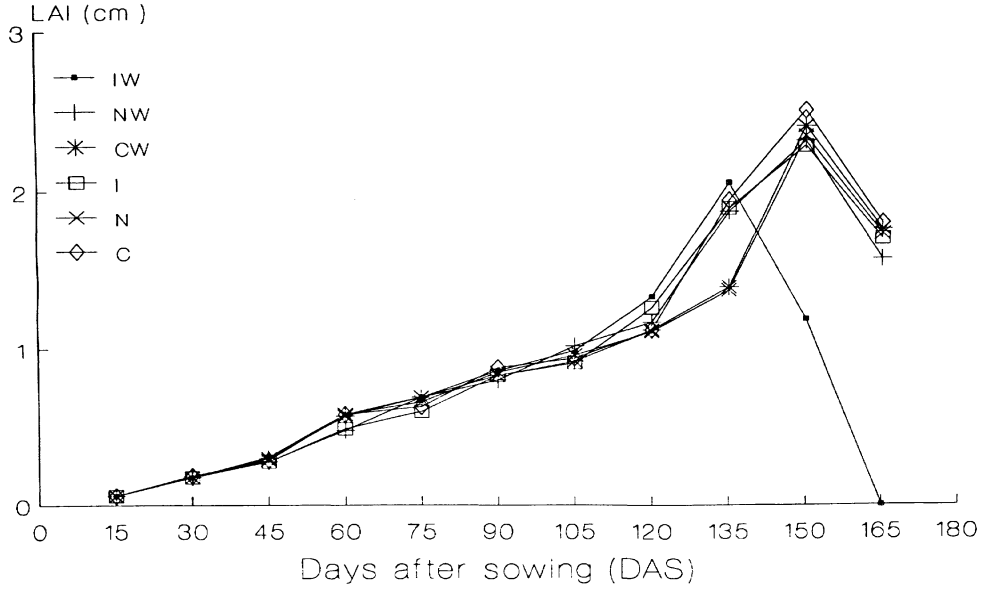


Fig.22 Leaf area index in 1994-95

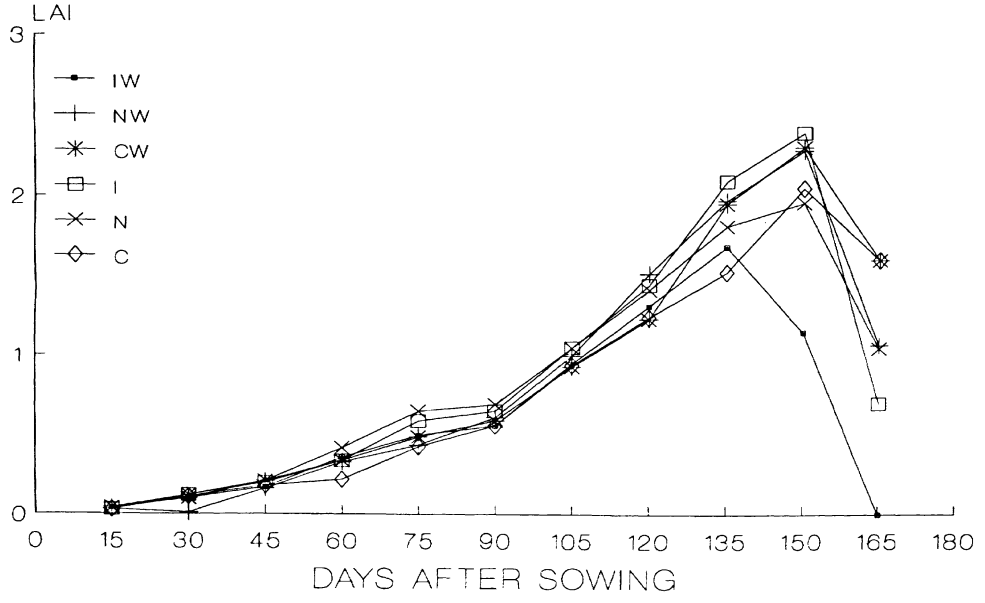


Fig.23 Leaf dry matter in 1993-94

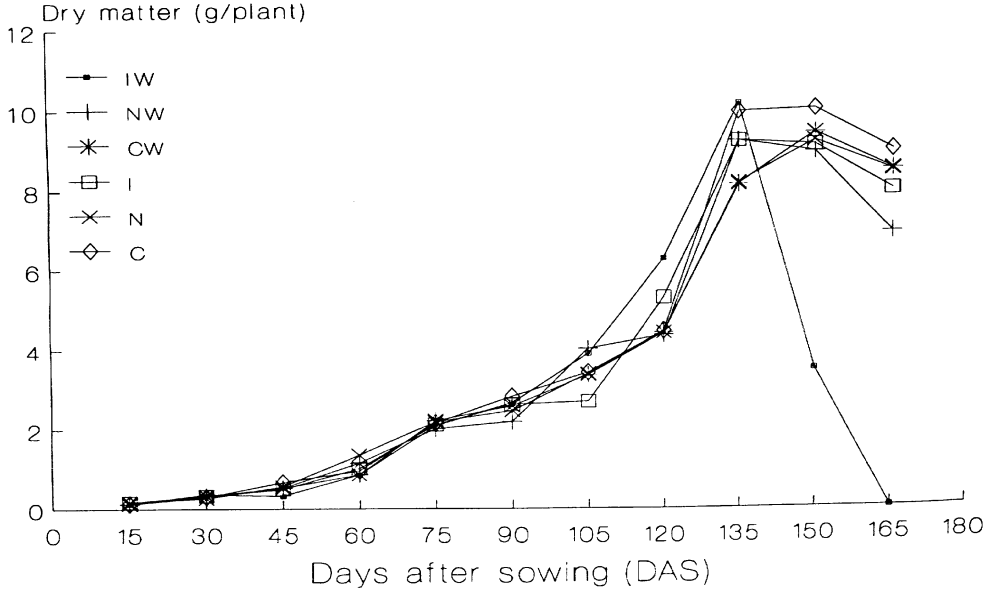


Fig.24 Leaf dry matter in 1994-95

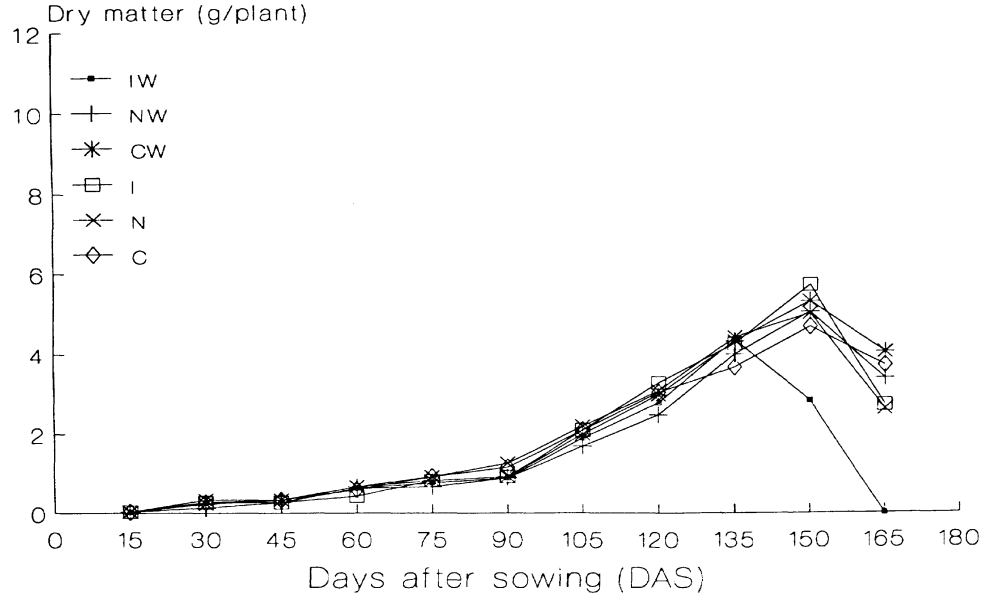


Fig.25 Stem dry matter in 1993-94

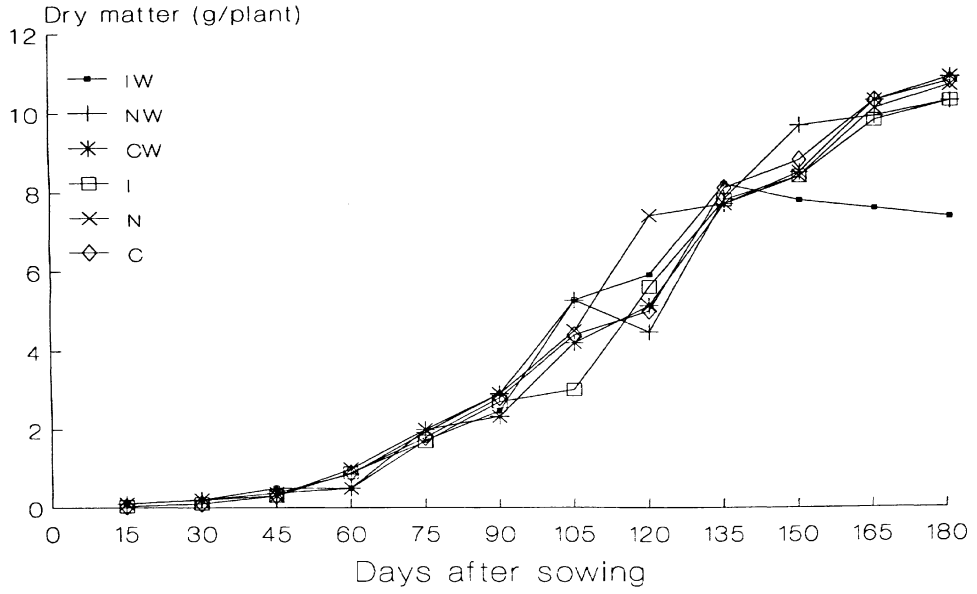


Fig.26 Stem dry matter in 1994-95

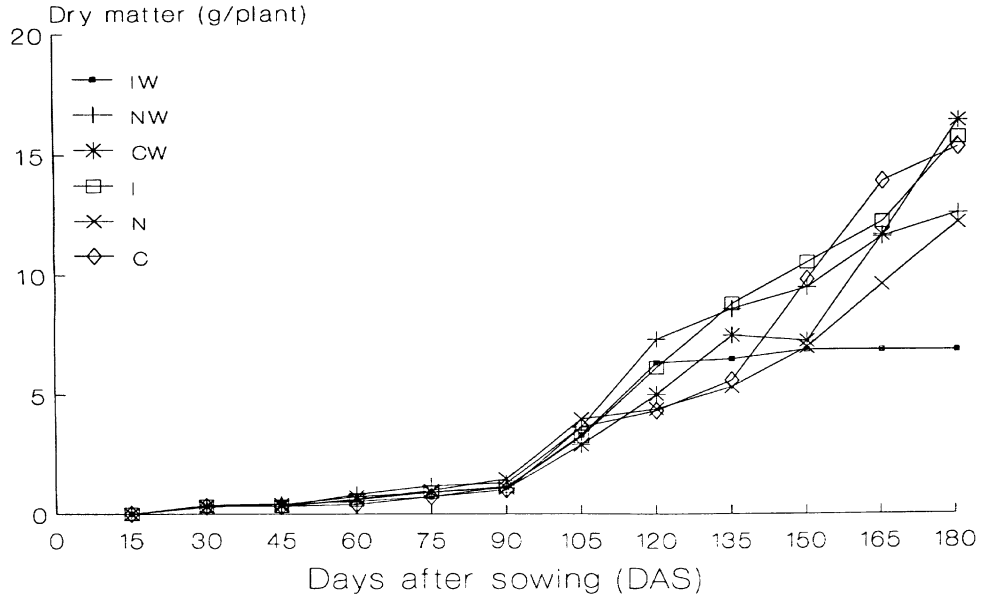


Fig.27 Pod dry matter in 1993-94

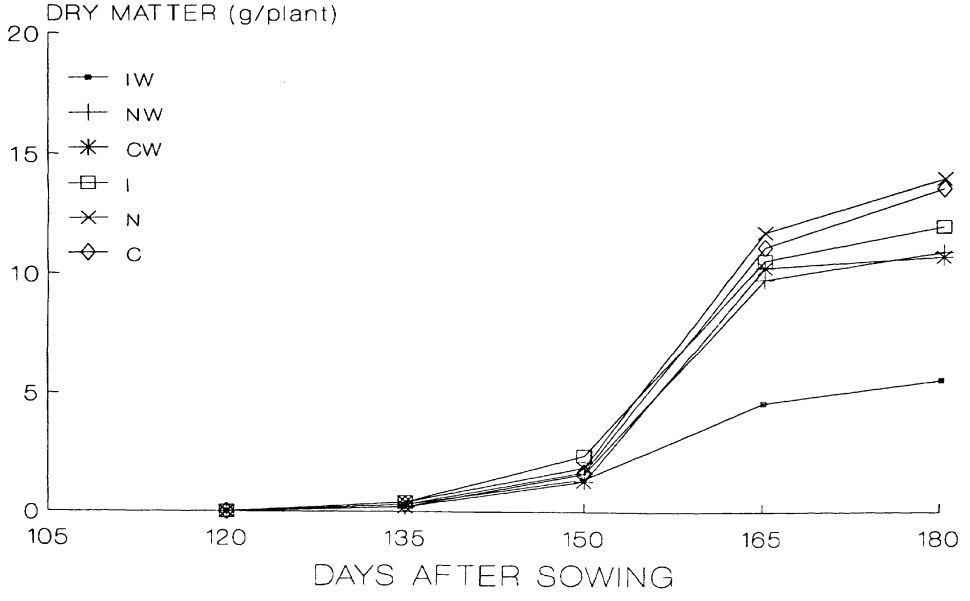


Fig.28 Pod dry matter in 1994-95

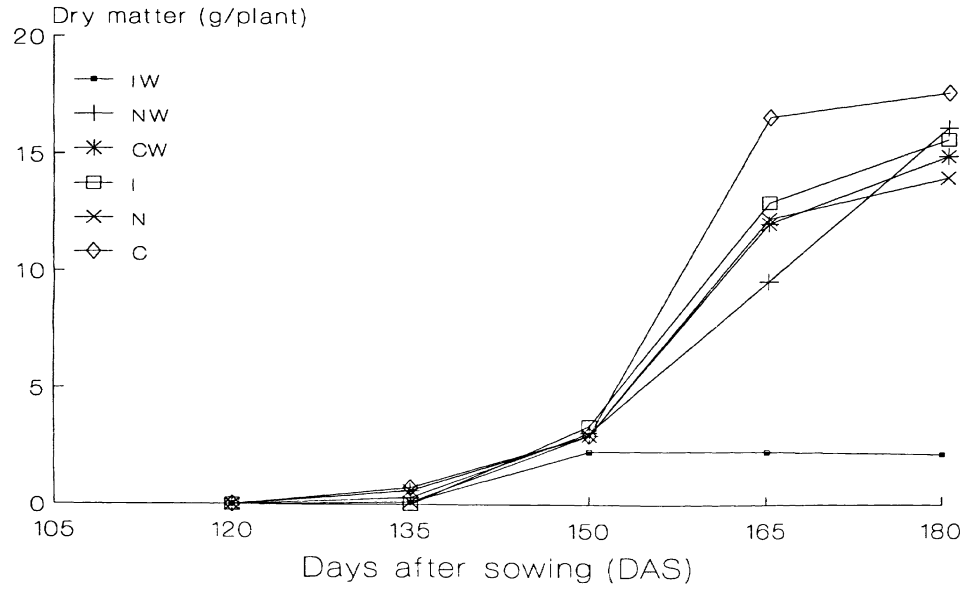


Fig.29 Seed dry matter in 1993-94

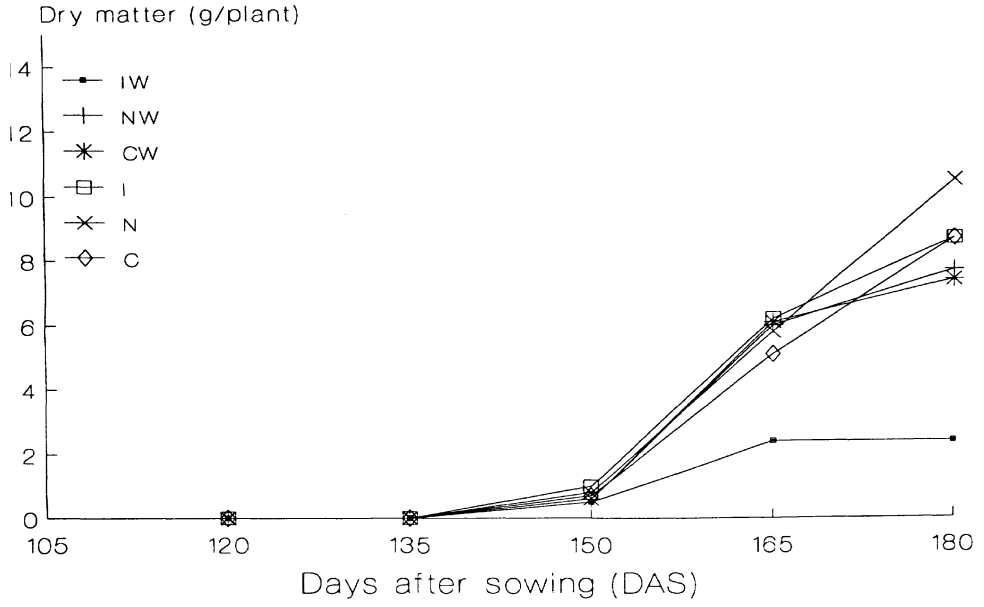
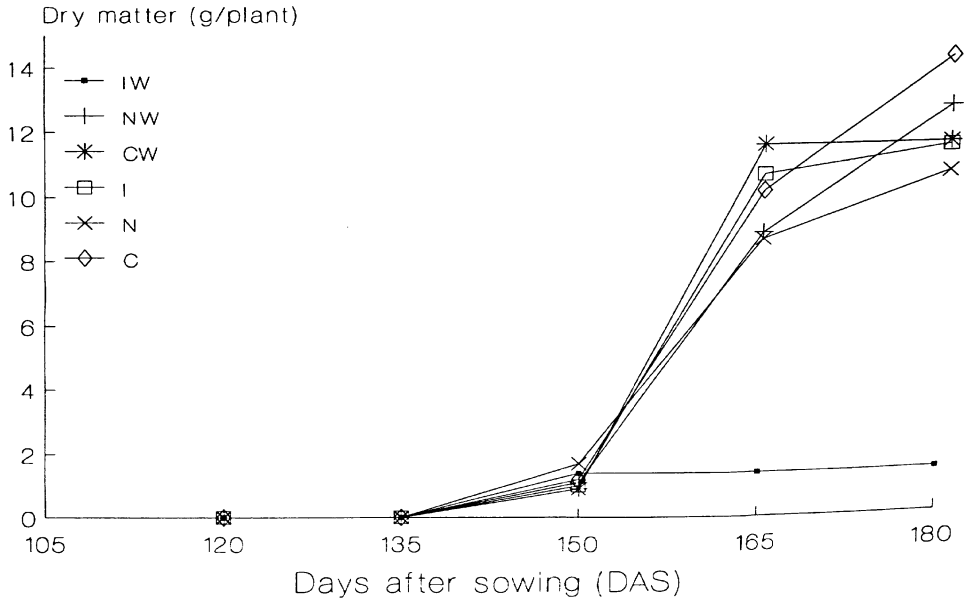


Fig.30 Seed dry matter in 1994-95



maximum LAI was observed at 150 DAS in all the treatments except IW, where the crop attained highest LAI at 135 DAS and declined steeply. In the other treatments the LAI declined from 150 DAS onwards as the leaves dried (Fig. 21).

A similar trend in LAI was observed in the year 1994-95, where it showed a gradual increase up to 90 DAS in all treatments, and a sharp increase up to 150 DAS in all treatments except IW where the increase was continued only up to 135 DAS. Later on there was a sharp decline in LAI in all the treatments up to 165 DAS. The maximum LAI was observed in I treatment followed by CW, NW, C, N and IW (Fig.22)

The maximum LAI in both year was similar in all the treatments (Table 10).

4.5.3 Leaf dry matter (g/plant)

In 1993-94, leaf dry matter (LDM) was monitored at fortnightly intervals and is presented in Figure 23.

There was a gradual increase in LDM up to 60 DAS in all the treatments. Later on a considerable increase was observed up to 120 DAS which steepened from 120 to 135 DAS. From 135 to 165 DAS, the LDM in all the treatments declined. The decline was gradual in all except IW treatment, where it was steep.

In 1994-95, a slow increase in LDM was observed up to 90 DAS in all the treatments followed by a gradual increase up to 150 DAS in all except IW, where the increase continued only up to 135 DAS. Later on a decline was observed in all treatments up to 165 DAS (Fig.24). The LDM in 1994-95 was less than that in 1993-94 for all the

treatments. In both years, there were no significant differences between the treatments (Table 11).

4.5.3 Stem and branches dry matter

The stem and branches dry matter (SBDM), recorded at fortnightly intervals in 1993-94, increased gradually up to 90 DAS in all the treatments except I, where a slow rate of growth was observed up to 105 DAS. Subsequently the SBDM increased rapidly up to 180 DAS in all the treatments except IW, which reached a plateau at 150 DAS (Fig.25).

The SBDM recorded at fortnightly intervals in 1994-95 increased very slowly up to 90 DAS. Thereafter the rate of growth was rapid except for IW, where the SBDM did not increase after 120 DAS (Fig.26).

The final stem dry matter in 1993-94 is presented in Table 11. Significantly less SBDM was observed with the IW treatment compared with all other treatments which were similar.

In 1994-95, significantly less SBDM was observed with IW treatment. The SBDM in the NW and N treatments was similar and differences between CW, I and C were not significant (Table 11). In the second year, the SBDM was slightly more than in 1993-94.

4.5.4 Pod dry matter

The pod dry matter (PDM), recorded at fortnightly intervals in 1993-94, increased slowly up to 150 DAS (Fig.27). From 150 to 165 DAS the increase in PDM was rapid

Table 11: Final dry matter of leaves, stems, pods and seed dry matter of chickpea under different treatments

Plant character	IW	NW	CW	I	N	C	CD
			1993-94				
*Leaf dry matter(g pl^{-1})	10.1	9.2	9.4	9.1	9.2	10.1	NS
Stem dry matter(g pl^{-1})	7.4	10.3	10.9	10.9	10.7	10.8	1.7
Pod dry matter(g pl^{-1})	5.6	11.0	10.8	12.1	14.1	13.7	5.0
Seed dry matter(g pl^{-1})	2.5	7.7	7.4	8.7	10.5	8.7	2.9
			1994-95				
*Leaf dry matter(g pl^{-1})	6.2	5.1	5.3	5.7	5.0	5.4	NS
Stem dry matter(g pl^{-1})	6.9	12.6	16.4	15.7	12.2	15.3	3.0
Pod dry matter(g pl^{-1})	2.2	16.2	15.0	15.1	14.1	17.7	5.6
Seed dry matter(g pl^{-1})	1.4	12.7	11.3	11.5	10.7	14.5	4.4

IW = Inoculated plots with additional wetness
NW = Naturally infected plots with additional wetness
CW = Chemically controlled plots with additional wetness
I = Inoculated plots under natural conditions
N = Naturally infected plots under natural conditions C = Chemically controlled plots under natural conditions
*At maximum LAI

in all the treatments with the sole exception of IW, where the gradual increase in PDM continued. After 165 DAS a slow rate of growth was observed in PDM in all treatments.

The PDM recorded at fortnightly intervals in 1994-95 increased gradually in all treatments up to 150 days (Fig.28). Subsequently it remained constant in IW, but there was a steep increase up to 165 DAS in all the other treatments which became more gradual from 165 to 180 DAS.

In 1993-94, the PDM at harvest in IW treatment was significantly less than all the other treatments. These were similar to each other. A similar trend was observed in 1994-95 for PDM (Table 11).

4.5.5 Seed dry matter

The seed dry weight (SDM) recorded at fortnightly intervals in 1993-94 increased gradually from 135 to 150 DAS in all treatments. In the case of IW, there was a gradual increase in SDM from 150 to 165 DAS followed by a plateau. In all the other treatments there was a fast rate of growth up to 165 DAS. Thereafter the increase was more gradual in all these treatments except N, where the fast rate of growth continued up to 180 DAS (Fig.29).

The SDM recorded at fortnightly intervals in 1994-95 increased gradually up to 150 DAS (Fig.30). After this the SDM in IW remained constant whereas it showed a steep increase up to 165 DAS in the rest of the treatments. After 165 DAS the SDM increased slowly in N, CW and I but continuously at a fast rate of growth in

NW and C.

In 1993-94, the final SDM in the IW treatment was significantly less than all the other treatments. The remaining treatments showed a non-significant difference with each other in seed dry matter at harvest. A similar trend was observed in 1994-95, but SDM was more than that in 1993-94 (Table 11).

4.5.6 Seed yield

In 1993-94, the seed yield in the IW treatment was significantly less than all other treatments (Table 12). Of the remaining treatments, the seed yield of NW was significantly less than the rest. The seed yields of all other treatments were similar.

In 1994-95, the seed yield of the IW treatment was significantly less than all other treatments (Table 12). The seed yields of NW and CW treatments were similar and N and C were also similar. The seed yield of N and C were significantly greater than those of NW and CW. The seed yield of the I treatment was significantly less than the yields of NW, CW, N and C. Ascochyta blight (DIS) caused an exponential [$Y = \exp(7.527 - 0.0189*DIS)$, $r = 0.97$] loss of chickpea seed yield in the field.

Table 12: Final seed yield, total dry matter and harvest index of chickpea under different treatments

Plant character	IW	NW	CW	I	N	C	CD
			1993-94				
Seed yield(kg ha^{-1})	431.1	1473.5	1662.5	1925.8	1803.0	1853.6	321.6
Total dry matter(kg ha^{-1})	2870	4791	4409	4444	5186	5030	282.4
Harvest Index	0.15	0.31	0.38	0.43	0.36	0.39	0.09
			1994-95				
Seed yield(kg ha^{-1})	306.7	1643.4	1715.2	1391.1	1936.2	1919.5	174
Total dry matter(kg ha^{-1})	3403	5248	4542	4942	4629	4799	756
Harvest Index	0.1	0.3	0.38	0.28	0.42	0.40	0.06

IW = Inoculated plots with additional wetness
NW = Naturally infected plots with additional wetness
CW = Chemically controlled plots with additional wetness
I = Inoculated plots under natural conditions
N = Naturally infected plots under natural conditions
C = Chemically controlled plots under natural conditions

4.5.7 Total dry matter

In 1993-94, the total dry matter for IW treatment was significantly less than each of the other treatments (Table 12). The dry matter of CW and I were similar to each other. The values for N and C were also similar to each other. A significant difference was observed between NW and C.

In 1994-95 the total dry matter was similar in all the treatments except IW where it was significantly less than all the other treatments (Table 12).

CHAPTER-V

DISCUSSION

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In this chapter, the results of the effect of meteorological variables on Ascochyta blight of chickpea evaluated empirically, in controlled environment studies and in crop field experiments described in chapter IV are discussed.

5.1 EMPIRICAL STUDY

5.2 LABORATORY EXPERIMENTS

5.3 FIELD EXPERIMENTS

5.1 EMPIRICAL STUDY

The results of the crop - weather - disease interaction study showed that the crop growth season of chickpea can be divided in two parts which can be referred to as non-risky and risky periods. This is based on weekly relationships between the disease index and each of seven weather variables.

5.1.1 Non-Risky Period

Phase I

During the active vegetative phase *i.e.* Standard Meteorological Weeks (SMWs) 1 to 8 (from 1st January to 25th February), no significant relationship was found with six of the weather variables. There was a significant correlation with rh in SMW 6 to 8 (from 5th to 25th February). This result indicates that this period is not risky for the

development of an epidemic. This may be because the temperature during this period remained between 4.5 to 21.9°C with a mean of 13.2°C, so for most of the time, the temperature remained below optimum for germination of pycnidiospores and growth of the fungus as reported by Bedi and Aujla, 1970 and Singh, 1984. The resulting slow rate of growth and development of the pathogen would increase the length of the incubation and latent periods. In these conditions the disease would develop slowly and delay the secondary disease cycle, so maintaining a non-epidemic condition. This view is supported by Hassani (1981), Reddy and Singh (1990), and Trapero-Cassas and Kaiser (1992).

Phase II

During maturity phase *i.e.* SMW 15 to 17 from 9th to 29th April, non-significant relationships between disease index and each of the seven weather variables very well depict this period as non-risky. This may be because the temperature as well as rh during this period remained unfavourable for the growth of the pathogen and the pathogen - host interaction. The temperature was usually between 16.4 to 37.4°C with a mean value of 26.9°C, so for most of the time, the temperature remained above optimum range as defined by Bedi and Aujla (1970) and Singh (1984), and pycnidiospore germination, fungal growth and sporulation would have been adversely affected. The high temperature during this period resulted in a mean rh of 66 per cent, much below the optimum range defined by Singh *et al.* (1982). They showed that a rh above 85 per cent is prerequisite for the disease development. The combination of high temperature and low rh would result in shortening of leaf wetness period so spores would neither be released from

pycnidia nor germinate. The disease cycle would therefore be broken, as supported by Hassani (1981), Reddy and Singh (1990), Royle and Butler (1986) and Wiltzien and Kaack (1984).

5.1.2 Risky Period

During the reproductive phase of the crop which coincides with SMW 9 to 14 from 26th February to 8th March, out of the seven meteorological variables tested, there were continuous and significant relationships between disease index and two weather variables *viz.* maximum temperature and evening rh. The maximum temperature showed an inverse relationship whereas, the evening rh showed a positive relationship. These relationships indicate that during the active reproductive phase, the high temperatures restricted disease development, whereas, an increase in evening rh accelerated the disease development. The most favourable period for the development of Ascochyta blight was from SMW 9 to 14 because the temperature remained between 8.7 and 31.0°C with a mean value of 20°C. This would have been within the optimum range of temperature most of the time, since 20°C was most favourable for fungal growth , sporulation and pycnidiospore germination (Bedi and Aujla, 1970; Singh, 1984). A range of temperature from 10 to 30°C for infection and disease development has been defined by various researchers, and this prevailed during this period. Within this range, the incubation and latent periods would have been short and the formation of pycnidia with viable pycnidiospores would have further resulted in adequate sporulation, producing a large number of spores. The rate of disease development during the second infection cycle

would have been rapid (Kaiser, 1973; Weltzien and Kaack, 1984; Reddy and Singh, 1990 and Trapero-Cassas and Kaiser, 1992).

The weekly average of rh during this period ranged from 32 to 81 per cent, so it is likely that upper limit of rh within the crop canopy will be in the range of 95 per cent. This difference in rh would be partly dur to transpiration from the actively growing crop, and would prolong the congenial conditions for infection and disease development. This argument is supported by the report of Singh *et al.* (1982) that rh above 85 per cent is optimum for disease development.

The empirical study on the crop - weather - disease interaction has delineated two weather variables *viz.* maximum temperature and evening rh out of seven possible variables as significant regulators of the disease during the reproductive phase. Mavi *et al.* (1992), also identified congenial weather variables for other diseases using a similar approach. This gave a strong indication that the combined effect of both the variables may further be explored for a more refined analysis.

On the basis of this hypothesis, these two weather variables were combined together by dividing evening rh with corresponding values of maximum temperature to give a humid thermal ratio (HTR) which showed a highly significant correlation (r=0.9) with the disease index. A similar approach has been used by Jhorar *et. al.* (1992) using two variables for karnal bunt of wheat.

Taking into consideration the close relationships of disease index with maximum temperature, evening rh and the humid thermal ratio, best fit functions were selected to use them as disease predictive models.

In case of maximum temperature, a linear inverse relationship was observed with disease index (DI) which explained 72 per cent of the variation in the disease. The relationship clearly indicates that 23 to 26°C is the most favourable weekly mean maximum temperature for development of the disease in epidemics. The role of rh, however, is not explained here. These results support the findings of Trapero-Casas and Kaiser (1992).

The asymptotic regression was selected as an appropriate function for evening rh. This explained 85 per cent of the variation in disease index and it illustrated that disease epidemics did not occur when the evening rh remained below 40 per cent. When evening rh exceed 40 per cent, it is likely that the periods of favourable rh would increase, leading to prolonged leaf wetness from early evening to late morning. This would accelerate the disease development provided the temperature was favourable to activate the pathogen. Such conditions can only be observed from the early evening to late morning or on cloudy and rainy days. On the other hand it is common for the rh to remain high during cooler parts of the day when temperature is unfavourable (low), resulting in little scope for disease development.

The combination of both evening rh and maximum temperature in the HTR brought more refinement to the results when fitted as a quadratic function. This explained 93 per cent of the variation in disease. An increase in DI was observed with increasing HTR values up to 2.3, when it reached a peak followed by a decrease in DI, indicating that the disease favouring HTR ranges from 1.3 to 3.3. Below this range, low rh coupled with high temperature proved to be unfavourable for disease development. On the other

hand when HTR was greater than 3.0, low temperature alone was the limiting factor for disease development (Fig.7).

In order of goodness of fit, the function fitted to HTR was followed by that for evening rh and lastly maximum temperature. These functions can be used as predictive models.

As evident from empirical study, temperature and moisture are the two most important factors which influences Ascochyta blight. This finding provide strong evidence to initiate a controlled environment study on these aspects.

5.2 LABORATORY EXPERIMENTS

Experiments were conducted in controlled environments at a constant temperature of 20°C to investigate the effects of leaf wetness duration, dryness duration, rh and light and darkness on various components of the disease cycle and on the overall development of Ascochyta blight. The information on the effects of temperatures on the disease were already available in sufficient details in literature. Studies on this aspect, therefore, was not taken up.

Spore germination was studied by giving different treatments of wetness and observations were recorded 42 h after applying the treatments. The results indicated that only 0.7 per cent of the spores germinated with 2 h of wetness and no infection resulted. The percentage of germinated spores, however, increased with increasing wetness duration indicating that spore germination requires a certain minimum threshold of wet period to cause infection. The germination percentage differed significantly among all the

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treatments. This indicated that some of the pycnidiospores require longer wetness to produce germ-tube. Spore penetration (0.3 %) was observed only at and above 4 h wetness indicating the minimum threshold for disease. The spore penetration, however, increased with increasing wetness duration showing that longer wetness favours disease development by maintaining the viability of germ tubes longer to increase the chances of penetration with a favourable temperature (Pandey and Chaube, 1986).

The effect of leaf wetness duration on infection was studied in controlled conditions. Infection did not occur with 2 h wetness, however, the disease appeared in traces with 3 h but the difference from 0 to 3 h was non-significant. With 4 h wetness, however, there was a marked increase in disease and it differed significantly from that with 2 h and 3 h wetness, indicating that the minimum requirement of wetness for significant infection is 4 h at 20°C. When the period of wetness increased from 4 to 6 h, a significant increase in the disease was observed indicating that a continuous and longer duration of wetness favours the disease. Traparo-Casas and Kaiser (1992) and Weiltz and Kaak (1984), however, reported that a minimum period of 6 h is required to cause significant infection. The results presented here, however, are supported with the generalised statements of Huber and Gillespi (1992) and Royle and Butler (1986) for other diseases. The difference between 18 h and 24 h remained non- significant showing that, for a single disease cycle, a wetness period of 18 h may be sufficient for maximum infection. It may also be concluded from the results that when a sufficient wetness period has already been provided, the disease may not show variation with subsequent short period fluctuations in wet and dry periods. The study, however, needs further investigation

to establish differences for wetness duration (if any) beyond 24 h because wetness beyond 24 h could cause significantly more disease than that with 18 h.

The chickpea plants inoculated and dried immediately under a fan were exposed to different humidity levels. The effect of continuously high rh for 48 h on these plants in the humidity chambers indicated that no infection occurred when rh was less then 95 per cent. However, at 95 per cent rh, the disease appeared in traces (0.8 %) and at 98 per cent it reached 24.8 per cent. This was significantly different from the other treatments. At 99 per cent rh, the disease further increased to 58.4 per cent and it was highest at 100 per cent rh, reaching 75.2 per cent. These results suggest that rh above 95 per cent has an exponential effect on the disease indicating that the moisture requirement of the pathogen is fulfilled even in the absence of a free film of water. Such conditions generally occur when nights are cloudy. The result supports the studies conducted in controlled conditions by Hassani (1981) which revealed that spores germinated at 98 per cent rh and increase rapidly at 100 per cent rh. Arauz and Sutton (1989) also thought that the pathogen may germinate in the presence of high humidity even without wetness. The studies conducted by Reddy and Singh (1990), however, included a wide range of humidity and did not specify the favourable range. Singh *et al.* (1982) reported that wet inoculated plants (without drying the plant surface) when exposed to rh > 85 per cent, the disease development was significantly high. This would have been possible if wetness on the plant surface persisted for a sufficient period to allow infection. This view was supported by the wetness study where significant disease developed with 4 h wetness. The effect of longer than 48 h of rh on the development of disease with an initial wet plant

surface, however, needs further investigation.

When the plants were exposed to different periods of dryness immediately after inoculation, the disease intensity was reduced at a fast rate initially (from 0 to 2 h dryness) with a reduced effect as the dryness duration increased (from 2 h to 8 h dryness). This may be caused by the germination and penetration of the pathogen being restricted due to the break in continuous wetness. When pycnidiospores are exposed to dry conditions after inoculation, they may loose viability due to desiccation affecting the infection process adversely. This result contradicts the findings of Traparo-Casas and Kaiser (1992). They reported that a dryness period of 6 h immediately after inoculation followed by 24 h wetness, resulted in a substantial increase in disease.

The study pertaining to effect of light and dark on infection indicated that continuously dark conditions resulted significantly more disease than light conditions. This may be due to the plant becoming predisposed in the dark and pathogen penetrating more aggressively through the cuticle. These hypotheses are supported by studies conducted by Benedict (1971) which revealed that with the disease caused by *Puccinia graminis* on barberry leaves, low light conditions induced penetration through the cuticle, so increasing the disease. Pandey and Chaube (1986) reported penetrations by *A. rabiei* through the cuticle by forming appresoria and ignoring open stomata.

In a sporulation study, the effects of wetness, high rh and darkness were studied. Sporulation with >95 per cent rh was more than that with 90 per cent. This may be due to the availability of more moisture in the air (with > 95 % rh) resulting in the development of more number of pycnidia as well as spores per pycnidia. Royle and Butler

(1986) stated that the sporulation is commonly favoured by high rh. The number of pycnidia as well as pycnidiospores further increased in the dark when compared with light. This is in agreement with the findings of Chauhan and Sinha (1973), which revealed that darkness favours sporulation. These results, however, differ from reports given by Nene (1982) and Kaiser (1973) from laboratory studies on artificial media. They reported that continuous light favoured sporulation more than continuous dark. Maximum sporulation was recorded when continuous wet conditions were provided to the diseased plants. The number of pycnidia as well as the pycnidiospores increased rapidly indicating the multiplicative effect of wetness. Similar effects were reported by Hassani (1981).

The controlled environment studies supported the results of the empirical study and further provided the base to initiate the experiments in the field to validate these results in natural conditions.

5.3 FIELD EXPERIMENTS

In the field investigations, experiments were conducted for two years to evaluate the effect of meteorological elements on the disease. Artificial wetness was provided to the plants for varied intervals, in addition to natural wetness, for one week intervals beginning on 129 DAS in 1993-94 and on 119 DAS in 1994-95. These were the dates when plants were inoculated artificially.

The results indicated that in both the years comparatively high disease intensity (30 % in 1993-94 and 17 % in 1994-95) was recorded in the IW treatment, where congenial environmental conditions were provided by maintaining continuous leaf wetness

for a week. The disease either appeared in traces or it failed to appear without inoculation, in the NW (naturally infected and wet) and CW (chemically protected and wet) treatments in both the years. In the plots with natural wetness the disease reached 8 per cent, in the inoculated treatment (I) whereas, in the naturally infected (N) and chemically protected (C) plots, the disease either remained nil or appeared in traces. The higher disease in the IW plots resulted from the availability of free moisture for sufficient time for infection, as on every day an additional eight hours of wetness was supplied to this treatment. In addition a virulent pathogen was provided, so germination and penetration would have proceeded with little restriction. Similar effects have been reported for the pathogen under laboratory conditions by Hassani (1981), Nene (1984), Weiltz and Kaak (1984) and Trapero-Casas and Kaiser (1992) who found that long wetness periods permit maximum spore germination and further provide better chances for the pathogen to penetrate the host by extending the viability of germ-tubes. The year to year variation in the level of primary infection may be due to the variation in temperature during the experimental periods. The temperature during the week immediately after inoculations remained about 5°C higher in 1993-94 approaching the optimal temperature range, compared to 1994-95.

In the treatment where the plants were provided artificial wetness but not inoculated artificially, the disease did not appear in the first year and in the second year it appeared in only traces. Much more disease appeared in the IW treatment, where inoculum was supplied along with additional wetness whereas, in the NW treatment, where additional wetness was supplied but inoculum was not supplied artificially, the

disease either was absent or appeared in traces in 1993-94. These results provide strong evidence that inoculum was a limiting factor in NW. This finding further provides a basis to explore the possibility of inoculum availability in the nature to cause epidemics. In the literature, however, researchers differ in their opinions regarding the availability of inoculum in the nature (Singh and Sharma, 1995). In the case of chemically protected plants with conditions congenial to disease (CW), the disease did not appear in the first year, and in the second year, it appeared only in traces confirming the effectiveness of the chemical spray. However the results from C, CW, N, NW were similar indicating that application of chemical spray in non-epidemic years does not give any economic benefit. This finding provides a strong base to develop a suitable forecasting system to make judicious use of chemicals while minimising adverse effect on the ecosystem. In 1993-94, more disease in the I treatment than in N appeared due to high humidity and/or wet conditions together with congenial temperature (higher by about 5°C) prevailing immediately after inoculation of the plots under natural conditions. In 1994-95, however, the disease did not appear in the I treatment, because the natural conditions were not congenial for infection immediately after inoculation (Fig. 1 to 4).

In the second infection cycle there was high disease intensity in IW treatment in both years probably due to the multiplicative effect of wetness and initial inoculum. The maximum disease in 1993-94 was 80 per cent compared with 89 per cent in 1994-95, possibly because the temperature in 1993-94 was around 22 to 23°C, and around 20°C in 1994-95. This might have played a significant role in establishing higher terminal disease in the second year. The results are in common with the findings of Wiltzin and

Kaak (1984). In 1993-94, only slight disease (1.1 %) developed in NW treatment compared with 7.5 per cent in 1994-95. This difference and a similar difference between years for the N treatment, indicate that significantly more disease developed in the second year due to the availability of inoculum and a favourable environment. Further spread of the disease was checked by an increasing air temperature associated with short periods of wetness and high humidity. The results support the views expressed by Reddy and Singh (1990) according to which high humidity and long wetness periods with favourable temperature would result in high infection and ultimately high disease intensity, but when the conditions are unfavourable, the disease progress is checked.

In an experiment conducted to study the effect of variable wetness, the results indicated that when continuous wetness in the field is prolonged, disease intensity increased significantly. This would have been caused by the infection process continuing for longer with prolonged wetness allowing more spores to germinate and penetrate the host, and increase the amount of disease. These results support the findings of the controlled environment studies discussed earlier and also the laboratory studies of Traparo-Casas and Kaiser (1992). Nene, (1984) also expressed similar views.

In the potted plant experiment, the per cent disease intensity remained comparatively high in the first, second, fifth and sixth exposure periods due to favourable moisture conditions with the evening rh remaining more than 40 per cent. However, the incubation periods were longer in first and second exposure periods because the temperature remained low *ie.* below 20°C (maximum temperature). The most favourable week for disease development was 5 th exposure period when the maximum disease

intensity of all the sets coincided with a seven day incubation period. The maximum temperature was 24°C and the minimum temperature was 10°C, The rh remained at 95 per cent for about 40 hours and rainfall occurred. With subsequent sets of inoculated plants, the disease remained comparatively low with a slight increase to seven per cent only in the 11th exposure period. The disease intensity remained low in these exposure periods because of unfavourable temperature and moisture conditions (Appandix-II) with slightly favourable moisture and temperature conditions during the 11th exposure period. The disease intensity during the weeks with high rh and wetness remained higher because the moisture requirement of pathogen for infection was fulfilled (partly). The empirical and controlled environment studies fully support these results. Also the views expressed by Reddy and Singh (1990), Kaiser (1973), Nene (1982), Weltzien and Kaak (1984) and Traparo-Casas and Kaiser (1992) support these findings.

The Incubation period decreased with time due to a seasonal rise in temperature. The inverse relationship between the incubation period and temperature is in accordance with the findings of controlled environment studies by Traparo-Casas and Kaiser (1992). The weeks with the highest disease intensity and the minimum incubation period are considered to be the most favourable periods for fast disease progress. These periods in the present investigation were from 13th to 20th February and from 27th March to 3rd April. A long period with these conditions might have lead to a disease epidemic.

The coefficients of correlation between disease on inoculated potted plants and

maximum, minimum and mean temperature in the field at Ludhiana showed a significant and inverse relationship with maximum temperature. With the average rh, there was a direct and highly significant relationship. These results indicate that the weather conditions in Punjab during the crop season are such that a reduction in maximum temperature and an increase in minimum rh (both representing day-time weather) result in high disease. There is also a strong, inverse relationship between the average temperature and the incubation period indicating that increased temperature reduces the incubation period. These results are supported by the findings of Traparo-Casas and Kaiser (1992). Short incubation periods have also been linked to severe disease and this is an area needing further investigations.

The coefficients of correlation between disease and maximum, minimum and mean rh indicated that rh is directly related to disease during the chickpea growing season. Maximum rh during first 1, 2 and 7 days, immediately after inoculation gave low coefficients of correlation but the effects of mean and minimum rh over the same period were greater. The coefficients of correlation (with mean rh) were 0.86, 0.85 and 0.76 respectively and minimum rh gave coefficients of correlation of 0.84, 0.84 and 0.74 respectively. These results indicate that the humidity is most important immediately after inoculation and its importance is slightly reduced with time.

The coefficients of correlation showed inverse relationships between the incubation period of the disease and maximum, minimum and mean temperatures at Ludhiana. The

maximum temperature in the first 1, 2 and 7 days after inoculation gave coefficients of correlation of -0.86, -0.85 and -0.90 respectively and the minimum temperature gave coefficients of correlation of -0.89, -0.86 and -0.79 respectively. The mean temperature in the first 1, 2 and 7 day after inoculation gave coefficients of correlation of -0.90, -0.89 and -0.87 respectively. These results indicate that the mean temperature throughout the week affect the incubation period inversely and is important to disease development.

Plant population, final plant height and maximum leaf area index (LAI) did not differ significantly between any of the treatments *viz.* IW, NW, CW, I, N and C. This indicates that the crop experienced similar environments in both the years and in all the treatments up to the date of inoculation *ie.* seed formation stage. This is evident from time taken to reach the seed formation stage (ranging from 121 to 129 DAS).

The time trend of different growth measurements during both crop seasons *ie.* leaf area index (LAI), leaf dry matter (LDM), stem and branches dry matter (SBDM), pod dry matter (PDM), seed dry matter (SDM), total biomass and yield were similar in all the treatments except for IW. In the IW treatment the conditions were congenial for the pathogen, leading to a disease epidemic which drastically reduced not only the source size but also dry matter accumulation in different plant parts such as leaves, stems, pods and seed.

The disease epidemic in the IW treatment shortened the grain filling period on one

hand, while on the other hand it significantly reduced the source size and sink capacity by reducing the pod dry matter. The reduced capacity to accumulate photosynthates was indicated through significant and large reductions in the dry matter of different plant parts including seed dry matter. This played a major role in significantly reducing seed yield in the IW treatment in both years.

CHAPTER-VI

SUMMARY AND CONCLUSIONS

Chickpea (Cicer arietimum L.) is an important food legume grown in ecologically diverse environments, where crop yields range widely. Large fluctuations in the productivity of chickpea commonly coincide with Ascochyta blight caused by *Ascochyta rabiei* (Pass). This disease is a major threat to chickpea production in several states of India, including the Punjab.

Ascochyta blight epidemics coincided with typical weather conditions shown by empirical relationships between the disease and meteorological variables. These relationships were investigated further in controlled environment and disease progress and its influence on the chickpea crop were investigated in the field.

In the empirical study, the growing season of chickpea was divided in risky and non-risky periods.

The vegetative phase of the crop (SMW 1 to 8) was non-risky. Coefficients of correlation between the disease and meteorological variables remained weak and non-significant, because temperature during this period was too low for germination of pycnidiospores and growth of the fungus.

The maturity phase (SMW 15 to 17) was also non-linear. The temperature and humidity remained unfavourable for growth and development of the pathogen and host-pathogen interaction. The host escape risk by attaining maturity before dominance of the

disease.

In the reproductive phase (SMW 9 to 14), variation in the maximum temperature and evening relative humidity gave strong relationships with the disease indicating that this period was risky.

The empirical study delineated maximum temperature and evening relative humidity out of seven weather variables as exerting most influence on disease development. These two variables were combined as the humid thermal ratio (HTR) and fitted to an appropriate function.

Maximum temperature during risky period gave a linear relationship with the disease index (DI = 5.17 -0.175 TMX), that explained 72 per cent of the variation in disease and indicated the range of 23 to 26°C as most favourable.

An asymptotic regression DI = 1- 30.94 * exp (-0.1*RHE) was the most appropriate function for evening relative humidity. This explained 85 per cent of the variation in disease and indicated that from SMW 9 to 14, an average of >40 per cent relative humidity during the day (in SMW 9 to 14), favoured disease development.

The combination of evening relative humidity and the corresponding maximum temperature in the form of HTR was fitted to a quadratic function (DI = -4.28 + 4.69*HTR - 1.05*HTR^2), which explained 93 per cent of the variation in disease. The disease appeared when the HTR ranged from 1.3 to 3.3 (2.3 ± 1) and reached maximum with an HTR value of 2.3.

The functions fitted with maximum temperature, evening relative humidity and the humid thermal ratio HTR can be used as tools to develop a disease forecasting model.

The findings of the empirical study provided sufficient evidence to initiate laboratory experiments to investigate the effects of temperature, relative humidity and leaf wetness on Ascochyta blight development.

Sufficient information on the effect of temperature on disease development is available in literature. Therefore, experiments in controlled environments were conducted at a constant temperature (either 20 or 24°C) to investigate the effect of moisture (relative humidity and wetness) on life cycle of *A. rabiei.*

Pycnidiospore germination and germ tube penetration increased linearly ($G = -0.40 + 0.855*WD$, $R^2 = 0.98$ and $P = -1.05 + 0.43*WD$, $R^2 = 0.91$) with wetness duration (WD). Spore germination (with 24 h wetness) started 12 h after inoculation and increased linearly ($G = -6.77 + 1.125*h$, $R^2 = 0.75$) with time up to 52 h (the end of the experiment).

The minimum wetness period of 4 h caused a little but significant disease and followed a non-linear (diminishing return) trend [$DI = 50 - 62.220155 \exp(-0.109329*WD)$, $R^2 = 0.97$].

Increaseing dryness duration (Dh) immediately after inoculation caused a linear decrease ($DI= 40.316 - 3.727*Dh$, $R^2 = 0.96$) in disease.

When the relative humidity aws less than 95 per cent, there was no infection following with wet inoculation with immediate drying of the plant surface. Relative humidity between 98 and 100 per cent, caused a steep and linear ($DI = -1015.33 +10.794*rh$, $R^2 = 0.85$) increase in the disease

Continuous darkness induced more infection in chickpea plants compared with

light probably due to predisposition of the plant enabling more penetration through the cuticle.

Maximum sporulation was found with continuous wetness which followed a non-linear increase {Py = 1200 - 1636.146 exp(-0.5468 WD), R^2 = 0.95} and {S = $6*10^8$ - $7.43*10^8$ exp(-0.467406*WD), R^2 = 0.98} with increasing wetness duration.

The results of the empirical study, validated with controlled environment experiments, provided a basis for field experiments to confirm the relationships between the disease with temperature and moisture (relative humidity and wetness).

In the field, the disease increased linearly with wetness duration in first and second infection cycles.

In un-inoculated plots Ascochyta blight either was absent or appeared only in traces, indicating inoculum was a major constraints to disease highlighted a need to initiate further investigation on this aspect.

Non-significant differences between C, CW, N and NW inferred that chemical spray in years with unfavourable weather for Ascochyta blight was uneconomical. The combined knowledge of inoculum load and anticipated weather would therefore minimize losses caused by additional (chemical) inputs.

High relative humidity favoured disease development in the field and could be used as good indicator for disease prediction.

The incubation period of *A. rabiei* was greatly influenced by temperature, permitting prediction of the appearance of disease symptoms.

Plant population, plant height and maximum LAI were not affected significantly, when the disease appeared in March but the disease drastically reduced yield of chickpea seed { Y=Exp (7.527-0.0189 DIS), r = -0.97}.

All three components of present investigations fully support each other. The results provide sufficient information to identify infection periods on the basis of prevailing weather and inoculum availability. This information can contribute to the development of a disease simulation model which could be used for weather-based advise for farmers to take timely control measures to check Ascochyta blight development.

Conclusions:

All three components of present investigation *ie* empirical, controlled environment and field studies highlighted the following conditions for Ascochyta blight development:

1. Availability of inoculum plays an important role in natural infection. It help to determine the epidemic and would facilitate the issue of disease forecasts.

2. The pathogen multiplies in the range fom 10 to 30°C and its development is most rapid between 15 and 25°C.

3. The organism is very responsive to rh and leaf wetness.

4. Moisture conditions immediately after inoculation are most important to the establishment of the pathogen.

5. Infection can only occur when the rh within the plant canopy is >95 percent. When the weekly mean of evening rh during months of March exceeds 40 per cent, disease epidemics are common.

6. At the optimum temperature, leaf wetness of at least 4 h is required for infection by *A. rabiei*. Prolonged wetness up to 24 h at the optimum temperature caused an asymptotic increase in a single infection cycle of the disease.

7. Once infection has taken place, temperature play a major role in disease development by affecting the incubation and latent periods.

8. Sporulation and release of pycnidiospores is maximum in wet conditions.

9. Ascochyta blight hampers the growth and development of plants and destroys developed plant parts leading to a total crop loss, in epidemics. The seed yield is reduced exponentially with increasing Ascochyta blight.

These findings provide a good base to identify infection periods from prevailing weather variables. Daily leaf wetness periods and temperature in the presence of sufficient

inoculum enable infection indices to be worked out. On the basis of these indices, a risk of Ascochyta blight can be assessed before the appearance of symptoms. The extent of sporulation can also be estimated to predict potential inoculum for successive disease cycles. The forecast values of temperature, rh and wetness can be estimated by analysis of synoptic weather charts and cloud pictures. An eastward track of western disturbances and deep upper layer troughs is kept under observation, as soon as they appear around 30°E. This system would give weather over north-western India (around 75°E and 30°N) about a week in advance which is sufficient lead time to predict work out favourable or unfavourable conditions for Ascochyta blight and to issue a weather-based advisory to farmers for efficient and judicious application of fungitoxicants as part of a disease management system for increased and sustainable chickpea production.

LITERATURE CITED

Anonymus, 1994. FAO production yearbook. Roam, Italy: FAO

Arauz,L.F. and Sutton, T.B. 1989. Influence of temperature and moisture on germination of ascospores and conidia of *Botryosphaeria obtusa*. *Phytopathology*. **79:** 667-74

Askerov,I.B. 1968. Ascochytosis of chickpea. *Zaschita rastenii ot Vredifeleii Baloznei, B,* 52-53 (In: *Rev. Ppl. Mycol.***47:** 369)

Bedi,P.S. and Aujla, S.S. 1970. Factors affecting the mycelial growth and the size of pycnidia produced *Phylosticta rabiei* (Pass.) Tort. incitant of gram blight in the Punjab. *J. Res.* (PAU), **4:** 606-09

Biscoe,P.V. and Gillagher, J.N. 1977. Weather dry matter production and yield. In: *Environmental Effects on Crop Physiology* (ed. Landsberg,J.J. and Cutting, C.V.), London, Academic

Butler,D.R. 1992. Daily pattern of dew-point temperature in a semi-arid climate. *Agric.Forest Meteorology* **60:** 267-78

Butler,D.R. 1993. How important is crop microclimate in chickpea Botrytis graymold. In: Recent advances in Botrytis graymold of chickpea: Summary proceedings of second working group meeting to disease collaborative research on Botrytis graymold of chickpea, 14-17 March, 1993, Rampur, Nepal (ed. Hawre,M.P., Gowda,C.L.L. and McDonald,D.), ICRISAT, Patancheru, A.P., 502 324, India

Butler,D.R., Reddy,R.K. and Wadia,K.D.R. 1995. Single plant chambers to control humidity for pathological studies. *Plant Pathology,* **44 (1):** 1-9

Chauhan,R.K.S. and Sinha,S. 1973. Effect of varying temperature, humidity and light during incubation in relation to disease development in blight of gram (*Cicer arietinum*) caused by *Ascochyta rabiei*. Proc. National Science Academy, India, B, **37:** 473-82

Coakley,S.M. 1988. Variation in climate and prediction of disease in plants. *Ann. Rev.* Phytopathol. **26:** 163-81

Colhun,J. 1973. Effect of environmental factors on plant disease. *Ann. Rev. Phytopathol.* **11:** 343-364

Dey,S.K. 1990. Basis of resistance to Ascochyta blight in chickpea (*Cicer arietinum* L.). Ph.D. Thesis, Punjab Agric. Univ., Ludhiana, pp. 180

Diekmann,M. 1992. Use of climatic parameters to predict the global distribution of Ascochyta blight on chickpea. *Plant Dis.* **76 (4):** 409-12

Edwards,D.A., Doley,D. and Rimmington,G.M. 1986. Plant development, pages 155-170. In: Modelling Plant growth and Development. Academic Press, Australia, pp. 235

Ellis,R.H., Lawn,R.J., Summerfield R.J., Qi,A., Roberts,E.H., Chay,P.M., Brouwer,J.B. Rose,J.L.,Yeates,S.J. and Sandover,S. 1994. Towards reliable prediction of time to flowering in six annual crops v. chickpea (*Cicer arietinum*). *Expt.Agric.* **30 (3):** 271-82

*Eshal,Y. 1967. Effect of sowing date on growth and seed yield components of chickpea (*Cicer arietinum* L.). *Israel J. of Agric. Res.* **17:**1993-97

Fisher,R.A. and Yates,F. 1963. Statistical Tables for Biological, Agricultural and Medical Research. Hafner Publ. Co. Inc., New York, pp: 146

Fitt,B.D.L. and Bainbridge,A. 1983. Recovery of *Pseudocenosforalla hepotrichodies* spores from rain splashed samples. *Phytopathology.* **106:** 177-82

Gill,D.S., Brar,H.S., Verma,M.M. and Bains, T.S. 1988a. Physiological evaluation of chickpea genotypes for temperature tolerance. *Crop Improvement.* **20 (1):** 101-10

Gill,D.S., Verma,M.M. and Brar,H.S., 1988b. Physiological response of chickpea (*Cicer arietinum* L.) genotypes under high inputs in cooler environments of northern India. *Indian. J. of Plant physiology.* **31 (4):** 413-17

Hassani,A.A.E. 1981. Modalities d'expression de la resistance d'um cultiver de pois chiche (*Cicer arietinum* L.) a *Ascochyta rabiei* (Pass.)Lab. University de Rennes I. France. A Ph.D. Thesis. pp. 41

Hawtin,G.C. and Singh,K.B. 1984. Prospectus and potential of winter sowing of chickpea in the Mediterranean region. Pages 7-16. Proc.Workshop on Ascochyta blight and winter sowing of chickpea (ed. Saxena.M.C. and Singh,K.B.). Martinus Nijhoff/Dr. W.Junk Publ., Hague, The Netherlands

Huber,L. and Gillespie,T.J. 1992. Modelling leaf wetness in relation to plant disease epidemiology. *Ann.Rev. Phytopathol.* **30:** 553-77

Huda,A.K.S. and Virmani,S.M. 1987. Agroclimatic environment of chickpea and pigeonpea. Pages 15-21. In; Adaptation of chickpea and Pigeonpea to Abiotic Stresses. Proceedings of Consultants' Workshop, 19-21 Dec. 1984, ICRISAT, Patancheru, A.P. 502 324, India

Jagdish Kumar, 1990. chickpea research at ICRISAT as related to Asia. Pages 53-56. In Enhancing Grain Legumes Research in India. Summary proceedings of the Asian Grain Legume Network Coordinators' Meeting, 10-12 Dec. 1990, ICRISAT, Patancheru, A.P. 502 324, India

Jhorar,O.P., Mavi,H.S., Sharma,I., Mahi,G.S., Mathauda,S.S. and Singh,G. 1992. A biometeorological model for forecasting karnal bunt disease of wheat. *Pl. Dis. Res.* **7 (2):** 204-209

Jodha,H.S. and SubaRao,K.V. 1987. Chickpea: World importance and distribution. Pages 1-10. In: The Chickpea. (Ed. Saxena,M.C. and Singh,K.B.), Wallingford, Oxon, U.K.: CAB International

Kaiser,W.J. 1973. Factors affecting growth sporulation pathogenicity and survival of *Ascochyta rabiei*. *Mycologia*. **65:** 444-57

Kaiser,W.J.1992. Epidemiology of *Ascochyta rabiei*. Pages 117-134. In: Disease resistance breeding in chickpea. Proceedings of consultative meeting on breeding for disease resistance in Kabuli chickpea, 6-8 March, 1989, ICARDA, Aleppo, Syria

Kaiser,W.J. and Muehlbauer,F.J. 1988. An outbreak of Ascochyta blight of chickpea in the Pacific North West USA, in 1987. *Int. Chickpea Newsletter*, **18:** 16-17

Kauser, A.G. 1965. Epiphytology of recent epiphytotics of gram blight in West Pakistan. *Pak. J. Agric. Sci.* **11:** 185-195

Luthra,J.C., Sattar,A. and Bedi,K.S. 1935. Life history of gram blight [*Ascochyta rabiei* (Pass.) Lab. = *Phylosticta rabiei* (Pass.) Tort., on *Cicer arietinum*] and its control in the Punjab. Punjab Agric. and Live-Stock, India, **5:** 489-98

Malik,B.A. and Tufail,M. 1984. Chickpea production in pakistan. Pages 229-236. Proc. of workshop on Ascochyta blight and winter sowing of chickpea (ed. Saxena,M.C. and Singh,K.B.), ICARDA, Aleppo, Syria

Mavi,H.S., Jhorar,O.P., Sharma,I., Singh,G., Mahi,G.S., Mathauda,S.S., Aujla,S.S. 1992. Forecasting karnal bunt disease of wheat - A biometeorological method. *Cer. Res. Comm.* **20:** 67-74

Murinda, M.V. and Saxena,M.C. 1983. Agronomy of faba beans, lentils and chickpeas. Pages 229-244. In: Faba beans, kabli chickpeas and lentils in the 1980s. Proceedings of International workshop on faba beans kabli chickpeas and lentils in the 1980s. (ed. Saxena,M.C. and Verma,S.). 16-20 May, 1983, ICARDA, Aleppo, Syria

Nene,Y.L.1984. A review of Ascochyta blight of chickpea (*C. arietinum* L.). Pages 17-37. In: Proceedings of Workshop on Ascochyta blight and winter sowing of chickpea (ed. Saxena.M.C. and Singh,K.B.). Martinus Nijhoff/Dr. W.Junk Publ., Hague, The Netherlands

Ooka,J.J and Kommedahl,T. 1977. Wind and rain dispersal of *Fusarium moniliforme* in corn fields. *Phytopathology*. **67:** 1023-26

Panse,V.G. and Sukhatme, P.V. 1978. Statistical methods for agricultural workers (revised by Sukhtme,P.V. and Amble,V.N.) ICAR, New Delhi, pp. 381

Prabhakar,M. and Saraf,C.S. 1990. Dry matter accumulation and distribution in chickpea (*Cicer arietinum*) as influenced by genotype phosphorus source and irrigation level. *Indian. J. of Agric. Sci.* **60 (3)** :204-06

Pundir,R.P.S., Reddy,K.N. and Mengesha,M.H. 1988. Chickpea germplasm catalog : Evaluation and analysis. ICRISAT, Patancheru, A.P. 502 324, India

Raffray,J.B. and Sequeira,L. 1971. Dark induction of sporulation in *Bremia lactucae*. *Can. J. Bot.* **49:** 237-39

Ram Singh and Mahender Pal, 1993. Survival of *Ascochyta rabiei* in infected plant debris at different temperatures and soil depths. *Indian Phytopathology*. **46 (4):** 406-08

Reddy,M.V., Nene,Y.L., Gurdip Singh and Bashir,M. 1991. Strategies for management of foliar diseases of chickpea. Pages 117-127. In: Chickpea in Nineties. Proceedings of second international workshop on chickpea improvement, 4-8 Dec. 1989, ICRISAT, Patancheru, A.P. 502 324, India

Reddy,M.V. and Singh,K.B. 1990. Relationship between temperature, relative humidity and Ascochyta blight development in winter sown chickpea in Syria. *Phytopathologica Mediterranea*. **29 (3):** 159-62

Roberts,E.H., Hadley,P. and Summerfield,R.J. 1985. Effect of temperature and photoperiod on flowering in chickpea (*Cicer atietinum* L.) *Annals of Botany*. **55:** 881-92

Roberts,E.H. and Summerfield,R.J. 1987. Measurement and prediction of flowering in annual crops. Pages 17-50. In: Manipulation of flowering. (ed. Atherton,J.G.), Cambridge: Cambridge Univ. Press

Royle,D.J. and Butler,D.R. 1986. Epidemiological significance of liquid water in crop canopies and its role in disease forecasting. Pages 139-156. In: Water fungi and plants. (ed. Ayres,P.G. and Boddy,L.), Cambridge : Cambridge Univ. Press

Saxena,M.C. 1983. Food legume improvement program at ICARDA - An overview. Pages 1-13. In: Faba beans, kabli chickpeas and lentils in the 1980s. Proceedings of International workshop on faba beans kabli chickpeas and lentils in the 1980s. (ed. Saxena,M.C. and Verma,S.). 16-20 May, 1983, ICARDA, Aleppo, Syria

*Sharma,S. and Sonakya,V.K. 1990. Impact of thermal and heliothermal requirements of chickpea cultivars on phenological changes and productivity. *Research and Development Reports.* **7 (1-2):** 41-46

Singh,G. 1987. Variability in *Ascochyta rabiei* (Pass.) Lab., the causal agent of chickpea blight. M.Sc. Thesis, PAU, Ludhiana, pp. 97

Singh,G. and Sharma,Y.R. 1995. Ascochyta blight of chickpea. In: International book series on IPM system on agriculture. (ed. Upadhyay,R.K. and Mukerji,K.G.), Oxford & IBH Publ.Co.Pvt.Ltd.,New Delhi (In press)

Singh,G., Singh,K. and Sashi-Kapoor, 1982. Screening for source of resistance of Ascochyta blight if chickpea. *International Chickpea Newsletter.* **6:** 15-17

Singh,M. 1984. Studies on Ascochyta blight of gram caused by *Ascochyta rabiei* (Pass.) Lab. M.Sc. thesis, PAU, Ludhiana, pp. 95

Sinha,S.K. 1977. Food legumes : Distribution, adaptability and biology of yield. FAO plant production and protection paper no. 3. Rome, Italy, FAO. pp. 124

Sinha,S.K. 1985. Morphological and physiological requirements of a productive plant of chickpea. Pages 209-218. In: Faba beans, kabli chickpeas and lentils in the 1980s. Proceedings of International workshop on faba beans kabli chickpeas and lentils in the 1980s. (ed. Saxena,M.C. and Verma,S.). 16-20 May, 1983, ICARDA, Aleppo, Syria

Summerfield,R.J., Minchin,F.R., Roberts,E.H. and Hadley,P. 1980. Effect of photoperiod and air temperature on growth and yield of chickpea (*Cicer arietinum* L.). Pages 121-149. In: Proceedings of chickpea improvement, 28 February - 2 March, 1979, ICRISAT, Patancheru, A.P. 502 324, India

Summerfield,R.J., Roberts,E.H. and Hadley,P. 1987. Photothermal effect on flowering in chickpea and other grain legumes. Pages 121-149. In: Adaptation of chickpea and pigeonpea to abiotic stress. Proceedings of the consultants' workshop, 19-21 Dec. 1984, ICRISAT, Patancheru, A.P. 502 324, India

Summerfield,R.J., Virmani,S.M., Roberts,E.H. and Ellis,R.H. 1991. Adaptation of chickpea to agroclimatic constraints. Pages 61-72. In: Chickpea in Nineties. Proceedings of second international workshop on chickpea improvement, 4-8 Dec. 1989, ICRISAT, Patancheru, A.P. 502 324, India

åSutton,J.C., Jems,T.D.W. and Rowel,P.M. 1984. BOTCAST - A forecaster for timing fungicides to control botrytis leaf blight of onion. University of Guelph circular no. EBO. 784, pp. 1-15

Sutton,T.B. and Jones,A.L. 1979. Analysis of factors affecting dispersal of *Padosphaera lancatricha* conidia. *Phytopathology*. **69:** 380-383

Trapero-Casas,A. and Jimenez-Dias, 1986. Influence of sowing date on Fusarium wilt and Ascochyta blight of chickpea in southern Spain. Int. Food Legume Conf. on Pea, Lentil, Faba bean and Chickpea, July 1986, Spokane Washington, USA, Idaho (Abst.)

Trapero-Casas,A., and Kaiser,W.J. 1992. Influence of temperature, wetness period, plant age and inoculum concentration on infection and development of Ascochyta blight of chickpea. *Phytopathology*. **82 (5):** 589-96

Trapero-Casas,A., and Kaiser,W.J. 1992. Development of *Didymella rabiei,* the teleomorph of *Ascochyta rabiei* on chickpea straw. *Phytopathology*. **82 (11):**1261-66

Vander Wal,A.F. 1978. Moisture as a factor in epidemiology and forecasting. In: Water deficit and plant growth: Water and plant disease, (ed. Kozlowski,T.T.), **5:**253-95, London, Academic

Verma,M.M., Singh,G., Sandhu,T.S., Brar,H.S., Sandhu,K. and Bhullar,K.S. 1981. Sources of resistance to gram blight and graymold. *International Chickpea Newsletter*. **4:** 14-16

Virmani,S.M., Faris,D.G. and Johnson,C. 1991. Agroclimatology of chickpea in India. In: Agroclimatology of Asian grain legumes chickpea, pigeonpea and groundnut. A research bulletin no. 14. pp.15-23

Weltzien,H.C. and Kaack,H.J. 1984. Epidemiological aspects of chickpea Ascochyta blight. Pages 35-44. In: Proceedings of Workshop on Ascochyta blight and winter sowing of chickpea (ed. Saxena.M.C. and Singh,K.B.). Martinus Nijhoff/Dr. W.Junk Publ., Hague, The Netherlands

*Zachoe,D.G., Panagopoulos,C.G. and Makris,S.A. 1963. Recherches sur la biologie, l'epidemiologie et al lutte contre l'anthracnose du pois-chiche. *Ann. Inst. Phytopathol. Benaki*, n.s. **5:** 167-92

*Original not seen

Appendix-I

Basis for the ratings of the disease in field

Scale Value	Class	Mid Value	Scale	Class	M i d
0	0	0	5	25.1-35	30.0
1	0.1-5	2.5	6	35.1-50	42.5
2	5.1-10	7.5	7	50.1-70	60.0
3	10.1-15	12.5	8	70.1-85	78.5
4	15.1-25	20.0	9	85.1-100	92.5

Appendix-II

Set #	Temperature (°C)		Relative humidity (%)	
	Minimum	Maximum	Evening	Morning
1	6.500	15.700	66.000	97.000
2	4.5000	18.100	46.000	96.000
3	4.7000	20.200	45.000	95.000
4	10.600	21.600	55.000	92.000
5	9.3000	20.400	61.000	95.000
6	8.2000	21.600	51.000	94.000
7	5.8000	21.000	37.000	91.000
8	7.3000	23.100	45.000	85.000
9	10.000	26.700	43.000	86.000
10	15.200	28.600	45.000	88.000
11	12.100	26.900	38.000	88.000
12	14.300	31.700	26.000	76.000
13	15.300	32.600	26.000	67.000